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EDITED BY MATTHEW HOTOPF

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Advising relatives of risk of Alzheimer’s disease

Liddell et al (2001) reviewed what knowledge we have on the genetic epidemiology of Alzheimer’s disease for the purpose of informing relatives of patients about their own risks. We read their review with interest; however, we disagree on several points.

First, in many cases of late-onset dementia, differentiating between the common causes of Alzheimer’s disease and vascular dementia is difficult. In everyday clinical practice even differentiating Alzheimer’s disease from Lewy-body disease and fronto-temporal dementia is not always feasible. To what extent these distinctions are relevant to genetic counseling with respect to late-onset dementia is not clear.

Second, the very high prevalence of dementia found in centenarians (Asada et al, 1996; Blansjaar et al, 2000) is not the only argument against a slowing down of the rate of increase in dementia over 85, 90 or 95 years of age. Meta-analyses, not included in the review, did not find evidence for such a slowing down (Gao et al, 1998; Jorm & Jolley, 1998). Therefore, the prevalence of dementia almost certainly increases substantially, exceeding 15% from the age of 85.

Most investigations attributed some three-quarters of late-onset dementia to Alzheimer’s disease. We agree that the literature indicates a three- to fourfold risk in first-degree relatives of patients with late-onset dementia (seven- to eightfold with two affected first-degree relatives). We can only conclude that this leads to a risk of one in three, if not higher, for those first-degree relatives who reach the age of 85 years. Obfuscating this information by showing graphs to anxious relatives is, in our opinion, not an appropriate reassurance. We feel that better consolation can be effected by proffering the view that most people do not reach the age of 85, and by explaining the slowly progressive course of most cases of late-onset dementia.


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Authors’ reply: For the most part, the points of disagreement that Drs Blansjaar and van Schaick raise are differences more of emphasis than of substance.

True, the diagnosis of the type of dementia, particularly in late old age, is not always easy. Dementia in later life is probably best described as a syndrome, the emergence of clinical dementia being dependent upon the interplay of two or more pathologies. The ‘Nun Study’ by Snowdon et al (1997) is probably one of the best demonstrations of this. Yet, it is believed that Alzheimer’s disease is a major cause of dementia in later life. Even without having seen the patient, one is going to be correct in a diagnosis of Alzheimer’s disease, or Alzheimer’s disease and cerebrovascular disease, 75% of the time. Rarer diagnoses, such as fronto-temporal dementia and Lewy-body disease, should suggest themselves if they are kept in mind, a careful history taken and the patient followed-up so that departures from the normal symptom progression for Alzheimer’s disease are noted. Of course, mistakes in diagnosis will occur, but we think that this will occur insufficiently frequently to compromise the very broad-brush approach to estimating the familial risk of dementia that we have advocated.

As to whether the rate of increase in the incidence and prevalence of dementia begins to slow or goes on increasing exponentially into extreme old age, this is a controversial area, which is, in fact, also highlighted in the two meta-analyses cited by Drs Blansjaar and van Schaick. Jorm & Jolley (1998) suggest that “the incidence rises exponentially up to the age of 90 years”. Gao et al (1998) suggest that “the acceleration of incidence rates for AD and dementia slows down with the increase in age, although we find no evidence of a rate decline”. Faced with such difficulties of interpretation, we can only commend the clarity of Blansjaar et al’s own study (2000), which suggests that the increase in dementia prevalence does not slow down in extreme old age.

We agree that the risk of a first-degree relative of a proband with Alzheimer’s disease developing the disorder once they reach the age of 85 may be one in three, if not higher. Perhaps this point could have been made more clearly in our review. The main point we tried to make was that the actual likelihood of surviving to age 85 and developing Alzheimer’s disease is lower. We disagree that showing graphs to anxious relatives is “obfuscating this information”, but we accept that Drs Blansjaar and van Schaick and, indeed, other clinicians may think differently.

In non-Mendelian Alzheimer’s disease it is difficult to estimate how much the risk increases as the number of affected first-degree relatives goes up, principally because few studies have addressed this issue. However, the ‘conjugal Alzheimer’s disease’ study of Bird et al (1993), which we cited, and the transmission study of Farrer et al (1990), which we did not cite, indicate that the risk increases substantially. With such pedigrees showing apparently high genetic loading for Alzheimer’s disease, we suggested that a psychiatrist seek the advice of a clinical geneticist.

Finally, we agree that it is often reassuring to point out that the course of dementia in late old age is usually more slowly progressive and more benign than dementia occurring in a younger person.
Treatment for Alzheimer’s disease in people with learning disabilities: NICE guidance

In January 2001 the National Institute for Clinical Excellence (NICE) published Guidance on the Use of Donepezil, Rivastigmine and Galantamine for the Treatment of Alzheimer’s Disease. The guidance indicates that the drugs should be made available within the National Health Service to people with mild to moderate Alzheimer’s disease whose minimal state examination (MMSE) score is above 12 points. The Institute’s guidance does not mention the use of anti-dementia drugs in people with learning disabilities and Alzheimer’s disease. Studies have shown that the prevalence of Alzheimer’s disease in those with learning disabilities is higher than in the normal population (Patel et al, 1993). This is likely to increase in the future because of the rising life expectancy of people with learning disabilities (Zigman et al, 1997). In Down’s syndrome, approximately 40% develop dementia of Alzheimer type by the age of 60 (Holland et al, 1998).

It is known that clinical evidence for the effectiveness of various psychiatric treatments in the learning disability population is scanty and specialists rely on evidence from the normal population. In this situation, a specialist in the psychiatry of learning disability might consider following the NICE guidance in treating dementia in the people under his or her care. However, there is a major problem, as NICE guidance suggests that treatment should be monitored by MMSE score but the MMSE cannot be used reliably in people with learning disabilities (Deb & Braganza, 1999). This means that NICE guidance on the use of anti-dementia drugs is not applicable to people with learning disabilities. This is likely to discourage specialists from prescribing treatment for some patients with a learning disability and Alzheimer’s disease who may benefit from it in future. In its guidance, NICE mentioned limitations on the use of the MMSE in people whose Alzheimer’s disease is complicated by dysphasia and whose first language is not English, but failed to identify that the MMSE is not standardised for people with learning disabilities who make up 2% of our population. The fact that this group of people, with a high prevalence of dementia, was completely ignored within the guidance is quite worrying. We appreciate that the guidance from NICE is not prescriptive and does not replace individual judgement; however, complete omission of learning disability could potentially exclude people from receiving beneficial treatment.


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Community care for mental disorders in developing countries: a perspective

Given the limitations of the existing model of community care for mental disorders in developing countries, Jacob (2001) has tried to construct another model and has focused on some of the constituent elements of such a model. Although Jacob insists on a potentially innovative approach to the provision of mental health services in developing countries, the framework within which to take forward the debate regarding community care fails to analyse in depth the sociopolitical and economic contexts in which community care is constructed. Owing to the strong emphasis placed upon discriminatory social and political structures, an analysis of what it is to be mentally ill, and the sociological and psychological implications of this, has largely been ignored.

I agree with Jacob that cooperation between governments and non-governmental organisations (NGOs) in providing community care will help in implementing health care policies. However, by their very nature, NGOs are heterogeneous and vary from large agencies operating in many countries (e.g. Oxfam, Save the Children Fund) to very small organisations operating at village level. Despite the growth of NGO activity in the past decade, there remain questions regarding their effectiveness in achieving their stated objectives (Nyoni, 1987). Evaluation of an NGO’s effectiveness can become something of a propaganda exercise, aimed more at impressing donor agencies than at a critical analysis of the NGO’s activities. A related issue concerns the mixed accountabilities of NGOs – ‘downwards’ to their collaborating partners and ‘upwards’ to their donor agencies. These issues result in difficulties of monitoring and enforcement (Brett, 1993).

We know that the lives of individuals with mental illnesses around the world are usually limited far more by prevailing social, cultural and economic constraints than by their illnesses. If this is the case, then the issues related to community care for people with mental disorders move from those of health to those of human rights. Their lives are hard indeed. Mental health professionals can help to change this state of affairs. Whether the issue is community care in urban London or in rural India, professionals who work on mental health...
health issues must extend their perspective beyond the bounds of policies and programmes if they are to help make a meaningful difference in the lives of those they serve.

This does not mean that policies and programmes are not needed – they clearly are. But the tendency to implement ready-made models could easily miss the needs of the target groups. Community care programmes have to be tied to broader social and economic policies and programmes. Mental health professionals can contribute their expertise to this process and work in conjunction with these people and their families to strengthen their voices in the arena of human rights.


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Mental health literacy in developing countries

The article on mental health literacy by Jorm (2000) was interesting and stimulating and we were encouraged to see reference to the mental health literature in developing countries. However, it portrayed a rather incomplete and negative view of the situation. It has rightly been pointed out that the beliefs in supernatural causes of mental disorders are more widely held and traditional sources of help, such as spiritual healers, are preferred over medical advice for a range of mental health problems in these countries. It was not mentioned, however, that mental health literacy is perhaps part of general literacy. In some developing countries where more than half of the population may be illiterate the dimensions of mental health literacy are totally different from those in Western countries. One corollary of this is that mental health professionals will have to adopt innovative approaches to mental health literacy.

The community mental health programme in Pakistan is an example. With a literacy rate of about 40%, it was not conceivable to adopt the approaches discussed by Jorm (2000). The innovative approaches adopted instead ranged from creating awareness at all levels of health personnel, including primary health care physicians, schoolchildren and teachers in the community, and collaborating with other sectors like traditional faith healers. One interesting attempt in this direction was to create awareness among schoolchildren and their teachers. We believe that the schoolchildren in rural areas are the eyes and ears of the community. In a study evaluating the effect of the school mental health programme it was shown that knowledge, attitudes and superstitions about mental health problems significantly improved in a group of schoolchildren, their friends and neighbours after implementation of the programme, compared with a control group (Rehman et al, 1998). More recently, the work has been extended to the detection of disabilities by children (Gater et al, 1999). This article, which gained the Barker Memorial Award, clearly showed the impact of improved mental health literacy through the strong messenger force of children in the rural community. An interesting impact of improved mental health literacy was its effect on general health indicators such as maternal mortality, infant mortality and the immunisation of children, which were significantly improved following integration of the mental health awareness campaign within primary health care (Maqsood et al, 2001). Similarly, educating the traditional faith healers assisted tremendously in identification and referral of people with mental illnesses for proper psychiatric treatment (Saeed et al, 2000). These are just a few examples of indigenous projects undertaken in the community, which highlight innovative approaches to mental health literacy in a developing country.

 Recently, the issue of the role of the World Health Organization in improving mental health literacy in developing countries has been debated (Mubashar & Saeed, 2000) and directions for developing cost-effective and sustainable mental health programmes have been outlined.


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Handedness and schizophrenia: genetic and environmental factors

I would like to comment on the conclusions reached by Sommer et al (2001) in their meta-analysis of the literature on cerebral lateralisation and anatomical asymmetry in schizophrenia.

The article opens with the claim that: “Right-handedness, left cerebral dominance for language and normal cerebral asymmetry are considered to be secondary to a dominant allele, the ‘right-shift factor’”. It must be kept in mind that this is only a hypothesis and has yet to be proven. Even if the centrality of this gene were demonstrated, the influence of environmental factors would still have to be accounted for. Otherwise, it would be impossible to explain the relatively common discordance for handedness in monozygotic twins: this discordance can only be due to disruption of the intrauterine neurodevelopment in one of the twins caused by the action of environmental factors. This is supported by Steinmetz et al (1995) in which magnetic resonance imaging of twins discordant for handedness showed that this discordance is mirrored by differences in brain lateralisation. Further confirmation of the importance of environmental factors in handedness comes from a Norwegian study by Salvesen et al (1993), in which a cohort of pregnant women were divided in two groups. Half of the mothers had real ultrasound scanning during pregnancy while the others had a sham investigation. When the children were examined after birth there was significant excess of left-handedness only in the group exposed to real ultrasonography.
An interesting study by Davis & Phelps (1995) showed that the concordance for schizophrenia in monozygotic twins discordant for handedness is much higher than in twins concordant for handedness (60 v. 32%). Again, this difference can be explained only by the presence of an environmental factor acting in utero, which disrupts the neurodevelopment causing schizophrenia and altering handedness. No genetic factor can explain it.

The evidence from the literature therefore directs towards an environmental factor, which acts during neurodevelopment disrupting handedness and predisposing for schizophrenia. This hypothesis is briefly considered in the article, but then dismissed because of the results of a meta-analysis. The authors compared patients suffering from schizophrenia with patients suffering from other neuropsychiatric disorders. Both groups showed an excess of left-handedness, but in the schizophrenia population the excess was significantly higher.

The authors reached the conclusion that this shows the involvement of a genetic mechanism, but this is only one of the possible explanations. Another possibility is that there are neuropsychiatric disorders that are not neurodevelopmental in origin. Another explanation is that in certain disorders the neurodevelopmental damage acts before or after the time when handedness is established.

It is therefore my opinion that the literature on handedness and schizophrenia, comprehensively reviewed by the authors, confirms that both genetic and environmental factors have to be accounted for in the aetiology of schizophrenia. The ‘right shift’ is still only a hypothesis and the meta-analysis by Sommer et al does not corroborate or refute it.


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**Stress management and schizophrenia**

Bellack et al (2000) have addressed the important question of delineating the core therapeutic components of carer-based stress management that account for the improved course of schizophrenic disorders found consistently when these methods have been integrated with optimal pharmacotherapy. Their conclusions deserve close scrutiny, particularly when they make strong statements about the relative cost-effectiveness of different approaches, without the benefits of any economic analysis. Their conclusion that cognitive–behavioural strategies aimed at enhancing the problem-solving of patients and their key caregivers do not contribute to the clinical benefits cannot be drawn definitively from the study they report.

This study is extremely complex and was not designed to evaluate the comparative effectiveness of the key components of cognitive–behavioural family interventions. All cases were offered 25 sessions of structured education in multi-family groups over 24 months. These educational groups aimed to assist patients and their caregivers in the management of their disorders and the stresses in their lives. However, half the sample was assigned at random to an additional 29 sessions of home-based education that employed active learning methods to enable the patient and carers to conduct weekly self-help sessions in the home. During these sessions they were expected to work on the personal problems and goals that they considered important. They were taught to use a problem-solving approach, with guide sheets to structure their discussions and to provide records of their plans. This home-based training ceased after 12 months, but the multi-family educational groups continued to 24 months.

From the outcome measures reported, there was no significant difference between the two stress management conditions in forestalling hospital admissions over the 24 months. Thus, the addition of the problem-solving training, which seldom requires more than 5 hours of teaching, was considered redundant. The method attributed to Falloon et al (1984) does not include the additional monthly educational groups used in this project, and it is probable that the combination of two approaches that emphasized somewhat different objectives may have proved confusing to some participants and excessive to others. We certainly observed that at times of crisis the therapists confused the two approaches. A definitive study that aimed to compare the benefits of the educational and problem-solving strategies would need to ensure that each approach was more clearly defined, and would have to control for the time participants were exposed to the contrasting methods and the therapist’s competence and enthusiasm for both methods. It may also be important to consider that maximum benefits might be reached with a lower-than-standard course of education for many cases, and that too much of a good thing may not produce the best results. It is important to note that similar multi-family group education approaches have not always proved successful (McCrearie et al, 1991) except where problem-solving training has been a core component (McFarlane et al, 1995).

Furthermore, it is interesting to refer to the earlier publication of this important study of combinations of various maintenance medication dosage strategies (Schoeler et al, 1997). For those cases receiving the care-based stress management approaches who were also maintained on optimal doses of medication throughout the 24 months, 19% of those offered the additional problem-solving training in the first 12 months were admitted to hospital in contrast to 31% of those receiving only the education group sessions. Although this difference does not quite achieve statistical significance, the trend is clear, and this is in accord with the consistent observation of somewhat greater efficacy of the problem-solving approach when it is more clearly integrated with mental health education (Falloon et al, 1999).

Authors’ reply: We appreciate Professor Falloon’s comments about our paper (Bellack et al., 2000), as well as his thoughts about the broader Treatment Strategies in Schizophrenia (TSS) project from which we drew the subjects of our paper. He is certainly correct that the study was not designed to systematically dismantle his behavioural family therapy (referred to as ‘applied family management’ in our study), and that we did not conduct a formal economic analysis of the outcomes.

Most of his comments refer to the parent TSS study, its design, its conduct and the interpretation of its results, and go substantially beyond the relatively limited questions that we addressed in our paper. The goal of our article was to examine the effects of family treatments on communication and whether changes in communication mediated patient outcomes. The TSS study compared behavioural family treatment with a less-structured family support programme. In Bellack et al. (2000) we reported that there were no differences between the two family treatments in communication or problem-solving, and changes in communication that may have occurred did not mediate outcomes of interest, including any difference in rehospitalisation.

The TSS study was, as Professor Falloon has written (Falloon et al., 1996), designed to compare two family treatments based on common assumptions and common principles. Applied family treatment (AFM) was based on the behavioural family therapy developed by Falloon and “differed from Supportive Family Treatment in intensity and in the site of delivery of treatment . . . Further, AFM has a behavioral focus, with the intent of providing specific training in communication and problem solving” (Falloon et al., 1996, page 47). Both family treatments included parallel family support groups, and the two treatments did not differ in their orientation, as Professor Falloon now suggests. Stress management was a feature of the two family interventions, but they were not conceptualised that narrowly. Falloon’s treatment (Falloon et al., 1996), in particular, was viewed as a comprehensive intervention that included case management and a multi-factorial educational component designed to modify patterns of communication within the family. He reports that the problem-solving component “seldom requires more than 5 hours of teaching”, yet his programme required 13 weekly sessions in the home followed by 13 bi-weekly sessions and then monthly sessions for up to an additional 6 months. Neither the data nor our own clinical observations support the thesis that most families are able to learn the targeted skills at all, let alone in 5 hours of training. In regard to cost benefit, it should be noted that there was no demonstrable benefit from this extensive training and the cost of more than a year of home visits would be prohibitive in most clinical settings: a statistical economic analysis would be redundant with this self-evident finding.

Professor Falloon makes two other assertions with which we disagree. First, it indicates that the monthly educational groups and the behavioural training were incompatible and that therapists “confused the two approaches”. On the contrary, the two approaches were designed to support one another by providing a common model of the illness to families in two different forums with the added benefit of multi-family support and sharing. Moreover, Professor Falloon provided quality control for the home visits but had no ongoing role in oversight of the multi-family support groups, so it is difficult to understand how he determined the existence of this putative confusion on the part of therapists.

Second, Professor Falloon notes that there was a difference in rehospitalisation between the two family treatments over 2 years under “optimal” medication conditions. The numbers cited are selected from a large analysis that tested the effects of the family treatment comparison as well as of medication condition. There was neither a statistically significant main effect of family treatment nor an interaction of family treatment and medication. The difference he cites is thus not appropriately described as “not quite” statistically significant. The clear and overarching conclusions to be drawn from the entire data set is that Falloon’s behavioural family therapy did not produce any differential benefit to family members or patients, despite its high cost. We strongly support clinical and humanitarian goals of improving the quality of interactions in families with a child suffering from schizophrenia, and of reducing stress experienced by family members and patients. However, we also believe that the TSS data provide a convincing argument that the behavioural treatment approach is not a useful or effective strategy for most families.


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Psychological therapies in anorexia nervosa

It is heartening to see trials of therapy for anorexia nervosa, the most lethal of psychiatric illnesses and the cinderella of research. Dare et al. (2001) have shown that over a year it is possible to effectively treat a severely ill group of young adults with poor prognostic features, and to do so on an out-patient basis.

I am surprised, though, that they feel able to conclude that “specialised psychotherapies are more effective than routine treatment”. The two therapies which came out ‘top’ (family therapy and focal psychoanalytic psychotherapy) were given by the same three highly experienced therapists, and the next best therapy (cognitive–analytic therapy) was given by trained specialists in eating disorders, whereas ‘routine treatment’ was provided by junior psychiatrists on 6-month rotations who had to hand over to colleagues during the year of patient contract.

Certainly, confidence in at least one model of therapy is an important component of an experienced therapist’s effectiveness, but for me the clearest implication of the study is that patients who suffer from this chronic condition do best with
continuity of care from the most experienced therapists. I am not sure we can say anything at all yet about choice of specific therapy.


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The paper by Dare et al (2001), on a trial of psychological treatments for anorexia nervosa, has two major shortcomings. The investigators planned for a year of weekly sessions of 50 minutes of psychoanalytic therapy; a year of weekly to 3-weekly sessions (60 to 75 minutes) of family therapy; 23 sessions (50 minutes) of cognitive–analytic therapy (CAT), and an unstated frequency of 30 minute sessions for 1 year for the ‘routine treatment’ group. The patients in the psychoanalytic arm ended up receiving a mean of 24.9 sessions as opposed to 12.9 for the CAT, 13.6 for the family therapy and 10.9 for the ‘routine’ arm. The differences in the numbers of sessions planned and those actually taking place has not been taken into account in evaluating the results. A summarised by Bergin & Garfield (1994), a large number of different studies show that more sessions are associated with greater improvements. However, the relationship is not linear and begins to taper off after 26 sessions: a figure almost reached by the patients in the psychoanalytic arm but far removed from that of the other three groups.

Not only did the ‘control’ group receive the fewest number of sessions, with each session lasting only 30 minutes, but as noted and implied by the authors: therapists assigned to this group had the least commitment to and experience in treating anorexia nervosa. The paper does not state how many therapists each patient ‘went through’ during the course of the study. All these factors would predispose to the formation of poor working alliances compared with the other groups. Thus, the poor results obtained by the ‘control’ group could be accounted for by a combination of fewer sessions of shorter duration and weak therapeutic alliances, rather than the superiority of specific psychological treatment models.


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I would like to comment on the Maudsley trial evaluating three psychotherapies for anorexia nervosa compared with routine treatment (Dare et al, 2001). I congratulate the team on their efforts in this study in a research area fraught with difficulties and for their major contribution to knowledge in the eating disorders field. The authors rightly conclude that little can be drawn from the study regarding the differential impact of the therapies used. However, the paper did not make clear the differences between the conditions other than the models of therapy. The experience and qualifications of therapists were stated for focal psychoanalytic therapy and family therapy but not for cognitive–analytic therapy (CAT) and one can only conclude that the CAT therapists were not trained or qualified in CAT. Also, the total contact hours in each condition varied widely. The longer the contact hours the more impact the therapy. Perhaps the trial indicates that to treat moderately severe anorexia nervosa effectively, trained and experienced therapists and/or over 15 contact hours (over 18 x 50-minute sessions) are required. The need for experienced staff delivering therapies of adequate length is well known within the field (e.g. Palmer et al, 2000) but may not be fully appreciated by those commissioning or funding services. These are perhaps more important variables affecting outcome than the specific therapeutic modality used.


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Author’s reply: We agree in part with the points made in these letters. Dr Okhain comments on the different treatment intensity between the conditions and in particular in the ‘control’ condition. The ‘control’ treatment was intended as a surrogate for placebo treatment. It is ethically difficult to have a placebo treatment for anorexia nervosa given the high morbidity of the condition and the lack of any placebo response. Our aim, therefore, was to have a ‘control’ condition similar to treatment as usual that would/could be offered in general adult psychiatry units. It could be argued that this therapy was better than that offered in many such positions in that regular supervision was given by an expert in eating disorders. Furthermore, the patients (2–3 per psychiatrist) were offered treatment for up to a year. We agree that in anorexia nervosa as in other conditions the therapeutic alliance is a key factor in response to therapy. We would argue that the specialist treatments have a specific focus on the therapeutic alliance. Indeed, it is perhaps noteworthy that the results of this study led to a change in the practice of cognitive–analytic therapy on the unit in that it is now preceded by a short course of motivational enhancement therapy to facilitate engagement (Treasure & Ward, 1997).

The number of sessions attended may be a sensitive marker of the therapeutic alliance in anorexia nervosa. For example, in a previous study comparing cognitive–behavioural therapy for anorexia nervosa with dietary management all patients dropped out of the dietary management group early in treatment (Serfaty, 1999).

We agree with Dr Morris that the important ‘take-home message’ is that specialised therapists following a specific therapeutic approach offer the best outcome in anorexia nervosa. This complements the analysis made by Nielsen et al (1998), in which he found that mortality was lower in regions of the country with specialised services. It is, therefore, of concern that such skills are in limited supply.


Quality of evidence in meta-analysis

Thase et al (2001) provide some evidence that venlafaxine is superior to selective serotonin reuptake inhibitors in terms of relapse rates. Although the authors are honest about the limitations of this meta-analysis, these need further exploration.

All meta-analyses should be based on a systematic review of the literature, which should include an exhaustive search for trials including those unpublished (grey data). Failure to do this could result in publication bias, because studies showing negative results or no differences are less likely to be published than those showing positive results. Failing to identify these missing studies may skew the results of this meta-analysis towards favouring venlafaxine. Although the authors identified a further 12 trials (not included in their analysis), there is no description of the search technique and it is possible that other trials were missed.

One way to identify possible publication bias is to construct a funnel plot (Fig. 1). This is a simple technique where effect size (in this case odds ratio taken from Table 3 of the paper) is plotted against the number of subjects in each study (Table 1). The principle of a funnel plot is that small studies are less precise and the precision of a study increases, approximating to the true effect, as the sample size gets larger. This produces an inverted funnel shape.

Data missing from the lower left segment of the plot suggests small negative studies have not been identified.

The authors do not include the 12 other trials they identified in their paper in the meta-analysis but go on to undertake a “qualitative review” of these trials. This ‘vote counting’ technique can be misleading as smaller trials are given as much weight as larger ones.

There would be a tendency for some evidence-based practitioners to disregard this paper completely. I think this is to miss the point of evidence-based medicine, which is not to be reductionist about evidence. Rather, we should use our skills in evidence-based medicine to decide where on a continuum between very good and very bad a particular paper lies, and use its conclusions accordingly.


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Placebo response in depression

An unstated conclusion to Gavin Andrews’ editorial (2001) is surely that placebo-controlled trials are absolutely essential to our understanding of the true effects of antidepressants. Without placebo trials Andrews’ main conclusion that the placebo effect is significant and worth potentiating would not be possible.

It is not sufficient to prove that new treatments are better than or equivalent to existing treatments because we do not know that the existing treatment is still better than ‘placebo’ treatments. Today’s ‘placebo’ treatment may not be the same as that of 10 or 20 years ago when the original placebo trials were done. Further, there may be considerable differences between groups with the same diagnosis. This is all well demonstrated in the study of tricyclic antidepressants in children. Since it was thought unnecessary and unethical to do placebo trials in children and adolescents, new antidepressants were tested only against existing ones and found to be effective in 50 to 70 per cent of cases. Only after 20 or so years of such trials were placebo trials done and the ‘placebo’ treatment (probably the accompanying environmental, individual and family treatment) was found to be just as effective as the drug. In this time numerous children were treated unnecessarily with tricyclic antidepressants and several may have died from cardiac arrhythmia. This was not an ethical way to introduce new drugs.

Among additional reasons for placebo controlled trials are first, that far more people have to take part in a trial comparing a new treatment with an existing treatment because the difference in effect is much less than with placebo. Thus, more people will be exposed to a new treatment with unknown side-effects. Second, placebo controlled trials are the only way to get accurate knowledge of side-effects: essential information for clinicians.

Thus, the statement by Andrews that “the existence of proven treatments would normally render placebo trials unethical” is unwarranted. I believe it is unethical not to use placebo controlled trials even when there is a proven therapeutic method (since no method is perfect), so long as there can be no lasting harm from delaying treatment and the subjects fully understand the risks and voluntarily consent. I urge researchers and clinicians to press the World Medical Association to modify the latest version of the Declaration of Helsinki (World Medical Association, 2000), which contains this restriction on placebo controlled trials.


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Does size matter?

I commend the article by Weich et al (2001) examining the effects of income inequality on mental health. Given the importance of psychosocial factors in Wilkinson’s (1996) thesis on inequality and health it is an important and long overdue contribution to this debate. Although this study was
cross-sectional in design, the inferences that can be drawn from the findings in support or opposition to Wilkinson’s hypothesis are contradictory. The finding that those on high incomes were more likely to report common mental disorders in areas of the greatest income inequality, fits comfortably within Wilkinson’s model depicting declining social cohesion, combined with increasing crime and fear. However the second finding, that those on low incomes reported a lower prevalence of common mental disorders in areas of high, rather than low income inequality may appear contrary to this model. However, this finding may help redirect research along an emerging avenue exploring the importance of the appropriate size/scale of comparisons. Recent research has explored this hypothesis at a much smaller scale, examining effects at the level of localities in England and Wales (Boyle et al, 1999). Wilkinson’s work contains an inherent contradiction on this issue. He argues that the societal level is the most appropriate scale of comparison to explore inequality and health, while basing much of his support for his thesis on studies at a much lower scale.

Weich et al’s second finding may reflect income differences in the scale of comparisons. Research exploring the mental maps of individuals, which may influence such comparisons, show dramatic differences by age, gender and particularly by income (Lynch, 1960). Although it is dated and overplayed, Stacey’s (1960) comment that “The basis of the upper class social circle is national, with the West End of London as its town centre, The Times as its local paper, and certain national events, e.g. Ascott, as its focal points”, helps demonstrate this point.

Britain is no exception to the international trend of increasing socially homogenous housing estates. Therefore, it is likely that those on the lowest incomes are living in poor, socially segregated areas. They may also be using more local scales of comparison, and in their eyes may in fact be living in areas of low income inequality. Further work is urgently needed to explore the importance of scale in the inequality and health model, paying particular attention to the effects of income, gender and age on such comparisons.


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**One hundred years ago**

**The private class of insane**

The London County Council has recently announced to medical practitioners that it has provided accommodation for about sixty female patients having a legal settlement in the county of London at the Manor House, Horton, Epsom, at a weekly charge, as at present fixed, of 1s., exclusive of clothing and special luxuries. Full particulars can be obtained from Mr. R. W. Partridge, clerk of the Asylums Committee. At the Claybury Asylum provision is also made for private patients who can claim a settlement in the county of London at a charge of 30s. a week, and for others at a charge of £2. This action of the London County Council has our hearty approval. Similar arrangements have been made in other counties, and the results have proved satisfactory in affording most needful accommodation to the poorer class of the private insane.

**REFERENCE**

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Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey
Advising relatives of risk of Alzheimer's disease
B. A. Blansjaar and H. W. van Schaick
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