Correspondence
EDITED BY MATTHEW HOTOPF

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Analysis of data on outcome of depression

The analysis of the data reported by Tuma (2000) is seriously flawed. In this report there are no primary outcome data for 26 (48%) of the elderly cohort and 8 (14%) of the younger adults. The eight elderly people developing dementia at the 4.5 years outcome point are included in the analysis of the outcome of depression but their depression outcome is not reported. Dementia is not the primary outcome in this study and, therefore, either subjects with dementia are excluded (as the author has done with natural deaths) or the depression outcome is reported. Presumably, they all survived or they would have been included as deaths.

This produces a serious bias and un-founded conclusions. For instance, if the eight subjects with dementia are excluded (as they must be if their depression outcome is not reported) then the elderly cohort at 4.5 years consists of 28 and not 36 subjects. Then, referring to Table 1, natural deaths removed, the outcome is lasting recovery 46% (not 36%), relapse and recovery 39% (not 30%), residual symptoms 7% (not 5.5%) and chronic 7% (not 5.5%). Of the elderly, 85% are recovered compared to 78% of younger adults.

If the eight dementia subjects were included and all had a lasting recovery from depression, or relapse with recovery, then the recovery rate is 88%. The conclusions reported for good outcome would be correct only if all eight subjects with dementia were included in the residual symptoms or chronic categories.

Of course, if all natural deaths had recovered from depression at the time of death, this would also paint a different picture. We all die but the issue here is whether we die happy or depressed.

It is critical that data are reported accurately. Misrepresentation of this sort could be extremely damaging.

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Author's reply: Dr Anderson is right in claiming that if patients with dementia are excluded from the calculations, the prognosis for the depression among the elderly will improve: but can dementia be regarded as a successful outcome from index depression which is incident in old age? This question may also be applied to those elderly subjects who had died at follow-up. As such, dementia and death were given special outcome categories in this study.

As to the depression status of the elderly subjects before death, they were: four died during their index illness; six achieved full recovery; two recovered, relapsed and recovered; five had chronic illness and one had dementia.

The depression status of the elderly subjects prior to developing dementia were: one recovered completely; six recovered, relapsed and recovered; and in one the depressive illness became chronic and dementia subsequently developed.

None of the younger adults recovered prior to their death but: three recovered, relapsed and recovered again; one developed chronic depressive illness; one developed post-stroke dementia; and three were classified as dead during the index illness (one by suicide).

Given this new information the reader may work out the figures accordingly.

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Breast-feeding and schizophrenia

We read with interest the article by Leask et al (2000). They conclude against any protective association of breast-feeding with development of adult psychosis.

The authors have used two UK national cohorts. In the 1958 cohort, data were last collected when the members were 33 years old, therefore missing out a significant number of possible cases, which could have given more power and would have thus reduced the possibility of type 2 error in this study with so few cases. In only 29 of 40 cases of ‘narrow schizophrenia’ were data on breast-feeding available, which means a loss of 27.5%. These are the very cases who could have missed breast-feeding totally. We are also very curious as to why the narrow definition was used when the point of interest is relevant to the whole spectrum of schizophrenic disorder (especially after using “adult psychosis” in the title of their paper). Although the selection bias is largely taken care of by the nested design of the study, there is scope for recall bias, as breast-feeding interviews took place as long as 7 years after birth in one and after two years in the other cohort.

The original study (McCreaddie et al, 1997), which the current study claims to refute, has a very strong logical appeal as it fits in nicely with the neurodevelopmental theory of schizophrenia implying diet, and therefore environment, and gene interaction. Again, this study also had a small sample of patients with data available only in 31% of cases (45/146). Of these cases, 77% were born between 1920 and 1960. However, the mothers were asked about the duration of breast-feeding with an expected precision of 1–2 weeks in 1989 only, again inviting recall bias. The other finding, which is difficult to explain away, is the fact that the siblings of these cases had a statistically similar pattern of breast-feeding, yet they did not develop schizophrenia.

In effect none of the studies can convincingly suggest any positive or negative association between breast-feeding and schizophrenia. This is doubly unfortunate as the clinical question asked has huge conceptual face validity and public health implications along with a very sensitive link with the neurodevelopmental understanding of schizophrenia.

Authors’ reply: Mukherjee & Galanis express enthusiasm for the hypothesis that breast-feeding protects the infant against later schizophrenia. This despite widely published evidence, referenced at the beginning of our article, for a lack of any substantial relationship between breast-feeding and cognitive, emotional and social development in children (i.e. a lack of predictive validity of abnormal central nervous system development).

We examined the hypothesis in two cohorts (the 1946 National Survey of Health and Development (n=4447) and the 1958 National Child Development study (n=18 856)) in which the possibility of recall bias does not arise because, in contrast with the earlier report, the data were prospectively collected with respect to outcome. We observe no evidence that an individual’s breast-feeding experience is significantly related to her/his later risk of schizophrenia.

May we suggest to those who wish to persuade us that the hypothesis is still viable that there is an onus to present findings from a larger and better-documented population.

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Changes in suicide rates or changes in suicide statistics
I read with interest both McClure’s (2000) article and the response by Rihmer et al (2000). Although both reports presented and discussed decreasing suicide rates in their countries since 1990, some important differences need to be highlighted. This letter will argue that results of the latter might have far fewer implications than those of the former.

First, I would agree that it is easier to evaluate outcome of isolated changes in some risk factors than to investigate several interrelated changes in many risk factors, some of these having opposite implications. For example, risk factors for suicide in England and Wales have been changing more or less continuously over the past decade, but there has been no abrupt political change with significant socio-economic consequences. However, in Hungary the changes since the late 1980s have led to improved (e.g. democracy) and worsened (significant increase in unemployment rates) socio-economic variables at the same time.

Second, no major changes have occurred in the official suicide statistics in England and Wales. On the other hand, recent political changes in Hungary might have had an impact on validity and reliability of death certification and reporting. The recording of cause of death could have been influenced by the renaissance of previously repressed Christianity in this country. Kel- leher et al (1998) have shown the effect of religion on the reporting of suicide rates. Open verdicts should be therefore also considered before such an extreme decline in suicide rates is reported.

Finally, Rihmer et al (2000) have thought about the possibility of a relationship between suicide rates in Hungary and recent improvements in mental health pol- icy in that country. This is not to disagree with their suggestion that better mental health care is beneficial for suicide prevention, but would it be reasonable to think that these have had more substantial effect than the Gotland study? The latter was systematically prepared, well-controlled and correctly evaluated. However, although significant, far more moderate decreases in suicide rates were noted in the pioneering work by Rutz et al (1995).


Information and education for carers of patients with Alzheimer’s disease
Marriott et al (2000) have shown the usefulness of focused interventions in reducing the burden on caregivers of patients with Alzheimer’s disease. The authors did not specify the kind of information provided to the carers in the control groups. We presume that they did not receive the kind of detailed information that was given to the caregivers in the study group. Thus, this study was not designed to compare the effects of giving information alone with an intervention programme, where giving information was only one of its components. Despite this, the authors had come to the conclusion that “providing information alone to the carer had no effect on burden”.

If one control group had received the initial three sessions of the intervention and was compared to the study group, then we would have known the efficacy of that component of the intervention. The study design does not allow us to come to conclusions about the relative efficacy of the different components of the intervention programme. So one could speculate that the first three sessions were crucial and mostly responsible for the improvement.

By dismissing the possibility that information alone could have desirable effects, the authors have underestimated its thera- peutic value. We disagree with the assertion of the authors that they found little evi- dence that information alone significantly reduced burden or had an impact on the patient. We are of the opinion that neither the study design nor their findings allow such conclusions. Effects of single-component interventions, like giving information and educating the caregiver, have to be evaluated thoroughly considering the potential for widespread application in the community, especially in developing regions of the world. There is an urgent need for developing and evaluating services that can be of use in developing countries (10/66 Dementia Research Group, 2000).


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Interventions that are costly and need highly trained professionals for implementation have serious limitations in such settings.


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Authors’ reply: Dr Shaji et al raise an important point in relation to the interpretation of trials of interventions with carers of people with dementia. In relation to our own study, information was provided in three 45-minute sessions by an experienced clinician, and supplemented by four written information booklets entitled “What are dementia and Alzheimer’s disease”, “Stress and the person with Alzheimer’s disease”, “Coping with caring” and “Advice about services”. The control group did not receive the information and education sessions. We carried out an analysis after the three sessions of information, which occurred at the beginning of the intervention, and there was no difference between the intervention and control groups at that time on any outcome variable. This finding has also been reported in trials of family intervention with carers of patients with serious mental illness (Tarrier et al, 1988). This is perhaps not surprising, as providing information and advice is notoriously poor at changing people's behaviour.

With regard to the method of the intervention, we utilised an integrated model described previously in relation to schizophrenia (Barrowclough & Tarrier, 1992). This takes an individualised approach and includes an assessment of the carer’s own model of coping. It is recognised that there are significant individual differences in the impact of education on carers managing older people with dementia. It may be that the information provided will enable those in the intervention group to utilise the later sessions more effectively.

We agree entirely with Dr Shaji et al that simple, straightforward strategies should be evaluated in carers of people with dementia, and that costly interventions should not be adopted unless they have been shown to be effective.


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Violence risk prediction in practice

Dolan & Doyle (2000) provide a helpful review of clinical and actuarial measures in violence risk prediction. The evidence shows that prediction can be significantly better than chance. However, they present only one half of the story. How well do the best instruments perform in the real clinical world where prediction leads to action, including restrictions on the liberty of patients regarded as dangerous? False positives are very serious from an ethical (including resource allocation) point of view. Here we encounter the ‘base rate’ problem that the authors inexplicably fail to mention.

The rate at which violent acts occur in the population of interest is critical to the predictive abilities of any instrument. The authors reproduce a receiver operator characteristics (ROC) curve of a well-performing instrument which, as they say, shows the trade-off between the true positive rate and the false positive rate (or conversely the true negative rate). Where that trade-off should lie depends on the relative costs of false positives v. false negatives. One usually looks at the point of maximum perpendicular distance from the diagonal line. For this ROC, a true positive rate of 0.7 and a false positive rate of 0.3 (equivalent to a true negative rate of 0.7) is probably the optimum. A test has to predict accurately who will be violent as well as who will not be violent. Although this ROC is statistically significant against chance at the P<0.001 level in predicting violence, how does it fare in practice?

It is difficult to describe how prediction instruments perform in a way that is easily comprehensible to non-mathematicians. Perhaps probability trees can help. Figure 1 shows a probability tree in which the essential data are presented in relation to a population in which 20% of patients will actually be violent during the follow-up period. Using the test represented by the ROC described, it can be seen that the positive predictive value, that is, the proportion of patients predicted by the test to be violent who indeed turn out to be violent, is 0.37. But this means also that the prediction will be wrong about six times out of ten. Perhaps a base rate of 20% is appropriate to some forensic populations.

![Fig 1 Probability tree for determining the predictive ability of a test for violence. The rate of violence in the population is 20%. The test has a true positive rate of 0.7 and a true negative rate of 0.7.](image-url)
In a community mental health service, even an inner-city one, the rate of violent acts, of any severity, over a 6-month period is more likely to be around 6% (Shergill & Szumkler, 1998). Substituting the figures 6 and 94 in the probability tree the reader will discover that the positive predictive value drops to 0.14; that is, the prediction will be wrong almost nine times out of ten. For very serious violence, perhaps at a rate of 1%, the test will be wrong about 97 times out of a 100. For homicides, at around 1 in 10 000 per annum committed by patients with a psychosis, prediction is meaningless.

Rare events are inherently difficult to predict. Even a test with an impossible 0.9 accuracy for both true positives and true negatives will be wrong more than nine times out of ten at a base rate of 1%. Thus highly statistically significant ROC curves look very limited indeed in their practical application in a community context. How unfair is it then that mental health services in the UK seem to be expected to prevent what is, in practice, unpredictable?


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**Australia with mental illness who smoke**

This Australian comparison to the editorial by McCreddie & Kelly (2000) demonstrates that the financial costs for Australian smokers with a mental illness, as for British subjects, are substantial.

As part of a detailed qualitative study of a public mental health service in Adelaide, South Australia, encompassing qualitative interviews with 24 community clients and a participant observation of the community and in-patient settings in which they have contact, I found that these smokers experience significant financial and social disadvantage as a consequence of their smoking. Within their community homes and hostels, and in-patient environments, there exists a significant reinforcing smoking culture in which cigarettes provide a central currency for many aspects of people’s lives. Smoking provides them with a source of control and autonomy in the face of overwhelming powerlessness, fear of illness relapse, and stigma. However, a vicious cycle of loss, debt and need serves to compound the predicaments of these smokers. Some basic data are presented in Table 1.

In Australia, the current average cost of one of the cheaper brands of cigarettes is $10.40 for a packet of 40 (from a survey of two supermarkets and two suburban convenience stores; recommended retail prices for the equivalent brands, as quoted by Phillip Morris and British American Tobacco Australia Ltd, were approximately $2 more). Of this, the amount returned to the government in excise is $7.79 (Australian Taxation Office, 2000). Therefore, a person with a mental illness who smokes 40 cigarettes per day gives to the government $54.53 per week in the form of tax, or $2835.56 per year. All participants in this study receive a government pension and most live alone in public rental accommodation. The current rate of the Disability Support Pension is $197.05 per week (Centrelink, 2000). Hence, such a person who smokes 40 cigarettes per day returns approximately 27.7% of their benefit to the Australian treasury.

Following the introduction of population-wide anti-smoking measures, there has been an overall reduction in the prevalence of smoking to about 25% of the Australian population. However, this is not the case for people with a mental illness. According to a National Mental Health Strategy survey (Jablensky et al, 1999), 73.3% of people with a psychotic illness smoke. With a prevalence of psychosis at 4.7 per 1000 population aged 18–64 years (Jablensky et al, 1999), there are probably at least 53 416 people with psychosis in Australia (Australian Bureau of Statistics, 2000a,b). If 73.3% smoke, and smoke on average 40 cigarettes per day, the contribution to the treasury is approximately $111 million per year. People with a mental illness are, through their smoking habit, contributing substantially to the cost of their own care.

For people with a mental illness the financial and personal consequences of their dependence on smoking impact on all aspects of their quality of life, and their ability to manage their mental illness. We are in danger of further polarising this population, already stigmatised by their mental illness, if the perpetuation of the poverty cycle in which they find themselves is not addressed.


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**Table 1 Characteristics of participants (n=24)**

<table>
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<tr>
<td>Quit attempts</td>
<td>Multiple</td>
<td>Multiple</td>
<td>0 to Multiple</td>
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**Lowered seizure threshold on olanzapine**

Olanzapine has been licensed in the UK since 1996 for schizophrenia. Along with other atypical antipsychotics it is being used increasingly, with roughly equivalent...
therapeutic effect but better side-effect profiles than more traditional antipsychotics (Lader, 1999).

A 30-year-old patient with paranoid psychosis for 5 years and seizures for 12 years, described on average two generalised seizures a year, improving with valproate. His psychosis had been controlled with zuclopenthixol for 2 years. He had normal electroencephalograms (EEGs) in 1986 and 1998, including a sleep study while taking zuclopenthixol but not valproate. His psychosis relapsed secondary to non-compliance with medication and so zuclopenthixol 400 mg twice weekly was recommenced. He improved, but owing to concerns over potential side-effects was changed to olanzapine 10 mg daily. Over the next 3 months he suffered increasing seizures culminating in a generalised or tonic-clonic seizure resulting in bilateral hemiplegic head fractures, one of which required internal fixation.

There was no metabolic or electrolyte disturbance. An EEG showed multifocal and generalised epileptiform discharges similar to those seen with clozapine, which are unusual for zuclopenthixol. They resolved on withdrawal of olanzapine and reinstitution of zuclopenthixol.

Conventional neuroleptics lower seizure threshold, yet this patient with a history of epilepsy had normal EEGs while on zuclopenthixol. Manufacturer’s trials gave a seizure rate, similar to other antipsychotics, of 0.88% patients (product data sheet, Eli Lilly). However, other epileptogenic factors were present in these patients and also in two subsequent case reports involving olanzapine and seizures (Lee et al, 1999; Wyderski et al, 1999).

Our patient thus represents the strongest case to date implicating olanzapine alone in lowering seizure threshold, with objective EEG support.

Post-marketing surveillance and case reports are a useful early warning system for reporting side-effects, for example, serendipity with cardiotoxicity and more recently olanzapine with impaired glucose tolerance. This serves to remind all practitioners of the importance of considering a possibly underemphasised side-effect within the context of a newly introduced therapy. Olanzapine should be used cautiously in patients who have a history of seizures.


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Olanzapine: concordant response in monozygotic twins with schizophrenia

There is growing evidence that genetic variation in several neurotransmitter systems (e.g. serotonergic) may influence the clinical response to different psychopharmacological drugs (Arranz et al, 1998, 2000). A previous paper (Vojvoda et al, 1996) described the concordant clinical response of a pair of monozygotic twins with schizophrenia when treated with clozapine. Now we report on two monozygotic twins concordant for DSM–IV (American Psychiatric Association, 1994) schizophrenia whose clinical response to olanzapine was also concordant.

The twins are now 60 years old. Twin 1 developed her first psychotic symptoms at age 21. Since then, she has been repeatedly admitted to hospital because of worsening of her psychiatric symptoms, never returning to her premorbid level of functioning. She was treated with a wide variety of conventional antipsychotics, always with a poor response. Prior to her first psychotic breakdown, she suffered a seizure, and was treated with phenobarbital and valproate. At age 58 years she was started on olanzapine building up to a high dose (20 mg daily) to control her symptoms. With this drug she had a good response (both in positive and negative psychotic symptoms) and an improvement in her level of functioning.

Twin 2 had her first psychotic episode and hospital admission at age 24. Subsequently, she was treated with different conventional antipsychotics as well as with clozapine, but never achieved a successful recovery. She needed several hospital treatments and suffered two seizures, with normal electroencephalogram while taking clozapine and levomepromazine, and agranulocytosis under clozapine treatment. Encouraged by her sister’s response to olanzapine, she was treated with 20 mg olanzapine daily. She showed a good response, soon improving in both positive and negative symptoms, and in her level of functioning. Each twin is now symptom-free, working and living unaidered. Their response to olanzapine treatment has been similar both in intensity and in the pattern of symptoms that have improved. To our knowledge, this is the first report describing monozygotic twins with similar illness characteristics who showed a similar response to olanzapine treatment. Our finding supports the view that, as with clozapine, genetic factors may be important in predicting response to olanzapine and other antipsychotic drugs.


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Penile self-mutilation

Self-injurious behaviour, self-mutilative behaviour or self-harming behaviour are defined as deliberate destruction of body tissue without conscious suicidal intent (Feldman, 1988). An alternative definition of self-injurious behaviour is repetitive, direct physical self-harm that is evidently not life-threatening (Herpertz, 1995). Some other terms such as autoagression, purposeful accidents and self-harm are also used. The three most commonly reported types of self-injurious behaviour are self-cutting of the skin, ocular self-mutilation and genital self-mutilation (Feldman, 1988). In Greilheimer & Groves’s (1979) study a majority of cases of male genital self-mutilation had psychosis. Cases of non-psychotic genital self-mutilation include men with character disorders and transsexuality. Many of the patients seemed influenced by religious factors, such as
beliefs involving sexual guilt. Meninger (1935) viewed circumcision among Jews as a ‘practical substitution’ of the foreskin for the entire genitalia. In India, we have not before come across any report of penile auto-amputation.

A 24-year-old male was referred from a surgical ward for psychiatric evaluation after he had severed his penis with a knife. He came from a rural farming background and had received four years of formal education (up to 8 years). From childhood, he was preoccupied with religious matters and was always ready to eschew material gains for the betterment of his fellow man. In adulthood, he decided to adopt a true religious life after deciding to forego married life and a family of his own. He became popular in his village and the people would come to him to seek his blessings and guidance. He wanted to fulfill all the obligations to attain Moksha (salvation). His extreme step of penile self-mutilation was also a step in the same direction as he did not want any sexual impulses to disturb him on his way to salvation. There was no past or family history of any psychiatric illness, chronic medical illness or drug misuse. On examination of his mental state, the patient was a pleasant and polite individual. Rapport was easily established. There was no evidence of any thought disorder, depression or perceptual abnormality. His orientation, memory and other higher mental functions were also normal. His explanation for penile self-mutilation was that he did not want to succumb to any sexual temptation which could obstruct his way to salvation.

The case is rare as he did not have any underlying psychiatric illness. His overvalued idea that sexual or married life is contradictory to religious life is also not compatible with Hinduism. The subject did not have any sexual preoccupations but in his apprehension to save himself from any forthcoming sexual temptations, he performed penile self-mutilation.


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**One hundred years ago**

**General paralysis in the Navy**

In the November number of the *Edinburgh Medical Journal* Surgeon F. H. A. Clayton, R. N., assistant medical officer at the Royal Naval Hospital, Yarmouth, publishes an analysis of the statistics of general paralysis as observed in the Royal Naval Asylum for a series of years, and discusses the question of its etiology, with special reference to sexual excess, syphilis, and alcoholism. An investigation of this disease as it occurs in the navy possesses the advantage that the inquiry is limited to a distinct class of men who are particularly subject to it, whose medical history since entry has been recorded, and whose physical condition, environment, and even mental characteristics are much alike. That seamen are more liable than officers to this disease appears from the fact that of 274 officers admitted in the last 25 years only 48 were paralytic cases, 12 of whom were warrant officers coming originally from the seamen class, whereas of 839 men 188 were paralytic cases. At present among 27 commissioned officers in the asylum there is no case, but, on the other hand, four out of six warrant officers and 18 out of 97 men come under that head. With respect to etiology Surgeon Clayton summarises his conclusions as follows: “Altogether, one inclines to accept the view that although syphilis or its toxins in many cases, by interference with nutrition, render liable to general paralysis many persons otherwise free, there is no evidence of direct connexion. The influences which act remotely are usually conditions tending to interference with nutrition and to promoting the growth of less highly organised tissues while the proximate influences probably act by lowering vitality. A ‘specific’ cause, as yet unknown, capable of developing the disease per se, though often aided by various factors, and which usually selects those apparently most healthy and vigorous both in mind and body, seems to be indicated by all the evidence.” As is well known, general paralyses always become bed-ridden and in the concluding paragraphs of his article Surgeon Clayton gives some useful hints for the prevention of bed-sores.

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