Integrated multidisciplinary approach for dementia care

To read the study on dementia care by Wolfs et al1 was a delight. They deserve a round of applause for not only conducting a trial in a very complex but essential service but also for demonstrating that an integrated multidisciplinary approach has a positive impact on dementia care. As a consultant old age psychiatrist in the English National Health Service, would I repeat this study?

In the 1970s, we experimented joint working with our colleagues in elderly medical care. At some district general hospitals, joint assessment wards were set up for older patients with complex medical and psychiatric problems. Although the idea looked attractive, the key issue for professionals was who provides and who is responsible for general care practitioners, geriatricians or old age psychiatrists. Unfortunately, the arranged marriage between the medical and psychiatric services ended in an amicable separation, if not divorce, at most places. This separation has not been helped by the fact that these services are delivered by separate hospital trusts. The situation is getting worse as many more hospitals are being managed by ever-growing-mega trusts.

Psychiatry services for older people are now well established across the UK, based on the principle of multidisciplinary working especially in the community. Dementia care has improved significantly with the introduction of memory assessment services across the UK. To bring physicians and psychiatrists together at the research-oriented teaching hospitals may be attractive, but to bring them together for integrated multidisciplinary assessment and diagnostic work does not hold any realistic future.

There are drawbacks in the Dutch study. Only 65% of patients agreed to participate. Health-related quality of life was the primary outcome. A difference of 10% or more between the intervention group and the control group had been determined as a clinically relevant difference, but the study resulted in only 9.6% group difference after 12 months. Moreover, the proportion of patients who improved more than 10% was only 39% compared with over 22% in the control group. Does this modest result justify integration of medical and psychiatric services for dementia care in the UK? The answer, I am afraid, is negative at the moment. The important lesson to learn, however, is to provide a dementia diagnostic service in terms of comprehensive assessment, reaching a diagnosis and communicating that to patients and their carers with a comprehensive care plan. I would be more interested in conducting a randomised controlled trial to evaluate the clinical effect of a diagnostic approach rather than the traditional assessment approach by the existing community mental health teams for older people.

Authors’ reply: Dr Jha feels that our study is flawed by an inclusion of only 65% of patients willing to participate. A participation rate of 65% is common in this type of study. We anticipated before the start of the study that the rate of non-participants might amount to 40%, and therefore the actual inclusion rate was higher than expected. More importantly, the two groups did not differ regarding relevant characteristics affecting the prognosis, such as age, diagnosis, gender and baseline cognition. Moreover, Dr Jha found the results of our study quite modest. We do not entirely agree with this: a difference of 9.6% between groups regarding health-related quality of life is higher than found in any pharmacological study in dementia so far. We furthermore emphasise that usual care in our region is provided by an active university medical centre and a community mental health service that have collaborated in the past on several projects. We therefore expect that the effects of our study may be underestimated, and would be higher in other regions. Indeed, a marriage between different disciplines involved in the care of people with dementia sometimes involves conflict and is dependent on the willingness to invest in the relationship. So far, a lot of work has already been performed and although the marriage still isn’t perfect, we think that it does have a realistic and happy future.

Anxiety disorders in mothers and their children

Schreier et al2 have found that the risk of anxiety disorders in children is increased when mothers have specific anxiety disorders such as social phobia and generalised anxiety disorder. They also claim that their findings confirm and extend the findings of Bijl et al.1 However, Bijl et al did not demonstrate that parental anxiety symptoms were significantly related to psychiatric disorders in the children, including anxiety disorders. In addition, anxiety disorders in the children were not related to most parental psychiatric symptoms. Moreover, their patients were older (adult children) than those in Schreier et al’s study and we are not certain whether it is possible to compare these two different populations.

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Email: imranmushtaq@doctors.org.uk; Marla Minn-Din, Department of Clinical Epidemiology and Medical Technology Assessment, University Hospital, Maastricht, The Netherlands. Email: f.verhey@np.unimaas.nl; Claire A. G. Wolfs, Department of Clinical Epidemiology and Medical Technology Assessment, University Hospital, Maastricht, University; Alfons Kessels, Carmen D. Dirksen, Department of Clinical Epidemiology and Medical Technology Assessment, University Hospital, Maastricht; Johan L. Severens, Department of Clinical Epidemiology and Medical Technology Assessment, University Hospital, Maastricht; Frans R. J. Verhey, Department of Psychiatry and Neuropsychology, University Hospital of Maastricht/Antierv Centre Limburg, PO Box 5000, 6202 A2 Maastricht, The Netherlands. Email: F.Verhey@np.unimaas.nl; Colin Campbell, Department of Psychiatry and Neuropsychology, University Hospital, Maastricht, The Netherlands. Email: f.verhey@np.unimaas.nl; Carmen D. Dirksen, Department of Clinical Epidemiology and Medical Technology Assessment, University Hospital, Maastricht; Johan L. Severens, Department of Clinical Epidemiology and Medical Technology Assessment, University Hospital, Maastricht; and Department of Health Organisation, Policy & Economics, Maastricht University, The Netherlands.

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Correspondence

Authors’ reply: As Mushtaq & Minn-Din correctly point out, Bijl et al\textsuperscript{1} did not find associations between prevalence rates of any psychiatric disorder (including anxiety disorders) in children and anxiety-related symptoms in parents. However, one must be aware of several methodological differences to our investigation: the results cited by Mushtaq & Minn-Din are based on 12-month prevalence rates and multivariate logistic regression analysis additionally controlling for childhood adversities and socio-demographic characteristics. We would like to clarify that the results of the Bijl et al paper are much more comparable with our study and that the results we are actually referring to are those based on life-time prevalence rates of psychiatric disorders in children without controlling for childhood adversities and reported separately for the various offspring disorders. Here, Bijl et al clearly report associations between anxiety in parents and children.

In addition, it is true that the adult children in the Bijl et al study were considerably older (18–65 years) than the offspring in our study (17–21 years at follow-up). We would like to add that there are other substantial ways in which the studies differ; for example, our use of assessment via direct interviews vs. family-history information.\textsuperscript{1} Nevertheless, we do not see why our claim that we confirm and extend the Bijl et al study should be problematic, especially when taking into account the low median for age at onset of anxiety disorders.\textsuperscript{2}

We would also like to point out that both studies were community-based so that the use of the term ‘patients’ by Mushtaq & Minn-Din is slightly misleading.


Cognitive–behavioural therapy for self-harm

We read Slee et al’s\textsuperscript{1} article with interest and concern. We believe there are major biases towards the treatment arm of this study which may invalidate their conclusions. Furthermore, our experience of working in a liaison psychiatry team receiving more than 1500 self-harm referrals a year leads us to question the applicability of the intervention given the characteristics of the study group.

At the outset, there are more participants in the treatment-as-usual (TAU) group shown to be depressed and this difference reaches statistical significance from the first follow-up at 3 months and gradually grows with each follow-up. Hence, it can be argued that the difference in outcome is a mere difference in depression and anxiety, which we know respond well to cognitive–behavioural therapy (CBT). Moreover, as the authors themselves admit, there was a trend from the beginning of higher suicidal cognitions in the TAU group, which assumed statistical significance from the first follow-up at 3 months. Furthermore, the authors have not attempted to match the extra time spent with participants in the CBT group with a similar amount of therapist/contact time in the TAU group. Masking (as acknowledged) of follow-up assessments was not undertaken. Therapists in the treatment group very actively pursued participants; this may have been the active ingredient rather than CBT. Sending postcards alone as an intervention significantly reduces the frequency of hospital-treated self-poisoning events.\textsuperscript{3} All these factors bias the results in favour of the treatment group. Despite these biases, the reported benefit in reducing self-harm was marginal and only statistically significant at 9 months, with questionable clinical significance.

The participants in this study differ very significantly from the individuals seen after self-harm by routine liaison psychiatry services. The self-harm definition used was very wide, including punching and head banging, which are not usually defined as self-harm by clinicians and not proven to be associated with higher suicide risk, unlike self-poisoning and self-cutting. No data are reported on the proportion of self-harm in the study which was of this milder nature. Right from the recruitment phase, participants with alcohol and drug misuse were eliminated. This clearly skews the population enormously since a very high proportion of our patients have comorbid issues. The treatment group in particular lost eight individuals before CBT was started, and all assessments and therapy sessions were then completed. We contend that this was a highly motivated and selected group likely to benefit from the intervention, and unrepresentative of the clinical population.

Short-term interventions for self-harm have not generally proved significant when explored in large-scale studies.\textsuperscript{3} It is therefore crucial that small randomised trials of CBT or other interventions are carefully designed to minimise bias, and we feel this study fell short of the design and reporting standards we would expect. We are also concerned that high-profile publication of such studies may lead to unwarranted implementation of interventions whose effect is unproven, and whose opportunity costs are great.


Authors’ reply: Kripalani et al express their concerns about biases towards the treatment arm of our study and the characteristics of our study group of patients who self-harm. With respect to biases towards the treatment arm, it should be noted that at the start of treatment no significant differences in anxiety, depression and suicidal cognitions were evident. Thus, the gradually growing difference in depression and suicidal cognitions from the first follow-up at 3 months and in anxiety at the 9-month follow-up in our opinion reflects a treatment effect. Just because the effects on secondary measures were stronger than on the target variable, we concluded that, as hypothesised, CBT primarily targeted maintaining factors of self-harm and that the specific self-harm effect was a secondary effect. Moreover, our study results remain silent on whether the treatment effects observed are attributable to specific ingredients of CBT or to the total package of CBT in addition to TAU. We agree with Kripalani et al, however, that the fact that assessments were not carried out to treatment

Andrea Schreier, Max Planck Institute of Psychiatry, Munich, Germany. Email: schreier@mpipi.de; Hans-Ulrich Wittchen, Max Planck Institute of Psychiatry, Munich, and Technical University of Dresden, Clinical Psychology and Psychotherapy, Dresden, Germany; Michael Höfler, Technical University of Dresden, Clinical Psychology and Psychotherapy, Dresden, Germany; Roselind Lieb, Max Planck Institute of Psychiatry, Munich, and University of Basel, Epidemiology and Health Psychology, Basel, Switzerland.

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Mukesh Kripalani, Northern Deanery, Tees, Esk and Wear Valleys NHS Trust, Darlington, UK. Email: drmkush@doctors.org.uk; Amanda Gash, Joe Reilly, Tees, Esk and Wear Valleys NHS Trust, Darlington, UK.

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group might have influenced outcome. With respect to characteristics of the study group, participants in our study manifested both self-poisoning (91%) and self-injury (9%) irrespective of the apparent purpose of the act, and therefore can be considered a representative sample of patients who self-harm. Of the contacted participants, only 7.3% were excluded because of schizophrenia or alcohol and drug misuse. Our final sample consisted of females (94%) with a long history of self-harm (77% reported 10 or more previous episodes of self-poisoning and/or self-injury) and severe psychological and psychiatric problems (on average four psychiatric diagnoses (mood and anxiety disorders in particular)). It is possible that CBT as an add-on to TAU is more likely to be effective for people with such chronic and severe self-harm. The fact that rate of withdrawal from CBT amounted to 17% underscores the feasibility of an intervention tailored to the needs of this particular group.

In conclusion, CBT appears to be an effective adjunct to TAU in chronic self-harm and further research on moderators and mediators of change seems warranted.

Virtual reality and paranoia

The use of virtual reality to create a ‘laboratory’ is promising. As someone who has played computer games and has used the London underground (‘tube’) trains almost daily for 4 years, I was interested in the observations that those who used the tube regularly were less likely to have persecutory thinking in virtual reality, whereas an experience of playing computer games was a strong predictor of paranoid thinking.1

I am not sure whether the observations can be justified by an assumption that the game-playing individuals were reacting because they automatically processed the computer characters as real. The use of a virtual reality environment may have introduced a bias not taken into account just by estimating the duration of game play.

Cognition and automatic thoughts are based on prior experiences. Has this study taken into account how prior gaming experiences. Has this study taken into account how prior gaming experience may affect one’s perception to a virtual reality environment, as opposed to a generalised cognition easily translated to the real world? Is there a possibility that the participants automatically processed the environment as being hostile thus making the findings ‘a strong predictor of paranoid thinking’ only in a virtual world?

The data provided in the paper fail to show the nature of gaming experience these people have had. Is it possible that a person who plays non-violent strategy games, or gambles online, will have a different experience of virtual reality compared with someone who plays first-person shooters where one of the primary objectives of the game would be to survive, keep safe distance and, of course, to ‘kill’ other players when they are in range? Also, would the findings be different if some of these people who played computer games spent their time in virtual reality social networking worlds such as ‘Second Life’?

If an experience of travelling on the tube regularly shows less likelihood of feeling persecuted in a virtual train ride, can it be said that a prior experience of a threatening virtual reality environment make those who play games more likely to feel persecuted in the chosen medium than they would otherwise be in the real life?  


Sunanda Ghosh, Hertfordshire Partnership Foundation Trust, UK. Email: sunanda.ghosh@gmail.com
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Freeman et al have used an innovative technique in a non-clinical population to confirm a high background prevalence of negative, mistrustful and fearful thoughts about others.1 Their paper may be helpful in encouraging healthcare professionals in their attempts to normalise rather than medicalise such thoughts, which are particularly common and pronounced in patients with neurotic and personality disorders.2

I am concerned, however, by the authors’ use of the word ‘paranoia’ to describe these thoughts. Freeman et al define paranoia as ‘the unfounded fear that others intend to cause you harm’, with reference only to an earlier publication by the main author; later in the paper the words ‘persecutory’ and ‘paranoid’ are used synonymously. This definition and usage are erroneous.

Varying definitions of paranoia exist in the literature but the correct meaning of ‘paranoid’ is ‘delusional’.3 With a Greek derivation and a literal meaning of ‘out of the mind’; German psychiatrists revived the term in the mid-19th century to describe conditions characterised by delusions, not only of persecution but also of grandeur.4 Later, Kraepelin, Bleuler and others variously attempted to classify paranoia, but central to all concepts was that it referred only to delusional rather than non-delusional ideation, and could include grandiose, jealous or somatic, as well as persecutory, delusions.4 Indeed, the ‘paranoid’ subtype of schizophrenia, still in use, refers to an illness dominated by hallucinations and delusions, and the latter need not be persecutory in nature.5

Of course, over the 20th century, the word has taken on an entirely different meaning outside psychiatry. Anecdotally, patients frequently report ‘paranoia’ as an unpleasant presenting complaint, despite the fact that, by its very nature, a fixed false belief cannot be viewed by its sufferer as a symptom. Similarly, mental health professionals commonly use the term erroneously, sometimes resulting in non-psychotic patients being inappropriately referred to specialist services for those with psychosis. I fear that Freeman et al’s rejection of the longstanding psychiatric definition of paranoia, in favour of its lay meaning, will only add to this unnecessary confusion.


Author’s reply: All too often the presence of paranoid thinking has only been given significance in relation to diagnosing illness. It has been viewed as a symptom that leads to a diagnosis and that, more or less, is the end of it. An alternative view is that the experience itself should take centre stage.1,2 Persecutory thinking is
important to understand and treat in its own right. Evidence is accumulating that paranoid ideation is on a spectrum of severity in the general population. Our study concerned the milder end of the experience, but it is of interest for understanding clinical paranoia.

Braithwaite's suggestion of an 'erroneous use of the word paranoia' contains an example of the problems of the traditional diagnostic approach to psychosis. The history of the term paranoia was originally described by Sir Aubrey Lewis.3 Lewis began his review of the fluctuations in the use of the word by noting that Hippocrates applied it to describe the delirium of high fever. Braithwaite does not wish to revert to this early use of the term, but takes a very traditional psychiatric delusion definition. This view is that paranoia only refers to a fixed false belief that the person cannot conceive of as a symptom. The problems with such a view of delusions have been laid out in many places over many years.4 A simple illustration of the difficulties is provided by asking: how strongly does the idea have to be held to be delusional (100% conviction, 99%, 90%, etc.)? Studies show that about a half of people with clinical delusions can conceive that they might possibly be mistaken. The empirical evidence indicates that delusions are complex multidimensional experiences that are not easily dichotomised into being present or absent. The other aspect of the objection is that paranoia can refer to all delusion subtypes. Undoubtedly, psychiatric researchers have used the term variably. In our work the definition of the experience being studied—called persecutory or paranoid ideation—is made explicit for readers, based on an earlier review.5 Therefore, the most salient point is that the phenomenon being explained is always clear.

Ghosh focuses on one of the predictors of paranoia in virtual reality: previous gaming experience. He provides helpful comment on the association. However, there are perhaps more interesting aspects of the study for psychiatry. Persecutory ideation in virtual reality was predicted by everyday occurrences of paranoid thought, suggesting that the results are more generally applicable to understanding the paranoia spectrum. Therefore, the identification of a number of emotional and cognitive processes (e.g. worry, self-esteem, cognitive flexibility) that predict paranoia is where the interest should lie for clinical paranoia. It is hoped that these aspects of the study also generate interest and debate.


**Semantic hyperpriming in schizophrenia**

Impairment of memory is one of the principal cognitive symptoms of schizophrenia. Pomarol-Clozet et al1 reported on a meta-analysis in which they evaluated the results of studies on semantic priming in schizophrenia. Semantic priming is a component of long-term implicit memory. They argued that hyperpriming (i.e. greater semantic priming in patients than healthy controls) could be an artefact of a general slowing in schizophrenia. As a consequence, these authors aimed to consider general slowing as a moderator variable in their statistical analysis. The measure of general slowing that they chose corresponded to the difference in response time between controls and patients, when prime and target were unrelated. In our opinion, this measure is not the most suitable as it reflects other cognitive processes. Individuals need to inhibit the prime so as to be able to process the target, since prime and target do not share any semantic relationship. Consequently, response time in an unrelated condition could be the expression of an accurate inhibitory process rather than of a general slowing as proposed by the authors. Some arguments support this view. First, we evaluated slowing in a simple reaction task in two different studies.2,3 Values were included as covariates in the analyses of covariance of priming effects. Despite confirming general slowing, there was evidence of significant increased priming in patients with schizophrenia compared with controls. Consequently, hyperpriming can be demonstrated even if general slowing is taken into account and controlled. Second, we demonstrated that the time required to inhibit an unrelated prime was significantly enhanced in patients with schizophrenia compared with healthy controls. General slowing was also controlled. Consequently, we demonstrated that the increased priming effect in patients compared with controls was mainly induced by increased time required to inhibit the unrelated prime. Our results support impairment of the inhibition of semantically unrelated information in patients with schizophrenia. Pomarol-Clozet et al suggested that 'the greater the slowing, the greater the amount of priming.' Given our results, an alternative explanation has to be considered. We suggest that hyperpriming in patients with schizophrenia could reflect decreased abilities to inhibit irrelevant information such as semantically unrelated information.


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**Laurent Lecardeur**, Centre de Recherche Fernand-Seguin, Montréal, Québec, H1N 3J6, Canada. Email: lecardeur@cyceron.fr; Sonia Dell’Itos, Centre Espaceur, CHU de Caen Centre d’Imagerie, Neurosciences et d’Application aux Pathologies, UMR 6232 14074 Caen, France; Emmanuel Stip, Centre de Recherche Fernand-Seguin Hôpital, Louis-H. Lafontaine, Montréal, Québec, Canada
doi: 10.1192/bjp.193.1.82

**Daniel Freeman**, Department of Psychology, Institute of Psychiatry, King’s College London, London SE5 8AF, UK. Email: D.Freeman@iop.kcl.ac.uk
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Richard Braithwaite
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