

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.

**Philip Spinhoven**, Unit of Clinical Psychology, Leiden University Institute for Psychological Research, and Department of Psychiatry, Leiden University Medical Centre, The Netherlands. Email: spinhoven@fsw.leidenuniv.nl; **Ella Arensman**, National Suicide Research Foundation, Cork, Ireland

doi: 10.1192/bjp.193.1.80b

## 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.<sup>1</sup>

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 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?

1 Freeman D, Pugh K, Antley A, Slater M, Bebbington P, Gittins M, Dunn G, Kuipers E, Fowler D, Garety P. Virtual reality study of paranoid thinking in the general population. *Br J Psychiatry* 2008; **192**: 258–63.

**Sunanda Ghosh**, Hertfordshire Partnership Foundation Trust, UK. Email: sunanda.ghosh@gmail.com

doi: 10.1192/bjp.193.1.81

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.<sup>1</sup> 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.<sup>2</sup>

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'.<sup>3</sup> 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.<sup>4</sup> 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.<sup>4</sup> 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.<sup>5</sup>

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.

- 1 Freeman D, Pugh K, Antley A, Slater M, Bebbington P, Gittins M, Dunn G, Kuipers E, Fowler D, Garety P. Virtual reality study of paranoid thinking in the general population. *Br J Psychiatry* 2008; **192**: 258–63.
- 2 Reid WH, Thorne SA. Personality disorders and violence potential. *J Psychiatr Pract* 2007; **13**: 261–8.
- 3 Hamilton M (ed). *Fish's Clinical Psychopathology* (2nd edn). Butterworth-Heinemann, 1985.
- 4 Gelder M, Gath D, Mayou R, Cowen P. *Oxford Textbook of Psychiatry* (2nd edn). Oxford University Press, 1996.
- 5 World Health Organization. *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines*. WHO, 1992.

**Richard Braithwaite**, Portsmouth City Teaching Primary Care Trust, Cavendish House, 18 Victoria Road, South Southsea, Hampshire PO5 2BZ, UK. Email: richard.braithwaite@ports.nhs.uk

doi: 10.1192/bjp.193.1.81a

**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.<sup>1,2</sup> 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.<sup>3</sup> 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.<sup>4</sup> 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.<sup>5</sup> 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 practice. These factors could be changed and thereby may lead to reductions in persecutory ideation. More broadly, the study highlights the large affective component to paranoid experience. It is hoped that these aspects of the study also generate interest and debate.

- 1 Freeman D, Bentall R, Garety P (eds). *Persecutory Delusions: Assessment, Theory and Treatment*. Oxford University Press, 2008.
- 2 Freeman D, Freeman J. *Paranoia: the 21st Century Fear*. Oxford University Press, 2008.
- 3 Lewis A. Paranoia and paranoid: a historical perspective. *Psychol Med* 1970; **1**: 2–12.
- 4 van Os J, Verdoux H. Diagnosis and classification of schizophrenia: categories versus dimensions, distributions versus disease. In *The Epidemiology of Schizophrenia* (eds RM Murray, PB Jones, E Susser, J van Os, M Cannon): 364–410. Cambridge University Press, 2003.
- 5 Freeman D, Garety PA. Comments on the content of persecutory delusions: does the definition need clarification? *Br J Clin Psychol* 2000; **39**: 407–14.

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

doi: 10.1192/bjp.193.1.81b

## Semantic hyperpriming in schizophrenia

Impairment of memory is one of the principal cognitive symptoms of schizophrenia. Pomarol-Clotet *et al*<sup>1</sup> 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.<sup>2,3</sup> 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-Clotet *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.

- 1 Pomarol-Clotet E, Oh TMSS, Laws KR, McKenna PJ. Semantic priming in schizophrenia: systematic review and meta-analysis. *Br J Psychiatry* 2008; **192**: 92–7.
- 2 Lecardeur L, Giffard B, Laisney M, Brazo P, Delamillieure P, Eustache F, Dollfus S. Semantic hyperpriming in schizophrenic patients. Increased facilitation or impaired inhibition in semantic association processing? *Schizophr Res* 2007; **89**: 243–50.
- 3 Lecardeur L, Brazo P, Dollfus S, Giffard B, Laisney M, Eustache F, Stip E. Does hyperpriming reveal impaired spreading of activation in schizophrenia? *Schizophr Res* 2007; **97**: 289–91.

**Laurent Lecardeur**, Centre de Recherche Fernand-Seguin, Montréal, Québec H1N 3V2, Canada. Email:lecardeur@cyceron.fr; **Sonia Dollfus**, Centre Esquirol, 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