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 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.

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Semantic hyperpriming in schizophrenia

Impairment of memory is one of the principal cognitive symptoms of schizophrenia. Pomarol-Clotet *et al*[1] 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-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.

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