between sessions 2 and 3. These sessions were randomised and counterbalanced, and no order effects were observed.

Finally, as Liang & Ho note, Professor Nutt commented on the two main effects of acute tryptophan depletion. That is, acute tryptophan depletion increased the dopamine response when patients took cocaine, and decreased it when the drug was absent. These opposite effects in the presence *v*. absence of drug might contribute to a core feature of substance misuse: i.e. increased incentive motivational states when drugs and highly salient drug cues are present, and decreases when these stimuli are absent.[5]

1 Linnet J, Peterson E, Doudet DJ, Gjedde A, Møller A. Dopamine release in ventral striatum of pathological gamblers losing money. *Acta Psychiatr Scand* 2010; **122**: 326–33.

2 Linnet J, Peterson E, Gjedde A, Doudet DJ. Inverse association between dopaminergic neurotransmission and Iowa Gambling Task performance in pathological gamblers and healthy controls. *Scand J Psychol* 2011; **52**: 28–34.

3 Campbell-Meiklejohn D, Wakeley J, Herbert V, Cook J, Scollo P, Ray MK, et al. Serotonin and dopamine play complementary roles in gambling to recover losses. *Neuropsychopharmacol* 2011; **36**: 402–10.

4 Abler B, Grön G, Hartmann A, Metzger C, Walter M. Modulation of frontostriatal interaction aligns with reduced primary reward processing under serotonergic drugs. *J Neurosci* 2012; **32**: 1329–35.

5 Leyton M. Conditioned and sensitized responses to stimulant drugs in humans. *Prog Neuropsychopharmacol Biol Psychiatry* 2007; **31**: 1601–13.

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Encephalitis and schizophrenia: a matter of words

The two recent articles[1,2] on the psychiatric manifestations of antibody-mediated encephalitis are important reminders that a well-informed differential diagnosis has far reaching implications for providing optimal patient care. It is indeed instructive to note that a marked recovery is possible with immunosuppressant therapy. Additionally, the need for close liaison with plasma exchange facilities, gynaecologists, neurologists and immunologists represents a novel departure for many practitioners, we presume. We did, however, have some concerns with the title of the Lennox *et al* editorial.[1] Describing the encephalitis as a treatable cause of schizophrenia jarred a little. First, we were concerned that the editorial title could give the impression that other causes of schizophrenia are not treatable. This brings to mind another excellent editorial, by Williams *et al*.[3] They proposed that we should use the term 'neuroleptic resistance' as opposed to treatment resistance when discussing clozapine therapy to avoid therapeutic nihilism. Second, is what is being described schizophrenia or a schizophrenia-like illness? The ICD-10[4] states that 'schizophrenia should not be diagnosed in the presence of overt brain disease.' As neuroimaging progresses, this stipulation might no longer be tenable. Is it preferable to refer to this type of presentation as a psychosis? However, these are minor quibbles and we will certainly view initial psychotic presentations differently as a consequence of these two important contributions to the psychiatric literature.

1 Lennox BR, Coles AJ, Vincent A. Antibody-mediated encephalitis: a treatable cause of schizophrenia. *Br J Psychiatry* 2012; **200**: 92–4.

2 Barry H, Hardiman O, Healy DG, Keogan M, Moroney J, Molnar PP, et al. Anti-NMDA receptor encephalitis: an important differential diagnosis in psychosis. *Br J Psychiatry* 2011; **199**: 508–9.

3 Williams L, Newton G, Roberts K, Finlayson S, Brabbins C. Clozapine-resistant schizophrenia: a positive approach. *Br J Psychiatry* 2002; **181**: 184–7.

4 World Health Organization. *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines*. WHO, 1992.

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Authors' reply: We suggest that the evidence shows that although antipsychotics are effective in alleviating some, although often not all, of the symptoms of schizophrenia, there is no evidence that they treat the underlying disorder. The editorial was highlighting the fact that the clinical syndrome of patients with psychosis and *N*-methyl-D-aspartate receptor antibodies is the same as those with schizophrenia, such that most patients with this new disorder have previously received diagnoses of schizophrenia. However, as O'Laughlin *et al* state, having an identifiable cause invalidates the diagnosis of schizophrenia according to ICD. We agree that a syndrome of psychoses is a better diagnostic construct. This situation is not unique to psychiatry. In epilepsies, despite the rapid advance in discovery of aetiological factors, the diagnosis remains based on the clinical presentation of the seizures.

Declaration of interest

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Loss of autism in DSM-5

I wish to comment on the phrase in the editorial by Tyrer & Craddock that in DSM-5 'the changes are largely cosmetic'.[1] This is probably correct for most of DSM-5 but not for autism, where a new, narrow definition of autism is proposed. The broader autism phenotype is accepted by professionals in this area of study. A new study has shown that only 60% of patients meet criteria for DSM-IV autism when they are assessed using the criteria of DSM-5 autism.[2] A second error in this area in relation to DSM-5 is that an aspect of autism has been split off into a new category called social communication disorder. ICD-11 has not made this error. These changes in DSM-5 in relation to autism are radical and will lead to patients losing their diagnosis and services.

1 Tyrer P, Craddock N. The bicentennial volume of the *British Journal of Psychiatry*: the winding pathway of mental science. *Br J Psychiatry* 2012; **200**: 1–4.

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