

Correspondence

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Neurohawks fight back

Bullmore *et al*¹ mount a defence of neuroscience in psychiatry, invoking history, a dawning golden age, Reil and Freud. Although ensuring that the curriculum for undergraduates and trainees should accurately reflect what is valuable for doctors wishing to understand and treat mental disorders, they do not fulfil the ambitions of their manifesto. Their argument against neuro-scepticism is weak. Specifically, physical models for mental disorder imply a particular position on psychology which is known as analytical behaviourism, and which effectively denies the existence of mind as a reasonable concept. They may wish to advance this view but either do not realise it or do not say so.

Neuroscience is a materialist enterprise that generates and examines hypotheses about brain function, which may inform new ways of looking at mental life: but psychiatry cannot be 'based' on neuroscience without becoming neurology. If psychiatrists cease to occupy the no man's land of unknowability, others will. The point about reductionism is a parallel problem. The kind of conversation that psychiatrists engage in with patients could well be better informed by neuroscience, but the reason for contemporary 'vague talk about neurotransmitters' is that the innumerable diagnostic categories invented in psychiatry bear no relation to discrete pathognomonic anomalies: nobody would base a diagnosis of schizophrenia on a brain image whether functional or structural. Patients do not need to see their brains light up to know that they are experiencing voices. In psychiatry there is an underrated crisis of validity, which many get around by claiming that psychiatry is where the rest of medicine was before the discovery of microbes and so on. An alternative view would be that schizophrenia, for example, is indeed a 'functional' disorder: an illness but not a disease, an illness that is culturally placed and to a great extent subjective in its essence.

Finally, the authors claim to refute the allegation that neuroscience is relatively bereft of therapeutic achievement. They fail to provide a single example of a 'neuroscientific' novelty since the 1960s that has transformed any really notable aspect of outcome in psychiatry. The one really big change, de-institutionalisation, could have occurred without any input from neuroscience at all; in fact, it was in large part a reaction against biomedicalism. It is doubtful that it would be deemed necessary to place yet another prominent polemical article in the *Journal* in defence of narrowly conceived neuroscientific hegemony within psychiatry, were this not the case.

- 1 Bullmore E, Fletcher P, Jones PB. Why psychiatry can't afford to be neurophobic. *Br J Psychiatry* 2009; **194**: 293–5.

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Bullmore *et al*¹ argue for psychiatry to continue to develop as a neuroscientific discipline, rebutting what they describe as 'neurophobic' views of mental illness. I share their enthusiasm for further understanding the biological basis of psychological conditions, and the article highlights an unhealthy division that continues to cause debate and disagreement in those treating mental illness. It often manifests itself in day-to-day clinical practice and is expressed by those that view mental illness as 'psychological' and those that look for a 'biological' explanation. Obviously the two cannot be separated – unless clinging to a Cartesian dualistic viewpoint, one must be optimistic that all mental life will eventually be mapped onto a neuronal substrate.

Proponents of both approaches would do well to familiarise themselves with David Marr,² acknowledged as the founder of computational neuroscience, and his concept of 'levels of analysis' which he applied to his seminal explanations of the visual system's information processing. He pointed out that one must be aware of the 'level' at which one is trying to explain a problem. Bullmore *et al* urge us to find explanations to mental functioning at the implementational level involving the biological substrate, i.e. genes, molecular and cellular interactions creating a complex system. Theories put forward by Beck and Seligman on explaining depression, for example, and Clark's work on panic disorder³ are set at a higher level of explanation and do not address the implementation of the processes. For example, Clark postulated that it is a catastrophic interpretation of body state that leads to a panic attack. This level of explanation offers a psychological mechanism but does not comment on the biological underpinning of the disorder. This does not mean that Clark's explanation of panic attacks claims the disorder to be 'psychological' rather than 'biological'. Instead, the explanation is set at a computational level and not an implementational level.

To understand that brain-based and psychological explanations are not mutually exclusive but that they offer different levels of explanation will help avoid unnecessary debate. We can no more afford to be 'neurophobic' than we can afford to be 'psycho-phobic'; understanding at every level is vital in moving psychiatry forward as a discipline of medicine.

- 1 Bullmore E, Fletcher P, Jones PB. Why psychiatry can't afford to be neurophobic. *Br J Psychiatry* 2009; **194**: 293–5.
- 2 Marr D. *Vision: A Computational Investigation into the Human Representation and Processing of Visual Information*. W.H Freeman, 1982.
- 3 Clark DM. A cognitive approach to panic disorder. *Behav Res Ther* 1986; **24**: 461–70.

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Bullmore *et al*¹ falteringly attempt to challenge 'neurophobic' positions in psychiatry, and then fail to present a persuasive argument for the increasing prominence of the neurosciences in psychiatry. They also contradict themselves in a number of places. For example, they argue that psychiatrists implicitly rely on neuroscience through prescribing drugs, suggesting that psychiatrists would not do so unless they believed that mental disorders are related to abnormal signalling between nerve cells,

but later on admit that the true mechanism of action of psychiatric drugs and the pathophysiology of mental disorders are unknown. Despite this, they conclude by advocating for more psychopharmacology in the MRCPsych curriculum.

Bullmore *et al* correctly highlight the false dichotomy between functional and organic disorders. However, they fail to acknowledge that disorders previously conceived as psychiatric, for which a neuropathology has been elucidated, are now considered neurological disorders and the preserve of neurologists. Huntington's disease and neurosyphilis are two examples. Consequently, they do not consider whether, if future neuroscientific research elucidates a neuropathology for the major mental disorders, these disorders would still be under the remit of psychiatrists. If not, perhaps there is little need for clinical psychiatrists to embrace the neurosciences.

They further note that objections to neurobiological research are based on concerns that the doctor–patient relationship would be fundamentally altered, to the patient's detriment. They argue that this is not the case for other medical specialties, where empathy and understanding are still important. However, Kleinman² notes that the doctor–patient relationship did indeed become a casualty of an increasingly scientific and technological medicine. Bullmore *et al* suggest that the neurosciences will reduce the stigma of mental illness. Yet, there is evidence that neurobiological models of mental disorder may actually increase stigmatising attitudes to the mentally ill and that clinicians who hold such views are less likely to involve patients in decisions about their care.³

They note the contention that physical models have not made any difference to clinical psychiatry, yet they provide no defence, only an optimistic future prediction that this will happen.

It is difficult to object to neurobiological research, but it is important to temper enthusiasm for its potential to revolutionise psychiatry. Not a single patient has benefitted from neurobiological research into psychiatry, and although psychopharmacology is one of the success stories of modern psychiatry, our drugs are the result of serendipity rather than a true understanding of the neural and molecular basis of the mental phenomena that underpin the experiences diagnosed as mental disorder. This research is extremely expensive and may be occurring at the cost of social, epidemiological and psychological research for which it is increasingly difficult to secure funding. In contrast, such research has created evidenced-based interventions for mental illness. For example, the finding that high expressed emotion in families is associated with greater relapse in schizophrenia led to the development of family intervention,⁴ and the finding that life events of an interpersonal nature were associated with the onset of depression led to the development of interpersonal therapy.⁵ Perhaps psychiatry cannot afford to be neurophobic, but no evidence for this has thus far been provided.

- 1 Bullmore E, Fletcher P, Jones PB. Why psychiatry can't afford to be neurophobic. *Br J Psychiatry* 2009; **194**: 293–5.
- 2 Kleinman A. *The Illness Narratives: Suffering, Healing and the Human Condition*. Basic Books, 1988.
- 3 Read J, Haslam N, Sayce L, Davies E. Prejudice and schizophrenia: a review of the mental illness is an illness like any other approach. *Acta Psychiatr Scand* 2006; **114**: 303–18.
- 4 Kuipers E, Leff J, Lam D. *Family Work for Schizophrenia: A Practical Guide (2nd edn)*. Gaskell, 2002.
- 5 Klerman GL, Weissman MM, Rounsaville BJ, Chevron ES. *Interpersonal Psychotherapy of Depression*. Basic Books, 1984.

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Psychiatry rests on the biopsychosocial model rather like a three-legged stool: remove any one of the legs and the stool, and psychiatry, fall over. Another three-legged stool might be that of emotion, cognition and behaviour, each is necessary, but insufficient, for understanding humans.

In 'Why psychiatry can't afford to be neurophobic',¹ Bullmore *et al* give a compelling picture of the complexity and explanatory power of genotype and phenotype in modern psychiatry and neuroscience. They expand phenotype to include behaviour and cognition, and also refer to Reil's vision of psychiatrists as physicians of the mind. Reil (1759–1813) coined the term 'psychiatry' and was concerned with the soul and soul organ, which he considered to be a product of the nervous system.² Reil's conception of the soul would be considerably wider than cognitive function and behaviour. Living during the Romantic period, he was concerned with what today might be called emotions, character and self-regulation.

It is difficult to do justice to the full breadth of neuroscience in an editorial; however, neuroscience and psychiatry are far broader than genes, cognition and the intervening processes. Although the nod is given to psychoanalysis and the importance of 'mental, interpersonal, developmental and therapeutic processes', and 'maternal deprivation and child abuse', there is no reference to emotion and its mental representation, affect, and the rapidly growing fields of affective neuroscience, attachment theory, affect regulation, mentalisation and developmental psychopathology.

Biology, ethology and palaeoanthropology have shown that social living has been the most important recent evolutionary pressure for brain development.³ Subjectivity is intrinsic to, and an emergent property of, our social brain.⁴ Ethology and attachment theory have shown how emotions are the glue of social interactions; from the moment of birth we are instinctually driven to engage with others: attachment behaviours, smiling and crying are genetically programmed. The representation of affect states in self and other (mentalisation) is vital to affect regulation and effective social adaptation; affect regulation and mentalisation are acquired through secure attachment relationships; and secure attachment, mentalisation and self-regulation contribute significantly to emotional resilience, which helps us to weather the challenges that life presents.^{5,6}

The danger of seeming to neglect the importance of emotion and relating (while emphasising the importance of cognition, molecules and genes) in psychiatry is that we risk promoting the disengagement from neuroscience that Bullmore *et al* argue so passionately against.

- 1 Bullmore E, Fletcher P, Jones PB. (2009). Why psychiatry can't afford to be neurophobic. *Br J Psychiatry* 2009; **194**: 293–5.
- 2 Marneros A. Psychiatry's 200th birthday. *Br J Psychiatry* 2008; **193**: 1–3.
- 3 Wills C. *The Runaway Brain*. Harper Collins, 1993.
- 4 Solms M, Turnbull O. *The Brain and the Inner World*. Other Books, 2002.
- 5 Fonagy P, Gergely G, Jurist E, Target M. *Affect Regulation, Mentalization, and the Development of the Self*. Karnac, 2004.
- 6 Sroufe LA, Egeland B, Carlson EA, Collins WA. *The Development of the Person. The Minnesota Study of Risk and Adaptation from Birth to Adulthood*. Guilford, 2005.

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Authors' reply: We thank the correspondents for their interest in our article¹ that, following Craddock's polemic,² we hoped would provoke some responses and debate. While we would dearly

like to agree with the Editor's suggestion³ that a belief in the importance of the brain marks us out as Cavaliers, we fear that the neuroscientific enterprise, marked by slow, painstaking data collection, hypothesis testing and incremental advances does not quite suit his analogy. Nor do we, in championing neuroscience, dismiss the importance of other levels of explanation as some of our respondents suggest. Our original editorial was clear on this. As for the suggestion that neuroscience is a form of behaviourism and must thereby deny the mind, we do hope that a brief survey of the past decade's cognitive neuroscientific literature refutes that concern.

McQueen is right to take us to task for forgetting emotion: this is an oversight in our article but not, we are happy to say, in the field, where affective and social neurosciences thrive. Blewett is also correct when he points out that major impacts on the lives of patients have arisen and continue to flow from phenomena that are meaningless when conceived solely within a neuroscientific framework.

We certainly do not demur from a biopsychosocial formulation; these are the three primary colours in which we paint our discipline and which make it more vibrant than other medical specialties. Rather, we point out that the 'bio-' aspect of psychiatry is getting brighter, stronger and, in our opinion, more useful such that, as a profession, we cannot afford to ignore it lest we do a disservice to our patients. To argue, as does Datta, that if we embrace this change then we shall be taken over by neurology is surely, as Johansson indicates, unflinchingly absurd.

After all, patients need good doctors first and foremost, and we believe that Reil conceived psychiatry as a broad discipline reflecting his own polymathematical abilities.

When we manage someone's arachnophobia with an appropriately eclectic mix of graded exposure, a selective serotonin reuptake inhibitor for comorbid depression, psychoeducation and family support we do not aim for them to live in a world populated by tarantulas, let alone become one. So, too, for psychiatry: in pointing out its neurophobic tendencies we aim to restore good function and allow it to move on. To us, this doesn't appear to be rocket science, just neuroscience.

- 1 Bullmore E, Fletcher P, Jones PB. Why psychiatry can't afford to be neurophobic. *Br J Psychiatry* 2009; **194**: 293–5.
- 2 Craddock N, Antebi D, Attenburrow M-J, Bailey A, Carson A, Cowen P, et al. Wake-up call for British psychiatry. *Br J Psychiatry* 2008; **193**: 6–9.
- 3 Tyrer P. From the Editor's desk. *Br J Psychiatry* 2009; **194**: 386.

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Psychopathic traits and studies of deception

Fullam *et al's* aim was 'to investigate the relationship between neural responses during deception and psychopathic personality traits'.¹ One of my main concerns is that what the authors referred to as 'deception' was not actually deception. The study participants were aware that the truth was known and they were being asked to 'lie' for the study. I do not believe this to be a good enough surrogate for deception.

For the purpose of the study, the word 'lie' was defined as 'the intentional giving of a false response and awareness that the response is false rather than a mistake'. I believe this definition to be inadequate. The definition does not take into account that participants were 'told' to provide untrue answers or the fact that the true answers were known by the assessors. This situation is

more comparable to a dramatic performance or acting rather than deception. A more appropriate definition of a lie would include the intent to deceive that is always present in a lie. These participants did not intend to deceive anyone with the 'false' answers, so they cannot be seen as lying.

Furthermore, the study adopts an approach that does not take into account the emotional and contextual elements involved in deception. The consequences of lying or not lying during the study were also incomparable to real life. This reduces the ecological validity of the study and makes the findings difficult to generalise.

The participants were also 'required' to make a motor response in order to select their answer. This adds further complexity to the analysis of the study results and further dents the ecological validity.

One of the main findings was that 'mean response times (seconds) were significantly slower during the lie condition'. Although the stated *P*-value (0.024) shows a statistically significant difference, the actual difference of a tenth of a second (the difference between 2.66 and 2.56 seconds) only equates to about 4% delay. In clinical terms this does not appear to be significant.

The functional magnetic resonance imaging (fMRI) does provide exciting opportunities for research, but the overall utility of this study appears to be very limited; further research of a higher quality is required in this fascinating but complex field.

To overcome some of the problems with the methodology, the researchers would actually have to deceive the participants regarding the aims of such a study. The British Psychological Society provides extensive guidance regarding the use of deception in research (www.bps.org.uk/the-society/code-of-conduct/ethical-principles-for-conducting-research-with-human-participants.cfm).

- 1 Fullam RS, McKie S, Dolan MC. Psychopathic traits and deception: functional magnetic resonance imaging study. *Br J Psychiatry* 2009; **194**: 229–35.

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Authors' reply: Dr Ehjaz appears to have misinterpreted the purpose of our study and his comments suggest a lack of awareness of the extensive literature examining the utility of fMRI for the detection of deception.^{1–4}

The primary goal of our study was to examine the influence of psychopathic personality traits on neural responses exhibited during deception. We used a direct replication of a previously published simple deception paradigm developed by Spence *et al's*⁵ and our definition of deception was lifted directly from Spence's work in this area. We have clearly acknowledged in the paper that the work presented needs to be replicated with more sophisticated paradigms, including those with an emotional component. The issues surrounding deception paradigm design are adequately covered in the existing literature.

Dr Ehjaz states that our main findings were the reported reaction time differences between the lie and truth conditions. This is not correct. The key findings lie in the modulation of deception-related blood oxygen level-dependent responses by personality traits. The response time data are reported as a direct replication of Spence *et al's*⁵ finding and indicate increased cognitive load associated with the production of a lie at the same time as withholding a truthful response. In neural terms, a mean response time difference of a tenth of a second is really rather significant.

- 1 Simpson JR. Functional MRI lie detection: too good to be true? *J Am Acad Psychiatry Law* 2008; **36**: 491–8.
- 2 Bles M, Haynes JD. Detecting concealed information using brain-imaging technology. *Neurocase* 2008; **14**: 82–92.
- 3 Sip KE, Roepstorff A, McGregor W, Frith CD. Detecting deception: the scope and limits. *Trends Cogn Sci* 2008; **12**: 48–53.
- 4 Spence SA. Playing Devil's advocate: the case against fMRI lie detection. *Legal and Criminological Psychology* 2008; **13**: 11–25.
- 5 Spence SA, Farrow TD, Herford AE, Wilkinson ID, Zheng Y, Woodruff PWR. Behavioural and functional anatomical correlates of deception in humans. *Neuroreport* 2001; **12**: 2849–53.

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Lithium in drinking water and food, and risk of suicide

Ohgami *et al*¹ reported lithium in drinking water (0.7–59 gm/l) and linked it to suicide rates. However, dietary lithium, which has received scant attention, is found in grains and vegetables, and to some extent animal-derived foods.² Hence, considering only lithium in drinking water may not be enough of a link to suicide rates. Dietary sources of lithium may actually have made the difference rather than just the drinking water. Differences in the prevalence of mood disorders with natural lithium levels acting as a prophylactic have been reported.^{3,4} Jathar *et al*³ assessed the lithium content of the daily diet (72.55–154.6 µg) and biological fluids, and hypothesised lithium to be a natural prophylactic. It will be interesting to see whether dietary and drinking water lithium levels have a direct impact on mood disorder prevalence, which in turn could explain the variation in suicide rates. And what about lithium-containing food cooked in lithium-containing tap water?

- 1 Ohgami H, Terao T, Shiotsuki I, Ishii N, Iwata N. Lithium levels in drinking water and risk of suicide. *Br J Psychiatry* 2009; **194**: 464–5.
- 2 Schrauzer GN. Lithium: occurrence, dietary intakes, nutritional essentiality. *J Am Coll Nutr* 2002; **21**: 14–21.
- 3 Jathar VS, Pendharkar PR, Pandey VK, Raut SJ, Doongaji DR, Bharucha MP, et al. Manic depressive psychosis in India and the possible role of lithium as a natural prophylactic. II – Lithium content of diet and some biological fluids in Indian subjects. *J Postgrad Med* 1980; **26**: 39–44.
- 4 Doongaji DR, Jathar VS, Satoskar RS. Manic depressive psychosis in India and the possible role of lithium as a natural prophylactic. I – Hypothesis. *J Postgrad Med* 1980; **26**: 34–8.

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The study by Ohgami *et al*¹ raises serious ethical issues related to the interpretation of research findings and, as a consequence, their possible application. While not denying that the findings are interesting and have caused a stir in the lay press and on the internet, we question the methodology and the possible implications if the results are taken seriously.

First, sociological reasons for suicide are important, and changing rates of suicide in many countries are linked to changes such as migration, poverty, relationships and economic issues. The finding that when gender was included in the analysis there was a difference in the significance levels between men and women (with

the results being less significant in women) is one such example. Adding lithium to tap water is not going to change these demographic and social factors that contribute to suicide rates, and not having accounted for at least some of these is a major limitation of the study. Second, although we agree with Young² in his commentary that more research is needed to prove or disprove this tantalising idea, it is also important to assess what the impact of different levels of tap-water lithium is going to be on thyroid function, pregnant women and on the unborn fetus. It is also important to assess whether tap-water levels of lithium directly correlate with serum lithium levels in the respective populations. The levels of lithium in body fluids in normal healthy controls have varied from 0.01 to 0.09 meq/l in one study,³ but there are no data about serum lithium levels among individuals attempting suicide. Maybe assessment of serum lithium levels among those with suicidal behaviour can be a place to start. More data are also needed on the role of low-dose lithium in individuals without mood disorders who are at risk of suicide.

Finally, several foods (particularly spices) are known to have relatively high levels of lithium as reported by a study in India several years ago.³ This study reported levels as high as 12 µg/g of lithium in tobacco and high levels in crude salt, rock salt and several spices. Maybe, until such time that we are certain about lithium's role in decreasing suicidality in non-psychiatric populations, it might be worth conducting randomised controlled trials with these foods in individuals with suicidal behaviour to see whether low doses of lithium really help.

Let us not throw the lithium out with the tap water yet!

- 1 Ohgami H, Terao T, Shiotsuki I, Ishii N, Iwata N. Lithium levels in drinking water and risk of suicide. *Br J Psychiatry* 2009; **194**: 464–5.
- 2 Young AH. Invited commentary on . . . Lithium levels in drinking water and risk of suicide. *Br J Psychiatry* 2009; **194**: 466.
- 3 Jathar VS, Pendharkar PR, Pandey VK, Raut SJ, Doongaji DR, Bharucha MP, et al. Manic depressive psychosis in India and the possible role of lithium as a natural prophylactic. II – Lithium content of diet and some biological fluids in Indian subjects. *J Postgrad Med* 1980; **26**: 39–44.

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Authors' reply: We thank Drs Chandra and Babu for their comments, but we would like to emphasise that we had never recommended the addition of lithium to drinking water supplies¹ because our findings are preliminary and yet to be conclusive.

First, we agree that sociological factors such as migration, poverty, human relations and economic issues may be associated with suicide rates, and have already admitted such limitations by stating 'other factors such as psychosocial and economic factors were not taken into consideration'.¹ Second, Drs Chandra and Babu state that it is also important to assess side-effects of lithium in tap water on thyroid function, pregnant women and the unborn fetus. Although it seems probable that these low levels of lithium are far below the levels required to produce side-effects, we agree with them. Third, they mention lithium levels in food, also raised by Drs Desai and Chaturvedi. This may be important because dietary lithium intake is estimated not to be a negligible quantity. For example, mean (s.d.) dietary lithium was reported to be: 1560 µg/day (980) in China; 1485 (1009) (Tijuana) and 939 (928) (Culiacan) in Mexico; 1090 (324) in Sweden; 1009 (324) in Denmark; 821 (684) (Texas), 650 (740) (New York) and 429 (116) (San Diego) in the USA; 812 (383) in Japan; 406 (383) in Germany; and 348 (290) in Austria.² Therefore, at the next stage,

it would seem necessary to measure serum lithium levels in participants, incorporating total lithium intake of both drinking water and food.

- 1 Ohgami H, Terao T, Shiotsuki I, Ishii N, Iwata N. Lithium levels in drinking water and risk of suicide. *Br J Psychiatry* 2009; **194**: 464–5.
- 2 Schrauzer GN. Lithium: occurrence, dietary intakes, nutritional essentiality. *J Am Coll Nutr* 2002; **21**: 14–21.

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Psychosis and catatonia as a first presentation of antiphospholipid syndrome

We report (with the patient's consent) a 28-year-old woman who presented with episodic psychosis and catatonia associated with antiphospholipid syndrome, with venous thromboembolism, rash, an acute phase response, and elevated liver enzymes. We know of no previous reports of catatonia associated with this syndrome.

She was admitted abroad in October 2007 with rapid-onset psychosis (persecutory delusions, visual/auditory hallucinations), confusion, and disorientation. She responded to quetiapine and lorazepam, and initially remained well after stopping medication. In July 2008 she deteriorated, with low mood, somatic and nihilistic delusions, and demotivation. She was admitted with catatonic stupor, staring and mutism. She improved with haloperidol, exhibiting severe distraction with thought block and hypersensitivity to background noise, before recalling visual/auditory hallucinations, confusion and delusions. In August 2008 she suffered a spontaneous popliteal vein thrombosis and a mild purpuric rash.

She had no personal or family history of psychiatric, auto-immune or thromboembolic disease, did not smoke or use recreational drugs, and took no medication except an oral contraceptive pill briefly before, and olanzapine the day before, admission (July 2008).

She had persistent elevations in alanine aminotransferase (79 U/l prior to quetiapine, peak 257 U/l), erythrocyte sedimentation rate (19–24 mm/h), and C-reactive protein (17 mg/l). Hepatic ultrasound showed mild diffuse echogenicity. Anticardiolipin antibodies were positive (22 IgMU/ml, August 2008; 25.4 IgGU/ml, October 2008; 18.0 IgMU/ml, November 2008 after immunosuppression). Antinuclear antibody was negative from October 2007 to August 2008, but weakly positive in October 2008. Rheumatoid factor likewise became positive.

Normal investigations included head magnetic resonance imaging, electroencephalography, blood count, renal/thyroid

function, electrolytes, calcium/phosphate, folate, cobalamin, ceruloplasmin, ammonia, lactate, porphyrins, amino/organic acids, complement, lupus anticoagulant, serology for hepatitis A/B/C, cytomegalovirus, Epstein–Barr virus, syphilis, *Toxoplasma*, and HIV; and antimitochondrial, anti-smooth muscle, anti-liver–kidney microsome, anti-thyroid peroxidase, anti-Hu/Ri/Yo, anti-voltage-gated potassium channel, anti-*N*-methyl-*D*-aspartate receptor, anti-myeloperoxidase, anti-proteinase-3, and anti- β_2 -glycoprotein-1 antibodies. Hepatic ultrasound showed mild diffuse echogenicity.

Following anticoagulation, haloperidol and venlafaxine, she was anticoagulated further (international normalised ratio 3:4) and immunosuppressed (azathioprine, prednisolone), leading to symptomatic resolution.

Vascular thrombosis and persistent antiphospholipid antibodies constitute antiphospholipid syndrome.¹ Catatonic immobility may have contributed to her thrombosis, but does not explain the immunophenotype. Oral contraceptives can exacerbate antiphospholipid syndrome; oral contraceptive use and antiphospholipid antibodies may be associated, but primarily for anti- β_2 -glycoprotein-1 antibodies.² Phenothiazines can induce antiphospholipid antibodies, but this has not been reported after quetiapine, olanzapine, or haloperidol. Although our patient may represent the first such occurrence, the spontaneous inflammation suggests an alternative interpretation. Research criteria for systemic lupus erythematosus were not met, but her inflammatory disorder may be an early stage of this disease. Psychosis and catatonia can occur in lupus. Antiphospholipid antibodies are associated with neuropsychiatric manifestations of systemic lupus erythematosus³ and psychosis *per se*.⁴

- 1 Sanna G, Bertolaccini ML, Hughes GR. Hughes syndrome, the antiphospholipid syndrome: a new chapter in neurology. *Ann N Y Acad Sci* 2005; **1051**: 465–86.
- 2 Poka R, Vad S, Lakos G, Bereczki Z, Kiss E, Sipka S. Increased titer of anti-beta2-glycoprotein I IgG antibody among factor V Leiden carriers during oral contraceptive use. *Contraception* 2004; **69**: 27–30.
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