Correspondence

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Neurohawks fight back

Bullmore et al1 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 neuroscience 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 and others will. The point about reductionism is a parallel problem.

Bullmore et al2 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 Descartian 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,3 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 disorder2 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.


Bullmore et al3 faltarily 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 neuroscience 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, Kleinman 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.

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 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, and the finding that 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.

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.


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Authors’ reply: We thank the correspondents for their interest in our article that, following Craddock’s polemic, we hoped would provoke some responses and debate. While we would dearly...
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Fullam et al’s aim was ‘to investigate the relationship between neural responses during deception and psychopathic personality traits’. One of my main concerns is 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).


Psychopathic traits and studies of deception

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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 al5 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’s5 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.


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Lithium in drinking water and food, and risk of suicide

Ohgami et al. 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. 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. Jathar et al. assessed the lithium content of the daily diet (72.55–154.64μg) 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?


The study by Ohgami et al. 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 Young 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, 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. This study reported levels as high as 12μg/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!

4 Babu state that it is also important to assess side-effects of lithium in the 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 (506) 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. Therefore, at the next stage,
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 (perseveratory 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, autoimmune 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 IgM U/ml, August 2008; 25.4 IgG U/ml, October 2008; 18.0 IgM U/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, antivoltage-gated potassium channel, anti-N-methyl-D-aspartate receptor, anti-myeloperoxidase, anti-proteinase-3, and anti-β2-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. 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-β2-glycoprotein-1 antibodies. 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 erythematosus and psychosis per se.

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