

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 benefited 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.

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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.

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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.