Correspondence

EDITED BY MATTHEW HOTOPF

Personality disorder: agency and responsibility

Much of the published reaction to the government's *Reforming the Mental Health Act* (Department of Health, 2000) has been ethical in tone, focusing upon whether it might be justifiable to detain people with personality disorders who have yet to commit a criminal offence. An implication has been that it is perhaps unreasonable to ask psychiatrists to treat the behaviours associated with personality disorders prior to conviction. However, an alternative view might be that, in these proposals, psychiatry is being hoist with its own petard.

At both the psychodynamic and biological ends of the speciality's spectrum there are research findings which suggest that personality disorder is a legitimate concern of psychiatry. Psychotherapists (particularly in the context of a therapeutic milieu) claim to be able to treat personality disorders. Neuroscientists report organic correlates (e.g. implicating prefrontal cortex). Hence, if one takes the evidence at face value, personality disorders are brain disorders amenable to psychotherapeutic intervention. Why should not a democratically elected government, concerned for the safety of its citizens, ask psychiatrists to do what we say we can do: treat mental disorder? After all, we detain other patients without them having to commit an offence.

Of course, the problem is that commentators from within the psychiatric profession either do not believe such findings or they do not wish to bear their consequences. Both the psychodynamic and biological accounts of personality disorder, if indiscriminately applied, appear to diminish personal responsibility. If personality disorder justifies mitigation in the forensic setting, then large numbers of people in society are walking about with a trump card, to be played should they ever go to court. This is not fanciful: there are many sophisticated patients who can effectively use this card to their short-term advantage in their dealings with members of community mental health teams. These individuals have *carte blanche* to commit immoral acts, an excuse, a reason (i.e. their personality disorder), and if they should murder or maim, it is the health professional who will be held to account. It has never been more important for the discipline of psychiatry to establish a coherent and consistently applied approach to agency and responsibility in the context of personality disorder.

Parenthetically, it is worth noting that there are severe limitations at both ends of the spectrum referred to above. The evidence that psychotherapeutic interventions treat personality disorder seems often to emerge from institutions with a vested interest in demonstrating success. While I do not question the integrity of the researchers concerned, perhaps their papers should include 'declarations of interest'. Also, the applicability of their findings to the real world seems limited: violent subjects with comorbid substance misuse are rarely accepted for psychotherapy lest they act out. On the biological side, despite the demonstration of correlates with psychopathy, it has to be admitted that these findings have yet to differentiate cause from effect. We have good evidence that when children learn a musical instrument, then they change the structure and function of motor regions in their brain. Might not discovering, learning and practising immoral conduct affect other brain regions similarly? Aristotle may have pre-empted us:

> "We learn how to make by making, men come to be . . . harp-players by playing the harp: exactly so, by doing just actions we come to be just" (*Nichomachean Ethics*: quoted in Dilman, 1999).

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Revisiting evolutionary psychology and psychiatry

At the risk of prolonging this non-meeting of minds, I must respond to the comments by Abed (2001) and Ayton (2000), passed on to me by Dr Lucas. No biologist could fail to agree with the great geneticist Theodosius Dobzhansky when he argued that nothing in biology makes sense except in the light of evolution – a point made at length in, for instance, my book *Lifelines* (Rose, 1998). However, we must distinguish between testable and untestable evolutionary speculations, and between determining and enabling conditions.

Despite Abed's assertion, I find it difficult to imagine what type of empirical study could reveal whether or not "the human psyche or mind [was] formed primarily during the Pleistocene", although quite clearly evolutionary processes have both enabled and limited humans in their creation of the wide variety of psychic, social and cultural styles evident in the world around us. But what *determines* our mental states and actions is for most useful purposes better understood by examining proximal causation rather than distal generalisation. And I am extremely surprised to find a psychiatrist, of all disciplines, adopting the cognitivist style of referring to the 'architecture' of the human mind as if this rigid, blueprint-evoking metaphor could encompass the richness of evolutionarily, developmentally, socially and culturally shaped mental experience.

Ayton (2000) suggests that there has been some sort of conspiracy in psychiatry to ignore biology. I am not a psychiatrist, but like any other scientist, I endeavour to defend the truth as I see it, while recognising that all our perceptions of such truths are formed within the metascientific context within which all living humans are embedded.

Abed, R. T. (2001) A defence of evolutionary psychology (letter). *British Journal of Psychiatry*, **179**, 267.

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The editorial by Abed (2000) and the subsequent correspondence cause me considerable concern as someone interested in the

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history and philosophy of the science. The suggestion that evolutionary psychology and psychiatry has been created over the past 20 years is surprising.

I would suggest that its origins are far more venerable and lie in the work of Ernst Haeckel (1834–1919), sometime Professor of Zoology at the University of Jena, who dominated the discussion of evolutionary theory in German-speaking Europe in the 19th century and who, indeed, published his theory of human evolution in 1868 (see Haeckel, 1879), 3 years prior to Darwin's *The Descent of Man* (1871). He is now perhaps best remembered for his 'biogenetic law' (i.e. that ontology recapitulates phylogeny). For human beings this means that the stages of human development replicate, in sequence, the stages of the development of the human race. In addition to biological recapitulation, Haeckel considered that the mind had also evolved and that one of the tasks of psychology was to trace this evolution.

The task was initially taken up by Freud (1950) and by Jung (1953), who started to compile historical evidence for his hypothesis of the collective unconscious in 1909. Indeed, throughout the *Collected Works* it is clear that Jung considered that ontogenesis in psychology corresponded to phylogenesis and that infantile thinking, as well as dreams, were "a re-echo of the prehistoric and the ancient".

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Freud, S. (1950) Totem and Taboo: Resemblances between the Psychic Lives of Savages and Neurotics. Reprinted (1953–1974) in the *Standard Edition of the Complete Psychological Works of Sigmund Freud* (trans. and ed. J. Strachey), vol. 13: London: Hogarth Press.

Haeckel, E. H. P. A. (1879) *The Evolution of Man: A Popular Exposition of the Principal Points of Human Ontogeny and Phylogeny* (trans. from the German edition of 1868). London: Beccles.

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Chromosome 22q11 deletion and brain tissue composition

We thank Eliez & Blasey (2001) for their kind comments about our paper (van Amelsvoort *et al*, 2001). However, we disagree that our paper implied that Eliez *et al* (2000) reported relatively smaller frontal lobe volumes and would like to draw their attention to the following. Normal brain maturation is accompanied by a reduction in cortical grey matter volume and an increase in white matter volume. Myelination typically progresses from posterior to anterior brain regions and occurs relatively late in frontal regions (where it continues into adulthood). Also, the maturational process from adolescence into adulthood is associated with a net volume reduction in frontal regions (Giedd *et al*, 1999; Sowell *et al*, 1999), and not a volume increase as Eliez & Blasey (2001) suggest. Consequently, we interpreted the relatively larger frontal lobe volumes found by Eliez *et al* (2000) in children and adolescents with velocardio-facial syndrome (VCFS) as compared with controls as possibly being caused by a relative delay in onset of 'maturational' grey matter reduction in VCFS. Our finding of a regional increase in volume of frontal grey matter and decrease in frontal white matter, in the absence of a difference in total frontal lobe (grey and white matter) volume, supports this interpretation and suggests that subtle differences in tissue composition occur which may reflect a delay in maturational processes (van Amelsvoort *et al*, 2001). Moreover, white matter abnormalities have been reported in VCFS and abnormal myelination could partially explain the abnormal, or delayed, maturational process. Future studies using longitudinal designs across this age span, and newer techniques such as diffusion tensor imaging, should be able to address this issue.

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Long-term potentiation and changes seen in depression

Reid & Stewart (2001) review evidence for a neurobiological basis of depression and it is suggested that brain plasticity plays a major role. These plasticity changes involve neuronal atrophy, neurogenesis, dendrite involution and formation, and long-term potentiation (LTP). Electroconvulsive therapy (ECT) and antidepressants enhance LTP and, as Reid & Stewart imply, the benefits (and adverse effects) of such treatments may be due to an enhancement or saturation of LTP. We question whether abnormal LTP *per se* is a critical neurobiological path to the changes seen in depression. We support the view that alterations in structural plasticity, as opposed to LTP, are more critical. Antidepressants, ECT, depression and stress can all modulate neuronal structure and LTP has been shown to be abnormal in models of depression and stress, but it does not follow that abnormal LTP is a prerequisite for these states, even though LTP is accepted to be important in, for example, associative learning.

In studies where LTP has been shown to alter neuronal structure, the increase in synaptic efficacy (assayed electrophysiologically) occurs within seconds to minutes but the earliest detected structural changes take at least 20 minutes (Yuste & Bonhoeffer, 2001). This time frame also does not correlate with the time required for the effects of antidepressant treatment (including ECT) – structural changes correlate better. Furthermore, although LTP is associated with morphological changes, these do not necessarily contribute to the potentiation (Yuste & Bonhoeffer, 2001). This casts doubt on the notion that the alterations in LTP are critical to the pathophysiological mechanism. We support the notion that the primary pathology is due to maladaptive neuronal structural change (Vaidya & Duman, 2001). The most likely reason why LTP can be affected by stress and depression,

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