Correspondence

EDITED BY MATTHEW HOTOPF

Evolutionary psychology revisited

Dr Abed's (2001) reply to my letter (Lucas, 2001) prompted me to contact Professor Steven Rose, one of the authors I cited (Rose & Rose, 2000), to check that I had not fundamentally misunderstood his position. It seemed to me unlikely that a neuroscientist would be, in Abed's words, "effectively in the camp that views the mind as a *tabula rasa*" (Abed, 2001). These are Professor Rose's comments:

> "Dr Abed both misstates the arguments of those of us who are critical of the claims of evolutionary psychology and is over-anxious to absolve its protagonists from charges of biological determinism. First, neither Professor Hilary Rose nor I, as the two editors of *Alas, Poor Darwin* (Rose & Rose, 2000), are evolutionary biologists. I am a neuroscientist whose research interest lies primarily in learning and memory and she is a sociologist of science. No neuroscientist could ever suggest that the mind was a Lockeian *tabula rasa*. As all my own writings make clear, any understanding of the human mind and brain needs to locate its structure and workings in the context of evolution and development, as well as social, cultural and technological history. For that matter, nothing the population geneticist Richard Lewontin has ever written could, to my knowledge, justify Abed's assertion concerning him and I would challenge Abed to find any quote which would support his assertion.
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> "In terms of evolutionary psychological theory, I dispute the claim made most strongly by evolutionary psychology's spokespeople that the 'architecture' of the human mind was laid down in the Pleistocene and there has not been evolutionary time since for any major change to occur. Cavalli-Sforza (2000), for one, has recently surveyed the substantial evidence of significant post-Pleistocene genetic change under selection pressures.
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> "Returning to Dr Abed's reply to Dr Lucas, if it is not 'biologically deterministic' to claim that humans possess innate 'cheat detector' modules or that men are innately programmed to prefer sex with younger women with specific hip : waist ratios, and women sex with older men – preferably with symmetrically shaped bodies which guarantee better orgasms – I am not sure what is. And I cannot believe that Abed quotes as a respectable source Thornhill & Palmer's (2000) claim that rape is an evolutionarily adaptive male strategy – that melange of scorpion fly data and human anecdote, so broadly condemned by the academic community.
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> "Finally, Abed parrots the attack made in Cosmides and Tooby's straw-person invention of what they call the 'standard social science model' that they argue dominates sociology. As Hilary Rose points out in *Alas, Poor Darwin*, there is little in European sociology which conforms to such a caricature. What is at stake is the autonomy of the social sciences as research fields from the imperialistic claims of an overly reductive biology at the hands of these new evolutionary fundamentalists. If evolutionary psychology were, as Abed claims, a 'hypothesis-driven empirical science', there would be little to complain about. Indeed, both biologists and social scientists should welcome it. The problem is that what currently passes for evolutionary psychology is little more than an untestable bunch of anecdotes based upon *a priori* ideological convictions."

I think further comment from me would be superfluous.

Abed, R. T. (2001) Evolution and psychiatry (letter). *British Journal of Psychiatry*, **178**, 179.

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Placebo response in antidepressant trials

The recent editorial by Gavin Andrews (2001) omitted some important considerations on the discussion of placebo response in clinical trials of antidepressants. These include the obligations of regulatory authorities in the appraisal of new treatments, numerous research-specific factors that contribute to the placebo response in a research environment, and the contribution of the scales currently employed as primary efficacy measures in depression trials to test the null hypothesis.

The demand by regulatory authorities for placebo-controlled trials in the evaluation of antidepressant therapies is supported by data published by Paul Leber, former Director of the Neuropharmacologic Products Division of the US Food and Drug Administration. Leber (1989, 1991) cited research studies where the antidepressant test agent was as effective as an already-approved active control medication, but in five of the six studies he cited, both were inferior to placebo. Given the notoriously poor sensitivity and interrater reliability of the Hamilton Rating Scale for Depression (HRSD), the usual primary efficacy instrument in US antidepressant clinical trials, this is not surprising to investigators who use the HRSD on a day-to-day basis. However, based on Leber's evidence alone, if regulatory agencies move toward the Declaration of Helsinki (World Medical Association, 2000) mandated non-inferiority trials as a basis for approving new drugs, then it is only a question of time before regulatory approval is given to drugs that very well might be less effective than placebo. The regulatory approval of an-inferior-to-placebo drug would ultimately be harmful to the large number of patients who would take this ineffective drug, in part being persuaded to do so on the basis of regulatory agency assurance of its efficacy. As all drugs have some side-effects in some patients, regulatory agencies must be able to affirm that a drug has been demonstrated to be better than 'nothing' (i.e. placebo) in conditions where 'nothing' has demonstrated benefits.

Andrews' summary of the mechanisms of placebo response in antidepressant clinical trials also omits several important considerations. Spontaneous remission of depression with time, the natural fluctuations of a chronic illness, and the encouragement that comes with being treated were the only factors he cited as contributors to placebo response. He does not cite the anxiety-lowering effect of receiving of a definitive diagnosis from a trusted expert physician in a clinical trial, and the increased sense of mastery and control that comes from patients' greater understanding of their illness as a result of the more unhurried

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and usually very extensive evaluation that occurs in clinical research practice compared with standard medical practice. He does not consider that these aspects of placebo response might be inherent in the environment of clinical trials, but are not generalisable to day-to-day general or psychiatric practice (although these differences are difficult to assess quantitatively). Other factors of the placebo response in clinical research are potentially amenable to change. These include potentially unhelpful-to-research, overly encouraging behaviours of those conducting the clinical trial, and false, overly optimistic patient assumptions and expectations about their outcome in the research trial, leading to inaccurate reporting of symptoms and thereby excessive response in those patients randomised to placebo. It is likely that increased efforts in patient and investigator education about how both patients and study site staff can be helpful in forming a productive research alliance and not generate 'wishful thinking' and the overly positive responses that might accompany it, is needed to reduce the costly, wasteful number of failed trials caused by excessive placebo response. In response to this need, I have recently proposed PREECT (Patient and Rater Education about Expectations in Clinical Trials), a two-component approach to the problem (Zimbroff, 2001). Briefly, the first component focuses on ensuring that clinical trial participants understand that they are entering a research alliance – not receiving regular medical care. The second component involves education of study site staff. Both anecdotal feedback and data lend support to the contention that PREECT reduces placebo response rates and, thus, could ultimately reduce the numbers of patients needed in placebo-controlled trials to achieve sufficient power to test the null hypothesis. Further exploration and validation of the PREECT approach would benefit patients and researchers involved in antidepressant trials, and would reduce the likelihood of an excessive placebo response.

Finally, a critique of the scales used as primary efficacy measures needs to be considered. This in itself is a thesis. The very fact that regulatory authorities in the USA are collaborating with researchers on tackling this issue lends support to its importance as a consideration in appraising this topic.

Andrews, G. (2001) Placebo response in depression: bane of research, boon to therapy. *British Journal of Psychiatry*, **178**, 192–194.

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Cannabis regimes

We read with interest MacCoun & Reuter's (2001) report on evaluating alternative cannabis regimes. In The Netherlands, drug policy is a topic of great interest. A scientific evaluation of policy regimes would be highly appreciated. MacCoun & Reuter's paper illustrates that this, however, is not an easy job.

As the authors stress, cross-national scientific evaluations are hampered by a lack of comparability due to methodological differences. Nevertheless, many studies summarised in their Table 1 are not methodologically comparable. MacCoun & Reuter compare the results of Dutch school surveys with those of population surveys in the USA. However, school surveys yield higher prevalence figures of substance use than population surveys do (Gfroerer *et al*, 1997). Furthermore, the age group "approximately 18" from the Dutch school survey is compared with the 18-year-old age group in the American national study. In The Netherlands schooling is compulsory until the age of 15–16 years, so 18-year-old high school students cannot be considered as representative of all 18-year-olds in our country. Among high school students aged 12–18 years we saw an increase in cannabis use in 1984–1996, but this had stabilised in 1999. The arguments that the rise may be associated with the coffee shop model and with a phenomenon the authors describe in terms of commercialisation and glamorisation do not quite convince us.

(a) The increase in cannabis prevalence coincides with a supposed increase in the number of coffee shops but this does not prove a causal relationship.

(b) About 80% of Dutch municipalities have no coffee shops at all (Bieleman & Goeree, 2000). Less than half of cannabis consumers purchase the drug in a coffee shop – the majority obtains it elsewhere (from a friend, a private house, sale on the street, courier services and take-away services).

(c) The authors do not present clear definitions of the concepts commercialisation and glamorisation. Coffee shops must adhere to the so-called AHOJ-G criteria, which include no advertising. The Public Prosecution Department proclaimed deviation from these criteria a nationwide criminal prosecution policy in 1991.

(d) The increase in cannabis use in the USA seems to have taken place much earlier than in Europe. The authors do not offer a plausible explanation for this trend but indicate the importance of non-policy factors.

(e) Countries with a high prevalence of drug use are more likely to experience a downward trend than countries with low prevalence figures. This is now the case in Europe: an ongoing increase in countries with previously low use levels, and stabilisation or even decline in countries with previous high prevalence figures, both in general population studies and in school surveys, confirm the tendency towards convergence (European Monitoring Centre for Drugs and Drug Addiction, 2000; Hibell *et al*, 2000). In the UK cannabis use among students was significantly lower in 1999 than in 1995, while in France cannabis prevalence increased steeply, although there are no coffee shops in France. In The Netherlands, among students between 1996 and 1999, not only cannabis use but also use of ecstasy, cocaine, heroin and amphetamines stabilised (de Zwart *et al*, 2000).

Clearly, trends in drug use are influenced by a complex interplay of factors.

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