Correspondence

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Implications of evolutionary theory for psychiatry

It may well have been a coincidence that the announcement of the ‘breaking’ of the human genetic code and the publication of an editorial on psychiatry and Darwinism in the Journal occurred within the same week, but it is to be hoped that both of these events signal a new beginning. Abed (2000) asks whether the time has come for psychiatry to reconsider Darwinism: in fact, one could argue that if psychiatry as a science is to survive, there is no other option. Since its first publication in 1859, Darwin’s evolutionary theory has transformed our understanding of the living world. The model has stood the test of time despite heavy resistance from religious groups, exploitation by Fascism and enthusiastic misinterpretation. The proliferation of papers on the subject in scientific journals over the past 30 years strongly suggests that it is here to stay. Evolutionary psychology has already established itself (Barkow et al., 1995). In contrast, only a few articles have been published by psychiatric journals, and evolutionary theory is largely ignored in psychiatric training worldwide.

If psychiatry has survived until now without using evolutionary theory, what would be the advantage of a theoretical shift? Psychiatry badly needs a theoretical framework (Kandel, 1998) that allows for the synthesis of knowledge accumulated by different schools that do not speak the same language and therefore do not interact with each other. Evolutionary theory is capable of integrating genetic, environmental, developmental and social explanations of behaviour and is therefore an excellent candidate (Leckman & Mayes, 1998). Furthermore, as Abed points out, the usefulness of the model can be tested by theory-driven research. Psychiatry has to take up the challenge. The application of modern evolutionary theory should lead to a more accurate understanding of human behaviour, including the origins and treatment of mental illness. Psychodarwinism became a term of abuse following atrocities perpetrated during the first half of the 20th century. It is time to learn the lessons of the past and move on. Attachment theory is one successful example of using evolutionary principles in psychiatry, and there will be more to come.


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Delighted though I was to see Abed’s editorial on evolutionary theory (Abed, 2000), I have reservations about its ability to provide an integrated scientific psychiatric view as the author implied. Rather, it provides a welcome additional frame of reference. Like all ultimate theories, it applies to everything but lacks power with specifics (for example, to clarify whether an antidepressant or psychotherapy is best for an individual patient). Evolutionary theory seldom generates new treatments. It offers ultimate causes over which we have no control.

Although I am an enthusiast of both, I am concerned that evolutionary theory has the same drawback as psychodynamic theory; it can accommodate any combination of facts. If I devise and test a theory that adolescent males will be less or more inclined to form lasting sexual relationships than older men, I can explain either. If they desire to form casual relationships, then I can argue that in the ancestral environment this benefited their genes at a stage in life when it was difficult to get a permanent mate. And if they do not, I can argue that their male ancestors propagated best by acquiring a mate in youth, reserving infidelity until later. Hence, it is difficult to establish whether a proximate or ultimate cause has determined the outcome. A true sociological explanation for the sexual strategies of adolescent males might be hidden by our adherence to evolutionary theory. Furthermore, the specific evolutionary mechanism alongside the sociological mechanism might be different from the one proposed.

An unmentioned benefit of evolutionary theory is reassurance. If cyclothymia was adaptive in the ancestral environment (by optimising peak function), then the risk of depression may have been increased in subsequent generations. Instead of ‘defective’ we can think of ourselves as highly adapted. When vandals wreck the playground where my children play I can reflect that this is normal behaviour for male primates. By exerting themselves against the environment they intimidate rivals – a pleasant zoological perspective preferable to saying that society is falling apart.


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Comments on the UK700 case management trial

The UK700 Group (2000) presents a comparative cost analysis of intensive case management (ICM) v. standard case management for patients with severe mental illness. It failed to find any significant difference in duration of in-patient treatment between the two groups at 2 years, and the cost of care was thus roughly equal. The authors conclude that “the policy of advocating intensive case management for all patients with severe psychosis is not supported….”

While the execution of the UK700 study is admirable in terms of its sheer number of subjects, the design is critically restricted by the very nature of the ‘intensive case management’ offered. Indeed, the mean number of contacts per client was 100 (s.d. = 64) v. 64 (s.d. = 30) in the control group; this equates to around one visit per week and one per fortnight, respectively. Comparison with our local (ICM) service shows that our case managers visit clients
far more regularly than this (daily, if necessary). While this might seem excessive, it appears that it is crucial to maintaining such patients in the community, and is ultimately cost-effective. Indeed, Preston & Fazio (2000) showed that for our ICM service, with a capped case-load per case worker of around 10 patients, and a mean number of annual community contacts of 164 (s.d.=20) v. 56 (s.d.=100) for non-intensive patients, in-patient bed-days fell dramatically (from a mean of 118 days (s.d.=113) per year before ICM, to 57 days (s.d.=91) in the second year of the ICM intervention). The control group showed no such reduction in bed-days, and the overall cost saving (factoring in the increased out-patient costs for the ICM group) at the end of the 2 years was AUS$81 475 for 65 patients (P<0.001).

Thus, it is important that the precise nature of the intervention is examined before dismissing ICM as a cost-effective model of service delivery.


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We read with interest the paper regarding the cost-effectiveness of intensive v. standard case management for severe psychotic illness (UK700 Group, 2000).

We feel very strongly that more comment should have been made on the topic of training (or lack of training) of the case managers involved. The findings of this large, well-designed trial are very similar to those of Muijen et al (1994), who found no difference in outcome between the use of community psychiatric nurses (CPNs) configured into case management teams v. CPNs working in a generic way. The main implication of this study was that merely reconfiguring services into different working arrangements provides no additional benefits to patients. However, it could be argued that benefits may accrue if training in research-based interventions is provided. Indeed, such training for CPNs and other health care professionals has been developed in the Thorn and similar programmes, which focus specifically on providing skills in assertive community treatment, family interventions, psychological interventions etc. One could argue that these groups of skills, which comprise what is loosely known as psychosocial interventions, are essential to effective case management.

In the UK700 study, we note that the case managers received a 2-day induction course in case management and an unspecified amount of instruction in outreach practice given by a team leader in the assertive community treatment service from Boulder, Colorado. It seems to us that such training input is insufficient to provide the skills necessary to deliver truly effective psychosocial care. (The Thorn programme comprises 36 days of direct training plus the equivalent of 50 days of further study and project work.) We are therefore not surprised that the case managers with smaller case-load sizes could not improve on the outcomes attained by those working with the more average size case-loads.

Surely studies of training per se are now needed, with random allocation of case managers to training in research-based interventions or to standard practice, and measurement of outcomes for both the trainees (in terms of skills acquisition and knowledge gain) and their patients (in terms of clinical outcomes).

We are at present spending enormous amounts of money on training throughout the National Health Service and yet the vast majority of this training remains completely unevaluated. Although randomised controlled trials of training interventions will be costly, the price of not knowing whether training makes a difference is much greater.


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Lithium and mortality

In their study of mortality in patients with affective disorder commenced on lithium, Brodersen et al (2000) paint an unfairly negative picture of the efficacy of lithium. They compared mortality in patients with affective disorders who were started on lithium, irrespective of their compliance with treatment, with that of the general population. This gives a false impression that lithium could increase mortality. To assess the efficacy of lithium, they should ideally have compared those who were compliant with the treatment with those who were not and also with the general population, as Kallner et al (2000) did. The latter study clearly demonstrates that even though affective disorder patients have an increased mortality compared with the general population, lithium has a definite antisuicidal effect. Moreover, in unipolar depression, suicide rates increased only after patients discontinued lithium. These two studies also show how the methodology can affect the findings.


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Authors’ reply: Gracious & Faludon find that our study of mortality in affective disorder patients commenced on lithium (Brodersen et al, 2000) paints an unfairly negative picture of lithium’s efficacy. They object to our intention-to-treat analysis of all patients commenced on lithium irrespective of compliance, which showed a significantly elevated standardised mortality ratio (SMR) of 2.5. They suggest that we should have compared compliant with non-compliant patients and with the general population, as did Kallner et al (2000).

Kallner et al actually reported – even in patients compliant with lithium – that mortality in general (SMR=1.6) and suicide in particular (SMR=14.0) were significantly elevated. They also found that mortality was even higher in non-compliant patients, a result which may very well be valid. However, comparison of compliant with non-compliant patients introduces a considerable selection bias, since patients are not randomly allocated to the two groups. Rather, patients with comorbidity, such as
drug and alcohol misuse and other predictors of negative outcome (Vestergaard et al., 1998) select themselves to the non-compliant patient group. Therefore, a finding that non-compliant patients fare worse than compliant patients may testify only to the existence of negative predictor variables among patients who were non-compliant, instead of supporting the efficacy of lithium treatment. Neither our study nor Kallner et al.’s allow conclusions as to whether or not lithium has specific antisuicidal effects exceeding what can be inferred from its ability to prevent recurrent illness episodes in affective disorder patients.

The efficacy of long-term prophylactic treatment with lithium has been questioned frequently (Moncrieff, 1995). We believe, as apparently do Gracious & Falodun, that despite its shortcomings lithium is a very helpful tool in the psychiatric armamentarium. Arguments that support the efficacy (or inefficacy) of long-term lithium treatment should, however, rest on sound scientific evidence.


Finding the evidence in forensic rehabilitation

Cure & Adams (2000) suggest that we managed to overlook 22,000 potential references including 2000 which apparently contain data relevant to our inquiries. Contrary to our belief, they also claim that the randomised trial is the preferred research methodology in forensic psychiatric rehabilitation.

These criticisms are, in our view, based on a poor understanding of the process of rehabilitating mentally disordered offenders, and reveal a blinkered approach to novel research strategies which may be of value in such atypical settings.

Cure & Adams cited three examples of the many quality studies they allege we overlooked in our review. All were published after the final submission of our paper, but are presumably presented as examples of the treatment and rehabilitation of mentally disordered offenders. Two of the cited reviews examine antipsychotic treatment (in people with learning disabilities and with acute schizophrenia) and the other is a review of sex offender treatment. These studies are, without doubt, most relevant to clinical forensic psychiatric practice. They do not, however, target the process of rehabilitation in a more general sense, as outlined in our paper. There is more to forensic work than drugs and specific programmes for certain offender groups.

Apparently, Cure & Adams fail to appreciate the difference between psychiatric work among forensic and non-forensic populations. That difference is the rationale for our remark that a randomised trial is not the method of choice in evaluating the outcome of forensic psychiatric rehabilitation. The crucial point is that allocation to forensic psychiatric treatment is not controlled by medical professionals but by legal authorities, refractory to the systematic and premeditated manipulation that some research requires. Although mentally disordered offenders, delivered by the courts to the hospitals, can be diverted into different treatment schemes, it is not feasible to maintain a predetermined course of rehabilitation. Important factors such as the length of incarceration, number and duration of leaves as well as external support by non-forensic caregivers, are not possible to randomise and control.

Randomised trials do not provide the only source of data on treatment efficacy, although where these trials are possible, valid and important data may be presented. Our paper did not pretend to review all articles related to the field of forensic psychiatric practice. Such magnificent and ambitious endeavours can only be embarked upon by the privileged few who are provided with considerable support from national funding institutions. Their reports may prove invaluable in guiding clinicians, assuming that the issues are correctly presented – a considerable responsibility. One obvious risk of the rapid growth of evidence-based medicine is its inhibiting effect on the advancement of the theory of clinical practice and its potentially discouraging effects on active contributors and reviewers of articles to medical journals.


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Arachnophobia: a practical management device

While not wishing to endorse a particular product or brand, I would like to report the effectiveness of a cheap and readily available device in the management of insect and spider phobia (the ‘Bug Katcha’, from Betterware). The device consists of a clear Perspex box with a sliding door mounted on a long handle, allowing the offending insect to be entrapped from a distance and released without manual contact.

Having in jest presented a severely spider-phobic psychiatrist friend with such an item, I was pleased to hear that its use had provided effective exposure in vivo and led to a marked reduction in symptoms of anxiety. She became able to talk about and to be in a room with spiders without displaying visible signs of arousal. As many non-arachnophobes prefer not to handle spiders directly, her functioning seems to have been restored to an acceptable level.

This device may provide a practical and cost-effective way to reduce the manifestations of simple insect and spider phobias.

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Thrombocytosis due to clozapine treatment: working towards an early marker for clozapine-induced agranulocytosis

Recently, Hampson (2000) reported thrombocytosis with clozapine, and serious consideration must be given to reports that
identify potential markers for the development of agranulocytosis. Haematological side-effects include leucopenia, neutropenia and thrombocytopenia (1–3% of patients); and anaemia, leucocytosis and thrombocytosis (<1% of patients) (American Hospital Formulary Service, 1997). Thrombocytosis reported with clozapine treatment may give evidence of the mechanism of agranulocytosis in some patients.

In some cases clozapine is discontinued if the differential white blood cell count shows an initial drop with starting clozapine treatment. If a re-challenge on clozapine results in either thrombocytosis or thrombocytopenia, this may be a result of an immune reaction, as both these platelet abnormalities are recognised features of such a reaction. (Note that it is now recommended that permanent withdrawal of clozapine should occur for leucopenia below $3 \times 10^9$ or neutrophil count below $1.5 \times 10^9$ (British Medical Association & Royal Pharmaceutical Society of Great Britain, 2000)).

Clozapine has a direct action on the haematopoietic stem cells of the bone marrow and can therefore trigger a reaction similar to an acute myeloid leukaemia or myeloproliferative disorder. It is hypothesised that an abnormal haematocrit and platelet abnormality could be seen if clozapine causes these side-effects via an immune reaction on the haemopoietic tissue. Karyotype analysis provides useful prognostic information in myelodysplastic syndrome (Provan, 1997), and is associated with clozapine response (Arranz et al, 1995). A high index of suspicion when reviewing the full blood count or karyotype analysis could lead to a marker before fatal agranulocytosis occurs as a result of clozapine.


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Etomidate-induced convulsion prior to electroconvulsive therapy

Choice of anaesthesia for electroconvulsive therapy (ECT) has become more limited in the past year owing to the non-availability of methohexital. As a result, there has been more use of etomidate in our hospital as an anaesthetic agent for ECT. We would like to draw your attention to a case of a spontaneous seizure after administration of etomidate.

A young man aged 19 years had been an in-patient for 3 months, following the onset of a schizoaffective disorder. He was being treated with a course of ECT and his medication from the start of this treatment was chlorpromazine $1000 \text{mg/day}$, procyclidine $15 \text{mg/day}$ and fluoxetine $20 \text{mg/day}$. There was no relevant past medical history or previous history of seizures. Prior to his tenth treatment of ECT he experienced a spontaneous generalised tonic-clonic seizure while being induced under anaesthetic. He was administered etomidate initially $26 \text{mg}$, which was increased to a dose of $30 \text{mg}$ as facial twitching was evident. He was then administered suxamethonium $75 \text{mg}$. However, the twitching continued into a full grand mal seizure lasting about 90 seconds, which was terminated by $10 \text{mg}$ diazepam given intravenously.

Recovery from anaesthesia was otherwise normal and there was no evidence of postictal confusion or other physical sequelae. Etomidate was used as an anaesthetic agent for this man’s nine other ECT sessions, to a maximum dose of $28 \text{mg}$ with no adverse effect. Improvement in this young man’s mood was maintained following this incident and it was decided to discontinue ECT.

Generalised seizures after short-term etomidate infusion have been reported during or after recovery from anaesthetic (Goroszeniuk et al, 1986; Krieger & Koemer, 1987; Hansen & Drenck, 1988). However, seizures have not yet been reported during induction of etomidate anaesthesia or while undergoing a course of ECT. There is no definite neuropharmacological explanation for this seizure-like activity of etomidate, which is thought to result from a disinhibition of subcortical activity, rather than a specific epileptogenic effect of the compound (Kugler et al, 1977).

Concurrent use of fluoxetine and chlorpromazine may have partly contributed, lowering the seizure threshold. However, in this case the slightly higher dose of etomidate seems the most likely causative agent, as all other medication had been prescribed at the above doses throughout the course of ECT.

This case emphasises the importance of minimising adverse effects during ECT by using the lowest effective dose of anaesthetic agent and carefully considering concurrent usage of other medication.


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