Aripiprazole in the treatment of primary delusional parasitosis

The review by Lepping *et al*[1] points out the difficulty of engaging patients with primary delusional parasitosis in psychiatric treatment owing to their poor insight. Our clinical experience fully supports this. The authors emphasise the lack of randomised controlled studies in this field and the limited, but promising, anecdotal literature on the use of atypical antipsychotics. Their systematic review did not identify any reports of aripiprazole in the treatment of primary delusional parasitosis. However, since this review was accepted for publication, a case report has appeared reporting the successful use of aripiprazole in an 85-year-old woman with primary delusional parasitosis.[2]

Aripiprazole has a unique pharmacological profile that includes partial agonist activity at dopamine D_2 and serotonin 5-HT_{1A} receptors. It has a favourable side-effect profile relative to other antipsychotics.[3] It is non-sedating and has little propensity to cause weight gain, extrapyramidal symptoms, prolactin elevation and metabolic disturbance. However, it can cause nausea and akathisia in some patients. The favourable tolerability profile may be a particular benefit in primary delusional parasitosis as these patients are often reluctant to consider antipsychotic treatment and tolerate medication poorly.

Aripiprazole has a long half-life (about 60 h) compared with other oral antipsychotics,[4] which means that occasional missed doses are less likely to affect clinical outcome. Consequently, aripiprazole may be particularly useful when intermitted adherence with medication is a problem, a situation often encountered in primary delusional parasitosis. Interestingly, in the five main studies of antipsychotic treatment of primary delusional parasitosis identified by Lepping *et al*,[1] the highest remission rate (73%) was in the only study that assessed antipsychotic depots.[5] Although the small sample sizes limit the value of cross-study comparisons, this result is consistent with the view that medication adherence is poor in primary delusional parasitosis and that treatments that can overcome this, in this case a depot antipsychotic, can lead to better outcomes.

In summary, although further evidence is needed to establish the efficacy of aripiprazole in primary delusional parasitosis, it seems reasonable to consider this drug when discussing treatment choices with patients.

Declaration of interest

P.M.H. has received fees for lecturing and consultancy from Bristol-Myers Squibb.

1 Lepping P, Russell I, Freudenmann RW. Antipsychotic treatment of primary delusional parasitosis: systematic review. *Br J Psychiatry* 2007; **191**: 198–205.

2 Rocha FL, Hara C. Aripiprazole in delusional parasitosis: case report. *Prog Neuropsychopharmacol Biol Psychiatry* 2007; **31**: 784–6.

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4 Mallikaarjun S, Salazar DE, Bramer SL. Pharmacokinetics, tolerability, and safety of aripiprazole following multiple oral dosing in normal healthy volunteers. *J Clin Pharmacol* 2004; **44**: 179–87.

5 Frithz A. Delusions of infestation: treatment by depot injections of neuroleptics. *Clin Exp Dermatol* 1979; **4**: 485–8.

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Authors' reply: Narayan *et al* suggest that the pharmacokinetics (half-life of 60–80 h) and side-effect profile of aripiprazole may make it particularly interesting for the treatment of primary delusional parasitosis where adherence to and engagement with treatment are the most significant challenges. They mention a case report of an 85-year-old woman with primary delusional parasitosis, who fully responded to aripiprazole.[1] It is interesting that our own systematic review showed that the best response rates were achieved with first-generation depot antipsychotics.[2]

However, other first-generation antipsychotics with relatively long half-lives did not differ from those with shorter half-lives. Experience with second-generation antipsychotics with long half-lives is limited to one case with partial remission to sertindole[3] and one recent case treated with paliperidone.[4] The level of efficacy of second-generation antipsychotics in delusional parasitosis is less than certain. Our own review showed that only 25% of patients treated with a second-generation antipsychotic achieved full remission in primary delusional parasitosis. The side-effect profile of aripiprazole may be advantageous with regard to the development of metabolic syndrome, but this is often less important in delusional parasitosis because of the often short period of time that patients agree to take medication. Furthermore, the common side-effect of insomnia is a real problem with aripiprazole in a patient group that already suffers from agitation and insomnia because of persistent itching.

We therefore do not agree that the current evidence available for aripiprazole makes it the substance of choice among second-generation antipsychotics for primary delusional parasitosis despite its favourable pharmacokinetic profile and low risk of metabolic and cardiac complications. There are substantially more successful reports on risperidone (more than 30) and olanzapine (more than 15) as well as our own positive experience with amisulpride than case reports on aripiprazole in both primary and secondary delusional parasitosis. However, clinical studies, not case reports, will be needed to further establish second-generation antipsychotics in delusional parasitosis and show their individual effects in this syndrome.

Declaration of interest

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1 Rocha FL, Hara C. Aripiprazole in delusional parasitosis: case report. *Prog Neuropsychopharmacol Biol Psychiatry* 2007; **31**: 784–6.

2 Lepping P, Russell I, Freudenmann RW. (2007) Antipsychotic treatment of delusional parasitosis: systematic review. *Br J Psychiatry* 2007; **191**: 198–205.

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150 years of evolutionary theory

'In the distant future I see open fields for more important researches. Psychology will be based on a new foundation, that of the necessary acquirement of each mental power and capacity by gradation.'[1]

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Charles Darwin first recorded his ideas on 'transmutation', a word used to signify the changeable nature of species, in 1844. However, he did not publish his ideas then but instead embarked on painstaking studies of molluscs and other subjects for many years in his home-based laboratory, publishing widely and making some novel discoveries in the area of mollusc biology. We will never know whether he would have got around to publishing his theory of evolution if it had not been for the work of a young naturalist called Alfred Russel Wallace, who forwarded his own (remarkably similar) ideas on the subject to Darwin in 1858. As a result, the two men had their findings jointly presented to the Linnaean Society one and a half centuries ago this year, on July 1st 1858,[2] an event that initially passed by relatively quietly, but that was soon to rock the scientific establishment and society as a whole. Darwin's notebooks prove that he had been developing his theories on 'transmutation' for the previous 20 years, based on his observations on the HMS Beagle and his own 'home-work' on molluscs and numerous other subjects. Potential reasons as to Darwin's delay in publishing his findings include his wish to produce as much supportive scientific evidence as possible, ambivalence about publishing a Godless theory in a religious society and, at a personal level, a reluctance to offend his devoted wife Emma, who was a devout Christian.[3]

Charles Darwin contributed directly to modern psychology and psychiatry in the form of his book *The Expression of the Emotions in Man and Animals*, which was effectively the first textbook on human evolutionary psychology and psychiatry.[4] His indirect contribution is far more significant, and involves the application by many others of evolutionary principles to psychology and psychiatry.[5–7] However, despite the universal acceptance of evolutionary theory in all branches of the biological sciences, evolution is effectively ignored in mainstream medicine and in psychiatry and psychology, partly because of difficulties with providing empirical evidence or 'proof', and partly because of the corruption and perversion of such ideas in the abuses of Social Darwinism and eugenics. Perhaps most importantly, there is only an emerging evidence base on the clinical applications of evolutionary theory to psychology and psychiatry, and this area remains a challenge to researchers and clinicians with an interest in the subject.

Despite these problems and shortcomings, an undeniable fact remains that evolution is one of the central platforms of biology and, if psychology and psychiatry are to be considered as belonging to the biological (as opposed to the social) sciences, then evolutionary theory must have relevance to the study of the human mind.[8]

In a time when 'biological psychiatry' has taken on hopelessly reductionistic connotations, for example relating the complexity of human emotions and psychopathology to often questionable and over-simplistic neurotransmitter theories, psychiatry and psychology were never more in need of the fresh perspectives that evolutionary theory would bring to the study of the human mind. A 21st-century presentation to the Linnaean Society is needed, this time on evolutionary psychology and psychiatry.

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Corrections

White-matter hyperintensities in first-episode psychosis. *BJP*, **193**, 25–30. Page 29, col. 1, 2nd sentence should read: Although several papers reported frontal[27,31] and frontoparietal[32,33] location of white-matter lesions in association with bipolar disorder, only one recent study[34] directly compared frontal deep white-matter hyperintensities between participants with bipolar disorder and controls, in an elderly population, and found increased frontal scores bilaterally in the bipolar disorder group.

Psychiatry in pictures: military psychiatry at the Maudsley, 1918. *BJP*, **192**, 439. Those photographed were, from left to right, standing: Captain Bernard Hart, Captain Frederick Golla, not known, not known, Captain G. W. B. James, possibly Captain Millais Culpin; seated: Captain W. H. R. Rivers, not known, Lt Colonel Atwood Thorne, Lt Colonel F. W. Mott. If any reader should know the identity of those not named, please contact Professor Edgar Jones (Edgar.Jones@iop.kcl.ac.uk).

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