certain trait – such as hypervigilance for possible dangers, as argued in the editorial – in a few members of a social group is potentially beneficial for the group, this would not affect the ability of each one of those hypervigilant individuals to spread their genes. At an individual level, it would be difficult to argue that hypervigilance would increase the overall chances of survival and procreation of a particular human. This individual would be cautious, but also seriously handicapped by an inability to trust others in the social group. Certain aspects of a human phenotype, such as psychotic symptoms, are not advantageous, but they have not been eradicated by evolution simply because they do not have a sufficient impact on survival before reproductive age. An evolutionary approach would not find any advantages in having bad teeth or weak coronary arteries, but the fact that these widespread human characteristics manifest themselves only after the individual has already had the chance to reproduce explains why it is that they are still – unfortunately – very much with us.

1 Kelleher I, Jenner JA, Cannon M. Psychotic symptoms in the general population – an evolutionary perspective. *Br J Psychiatry* 2010; **197**: 167–9.

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Authors' reply: We thank readers for their interest in our editorial.[1] The main purposes of the editorial were threefold: (i) to highlight the relatively recent identification and characterisation of a non-clinical psychosis population (for review see Kelleher & Cannon[2]); (ii) to point out that there might be important overlap in the genetics of the clinical and non-clinical psychosis phenotypes; and (iii) to discuss the potential value of this population for empirically testing evolutionary theories of psychosis.

Dr Euba points out that hypervigilance may lead to an individual being 'handicapped by an inability to trust others in the social group' and as a result being less likely to procreate. However, hypervigilance is not in itself a disadvantage. In fact, the more vigilant an animal, the more likely it is to identify threats such as predators and to protect both itself and its progeny, allowing the propagation of associated genes. Increasing levels of vigilance, however, would promote survival of the organism and its progeny only to a point. As this trait becomes ever more pronounced, it would eventually lead to the dysfunction identified by Euba – paranoia. Nesse referred to this as cliff-edged fitness,[3] whereby traits may increase fitness up to a critical threshold, but beyond this point, fitness drops precipitously (the cliff edge here being the transition from hypervigilance into paranoia). Thus, while in its extreme form – paranoia – hypervigilance will be negatively selected owing to negative fitness consequences, a 'subthreshold' level of this trait would be positively selected.

We agree with Treffurth that it is possible that non-clinical psychotic symptoms may be neither advantageous nor disadvantageous and that associated genes may have been passed on alongside other fitness-enhancing phenotypes. Our argument, however, is that if, as has been suggested by many researchers to date, the genetics of psychosis encode for positive as well as negative traits, then people with the recently characterised non-clinical psychosis phenotype may provide a valuable population in which to conduct empirical research.

Hubbeling makes the very point that we wished to emphasise in our editorial – that the non-clinical psychosis phenotype provides us with a population in which to test hypotheses about the evolutionary benefit of psychosis genes. It is clear why genes that promote certain traits, such as language development, hypervigilance and complex social cognition, would be selected in evolution. The 'how' questions, as Hubbeling points out, require attention, for instance how these traits differ in (non-psychotic) persons with psychosis genes compared with persons without (or with fewer) psychosis genes. This type of research is precisely what we wish to encourage by highlighting the validity of the non-clinical psychosis phenotype for empirical investigation. This population provides a potentially valuable means for moving beyond 'just-so' stories[4] into the realm of testable hypotheses.

1 Kelleher I, Jenner JA, Cannon M. Psychotic symptoms in the general population – an evolutionary perspective. *Br J Psychiatry* 2010; **197**: 167–9.

2 Kelleher I, Cannon M. Psychotic-like experiences in the general population: characterizing a high-risk group for psychosis. *Psychol Med* 2010; May 19: 1–6 (Epub ahead of print).

4 Kipling R. *Just So Stories*. Macmillan, 1902.

3 Nesse RM. Cliff-edged fitness functions and the persistence of schizophrenia. *Behav Brain Sci* 2004; **27**: 862–3.

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Corrections

Auditory hallucinations and brain structure in schizophrenia: voxel-based morphometric study. *BJP*, **196**, 412–413. All correlations reported in this paper are negative (i.e. the higher the hallucination scores, the smaller the gray matter values). There were no positive correlations.

Nalmefene in the treatment of pathological gambling: multicentre, double-blind, placebo-controlled study. *BJP*, **197**, 330–331. Page 330, col. 1, the fifth sentence should read: 'In a double-blind, 16-week multicentre trial ($n = 207$), various doses of nalmefene (25, 50, 100 mg/day) showed similar efficacy, but premature discontinuation was common (drop-out rate: 66%).[4,]'

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