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.

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’ into the realm of testable hypotheses.


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).


Corrections

Auditory hallucinations and brain structure in schizophrenia: voxel-based morphometric study. BIP, 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. BIP, 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%).’


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).


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