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Editorial

Psychotic symptoms in the general population – an evolutionary perspective

Ian Kelleher, Jack A. Jenner and Mary Cannon

Summary
Our ideas about the intrinsically pathological nature of hallucinations and delusions are being challenged by findings from epidemiology, neuroimaging and clinical research. Population-based studies using both self-report and interview surveys show that the prevalence of psychotic symptoms is far greater than had been previously considered, prompting us to re-evaluate these psychotic symptoms and their meaning in an evolutionary context. This non-clinical phenotype may hold the key to understanding the persistence of psychosis in the population. From a neuroscientific point of view, detailed investigation of the non-clinical psychosis phenotype should provide novel leads for research into the aetiology, nosology and treatment of psychosis.

Declaration of interest
J.A.J. is director of Jenner Consultants, Haren, The Netherlands, which provides advice and training on psychological treatment of psychosis.

Ian Kelleher (pictured) is a postgraduate student in the Department of Psychiatry at the Royal College of Surgeons in Ireland, Dublin. Jack A. Jenner is emeritus associate professor in the Department of Psychiatry at the University Medical Center Groningen and the Mental Health Care Foundation in Friesland, The Netherlands. Mary Cannon is an associate professor in the Department of Psychiatry at the Royal College of Surgeons in Ireland and a consultant psychiatrist in the Department of Psychiatry, Beaumont Hospital in Dublin.

Psychosis is the price we pay for being what we are. And how unfair, how bitterly unfair it is that the price is not shared around but paid by one man in a hundred for the other ninety-nine.

(Sebastian Faulks, *Human Traces*)[1]

Hallucinatory experiences are as old as humankind and until the nineteenth century these experiences were generally attributed to mystical or divine sources such as gods or demons, whereas in modern times, hallucinations have generally been regarded as pathological and as signs of illness.[2] Recently, however, our ideas about hallucinations and delusions are being challenged by findings from epidemiology. A new viewpoint is emerging. Simply put, hallucinations and delusions are more common than we think. Population-based studies using both self-report and interview surveys show that the prevalence of psychotic symptoms is far greater than had been previously considered, with a recent meta-analysis suggesting a prevalence rate of 5–8% in the general population, which is about ten times higher than the prevalence of diagnosed psychotic disorders.[3] Prevalence rates of such symptoms may be even higher among young people. Ten years ago, Poulton *et al*[4] reported that 14% of 11-year-old children in the Dunedin birth cohort reported psychotic symptoms on interview and showed that these symptoms were associated with a 5- to 16-fold increased rate of psychotic illness in early adulthood, depending on the strength of the initial symptoms. Since then, large, population-based studies surveying psychotic symptoms among adolescents have found rates of 9–14% in interview-based studies,[5,6] and greater than 25% in some studies using self-report questionnaires.[7–9] Positive answers on self-report questionnaires have been validated on clinical interview.[9] Therefore it is becoming increasingly clear that a sizable minority of young people experience psychotic symptoms.

This high prevalence of non-clinical psychotic symptoms in the population prompts us to re-evaluate these symptoms in the light of evolutionary theory.[10] In this editorial we discuss how this non-clinical phenotype (i.e. psychotic symptoms) may hold the key to understanding the persistence of psychosis in the population and provide a new perspective on aetiology and treatment.

Evolutionary theories of psychosis

Psychosis is highly heritable and exerts strong negative fitness effects. Despite this apparent disadvantage, schizophrenia maintains a relatively stable prevalence worldwide. Several theories drawing on the Darwinian paradigm of selective advantage have been formulated to explain the persistence of psychosis in the human population. Crow's 'speciation' hypothesis argues that psychosis is the 'price that *Homo sapiens* pays' for development of language.[11] Burns proposed that schizophrenia is a 'costly by-product' in the evolution of complex social cognition[12] and Nesse developed this idea further in terms of 'cliff-edge' fitness,[13] whereby certain traits may increase fitness up to a critical threshold, but beyond this point, fitness falls precipitously. For instance, strong tendencies to use metarepresentation and theory of mind can increase the ability to predict other people's behaviours and discern their intentions, but, as Nesse explains, 'it is only one step further, over the cliff's edge of psychotic cognition . . . to finding secret meanings and evidence for conspiracies in other people's most casual gesture'.[13] Recently, Dodgson & Gordon have proposed that certain types of hallucinations could be viewed as evolutionary by-products of a cognitive system designed to detect threat since, from a survival perspective, it is much worse to fail to recognise a threat such as the sound of an approaching predator than to mistakenly believe that a predator is approaching when it is not.[14] Evolution may therefore favour a selective skew towards propagation of genes that promote false positives over false negatives, thus resulting in 'hypervigilance hallucinations' in the population.[14] In social or pack animals, such as humans, hypervigilance is not necessary for every member, but the presence of this trait in at least some members stands to benefit the entire group. Such a trade-off allows the persistence of advantageous traits even in the presence of increased risk of disorder.

Implications for genetic studies

The possibility that vulnerability to psychosis may be a by-product of 'normal' human brain evolution, may offer an explanation for

the difficulty in locating a genetic 'point of rarity' between individuals with schizophrenia and controls.[15,16] We would also suggest that the limited success in findings in schizophrenia to date may be a result of shared genetic variation between the clinical (disease) phenotype and the non-clinical (symptom) phenotype. Genetic research thus far has used the simple dichotomy of people with psychotic disorder compared with the general population. However, the relatively high prevalence of the non-clinical psychosis phenotype in the general population may have masked genes of importance. Rather than searching for genetic variation only between individuals with psychotic disorders and the rest of the population (which contains, as we have discussed, significant numbers of individuals with the non-clinical phenotype), it may prove more fruitful to investigate genetic variation among individuals with the non-clinical phenotype in the general population (plus or minus individuals with the disease phenotype) compared with the individuals without such symptoms. Furthermore, an understanding of these genes and their functions may hold the key to explaining the persistence of psychosis in the population. It is possible that there are evolutionary advantages associated with genes that contribute to the non-clinical phenotype but which, in a fitness trade-off, also increase the risk for psychotic disorder. If, for example, as suggested by cliff-edge fitness theory,[10] psychosis results from the development of certain useful traits beyond an optimal peak, we might find that people with the non-clinical psychosis phenotype demonstrate advantageous traits compared with the rest of the population in, for example, certain language, cognitive or metacognitive skills. Detailed investigation of the non-clinical psychosis phenotype might, therefore, allow us to uncover a Darwinian explanation for the persistence of psychosis genes in the general population that has not been possible by looking only at psychotic disorder.

Implications for diagnosis and treatment

The current diagnostic nosological systems are undergoing revision. Evolutionary theories lend support for the idea of a continuum approach to the diagnosis of psychosis[15] and provide some clues as to why the traditional Kraepelinian or categorical approaches to psychosis are beginning to prove problematic.[16] Breaking down the categorical barriers may also help reduce the stigma associated with psychosis, which remains one of the greatest obstacles standing in the way of recovery and rehabilitation. Taking an evolutionary perspective may also lead to changes in how we think about treatment – for instance the idea of schizophrenia as a disorder of social brain development points to the importance of family therapy and social skills training. Some therapies such as hallucination-focused integrative therapy already incorporate such elements alongside more traditional cognitive–behavioural therapy approaches.[17] The idea of hallucinations as a 'hypervigilance' reaction also suggests the value of engaging directly with the content of the symptoms in psychosis and acknowledging the fact that some individuals may wish to preserve positive or 'useful' voices that they feel support or help them.[17]

Broadening the evolutionary perspective

An evolutionary approach may also prove fruitful in other areas of psychiatry. Higher than expected prevalences have been reported for depression and anxiety. Prospective studies are showing that up to half (and perhaps even a majority) of the population can expect one or more episodes of depression over their lifetime.[18,19] Taking an evolutionary perspective, one could speculate that the tendency for unhappiness and dissatisfaction is intrinsic to the human species (man as the 'unhappy ape') and this is one of the features that has been a contributory factor in the success of our species, driving *Homo sapiens* onwards, even to the surface of the moon, in search of new terrain and challenges. Applying evolutionary theories to depression may also lead to new approaches to treatment and novel aetiological theories.[10]

Conclusion

Charles Darwin's anniversary year has ended but that does not mean we should put the theory of evolution back on the shelf and dust it off only when the next anniversary comes round. As Theodosius Dobzhansky famously stated, 'Nothing in biology makes sense except in the light of evolution'.[20] From a neuroscientific point of view, detailed investigation of the non-clinical psychosis phenotype should provide novel leads for research into the aetiology, nosology and treatment of psychosis and other mental disorders.

Ian Kelleher, MSc, Department of Psychiatry, Royal College of Surgeons in Ireland, Dublin, Ireland; **Jack A. Jenner**, MD, PhD, University Medical Center Groningen, Groningen and the Mental Health Care Foundation, Friesland, The Netherlands; **Mary Cannon**, MD, PhD, Department of Psychiatry, Royal College of Surgeons in Ireland and Department of Psychiatry, Beaumont Hospital, Dublin

Correspondence: Mary Cannon, Department of Psychiatry, Royal College of Surgeons in Ireland, Education and Research Centre, Beaumont Hospital, Dublin 9, Ireland. Email: marycannon@rcsi.ie

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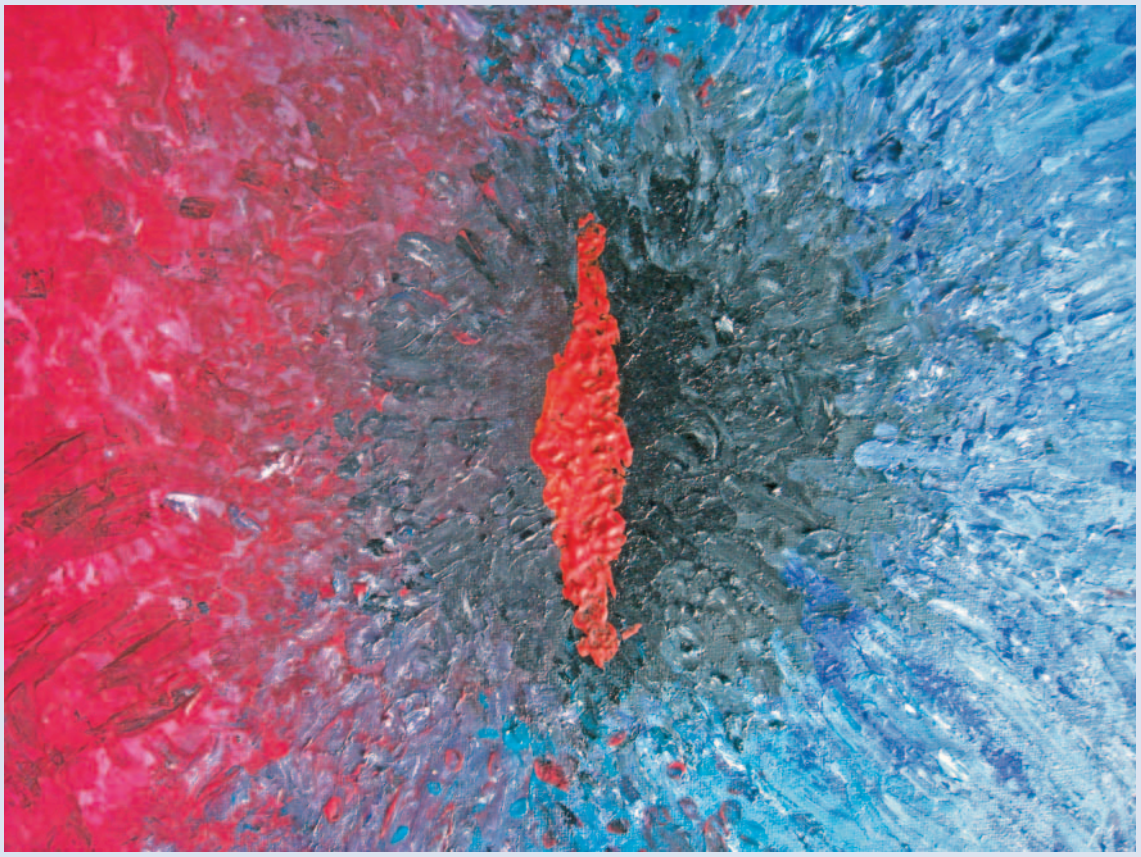
psychiatry in pictures

Bipolar (2008)

Alice Hatter (b. 1979)



Alice Hatter decided to leave her medical studies after developing a bipolar illness. This decision was not made because she felt that her illness prevented her from continuing or coping with her studies, but because her outlook on life changed afterwards. Being able to express herself and her experiences through the medium of art has been a great help to her while trying to combat her illness. She had always enjoyed the arts when growing up and hopes to develop this area of her life further. Alice's interest lies in the link between creativity and mental illness, in particular affective disorders, and she hopes in the future to have the opportunity to look more closely at this area of mental health.

This picture represents Alice's idea and experience of a bipolar illness. One side is blue and the other is pink, representing depression and mania, respectively. The colours get deeper as you go to the centre where the core appears black – but it is actually black on the depression side and very dark purple on the mania side. The centre is open to interpretation – the red can either represent 'rage' or 'anger' which is at the centre of the illness but gets buried under the other emotions; or it could be the rawness of the person when the 'end point' of mania or depression is reached; it can be like falling into the depths and being exposed and naked. Around the top and bottom edges of the picture the colours merge into one another to give a turquoise colour eventually which Alice feels represents the 'normal' mood state, but the fact that all the colours merge like a spectrum is still preserved as, like the illness, it is a continuum rather than specific discrete entities.

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