that the best-fitting model was one in which cigarette smoking (or nicotine dependence) predicted depression. In the original study, we reported on analyses of nicotine dependence symptoms and symptoms of depression in order to maintain a focus on measures germane to psychiatry, in view of the scope of this Journal.

Finally, Dr Sheikh argues that depression must be caused by nicotine withdrawal rather than smoking. However, Benowitz\(^3\) has shown that active smokers go through several withdrawal phases during each day, and that these withdrawal phases are one of the factors that causes self-administration of nicotine. Therefore, it could also be argued that depressive symptomatology may be increased among active smokers because of this continual cycle of withdrawal and satiety.


**Evolution and non-clinical psychotic symptoms**

In their recent editorial, Kelleher et al\(^1\) emphasised the importance of evolutionary theory for explaining the persistence of psychotic symptoms, depression and anxiety in humans. The authors did not mention the difference between proximate and ultimate explanations, in other words between ‘how’ and ‘why’ explanations,\(^2\) and this could make their argument for using explanations, in other words between ‘how’ and ‘why’ symptoms, depression and anxiety in humans. The authors did not mention the importance of evolutionary theory for explaining the persistence of psychotic traits is beneficial but too many are detrimental.

In their recent editorial, Kelleher et al\(^1\) emphasised the importance of evolutionary theory for explaining the persistence of psychotic symptoms, depression and anxiety in humans. The authors did not mention the difference between proximate and ultimate explanations, in other words between ‘how’ and ‘why’ explanations,\(^2\) and this could make their argument for using evolutionary theory in psychiatric research more specific. In the development of treatments one needs an explanation at the proximate level, whereas the ultimate level can be necessary for generating hypotheses.

In evolutionary-based research the challenge is to find not which behaviour is beneficial now, but which behaviour has been advantageous for the procreation of ancestors in the past. This is the ultimate-level explanation. We know very little about our human ancestors and hypotheses can easily become ‘just-so’ stories with limited predictive value. Therefore rigorous testing at the how level is required.\(^3\) Furthermore, there are complicating factors such as cliff-edged fitness,\(^4\) whereby a limited number of traits is beneficial but too many are detrimental.

The possible theories for psychosis or schizophrenia mentioned by Kelleher et al\(^1\) vary enormously. It might have something to do with language development, complex social cognition, hypervigilance or with something completely different. However, all these theories need to be further developed to generate hypotheses at the how level, for example how language/hypervigilance/social cognition skills differ in humans with genes associated with schizophrenia or in family members of people with schizophrenia. The aim is to explain psychiatric disorders at the proximate level, because that is needed to find the best possible treatment.


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I found the editorial by Kelleher et al\(^1\) both stimulating and thought provoking. However, it is important to bear in mind that a given characteristic must either promote or hinder an individual’s chances of survival and procreation if it is going to have an impact on natural selection. Even if the presence of a