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Adverse childhood experiences and theory of mind

The recent paper by Upthegrove *et al*[1] provided an extensive study evaluating the association of childhood events with later development of psychosis. Hypotheses were partially supported and demonstrated that although childhood trauma (e.g. family disruption, abuse) was not associated with the development of psychosis, the authors did find a significant association between childhood abuse and hallucinations that were mood congruent or abusive in their content. More specifically, the authors identified that childhood sexual abuse, experiencing a victimising event (such as bullying) and death of a loved one were all significantly linked to hallucinations, even after controlling for the effects of cannabis use. The authors also suggest that childhood events did not show any association with the development of depressive or persecutory delusions.

This observed result is suggested to question cognitive models of delusions, where the precipitator, i.e. childhood trauma, leads to a state of arousal in the individual, which leads to inner–outer confusion and can cause unusual sensory/perceptual experiences.[2] Cognitive models suggest that it is the cognitive biases around these unusual experiences, the search for meaning of these experiences, and the core beliefs about the self, others and the world, which lead to the explanation that gives rise to the 'threat belief' or persecutory delusion. Therefore the authors suggest that it may be later-life experiences rather than childhood experiences which feed into this model, leading to the delusion.

Instead, it is possible that the neuropsychological theory of mind[3] can provide an alternative explanation for the development of persecutory delusions, whereby the individual develops false beliefs about the intentions of other people due to impairments in mentalising, which leads to the development of the 'threat belief' or delusion.[4] Frith[3] proposed that patients with schizophrenia develop theory-of-mind skills appropriately, but suggested that they experience a loss of these skills during a psychotic episode. Further research into this area has demonstrated that theory-of-mind deficits are not simply 'state characteristics', as patients with schizophrenia in remission still display significantly impaired theory-of-mind skills compared with healthy controls, demonstrating that theory-of-mind impairments are likely to be trait characteristics.[5] It is possible that it is a combination of adverse childhood experiences and impaired theory-of-mind skills that have a cumulative effect of leading to persecutory delusions, hence the findings in Upthegrove *et al*'s study.

1 Upthegrove R, Chard C, Jones L, Gordon-Smith L, Forty L, Jones I, et al. Adverse childhood events and psychosis in bipolar affective disorder. *Br J Psychiatry* 2015; **206**: 191–7.

2 Freeman D, Garety PA, Kuipers E, Fowler D, Bebbington PE. A cognitive model of persecutory delusions. *Br J Clin Psychol* 2002; **41**: 331–47.

3 Frith CD. *The Cognitive Neuropsychology of Schizophrenia*. Lawrence Erlbaum Associates, 1992.

4 Blackwood NJ, Howard NJ, Bentall RP, Murray RM. Cognitive neuropsychiatric models of persecutory delusions. *Am J Psychiatry* 2001; **158**: 527–39.

5 Bora E, Yucel M, Pantelis C. Theory of mind impairment in schizophrenia: meta-analysis. *Schizophr Res* 2009; **109**: 1–9.

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Authors' reply: We would agree that deficits of theory of mind may prove one mechanism that differentiates pathways to delusional belief rather than hallucinations. Indeed, a symptom-specific approach to research across diagnostic groups is increasingly called for. National Institute of Mental Health Research Domain Criteria encourage this approach, and there is already a volume of research on the causes of hallucinations, and childhood experiences therein.[1–3] Persecutory delusional beliefs – and a specific neurobiology for these – have also been investigated, with significant results centring around salience, prediction error and social cognitive processing.[4–6] Further focus on the distinction between primary and secondary delusions, and those with content outside the persecutory, is also needed.

1 Insel T, Cuthbert B, Garvey M, Heinssen R, Pine DS, Quinn K, et al. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *Am J Psychiatry* 2010; **167**: 748–51.

2 Sitko K, Bentall RP, Shevlin M, Sellwood W. Associations between specific psychotic symptoms and specific childhood adversities are mediated by attachment styles: an analysis of the National Comorbidity Survey. *Psychiatry Res* 2014; **217**: 202–9.

3 Modinos G, Costafreda SG, van Tol MJ, McGuire PK, Aleman A, Allen P. Neuroanatomy of auditory verbal hallucinations in schizophrenia: a quantitative meta-analysis of voxel-based morphometry studies. *Cortex* 2013; **49**: 1046–55.

4 Upthegrove R, Ross K, Brunet K, McCollum R, Jones L. Depression in first episode psychosis: the role of subordination and shame. *Psychiatry Res* 2014; **217**: 177–84.

5 Blackwood NJ, Howard RJ, Bentall RP, Murray RM. Cognitive neuropsychiatric models of persecutory delusions. *Am J Psychiatry* 2001; **158**: 527–39.

6 Corlett PR, Fletcher PC. Delusions and prediction error: clarifying the roles of behavioural and brain responses. *Cogn Neuropsychiatry* 2015; **20**: 95–105.

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Identification, diagnosis and treatment of prosopagnosia

Prosopagnosia is characterised by impaired face recognition in the absence of brain injury. There is a growing corpus of research on prosopagnosia, which helps elucidate the neurocognitive mechanisms underlying typical and atypical face perception.[1] Prevalence rates are estimated to be as high as 2% and it can have far-reaching psychosocial consequences. However, despite consideration as a 'neurodevelopmental disorder', prosopagnosia