The clinical relevance of appraisals of psychotic experiences

It is not psychotic experiences in themselves but the way in which we appraise, or make sense of, them that determines their clinical relevance, and provides the key focus of psychological therapy. Psychotic experiences do not inevitably cause distress, impair functioning or result in psychiatric diagnosis. Extensive empirical findings indicate that these experiences can occur in the absence of a "need for care"[1].

What, therefore, determines clinical pathological outcomes? Cognitive models of psychosis[2] outline how the appraisals which people make shape both the content of psychotic experiences and the meaning that is attributed to them, bridging the gap between phenomenological and neurobiological accounts of their occurrence[3]. Characteristic appraisals, for example, of psychotic experiences as betokening threat, and rendering the self as vulnerable or worthless, are associated with need for care. These appraisals in turn are influenced by the psychological (i.e., cognitive, affective and behavioural) processes which have developed in the context of a person's genes, biology and socio-environmental experiences[4].

A case example illustrates our proposition. James grew up in poverty, experienced bullying and was raped during his teenage years. These early experiences led to distressing beliefs that he was weak and others would harm him, and he tended to be alert to potential threats. As adolescence developed into adulthood, jobless, James became increasingly isolated and rarely went outside. James felt very on-edge, and his sleep was disturbed. One day, he heard whispers that sounded critical, which he was sure were people talking about him. He became more anxious and struggled to take care of himself. He started using cannabis. The voices suddenly got more intense, telling him "you are nothing and are going to get it". James just knew this was a sign he would never escape others' persecution, and he became even more guarded and avoidant. James felt completely helpless and had no hope for his future.

James's difficulties highlight how adverse life experiences contribute to negative appraisals about the self and others, which can – in the presence of a range of affective, cognitive, behavioural, social and biological factors – trigger and shape psychotic experiences and the meaning that is attributed to them. James's voices reflect the themes of how he views himself and others; and his appraisals ("I am cursed") and their consequences ("I am helpless") also mirror his negative beliefs.

But note it is not just the content of appraisals that is of clinical relevance, but also the processes by which people reach such conclusions and how they react to them. A certain type of thinking style, *fast thinking*[5], is particularly associated with threatening appraisals in psychosis, and is characterized by a tendency to "jump to conclusions", to have high conviction in one's instincts, and to fail to consider alternative explanations[6]. Worry and ruminative thinking further maintain distressing interpretations, together with threat-focused attention, memory biases and understandable, but unhelpful, avoidant "safety behaviours" which act to prevent disconfirmation of fears[6].

The focus of cognitive-behavioural therapy for psychosis (CBTp) is therefore on understanding and exploring these appraisals of psychotic experiences and the thinking contributing to them, with the goal of supporting people to become less distressed and more able to live a personally meaningful life. The evidence base for CBTp is now consistent in demonstrating benefits for psychotic symptoms[7]. Developing trust and safety in the therapeutic relationship is the foundation of CBTp, as for other therapies, and requires skilful competence, given the nature of people's beliefs and the marked interpersonal difficulties they have often experienced.

An empathic and collaborative approach is essential, conveying a spirit of open enquiry, including the "suspension of disbelief" regarding the veracity of appraisals[8]. Directly challenging these appraisals and presenting contradictory evidence is counter-therapeutic, as it risks invalidating people's subjective experience, and may paradoxically increase their conviction and distress.

However, empathic engagement alone is insufficient to bring about clinically significant improvements in people with psychosis. A key mechanism of change in CBTp, consistent with psychodynamic approaches, is the development of reflective functioning or the ability to make sense of one's own mind and that of others, in order to understand behaviour[9]. Specifically, belief flexibility or s*low thinking* is fundamental to adaptive psychological functioning, and involves reflective curiosity and generation of alternative ideas[5]. There is now evidence that therapy which targets improvements in belief flexibility specifically diminishes paranoia[10].

So, whilst a developmental perspective is valuable in aiding self-understanding, the key therapeutic focus is on identifying and modifying day-to-day cycles which maintain occurrence of distressing appraisals of psychotic experiences. As well as fast thinking processes, these include sensitivity to stress, threat anticipation, negative affect, ruminative worrying and safety behaviours[6].

The synthesis of an individualized narrative provides an account of the range of probable factors that contribute to distressing appraisals, with the goal of increasing people's awareness of the mechanisms by which they attribute meaning to their experiences. CBTp can be seen as a process of "sowing seeds" to support the germination of alternative, less distressing explanations, which over time become more adaptive appraisals of psychotic experiences[11]. This then supports behavioural experimentation in daily life, to explore the impact of modifying these and trying out different ways of managing stressful, but valued activities, with experiential learning gradually reinforcing safer appraisals of experience.

CBTp mirrors the naturalistic process through which we derive meaning from our life experiences to support adaptive functioning. However, sustaining this without support, given heightened vulnerability to stress, is a significant challenge. An important target for future research is the facilitation of enduring generalization of therapy gains to everyday life. To address

this, our research team is trialling a digital therapy called *SlowMo* that targets problematic fast thinking to modify distressing appraisals of psychotic experiences and thereby reduce paranoia[10]. A *SlowMo* mobile app (see www.slowmotherapy.co.uk) assists people to *slow down for a moment* in their daily life to notice new information and develop safer thoughts, thereby aiming to optimize the clinical relevance of adaptive appraisals of psychotic experiences to real life.

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Mating, sexual selection, and the evolution of schizophrenia

For over fifty years, evolutionary theorists have sought to understand the biological roots of our species' vulnerability to schizophrenia – a debilitating disorder that has a relatively high incidence despite being associated with markedly reduced fertility (the so-called "schizophrenia paradox"). While some models treat the entire spectrum of schizophrenia as a manifestation of biological dysfunction, others postulate that psychosis proneness (schizotypy) or even psychotic symptoms may confer adaptive benefits through enhanced survival or reproduction (or they used to do so during our evolutionary history)[1].

Adaptive models of this kind face some formidable challenges. In addition to the low fertility of patients – which is not balanced out by that of their close relatives – and the evidence of reduced IQ and neural integrity in schizophrenia, they need to account for the role played by deleterious *de novo* mutations (including rare copy number variations), which explain a larger share of schizophrenia risk than common genetic variants[1,2].

Schizophrenia is a heterogeneous category, and any comprehensive explanation is likely to require a combination of models. At the same time, theory and evidence increasingly point to mating as a contributing factor in the evolution of psychosis proneness. The sexual selection model (SSM) was first advanced by Nettle[3] and refined by Shaner et al[4]. According to this model, schizophrenia is a maladaptive condition, but schizotypal traits – in particular positive schizotypal traits such as magical thinking, ideas of reference, and unusual perceptual experiences – are associated with enhanced verbal and artistic creativity and, as a result, lead to increased success in courtship and mating. The hypermentalistic cognitive style of schizotypal individuals involves a heightened focus on others' thoughts and emotions, which may also contribute to courtship success[5,6]. Consistent with this hypothesis, several studies have shown that positive schizotypy is associated with artistic creativity, a larger number of sexual partners, and a preference for uncommitted sexual relatioships[7]. Also, a moderate degree of reduction in white matter integrity has been linked to creative thinking and imagination[8].

But how does this model account for the role of rare mutations in schizophrenia? Most sexually selected traits are fitness indicators in that they correlate with the organism's underlying condition, including good nutrition, absence of parasites, low levels of harmful mutations, and so on. Other traits may evolve as amplifiers by further increasing the condition sensitivity of fitness indicators. In a nutshell, the SSM hypothesizes that verbal and artistic creativity are fitness indicators, whereas schizotypy functions at least in part as an amplifier trait[4]. In other words, high schizotypy increases the risk of schizophrenia in people who carry many harmful mutations and/or are exposed to high levels of stress and infections; however, the same traits boost mating success in people with low mutation load and few developmental stressors. Of course, contraception and other evolutionary novel aspects of modern societies may attenuate or break the link between mating success and actual reproduction.

The SSM potentially explains the logic of several risk factors for schizophrenia, from harmful mutations and low IQ (which is also affected by mutation load, especially at the low end of the distribution) to early infections and stressful life events. In addition, specific stressors such as migration into a minority population may partly operate by exacerbating competition for mates in adolescence and early adulthood. Most importantly, the SSM offers a potential solution to the paradox of low fertility in patients and their close relatives. According to the model, the low fertility of patients is not caused by schizotypy alone, but rather by the interaction between schizotypy and fitness-reducing factors such as mutations and adversity. Close relatives of schizophrenics are likely to share some of the same factors, both genetic and environmental. As a result, they can also be expected to show reduced fertility, though less dramatically so than patients[9]. If the model is correct, the crucial comparison would be that between the close relatives of schizophrenics and people with similarly high levels of schizotypy but without a diagnosed relative.

At the genetic level, it is important to appreciate that the SSM postulates the existence of at least two distinct sources of