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

schizophrenia risk: a) rare and *de novo* mutations, which are overwhelmingly harmful and subject to negative selection; and b) schizotypy-increasing alleles, which should be relatively common and evolve under balancing selection (a regime of alternating positive and negative selection on the same allele)[9]. Common variants that influence IQ may represent a third independent source of risk. The SSM also predicts a unique pattern of genotype-by-genotype interaction – namely, the same deleterious mutations should have a stronger effect on the risk for schizophrenia when they occur on a background of schizotypy-increasing common variants. To my knowledge, this hypothesis has never been tested in genetic research. A recent study found that strong negative selection on rare mutations contributes to maintain variation in other, physically close genes on the same chromosomes[2]. However, the authors did not test whether balancing selection may also contribute to maintain a certain amount of common genetic variation, independent of deleterious mutations.

Mating is only one component of an organism's fitness, and needs to be balanced against other critical tasks. Examples are skills acquisition, feeding, and protection of the offspring. The decisions made in allocating time and energy to these investments determine an individual's life history strategy. Life history strategies have wide-ranging implications for personality, behavior, and physiology. In humans, "fast" strategies are associated with heightened mating effort, precocious sexuality, low investment in stable couple relationships (which are conducive to parenting effort), impulsivity and risk-taking, and broad personality traits such as low agreeableness and conscientiousness. "Slow" strategies are associated with lower mating and higher parenting effort, delayed sexuality, fewer partners, self-control and risk aversion, and high agreeableness and conscientiousness.

Life history concepts can be used to develop a broad-band evolutionary taxonomy of mental disorders[10]. In this framework, schizophrenia spectrum disorders can be classified as fast spectrum conditions, together with borderline personality disorder, antisocial and conduct disorders, and eating disorders marked by behavioral dysregulation. Above and beyond their differences, these disorders share a functional link with fast life history-related traits such as heightened mating effort and impulsivity, and form a comorbidity network with common risk factors and developmental correlates[6,7,10]. They can be contrasted with slow spectrum conditions, such as obsessive-compulsive personality disorder, at least a subtype of autism spectrum disorder (mainly in the high-functioning range), and eating disorders characterized by elevated conscientiousness and self-control.

The taxonomy sketched above is still provisional and open to substantial revisions. Even so, simulations show that the life history model is already capable of reproducing the large-scale empirical structure of mental disorders, including the internalizing-externalizing distinction and the emergence of a general "p factor" of psychopathology[10]. A life history approach recasts the SSM within a broader theoretical framework and integrates its insights with those of other evolutionary models, such as the diametrical model of autism and psychosis advanced by Crespi and Badcock[5]. Together, these developments are starting an exciting new chapter in the evolutionary study of schizophrenia, with novel predictions to test and unexplored implications for epidemiology, prevention, and treatment.

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Validity and utility of the general factor of psychopathology

Psychopathology can be viewed as a variety of symptoms that are organized into first-order dimensions by their correlations. Critically, these first-order dimensions are themselves robustly correlated[1]. These correlations are problematic for categorical taxonomies[2], but provide essential information about the nature of psychopathology[3-5]. Correlations among first-order dimensions vary in magnitude, with stronger correlations among some dimensions yielding second-order factors, particularly internalizing and externalizing factors[6].

These second-order factors do not completely capture the correlations among dimensions of psychopathology, however. Rather, second-order internalizing and externalizing factors are themselves substantially correlated. We provided evidence that the correlations between internalizing and externalizing factors can be explained by a general factor of psychopathology on which every first-order dimension loads[7]. This finding has been replicated many times across the lifespan[4]. Most studies examined only prevalent forms of psychopathology, but several showed that bipolar disorder, schizophrenia and autism are strongly related to the general factor of psychopathology, suggesting that this factor is very general indeed[4].

Before deciding that the general factor of psychopathology is useful, we must know if it is only an artifact of systematic measurement error. The general factor almost certainly partly reflects nuisance correlations due to the same informant reporting on all psychopathology dimensions, but it must also capture something