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Social Predictors of Psychotic Experiences: Specificity and Psychological Mechanisms

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It has become widely accepted that the psychotic disorders are endpoints of atypical developmental trajectories indexed by abnormal emotional and cognitive development early in life. However, the role of environmental factors in determining these trajectories has received relatively little attention. In this article, we argue that (1) the influence of environment on psychosis can best be understood if we focus on specific types of psychotic experiences such as hallucinations and delusions, (2) these symptoms are the products of specific cognitive biases and deficits, and (3) the development of these particular patterns of cognitive functioning is influenced by specific kinds of environmental adversity. This approach is at variance with more conventional approaches because it suggests that each type of experience, rather than being the manifestation of a common underlying illness process, is a product of a specific set of causal variables. Importantly, these variables include environmental determinants, although not to the exclusion of endogenous factors such as neurodevelopmental impairment or genetic vulnerability. We discuss the implications of this approach for neurobiological and genetic research into psychosis, as well as clinical practice.

Key words: hallucinations/delusions/trauma/victimization/sexual abuse

Research into psychotic experiences has usually been guided by the assumption that they are symptoms of discrete diseases such as schizophrenia. The limitations of this paradigm are well known. There appears to be a continuum between psychotic and ordinary experiences,[1–3] and recent epidemiological studies have shown that about 10% of the population has experienced hallucinations and delusions[4–6] compared with an estimated lifetime risk of schizophrenia of under 1%[7] and an estimated annual incidence rate varying between 5/100 000 and 43/100 000.[8] Psychotic experiences do not cluster into syndromes corresponding to diagnoses such as schizophrenia and bipolar disorder, and many patients experience symptoms of more than one diagnosis.[9,10] The search for vulnerability genes for specific psychiatric disorders has not yielded consistent findings.[11,12] At best, vulnerability to psychosis is associated with many genes of small effect, most linked to more than one diagnosis.[13,14] It seems increasingly likely that these genes interact with environmental factors.[15] Studies of the psychological mechanisms involved in different types of psychotic experience suggest that they are relatively uninfluenced by broad cognitive deficits thought to be indicative of neuropsychological impairment, which are also diagnostically nonspecific.[16–18] Rather, as we will discuss later, each type of experience appears to be the product of specific biases in reasoning and cognition.

The Influence of Adversity on Psychosis

It is widely accepted that psychotic symptoms are the endpoints of abnormal developmental trajectories.[19,20] Although the role of environmental influences and developing psychological processes in determining these trajectories has been neglected,[21] a growing body of evidence suggests that experiences of adversity may play an important role. For example, the risk of being diagnosed as psychotic is increased by × 4-8 in ethnic minorities living in the United Kingdom[22,23] and elsewhere,[24–28] and this effect is almost certainly a consequence of environmental influences.[29] Incidence rates are greatest in those immigrants who are living in neighborhoods in which they form a clear minority,[30,31] suggesting that discrimination,[32,33] experiences of social defeat and powerlessness,[34] and/or lack of social support may be important in conferring risk of illness.

Recent research has also confirmed an association between exposure to the urban environment, especially in early life, and both clinical[35,36] and subclinical psychosis.[37] Ethnicity, drug use, neuropsychological impairment, birth complications, and childhood socioeconomic status have been ruled out as mediating variables,[38]

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suggesting again that social factors must be important. In this context, it is worth noting that many urban areas probably provide the toxic social circumstances that have been implicated in the high rates of psychosis found in immigrant communities, eg, experiences of victimization and powerlessness.[39]

Although researchers studying familial expressed emotion have often been at pains to argue that "We consider that families do not exert a *causal* influence" on the development of psychosis,[40] it seems likely that aspects of family relationships are indeed relevant. One line of research has explored the importance of relationships with attachment figures. An insecure attachment style has been reported in association with psychotic symptoms in both nonclinical[41–46] and patient samples.[47,48] Although these findings are based on cross-sectional comparisons in which participants' current styles of relating to others are assessed rather than the quality of past relationships, prospective data suggest that disrupted attachment relations may be causal. In a birth cohort study, risk of psychosis in adulthood was raised by a factor of 4 if the mother, during pregnancy, reported that a baby was unwanted.[49] Separation from parents in early life has been found to predict an increased risk of psychosis in genetically vulnerable children,[50,51] and the association between immigrant status and severe mental illness may be at least partially explained by the high rates of early separation in migrant populations.[52] Adolescents at high genetic risk of psychosis have also been found to be at increased risk of psychosis in later life if they report adverse relationships with their parents.[53]

A second line of research into family functioning has implicated "parental communication deviance," a style of communicating with offspring that is vague, fragmented, and contradictory.[54,55] Although early studies of this phenomenon were criticized on methodological grounds,[56] it was later reported that parental communication deviance and criticism/hostility predicted later psychosis among nonpsychotic child guidance attendees,[57,58] reflecting bidirectional interactions between psychopathology in the children and parental behavior.[59] More recently, a Finnish adoption study found that children at genetic risk of psychosis were more likely to become psychotic in later life if raised by adoptive parents with communication deviance.[60,61]

People with psychosis also report very high rates of trauma prior to illness.[62–65] Sexual abuse has been specifically investigated, although most studies have been criticized on the grounds of poor methodology.[66] In a recent epidemiological study, it was reported that the probability of experiencing psychosis given a history of sexual abuse was approximately 15 times greater than the probability without such a history.[67] There is also evidence that psychotic patients with a history of trauma often experience persistent interpersonal difficulties that may prevent them from engaging effectively with services, thereby preventing them from obtaining long-term benefit from treatment.[68]

These studies have almost all involved grouping people according to diagnoses such as schizophrenia. However, some of these effects may be symptom specific. Several studies have reported that early trauma, and especially childhood sexual abuse, specifically increases the risk of later hallucinations in both schizophrenia and bipolar patients.[69–73] On the other hand, insecure attachment appears to be specifically associated with paranoia and not hallucinations.[45,46] Evidence that discrimination or victimization plays a specific role in the development of paranoid beliefs has emerged from a population survey in the United States and Mexico,[39] from a prospective population-based study in Holland,[32] and from patients' retrospective reports of their experiences of intrusive[74,75] and threatening[76] life events (as noted above, this effect may contribute to the elevated rates of psychosis in immigrant populations). Finally, a specific association has been reported between thought disorder and communication deviance in parents,[77] reflecting an interaction between environmental transmission and genetic vulnerability.[78]

These associations between different kinds of adversity and specific symptoms can only be understood in the context of the psychological mechanisms thought to be important in each type of symptom. In the following 2 sections we discuss, in particular, recent research on those processes implicated in auditory-verbal hallucinations (AVHs) and paranoid belief systems.

Adversity and AVHs

Physiological data indicate that AVHs are accompanied by subvocalization (covert activation of the speech muscles) and activations of brain systems involved in the generation and monitoring of speech.[79–84] These observations have led many investigators to conclude that AVHs arise when inner speech is misattributed to a source that is alien to the self.[85–87] Consistent with this account, people with AVHs, when engaged in source-monitoring tasks (in which they are required to discriminate between externally generated and self-generated words), show a bias toward assuming that the source of their experiences is external to the self[88,89] especially when attending to emotionally salient material[90,91] (see [92] for a review). There is evidence that the source-monitoring judgments of people with AVHs are influenced by top-down processes, such as expectations about what kinds of events are likely to occur.[93–96] Like obsessive-compulsive patients suffering from intrusive thoughts,[97] patients with AVHs also report abnormal metacognitive beliefs (eg, the belief that the failure to control one's own thoughts is catastrophic), leading them to use dysfunctional strategies in attempts to regulate their own mental processes.[98–100]

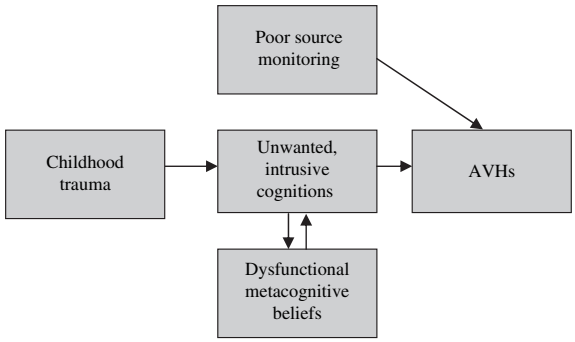

Fig. 1. Hypothesized Pathway From Childhood Trauma to Auditory-Verbal Hallucinations.

These findings point to a tentative explanatory model of the association between trauma and AVHs in which it is assumed that poor source monitoring is a vulnerability factor for this symptom (see figure 1). Research on post-traumatic stress disorder shows that traumatic experiences can lead to intrusive thoughts that occur spontaneously, without cognitive effort.[101] Intrusive thoughts of this kind are especially likely to occur during periods of stress, during which inner speech takes an expanded dialogic form.[102] Studies with healthy people show that low-effort cognitions are most difficult to source monitor.[103] Hence, individuals who are poor at source monitoring are likely to misattribute the self-generated mental contents that are the sequelae of trauma. The abnormal metacognitive beliefs apparent in people with AVHs may be a causal factor in their use of self-defeating strategies (such as thought suppression) to control these kinds of thoughts.[99]

Adversity and Paranoid Delusions

A different profile of cognitive biases and deficits is evident in the case of paranoid delusions. A core process in this kind of belief system is the expectation that negative interpersonal interactions will be experienced in the future.[104–106] Research has highlighted a number of psychological mechanisms that might lead to this expectation.

Psychotic patients tend to "jump to conclusions" on tasks in which they are required to construct hypotheses on the basis of sequentially presented information,[107–109] an effect that becomes more pronounced when reasoning about emotionally salient material.[110,111] Although the cause of this bias is poorly understood, there is evidence that this bias is specifically related to delusions.[112,113]

It has also been suggested that beliefs about persecution might arise as a consequence of losing the ability to understand the mental states of others, leading to the assumption that others have malign intentions toward the self.[114] Consistent with this hypothesis, some studies have found "theory of mind" (ToM) deficits in patients with paranoid delusions[113,115–117] although they have also been reported in patients suffering from other psychotic symptoms,[118,119] raising questions about their specificity.[120]

There is more consistent evidence that self-esteem–related processes play an important role in paranoid delusions. Trower and Chadwick[121] have argued that it is important to distinguish between patients with "bad-me" paranoia (in which persecution is believed to be deserved) and "poor-me" patients who believe that their persecution is undeserved. They have reported that negative beliefs about the self are prominent in the former group but defended against in the latter group.[122] However, the status of this distinction is controversial because bad-me beliefs seem to be rare in psychiatric samples.[123] In a recent longitudinal study, the majority but not all of psychotic patients had poor-me beliefs, but over time patients sometimes changed in their estimation of the extent to which they deserved to be persecuted.[124] We have found highly negative self-esteem in all paranoid patients,[106] although (consistent with previous research[122]) the effect was weaker in poor-me than bad-me patients. We have also used longitudinal methods to show that the self-esteem of paranoid patients is highly unstable over periods of minutes[125] and years.[126] In a nonclinical study, negative self-esteem partially mediated the association between insecure attachment and paranoia.[45]

Several investigators have found that paranoid patients report an extreme external-personal locus of control,[127–129] and, using the related construct of attributional (explanatory) style, it has been shown that they tend to attribute negative events to causes external to the self[127,130,131] especially those that implicate the intentional actions of others rather than situational factors.[132] This effect for attributions seems to be restricted to patients who suffer from poor-me delusions[124] and/or who are both paranoid and grandiose[133] and is not found in nonclinical paranoid samples.[134–136]

These observations have led to attempts to construct integrative theories by exploring the relation between the different kinds of psychological abnormalities that have been observed; eg, by studying whether ToM deficits[137] or hasty decision making[138] affect the way that individuals with negative self-esteem explain and anticipate negative events. In a recent, large-scale study that attempted to investigate a range of psychological mechanisms in relation to paranoia in a transdiagnostic sample (schizophrenia spectrum patients and patients with major depression), it was found that all the above-mentioned processes (jumping to conclusions, poor ToM skills, negative self-esteem, and attributional abnormalities) contributed to persecutory delusions.[139]

These findings point to a tentative explanatory model of the association between insecure attachment, experiences of chronic victimization, and paranoid beliefs (see figure 2). Insecure attachment in adulthood is associated with low self-esteem and difficulty in trusting others.[140] Especially in individuals with the preoccupied and fearful

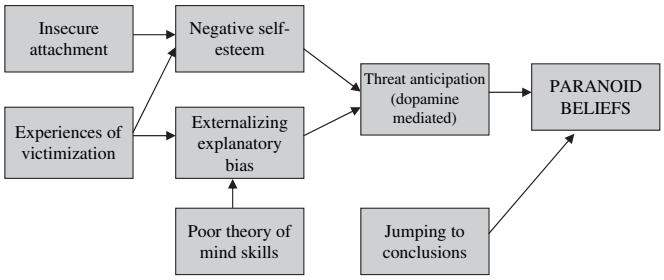

Fig. 2. Hypothesized Pathway From Adversity to Paranoid Beliefs.

attachment styles (which are associated with a negative model of the self[141]), repeated experiences of victimization are likely to exacerbate negative self-esteem while provoking an externalizing explanatory style in which negative events are assumed to be caused by powers external to the self. This is especially likely to be the case if, in the absence of well-developed ToM skills, the individual is unable to attribute the negative actions of others to situational factors.[137] (Consistent with this part of this model, a genetic high-risk study has reported that, in adolescents at high genetic risk of psychosis, an external locus of control predicted the later development of illness.[142]) These characteristics will, in turn, lead to a tendency to anticipate social threats and hence paranoid beliefs. (Elsewhere, we have argued that the computational processes underlying the stage of threat anticipation in this model may be implemented by striatal dopamine neurons.[143,144]) Finally, we hypothesize that a jumping-to-conclusions style of reasoning will prevent reality testing and will therefore serve to maintain paranoid beliefs once established.

Implications for Neurobiological Research

The accounts we have given of auditory hallucinations and paranoid delusions suggest plausible pathways that explain how specific symptoms arise understandably from specific kinds of adversity. They are consistent with some previous accounts that have emphasized the impact of adversity on biological and psychological functioning, eg, the traumagenic neurodevelopmental model of schizophrenia of Read et al,[145] except that we have argued for a focus on specific types of behavior and experience—symptoms—rather than broad diagnoses. In addition to bridging Jaspers'[146] distinction between causal explanation and understanding, models of the kind we have proposed have a number of important virtues. First, they help explain why adversity sometimes leads to psychosis and sometimes does not (because specific types of adversity are hypothesized to be associated, through interaction with other variables, with specific symptoms rather than with broad diagnoses such as "schizophrenia"). Second, they suggest specific hypotheses that can be tested in future retrospective and longitudinal investigations. Third, they have important implications for research into the neurobiology of psychosis.

In this context, it is important to note that the models we have suggested do not give primacy to either environmental or biological variables. Although many in psychiatry would sign up to Engell's[147] biopsychosocial framework in principle, there has been too often a tendency to assume that these 2 domains of explanation represent different universes of causation, leading to "a mechanical notion that admits the influence of both biology and experience but insists on dividing the total variance into percentages."[148] This kind of dualism is evident, eg, in many diathesis-stress[149] or 2-hit[150] models of psychosis onset.

For progress to be achieved, it will be necessary to recognize and explore more complex relationships between environmental and biological processes. For example, substantial efforts have been directed to the detection of both structural and functional neuroanatomical abnormalities in psychotic patients. One obvious implication of the account we have given—that researchers might explore the extent to which these abnormalities are related to specific symptoms—is already widely accepted. Electrophysiological[80] and magnetic resonance studies[151] have been used to study the neuropsychological mechanisms that lead inner speech to be misattributed to an external source in the case of hallucinations. However, a perhaps less obvious implication is that life history may be an important confound that will need to be controlled for in order to understand the way that brain abnormalities contribute to psychosis. For example, structural neuroimaging studies of victims of sexual abuse and other traumas have sometimes reported findings similar to those reported in psychotic patients, such as thinning of the corpus callosum,[152,153] loss of volume in the anterior cingulate cortex,[154,155] and reduced hippocampal volume.[156,157] Similarly, in animal studies it has been demonstrated that chronic experiences of victimization lead to sensitization of the basal ganglia dopamine system,[34] which may help to explain abnormal dopaminergic functioning in acute psychosis.[158] As Read et al[145] have pointed out, it is therefore entirely possible that findings which are usually taken as evidence of endogenous dysfunction are in fact indices of more complex environment × neurobiology interactions. In future structural and functional neuroimaging studies, it will therefore be important to determine the extent to which the abnormalities observed in patients are the consequence of these kinds of interactions.

The observation that psychotic symptoms arise from specific kinds of adversity also has implications for genetic research, which has so far failed to yield replicable associations between specific genes and psychotic illness.[11,12] Some investigators have already tried to resolve inconsistencies in the literature by searching for genes relating to specific symptoms,[159] but it is too early to tell

whether this approach will be fruitful. More interestingly, the models we have outlined suggest ways of approaching genetic influences in psychosis that see them as resilience rather than risk factors, an approach that is already being explored with respect to nonpsychotic disorders.[160] Specifically, we have proposed that hallucinations and delusions are expected consequences of certain kinds of adversity, and it might therefore be fruitful to explore whether there are genes that protect against psychosis in these circumstances. For example, the A1 allele of the *DRD2-TAQ-IA* polymorphism is known to reduce D_2 receptor density by up to 30%, resulting in a reduced ability to learn to avoid negative consequences.[161] As we have argued that abnormal dopaminergic responses underlie the increased anticipation of threat and consequent avoidance behavior of paranoid patients,[143] it seems possible that this allele will protect against persecutory delusions. Testing a hypothesis of this kind would involve comparing chronically victimized nonparanoid individuals with patients suffering from persecutory delusions (in which the allele would be expected to have a close to zero prevalence) and would require different statistical models to those used to detect positive associations between genes and psychosis.

Clinical Implications

We have already noted that patients who have a history of trauma often fail to engage adequately with services, preventing them from obtaining long-term benefit from treatment.[68] It is possible that this happens because they feel that clinicians who do not acknowledge their experiences are unable to adequately address their needs. For this reason alone, all patients with psychosis should be assessed for early trauma, a task for which clinicians may need to receive special training.[162]

The account we have given of the relationship between adversity and psychosis has a number of further clinical implications, some of which may be counterintuitive. For example, if, as we have proposed, excessive dopamine-mediated threat anticipation is related to poor-me but not bad-me paranoid delusions, it follows that only poor-me delusions will respond to antipsychotics. (Consistent with this prediction, the psychotic symptoms of depressed patients appear to be poorly responsive to dopamine-blocking drugs.[163]) Less obviously, perhaps, if adversity leads to sensitization of the dopamine system,[34] it might be predicted that patients whose symptoms are a response to trauma will show a greater initial response to antipsychotics than patients whose psychosis is not trauma related. With respect to psychological therapies, our approach highlights the importance of identifying the role of adversity within any psychological formulation of an individual patient's difficulties prior to deciding on the focus of treatment. Recently, some cognitive behavior therapists have developed specific strategies for working with severely mentally ill patients when trauma is implicated in their difficulties.[164,165]

Accounts of mental illness that acknowledge the complex interplay between biological and environmental causal processes may also have implications for the way that mental health services are organized. It has been noted that, despite the widespread adoption of a biopsychosocial approach in principle, the pluralism evident in modern services is more often the consequence of negotiation between competing disciplines who favor different therapies (eg, pharmacological or psychological interventions) rather than of genuine attempts to forge integrative approaches to understanding and treating patients.[166] At worst, this leads to patients receiving multiple, poorly coordinated interventions irrespective of their needs. Hopefully, a better understanding of the pathways to psychosis will eventually lead to a more rational, scientifically based approach to treatment in which interventions are tailored according to their proven ability to influence processes that are known to be important in each particular patient's difficulties.

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