

## Social Predictors of Psychotic Experiences: Specificity and Psychological Mechanisms

Richard P. Bentall<sup>1,2</sup> and Charles Fernyhough<sup>3</sup>

<sup>2</sup>School of Psychology, University of Bangor, Gwynedd, UK;

<sup>3</sup>Department of Psychology, Durham University, Durham, UK

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,<sup>1-3</sup> and recent epidemiological studies have shown that about 10% of the population has experienced hallucinations and delusions<sup>4-6</sup> compared with an estimated lifetime risk of

schizophrenia of under 1%<sup>7</sup> and an estimated annual incidence rate varying between 5/100 000 and 43/100 000.<sup>8</sup> 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.<sup>9,10</sup> The search for vulnerability genes for specific psychiatric disorders has not yielded consistent findings.<sup>11,12</sup> At best, vulnerability to psychosis is associated with many genes of small effect, most linked to more than one diagnosis.<sup>13,14</sup> It seems increasingly likely that these genes interact with environmental factors.<sup>15</sup> 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.<sup>16-18</sup> 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.<sup>19,20</sup> Although the role of environmental influences and developing psychological processes in determining these trajectories has been neglected,<sup>21</sup> 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  $\times 4-8$  in ethnic minorities living in the United Kingdom<sup>22,23</sup> and elsewhere,<sup>24-28</sup> and this effect is almost certainly a consequence of environmental influences.<sup>29</sup> Incidence rates are greatest in those immigrants who are living in neighborhoods in which they form a clear minority,<sup>30,31</sup> suggesting that discrimination,<sup>32,33</sup> experiences of social defeat and powerlessness,<sup>34</sup> 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<sup>35,36</sup> and subclinical psychosis.<sup>37</sup> Ethnicity, drug use, neuropsychological impairment, birth complications, and childhood socioeconomic status have been ruled out as mediating variables,<sup>38</sup>

<sup>1</sup>To whom correspondence should be addressed; School of Psychology, University of Bangor, Brigantia Building, Penrallt Road, Bangor, Gwynedd LL57 2AS, UK; tel: +44 1248 383624, fax: +44 1248 382599. e-mail: richard.bentall@bangor.ac.uk.

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.<sup>39</sup>

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,<sup>40</sup> 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<sup>41–46</sup> and patient samples.<sup>47,48</sup> 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.<sup>49</sup> Separation from parents in early life has been found to predict an increased risk of psychosis in genetically vulnerable children,<sup>50,51</sup> 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.<sup>52</sup> 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.<sup>53</sup>

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.<sup>54,55</sup> Although early studies of this phenomenon were criticized on methodological grounds,<sup>56</sup> it was later reported that parental communication deviance and criticism/hostility predicted later psychosis among nonpsychotic child guidance attendees,<sup>57,58</sup> reflecting bidirectional interactions between psychopathology in the children and parental behavior.<sup>59</sup> 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.<sup>60,61</sup>

People with psychosis also report very high rates of trauma prior to illness.<sup>62–65</sup> Sexual abuse has been specifically investigated, although most studies have been criticized on the grounds of poor methodology.<sup>66</sup> 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.<sup>67</sup> 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,

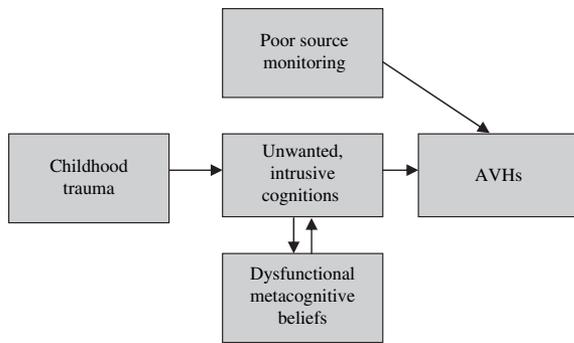
thereby preventing them from obtaining long-term benefit from treatment.<sup>68</sup>

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.<sup>69–73</sup> On the other hand, insecure attachment appears to be specifically associated with paranoia and not hallucinations.<sup>45,46</sup> 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,<sup>39</sup> from a prospective population-based study in Holland,<sup>32</sup> and from patients’ retrospective reports of their experiences of intrusive<sup>74,75</sup> and threatening<sup>76</sup> 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,<sup>77</sup> reflecting an interaction between environmental transmission and genetic vulnerability.<sup>78</sup>

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.<sup>79–84</sup> 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.<sup>85–87</sup> 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<sup>88,89</sup> especially when attending to emotionally salient material<sup>90,91</sup> (see <sup>92</sup> 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.<sup>93–96</sup> Like obsessive-compulsive patients suffering from intrusive thoughts,<sup>97</sup> 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.<sup>98–100</sup>



**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.<sup>101</sup> Intrusive thoughts of this kind are especially likely to occur during periods of stress, during which inner speech takes an expanded dialogic form.<sup>102</sup> Studies with healthy people show that low-effort cognitions are most difficult to source monitor.<sup>103</sup> 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.<sup>99</sup>

### 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.<sup>104–106</sup> 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,<sup>107–109</sup> an effect that becomes more pronounced when reasoning about emotionally salient material.<sup>110,111</sup> Although the cause of this bias is poorly understood, there is evidence that this bias is specifically related to delusions.<sup>112,113</sup>

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.<sup>114</sup> Consistent with this hypothesis, some studies have found “theory of mind” (ToM) deficits in patients with paranoid delusions<sup>113,115–117</sup> although they have also been reported in patients suffering from

other psychotic symptoms,<sup>118,119</sup> raising questions about their specificity.<sup>120</sup>

There is more consistent evidence that self-esteem-related processes play an important role in paranoid delusions. Trower and Chadwick<sup>121</sup> 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.<sup>122</sup> However, the status of this distinction is controversial because bad-me beliefs seem to be rare in psychiatric samples.<sup>123</sup> 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.<sup>124</sup> We have found highly negative self-esteem in all paranoid patients,<sup>106</sup> although (consistent with previous research<sup>122</sup>) 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<sup>125</sup> and years.<sup>126</sup> In a nonclinical study, negative self-esteem partially mediated the association between insecure attachment and paranoia.<sup>45</sup>

Several investigators have found that paranoid patients report an extreme external-personal locus of control,<sup>127–129</sup> 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<sup>127,130,131</sup> especially those that implicate the intentional actions of others rather than situational factors.<sup>132</sup> This effect for attributions seems to be restricted to patients who suffer from poor-me delusions<sup>124</sup> and/or who are both paranoid and grandiose<sup>133</sup> and is not found in nonclinical paranoid samples.<sup>134–136</sup>

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<sup>137</sup> or hasty decision making<sup>138</sup> 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.<sup>139</sup>

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.<sup>140</sup> Especially in individuals with the preoccupied and fearful

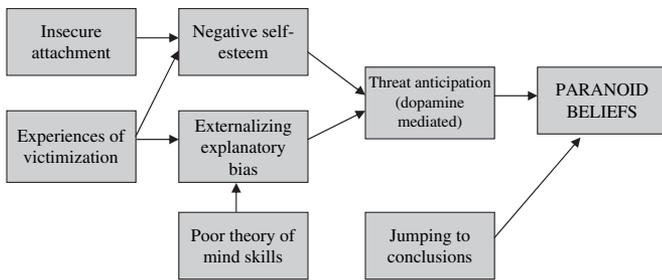


Fig. 2. Hypothesized Pathway From Adversity to Paranoid Beliefs.

attachment styles (which are associated with a negative model of the self<sup>141</sup>), 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.<sup>137</sup> (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.<sup>142</sup>) 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.<sup>143,144</sup>) 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,<sup>145</sup> 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<sup>146</sup> 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<sup>147</sup> 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.”<sup>148</sup> This kind of dualism is evident, eg, in many diathesis-stress<sup>149</sup> or 2-hit<sup>150</sup> 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<sup>80</sup> and magnetic resonance studies<sup>151</sup> 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,<sup>152,153</sup> loss of volume in the anterior cingulate cortex,<sup>154,155</sup> and reduced hippocampal volume.<sup>156,157</sup> Similarly, in animal studies it has been demonstrated that chronic experiences of victimization lead to sensitization of the basal ganglia dopamine system,<sup>34</sup> which may help to explain abnormal dopaminergic functioning in acute psychosis.<sup>158</sup> As Read et al<sup>145</sup> 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.<sup>11,12</sup> Some investigators have already tried to resolve inconsistencies in the literature by searching for genes relating to specific symptoms,<sup>159</sup> 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.<sup>160</sup> 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<sub>2</sub> receptor density by up to 30%, resulting in a reduced ability to learn to avoid negative consequences.<sup>161</sup> As we have argued that abnormal dopaminergic responses underlie the increased anticipation of threat and consequent avoidance behavior of paranoid patients,<sup>143</sup> 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.<sup>68</sup> 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.<sup>162</sup>

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.<sup>163</sup>) Less obviously, perhaps, if adversity leads to sensitization of the dopamine system,<sup>34</sup> 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.<sup>164,165</sup>

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.<sup>166</sup> 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.

### References

1. Chapman LJ, Chapman JP. Scales for rating psychotic and psychotic-like experiences as continua. *Schizophr Bull.* 1980; 6:477–489.
2. Chapman LJ, Chapman JP, Raulin ML. Scales for physical and social anhedonia. *J Abnorm Psychol.* 1976;85:374–382.
3. Claridge GS. Can a disease model of schizophrenia survive? In: Bentall RP, ed. *Reconstructing Schizophrenia*. London, UK: Routledge; 1990:157–183.
4. Poulton R, Caspi A, Moffitt TE, Cannon M, Murray R, Harrington H. Children's self-reported psychotic symptoms and adult schizophreniform disorder: a 15-year longitudinal study. *Arch Gen Psychiatry.* 2000;57:1053–1058.
5. Tien AY. Distribution of hallucinations in the population. *Soc Psychiatry Psychiatr Epidemiol.* 1991;26:287–292.
6. van Os J, Hanssen M, Bijl RV, Ravelli A. Strauss (1969) revisited: a psychosis continuum in the normal population? *Schizophr Res.* 2000;45:11–20.
7. Jablensky A. Schizophrenia: the epidemiological horizon. In: Hirsch SR, Weinberger DR, eds. *Schizophrenia*. Oxford, UK: Blackwell; 1995:206–252.
8. McGrath JJ. Myths and plain truths about schizophrenia epidemiology. *Acta Psychiatr Scand.* 2005;111:4–11.
9. Kendell RE, Brockington IF. The identification of disease entities and the relationship between schizophrenic and affective psychoses. *Br J Psychiatry.* 1980;137:324–331.
10. Kendell RE. The major functional psychoses: are they independent entities or part of a continuum? Philosophical and conceptual issues underlying the debate. In: Kerr A, McClelland H, eds. *Concepts of Mental Disorder: A Continuing Debate*. London, UK: Gaskell; 1991.
11. Crow TJ. The emperors of the schizophrenia polygene have no clothes. *Psychol Med.* 2008; 10.1017/S0033291708003395, Published online April, 21 2008.
12. Sanders AR, Duan J, Levinson DF, et al. No significant association of 14 candidate genes with schizophrenia in a large

- European ancestry sample: implications for psychiatric genetics. *Am J Psychiatry*. 2008;165:497–506.
13. Bramon E, Sham P. The shared genetic architecture which underlies schizophrenia and bipolar disorder. In: McDonald C, Schulze K, Murray RM, Wright P, eds. *Schizophrenia: Challenging the Orthodox*. London, UK: Taylor-Francis; 2004:173–181.
  14. Craddock N, O'Donovan MC, Owen MJ. The genetics of schizophrenia and bipolar disorder: dissecting psychosis. *J Med Genet*. 2005;42:193–204.
  15. McGuffin P. Nature and nurture interplay: schizophrenia. *Psychiatr Prax*. 2004;31:S189–S193.
  16. Green MF, Nuechterlein KH. Should schizophrenia be treated as a neurocognitive disorder? *Schizophr Bull*. 1999;25:309–319.
  17. Wykes T. Cognitive remediation is better than cognitive behaviour therapy. In: McDonald C, Schulze K, Murray RM, Wright P, eds. *Schizophrenia: Challenging the Orthodox*. London, UK: Taylor-Francis; 2004:163–171.
  18. Keefe RSE, Bilder RM, Harvey PD, et al. Baseline neurocognitive deficits in the CATIE schizophrenia trial. *Neuropsychopharmacology*. 2006;31:2033–2046.
  19. Jones PB, Rodgers B, Murray RM, Marmot MG. Child developmental risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet*. 1994;344:1398–1402.
  20. Marenco S, Weinberger DR. The neurodevelopmental hypothesis of schizophrenia: following a trail of evidence from cradle to grave. *Dev Psychopathol*. 2000;12:501–527.
  21. Bentall RP, Fernyhough C, Morrison AP, Lewis S, Corcoran R. Prospects for a cognitive-developmental account of psychotic experiences. *Br J Clin Psychol*. 2007;46:155–173.
  22. Harrison G, Owens D, Holton A, Neilson D, Boot D. A prospective study of severe mental disorder in Afro-Caribbean patients. *Psychol Med*. 1988;18:643–657.
  23. Fearon P, Kirkbride JB, Morgan C, et al. Incidence of schizophrenia and other psychoses in ethnic minority groups: results from the MRC AESOP study. *Psychol Med*. 2006;36(11):1541–1550.
  24. Bresnahan M, Begg M, Brown AS, et al. Race and risk of schizophrenia in a US birth cohort: another example of health disparity? *Int J Epidemiol*. 2007;36:751–758.
  25. Cantor-Graae E, Pedersen CB, McNeil TF, Mortensen PB. Migration as a risk factor for schizophrenia: a Danish population-based cohort study. *Br J Psychiatry*. 2003;182:117–122.
  26. Selten J-P, Veen N, Feller W, et al. Incidence of psychotic disorders in immigrant groups to The Netherlands. *Br J Psychiatry*. 2001;178:367–372.
  27. Smith GN, Boydell J, Murray RM, et al. The incidence of schizophrenia in European immigrants to Canada. *Schizophr Res*. 2006;87:205–211.
  28. Zolkowska K, Cantor GE, McNeil TF. Increased rates of psychosis amongst immigrants to Sweden: is migration a risk factor for psychosis? *Psychol Med*. 2001;31:669–678.
  29. Rutter M, Pickles A, Murray R, Eaves LJ. Testing hypotheses on specific environmental causal effects on behavior. *Psychol Bull*. 2001;127:291–324.
  30. Boydell J, van Os J, McKenzie J, et al. Incidence of schizophrenia in ethnic minorities in London: ecological study into interactions with environment. *Br Med J*. 2001;323:1–4.
  31. Veling W, Susser E, van Os J, Mackenbach JP, Selten JP, Hoek HW. Ethnic density of neighborhoods and incidence of psychotic disorders among immigrants. *Am J Psychiatry*. 2008;165:66–73.
  32. Janssen I, Hanssen M, Bak M, et al. Discrimination and delusional ideation. *Br J Psychiatry*. 2003;182:71–76.
  33. Veling W, Selten JP, Susser E, Laan W, Mackenbach JP, Hoek HW. Discrimination and the incidence of psychotic disorders among ethnic minorities in the Netherlands. *International Journal of Epidemiology*. 2007;36:761–768.
  34. Selten J-P, Cantor-Graae E. Social defeat: risk factor for psychosis? *Br J Psychiatry*. 2005;187:101–102.
  35. Pedersen CB, Mortensen PB. Evidence of a dose-response relationship between urbanicity during upbringing and schizophrenia risk. *Arch Gen Psychiatry*. 2001;58:1039–1046.
  36. Allardyce J, Boydell J. Review: the wider social environment and schizophrenia. *Schizophr Bull*. 2006;32:592–598.
  37. van Os J, Hanssen M, Bijl RV, Vollebergh W. Prevalence of psychotic disorder and community level of psychotic symptoms: an urban-rural comparison. *Arch Gen Psychiatry*. 2001;58:663–668.
  38. van Os J. Does the urban environment cause psychosis? *Br J Psychiatry*. 2004;184:287–288.
  39. Mirowsky J, Ross CE. Paranoia and the structure of powerlessness. *Am Sociol Rev*. 1983;48:228–239.
  40. Kuipers L, Birchwood M, McCreadie RD. Psychosocial family intervention in schizophrenia: a review of empirical studies. *Br J Psychiatry*. 1992;160:272–275.
  41. Berry K, Wearden A, Barrowclough C, Liversidge T. Attachment styles, interpersonal relationships and psychotic phenomena in a non-clinical student sample. *Pers Individ Dif*. 2006;41:707–718.
  42. Cooper ML, Shaver PR, Collins NL. Attachment style, emotion regulation, and adjustment in adolescence. *J Pers Soc Psychol*. 1998;74:1380–1397.
  43. MacBeth A, Schwannauer M, Gumley A. The association between attachment style, social mentalities, and paranoid ideation: an analogue study. *Psychol Psychother*. 2008;81:79–83.
  44. Mickelson KD, Kessler RC, Shaver PR. Adult attachment in a nationally representative sample. *J Pers Soc Psychol*. 1997;73:1092–1106.
  45. Pickering L, Simpson J, Bentall RP. Insecure attachment predicts proneness to paranoia but not hallucinations. *Pers Individ Dif*. 2008;44:1212–1224.
  46. Meins E, Jones SR, Fernyhough C, Hurndall S, Koronis P. Attachment dimensions and schizotypy in a non-clinical sample. *Pers Individ Dif*. 2008;44:1000–1011.
  47. Dozier M, Stevenson AL, Lee SW, Velligan DI. Attachment organization and familiar overinvolvement for adults with serious psychopathological disorders. *Dev Psychopathol*. 1991;3:475–489.
  48. Dozier M, Lee SW. Discrepancies between self and other-report of psychiatric symptomatology: effects of dismissing attachment strategies. *Dev Psychopathol*. 1995;7:217–226.
  49. Myhrman A, Rantakallio P, Isohanni M, Jones P. Unwantedness of pregnancy and schizophrenia in the child. *Br J Psychiatry*. 1996;169:637–640.
  50. Agid O, Shapira B, Zislin J, et al. Environment and vulnerability to major psychiatric illness: a case control study of early parental loss in major depression, bipolar disorder and schizophrenia. *Mol Psychiatry*. 1999;4:163–172.
  51. Parnas J, Teasdale TW, Schulsinger H. Institutional rearing and diagnostic outcome in children of schizophrenic mothers. A prospective high-risk study. *Arch Gen Psychiatry*. 1985;42:762–769.

52. Morgan C, Kirkbride J, Leff J, et al. Parental separation, loss and psychosis in different ethnic groups: a case-control study. *Psychol Med.* 2007;37:495–503.
53. Schiffman J, LaBrie J, Carter J, et al. Perception of parent-child relationships in high-risk families, and adult schizophrenia outcome of offspring. *J Psychiatr Res.* 2002;36:41–47.
54. Singer MT, Wynne LC. Thought disorder and family relations of schizophrenics IV. Results and implications. *Arch Gen Psychiatry.* 1965;12:201–212.
55. Singer MT, Wynne LC. Thought disorder and family relations of schizophrenics III. Methodology using projective techniques. *Arch Gen Psychiatry.* 1965;12:187–200.
56. Hirsch SR, Leff JP. Parental abnormalities of verbal communication in the transmission of schizophrenia. *Psychol Med.* 1971;1:118–127.
57. Goldstein MJ. The UCLA high-risk project. *Schizophr Bull.* 1987;13:505–514.
58. Goldstein MJ. Adolescent behavioral and intrafamilial precursors of schizophrenia spectrum disorders. *Int Clin Psychopharmacol.* 1998;13(suppl 1):101.
59. Cook WL, Strachan AM, Goldstein MJ, Miklowitz DJ. Expressed emotion and reciprocal affective relationships in disturbed adolescents. *Fam Process.* 1989;28:337–348.
60. Tienari P, Wynne LC, Sorri A, et al. Long term follow-up study of Finnish adoptees. *Br J Psychiatry.* 2004;184:214–222.
61. Wahlberg K-E, Wynne LC, Oja H, et al. Gene-environment interaction in vulnerability to schizophrenia: findings from the Finnish Adoptive Family Study of Schizophrenia. *Am J Psychiatry.* 1997;154:355–362.
62. Goodman LA, Rosenberg SD, Mueser K, Drake RE. Physical and sexual assault history in women with serious mental illness: prevalence, correlates, treatment, and future research directions. *Schizophr Bull.* 1997;23:685–696.
63. Greenfield SF, Strakowski SF, Tohen M, Batson SC, Kolbrener ML. Childhood abuse in first episode psychosis. *Br J Psychiatry.* 1994;165:415.
64. Mueser KT, Goodman LB, Trumbetta SL, et al. Trauma and posttraumatic stress disorder in severe mental illness. *J Consult Clin Psychol.* 1998;66:493–499.
65. Neria Y, Bromet EJ, Sievers S, Lavelle J, Fochtmann LJ. Trauma exposure and posttraumatic stress disorder in psychosis: findings from a first-admission cohort. *J Consult Clin Psychol.* 2002;70:246–251.
66. Bendall S, Jackson HJ, Hulbert C, McGorry PD. Childhood trauma and psychotic disorders: a systematic, critical review of the evidence. *Schizophr Bull.* 2008;34:568–579.
67. Bebbington P, Bhugra D, Bhugra T, et al. Psychosis, victimisation and childhood disadvantage: evidence from the second British National Survey of Psychiatric Morbidity. *Br J Psychiatry.* 2004;185:220–226.
68. Mueser KT, Rosenberg S, Goodman LA, Trumbetta SL. Trauma, PTSD, and the course of severe mental illness: an interactive model. *Schizophr Res.* 2002;53:123–143.
69. Ross CA, Anderson G, Clark P. Childhood abuse and the positive symptoms of schizophrenia. *Hosp Community Psychiatry.* 1994;42:489–491.
70. Read J, Agar K, Argyle N, Aderhold V. Sexual and physical abuse during childhood and adulthood as predictors of hallucinations, delusions and thought disorder. *Psychol Psychother.* 2003;76:1–22.
71. Hammersley P, Dias A, Todd G, Bowen-Jones K, Reilly B, Bentall RP. Childhood trauma and hallucinations in bipolar affective disorder: a preliminary investigation. *Br J Psychiatry.* 2003;182:543–547.
72. Kilcommons A, Morrison AP. Relationship between trauma and psychosis: an exploration of cognitive and dissociative factors. *Acta Psychiatr Scand.* 2005;112:351–359.
73. Shevlin M, Dorahy M, Adamson G. Childhood traumas and hallucinations: an analysis of the National Comorbidity Survey. *J Psychiatr Res.* 2007;41:222–228.
74. Harris T. Recent developments in the study of life events in relation to psychiatric and physical disorders. In: Cooper B, ed. *Psychiatric Epidemiology: Progress and Prospects.* London, UK: Croom Helm; 1987:81–102.
75. Day R, Neilsen JA, Korten A, et al. Stressful life events preceding the onset of acute schizophrenia: a cross-national study from the World Health Organization. *Cult Med Psychiatry.* 1987;11:123–206.
76. Fuchs T. Life events in late paraphrenia and depression. *Psychopathology.* 1999;32:60–69.
77. Docherty NM, Rhinewine JP, Labhart RP, Gordinier S. Communication disturbance and family psychiatric history in parents of schizophrenic patients. *J Nerv Ment Dis.* 1998;186:761–768.
78. Wahlberg KE, Wynne LC, Oja H, et al. Thought disorder index of Finnish adoptees and communication deviance of their adoptive parents. *Psychol Med.* 2000;30:127–136.
79. Dierks T, Linden DEJ, Jandi M, et al. Activation of Heschl's Gyrus during auditory hallucinations. *Neuron.* 1999;22:615–621.
80. Ford JM, Mathalon DH. Electrophysiological evidence of corollary discharge dysfunction in schizophrenia during talking and thinking. *J Psychiatr Res.* 2004;38:37–46.
81. Gould LN. Verbal hallucinations and activity of vocal musculature. *Am J Psychiatry.* 1948;105:367–372.
82. Inouye T, Shimizu A. The electromyographic study of verbal hallucination. *J Nerv Ment Dis.* 1970;151:415–422.
83. Jones SR, Fernyhough C. Neural correlates of inner speech and auditory verbal hallucinations: a critical review and theoretical integration. *Clin Psychol Rev.* 2007;27:140–154.
84. McGuire PK, Shah GMS, Murray RM. Increased blood flow in Broca's area during auditory hallucinations. *Lancet.* 1993;342:703–706.
85. Hoffman RE. Verbal hallucinations and language production processes in schizophrenia. *Behav Brain Sci.* 1986;9:503–548.
86. Bentall RP. The illusion of reality: a review and integration of psychological research on hallucinations. *Psychol Bull.* 1990;107:82–95.
87. Frith CD. *The Cognitive Neuropsychology of Schizophrenia.* Hillsdale, NJ: Lawrence Erlbaum; 1992.
88. Bentall RP, Slade PD. Reality testing and auditory hallucinations: a signal-detection analysis. *Br J Clin Psychol.* 1985;24:159–169.
89. Rankin P, O'Carroll P. Reality monitoring and signal detection in individuals prone to hallucinations. *Br J Clin Psychol.* 1995;34:517–528.
90. Morrison AP, Haddock G. Cognitive factors in source monitoring and auditory hallucinations. *Psychol Med.* 1997;27:669–679.
91. Johns LC, Rossell S, Frith C, et al. Verbal self-monitoring and auditory hallucinations in people with schizophrenia. *Psychol Med.* 2001;31:705–715.
92. Ditman T, Kuperberg GR. A source-monitoring account of auditory verbal hallucinations in patients with schizophrenia. *Harv Rev Psychiatry.* 2005;13:280–299.

93. Al-Issa I. Sociocultural factors in hallucinations. *Int J Soc Psychiatry*. 1978;24:167–176.
94. Al-Issa I. The illusion of reality or the reality of an illusion: hallucinations and culture. *Br J Psychiatry*. 1995;166:368–373.
95. Haddock G, Slade PD, Bentall RP. Auditory hallucinations and the verbal transformation effect: the role of suggestions. *Pers Individ Dif*. 1995;19:301–306.
96. Young HF, Bentall RP, Slade PD, Dewey ME. The role of brief instructions and suggestibility in the elicitation of hallucinations in normal and psychiatric subjects. *J Nerv Ment Dis*. 1987;175:41–48.
97. Wells A, Papageorgiou C. Relationships between worry, obsessive-compulsive symptoms and meta-cognitive beliefs. *Behav Res Ther*. 1998;36:899–913.
98. Morrison AP, Wells A. Metacognition across disorders: a comparison of patients with hallucinations, delusions, and panic disorder with non-patients. *Behav Res Ther*. 2003;41:251–256.
99. Jones SR, Fernyhough C. The roles of thought suppression and metacognitive beliefs in proneness to auditory verbal hallucinations in a non-clinical sample. *Pers Individ Dif*. 2006;41:1421–1432.
100. Garcia-Montes JM, Cangras A, Perez-Alvarez M, Fidalgo AM, Gutierrez O. The role of meta-cognitions and thought control techniques in predisposition to auditory and visual hallucinations. *Br J Clin Psychol*. 2006;45:309–317.
101. Brewin C. *Posttraumatic Stress Disorder: Malady or Myth?*. New Haven, Conn: Yale University Press; 2003.
102. Fernyhough C. Alien voices and inner dialogue: towards a developmental account of auditory verbal hallucinations. *New Ideas Psychol*. 2004;22:49–68.
103. Johnson MK, Hashtroudi S, Lindsay DS. Source monitoring. *Psychol Bull*. 1993;114:3–28.
104. Corcoran R, Ciummins S, Rowse G, et al. Reasoning under uncertainty: heuristic judgments in patients with persecutory delusions or depression. *Psychol Med*. 2006;36:1109–1118.
105. Kaney S, Bowen-Jones K, Dewey ME, Bentall RP. Frequency and consensus judgements of paranoid, paranoid-depressed and depressed psychiatric patients: subjective estimates for positive, negative and neutral events. *Br J Clin Psychol*. 1997;36:349–364.
106. Bentall RP, Kinderman P, Howard R, et al. Paranoid delusions in schizophrenia and depression: the transdiagnostic role of expectations of negative events and negative self-esteem. *J Nerv Ment Dis*. 2008;196:375–383.
107. Garety PA, Hemsley DR, Wessely S. Reasoning in deluded schizophrenic and paranoid patients. *J Nerv Ment Dis*. 1991;179:194–201.
108. John CH, Dodgson G. Inductive reasoning in delusional thought. *J Ment Health*. 1994;3:31–49.
109. Dudley REJ, John CH, Young AW, Over DE. Normal and abnormal reasoning in people with delusions. *Br J Clin Psychol*. 1997;36:243–258.
110. Dudley REJ, John CH, Young AW, Over DE. The effect of self-referent material on the reasoning of people with delusions. *Br J Clin Psychol*. 1997;36:575–584.
111. Young HF, Bentall RP. Probabilistic reasoning in deluded, depressed and normal subjects: effects of task difficulty and meaningful versus nonmeaningful materials. *Psychol Med*. 1997;27:455–465.
112. Fine C, Gardner M, Craigie J, Gold I. Hopping, skipping or jumping to conclusions? Clarifying the role of the JTC bias in delusions. *Cognit Neuropsychiatry*. 2007;12:46–77.
113. Corcoran R, Rowse G, Moore R, et al. A transdiagnostic investigation of theory of mind and jumping to conclusions in paranoia: a comparison of schizophrenia and depression with and without delusions. [Published online ahead of print November 16, 2007]. *Psychol Med*. 2007; 10.1017/S0033291707002152.
114. Frith C. Theory of mind in schizophrenia. In: David AS, Cutting JC, eds. *The Neuropsychology of Schizophrenia*. Hove, UK: Erlbaum; 1994:147–161.
115. Corcoran R, Cahill C, Frith CD. The appreciation of visual jokes in people with schizophrenia: a study of ‘mentalizing’ ability. *Schizophr Res*. 1997;24:319–327.
116. Craig J, Hatton C, Bentall RP. Persecutory beliefs, attributions and Theory of Mind: comparison of patients with paranoid delusions, Asperger’s Syndrome and healthy controls. *Schizophr Res*. 2004;69:29–33.
117. Frith C, Corcoran R. Exploring ‘theory of mind’ in people with schizophrenia. *Psychol Med*. 1996;26:521–530.
118. Drury VM, Robinson EJ, Birchwood M. ‘Theory of mind’ skills during an acute episode of psychosis and following recovery. *Psychol Med*. 1998;28:1101–1112.
119. Sarfati Y, Hardy-Bayles MC, Brunet E, Widloecher D. Investigating theory of mind in schizophrenia: influence of verbalization in disorganized and non-disorganized patients. *Schizophr Res*. 1999;37:183–190.
120. Brune M. ‘Theory of mind’ in schizophrenia: a review of the literature. *Schizophr Bull*. 2005;31:21–42.
121. Trower P, Chadwick P. Pathways to defense of the self: a theory of two types of paranoia. *Clin Psychol*. 1995;2:263–278.
122. Chadwick P, Trower P, Juusti-Butler T-M, Maguire N. Phenomenological evidence for two types of paranoia. *Psychopathology*. 2005;38:327–333.
123. Fornells-Ambrojo M, Garety P. Bad me paranoia in early psychosis: a relatively rare phenomenon. *Br J Clin Psychol*. 2005;44:521–528.
124. Melo S, Taylor J, Bentall RP. ‘Poor me’ versus ‘bad me’ paranoia and the instability of persecutory ideation. *Psychol Psychother*. 2006;79:271–287.
125. Thewissen V, Bentall RP, Lecomte T, van Os J, Myin-Germeys I. Fluctuations in self-esteem and paranoia in the context of everyday life. *J Abnorm Psychol*. 2008;117:143–153.
126. Thewissen V, Myin-Germeys I, Bentall RP, de Graaf R, Vollenberg W, van Os J. Instability in self-esteem and paranoia in a general population sample. *Soc Psychiatry Psychiatr Epidemiol*. 2007;42:1–5.
127. Kaney S, Bentall RP. Persecutory delusions and attributional style. *Br J Med Psychol*. 1989;62:191–198.
128. Lasar M. Cognitive evaluation of action in chronic schizophrenia: locus of control beliefs in an inpatient group. *Psychol Bietraege*. 1997;39:297–311.
129. Rosenbaum M, Hadari D. Personal efficacy, external locus of control, and perceived contingency of parental reinforcement among depressed, paranoid and normal subjects. *J Abnorm Psychol*. 1985;49:539–547.
130. Candido CL, Romney DM. Attributional style in paranoid vs depressed patients. *Br J Med Psychol*. 1990;63:355–363.
131. Fear CF, Sharp H, Healy D. Cognitive processes in delusional disorder. *Br J Psychiatry*. 1996;168:61–67.
132. Kinderman P, Bentall RP. Causal attributions in paranoia: internal, personal and situational attributions for negative events. *J Abnorm Psychol*. 1997;106:341–345.
133. Jolley S, Garety P, Bebbington P, et al. Attributional style in psychosis: the role of affect and belief type. *Behav Res Ther*. 2006;44:1597–1607.

134. Janssen I, Versmissen D, Campo JA, Myin-Germeys I, van Os J, Krabbendam L. Attributional style and psychosis: evidence for externalizing bias in patients but not individuals at high risk. *Psychol Med.* 2006;27:1–8.
135. McKay R, Langdon R, Coltheart M. Paranoia, persecutory delusions and attributional biases. *Psychiatr Res.* 2005;136:233–245.
136. Martin JA, Penn DL. Social cognition and subclinical paranoid ideation. *Br J Clin Psychol.* 2001;40:261–265.
137. Kinderman P, Dunbar RIM, Bentall RP. Theory of mind deficits and causal attributions. *Br J Psychol.* 1998;71:339–349.
138. Merrin J, Kinderman P, Bentall RP. Jumping to conclusions and attributional style in patients with persecutory delusions. *Cognit Ther Res.* 2007;31:741–758.
139. Bentall RP, Rowse G, Shryane N, et al. The cognitive and affective structure of paranoid delusions: a transdiagnostic investigation of patients with schizophrenia spectrum disorders and depression. *Arch Gen Psychiatry.* In press.
140. Mikulincer M. Attachment style and the mental representation of self. *J Pers Soc Psychol.* 1995;69:1203–1215.
141. Bartholomew K, Horowitz LM. Attachment styles among young adults: a test of a four-category model. *J Pers Soc Psychol.* 1991;61:226–244.
142. Frenkel E, Kugelmass S, Nathan M, Ingraham LJ. Locus of control and mental health in adolescence and adulthood. *Schizophr Bull.* 1995;21:219–226.
143. Moutoussis M, Williams J, Dayan P, Bentall RP. Persecutory delusions and the conditioned avoidance paradigm: towards an integration of the psychology and biology of paranoia. *Cognit Neuropsychiatry.* 2007;12:495–510.
144. Moutoussis M, Bentall RP, Williams J, Dayan P. A temporal difference account of avoidance learning. *Network Comput Neural Syst.* 2008;19:137–160.
145. Read J, Perry BD, Moskowitz A, Connolly J. A traumagenic neurodevelopmental model of schizophrenia. *Psychiatry Interpers Biol Process.* 2001;64:319–345.
146. Jaspers K. Causal and ‘meaningful’ connexions between life history and psychosis. In: Hirsch SR, Shepherd M, eds. *Themes and Variations in European Psychiatry: An Anthology.* Bristol, UK: John Wright & Sons; 1913/1974:81–93.
147. Engell GL. The clinical application of the biopsychosocial model. *Am J Psychiatry.* 1980;137:535–544.
148. Freedman AM. The biopsychosocial paradigm and the future of psychiatry. *Compr Psychiatry.* 1995;36:397–406.
149. Zubin J, Spring B. Vulnerability: a new view of schizophrenia. *J Abnorm Psychol.* 1977;86:103–126.
150. Bayer TA, Falkai P, Maier W. Genetic and non-genetic vulnerability to schizophrenia: the basis of the ‘two hit hypothesis’. *J Psychiatr Res.* 1999;33:543–548.
151. Allen P, Amaro E, Fu CHY, Williams SCR, Brammer MJ, Johns LC. Neural correlates of the misattribution of speech in schizophrenia. *Br J Psychiatry.* 2007;190:162–169.
152. Teicher MH, Tomoda A, Andersen SL. Neurobiological consequences of early stress and childhood maltreatment: are results from human and animal studies comparable? *Ann N Y Acad Sci.* 2006;1071:313–323.
153. Downhill JE, Buchsbaum MS, Wei T, et al. Shape and size of the corpus callosum in schizophrenia and schizotypal personality disorder. *Schizophr Res.* 2000;47:193–208.
154. Kitayama N, Quinn S, Bremner JD. Smaller volume of anterior cingulate in abuse-related posttraumatic stress disorder. *J Affect Disord.* 2006;90:171–174.
155. Job DE, Whalley HC, McConnell S, Glabus M, Johnstone EC, Lawrie SM. Structural gray matter differences between first-episode schizophrenics and normal controls using voxel-based morphometry. *Neuroimage.* 2002;17:880–889.
156. Nemeroff C, Bremner JD, Foa EB, Mayberg HS, North CS, Stein MB. Posttraumatic stress disorder: a state-of-the-science review. *J Psychiatr Res.* 2006;40:1–21.
157. Nelson MD, Saykin AJ, Flashman LA, Riordan HJ. Hippocampal volume reduction in schizophrenia as assessed by magnetic resonance imaging: a meta-analytic study. *Arch Gen Psychiatry.* 1998;55:433–440.
158. Laruelle M, Abi-Dargham A, Gil R, Kegeles L, Innis R. Increased dopamine transmission in schizophrenia: relationship to illness phases. *Biol Psychiatry.* 1999;46:56–72.
159. Schulze TG, Ohlraun S, Czerski PM, et al. Genotype-phenotype studies in bipolar disorder showing association between the DAOA/G30 locus and persecutory delusions: a first step towards a molecular genetic classification of psychiatric phenotypes. *Am J Psychiatry.* 2005;162:2101–2108.
160. Enoch M-A. Genetic and environmental influences on the development of alcoholism. *Ann N Y Acad Sci.* 2007;1094:193–201.
161. Klien TA, Neumann J, Reuter M, Hennig J, von Cramon DY, Ullsperger M. Genetically determined differences in learning from errors. *Science.* 2007;318:1642–1645.
162. Read J, McGregor K, Coggan C, Thomas DR. Mental health services and sexual abuse: the need for staff training. *J Trauma Dissociation.* 2006;7:33–50.
163. Wijkstra L, Lijmer J, Balk F, Geddes J, Nolen WA. Pharmacological treatment for psychotic depression. <http://www.cochrane.org>. April 12, 2008.
164. Callcott P, Turkington D. CBT for traumatic psychosis. In: Larkin W, Morrison AP, eds. *Trauma and Psychosis: New Directions for Theory and Therapy.* London, UK: Routledge; 2006:222–238.
165. Larkin W, Morrison AP. Relationships between trauma and psychosis: from theory to therapy. In: Larkin W, Morrison AP, eds. *Trauma and Psychosis: New Directions for Theory and Therapy.* London, UK: Routledge; 2006:259–282.
166. Pilgrim D. The biopsychosocial model in Anglo-American psychiatry: past, present and future? *J Ment Health.* 2002;11:585–594.