**Finding a solution to psychosis: the emergence of a new path**

Xin YU

**Summary:** The transition from a dichotomous diagnostic classification system to the more holistic approach to understanding mental disorders engendered by the so-called biopsychosocial model has definite advantages, but it runs the risk of sacrificing methodological rigor to achieve all-inclusiveness. The Special Article by Bebbington on understanding psychosis in this issue attempts to show that high-quality psychosocial epidemiological research on the development of psychosis can, at least partially, overcome these limitations. Bebbington’s emphasis on the importance of non-psychotic symptoms such as disturbance in sleep and mood in the development of psychosis provides a new perspective on the conceptualization of psychosis, but I remain unconvinced about the usefulness of such symptoms in the differentiation of valid sub-categories of schizophrenia or other psychoses.

**Key words:** dichotomous classification; biopsychosocial model; psychotic disorders; psychosis; psychiatric epidemiology

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Dichotomous classifications of diagnoses used to be very popular both in psychiatry and in general medicine. In psychiatry this approach engendered several widely popularized distinctions: organic versus functional, neurotic versus psychotic, and genetic versus environmental.[1] This kind of dichotomy had practical advantages in the past when the limited repertoire of treatments available for mental disorders (e.g., electroconvulsive therapy, catharsis, large oral administration of reserpine, injection of tranquilizers, etc.) often had quite serious adverse effects and, thus, were intended for individuals who had severe mental disorders. More recently the value of dichotomous approach to diagnostic classifications has been challenged in psychiatry and in medicine more generally. One alternative approach that arose in the 1970s was the so-called bio-psycho-social model (BPSM),[2] a model that remains popular at the present. Many academics continue to advocate for the application of the BPSM in medical education, research, and practice.[3]

Moving from dichotomous conceptualizations of health and disease to a more nuanced understanding based on the BPSM is certainly a progressive step. However, I am not sure that it is best to use the BPSM as the foundation on which to build our understanding of mental disorders. The BPSM offers a Yin-Yang Tai Ji diagram of causes that encompasses everything, but its all-inclusive appeal conceals its lack of methodological rigor. Ghaemi discussed the advantages and disadvantages of the BPSM, and pointed out that overemphasizing the BPSM may lead psychiatry research into agnosticism.[4] Although psychiatric disorders are complicated, the constant emergence of new technologies and discoveries brings us closer and closer to the goal of understanding the brain and the disorders of the brain. Bebbington’s Special Article in this issue, ‘Unravelling psychosis: psychosocial epidemiology, mechanism, and meaning’,[5] is an attempt to avoid agnosticism and draw a roadmap of the development of psychosis that integrates these various approaches.

Bebbington cites an extensive literature of epidemiological studies, most of which go well beyond simple head counts and descriptive epidemiology, to analyze the development and prognosis of psychosis cross-sectionally and longitudinally. This review highlights an unfamiliar side of the ‘familiar’ psychotic disorders: non-psychotic symptoms, especially mood symptoms, play an important role in the onset and progression of psychosis. Even for schizophrenia, a condition that has an estimated genetic heritability of 80%, psychosocial factors have a major impact on the development and course of the disorder. There are over 300 known susceptibility genes for schizophrenia, each with its own specific functional pathway and
Moreover, it is highly unlikely for one individual with schizophrenia to carry more than two susceptibility genes. For such a genetically heterogeneous population to show similar psychotic symptoms, the power of environmental factors in molding this relatively consistent symptomatic presentation must be considerable.

Bebbington’s analysis of studies about early intervention is particularly useful. He introduced intervention research targeting adolescents with non-psychotic symptoms, such as poor sleep quality and disturbed mood, in order to prevent their progression to psychotic symptoms. This approach has been supported by various studies. For example, Arango emphasized that attention should be paid to mood problems in adolescents, and recommended the use of medication (when necessary) to prevent their progression to psychosis. Yung and colleagues introduced special services for adolescents in Australia, which they consider an effective method to prevent the development of psychosis. Although these interventions are costly and may be ethically controversial, they should be promoted to limit the long-lasting damage that psychosis can have for affected individuals and their families.

On the other hand, I disagree with some of Bebbington’s arguments. Psychotic episodes are unique psychological experiences, the core characteristic of which is the loss or severe damage of contact with reality. Psychotic episodes are frequently accompanied with many so-called non-psychotic symptoms, but the diagnostic and prognostic value of these symptoms should not be overestimated. Unrealistic perceptions and distorted beliefs are much rarer than mood and sleep disturbances, so achieving the goal of improving the validity and predictive power of diagnostic criteria may be best achieved by basing sub-classifications of schizophrenia (and other psychoses) on the characteristics of the psychotic symptoms rather than on the presence or absence of concurrent sleep or mood problems. Bebbington uses the reported effectiveness of cognitive behavioral therapy for psychosis (CBT-p) as confirmation of the important role psychosocial factors play in the onset and development of psychosis; I find this argument no more convincing than the assertion that improvement of psychotic symptoms after administration of an antipsychotic medication is evidence of the presence of a psychotic disorder. I agree with Bebbington that CBT-p should be studied further to explore its target symptoms and to better understand the psychological and biological mechanisms underlying its effectiveness in individuals with psychotic conditions, but it is misleading to infer the etiology of an illness based on the effectiveness of a treatment method.

Psychosis is similar to a difficult mathematical problem for which there are multiple solutions. Bebbington has bypassed the traditional entree to the problem – hallucinations and delusions – seeking an alternative pathway that starts with psychosocial environmental factors and common psychological symptoms to arrive at an alternative conceptualization of psychotic disorders. None of these solutions is perfect, but Bebbington has helped us understand that there may be multiple, equally-valid approaches to understanding and, eventually, addressing the problem of psychotic disorders such as schizophrenia.

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Dr. Xin Yu is Professor of Clinical Psychiatry at the Peking University Institute of Mental Health. He is the current President of the Chinese Society of Psychiatry, Founding president of the Chinese Psychiatrists Association, and Vice Chairman of the China Alzheimer’s Association. Dr. Yu has received training in Psychiatry in China and also received fellowships to study abroad at St. George Hospital, Melbourne University, Australia and at John Hopkins University, USA. Dr. Yu’s major areas of research include: dementia, late life depression and psychosis, substance abuse, first onset schizophrenia, neurocognitive function of HIV/AIDS, and bipolar disorder. He has co-authored more than 70 original articles and 15 book chapters. Dr. Yu is also on the editorial board of more than 10 domestic and international peer-reviewed journals, and is the editor-in-chief of the Chinese Mental Health Journal.