

Stress-reactivity in psychosis: Evidence for an affective pathway to psychosis

Inez Myin-Germeys^{a,b,c,*}, Jim van Os^{a,d}

^a *Department of Psychiatry and Neuropsychology, South Limburg Mental Health Research and Teaching Network, EURON, Maastricht University, Maastricht, The Netherlands*

^b *Section Social Cognition, Mondriaan Zorggroep, Heerlen, The Netherlands*

^c *School of Psychological Sciences, University of Manchester, UK*

^d *Division of Psychological Medicine, Institute of Psychiatry, London, UK*

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Abstract

This paper will review a series of studies using the Experience Sampling Method that suggest that altered sensitivity to stress is an endophenotype for psychosis. The Experience Sampling Method is a structured diary technique allowing the assessment of emotional reactivity to stressors occurring in normal daily life. Elevated emotional reactivity to stress was found in subjects vulnerable to psychosis, suggesting that affective responses to stressors in the flow of daily life are an indicator of genetic and/or environmental liability to psychosis. Indeed, the small stressors in daily life associated with affective responses also predict more intense moment-to-moment variation of subtle positive psychotic experiences.

Increased emotional reactivity was found to be independent from cognitive impairments, and argued to constitute evidence of an affective pathway to psychosis that may underlie a more episodic, reactive, good-outcome type of psychosis. Evidence for this hypothesis was found in data suggesting that the experience of stressful life events and early trauma were associated with increased stress-sensitivity, and that women were more likely to display elevated stress-reactivity. These findings are discussed in the light of recent biological and psychological mechanisms.

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1. Introduction

The search for causal mechanisms of psychotic disorders has led to an increased interest in the study of underlying vulnerability substrates, the so-called endophenotypes (Tsuang, 2001; Weinberger, 1999). Endophenotypes are more closely related to the genetic aetiology and its interaction with environmental risks, and thus may reveal underlying causal mechanisms that lead to the development of psychotic symptoms (Claridge, 1994). Since schizophrenia is characterized by a heterogeneous clinical expression and since multiple factors are argued to be on the causal pathway to psychosis, it is attractive to hypothesize that several endophenotypes contribute to the development of psychosis,

* Corresponding author. Department of Psychiatry and Neuropsychology, South Limburg Mental Health Research and Teaching Network, EURON, Maastricht University, Maastricht, The Netherlands.

E-mail address: i.germeys@sp.unimaas.nl (I. Myin-Germeys).

possibly independent from each other. The current paper will focus on aberrations in sensitivity to stress that may be considered an endophenotype of psychosis, reflecting underlying gene-environment interactions associated with the impact of trauma and stressful life events in vulnerable individuals, and will give an overview of accumulating evidence for what we have called an “affective pathway to psychosis”.

2. Stress and psychosis

It has long been acknowledged that stress is an important factor in the development of psychosis. The vulnerability-stress model (Katschnig, 1991; Nuechterlein & Dawson, 1984; Zubin & Spring, 1977) has been widely accepted as a heuristically useful framework for the study of the etiology and clinical course of schizophrenia. According to this model, psychiatric symptoms emerge whenever a threshold of stressors exceeds the individual’s vulnerability level, the latter being a stable within-person characteristic (Zubin, Magaziner, & Steinhauer, 1983). It has been reported that patients suffering from schizophrenia are sensitive to life events (Bebbington et al., 1993; Lukoff, Snyder, Ventura, & Nuechterlein, 1984). Life events have consistently been found to influence course and outcome of psychotic disorders. Increased numbers of life events have been associated with higher levels of symptomatology and increased relapse rates (Bebbington et al., 1993, 1996; Carr et al., 2000; Lukoff et al., 1984; Norman & Malla, 1993). Critical family environments are associated with alterations in illness course in schizophrenia (Bebbington & Kuipers, 1994; Brown, Birley, & Wing, 1972; Butzlaff & Hooley, 1998; Vaughn & Leff, 1976). Recent epidemiological work has demonstrated that the stresses of urban life consistently increase the risk to develop psychosis (van Os, Hanssen, Bijl, & Vollebergh, 2001), especially in subjects who are vulnerable to psychosis (Krabbendam & van Os, 2005; van Os, Hanssen, Bak, Bijl, & Vollebergh, 2003). Victimization and the experience of childhood trauma are also associated with increased levels of clinical and sub-clinical psychosis (Bebbington et al., 2004; Janssen et al., 2004; Lataster et al., 2006; Read, van Os, Morrison, & Ross, 2005). Furthermore, small daily events have been reported to be important predictors of psychological symptoms in general (Kanner, Coyne, Schaefer, & Lazarus, 1981; Monroe, 1983), of subjective distress (Norman & Malla, 1991), and of relapse rates in schizophrenia (Malla, Cortese, Shaw, & Ginsberg, 1990). Thus, there is convincing evidence that stress indeed is involved in the etiology of psychosis. However, it is less clear how these stressors impact on the subject who is vulnerable to develop psychosis. The stress-vulnerability model is essentially interactional, but most research to date has focused on either the indicator of vulnerability or the stressor, whereas their interplay has rarely been examined.

3. Emotional reactivity to stress in daily life

Sensitivity to stress can be assessed with questionnaires assessing the personality trait “Neuroticism”, a trait characterized by increased mood reactivity and sensitivity to stress. Patients with schizophrenia have been characterized by higher levels of neuroticism (Horan, Subotnik, Reise, Ventura, & Nuechterlein, 2005), and neuroticism may be associated with the severity of positive symptoms (Lysaker, Lancaster, Nees, & Davis, 2003). Neuroticism also increases the risk to develop both non-clinical and clinical psychosis (Krabbendam et al., 2002; Van Os & Jones, 2001). A more sensitive approach to elucidate the underlying mechanism through which stress impacts on subjects that are vulnerable to psychosis, is to measure stress-sensitivity in experimental conditions. A number of studies by Docherty and colleagues using a positive and negative memory speech task suggested that patients with psychosis reported higher levels of stress and affective reactivity of speech in the stress condition (Cohen & Docherty, 2004; Docherty, Hall, & Gordinier, 1998; Docherty, Rhinewine, Nienow, & Cohen, 2001). They also reported that patients with psychosis scored higher on trait arousability, which was significantly associated with reported stress during the speech task (Dinzeo, Cohen, Nienow, & Docherty, 2004). Horan and Blanchard, using a social-role play task, suggested that trait negative affect and maladaptive coping accounted for one quarter of the variance in negative mood during assertion role-play test scenes in individuals with schizophrenia (Horan & Blanchard, 2003). Other studies investigated the impact of stress on biological measures, such as pituitary–adrenal axis activation, administering both psychosocial and metabolic stress tasks. These studies reported conflicting results with some studies showing exaggerated adrenocorticotropin and cortisol release in patients with psychosis (Elman et al., 1998; Walsh, Spelman, Sharifi, & Thakore, 2005) while other studies found no difference between patients and controls (Breier, Davis, Buchanan, Moricle, & Munson, 1993) or even a blunted cortisol response (Jansen et al., 1998; Jansen, Gispens-de Wied, & Kahn, 2000; Marcelis, Cavalier, Gielen, Delespaul, & van Os, 2004).

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