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Responses to stress in patients with psychotic disorders compared to persons with varying levels of vulnerability to psychosis, persons with depression and healthy controls

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ABSTRACT

Background and Objectives: An experimental design was used to test whether self-reported, psychophysiological and symptomatic stress-responses increase as a function of the underlying vulnerability to psychosis as proposed by vulnerability-stress-models.
Methods: Stress-responses of participants with psychotic disorders (*PSY*, $n = 35$) were compared to those of participants with attenuated positive symptoms (*AS*, $n = 29$), first-degree relatives of persons with psychotic disorders (*REL*, $n = 26$), healthy controls (*HC*, $n = 28$) and controls with depression (*DEP*, $n = 30$). Using a repeated measures design, participants were assigned to a noise stressor, a social stressor and a no stress condition in random order. Stress-responses were assessed via self-report, salivary cortisol levels, heart rate and skin conductance levels. State-paranoia and depression were assessed with clinical scales.
Results: PSY reported to be significantly more stressed than *HC*, *AS* and *REL* across all conditions which went along with increased heart rate and decreased overall cortisol release. In contrast, *AS* showed elevated levels of cortisol. *PSY* showed a stronger response of self-reported stress to the noise condition compared to the no stress condition than *HC*, but no stronger response than the other samples. Furthermore, the stressors did not trigger stronger psychophysiological responses or symptom-increases in *PSY*.
Limitations: The social stressor was brief and not individualized and did not have an effect on cortisol.
Conclusions: The findings support the notion that subjective stress-responsiveness increases with vulnerability, but not the assumption that symptoms arise directly as a function of stress and vulnerability. Also, the generally high levels of arousal seem to be more relevant to psychosis than the responsiveness to specific stressors.

1. Introduction

Since the late 1970s stress has been ascribed a central role in the pathogenesis of psychotic disorders (Nuechterlein & Dawson, 1984; Zubin & Spring, 1977). Vulnerability stress models differ in detail, but all assume that people have varying levels of vulnerability, and that the likelihood of psychotic symptoms is a function of the extent of vulnerability and stress that the individual encounters. The mechanisms that translate stress into psychotic symptoms are proposed to be decreased neuropsychological functioning resulting in a breakdown of information processing abilities in earlier models (Nuechterlein & Dawson, 1984; Zubin & Spring, 1977) and reasoning biases, perceptual anomalities and emotion-processing in more recent model variants for positive symptoms (Blackwood, Howard, Bentall, & Murray, 2001; Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001). Vulnerability stress models have a high face validity and are used as a basis of psycho-educational approaches that aim to help patients to monitor early symptoms by reducing stress in their every-day lives. Nevertheless, the evidence for the basic notion that stress triggers psychotic symptoms is not fully conclusive.

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A common method to assess the impact of external stressors on psychopathology has been to retrospectively investigate life-events in periods followed by an increase in symptoms. Most of these studies found life-events to be precipitants of acute psychosis (Beards, Gayer-Anderson, Borges, Dewy, & Fisher, 2013). However, this retrospective methodology has been criticized (Philipps, Francey, Edwards, & McMurray, 2007), the major problem being that it does not answer the question whether stressful events cause psychosis. For instance, a patient might report that having been left by his wife triggered an episode of psychosis, while in fact being left was the consequence of prodromal symptoms.

More recent research has focused on the association of psychosis and everyday stressors, such as migration, isolation and discrimination (Cantor-Graae & Selten, 2005; Veling et al., 2007), urbanicity (Lederbogen et al., 2011; Weiser et al., 2007), or exposure to relatives with high-expressed emotion (Cutting, Aakre, & Docherty, 2006). Myin-Germeys and van Os (2007) took this approach several steps further: Using the Experience Sampling Method (ESM) to assess responses to hassles occurring in the participants' daily lives they found a clear association between minor stressors and the intensity of psychotic experiences in patients and their first-degree relatives. Although the cross-lagged analyses in the ESM studies indicate that stressors precede psychosis, the interpretation of the causal direction between stress and symptoms remains difficult. The report of stressors is not independent of the clinical status of the participants and therefore likely to be influenced by a number of disorder-related factors, including recall and attention biases (Philipps et al., 2007), illusory correlations or causal inferences which have been found in patients with psychotic disorders (Beer, Moritz, & Lincoln, 2012). Therefore, additional studies that control for the type and intensity of stressors are needed.

Beyond the question of causality, several relevant aspects related to the investigation of vulnerability-stress models need noting. One is that the stress response takes place on several levels, including psychophysiological responses. While some studies have assessed neuro-endocrine responses (Jansen, Gispen-de Wied, & Kahn, 2000; Thompson et al., 2007), autonomic responses have been neglected. Furthermore, few studies have differentiated between different types of stressors, such as social and non-social stressors. Also, it has been emphasized that more attempts need to be made to systematically compare the stress-responses of groups that differ in vulnerability, such as first-degree relatives and high- risk groups (Beards et al., 2013; Philipps et al., 2007). Finally, as stressors also tend to precede depression (Kessler, 1997; Stroud, Davila, & Moyer, 2008) more research is needed to assess whether psychosis is associated with a distinct stress response compared to depression.

To summarize, the existing body of research supports a connection between stress and psychosis but is less definite with regard to its causal directions. To provide a more conclusive answer to this question, the present study used an experimental design to investigate the impact of a noise and a social stressor on self-reported and psychophysiological stress parameters and paranoid symptoms in persons with varying levels of vulnerability to psychosis.

The study was preceded by two pilot studies demonstrating that a) paranoid beliefs increased in response to a noise stressor in healthy individuals and that this increase was moderated by baseline-vulnerability to psychosis (Lincoln, Peter, Schäfer, & Moritz, 2009) and b) that paranoid beliefs increased in response to a noise stressor in participants with psychotic disorders compared to healthy controls (Moritz et al., 2011). The present study extended on these findings and hypothesized (a) that the self-reported and psychophysiological (heart rate (HR), skin conductance level (SCL), salivary cortisol) response to induced stress will vary as a function of vulnerability (psychotic disorder, attenuated symptoms, first-degree-relatives, healthy) and (b) that psychotic symptoms will increase as a function of vulnerability and stress. Finally, in order to estimate the diagnosis-specificity, we compared the stress responses of the sample with psychotic disorders with those of a sample of patients with depression and contrasted the impact that stress exerts on paranoid symptoms with the impact it has on symptoms of depression.

2. Method

2.1. Design

The study was conducted in Hamburg and Marburg (Germany) as a randomized repeated measures design. Participants with psychotic disorders (*PSY*) were compared to healthy persons with attenuated positive symptoms (*AS*), first-degree relatives of persons with psychotic disorders (*REL*), participants with depression (*DEP*) and healthy controls (*HC*) in regard to subjective stress ratings, psychophysiological parameters (HR, SCL and cortisol) and psychotic symptoms within a no stress, a noise stress and a social stress condition. Subjective stress-ratings and psychophysiological stress parameters were assessed prior to each condition (baseline) and five additional times within each condition (compare Fig. 1). Symptoms were assessed at the end of each condition, while the stressors were still present.

This design allows us to test the differential influence of the stress conditions in the different samples (group × condition interactions). Due to the repeated assessments of the stress-parameters the design also allows us to test whether certain groups show stronger stress-responses from baseline to subsequent measurements dependent on condition (condition × time × group interactions).

Time in min	-14	-10	00[1]	10	20	30	40	50	60	70	80
Stressor											
HR & SCL	Test	Baseline		Time 2		Time 3		Time 4		Time 5		Time 6	
Cortisol			B		2		3		4		5		6
VAS-S			1		2		3		4		5		6
Symptoms													

Note. [1]The Experimental manipulation of stress (no stress versus noise, versus social stress) begins at time zero; HR=Heart rate; SCL=Skin Conductance Level; VAS-S=Visual Analogue Scale to assess the subjective stress response; Symptoms=Assessment of state paranoia and depression

Fig. 1. Assessment times during the stress conditions.