Schizophrenia Research xxx (2016) xxx–xxx

Contents lists available at ScienceDirect

Schizophrenia Research

journal homepage: www.elsevier.com/locate/schres

Experimentally induced psychosocial stress in schizophrenia spectrum disorders: A systematic review

Claudia Lange *[,1], Lorenz Deutschenbaur [1], Stefan Borgwardt, Undine E. Lang, Marc Walter, Christian G. Huber

Department of Psychiatry, University of Basel, Basel, Switzerland

ARTICLE INFO

Article history:
Received 14 June 2016
Received in revised form 22 September 2016
Accepted 4 October 2016
Available online xxxx

Keywords:
Psychosocial stress
Schizophrenia spectrum disorder
HPA axis
Heart rate

ABSTRACT

Background: There is evidence that exposure to social stress plays a crucial role in the onset and relapse of schizophrenia; however, the reaction of patients with schizophrenia spectrum disorder (SSD) to experimentally induced social stress is not yet fully understood.
Method: Original research published between January 1993 and August 2015 was included in this systematic literature research. Social stress paradigms, reporting subjective responses to stress measures, plasma or saliva cortisol, or heart rate (HR) in patients with SSD were included. 1528 articles were screened, 11 papers (390 patients) were included.
Results: Three main findings were attained concerning chronically ill patients: (1) overall similar subjective responses to stress ratings between SDD patients and controls, (2) no group differences in cortisol response to psychosocial stress and (3) an increase in HR after the stress exposure was seen in patients and controls. The study examining first-episode patients found higher subjective responses to stress and lower stress-induced cortisol levels.
Conclusion: The results indicate that first-onset medication free patients may show differences in subjective responses to stress measures and cortisol release while chronically ill patients display no differences in subjective and cortisol response. This may be the correlate of a pathophysiological dysfunction of the hypothalamic-pituitary-adrenal axis prior or at the onset of SSD and a subsequent change in dysregulation during the course of the illness. Given the paucity of studies investigating psychosocial stress in SSD and the pathophysiological relevance of psychosocial stress for the illness, there is need for further research. (PROSPERO registration number: CRD42015026525)

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1. Introduction

Schizophrenia spectrum disorders (SSD) are major mental illnesses that are characterized by positive and negative symptoms, as well as cognitive impairment (Dinzeo et al., 2008). The onset and the course of SSD are regulated by an interplay of biological factors, vulnerability, and psychosocial influences (Meltzer et al., 2001; Norman and Malla, 1993).

There is evidence that patients with SSD are exposed to higher rates of psychosocial stress: For example, it has been reported that patients with SSD often live a stressful social life with limited social support and critical family environment (Lukoff et al., 1984; van Winkel et al., 2008).

In SSD, stress is not only a consequence of the illness (Rusch et al., 2015), but also an important factor modulating the course of the disease (Mizrahi, 2015): The neural diathesis-stress model of Walker and Diforio (1997) (Walker and Diforio, 1997) assumes that stress is a trigger factor for transition to psychosis in patients with a pre-existing vulnerability. In line with this hypothesis, a meta-analysis by Beards et al. found about 3-fold increased odds of adverse life events in the period prior to psychosis onset (Beards et al., 2013). Psychosocial stress can also be regarded as a cause triggering recurrent psychotic episodes (Mizrahi, 2015). Overall, there seem to be a coincidence of adverse life events and the onset or exacerbation of psychotic symptoms (for review see van Winkel et al., 2008; Dinzeo et al., 2004).

In addition, psychosocial stressors such as childhood trauma appear to play a significant role for the development of a vulnerability for SSD: A recent meta-analysis found that childhood trauma scores are substantially higher in patients with ultra high risk for psychosis compared with healthy controls (Kraan et al., 2015). Furthermore, the stress of mothers during pregnancy – for example the death of the father before the child's birth (Huttunen and Niskanen, 1978), or exposure to war during pregnancy (Maslina et al., 2008) – can also be considered as a risk factor: research found that prenatal maternal stress is associated with the development of SSD in the children.

* Corresponding author at: Department of Psychiatry, University of Basel, Wilhelm Klein-Strasse 27, CH-4012 Basel, Switzerland.
E-mail address: claudia.lange@upkbs.ch (C. Lange).
[1] Equal contribution.

http://dx.doi.org/10.1016/j.schres.2016.10.008
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Please cite this article as: Lange, C., et al., Experimentally induced psychosocial stress in schizophrenia spectrum disorders: A systematic review, Schizophr. Res. (2016), http://dx.doi.org/10.1016/j.schres.2016.10.008

In addition, patients with schizophrenia report an increased feeling of uncontrollability in these stressful events (Horan et al., 2005). Uncontrollability is one of the main factors in eliciting a physiological stress response (Dickerson and Kemeny, 2004). In summary, current literature suggests that psychosocial stress constitutes an important factor in the clinical course of SSD, which can facilitate transition to psychosis, trigger the onset of a psychotic episode, and can aggravate psychotic symptoms (Mizrahi, 2015).

Psychosocial stress reactions have been intensively investigated in experimental settings over the past decades. Inducing stress in an experimental setting is a good way to imitate real life stressors (Allen et al., 2014). These tests serve to examine the stress response more directly, leading to subjective responses to stress, constituting a potent challenge of the hypothalamic-pituitary-adrenal axis, and eliciting vegetative stress reactions (Kirschbaum and Hellhammer, 1999). The biomarkers mostly investigated in stress research are cortisol and heart rate (HR). The hypothalamic-pituitary-adrenal axis (HPA axis) is commonly studied as the physiological system that acts in response to stressors of everyday life. It regulates the release of the stress hormone cortisol (Kirschbaum and Hellhammer, 1999). Psychosocial stress commonly leads to an increased HR and to the activation of the HPA axis, inducing the release of significantly more cortisol compared to a resting situation.

HPA axis abnormalities can be involved in the development of psychiatric illnesses and are often seen in psychiatric patients. Panic disorder (Petrowski et al., 2012; Schreiber et al., 1996), substance use disorder (Walter et al., 2008; Walter et al., 2011), and depression (Claes, 2009; Holsboer et al., 1992; Horstmann and Binder, 2011) are associated with a dysregulated HPA axis function. As detailed above, neuroendocrinological studies have shown that a dysfunction of the HPA axis might also play a role in the pathophysiology of schizophrenia (Mizrahi, 2015; Walker and Diforio, 1997; Altamura et al., 1999; Guest et al., 2011). To date, there is a narrative review examining the role of stress in general and the HPA axis for the course of schizophrenia (Walker et al., 2008) and covering the literature until 2007, a recent systematic review of the activity of the hypothalamic-pituitary-adrenal axis only pertaining to first-episode psychosis (Borges et al., 2013), and a meta-analysis comparing HPA axis activation in schizophrenia and depression (Ciufolini et al., 2014) including only a limited subset of three studies on SSD. However, there is no current systematic review focused on experimentally induced psychosocial stress as an important parameter for the development and course of SSD, examining the subjective responses to stress and vegetative stress response in addition to HPA axis functioning, and covering the broad diagnostic range of schizophrenia-spectrum disorders.

2. Aim/Objective

This systematic review aims to give an overview on all studies published from January 1993 to August 2015 using psychosocial stress tasks to examine the emotional response, as well as the two most frequently employed biomarkers of acute stress response, namely cortisol and heart rate, in patients with schizophrenia spectrum disorders.

3. Method

For this review, we performed a computer-based systematic literature search using PubMed and Web of Knowledge. A combination of keywords, one part pertaining to experimental induction of psychosocial stress ("cortisol", "stress", or "HPA axis") and one pertaining to the group of patients with schizophrenia spectrum disorder ("schizophrenia", "schizoaffective", "psychotic" or "psychosis") was employed using explosion of search terms. Our literature search included original work published from January 1993 until August 2015. The start date was chosen as the development of the Trier Social Stress Test (TSST) in 1993 preceded a substantial increase of research using standardized experimental protocols to induce psychosocial stress. Potentially relevant articles were identified, retrieved, and assessed for possible inclusion in this review by author CL. The reference lists of all retrieved articles were additionally screened for further applicable original papers (CL). Review articles were not considered. The screening of the articles was performed in a three-step procedure: The first step determined whether an article might be appropriate for this review based on the article's title and abstract. In the second step, full-text articles were examined to decide whether they should be included based on the in- and exclusion criteria (see below). Lastly, the selected studies were screened for potential sample overlap.

3.1. In- and exclusion criteria

Psychosocial stress tasks had to include an uncontrollable (e.g. time pressure, unsolvable task) and/or an evaluating (e.g. speech in front of a panel) characteristic. Tasks investigating chronic stress exposure (e.g. caregiving) and naturalistic observations (e.g. class examinations) were not included. To be included, studies had to examine at least one of the following outcome parameters: subjective responses to stress, HPA axis activity in response to psychosocial stress, or heart rate. Cortisol levels had to be analyzed with a standard biological assay; only reports on plasma cortisol or free/saliva cortisol were used. Studies examining HPA axis response to biological/pharmacological challenges (e.g. DEX/CRH test) or only reporting adrenocorticotropic hormones (ACTH) as measures of HPA axis response were excluded.

Patients had to have a DSM-IV or ICD-10 diagnosis of a schizophrenia spectrum disorder (i.e., paranoid, disorganized, catatonic, undifferentiated and residual schizophrenia; schizophreniform disorder; brief psychotic disorder; delusional disorder; shared psychotic disorder; schizoaffective disorder; excluding drug induced psychotic disorder). Furthermore, studies investigating only individuals under the age of 18 were not included. We included only studies written in English.

Out of 1528 screened papers, 13 papers matched inclusion criteria based on the article's title and abstract (cf. Fig. 1). Of these, 2 papers were excluded after evaluation of the full-text articles: one because patients had not been diagnosed with schizophrenia spectrum disorder (Smith and Lenzenweger, 2012), and one because the study did not use a psychosocial stress task (Dinzeo et al., 2004). Overall, 11 papers were included in the qualitative review.

To assess the methodological quality of the current review, we used the PRISMA 2009 checklist (Moher et al., 2009). While seven items of the checklist pertained to meta-analyses and were not applicable, 17 out of the remaining 20 items were fulfilled (items 1–11, 17, 18, and 24–27), indicating an overall good methodological quality. The study protocol was registered in the International Prospective Register of Systematic Reviews (PROSPERO, n.d; registration number: CRD42015026525) prior to the completion of data extraction.

4. Results

The main results including level of significance can be found in Table 1.

We included 11 studies examining 420 patients with SSD. Sample overlap was found between Brenner et al., 2009 and Brenner et al., 2011 (Brenner et al., 2009; Brenner et al., 2011), both reporting findings from the same 30 patients and 29 controls. Overall, 7 studies investigated subjective responses to stress, 8 studies investigated cortisol and 7 studies investigated heart rate. Only one study examined first-onset medication naïve SSD patients (van Venrooij et al., 2012), all other studies included chronically ill SSD patients.

4.1. Subjective responses to psychosocial stress

To our knowledge, only one study has investigated the subjective response to psychosocial stress in first episode, medication naïve patients

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