



Bridging psychophysiological and phenomenological characteristics of psychosis – Preliminary evidence for the relevance of emotion regulation

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ARTICLE INFO

Article history:

Received 24 August 2015

Received in revised form 7 October 2015

Accepted 23 October 2015

Available online 1 November 2015

Keywords:

Schizophrenia

Stress

Heart rate variability

Cortisol

ABSTRACT

In psychosis, the alleged increased subjective stress-sensitivity is reflected in abnormal physiological arousal such as higher heart rate, elevated skin conductance levels, decreased vagal activity, and unusual cortisol levels. Despite ongoing research, possible mechanisms that explain the interplay between the phenomenological (i.e., subjective stress and symptoms) and psychophysiological processes are not thoroughly understood. Building on the model of neurovisceral integration by Thayer and Lane (2000) that focuses on regulative mechanisms, we postulate that emotion regulation will be associated with vagal activity, and with both subjective and physiological stress. In the present analysis, we used data from a baseline relaxation period including a 5-minute assessment of heart rate variability (HRV), salivary cortisol, and momentary subjective stress ratings from a sample of 19 participants with psychosis (mean age = 40.9, $SD = 11.1$; 36.8% female). Emotion regulation modification skills were assessed for specific emotions (i.e., stress and arousal, anxiety, anger, sadness, shame) if these were present during the previous week. Vagal HRV was significantly and moderately associated with emotion regulation. Both stress parameters (i.e., cortisol, subjective stress) were significantly associated with emotion regulation, but not with HRV. We provide preliminary support for the notion that emotion regulatory processes represent a crucial link between phenomenological and psychophysiological phenomena in psychosis. A potential model that ascribes emotion regulation a central role in the restoration of homeostasis is discussed. Future studies are needed to verify its generalizability and predictive value.

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1. Introduction

“Autonomic regulation, attentional regulation, and affective regulation allow an organism to meet the challenges of an ever-changing environment”.

Thayer and Lane (2000, p. 214)

According to vulnerability-stress models of schizophrenia, vulnerability characteristics interact with environmental challenges in causing symptoms (Nuechterlein and Dawson, 1984; Zubin and Spring, 1977). Consequently, research on how psychosis develops has focused on the role of stressors and stress responses (Holtzman et al., 2013; Phillips et al., 2007). For example, individuals with psychosis have been found to show increased subjective stress in daily life (Myin-Germeys and van Os, 2007) and in laboratory experiments (Lincoln et al., 2015; Moritz et al., 2011). Moreover, higher stress-sensitivity in psychosis

compared to healthy controls seems to be reflected in elevated levels of physiological arousal, indicated by higher heart-rate (e.g., Dinzeo et al., 2008; Lincoln et al., 2015), elevated skin conductance levels (e.g., Schell et al., 2005; Zahn and Pickar, 2005), decreased vagal activity (Clamor et al., in press), and an altered hypothalamic-pituitary-adrenal axis (e.g., Bradley and Dinan, 2010). However, possible mechanisms that explain the interplay between the phenomenological (i.e., subjective stress and symptoms) and psychophysiological processes are not well understood. The model of neurovisceral integration by Thayer and Lane (2000) that focuses on impaired emotion regulation stemming from disinhibition of particular neuronal networks could be a promising starting point to understand the underlying pathogenicity. In this model, physiological systems that provide an index of neural integration and flexible control over the periphery indicate to what extent arousal and threat activation may be inhibited.

Williams et al. (2004) found a reduced activity in regions of the medial prefrontal cortex in paranoid psychosis, which was accompanied by a dysregulation in arousal and in pathways to the amygdala. Interestingly, these regions have been associated with autonomic regulation, specifically with heart rate variability (HRV). Impairments in prefrontal cortical regions that inhibit amygdala activity are suggested to lead to

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less inhibitory control via the nervus vagus, stimulating a constant perception of threat and weakened safety detection (Thayer et al., 2009, 2012). Beyond influencing the autonomic nervous system, the nervus vagus is assumed to inhibit the regulation of other allostatic systems, including the hypothalamic–pituitary–adrenal axis (Thayer and Sternberg, 2006). In accordance with this notion, increased cortisol levels were found to be related to decreased HRV (Thayer and Sternberg, 2006) and low HRV was found to be associated with an impaired recovery from stress and thus prolonged heightened cortisol levels (Weber et al., 2010).

Finally, emotion regulation has been suggested to be established by top-down inhibitory control of prefrontal over limbic brain areas (Morris et al., 2012). Consistent with a potentially shared network, low HRV has been proposed to be accompanied by deficits in emotion regulation (Thayer and Lane, 2000). This association recently received empirical support in healthy students (Williams et al., 2015). Moreover, several studies have investigated potential difficulties in emotion regulation and found these to be prominent in people with or vulnerable to psychosis (e.g., O'Driscoll et al., 2014; Westermann and Lincoln, 2011). Particularly, more dysfunctional regulation strategies (Livingstone et al., 2009), less acceptance strategies (Perry et al., 2011), and more impairment in emotion awareness and regulation (Kimhy et al., 2012) were found in comparison with healthy controls. Building on this research, we argue that difficulties in emotion regulation could be the missing link between impaired physiological adaptability and stress phenomena in individuals with psychosis. If emotion regulation does not restore homeostasis adequately, increased cortisol levels and subjective stress will follow, which are then likely to prompt perceived threat and subsequently symptoms.

As a preliminary test of this assumption, the present study investigates putative links between low HRV, difficulties in emotion regulation, and subjective and biological stress parameters in a sample of patients with psychosis. Derived from the postulation of impairment in the proposed shared neuronal network, we expect that low vagal HRV as an index of decreased adaptability and threat inhibition is associated with an impaired ability to modify emotions, irrespective of the subjective or physiological stress level. Additionally, we assume that low HRV and impaired emotion regulation will be associated with increased salivary cortisol levels and increased subjective stress levels.

2. Methods

The data used in the present analysis was collected in a study on stress-sensitivity in psychosis, for which a detailed description is provided in Lincoln et al. (2015). The original study included and compared participants with varying levels of vulnerability to psychosis. Within this sample we have previously found the participants with psychosis to show an increased stress-response, decreased HRV, and fewer emotion regulation skills than healthy controls (Clamor et al., 2014; Lincoln et al., in press). For the purpose of testing our hypotheses, we use the baseline data provided by the sample of patients with psychosis.

2.1. Participants

The participants were recruited from in- and outpatient treatment settings. From the total participant group ($N = 35$), 16 were excluded for the present analyses. Reasons were nonsufficient quality or absence of five-minute baseline blood volume pulse data ($n = 2$) or cortisol data ($n = 1$), current intake of cardiac medication (e.g., beta-blockers; $n = 7$), of unknown medication ($n = 1$) or of oral/intravenous medication from the group of steroids ($n = 3$), and smoking within 30 min prior to testing ($n = 2$). Of the final sample ($n = 19$), 11 were acutely psychotic and eight were remitted ($n = 16$ with a diagnosis of schizophrenia, and $n = 3$ with a diagnosis of a schizoaffective disorder). The mean age was 40.9 years ($SD = 11.1$), 36.8% of the group were female. The mean scores on the Positive and Negative Syndrome Scale (PANSS;

Kay et al., 1987) were 16.3 ($SD = 5.4$) for the positive syndrome, 14.7 ($SD = 4.6$) for the negative syndrome, and 33.3 ($SD = 6.8$) for the general psychopathology score. The majority was taking antipsychotic medication ($n = 15$).

2.2. Materials and procedures

HRV was recorded in a seated position at the baseline relaxation period (first day of the experiment) with an interval length of 5 min. It was measured via the blood volume pulse from the thumb of the non-dominant hand applying photoplethysmography (sampling rate: 256/s). The HRV analyses were conducted directly from the inter-beat intervals with the software ARTiiFACT, version June 2011 (Kaufmann et al., 2011) and automatically detected artifacts were corrected using cubic spline interpolation (see also Clamor et al., 2014). The present analyses include the time-domain HRV index of the root mean square of successive normal-to-normal interval differences (RMSSD) due to its primarily vagal indication (see Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology, 1996).

For the same baseline period, salivary cortisol was collected via Salivette® salivary collection device (Sarstedt, Nümbrecht, Germany). The cortisol level was determined by radioimmunoassay (DRG Instruments GmbH, Marburg, Germany) with inter-assay and intra-assay coefficients of variation below 8% and detection limits of 0.1 ng/mL.

After the baseline, we assessed the perceived stress with a visual analogue scale (based on Gaab et al., 2003). The participants indicated the extent to which they felt “I feel stressed by the situation.” on a 10 cm line (0 = “not at all” to 10 = “very intensely”).

To measure the ability to modify emotions, we used the modification subscale of the German version of the emotion specific Emotion-Regulation Skills Questionnaire (ERSQ-ES, Ebert et al., 2013). The participants provided an estimate of the highest intensity of a specific emotional state within the previous week (i.e., stress and arousal, anxiety, anger, sadness, shame). If an emotion was rated as having been present (i.e., ≥ 1 on a Likert-scale from 0 = absence to 10 = extreme presence), the participants' ability to modify the respective emotion (e.g., anger) was then assessed with three items (e.g., “I was able to influence my anger”) on a Likert-type scale (0 = “not at all” to 4 = “almost always”). Then, we computed the mean across all different emotions, with higher scores indicating greater skills. In a large clinical and non-clinical validation sample, the German ERSQ-ES met the theoretically based factorial structure, had good psychometric properties (Cronbach's $\alpha \geq .83$ for the modification scale), and showed good discriminant and convergent validity (Ebert et al., 2013).

2.3. Statistical analyses

We calculated partial correlations between HRV, emotion regulation, cortisol level, and subjective stress. For each of the specific correlations, all other respective correlations were partialled out. Analyses were conducted with the software “R 3.2.0” (R Core Team, 2015) and the package “qgraph 1.3.1” (Epskamp et al., 2012). Due to the directed hypotheses, the one-tailed significance level was set as $\alpha = .05$.

3. Results

As expected, HRV was significantly positively associated with emotion regulation, which in turn was associated negatively with the level of subjective stress (see Fig. 1, left). In contrast to our expectation, the cortisol and subjective stress levels were only significantly negatively associated with emotion regulation, not directly with HRV (see Fig. 1).

When subjective and physiological stress parameters were included simultaneously, the strongest remaining association was between HRV and emotion regulation, followed by a statistical trend for the negative correlation between emotion regulation and cortisol level (see Fig. 2).

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