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## **DISSERTATION**

# **The interplay between vulnerability factors, stress reactivity and paranoid symptoms: Emotion regulation and traumatic experiences in focus**

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### **Abstract**

Over the last few decades, vulnerability stress models have been applied to explain the etiology of psychosis. Thereby, the models propose that symptoms develop as a result of an interaction between the individual's vulnerability and its reactivity to stressors. Thus, research on both components of these models – vulnerability factors and stress reactivity – is crucial in order to gain better understanding of the etiology of psychosis and to explain how paranoid beliefs as a core symptom of this disorder emerge, maintain, and exacerbate.

The overarching aim of this dissertation was therefore to examine stress reactivity, its association with paranoid symptoms, and to further test the vulnerability stress models' assumptions by focusing on two vulnerability factors – emotion regulation and traumatic experiences. Psychological and biological indicators of the stress level were investigated regarding their relevance to the stress reactivity in healthy individuals (Study I). Furthermore, the path from stress reactivity in everyday life to paranoid symptoms was tested in individuals with attenuated psychotic symptoms (Study II/III) and in individuals diagnosed with a psychotic disorder (Study IV). Moreover, it was evaluated whether the association between stress reactivity and paranoid symptoms in everyday life is bi-directional or uni-directional (Study II and Study IV). Finally, adaptive and maladaptive emotion regulation as well as different characteristics of traumatic experiences were tested as potential vulnerability factors for an elevated stress reactivity (emotion regulation: Study I) and for the path from stress reactivity to paranoid symptoms in everyday life (emotion regulation: Study II; traumatic experiences: Study III).

Overall, the conducted studies corroborated the evidence that concurrent assessment of psychological and biological indicators of the stress level is of importance when investigating stress reactivity (Study I) and that both psychological and biological stress reactivity are relevant temporal predictors of paranoid symptoms (Study II/III and Study IV). Furthermore, findings of Study IV elucidated emotional processes before, during, and after paranoid symptoms by highlighting the role of anxiety as a precedent of paranoid symptoms and anger as a consequence. Maladaptive emotion regulation but not adaptive emotion regulation was found to be associated with stress reactivity (Study I) and to act as a moderator for the path from stress reactivity to paranoid symptoms (Study II). Finally, frequent trauma, younger age when trauma occurred, and physical trauma were moderators of the path from stress reactivity to paranoid symptoms (Study III). Despite limitations, which are discussed critically, this dissertation contributes to the understanding of the association between stress reactivity and paranoid symptoms and expands our knowledge on the relevance of emotion regulation and traumatic experiences for the process of symptom formation. In the long run, the findings could be utilized to improve both prevention and treatment.

Suspiciousness: *“Do you wish me a good morning, or mean that it is a good morning whether I want it or not; or that you feel good this morning; or that it is a morning to be good on?”*

— J.R.R. Tolkien, *The Hobbit*

## **1. Theoretical background**

### **1.1 Schizophrenia and other psychotic disorders**

According to the Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5; American Psychiatric Association, 2013), a diagnosis of schizophrenia requires at least two of the core symptoms (delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior, and negative symptoms) to be present for a minimum of a one-month period of time. Moreover, one of the symptoms must be either delusions, hallucinations, or disorganized speech. Delusions, as one of the core symptoms, are defined as “fixed beliefs that are not amenable to change in light of conflicting evidence.” (American Psychiatric Association, 2013, p. 87). Within delusions, persecutory delusions (i.e., paranoia) represent the most common form and they are characterized by the belief of the individual that “harm is directed at him or herself, and is ongoing or anticipated in the future” (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001, p. 1148).

With the lifetime prevalence averaging at 0.7 % (McGrath, Saha, Chant, & Welham, 2008), schizophrenia affects more than 21 million individuals worldwide (WHO, 2018). The consequences are severe: the recovery prognosis is poor, the life expectancy is reduced by 20 to 25 years compared to the general population due to suicide rates and cardiovascular diseases (Saha, Chant, & McGrath, 2007), and the premature all-cause mortality is elevated two to three times (McGrath et al., 2008). Moreover, in addition to these personal consequences for those affected by the diagnoses, there are severe societal consequences – costs resulting from early retirements or long-term sick leaves of those affected by the diagnosis, overcrowded waiting rooms in psychiatrists’ and psychotherapists’ offices, and high treatment costs – that altogether further emphasize the importance of research on schizophrenia.

The past few decades have been marked by significant changes in the understanding and treatment of schizophrenia and other psychotic disorders. Although antipsychotic medication is still considered to be the primary method of treatment and has been increasingly used to dampen psychotic symptoms such as delusions and hallucinations and to prevent relapses, recent reviews discuss rather discouraging results. Specifically, the medication appears not to be as effective as hoped in the long term, and its intake is accompanied with severe adverse side effects that strongly deteriorate the patients’ health status (Gøtzsche, Young, & Crace, 2015; Moncrieff, 2015; Whitaker, 2016). As a consequence, a focus has been increasingly put on the development of cognitive-behavioral

interventions tailored specifically for this group of patients (Mehl, Werner, & Lincoln, 2015). Although the first results of such interventions seem to be promising, the road to effective treatments of psychosis is long and the understanding of the formation, exacerbation, and maintenance of schizophrenia and its core symptoms is a prerequisite. For this reason, this dissertation is comprised of studies investigating the experience of paranoia as one of the core symptoms of schizophrenia and focuses on the triggers and vulnerability factors that contribute to the formation of paranoid symptoms as well as on processes that possibly contribute to their exacerbation and maintenance.

### **1.2 The formation of paranoia explained by the vulnerability stress model of psychosis**

The etiology of psychosis has been investigated from different perspectives ranging from biological approaches that explain psychosis as a result of neurodevelopmental abnormalities (e.g., Fatemi & Folsom, 2009; Murray & Lewis, 1987), to cognitive models of psychosis that discuss the importance of the dysfunctional cognitive processes and emotions for the etiology of psychosis (e.g., Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002; Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001; Preti & Cella, 2010). Another model that has been widely used to explain the etiology of psychosis is the vulnerability stress model (Nuechterlein & Dawson, 1984; Zubin & Spring, 1977) that integrates biological, cognitive and social risk factors. In particular, in the vulnerability stress model, the „vulnerability“ part is considered to be comprised of a variety of risk factors, from genetics, peri- and postnatal factors, over traumatic experiences and social defeat factors that include bullying, low social rank, and social network, to cognitive schemata, dysfunctional emotion regulation, and coping skills. Alone and combined, possibly additively, these vulnerability factors render a person more or less reactive to stressors, which represents the second part of the model. Once the equation is just about right, a threshold of stressors exceeds the vulnerability level of the individual and paranoid symptoms emerge (Zubin, Magaziner, & Steinhauer, 1983). Thereby, according to the hypothesis of psychosis continuum that describes psychotic symptoms as continuous phenomena (Linscott & van Os, 2010), paranoid symptoms range from vague suspiciousness to severe persecutory delusions and can be present not only in clinical groups but also in general population (Bebbington et al., 2013). Based on the vulnerability stress model and relying on the hypothesis of psychosis continuum, in the present dissertation I focused on different facets of stress reactivity and two specific vulnerability factors – emotion regulation and traumatic experiences.

## **1.3 Stress as a central component of the vulnerability stress model**

### **1.3.1 Different facets of stress**

The adaptive functioning of organisms may be explained by the constant regulation or maintenance of homeostasis so that internal conditions remain stable and relatively constant. In particular, once homeostasis is threatened, the individual experiences a stress reaction, which represents an attempt to adapt to changed circumstances (Chrousos, 2009). Thereby, a variety of triggers (i.e., stressors) of stress reaction are imaginable, ranging from major events threatening the vitality of the organism, such as injuries, to minor events, so called daily hassles, such as missing an important appointment or having an argument. Whether or not specific stressors are perceived as such is highly individual. That is, the stress reaction emerges only when the individual perceives the environment as overwhelming and their own resources as insufficient (Lazarus & Folkman, 1984). Mason (1968) described three central psychological preconditions for a stress reaction to take place: The situation needs to be interpreted as being novel, and/or as unpredictable, and/or the individual perceives that it has no control over the situation. Generally, a stress reaction is reflected in the response to and in the subsequent recovery from the stressor (see Figure 1). Thereby, a complex interplay of different systems takes place to orchestrate the stress reaction that is comprised of psychological and biological indicators of stress level.

In particular, biological stress level is top-down regulated by central nervous system processes and by limbic system that together process the information of threat cognitively and emotionally. These two systems then forward the information to hypothalamus that is closely related with two major stress response systems – hypothalamus pituitary adrenal (HPA) axis and the autonomous nervous system (ANS). Thereby, once the stress response is triggered, neurons in the hypothalamus release corticotropin-releasing hormone (CRH). The release of CRH then triggers the pituitary gland to secrete and release adrenocorticotropin hormone (ACTH). The ACTH then travels in the blood, reaches adrenal glands and triggers the secretion of the so-called stress hormones. These stress hormones can be divided into two classes: glucocorticoids (i.e., cortisol) and catecholamines (i.e., epinephrine and norepinephrine). Glucocorticoids specifically facilitate the energy substrates in the body and enable the organism to adequately adapt to changing demands of the environment. The activation of the HPA axis is therefore adaptive in acutely stressful situations. However, a prolonged activation of the HPA axis could represent a risk to the organism by causing suppressed immune functions, or increased blood pressure (for a review, see McEwen, 1998, 2000). A commonly used biomarker of acute HPA axis activation in stress research is salivary cortisol. Although the HPA axis is far more complex than reflected in salivary cortisol levels, this biomarker has been proven to be a reliable stress

level measure (Hellhammer, Wüst, & Kudielka, 2009). During a stress response, along with the psychological response and the HPA axis activation, sympathetic nervous system (SNS) as a part of the ANS activates with the aim to re-establish the homeostasis of the organism, at the same time suppressing the parasympathetic nervous system (PNS; Akselrod et al., 1981). This leads to the secretion of the catecholamines epinephrine and norepinephrine into the blood stream causing vasoconstriction of most blood vessels, including many of those in the skin, the digestive tract, and the kidneys, as well as increased blood pressure, increased muscle tension and a change in heart rate and heart rate variability. Hence, the SNS is responsible for priming the body for action, regulating the fight-or-flight response (Chrousos, 2009). In this dissertation, the heart rate that is innervated by both SNS and PNS was utilized as a measure of the activation of the ANS and the autonomic stress level.

Following the offset of the stressor the stress recovery takes place. Here, via the hypothalamic paraventricular nucleus, the activation of the HPA axis gets inhibited (i.e., glucocorticoid negative feedback; Herman & Cullinan, 1997) leading to the inhibition of cortisol secretion. Furthermore, the SNS gets inhibited as well and the PNS takes on control, allowing the organism to return to the resting state, thereby decreasing the heart rate and returning other bodily functions to the state of “rest and digest”.

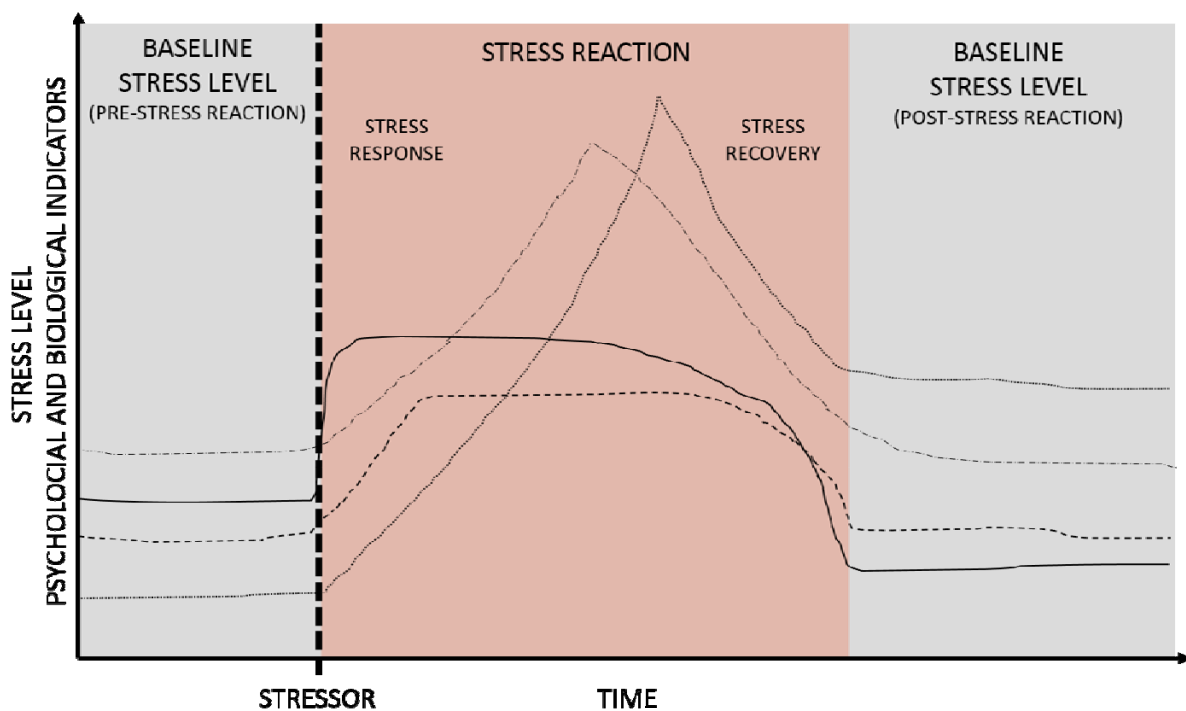


Figure 1. Graphical illustration of exemplary psychological and biological stress level indicators prior to, during and following exposure to a stressor.

### **1.3.2 Stress reactivity and psychosis**

Based on the vulnerability stress models, it has been proposed that individuals vulnerable to psychosis have an increased stress reactivity (Myin-Germeys & van Os, 2007; Myin-Germeys, van Os, Schwartz, Stone, & Delespaul, 2001) that could be reflected in higher stress levels in response to a stressor. Although the terms “stress sensitivity” and “stress reactivity” have often been used interchangeably, in this dissertation, I use the term stress reactivity to refer to the phenomenon of an elevated stress response.

A vast number of studies have shown an elevated stress reactivity in individuals with psychosis. These findings can be classified according to the indicators of stress levels used. When it comes to psychological indicators, experimental studies have found that individuals with psychosis and those at risk for psychosis show a stronger increase in negative affect, subjective appraisal and/or symptoms in response to stressors in comparison to healthy individuals (Ellett, Freeman, & Garety, 2008; Lincoln, Köther, Hartmann, Kempkensteffen, & Moritz, 2015; Veling, Pot-Kolder, Counotte, Van Os, & Van Der Gaag, 2016). Furthermore, these findings have been corroborated by experience sampling studies (ESM) that show individuals with psychosis and those at risk to have an elevated negative affect in response to daily hassles in comparison to healthy individuals (Lataster et al., 2009; Myin-Germeys & van Os, 2007; Myin-Germeys et al., 2001; Reininghaus et al., 2016). Moreover, this elevated negative affect seems to be related to the increase of paranoid symptoms in everyday life (Ben-Zeev, Ellington, Swendsen, & Granholm, 2011; Kramer et al., 2014; Thewissen et al., 2011). Finally, Van Der Steen and colleagues (2017) reported a stronger association between negative affect and paranoid symptoms in high risk groups than in patients with psychosis and Reininghaus and colleagues (2016) found this association to be even higher in first episode individuals than in a high-risk group, suggesting that reactivity to stress could be especially relevant to the unfolding of the disorder.

When it comes to the findings on the biological counterparts of stress reactivity, various studies have reported on a dysregulation of the HPA axis in psychosis (Chaumette et al., 2016; Shah & Malla, 2015). Furthermore, it has been shown that also individuals at genetic risk for psychosis have an elevated endocrine stress reactivity in daily life, in comparison to healthy individuals (Collip et al., 2011). Finally, in another ESM study, Boettger and colleagues (2006) found individuals with acute psychosis to have an increased heart rate and an altered autonomic variability in comparison to healthy controls, suggesting an elevated stress reactivity of ANS as well. Hence, the association between elevated stress reactivity and psychosis symptoms could be expected for both psychological and biological indicators of stress level.

Although, as outlined above, stress reactivity is reflected in both psychological and biological indicators of stress level, only few studies have concurrently assessed different

indicators, such as endocrine, autonomic and psychological (i.e., affective) stress level. Due to their rather low convergence (Mauss & Robinson, 2009) and different facets of reactivity that these indicators capture (Allen, Kennedy, Cryan, Dinan, & Clarke, 2014) it is however of importance to investigate these indicators concurrently. In one such study on individuals with varying vulnerability to psychosis, Lincoln and colleagues (2015) found psychological stress level to be more responsive to experimentally induced stress than biological stress level indicators. Furthermore, in one of very few studies that investigated autonomic activity in daily life of patients with psychosis, Kimhy and colleagues (2010) found negative affect and heart rate to be uncorrelated. At the same time they found negative affect to be associated with other ANS parameters, such as heart rate variability. Thus, it seems that the stress reactivity in patients with psychosis could be characterized by a complex pattern of a maladaptive hyper- and hypo-reactivity of different stress level indicators highlighting the importance of their concurrent measurement.

#### **1.4 The exacerbation and maintenance of paranoid symptoms – a vicious cycle of stress reactivity and paranoia?**

Whereas stress reactivity is able to explain the emergence of psychotic symptoms, it fails to explain the exacerbation or maintenance of such symptoms. Therefore, it is important to link the vulnerability stress models with the models explaining these processes. In the last two decades, cognitive models of psychosis (Bentall et al., 2001; Freeman et al., 2002; Garety et al., 2001) started to acknowledge the central role of affective processes for paranoid symptoms. As Freeman and colleagues (2002) propose in their cognitive model of persecutory delusions, an elevated anxiety could lead to paranoid symptoms and these could in turn lead to more anxiety, which could represent the process of the exacerbation and maintenance of paranoid symptoms. Such vicious cycle of symptoms and emotional processes has also been described in other disorders: For instance, in major depression, negative affect and negative cognitions are considered to lead to more depressive symptoms, and depressive symptoms to lead to even stronger negative affect and negative cognitions, further exacerbating the depressive symptoms (Teasdale, 1983). Hence, we could assume that exacerbation and maintenance of paranoid symptoms could as well be determined by such a vicious cycle.

While elevated negative affect in response to stressors has been increasingly investigated as a trigger and predecessor of paranoid symptoms in the context of the vulnerability stress models (Ben-Zeev et al., 2011; Ellett et al., 2008; Freeman et al., 2015; Kramer et al., 2014; Lincoln, Lange, Burau, Exner, & Moritz, 2010; Thewissen et al., 2011; Veling et al., 2016), the question of whether or not paranoid symptoms act as a stressor that triggers more negative affect, leading to a vicious cycle and contributing to the exacerbation of symptoms has been neglected. Few studies that focused on this research question have

found paranoid symptoms to be followed by affective consequences (Campbell & Morrison, 2007; Moritz & Van Quaquebeke, 2014; Van Rossum, Dominguez, Lieb, Wittchen, & Van Os, 2011). In contrast to these studies, in a longitudinal study on patients with psychosis, Fowler and colleagues (2012) found only a uni-directional path from negative cognitions and depressed mood to paranoid symptoms. Hence, the findings on bi-directional associations between negative affect and paranoid symptoms are sparse and somewhat contradicting, which highlights the need for further investigation of this potential process of symptom exacerbation and maintenance. Furthermore, considering that negative affect investigated in previous studies only reflects the psychological stress level, the research on this process should be expanded to biological indicators.

Another important aspect in regard to the exacerbation and maintenance of paranoid symptoms focuses on the relevant emotions for the psychological stress level and their contribution to the hypothesized vicious cycle of paranoid symptoms. In particular, previous research has strongly focused on anxiety and sadness as the most relevant emotions for paranoid symptoms (Ben-Zeev et al., 2011; Ben-Zeev, Morris, Swendsen, & Granholm, 2012; Freeman et al., 2012; Freeman & Fowler, 2009; Lincoln et al., 2010; Thewissen et al., 2011). However, there is also empirical evidence highlighting the importance of other emotional states, such as shame or anger. Specifically, shame has been found to be associated to paranoid symptoms (Matos, Pinto-Gouveia, & Gilbert, 2013), and to moderate the association between stressful life events and paranoid symptoms (Johnson et al., 2014). Furthermore, the relevance of anger has been highlighted by large-scale prospective studies showing that delusions predicted anger and that anger in turn predicted violent behavior (Coid et al., 2013; Ullrich, Keers, & Coid, 2014). Hence, concurrently assessing and analyzing different emotional states could be beneficial to better understand which exact emotions take place before, during, and after paranoid symptoms, possibly contributing their exacerbation and maintenance.

## **1.5 A 1000 pieces puzzle called “vulnerability” – focus on emotion regulation and traumatic experiences**

### **1.5.1 Emotion regulation**

Emotion regulation is considered as „the process by which individuals influence which emotions they have, when they have them, and how they experience these emotions” (Gross, 1998, p. 275). Thereby, we can differentiate between adaptive and maladaptive emotion regulation according to the outcomes that it facilitates (Aldao & Nolen-Hoeksema, 2012). Specifically, adaptive emotion regulation incorporates strategies (e.g., reappraisal, acceptance, or positive referencing) that have been found to be associated with positive outcomes and protective against psychopathology, whereas maladaptive strategies (e.g.,



suppression, rumination, or catastrophizing) have been found to be associated with negative outcomes and an increased risk for psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010). However, some studies suggest that based on their psychometric properties, these individual strategies can hardly be seen as separate constructs and rather represent two factors: the factor of the adaptive and the factor of the maladaptive emotion regulation (Aldao & Nolen-Hoeksema, 2010; Garnefski, Kraaij, & Spinhoven, 2001).

The importance of emotion regulation as a vulnerability factor is reflected in studies showing that individuals with psychosis apply more maladaptive and less adaptive emotion regulation strategies compared to healthy individuals (for a review, see O'Driscoll, Laing, & Mason, 2014). Moreover, cross-sectional (Moritz et al., 2016; Simpson, MacGregor, Cavanagh, & Dudley, 2012; Westermann & Lincoln, 2011), experimental (Boden & Berenbaum, 2007; Lincoln, Sundag, Schlier, & Karow, 2017; Lincoln, Hartmann, Köther, & Moritz, 2015a), and ESM (Hartley, Haddock, Vasconcelos, Emsley, & Barrowclough, 2014) studies offer evidence that emotion regulation strategies have an impact on paranoid symptoms in clinical and non-clinical samples. At the same time, there is solid empirical evidence that different emotion regulation strategies are related to how individuals in general population perceive stressors and respond to them (Butler et al., 2003; Carlson, Dikecligil, Greenberg, & Mujica-Parodi, 2012; Gross & John, 2003; Lam, Dickerson, Zoccola, & Zaldivar, 2009; Mauss, Cook, Cheng, & Gross, 2007; Memedovic et al., 2010; Thomsen, Mehlsen, Christensen, & Zachariae, 2003; Zoccola & Dickerson, 2012). Notwithstanding the fact that stress reactivity and paranoid symptoms are closely related and that both seem to be predicted by emotion regulation, only few studies investigated whether or not emotion regulation acts as a moderator of the path from stress reactivity to paranoid symptoms. Specifically, in one such a cross sectional study, Jones and Fernyhough (2008) found the interaction of high suppression and high anxiety to predict paranoid symptoms in a non-clinical sample. In a more recent ESM study, Nittel and colleagues (2018) also found maladaptive emotion regulation strategy suppression to moderate the association between negative affect and paranoid symptoms in everyday life of patients with psychosis. Hence, previous findings allow to hypothesize that adaptive and maladaptive emotion regulation act as moderators of the path from stress reactivity to paranoid symptoms but it needs further empirical investigation to corroborate this notion.

Finally, studies suggest that maladaptive emotion is more closely positively related to psychopathology in comparison to negative relation of adaptive emotion regulation and psychopathology (Aldao & Nolen-Hoeksema, 2012; Aldao & Nolen-Hoeksema, 2010; Aldao, Nolen-Hoeksema, & Schweizer, 2010). This has been corroborated by a recent prospective study, where the change of scores in maladaptive emotion regulation was related to a change in psychopathology scores, whereas the change in adaptive emotion regulation was

not (Moritz, Jahns, et al., 2016). Thus, research findings increasingly point towards the higher importance of maladaptive emotion regulation in comparison to adaptive emotion regulation.

### **1.5.2 Traumatic experiences**

The literature on traumatic experiences as a vulnerability factor for psychosis provides overwhelming evidence that experiencing trauma leaves a permanent mark in people's lives rendering them three to twelve times more vulnerable to develop psychosis (Janssen et al., 2004; Spauwen, Krabbendam, Lieb, Wittchen, & Van Os, 2006; Varese et al., 2012). Furthermore, a large body of research yielded evidence that traumatic experiences are associated with psychotic symptoms across the continuum of psychosis (Addington et al., 2013; Arseneault et al., 2011; Bailey et al., 2018; Lataster et al., 2006; Shevlin et al., 2007; Thompson et al., 2009). At the same time, narrowing down the particular characteristics of such traumatic experiences would be important in order to enable more specific detection of vulnerable individuals and thereby promote prevention. In particular, traumatic experience can be differentiated by the age when a trauma took place, the frequency and recurrence of traumatic experiences, as well as by the type of experienced trauma. However, findings on the association between psychotic symptoms and different characteristics of traumatic experiences are rather sparse and ambiguous. If we consider the age as a factor, previous studies have mostly focused on the childhood trauma. Although from developmental perspective this could be understandable, there is evidence that recent trauma could also be of importance. In particular, Lataster and colleagues (2012) found longitudinal associations between both early and recent trauma with an increased risk for psychosis. At the same time more recent studies corroborate the perspective that early trauma is especially important by showing trauma at a younger age to represent a particularly strong vulnerability factor for psychopathology (Powers et al., 2016; Russo et al., 2014). There is some further evidence that the age at which a trauma took place could be a factor that determines which symptoms an individual subsequently develops: Alameda and colleagues (2016) reported early trauma to predict more positive and negative psychosis symptoms and affective symptoms, whereas later trauma to be associated only with more negative symptoms. The role of the type of trauma as a vulnerability factor is similarly inconclusive, with some authors specifying physical trauma as the most relevant predictor of psychosis (Fisher et al., 2010; Rubino, Nanni, Pozzi, & Siracusano, 2009) and others finding only sexual trauma to be relevant (Thompson et al., 2014). Furthermore, trauma can also be specified as intentional (e.g., physical violence or abuse) versus non-intentional (e.g., natural catastrophe), whereby intentional trauma has been found to be a crucial type of trauma in regard to psychosis (Gibson, Alloy, & Ellman, 2016; van Nierop et al., 2014). Finally, the frequency of trauma and

its recurrence as a vulnerability factor also remains unresolved, with Powers and colleagues (2016) reporting no association between the frequency of trauma and risk of psychosis, and two other studies finding a dose-response relation with more frequent trauma being associated with a higher risk for psychosis (Rubino et al., 2009; Shevlin et al., 2007).

Apart from this conflicting evidence on trauma characteristics, the association between traumatic experiences and the risk of psychosis is still rather superficially researched. Specifically, although it seems to be clear that traumatized individuals carry a heightened risk for psychosis, it is questionable by which mechanism this risk acts. One postulated mechanism is that the association between traumatic experiences and paranoid symptoms takes place via elevated stress reactivity (a mediation model). Possibly, trauma induces increased stress levels, prolonged negative affective states such as fear, anger, or sadness (Amstadter & Vernon, 2008) and elevated stress reactivity (Glaser, van Os, Portegijs, & Myin-Germeys, 2006) that in turn have often been found to be a predictor of paranoid symptoms (Ben-Zeev et al., 2011; Fowler et al., 2012; Freeman et al., 2015; Jaya, Ascone, & Lincoln, 2017; Kramer et al., 2014; Lincoln, Peter, Schäfer, & Moritz, 2010; Oliver, O'Connor, Jose, McLachlan, & Peters, 2012; Thewissen et al., 2011). Such mediating mechanism has been corroborated by Freeman and Fowler (2009) and Bebbington and colleagues (2011), who found negative emotions to act as a mediator of the link between trauma and paranoia. Another possible mechanism could be reflected in a moderation model. In particular, although stress reactivity and paranoia are associated, it is also evident that not everyone who experiences strong stress reactivity develops paranoid symptoms as a consequence. In light of the vulnerability stress model, we could assume that traumatic experiences act as a vulnerability factor that strengthens the path between stress reactivity and paranoid symptoms (a moderation model). In a recent study, van Nierop and colleagues (2018) found that the association between stress reactivity in everyday life and symptom development at 14-month follow-up was only significant in individuals with trauma exposure and a mixed phenotype of psychopathology (co-occurrence of affective, psychotic, and anxiety symptoms). Furthermore, Rauschenberg and colleagues (2017) found that only in help-seeking adolescents and young adults but not in their siblings or healthy controls, experience of abuse and neglect was associated with an elevated negative affect and psychotic experience in response to daily stress. Hence, these studies offer some evidence that a moderation model may hold true. Therefore, studies are required to test both proposed mechanisms to compare their contribution to explaining the formation of paranoid symptoms and to expand existing findings by investigating different characteristics of traumatic experiences.

## 2. Goals and relevance – where do we go from here

The overarching goal of this dissertation was to investigate the central component of the vulnerability stress models – stress reactivity – and to explore its association with paranoid symptoms. Thereby, the focus has been set on investigating how the association between stress reactivity and paranoid symptoms is influenced by two potential vulnerability factors for psychosis: emotion regulation and traumatic experiences. The relevance of this dissertation is threefold: First and foremost, it adds to the understanding of stress reactivity and puts the vulnerability stress model as arguably the most relevant etiological model of psychosis to an empirical test, thereby closing important research gaps. Second, focusing on the temporality the association between stress reactivity and paranoid symptoms, this dissertation aims to explore processes that take place before during and after paranoid symptoms, possibly contributing to their exacerbation and maintenance. Third, by incorporating the experience sampling method that is integrated in the everyday lives of participants, this dissertation tests if the hypothesized processes can also be identified in an ecologically valid manner, rendering the results particularly representative and relevant for individuals' daily lives. Considering the increased focus of the scientific community on the treatment options apart from psychopharmacological treatment for patients diagnosed with schizophrenia or other psychotic disorders, the contribution of this dissertation to the research field is of importance. Assuming that we could narrow down the specific characteristics of traumatic experiences that are relevant for the formation of psychosis, the identification of vulnerable individuals would be more specific and enable better prevention programs. Furthermore, if emotion regulation moderates the path from stress reactivity to paranoid symptoms, discovering which specific indicators of stress level could be influenced by maladaptive or adaptive emotion regulation would enable the development of more specific and more efficient interventions. Finally, understanding the emotional processes that take place before, during and after paranoid symptoms occur would enable the integration of such interventions “at the right spot and at the right time”.

Specifically, in this dissertation I postulate the following research questions:

1) *Which indicators of stress level (negative affect, endocrine, autonomic, and symptomatic) determine stress reactivity and how does stress reactivity contribute to the formation of paranoid symptoms?* Considering different psychological and biological facets of stress, multiple indicators of stress level need to be measured (Study I and Study II). To gain insights into the characteristics of stress reactivity in general, the stress level indicators should in a first step be examined in a healthy population (Study I).

2) *Does emotion regulation have an impact on stress reactivity, and does it act as a moderator for the pathway from stress reactivity in daily life to paranoid symptoms?* Assuming that emotion regulation represents a vulnerability factor, maladaptive and adaptive

emotion regulation should directly predict stress reactivity (Study I) and also moderate the path from stress reactivity to paranoid symptoms in everyday life (Study II). Furthermore, considering the continuum of psychosis, the moderating effect should already be evident in individuals who only experience attenuated psychotic symptoms (Study II).

3) *Does trauma act as a moderator from stress reactivity in daily life to paranoid symptoms and which characteristics of traumatic experiences play an important role in this process: frequency and recurrence of trauma, age at trauma, and/or type of trauma?* Assuming that traumatic experiences as a vulnerability factor cannot be reduced only to their presence or absence, various characteristics of traumatic experiences need to be measured and tested for their role as moderators of the path from stress reactivity to paranoid symptoms (Study III).

4) *Do stress reactivity and paranoid symptoms in everyday life form a vicious cycle of paranoia exacerbation and which emotions in particular act as predecessors, concomitants and consequences of paranoid symptoms?* Assuming that paranoid symptoms act as a stressor, it could be expected that they are followed by elevated stress reactivity that then further exacerbates or maintains paranoid symptoms. In line with the hypothesis of the psychosis continuum this should hold true in both the subclinical (Study II) and the clinical (Study IV) sample. Furthermore, a differentiated analysis of emotions that the stress reactivity is comprised of is required to specify this model and derive possible interventions from it (Study IV).

### **3. Study I: Different facets of stress and the role of emotion regulation for the stress reactivity**

Krkovic, K., Clamor, A., & Lincoln, T. M. (2018). Emotion regulation as a predictor of the endocrine, autonomic, affective, and symptomatic stress response and recovery. *Psychoneuroendocrinology*, 94, 112-120.

#### **3.1 Introduction**

Elevated reactivity to stressors represents a central assumption of the vulnerability stress model that is commonly applied to explain the development of psychosis (Nuechterlein & Dawson, 1984; Zubin & Spring, 1977). This model further suggests that specific vulnerability factors increase this stress reactivity and strengthen its association with psychotic symptoms. As of now, however, the complexity of stress reactivity that is reflected in various psychological and biological indicators of stress level, is still not well understood. Specifically, previous research suggests that when investigating stress reactivity, due to the low convergence of different stress level indicators, a concurrent measurement of both

psychological indicators such as negative affect, and biological indicators such as ANS activation and HPA axis activation is necessary (Allen et al., 2014; Mauss & Robinson, 2009). In the present study, we focused on the investigation of how different stress level indicators correlate during stress response and recovery. Furthermore, we aimed to investigate whether or not deficits in emotion regulation act as a vulnerability factor for a stronger stress reactivity across the different indicators of stress level.

### **3.2 Method**

We recruited a community sample including 67 individuals. Participants first completed an online baseline questionnaire battery from home. Subsequent to this, all participants underwent stress manipulation with the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) on site. Indicators of the stress level were measured before (i.e., pre-TSST) and after the TSST (i.e., post-TSST), and after a 10 minute recovery phase following the TSST (i.e., post-rest).

Emotion regulation at baseline was measured with the German version of the emotion specific Emotion-Regulation Skills Questionnaire (ERSQ-ES; Ebert, Christ, & Berking, 2013) and the German version of the Cognitive Emotion Regulation Questionnaire (CERQ; Garnefski & Kraaij, 2007; Loch, Hiller, & Witthöft, 2011). The maladaptive emotion regulation was measured with the maladaptive emotion regulation subscale of the CERQ, whereas adaptive emotion regulation was calculated as a composite score from the ERSQ-ES and adaptive emotion regulation subscale of the CERQ. The indicators of the stress level were assessed as follows: Negative affect was assessed with four items capturing self-ratings of momentary anxiety, sadness, anger, and shame. Furthermore we assessed depressive symptoms and paranoid symptoms as an additional measure of the psychological stress level with an ultra-short version of the Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977) [German: Allgemeine Depressions-Skala (ADS; Hautzinger, 2012)], and with a five item version of the Paranoia Checklist (Schlier, Moritz, & Lincoln, 2016; based on Freeman et al., 2005). Salivary cortisol was collected via a salivary collection device as an indicator of the HPA axis activity. Mean heart rate was calculated for five minute intervals for the pre-TSST, post-TSST, and post-rest phase as an indicator of autonomic arousal.

To address the postulated hypotheses, partial correlations and general linear models (GLM) for repeated measures were calculated, whereby all predictors were centred around the group mean.

### **3.3 Main results**

All psychological indicators (negative affect, depressive and paranoid symptoms) were intercorrelated with partial correlations ranging from  $r = .307$  to  $r = .555$  pre-TSS, from  $r$

=.341 to  $r = .680$  post-TSST, and from  $r = .649$  to  $r = .781$  post-rest (all  $p_s < .05$ ). Salivary cortisol and heart rate as biological indicators correlated significantly pre-TSST with  $r = .341$  but were not significantly correlated post-TSST and post-rest. All partial correlations between psychological and biological indicators were non-significant.

GLM was significant for all indicators of stress level ( $p < .001$ ). The Bonferroni adjusted pairwise comparisons showed significant increases in heart rate, negative affect, depressive symptoms, and paranoid symptoms from pre-TSST to post-TSST ( $p < .05$ ), followed by a significant decrease in these indicators from post-TSST to post-rest ( $p < .05$ ). Salivary cortisol showed a significant linear increase ( $p < .05$ ) from pre-TSST to post-TSST, and from post-TSST to post-rest.

Furthermore, the results of the GLM showed no interaction effect of adaptive emotion regulation  $\times$  time-point on any of the stress level indicators (all  $p_s > .05$ ). The interaction effect of maladaptive emotion regulation  $\times$  time-point (i.e., pre-TSST, post-TSST, post-rest) on negative affect was significant,  $F(1.57, 97.55) = 4.33, p < .05, \eta^2_{\text{partial}} = .065$ , and we found a trend interaction effect on salivary cortisol,  $F(1.48, 83.10) = 3.33, p = .055, \eta^2_{\text{partial}} = .056$ . Within subject contrasts showed that maladaptive emotion regulation was only a significant predictor of the increase in negative affect from pre-TSST to post-TSST,  $F(1, 62) = 5.78, p < .05, \eta^2_{\text{partial}} = .085$ , with higher maladaptive emotion regulation predicting stronger increase of negative affect in response to stressor. The same pattern was found for salivary cortisol, where maladaptive emotion regulation was only a significant predictor of the change from pre-TSST to post-TSST,  $F(1, 57) = 4.68, p < .05, \eta^2_{\text{partial}} = .076$ . Thereby, individuals with high maladaptive emotion regulation showed a blunted response to the stressor in comparison to other individuals who showed a linear increase. The interaction effect of maladaptive emotion regulation  $\times$  time-point on heart rate, depressive and paranoid symptoms was not significant (all  $p_s > .05$ ).

### 3.4 Discussion

In this quasi-experimental study, we found that individuals responded to a social-evaluative stressor not only psychologically (e.g., feeling anxious or angry) and biologically (i.e., increased cortisol and heart rate) but also “symptomatically”, with an increase in depressive and paranoid symptoms. At the same time, after the rest phase, individuals were able to down-regulate and recover in all indicators of the stress level, except for salivary cortisol. For salivary cortisol we found a linear increase across time-points, which suggests that we only captured the stress response but not the recovery from the stressor. In line with previous studies, psychological and biological indicators were not correlated, indicating a low convergence (Mauss & Robinson, 2009; Shah & Malla, 2015). In line with previous findings, maladaptive emotion regulation was a predictor of stress reactivity, with a significant effect

on negative affect and a trend significant effect on salivary cortisol. Specifically, the group of participants with particularly high scores in maladaptive emotion regulation peaked in salivary cortisol before the stress induction and showed a linear decrease of the salivary cortisol response over the consecutive time-points. At the same time, this group of individuals reported the highest negative affect after the stress induction compared to the other groups. This suggests an endocrine hypo-reactivity and affective hyper-reactivity in individuals with maladaptive emotion regulation. However, we were not able to confirm our hypothesis that emotion regulation would have an effect on symptomatic stress reactivity. Emotion regulation also did not moderate stress reactivity measured as elevated heart rate.

To conclude, this study highlights the importance of investigating different indicators of stress level concurrently. Furthermore, it adds to recent findings by suggesting that maladaptive emotion regulation plays a stronger role for the stress reactivity than adaptive emotion regulation (Aldao et al., 2010; Aldao & Nolen-Hoeksema, 2012; Moritz, Jahns, et al., 2016). Therefore, interventions targeting maladaptive emotion regulation strategies may be promising to help individuals gain control over their stress reactivity. Nevertheless, other vulnerability factors, especially those possibly influencing the symptomatic stress reactivity should be investigated in future studies.

#### **4. Study II: Stress reactivity and paranoid symptoms in everyday life: Emotion regulation as a vulnerability factor**

Krkovic, K., Krink, S., Lincoln, T. M. (2018). Emotion regulation as a moderator of the interplay between self-reported and physiological stress and paranoia. *European Psychiatry*, 49, 43-49. DOI:10.1016/j.psyneuen.2018.04.028

##### **4.1 Introduction**

A vast number of empirical studies corroborates the notion that individuals with psychosis and those at risk for psychosis show elevated stress reactivity in everyday life in comparison to healthy individuals and that this reactivity is associated with psychotic symptoms (e.g., Ben-Zeev et al., 2012; Kramer et al., 2014). Thereby, etiological models of paranoid symptoms assume that paranoid symptoms also act as stressors on their own, leading to increased stress levels as a consequence (Freeman et al., 2002). A few studies corroborate this notion with the finding that different emotions emerge as a consequence of paranoid symptoms (Campbell & Morrison, 2007; Moritz & Van Quaquebeke, 2014; Van Rossum et al., 2011). In contrast, Fowler et al. (2012) found only a one-directional association of negative cognitions and depressed mood with paranoid symptoms. In this study, we investigated temporal aspects of the association between stress reactivity and



paranoid symptoms: whether or not stress reactivity in everyday life triggers paranoid symptoms and whether or not, in turn, paranoid symptoms trigger stress reactivity. Such a bi-directional association would imply the existence of the vicious cycle that could drive the exacerbation and maintenance of paranoid symptoms. Furthermore, we expanded previous ESM research by including both psychological and biological indicators of stress level (i.e., negative affect and heart rate). Finally, we focused on emotion regulation as a vulnerability factor that could possibly alter the association between stress reactivity and paranoid symptoms. We thereby hypothesized that deficits in emotion regulation would render individuals more vulnerable to experience paranoid symptoms subsequent to elevated stress levels.

## **4.2 Method**

We investigated the research questions by applying the ESM design on 64 individuals with attenuated psychotic symptoms. The participants were pre-screened with the Community Assessment for Psychic Experiences (Stefanis et al., 2002) with the cut-off for positive subscale set at eight. Emotion regulation was measured at baseline. For adaptive emotion regulation, a composite score was calculated from two questionnaires – the German version of the emotion specific Emotion-Regulation Skills Questionnaire (ERSQ-ES; Ebert, Christ, & Berking, 2013) and the adaptive subscale of the Cognitive Emotion Regulation Questionnaire (CERQ; Garnefski & Kraaij, 2007; Loch, Hiller, & Witthöft, 2011). Maladaptive emotion regulation was assessed with the maladaptive subscale of the CERQ.

The baseline questionnaire battery was administered online and participants completed it from their homes. Upon arrival, an ambulatory electrocardiogram sensor was attached to participants' chest. Participants received a smartphone, the ESM assessment was activated, and participants left the premises. In the following 24 hours, between 9 AM and 10 PM, participants were asked to fill out a short questionnaire every 20 minutes. Stress reactivity was operationalized as level of negative affect and level of heart rate. The negative affect was assessed with ten items referring to the previous 20 minutes. The heart rate was measured continuously over the 24 hours and analysed as a mean heart rate between two ESM questionnaires. Paranoid symptoms were assessed with a three item version of the Paranoia Checklist (Schlier, Moritz, & Lincoln, 2016; based on Freeman et al., 2005).

To answer our hypotheses, we applied multilevel analyses by calculating linear mixed-effect models with random intercept and random slope. To investigate the temporal associations, independent variables that were repeated-measures were time lagged and person-mean centred. Independent variables that were on a between-person level were grand-mean centred.

### 4.3 Main results

After controlling for preceding paranoid symptoms,  $b = 0.209$ ,  $SE = 0.035$ ,  $p < .001$ , 95% CI [0.139, 0.279], and for time effects,  $b = -0.004$ ,  $SE = 0.003$ ,  $p = .207$ , 95% CI [-0.009, -0.002], both preceding negative affect,  $b = 0.238$ ,  $SE = 0.037$ ,  $p < .001$ , 95% CI [0.165, 0.312], and heart rate,  $b = 0.004$ ,  $SE = 0.002$ ,  $p = .033$ , 95% CI [0.000, 0.006], were significant predictors of the subsequent paranoid symptoms. Preceding paranoid symptoms were not a significant predictor for either the subsequent heart rate nor for the negative affect (all  $p_s > .05$ ).

Maladaptive emotion regulation significantly moderated the path from preceding negative affect to subsequent paranoid symptoms,  $b = 0.188$ ,  $SE = 0.071$ ,  $p = .011$ , 95% CI [0.045, 0.331]. Maladaptive emotion regulation was not a significant moderator of the path from preceding heart rate to subsequent paranoid symptoms,  $b = 0.006$ ,  $SE = 0.003$ ,  $p = .090$ , 95% CI [-0.001, 0.012].

Adaptive emotion regulation was neither a significant moderator for the path from negative affect to paranoid symptoms nor for the path from heart rate to paranoid symptoms (all  $p_s > .05$ ).

### 4.4 Discussion

In the present study we were able to corroborate previous findings (e.g., Ben-Zeev et al., 2011; Kramer et al., 2014; Thewissen et al., 2011) that stress reactivity is temporally predictive of subsequent paranoid symptoms. Moreover, the results add to these previous findings by showing that this is not only the case for the negative affect as an indicator of the stress level but also for the heart rate as a biological indicator. Surprisingly, however, we did not find preceding paranoid symptoms to be predictive of subsequent stress reactivity in everyday life. This is in contrast to previous findings from studies that found paranoid symptoms to be followed by emotional consequences (Campbell & Morrison, 2007; Moritz & Van Quaquebeke, 2014; Van Rossum et al., 2011). Our results are in accordance with a longitudinal study by Fowler and colleagues (2012) where only uni-directional associations were found in a patient sample. These diverging results could be explained by different methodologies (experimental, prospective, qualitative, and ESM) used in existing studies. Therefore, further ESM studies on this topic are required to corroborate the uni-directionality of the association between stress reactivity and paranoid symptoms in everyday life. Furthermore, a clinical sample is required to test whether these results generalize to the group of individuals with severe, clinically relevant symptoms of paranoia. If so, the assumption of the “vicious cycle of paranoid symptoms” that we postulate in this study would need to be revised.

Finally, we found maladaptive emotion regulation, but not adaptive emotion regulation, to moderate the association from preceding negative affect to subsequent paranoid symptoms. Thereby, in individuals who reported to use more maladaptive emotion regulation strategies, the association between negative affect and subsequent paranoid symptoms was stronger than in those who reported to use less maladaptive strategies. This indicates that maladaptive emotion regulation plays a more important role as a risk factor for the formation of positive symptoms than the deficits in the adaptive emotion regulation. The absence of a moderation effect for the path from heart rate to paranoid symptoms needs to be further addressed in future studies.

### **5. Study III: Stress reactivity and paranoid symptoms in everyday life: Traumatic experiences as a vulnerability factor**

Krkovic, K., Schlier, B., & Lincoln, T. M. (in press). An experience sampling study on the nature of the interaction between traumatic experiences, negative affect in everyday life, and threat beliefs. *Schizophrenia research*.

#### **5.1 Introduction**

A large body of literature shows trauma and psychotic symptoms to be associated across the psychosis continuum (Addington et al., 2013; Arseneault et al., 2011; Bailey et al., 2018; Lataster et al., 2006; Shevlin et al., 2007; Thompson et al., 2009). Considering that trauma is associated with elevated negative affect as well (Amstadter & Vernon, 2008) and that individuals with a history of trauma show increased stress reactivity in everyday life (Glaser et al., 2006), it can be hypothesized that stress reactivity plays a central role for this association. However, the exact mechanism of the association remains unclear: whether trauma influences psychotic symptoms via stress reactivity (mediation model), or if stress reactivity is more likely to translate into psychotic symptoms in those who experienced trauma than in individuals who report no traumatic experiences (moderation model). So far, studies have found empirical evidence for both the mediation (Bebbington et al., 2011; Freeman & Fowler, 2009; Marwaha & Bebbington, 2015) and the moderation (Rauschenberg et al., 2017; van Nierop et al., 2018) model, but none of the existing studies has tested both models. For this reason in this study we tested both models in the same sample. Furthermore, although various studies investigated important characteristics of traumatic experiences for psychosis (Fisher et al., 2010; Lataster et al., 2012; De Loore et al., 2007; Powers et al., 2016; Rubino et al., 2009; Russo et al., 2014; Shevlin et al., 2007; Thompson et al., 2014), many questions still remain open that we aimed to shed light on in the present study – whether or not the age at the time of the traumatic experience matters, if the type of

trauma acts as an additional risk factor for experiencing paranoid symptoms in everyday life, or if repeated traumatic experiences increase the risk for experiencing paranoid symptoms in everyday life.

## 5.2 Method

This study was based on the same data-set as Study II (see above for details). Traumatic experiences were assessed at baseline using the Trauma History Questionnaire (THQ; Hooper, Stockton, Krupnick, & Green, 2011), which is a 24-item self-report questionnaire referring to potentially traumatic experiences. Trauma frequency was defined as the number of reported different traumatic experiences. Moreover, if any experience took place more than once, trauma was characterized as reoccurring. Furthermore, the participants provided their age at the time of the first traumatic experience. The following types of trauma were assessed: crime related intentional, non-intentional, sexual, and physical. We applied multilevel analysis and calculated linear mixed-effect models with random intercept and random slope for the mediation and moderation analyses.

## 5.3 Main results

Due to non-significant direct effects from different trauma characteristics to paranoid symptoms, mediation analysis was only performed for trauma frequency and sexual trauma that were directly related to paranoid symptoms in everyday life. The results showed no substantial change in direct effects from any of the tested trauma characteristics to paranoid symptoms after including negative affect as mediator (all  $p_s > .05$ ).

There was a significant interaction effect of trauma frequency  $\times$  negative affect,  $b = 0.023$ ,  $SE = 0.009$ ,  $p = .016$ , 95% CI [0.005, 0.041], a significant interaction effect of age at first trauma  $\times$  negative affect,  $b = -0.015$ ,  $SE = 0.007$ ,  $p = .040$ , 95% CI [-0.029, -0.001], and a significant interaction effect of physical trauma  $\times$  negative affect,  $b = 0.168$ ,  $SE = 0.074$ ,  $p = .026$ , 95% CI [0.018, 0.317], on subsequent paranoid symptoms.

All interaction effects between trauma characteristics and heart rate on subsequent paranoid symptoms were non-significant (all  $p_s > .05$ ).

## 5.4 Discussion

Findings of our study suggest that more frequent, recurring traumatic experiences and sexual trauma were predictive of experiencing more paranoid symptoms in everyday life. Furthermore, we found more support for the moderation model than the mediation model, with higher frequency, younger age and physical trauma, but not the other trauma characteristics, strengthening the path from negative affect to paranoid symptoms. Our results are in line with two other ESM studies that investigated this research question

(Rauschenberg et al., 2017; van Nierop et al., 2018). Considering that psychological mechanisms of psychosis formation take place on both “macro” level (i.e., distal, life-span related) and “micro” level (i.e., proximal, over the short period of time) (Reininghaus, Depp, & Myin-Germeys, 2016), it is possible that whereas a moderating role of traumatic experiences can be found on a micro level, different mechanisms act on a macro level. Furthermore, the finding that some characteristics of trauma act as direct predictors of paranoid symptoms and others act as moderators from negative affect to paranoid symptoms suggests different mechanism paths that could lead to paranoid symptoms. Contrary to our expectation, we did not find the same result pattern for the path from elevated heart rate in everyday life to paranoid symptoms. Due to the novelty of this research question, replication studies are needed as well as the extension of the assessment of stress reactivity for more specific biological indicators such as salivary cortisol.

#### **6. Study IV: Formation, exacerbation and maintenance of paranoid symptoms: The role of emotional processes**

Krkovic, K., Schlier, B., Clamor, A. & Lincoln, T. M. (2018). *Do paranoid beliefs have an emotion regulatory role? An analysis of the temporal interplay of emotions and paranoid symptoms in daily life*. Manuscript submitted for publication.

##### **6.1 Introduction**

Previous studies and findings of this dissertation clearly show that stress reactivity plays a role in the formation of positive symptoms such as paranoia. Thereby, negative affect as a psychological indicator of stress level has been postulated as an especially important predictor of paranoid symptoms as well as a consequence of experiencing paranoia (Bentall et al., 2001; Freeman et al., 2002; Preti & Cella, 2010). However, negative affect is a broad concept and represents a conglomerate of different emotional states such as anxiety, anger, or shame. In order to enable focused interventions in psychotherapy, it is essential to understand which exact emotions play a role in the formation, exacerbation and maintenance of paranoid symptoms. The vast majority of previous studies has focused on anxiety and sadness and their role as triggers of paranoia (Ben-Zeev et al., 2011; Freeman et al., 2012; Freeman & Fowler, 2009; Lincoln et al., 2010; Thewissen et al., 2011). Nevertheless, several other studies point to the possible role of other emotions, such as anger, guilt or shame, in this process (Coid et al., 2013; Johnson et al., 2014; Matos et al., 2013; Ullrich et al., 2014). However, since previous studies mostly focused on single emotions, further studies are needed that concurrently measure different emotional states. Therefore, the goal of this study was to investigate which emotions – anxiety, anger, sadness, guilt and shame –

contribute to the formation of paranoid symptoms in the everyday life of individuals diagnosed with schizophrenia or other psychotic disorders. Furthermore, as the extension of Study II, we explored the existence of the “vicious cycle” in a clinical sample, hypothesizing that specific emotions will be temporally predictive of paranoid symptoms, and that in turn, paranoid symptoms will be temporally predictive of respective subsequent emotions. This vicious cycle could represent the process of symptom exacerbation and maintenance.

## 6.2 Method

We tested our hypotheses in a sample of 30 individuals diagnosed with either schizoaffective disorder, schizophrenia, or a schizophreniform disorder. Participants were interviewed with a diagnostic interview, The German Version of the Structured Clinical Interview for DSM-IV Axis I disorders (SCID-I; Wittchen, Zaudig, & Fydrich, 1997) and The Psychotic Symptom Rating Scales (PSYRATS; Haddock, McCarron, Tarrier, & Faragher, 1999) to confirm diagnosis. Computer-based baseline questionnaires were performed. Afterwards, participants received a smartphone with ESM questionnaires. For seven consecutive days, the smartphones were programmed to alert the participants ten times per day in 90 minutes intervals between 9 AM and 10 PM. Self-ratings of anxiety, sadness, anger, shame and guilt were assessed in the ESM. Paranoid symptoms in the ESM were assessed with a five item version of the Paranoia Checklist (Schlier, Moritz, & Lincoln, 2016, based on Freeman et al., 2005).

We calculated linear mixed-effect models with random intercepts and fixed slopes. Variability of intercepts was modelled for participants and for days of assessment. For time-lagged models, we applied linear mixed-effect models with random intercept and the within-person autoregressive slope (i.e., paranoid symptoms at  $t-1$  predicting paranoid symptoms at  $t$  was estimated at person-level and allowed to vary randomly across individuals). Independent variables that were repeated measures were centred around the person-mean.

## 6.3 Main results

When controlled for preceding paranoid symptoms, only preceding anxiety predicted subsequent paranoid symptoms,  $b = 0.059$ ,  $SE = 0.020$ ,  $p = .003$ , 95% CI [0.021, 0.098]. The more anxiety at one time-point was reported, the more paranoid symptoms were reported on the following time-point.

The association between specific emotions and paranoid symptoms measured at the same time-point was significant for all tested emotions (all  $p_s < .05$ ), except for sadness that was marginally non-significant ( $p = .059$ ).

In a reversed model, only a model with preceding paranoid symptoms predicting subsequent anger was significant  $b = 0.156$ ,  $SE = 0.056$ ,  $p = .005$ , 95% CI [0.046, 0.153],

while controlling for the respective emotion on a preceding time-point. The more paranoid symptoms were reported at one time-point, the more anger was reported at the following time-point.

#### **6.4 Discussion**

As expected based on previous findings (Ellett et al., 2008; Freeman et al., 2015; Lincoln et al., 2010; Veling et al., 2016), anxiety was a temporal predictor of paranoid symptoms. Surprisingly, sadness was not temporally associated with subsequent paranoid symptoms in this study. Furthermore, at the time-points where stronger paranoid symptoms were reported, participants experienced a diffuse state of various negative emotions, as reflected in high correlations between paranoid symptoms and all measured negative emotions at the same time-point, except for sadness. One explanation of this result could be that during the symptom phases, individuals have difficulties to correctly differentiate between emotions, which is in line with findings that patients show deficits in awareness and understanding emotions (Kimhy et al., 2012; Lincoln, Hartmann, Köther, & Moritz, 2015b). Finally, we did not find an emotion-specific vicious cycle of paranoia: While anxiety was predictive of subsequent paranoid symptoms, paranoid symptoms were predictive of subsequent anger. This could indicate a specific regulatory process, where paranoid symptoms relieve anxiety and a diffuse emotional state during symptom phases by applying external attributions that in turn activate anger as a lead emotion. Potentially, such process could be beneficial in short-term, so that the exacerbation and maintenance of paranoid symptoms could be fitted into a reinforcement learning model: short-term positive consequence of anxiety and diffuse emotional state translating into anger facilitates the experience of paranoid symptoms, that in long-term becomes more maladaptive by strengthening the perception of environment as threatening and further elevating anxiety that exacerbates and maintains paranoid symptoms. This assumption needs to be addressed in future studies that would examine both short-term and long-term consequences of paranoid symptoms.

### **7. General discussion**

The goal of this dissertation was to investigate the assumptions of the vulnerability stress model and to extend the knowledge on the process of the formation, exacerbation and maintenance of paranoia as one of the core symptoms of psychosis. The insights gained in four studies will be discussed here by focusing on the following overarching topics: different indicators of stress level and their association with paranoid symptoms in everyday life, the process of formation, exacerbation and maintenance of paranoid symptoms, and the

relevance of emotion regulation and traumatic experiences as vulnerability factors for the formation of paranoid symptoms.

### **7.1 Different indicators of stress level and their association with paranoid symptoms in everyday life**

In Study I, the induction of social-evaluative stress triggered both psychological and biological stress responses, which was reflected in the significant increases in stress level measured as negative affect, depressive symptoms, paranoid symptoms, heart rate, and salivary cortisol. Furthermore, the stress level decreased significantly in all stress level indicators after the recovery, except for cortisol that showed a linear increase across the time-points due to its latency in response. Importantly, the tested indicators of stress level showed varying inter-correlations: whereas negative affect, depressive symptoms and paranoid symptoms as psychological indicators of stress level were significantly correlated, heart rate and cortisol as biological indicators of stress level showed very low inter-correlations. Moreover, psychological and biological measures of stress level were not significantly correlated with each other. This result adds to the existing literature on the divergence between psychological and biological indicators of stress level (Allen et al., 2014; Campbell & Ehler, 2012; Mauss & Robinson, 2009). The reasons for such divergence cannot be elucidated from this dissertation. However, it can be speculated that there are moderators that could influence the convergence of different measures that need to be further investigated. One such moderator of the association could be chronic stress. In particular, as Shah and Malla (2015) discuss in their review on different indicators of stress level in schizophrenia, it is possible that chronic stressors could alter biological acute stress reactivity but not necessarily psychological stress reactivity, so that acute biological and psychological stress reactivity may be more strongly associated in those with low chronic stress. Furthermore, we could speculate that psychological and biological systems could act to some extent independently and that, as Mauss and Robins (2009) discuss in their review on measurement of emotions, such dissociation between measures may not be an indicator of a dysregulated system but represent a rather healthy phenomenon. In Study II/III the relevance of a concurrent assessment of different indicators of stress level has been corroborated by the finding that not only a negative affect predicted the subsequent paranoid symptoms in everyday life but also that the preceding heart rate as a biological indicator acted independently as a predictor of paranoid symptoms. This further supports the explanation that to some extent biological and psychological response systems could act independently, as shown in studies that find that suppressing biological stress responses does not affect the psychological response (Ali, Nitshke, Cooperman, & Pruessner, 2017). Hence, Study I and Study II both highlight the importance of measuring different indicators



concurrently when trying to capture stress reactivity and its relevance to the emergence of psychotic symptoms.

Additionally, Study I and Study II offer further evidence on the “hypothesis of psychosis continuum”, in line with studies showing that individuals of varying vulnerability experience paranoid symptoms (e.g., Bebbington et al., 2013; Freeman et al., 2011; Freeman et al., 2005) Specifically, although in Study I the indicators of stress level were investigated during a stress reaction in healthy individuals, participants also responded to the stressor with a significant increase in paranoid and depressive symptoms. This implies that a symptomatic response to stressors is not only to be found in individuals with a specific mental illness diagnosis but also in the general population. Furthermore, the association between psychological and biological stress levels and subsequent paranoid symptoms that was found in Study II in a sample with attenuated psychotic symptoms provides additional evidence that the path from everyday stress reactivity to paranoia may be relevant even before the unfolding of the disorder.

Summarized, the findings of this dissertation corroborate the notion of the importance of different facets of stress level and their association with paranoid symptoms in both experimental and everyday life settings in non-clinical samples.

## **7.2 The process of formation, exacerbation and maintenance of paranoid symptoms**

In both studies that investigated the research question of a vicious cycle as an underlying process of paranoia formation, exacerbation and maintenance (Study II and IV), the hypothesis that stress level will predict subsequent paranoid symptoms and that paranoid symptoms will act as a stressor that further elevates the individual’s stress level was not confirmed. In particular, in Study II, in individuals with attenuated psychotic symptoms, there was only a uni-directional association of both previous negative affect and heart rate with subsequent paranoid symptoms. Similarly, in a clinical sample in Study IV, whereas specifically anxiety temporally predicted subsequent paranoid symptoms, paranoid symptoms only temporally predicted subsequent anger. Hence, also here, the vicious cycle was not directly confirmed.

Nevertheless, a more differentiated analysis in Study IV, where negative affect was examined separately for different emotions, offers valuable insights that could explain the process of the formation, exacerbation and maintenance of paranoid symptoms. First and foremost, this study corroborates the particularly important role of anxiety as a predictor of paranoid symptoms, which is in line with the assumption of Freeman and colleagues (2002) that anxiety and paranoid symptoms have a common theme in the “anticipation of danger”, making feelings of anxiety especially important to paranoid symptoms. Furthermore, Study IV shows that paranoid symptoms are accompanied with a mixture of negative emotions

(anxiety, guilt, shame, and anger) that could be interpreted as a diffuse, overwhelming negative state. As a result of paranoid symptoms however, only increased anger was evident. It can be assumed that paranoid symptoms, which represent externalizing attributional processes that take place in anxiety eliciting situations (Bentall et al., 2001), and are accompanied by overwhelming negative emotions, could have a regulatory purpose. In particular, although the measured emotions of anxiety, guilt, shame, and anger are all negative emotional states, anger represents the only negative emotion that facilitates the approach to the threatening situation instead of the avoidance of threat (Kashdan et al., 2015). Hence, if in anxiety eliciting situations the paranoid symptoms lead to a feeling of anger this could be in short-term beneficial for the individual. In terms of reinforcement learning this would facilitate the maintenance or even exacerbation of paranoid symptoms. This short-term benefit of paranoid symptoms has been found in previous studies showing that paranoid explanations for stressors could have a protective effect on self-esteem (Lincoln, Stahnke, & Moritz, 2014) and that paranoid stress reactivity is predictive of better autonomic recovery after a stressor (Clamor & Krkovic, 2018). However, this poses a question of what the consequence of paranoid symptoms is in the long-term: Possibly, regularly applying paranoid explanations in anxiety eliciting situations could lead to a self-affirming perception of a continuously hostile environment and generate more anxiety in the long run, that could eventually lead into an extended vicious cycle of anxiety, paranoid symptoms, anger, and subsequent anxiety due to threat anticipation.

Taken together, this dissertation does not directly support the existence of a vicious cycle between stress reactivity and paranoid symptoms neither in the non-clinical nor in the clinical sample but elucidates the exacerbation and maintenance of symptoms by highlighting the specific importance of anxiety as a precedent of paranoid symptoms and anger as its consequence.

### **7.3 Relevance of potential vulnerability factors for the formation of paranoid symptoms – focus on emotion regulation**

The results of Study I and Study II point to the stronger, more prominent role of maladaptive emotion regulation as a predictor of stress reactivity and as a moderator of the path from stress reactivity to paranoid symptoms, compared to adaptive emotion regulation. In particular, in the quasi-experimental Study I, higher score in the use of maladaptive emotion regulation predicted a stronger increase in negative affect in response to the stressor, suggesting a stronger stress reactivity in this group of individuals. The results for the endocrine stress response were only trend level significant, however, they yielded a pattern worth discussing. Namely, whereas the medium and low maladaptive emotion regulation scores were associated with an expected endocrine response with salivary cortisol

linearly increasing, particularly high scores in maladaptive emotion regulation were associated with a different stress reactivity pattern. The post-hoc results showed that these participants peaked in their salivary cortisol level before stress induction and that their cortisol levels then decreased linearly over the consecutive time-points. Hence, the group of participants with high maladaptive emotion regulation peaked in their salivary cortisol before the stressor and at the same time they reported the highest levels of negative affect after the stressor, compared to other groups. These results are in line with studies that have shown an influence of specific emotion regulation strategies on psychological and biological stress level (e.g., Bond & Bunce, 2003; Carlson et al., 2012; Denson, Creswell, Terides, & Blundell, 2014; Lam et al., 2009; Lincoln et al., 2015a; Mauss et al., 2007; Memedovic et al., 2010; Thomsen et al., 2003; Zoccola, Dickerson, & Zaldivar, 2008) and expand these by emphasizing the particular role of maladaptive emotion regulation for psychological and endocrine stress reactivity. Furthermore, the diverging pattern of psychological and endocrine results depending on emotion regulation offers insights into the acute stress reactivity of vulnerable individuals. Similar to our results, also Villada and colleagues (2016) found two clusters: one was comprised of individuals who had low trait anxiety and high active coping and showed low psychological and high endocrine response, and another cluster of individuals who had high trait anxiety and low scores on active coping and showed high psychological but low endocrine response. Taken together, these results offer support for the dysfunctionality of hypo-reactivity of the HPA axis during acute stress reactions. This is in line with the findings reviewed by Shah and Malla (2015) suggesting that blunted acute reactivity of the HPA axis could result from a chronically hyper-activated HPA axis and represents an “accumulated exhaustion” of the HPA axis in vulnerable individuals. Furthermore, this finding is in accord with the coherence/compensation model of stress response systems of Andrews, Ali and Pruessner (2013), who discuss that a hypo-activation of one system could lead to a hyper-activation of another system. Summarized, results of Study I indicate that an endocrine hypo-reactivity and affective hyper-reactivity are evident in vulnerable individuals even before the emergence of a disorder and that emotion regulation as a vulnerability factor could represent a significant determinant of such dysregulation.

Interestingly, in Study I, increases in depressive and paranoid symptoms in response to the stressor were not predicted by the maladaptive emotion regulation. However, in Study II, maladaptive emotion regulation moderated the link between negative affect and paranoia. In particular, participants who reported higher use of maladaptive emotion regulation also reported more paranoid symptoms after experiencing negative affect in their daily life. This implies that maladaptive emotion regulation could be a risk factor for formation and exacerbation of paranoid symptoms specifically by strengthening the path from daily stress reactivity to paranoid symptoms.

The findings of Study I and Study II together suggest that adaptive emotion regulation does not act as a vulnerability factor for stress reactivity and formation of paranoid symptoms. The stronger relevance of maladaptive compared to adaptive emotion regulation is in line with accumulating research in recent years showing maladaptive strategies to be more strongly related to general psychopathology (Aldao et al., 2010; Aldao & Nolen-Hoeksema, 2012; Moritz, Jahns, et al., 2016) and paranoid symptoms (Westermann, Boden, Gross, & Lincoln, 2013), compared to adaptive emotion regulation. Thus, individuals who report to regularly employ adaptive emotion regulation do not seem to fare better when it comes to stress reactivity and paranoid symptoms. Possibly, due to cognitive overload and the inhibition of prefrontal functions during a stress response (Schwabe & Wolf, 2009), individuals may fail to apply adaptive strategies (Raio, Orederu, Palazzolo, Shurick, & Phelps, 2013). Furthermore, it could be assumed that adaptive emotion regulation is not protective per se, but only in the presence of maladaptive strategies and has a compensatory role (Aldao & Nolen-Hoeksema, 2012).

Surprisingly, in both studies that measured heart rate as an indicator of biological stress level (Study I and Study II) there was no influence of emotion regulation on this indicator or on the path from stress level to paranoid symptoms. One explanation for this could be that this pathway is influenced by the behavioral rather than cognitive strategies, for instance changing the pace of breathing, or muscle relaxation. Nevertheless, this speculative explanation requires further empirical investigation.

Taken together, maladaptive emotion regulation seems to act as a vulnerability factor for psychological hyper-activity and endocrine hypo-activity during an acute stress response, and at the same time as a moderator of the path from negative affect to paranoid symptoms in everyday life. Therefore, reduction of maladaptive strategies could represent a crucial target in therapeutic interventions to prevent the development of paranoid symptoms.

#### **7.4 Relevance of potential vulnerability factors for the formation of paranoid symptoms – focus on traumatic experiences**

The assumption that various characteristics of traumatic experiences would contribute differently to explaining the path from stress reactivity to paranoid symptoms in everyday life was corroborated by the results of Study III. Specifically, when it comes to the direct link between trauma characteristics and paranoid symptoms, more frequent, recurring trauma and sexual trauma were predictive of reporting more paranoid symptoms in everyday life. Paranoid symptoms had no direct link to the age at which the trauma took place, non-intentional trauma, intentional trauma related to crime and physical trauma. The finding that sexual but not physical trauma was directly associated with paranoid symptoms contradicts studies by Fisher and colleagues (2010) and Rubino and colleagues (2009) who found

physical trauma to be the strongest and most robust predictor of psychosis. The reason for the divergence of this result from previous findings could be that in contrast to our study, Fisher and colleagues (2010) and Rubino and colleagues (2009) did not investigate the association between abuse and specific symptoms but rather the association of abuse with psychosis as a diagnostic category. Results of this dissertation could be better explained by the findings of the study by Murphy and colleagues (2012) where paranoia had a role as a mediator between sexual abuse and psychosis. Furthermore, In line with this, in the most recent meta-analysis Bailey and colleagues (2018) found that compared to other traumatic experiences, childhood sexual abuse was most robustly related with delusions. Altogether, this suggests that diverging findings could be explained by their focus on either psychosis or paranoid symptoms and that sexual abuse could be especially relevant to explaining development of paranoia.

With regard to the mechanism that links trauma to everyday stress reactivity and paranoia, together with two recent ESM studies (Rauschenberg et al., 2017; van Nierop et al., 2018) findings of this dissertation provide additional support for the moderation model suggesting that higher frequency, younger age and physical trauma strengthen the path from negative affect in daily life to paranoia. The emotional processing theory (Foa and Kozak, 1986, Foa et al., 1989) could offer an explanation for this finding. Possibly, these trauma characteristics facilitate the activation of fear structures that serve to escape or avoid danger in response to stress, which in turn strengthens the threat beliefs. Furthermore, finding physical but not sexual trauma to be a significant moderator was surprising given that sexual trauma was directly associated with paranoid symptoms. Nevertheless, this could suggest that different types of trauma influence paranoid symptoms in a different manner, some directly and some by strengthening the path from stress reactivity to paranoia. Possibly there could even be an additive effect and interactions between different trauma characteristics, as was found by Fisher and colleagues (2010) who reported that physical abuse from the main mother figure before the age of 12 showed the most robust associations with psychosis. Such additive effects need to be investigated in future studies.

In Study III, the association between heart rate as a biological indicator of stress reactivity and paranoid symptoms in everyday life was not moderated by any trauma characteristic. Together with the findings of Study II, which show no moderation effect of emotion regulation on this path, it could be assumed that the path from elevated autonomic arousal to paranoid symptoms is somewhat independent from the path from negative affect to paranoid symptoms. It requires theoretical considerations that I propose in the Outlook section of this dissertation and future empirical studies to examine possible moderators of this path.

## 7.5 Limitations

This dissertation has several important limitations that need to be considered when interpreting the results. These will be outlined here for all studies. Considering that Study II and Study III were parts of the same project, their limitations will be presented jointly.

The major limitation of Study I is its quasi-experimental design – without a control condition, it remains unclear whether the change in stress level indicators can be attributed to the applied stressor, or if it is a result of the time effect or assessment situation in general. Furthermore, although Study I offers insights into what a stress response and recovery are constituted of and it further elucidates how emotion regulation relates to acute stress reactivity thus making its findings valuable for the research on stress reactivity in psychosis, future studies should employ a sample that includes individuals across the continuum of psychosis. Only by including participants ranging from those without any psychotic experiences to those with a severe psychosis, conclusions can be made regarding the differences between a healthy and a pathological stress reactivity.

For Study II and Study III it is necessary to discuss the limitations of the study design. Specifically, in order to include the ambulatory assessment of the heart rate that represents a biological counterpart of a psychological stress level, the ESM was limited to only 24 hours. This short period of time limited the representativeness of the results for everyday life. To compensate for such a short ESM period a high number of assessment points during the day was included. However, this high frequency assessment could have triggered specific assessment reactivity in the participants, such as increased burden or awareness of cognitive and affective states (Vachon, Rintala, Viechtbauer, & Myin-Germeys, 2018). Furthermore, the assessment of negative affect was to some extent retrospective since it referred to the time period between two assessment time-points, which limits the “momentary” nature of the ESM method. Another limitation could be seen in the sample that primarily consisted of students. This could have potentially limited the generalizability of the findings to the general population where lower education and socio-economic status could be expected.

There are further limitations that are specific for Study III. In particular, the assessment of trauma relied on self-report that is prone to memory bias. Furthermore, the THQ used in this study to assess trauma does not collect data on perceived severity of the trauma, which has been considered important to determining the risk of psychosis (Kelleher et al., 2013; Spauwen et al., 2006). Additionally, the subsamples with specific trauma types were relatively small, so that it is possible that for some effects the power to detect differences was too low.

The major limitation of Study IV is that it solely relied on data from self-reports. As other studies of this dissertation show, complementing psychological indicators of stress

level with endocrine and autonomic indicators could be highly relevant. However, at this point, an ambulatory assessment of such biological indicators over an extended period of time is restricted. As in Study II, also here the repeated measures could have led to specific reactivity in participants. Furthermore, this study was based on the assumption that participants were able to recognize and name their emotional states. However, previous research has shown that patients with psychosis have higher values on alexithymia (O'Driscoll et al., 2014) that could have biased our results. One further limitation is that the sample included individuals with the diagnosis of a schizo-affective disorder. It could be expected that these individuals have different patterns of affectivity than those with non-affective psychosis (e.g., larger and more frequent fluctuations of specific emotions). Due to small subsamples a separately analysis of diagnosis-subgroups was not possible.

## **7.6 Clinical implications**

Despite the limitations outlined in the previous section, this dissertation significantly contributes to the research on processes involved in formation, exacerbation and maintenance of paranoid symptoms. Studies I, II, and III together imply the complexity of stress reactivity and highlight the relevance of not only psychological but also biological indicators of stress level. Therefore, addressing these in therapeutic settings could be of importance. In particular, this dissertation clearly speaks for focusing interventions on the reduction of negative affect in response to minor stressors in everyday life. At the same time, considering the complexity of the stress reactivity, such interventions should also aim to reduce autonomic arousal that seems to be predictive of paranoid symptoms in everyday life. For instance, breathing biofeedback or muscle relaxation that have been found to have an impact on the autonomic arousal (Lehrer, 2017; Lehrer & Gevirtz, 2014) could be good candidates for such interventions.

In light of the findings of this dissertation on the relevance of maladaptive emotion regulation for negative affect and its path to paranoid symptoms, interventions aiming at the reduction of the use of such maladaptive strategies could be indicated. The results also imply that teaching adaptive emotion regulation in the course of treatment may not be sufficient. If future studies would replicate our findings, this could have strong influence on clinical practice. In particular, mindfulness-oriented interventions that have been increasingly applied to practice acceptance towards negative emotional states and anxiety eliciting thoughts (for a review on mindfulness approaches in psychosis, see Aust and Bradshaw, 2017) would need to be complemented with interventions that explicitly aim to reduce maladaptive strategies. The transdiagnostic affect regulation training by Berking and Whitley (2014) could be a promising approach to preventing the development and exacerbation of paranoid symptoms in everyday life. Specifically, such emotion regulation trainings aim to reduce maladaptive

emotion regulation strategies and at the same time provide strategies to become aware of, identify and understand emotions with regard to triggering situations and underlying cognitions, which could be valuable for symptom phases that were found to be emotionally overwhelming in Study IV. Furthermore, emotion regulation trainings teach strategies to consciously modify, tolerate or accept emotions such as anxiety that was an important trigger of paranoid symptoms in Study IV.

Finally, the findings of this dissertation could be integrated in individuals' explanatory models that serve as the foundation for interventions in cognitive behavioral therapy. Specifically, the close association between specific characteristics of trauma and paranoid symptoms could help raise the understanding of "why did I develop exactly this disorder" at the beginning of the therapy as a part of psychoeducation that is considered to be an important precondition for a successful treatment (Bäumli, Froböse, Kraemer, Rentrop, & Pitschel-Walz, 2006). The reinforcement learning model of paranoia connecting specific negative emotions with symptoms could be helpful to derive the learning mechanisms and their short-term versus long-term consequences for the patient. Last but not the least, identifying vulnerable individuals could improve preventive measures that could be offered to these individuals to prevent negative affect from translating into symptoms.

## **7.7 Outlook**

The complexity of stress reactivity and its association with paranoid symptoms need further investigation. Specifically, future ESM studies should aim to include different biological indicators of stress level over a longer period of time to substantiate the findings of this dissertation and to ensure the generalizability of the results for everyday life. For instance, expanding the ambulatory assessment of heart rate to a longer period of time and complementing it with the ambulatory assessment of salivary cortisol would offer more comprehensive information on stress reactivity in daily life.

In this dissertation none of the hypothesized moderators influenced the path from elevated heart rate to paranoid symptoms in everyday life. In the first step, this finding needs to be replicated. In the second step, potential factors should be derived from existing models as well as research that could be relevant to this path. In the present work I suggest that it is more likely that behavioral strategies could play a role for this path than emotion regulation and trauma that were tested within this dissertation. In particular in Study I, where heart rate was not determined by emotion regulation, we observed behavioral strategies in some participants, which they used to cope with the situation: clenching their fists, taking deep breaths, looking away or focusing on one spot. This could be a relevant hint for future research: whereas breathing pace and muscle relaxation have been found to influence autonomic arousal (Lehrer, 2017; Lehrer & Gevirtz, 2014), it remains to be investigated whether or not such behavioral strategies could weaken the path to paranoid symptoms.



With regard to the missing moderating effect of adaptive emotion regulation on the stress reactivity and the path from negative affect and paranoid symptoms, new hypotheses for future research could be generated. Specifically, adaptive strategies such as reappraisal or putting into perspective require the ability to gather and integrate new, relevant information. Therefore, for adaptive emotion regulation to moderate the path from negative affect to paranoid symptoms, unbiased cognition could be a precondition. This is however where individuals vulnerable to psychosis seem to have difficulties (e.g., Dudley, Taylor, Wickham, & Hutton, 2016; Ross, McKay, Coltheart, & Langdon, 2015). Hence, cognitive bias could aggravate the use of adaptive emotion regulation so that the effect from negative affect to paranoid beliefs cannot be successfully moderated. This hypothesis requires empirical tests where both cognitive bias and emotion regulation could be assessed at the same time to examine if their interaction has an effect on the path from negative affect to paranoia.

Furthermore, when it comes to traumatic experiences as a vulnerability factor for psychosis, relying on the sociodevelopmental-cognitive model of schizophrenia by Howes and Murray (2014), it could now be tested if dopamine system (dys-)regulation that is often found in psychosis can be linked to specific characteristics of trauma identified in our study and to examine how these relate to other etiological factors. Specifically, whereas this dissertation points to the importance of emotion regulation and traumatic experiences as vulnerability factors for psychosis, the additive effect of different vulnerability factors and their interplay remain unanswered. To address this research question, large-scale longitudinal studies are required that could analyze such interaction effects with sufficient power and at the same time enable time-lagged analyses that would inform us specifically regarding the process of symptom formation. Based on vulnerability stress models, however, an additive effect could be expected. Moreover, studies comparing the findings of this dissertation with findings in other mental disorders are needed to get more insights into the specificity of the effects found in this dissertation. In particular, this dissertation focused on paranoia as one of the lead symptoms of schizophrenia and other psychotic disorders. However, considering that emotion regulation, traumatic experiences and affective processes could be relevant for other disorders as well, it could be assumed that processes found in this dissertation are more general for psychopathology than specific for psychosis. Therefore, future research should focus on the investigation of the specificity and whether or not here investigated processes have more relevance to psychosis than to other psychopathology.

Finally, while this dissertation substantiates and expands the etiological understanding of psychosis, in the last paragraph I want to encourage future researchers to invest more effort into the research on prevention. For one, social programs reducing violence in families and outreach programs offering support to trauma victims could have a large impact and reduce the prevalence rates of psychotic disorders. Moreover,

implementing emotion-focused prevention programs as early as in elementary schools could possibly be an important step towards a mindful society and represent a chance for vulnerable individuals to learn how to regulate their stress reactivity and become more resilient before the first symptoms emerge. Whether or not such prevention programs are effective remains to be investigated but bearing in mind personal and societal costs of schizophrenia, all efforts towards prevention research must be considered justified.

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## Appendix A: Study I

Krkovic, K., Clamor, A., & Lincoln, T. M. (2018). Emotion regulation as a predictor of the endocrine, autonomic, affective, and symptomatic stress response and recovery. *Psychoneuroendocrinology, 94*, 112-120.





## Emotion regulation as a predictor of the endocrine, autonomic, affective, and symptomatic stress response and recovery



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### ABSTRACT

Stress is associated with the development of mental disorders such as depression and psychosis. The ability to regulate emotions is likely to influence how individuals respond to and recover from acute stress, and may thus be relevant to symptom development. To test this, we investigated whether self-reported emotion regulation predicts the endocrine, autonomic, affective, and symptomatic response to and recovery from a stressor. Social-evaluative stress was induced by the Trier Social Stress Test (TSST) in  $N = 67$  healthy individuals (53.7% female,  $M_{age} = 29.9$ ). Self-reported habitual emotion regulation skills were assessed at baseline. We measured salivary cortisol, heart rate, negative affect, state depression and state paranoia at three time points: pre-TSST, post-TSST, and after a 10 min recovery phase. Repeated-measures ANOVA showed all indicators to significantly increase in response to the stressor ( $p < .001$ ) and decrease during the recovery phase ( $p < .001$ ), except for salivary cortisol, which showed a linear increase ( $p < .001$ ). The habitual use of maladaptive emotion regulation (e.g., rumination, catastrophizing) significantly predicted an increased affective and reduced cortisol response. Adaptive emotion regulation (e.g., acceptance, reappraisal) was not predictive of the stress response for any of the indicators. Neither type of emotion regulation predicted response during the stress recovery phase. Individuals who habitually resort to maladaptive emotion regulation strategies show a stronger affective and a blunted endocrine stress response, which may make them vulnerable to mental health problems. However, further research is needed to identify the full scope of skills required for effective stress-regulation before this knowledge can be used to develop effective prevention programs.

### 1. Introduction

Vulnerability-stress-models of psychopathology propose that vulnerable individuals show an increased sensitivity to acute stressors that is reflected in stronger affective, endocrine, and autonomic response, possibly accompanied by a “symptomatic” response. Dating back to the 1980’s and earlier, this model has been used to explain the development of mental disorders, such as depression (e.g., Beck, 1987; Bebbington, 1987) and psychosis (e.g., Zubin and Spring, 1977), which has inspired research on the phenomenon of stress-sensitivity in the context of these mental disorders.

In order to investigate the stress-sensitivity, researchers have applied different methods ranging from experimental designs, where the stress is induced in the laboratory, to the experience sampling method (ESM), where data is collected via electronic diaries in everyday life. In one ESM study, Myin-Germeys et al. (2003) found that both individuals with psychosis and those with depression exhibit elevated emotional sensitivity to daily stressors. Similarly, individuals at risk for psychosis

show an increased emotional response to stressors in everyday life (Lataster et al., 2009) and an increased endocrine response to daily stress (Collip et al., 2011). Experimental studies have also found both emotions and symptoms to increase in response to stressors in individuals with psychosis (e.g., Ellett et al., 2008; Freeman et al., 2015; Lincoln et al., 2015b; Veling et al., 2016). Hence, there is solid empirical evidence that psychosis and depression are associated with stronger emotional responses to stress, which tend to translate into symptoms.

This raises the question by which specific vulnerability factors the emotional stress response is being driven. It seems intuitive to expect that one such vulnerability factor could be impaired emotion regulation (ER). In some regards ER is a similar concept to coping (Compas et al., 2014; Wang and Saudino, 2011). However, in contrast to coping, which has been defined as “constantly changing cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person” (Lazarus and Folkman, 1984, p. 141), the concept of ER specifically refers to “the

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process by which individuals influence which emotions they have, when they have them, and how they experience these emotions" (Gross, 1998, p. 275). Thus, ER is more narrowly defined than coping and is likely to be the more suitable concept when it comes to understanding the emotional stress response that precedes psychotic symptoms.

In healthy individuals, different ER strategies were found to be important in relation to different aspects of subjective stress responses. In particular, the habitual use of rumination (i.e., focusing attention on the situation and the emotion) was associated with increased negative mood-congruent thinking and with increased endocrine reactivity or delayed recovery (e.g., Thomsen et al., 2003; Zoccola and Dickerson, 2012; Zoccola et al., 2008). Studies also demonstrate negative effects on well-being when negative emotions are suppressed (i.e., inhibiting behaviors associated with emotional responding; e.g., Butler et al., 2003; Gross and John, 2003), whereas accepting emotions was found to have beneficial effects on mental health (Bond and Bunce, 2003). Empirical results on habitual reappraisal are mixed, with some studies reporting a negative association with self-reported negative affect and physiological responses (e.g., Carlson et al., 2012; Memedovic et al., 2010; Mauss et al., 2007) suggesting a resilience function of reappraisal. In contrast, other studies found no effect of reappraisal on self-reported stress and a positive association with physiological responses (e.g., Denson et al., 2014; Lam et al., 2009). Although previous work indicates that specific ER strategies such as rumination or reappraisal are relevant to stress responses and well-being in healthy individuals, it also suggests that different strategies tend to load on the same latent factor rather than representing separate constructs (Aldao and Nolen-Hoeksema, 2010; Garnefski et al., 2001). As a consequence, different strategies are often subsumed into "maladaptive" strategies if their habitual use is considered to be less advantageous (e.g., rumination, suppression), and into "adaptive" strategies if their habitual use is considered as more advantageous (e.g., reappraisal, acceptance). It has been shown that the habitual use of maladaptive strategies is related to negative outcomes such as psychopathology, whereas the habitual use of adaptive strategies has been found to protect against psychopathology (Aldao et al., 2010).

Specifically, increased habitual use of maladaptive strategies has been found to be associated with depression (Aldao et al., 2010) and psychosis (O'Driscoll et al., 2014). Furthermore, several studies point to the lack of habitual use of adaptive strategies as a risk factor for psychosis and depression (e.g., Joormann and Stanton, 2016; Kimby et al., 2012; Lincoln et al., 2015a,b; Perry et al., 2011; van der Meer et al., 2009). Furthermore, in an at-risk sample lower levels of habitual use of adaptive and higher levels of maladaptive ER accounted for the increase in paranoid beliefs after social exclusion (Lincoln et al., 2017b). Thus, increased habitual use of maladaptive and limited habitual use of adaptive ER could alter the stress response and therefore represent a risk factor for the development of depression and psychosis in the longer term.

However, there are two pitfalls of the available research. One is that a stress response includes various indicators, such as an endocrine, autonomic, affective, and symptomatic response. So far, however, most studies have focused on either subjective stress indicators or cortisol responses alone. Few studies have concurrently assessed numerous stress indicators and those that did have found some indicators (e.g., subjective stress) to be more responsive to experimentally induced stress than others (e.g., cortisol) in people with psychosis or depression (e.g., Lincoln et al., 2015a,b). This aligns with studies pointing to a dysregulated hypothalamic-pituitary-adrenal (HPA) axis in these disorders (Chaumette et al., 2016; Shah and Malla, 2015; Stetler and Miller, 2011). Other studies point to compensatory patterns of different stress indicators (Ali et al., 2017). This indicates that a pathological stress response could be characterized by a complex pattern of hyper- and hyposensitivity of different stress indicators, which would require a comprehensive, concurrent measurement of various stress indicators in order to be detected.

The other pitfall is that although the definition of emotion regulation also includes influencing the *duration* of the emotion, research has mostly focused on the acute stress response. This is problematic, as a prolonged recovery from a stressor could represent an additional burden for individuals. The few studies that considered this aspect have been summarized in a review by Zoccola and Dickerson (2012) who discuss the mixed findings on whether or not the increased rumination is associated with a delayed cortisol decrease after the cessation of a stressor in healthy individuals. Furthermore, Lewis et al. (2017) found that in healthy individuals, the adaptive ER strategy reappraisal was associated with greater cortisol recovery after a stressor. Thus, there is some indication that ER strategies could have an impact on the recovery from a stressor, but this evidence is limited to the endocrine stress recovery.

The present study thus extends on the existing research in this field by testing the hypothesis that higher scores in habitual maladaptive and lower scores in habitual adaptive ER skills will predict 1) a stronger acute stress response and 2) a weaker recovery from a social evaluative stressor, which will be evident across a broad spectrum of stress indicators, including salivary cortisol, heart rate, affect, and symptoms.

## 2. Methods

### 2.1. Participants

Participants were recruited by print adverts in the facilities of the Universität Hamburg, churches, job centers, residential homes in Hamburg, and via several internet postings (e.g., Facebook, Stellenwerk, Ebay-Kleinanzeigen). To be included, the participants needed to be 18 years or older, have sufficient command of the German language, have no mental disorder and be able to provide informed consent. Additional exclusion criteria involved factors that could possibly influence the physiological stress response: any kind of medication, hormonal contraception, diagnosed cardiac or thyroid disorders, smoking, drug use, as well as eating, caffeine intake or sports within two hours prior to testing.

The final sample consisted of 67 participants (53.7% female,  $M_{age} = 29.94$ ,  $SD_{age} = 12.25$ ). About one third of the participants (29.9%) reported working full-time and 58.2% were students. The majority of the participants reported having a university degree (36%) or a general qualification for university entrance (A-level equivalent, 35%), 18% reported having a lower school degree and 11% reported having a professional training degree. Participants' mean body mass index (BMI) was  $M_{BMI} = 23.18$  ( $SD_{BMI} = 3.61$ ).

### 2.2. Design and procedure

The procedure is depicted in Fig. 1. The first part of the study consisted of a questionnaire battery that the participants completed online. After this, they were invited to participate in the second part of the study that took place at the Universität Hamburg. All appointments took place between 10AM and 7PM. After arriving at the lab, the participants were informed about the procedure and signed an informed consent, after which an ambulatory electrocardiogram (ECG) was attached to the participants' chest, pre-TSST heart rate was measured (HRT1; see Fig. 1) and the first (i.e., pre-TSST) assessment of salivary cortisol, negative affect, state depression and paranoia took place (t1; see Fig. 1). Following this, the participants completed cognitive assessment tests (ca. 30 min). Next, all participants underwent the Trier Social Stress Test (TSST; Kirschbaum et al., 1993). The test consisted of an anticipation period (3 min) where the participants were asked to prepare a speech and a test period where the participants had to deliver a speech (5 min) and then perform mental arithmetic tasks (5 min) in front of an audience. During the speech task, the second measurement of heart rate took place (HRT2; see Fig. 1). Immediately after the stress manipulation, the second assessment of salivary cortisol, negative

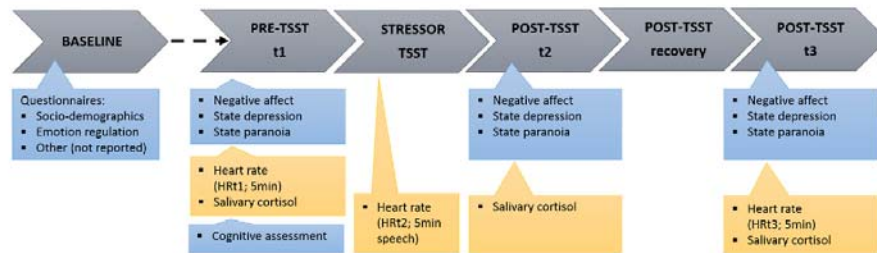


Fig. 1. Graphic procedure. TSST = Trier Social Stress Test; t1 = time-point before the TSST; t2 = time-point immediately after the TSST; t3 = time-point after 10 min recovery phase; HRT1 = time-point of the mean heart rate baseline before the TSST; HRT2 = time-point of the mean heart rate during the 5 min speech phase; HRT3 = time-point of the mean heart rate during the first 5 min of the recovery phase.

affect, state depression and paranoia was conducted (t2, see Fig. 1). The last part of the study included a 10 min recovery phase during which participants were left alone in a neutral environment where they could sit down on a chair and relax. Following that, the heart rate (HRT3), salivary cortisol, negative affect, depression and paranoia symptoms were assessed for the last time (t3, see Fig. 1). At the end, participants were debriefed and received a monetary compensation of 25€ (10€ per hour). The project was approved by the local ethics committee.

### 2.3. Assessment instruments

The present study was part of a larger project, in which further questionnaire assessments were included in the online questionnaire assessment (i.e., demographic information, assessments of chronic stress, psychosocial stressors, and traumatic experiences). After the pre-TSST stress assessment and before the stress induction, the Wisconsin Card Sorting Test (WCST; Heaton et al., 1993), a variant of the Beads-Task paradigm (Moritz et al., 2011), and the Young Schema Questionnaire (YSQ-S3; Young, 2006) were administered but were not analyzed for the purpose of this study. Here, we describe only those assessments relevant to this study.

#### 2.3.1. Baseline assessment of emotion regulation

Because no questionnaire captures the full range of ER strategies relevant to psychosis or depression, we used two different questionnaires to assess adaptive and maladaptive strategies: the German version of the emotion specific Emotion-Regulation Skills Questionnaire (ERSQ-ES, Ebert et al., 2013) and the German version of the Cognitive Emotion Regulation Questionnaire (CERQ, Garnefski and Kraaij, 2007; Loch et al., 2011).

**2.3.1.1. Adaptive emotion regulation strategies.** The ERSQ-ES measures adaptive emotion regulation separately for stress, anxiety, anger, sadness, and shame. The individual thereby estimates the highest intensity of each emotion during the previous week on an 11-point scale. For each emotion that occurred in the previous week, specific emotion regulation strategies are assessed on a Likert-type scale ranging from 0 (“not at all”) to 4 (“almost always”). The strategies encompass clarity, understanding, acceptance, tolerance, self-support, willingness to confront situations causing undesired emotions when necessary to attain personally relevant goals, and modification. The ERSQ-ES has shown good to excellent psychometric properties in large clinical and non-clinical samples (Ebert et al., 2013). The following additional adaptive strategies were assessed with the CERQ: acceptance, positive refocusing, refocusing on planning, positive reappraisal, and putting into perspective. The German version of the CERQ has acceptable to good psychometric properties (Loch et al., 2011). We calculated a composite score from adaptive strategies assessed with ERSQ-ES and CERQ by calculating the mean score across adaptive strategies assessed

by the two questionnaires. The internal consistency of the composite adaptive score was good in our sample (Cronbach's  $\alpha = 0.83$ ).

**2.3.1.2. Maladaptive emotion regulation strategies.** The habitual use of maladaptive emotion regulation strategies was measured with the maladaptive emotion regulation subscale of the CERQ, composed of the strategies self-blame, catastrophizing, rumination/focus on thought, and blaming others. A total score was calculated as a mean across different maladaptive strategies. The internal consistency of the maladaptive subscale in our sample was acceptable (Cronbach's  $\alpha = 0.75$ ).

#### 2.3.2. Assessment of stress indicators

**2.3.2.1. Negative affect.** The affective stress response was assessed at t1, t2 and t3 (see Fig. 1) with four items rated on a 10-point scale ranging from 1 (“does not apply at all”) to 10 (“applies very much”) capturing self-ratings of momentary anxiety, sadness, anger, and shame (e.g., “I feel annoyed/angry/mad”; “I feel anxious/fearful/afraid”). This scale has been used in previous studies where stress was induced in a laboratory (Lincoln et al., 2015a,b), or in an experience sampling study where affective responses were tracked in everyday life (Krkovic et al., 2018), and has been shown to be sensitive to change and therefore suitable for the measurement of the stress response.

**2.3.2.2. Salivary cortisol.** The HPA axis activation in response to the stressor was assessed via salivary cortisol. It was collected at t1, t2 and t3 (see Fig. 1) with a salivary collection device (Salivette<sup>®</sup>, Sarstedt, Germany). Saliva samples were stored at  $-50^{\circ}\text{C}$  until the end of the data collection and at  $-18^{\circ}\text{C}$  until the data processing. Cortisol concentration was determined by a luminescence assay (IBL).

**2.3.2.3. Heart rate.** The autonomic nervous system response was measured as heart rate per minute using a sensor designed for collecting an ambulatory electrocardiogram (Movisens ECGmove). ECGmove is a small  $62.3 \times 38.6 \times 11.5\text{ mm}$  sensor attached to electrodes that were placed on the left side of the chest and collected the signal continuously. For the purposes of the present analysis, a mean heart rate for five minutes intervals was calculated at HRT1, HRT2, and HRT3 (see Fig. 1). We performed the correction of the peak detection manually in Kubios (Version 2.2 and 3.0 Premium). If artefacts occurred due to movement or disturbances in electrode fixation, medium automatic correction was conducted.

**2.3.2.4. Psychopathology.** As an indicator of a symptom response, we included a brief assessment of state symptoms of paranoia and depression at t1, t2, and t3 (see Fig. 1). State depression was assessed with a five item ultra-short version of the German version of the Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977) [German: Allgemeine Depressions-Skala (ADS), Hautzinger and Bailer,

1993] rated on a 10-point scale ranging from 1 (“does not apply at all”) to 10 (“applies very much”) and referring to the moment of the assessment. For the purpose of the state measurement, we used the items that we identified as sensitive to change in an unpublished analysis of the dataset published by Lincoln et al. (2015a,b) (e.g., “I have trouble keeping my mind on what I am doing”; “I can’t get going”; see Appendix A in the supplementary material). State paranoia symptoms were assessed with a five item version of the Paranoia Checklist (Freeman et al., 2005) rated on a 10-point scale ranging from 1 (“does not apply at all”) to 10 (“applies very much”) (e.g., “I need to be on my guard against others”; “My actions and thoughts might be controlled by others”). This short version has been validated by Schlier et al. (2016) and shown to be sensitive to change in momentary assessments.

#### 2.4. Statistical analyses

We used IBM SPSS Statistics software (version 22.0) for data analyses. First, in order to test how different indicators of stress relate to one another, we calculated partial correlations between different indicators of stress within each time-point, partialling out the respective other indicators and scores at previous time-points. Due to the latency of cortisol response, we additionally correlated self-report measures and heart-rate at t2 with salivary cortisol at t3 while controlling for scores at previous time-points. Furthermore, we calculated correlations between the emotion regulation variables and all stress indicators prior to TSST.

In order to test whether ER predicts the stress response and recovery, we ran linear models (GLM) for repeated measures. All predictors were centered around the group mean. In a first step, we analyzed the time effects (change in scores from t1 to t2 and to t3) by running the GLMs for each stress response indicator as a repeated measure dependent variable without including predictors. Pairwise comparisons with Bonferroni adjustment for multiple comparisons and the test of within-subjects contrasts informed which exact time-points differed (t1–t2; t2–t3). Then, for each stress response indicator, we separately analyzed whether the stress response depended on the ability to regulate emotions by adding the adaptive and maladaptive emotion regulation scores as continuous predictors. Tests of within-subjects contrasts informed on which exact time-points differed (t1–t2; t2–t3) in interaction with the predictors. Finally, we tested whether the results remain stable after controlling for possible confounding variables – gender, age, BMI, and time of the assessment (in order to account for the circadian rhythm) – by adding these as additional covariates.

For exploratory purposes, we conducted an additional repeated measures GLMs with the nine different ER strategies assessed in the CERQ as predictors.

### 3. Results

#### 3.1. Descriptive statistics

The mean scores and standard deviations of the emotion regulation scores at baseline, as well as negative affect, cortisol, heart rate, state depression, and state paranoia at t1, t2, and t3 are presented in Table 1.

#### 3.2. Correlation between affective, endocrine, autonomic, and symptomatic stress indicators and emotion regulation

All self-report measures (negative affect, state depression and state paranoia) were substantially intercorrelated with partial correlations ranging from  $r = 0.307$  to  $r = 0.555$  at t1, from  $r = 0.341$  to  $r = 0.680$  at t2, and from  $r = 0.649$  to  $r = 0.781$  at t3. Salivary cortisol and heart rate correlated significantly at t1 with  $r = 0.341$  but were not significantly correlated at t2 and t3. All correlations between self-report measures and physiological measures were non-significant. Correlations with salivary cortisol at t3 showed a significant partial

**Table 1**

Mean, standard deviation and range of emotion regulation scores at baseline, and stress indicators before and after a stressor and after a recovery phase.

	N	M	SD	Range
Maladaptive emotion regulation	67	1.50	0.57	0.42–3.17
Adaptive emotion regulation	67	5.20	1.03	3.14–7.83
Negative affect t1	67	0.49	0.64	0.00–2.50
Negative affect t2	65	2.02	2.09	0.00–8.25
Negative affect t3	66	0.82	1.52	0.00–6.00
Salivary cortisol t1	62	4.97	3.98	1.04–20.03
Salivary cortisol t2	61	6.89	5.08	1.13–28.69
Salivary cortisol t3	61	8.56	6.53	0.49–36.75
Heart rate HRt1	58	78.04	12.16	52.77–109.09
Heart rate HRt2	59	100.43	17.65	66.76–136.16
Heart rate HRt3	58	76.21	11.69	51.58–107.13
State depression t1	67	0.84	0.84	0.00–3.20
State depression t2	65	1.78	1.48	0.00–6.80
State depression t3	66	0.91	1.11	0.00–5.20
State paranoia t1	67	0.69	0.80	0.00–4.00
State paranoia t2	65	2.83	1.68	0.00–8.40
State paranoia t3	66	0.98	1.28	0.00–4.60

Notes. t1 = time-point before Trier Social Stress Test; t2 = time-point after Trier Social Stress Test; t3 = time-point after 10 min recovery phase; HRt1 = five minutes period prior to Trier Social Stress Test; HRt2 = five minutes during the speech task in Trier Social Stress Test; HRt3 = first five minutes of the recovery phase.

correlation with heart-rate at t2,  $r = 0.344$ ,  $p = .021$ , but no significant partial correlation with any self-report measure at t2 (negative affect:  $r = 0.050$ ,  $p = .746$ ; depression:  $r = 0.084$ ,  $p = .584$ ; paranoia:  $r = 0.237$ ,  $p = .117$ ).

Correlations between adaptive ER and stress indicators prior to TSST ranged from  $r = 0.020$  with heart rate to  $r = -0.170$  with depression (all  $p_s > .05$ ). Maladaptive ER significantly correlated with self-reported measures prior to TSST (negative affect:  $r = 0.383$ ,  $p = .001$ ; depression:  $r = 0.514$ ,  $p < .001$ ; paranoia:  $r = 0.279$ ,  $p = .022$ ), whereas the correlations of maladaptive ER with heart rate and salivary cortisol were non-significant ( $p > .05$ ).

#### 3.3. Stress response across the time-points

Mauchly’s test indicated that the assumption of sphericity was violated for the main effects for all dependent variables. Therefore, degrees of freedom were corrected using Greenhouse-Geiser estimates of sphericity. Due to their skewness, salivary cortisol data and heart rate data were log-transformed.

All indicators of the stress response per time-point are depicted in Fig. 2. As presented in Table 2, results of the GLM analyses for repeated measures revealed a significant stress response and a significant recovery for all stress indicators: The Bonferroni adjusted pairwise comparisons showed significant increases in heart rate, negative affect, state depression and paranoia from t1 (pre-TSST) to t2 (post-TSST), followed by a significant decrease in these indicators from t2 (post-TSST) to t3 (post-TSST after a 10 min recovery). Salivary cortisol showed a linear change with a significant increase from t1 to t2 and from t2 to t3.

#### 3.4. Emotion regulation as a predictor of the stress response and recovery

##### 3.4.1. Adaptive emotion regulation

As presented in Table 3, the results of the GLM revealed no significant main effects of adaptive emotion regulation on any of the stress indicators at any of the three time-points. Furthermore, there was no interaction effect of adaptive emotion regulation  $\times$  time-point on any of the stress indicators. All results remained stable after controlling for the confounding effect of gender, age, BMI, and time of the assessment.

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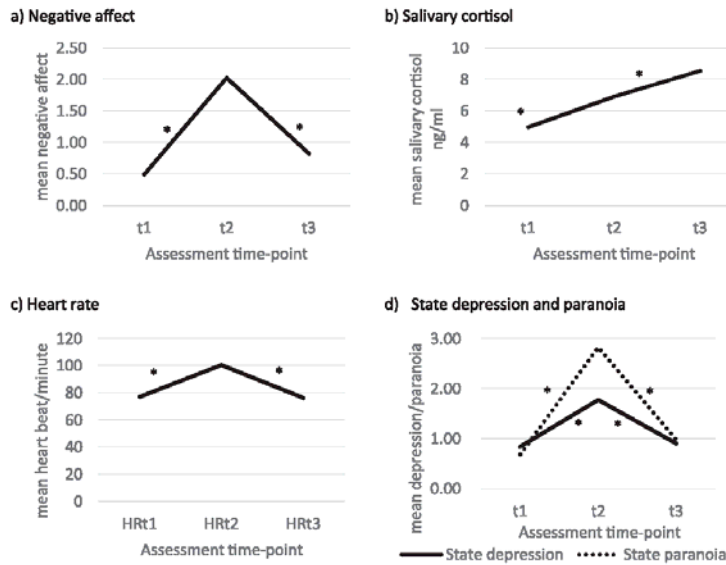


Fig. 2. Stress response to and recovery from the Trier Social Stress Test (TSST). t1 = time-point before the TSST; t2 = time-point immediately after the TSST; t3 = time-point after 10 min recovery phase; HRT1 = time-point of the mean heart rate before the TSST; HRT2 = time-point of the mean heart rate during the 5 min speech phase; HRT3 = time-point of the mean heart rate during the first 5 min of the recovery phase; \* $p < .005$ .

### 3.4.2. Maladaptive emotion regulation

Maladaptive emotion regulation was significantly associated with the mean scores of negative affect, depression and paranoia symptoms at all consecutive time-points but not with the endocrine and autonomic indicators of stress. As depicted in Table 3, there was a significant interaction effect of maladaptive emotion regulation  $\times$  time-point on negative affect and a trend effect on salivary cortisol. The analyses of within subject contrasts showed that maladaptive emotion regulation was a significant predictor of the increase in negative affect from t1 to t2 (stress response:  $F(1, 62) = 5.78, p < .05, \eta^2_{\text{partial}} = 0.085$ ) but not of the decrease from t2 to t3 (stress recovery:  $F(1, 62) = 0.30, p = .59, \eta^2_{\text{partial}} = 0.005$ , see Fig. 3). The same pattern was found for salivary cortisol, where maladaptive emotion regulation was a significant predictor of the change from t1 to t2 ( $F(1, 56) = 4.62, p < .05, \eta^2_{\text{partial}} = 0.076$ ) but not of the change from t2 to t3 ( $F(1, 56) = 0.39, p = .54, \eta^2_{\text{partial}} = 0.007$ , see Fig. 3). The interaction effect of maladaptive ER  $\times$  time-point on heart rate, depression and paranoia symptoms was not significant (Table 3).

All results remained stable after controlling for the confounding effect of gender, age, BMI, and time of the assessment.

### 3.5. Exploratory analyses of different emotion regulation strategies as predictors of the stress response and recovery

The interaction between acceptance  $\times$  time-point ( $p = .013$ ) and planning  $\times$  time-point ( $p = .046$ ) significantly predicted negative affect. Negative affect was significantly predicted by the interaction between catastrophizing  $\times$  time-point ( $p = .001$ ) and by blaming others  $\times$  time-point ( $p = .005$ ). Heart-rate was significantly predicted by the interaction rumination  $\times$  time-point ( $p = .035$ ). Detailed results of the exploratory analyses can be found in the Appendix B in the supplementary material.

## 4. Discussion

In the present study, we investigated whether habitual emotion regulation determines the response to and recovery from an acute social-evaluative stressor. As expected, we found that the stress induction with TSST resulted in significant increases in all of the tested indicators of stress. Following the recovery phase, we found a significant decrease of all tested indicators except for salivary cortisol, which showed a linear mean increase across the three consecutive time-points. Thus, we found that healthy individuals react to acute stressors not only subjectively (i.e., feeling stressed) and physiologically (i.e., increased cortisol, higher heart rate) but also “symptomatically”, by an increase in

Table 2

General linear model analyses for repeated measures of different stress response and recovery indicators in the Trier Social Stress Test.

	$F$ (df, error df)	$p$	$\eta^2_{\text{partial}}$	$M_{2-1}$ (SD)	Post hoc $M_{2-1}$ (SD)	$M_{1-3}$ (SD)
Negative affect	34.33 (1.61, 102.76)	< .001	0.349	1.515 (0.229)***	1.238 (0.203)***	-0.277 (0.142)
Salivary cortisol	17.54 (1.46, 84.72)	< .001	0.232	0.126 (0.032)***	-0.079 (0.026)**	-0.205 (0.044)***
Heart rate	230.44 (1.39, 72.14)	< .001	0.816	0.117 (0.008)***	0.120 (0.007)***	0.003 (0.004)
State depression	30.41 (1.58, 101.20)	< .001	0.322	0.938 (0.160)***	0.858 (0.135)***	-0.090 (0.098)
State paranoia	103.54 (1.64, 105.02)	< .001	0.618	2.118 (0.185)***	1.892 (0.174)***	-0.225 (0.119)

Notes. \*\*\* $p < .001$ ; \*\* $p < .01$ ; \* $p < .05$ ; Post hoc = test for pairwise comparisons with Bonferroni correction;  $M_{2-1}$  = mean difference between the time-point immediately after stress induction and the time-point before stress induction;  $M_{2-3}$  = mean difference between the time-point immediately after stress induction and the time-point after a 10 min recovery;  $M_{1-3}$  = mean difference between the time-point before stress induction and the time-point after a 10 min recovery.

**Table 3**  
Main effect of adaptive and maladaptive emotion regulation on indicators of stress and interaction effect between emotion regulation and assessment time-point.

	<i>F</i> ( <i>df</i> , <i>error df</i> )	<i>p</i>	$\eta^2_{\text{partial}}$
<i>Negative Affect</i>			
Adaptive ER	1.94 (1, 62)	.168	0.030
Maladaptive ER	17.08 (1, 62)	< .001	<b>0.216</b>
Adaptive ER × time-point	0.41 (1.57, 97.55)	.613	0.007
Maladaptive ER × time-point	4.33 (1.57, 97.55)	.023	<b>0.065</b>
<i>Salivary cortisol</i>			
Adaptive ER	0.02 (1, 56)	.886	0.000
Maladaptive ER	0.04 (1, 56)	.841	0.001
Adaptive ER × time-point	0.45 (1.48, 83.10)	.581	0.008
Maladaptive ER × time-point	3.33 (1.48, 83.10)	.055	<b>0.056</b>
<i>Heart rate</i>			
Adaptive ER	0.04 (1, 50)	.836	0.001
Maladaptive ER	0.02 (1, 50)	.883	0.001
Adaptive ER × time-point	0.35 (1.37, 68.59)	.624	0.007
Maladaptive ER × time-point	0.23 (1.37, 68.59)	.712	0.005
<i>State depression</i>			
Adaptive ER	2.13 (1, 62)	.149	0.033
Maladaptive ER	24.33 (1, 62)	< .001	<b>0.282</b>
Adaptive ER × time-point	0.06 (1.58, 97.92)	.909	0.001
Maladaptive ER × time-point	0.73 (1.58, 97.92)	.456	0.012
<i>State paranoia</i>			
Adaptive ER	0.57 (1, 62)	.453	0.009
Maladaptive ER	8.15 (1, 62)	.006	<b>0.116</b>
Adaptive ER × time-point	0.27 (1.62, 100.67)	.716	0.004
Maladaptive ER × time-point	0.96 (1.62, 100.67)	.369	0.015

Notes. ER = Emotion regulation. **Bold** = significant effects ( $p < .05$ ).

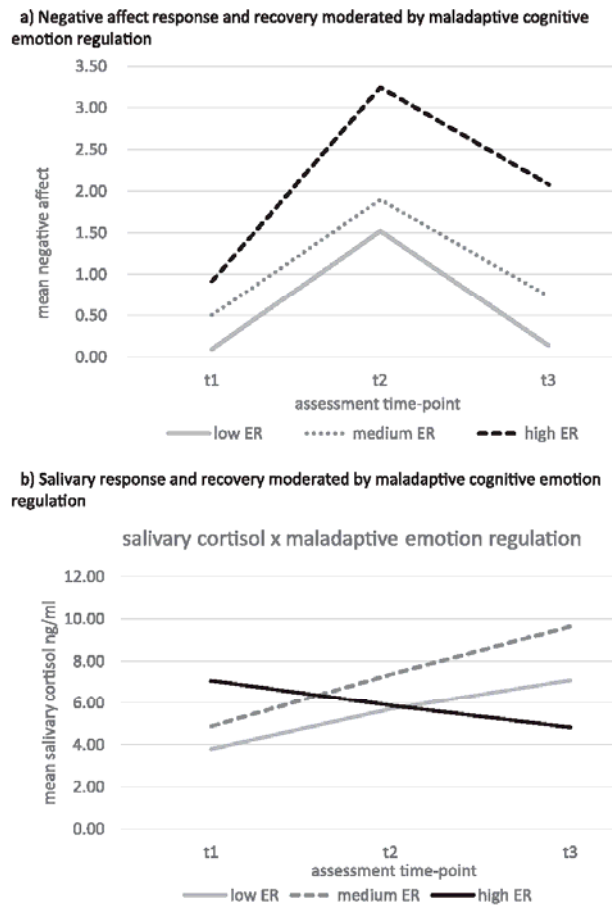
state depression and state paranoia.

In line with our hypothesis, we found habitual maladaptive ER to be predictive of the stress response. However, this was only the case for some of the stress indicators. In particular, we were able to corroborate previous findings that habitual maladaptive ER was related to the subjective stress response. However, there was only a non-significant trend for the association between habitual maladaptive ER and the endocrine stress response. Interestingly, the post-hoc results showed the group of participants with particularly high scores in habitual maladaptive ER to peak before the TSST and to show a linear decrease of the salivary cortisol response over the consecutive time-points and at the same time to report the highest subjective levels of stress after the TSST compared to other groups. In contrast, the participants who had medium and low habitual maladaptive ER scores showed the expected endocrine response with salivary cortisol linearly increasing and a somewhat lower subjective stress response and subjective recovery compared to the high maladaptive ER group. These results are in line with a small laboratory study by Villada et al. (2016), who investigated reactions to the TSST in 35 healthy participants and found two clusters of responders – one with a low subjective stress response and high endocrine response who were characterized by low trait anxiety and high scores on active coping and another cluster with high subjective and low endocrine responses who were characterized by high trait anxiety and low scores on active coping. One explanation for such a reversed trend in the group with high maladaptive ER in our study could be that the cortisol level prior to the TSST reflected anticipatory stress followed by recovery during the manipulation in this group. However, the high self-reported stress response seems to speak against this interpretation. An alternative explanation for the discrepancy of the endocrine and subjective stress response could be that the endocrine response was dampened due to a specific HPA axis dysregulation resulting in endocrine hypo-sensitivity, but replication is required to substantiate this interpretation. Future studies should thus investigate this discrepancy between subjective and endocrine response and include a longer sampling period prior to and after the stress

manipulation. Moreover, longitudinal research is needed to tease out the temporal precedence of hypo-sensitivity and difficulties in ER in order to understand whether maladaptive ER predicts the HPA dysregulation or vice versa or whether they are simply two sides of the same coin. This type of research could also take into account the known association of early adversity with both HPA dysregulation (e.g., Carpenter et al., 2007; Heim Newport et al., 2000; Strüber et al., 2014) and habitual ER (e.g., Carvalho-Fernando et al., 2014; Lincoln et al., 2017a).

As expected, higher maladaptive ER at baseline predicted a stronger increase in negative affect as a response to the stressor, which further corroborates studies finding maladaptive ER to be associated with dysfunctional outcomes such as stronger negative affect (Campbell-Sills et al., 2006; Krcovic et al., 2018) and mental disorders (e.g., Nolen-Hoeksema et al., 2007, 2008; Westermann et al., 2013). However, contrary to findings of these studies and our expectations, increases in state depression and paranoia in response to the stressor were not predicted by habitual maladaptive ER. Possibly, it requires other or additional vulnerability factors such as cognitive biases (Everaert et al., 2017) or negative schemata (Beck et al., 2017) for the affective and physiological stress-response to translate into symptoms. Furthermore, other explanations for the association between habitual maladaptive ER and stress reactivity found in this study should be discussed. In particular, due to the social-evaluative nature of the TSST, it is possible that individuals with high trait social anxiety react to the stressor with higher negative affect in response to a stressor, as has been found in previous studies (e.g., Krämer et al., 2012; von Dawans et al., 2017). If this were the case, maladaptive ER may have only acted as a proxy for social anxiety that was not tested in this study. However, although ER plays a role in social anxiety disorder (Jazaieri et al., 2015) the strength of this association is only moderate and ER has not been found to predict anxious responding to a social stressor (Helbig-Lang et al., 2015).

Contrary to our expectations, we did not find an association between habitual use of adaptive ER and any of the stress indicators. Thus, when it comes to the acute stress response, people who report to regularly employ adaptive ER strategies do not seem to fare better. This corroborates and extends previous findings showing that maladaptive ER could be a more relevant risk factor for psychopathology than the adaptive ER (Aldao et al., 2010; Aldao and Nolen-Hoeksema, 2012). A potential reason for this is that individuals may fail to apply adaptive strategies during an acute stressor, as found in a study conducted by Raio et al. (2013). This could be due to cognitive overload and the restriction of prefrontal functions that are found during a stress response (Schwabe and Wolf, 2009). In order to further pursue this question, future studies should include paradigms that allow the measurement of ER both habitually and in the presence of a stressor. Furthermore, it is possible that adaptive ER strategies are not protective per se, but only in the presence of maladaptive strategies. For instance, Aldao and Nolen-Hoeksema (2012) found that adaptive ER was negatively related to psychopathology only at high levels of maladaptive ER, indicating a compensatory role of adaptive ER. Another reason for the lack of an association between the habitual use of adaptive ER and the acute stress response could be that an acute stress response is not necessarily a dysfunctional process but is also an adaptive attempt of the organism to respond to or prepare for potential threat. Nevertheless, one would expect the use of adaptive ER to help people to recover more rapidly from the acute stress response, which was not supported by our data. Thus, either the habitual adaptive ER does not get employed in the recovery phase either, or it does not help an individual to recover more quickly. This does not exclude the possibility that other, possibly more behavioral coping strategies are employed, such as focusing on breathing techniques, diverging attention or using muscle relaxation. This was evident in the stress induction where we observed participants closing their eyes, clenching their fists, or focusing on one point in the room while performing the TSST speech or arithmetic task.



**Fig. 3.** Stress response and recovery moderated by maladaptive emotion regulation. t1 = time-point before Trier Social Stress Test (TSST); t2 = time-point immediately after the TSST; t3 = time-point after 10 min recovery phase; low ER = maladaptive emotion regulation score on CERQ < median - 1 SD; medium ER = maladaptive emotion regulation score on CERQ = median  $\pm$  1 SD; high ER = maladaptive emotion regulation score on CERQ > median + 1 SD.

It should be noted that the present study focused on the self-reported habitual use of ER strategies. What remains unanswered is whether or not individuals actually applied these strategies when they were confronted with the stressor in the laboratory, whether they applied them successfully, and whether or not the participants perceived these strategies as effective, which are all important aspects of emotion regulation (Gutentag et al., 2017). Nevertheless, our results signal that the habitual tendency to use maladaptive strategies is associated with stronger subjective responses in an acute stress situation.

Furthermore, the explorative analyses suggest that both adaptive and maladaptive strategies (i.e. planning, acceptance, catastrophizing, and blaming others) could be of importance for the stress reactivity. Nevertheless, due to the low power of this analysis and alpha error accumulation we will refrain from further interpretation and encourage further research to analyze the function of the individual strategies more thoroughly.

The results of the present study need to be considered in the light of several limitations. First, due to funding constraints, our design did not include a sufficient number of assessment time-points to track

endocrine stress recovery. With only two post-TSST assessment time-points within 10 min, this study only allows to draw conclusions in regard to the endocrine stress increase, but not in regard to the return to pre-TSST level. We would thus recommend that future studies include a larger number of salivary cortisol samples over a longer recovery phase (for an overview of usual sampling plans, Kirschbaum and Hellhammer, 1994). Second, endocrine and autonomic activation are sensitive to many confounding variables (e.g., contraceptives, coffee, and smoking). Although we accounted for these variables by considering them as covariates in our analyses, the assessment relied (as in most other studies) on self-report of the participants that could have been biased. Furthermore, we considered the assessment time as a covariate to account for possible circadian effects, however, restricting the assessment time would have been more appropriate. Finally, since the WCST may be perceived as stressful, the pre-TSST stress assessment was performed before the WCST was administered. Therefore it is possible that the observed stress response was not only due to the TSST but also partly a result of a carry-over effect of the cognitive assessment.

To conclude, the present study suggests that people who tend to use

maladaptive ER strategies show a stronger affective and a reduced endocrine stress response. This indicates that maladaptive ER could represent a risk factor for depression and psychosis by altering the stress response. However, future studies are needed to further investigate the nature of the relation between maladaptive ER and endocrine stress response and to explore which exact aspects of maladaptive ER determine the hyposensitivity of the HPA axis and the elevated negative affect in the face of stress. Interventions targeting maladaptive ER strategies may be promising to help individuals gain control over their affective and physical reactions to stress and thus become more resilient to stressors that are an inevitable part of their everyday life.

#### Conflict of interest

The authors state that no potential conflict of interest are associated to this manuscript.

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#### Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.psychneuen.2018.04.028>.

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## Appendix B: Study II

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## Original article

## Emotion regulation as a moderator of the interplay between self-reported and physiological stress and paranoia



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## ABSTRACT

Experience sampling method (ESM) studies have found an association between daily stress and paranoid symptoms, but it is uncertain whether these findings generalize to physiological indicators of stress. Moreover, the temporality of the association and its moderating factors require further research. Here, we investigate whether physiological and self-rated daily stress predict subsequent paranoid symptoms and analyze the role of emotion regulation as a putative moderator. We applied ESM during 24 h to repeatedly assess heart rate, self-rated stress, and subclinical paranoia in a sample of 67 psychosis-prone individuals as measured with Community Assessment for Psychotic Experiences (CAPE). Adaptive and maladaptive emotion regulation was assessed at baseline with the Emotion Regulation Skills Questionnaire (ERSQ-ES) and the Cognitive Emotion Regulation Questionnaire (CERQ). Linear mixed models were used to analyze the data. Heart rate ( $b = 0.004, p < 0.05$ ) and self-rated stress ( $b = 0.238, p < 0.001$ ) predicted subsequent paranoia. The reverse effect, paranoia as a predictor of subsequent heart rate ( $b = 0.230, p = 0.615$ ) or self-rated stress ( $b = -0.009, p = 0.751$ ) was non-significant. Maladaptive emotion regulation was a significant predictor of paranoia ( $b = 0.740, p < 0.01$ ) and moderated the path from self-rated stress to paranoia ( $b = -0.188, p < 0.05$ ) but not the path from heart rate to paranoia ( $b = 0.005, p = 0.09$ ). Our findings suggest a one-way temporal link between daily stress and paranoia and highlight the importance of emotion regulation as a vulnerability factor relevant to this process.

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## 1. Introduction

Studies applying an experience sampling method (ESM) have found that individuals with psychosis and those at risk show elevated self-reported negative affect in response to daily hassles, which is commonly referred to as “stress-sensitivity” or “stress-reactivity” [1]. Moreover, ESM studies find self-reported daily stress to be associated with psychotic symptoms in clinical and non-clinical samples [2–6], and in the most recent study, Van Der Steen et al. [7] found that this association is even larger in high risk groups than in patients. Daily stress thus appears to be especially relevant to the development of psychotic symptoms even before the unfolding of the full disorder. However, several issues require further clarification.

One of these issues is related to the operationalization of stress. Researchers using experimental designs to investigate stress-reactivity have widely acknowledged the importance to assess both physiological and psychological parameters of stress [8] in

order to gain a fuller understanding of the processes involved. In contrast, most ESM studies are limited to self-reported stress. In one of few ESM studies in the field of psychosis that investigated the activation of the autonomous nervous system (ANS) as a physiological marker of stress, Kimhy and colleagues [9] found no evidence for an association between self-reported stress and heart rate. They did, however, find self-reported stress to be associated with other ANS parameters, such as sympathovagal balance. Another ESM study found acute psychosis patients to have an increased heart rate and an altered autonomic variation in comparison to healthy controls [10]. These studies show alterations in physiological stress in the context of daily life in patients with psychosis. They also indicate that physiological stress parameters capture different aspects of stress than self-report. To further corroborate this notion and better understand the link between stress and symptom formation, research on the association between physiological stress, self-reported stress, and psychotic symptoms within a daily life context is required.

Another issue related to the association between daily stress and psychotic symptoms refers to its temporality. So far, there is only limited evidence for the assumption that stress precedes (rather than follows from) psychotic symptoms [11,12]. Moreover,

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few ESM studies have aimed to investigate the reversed pathway in which psychotic symptoms trigger stress [7,13,14]. This is surprising as – considering the literature on the distress related to psychotic symptoms [13,15] – a vicious circle, in which stress and symptoms are driving each other can be expected.

Finally, given that we can corroborate the assumption that stress predicts an exacerbation of psychotic symptoms in the earliest stage of disorder formation, the question arises whether we can identify specific factors that make people vulnerable to developing psychotic symptoms in the face of stress. Recent research suggests that deficits in emotion regulation (ER) could constitute a crucial vulnerability factor [16,17]. Compared to healthy controls, individuals with psychosis use more maladaptive and less adaptive ER [18–22]. Furthermore, ER seems to be related to physiological and self-reported stress [23,24]. In a laboratory experimental study, Lincoln et al. [24] found adaptive ER to moderate the association between induced stress and paranoid symptoms, indicating that ER could be a protective factor that prevents stress from translating into paranoid symptoms. However, this assumption needs further corroboration by testing to which extent the habitual use of adaptive or maladaptive ER skills makes people vulnerable to responding to stress with psychotic symptoms in daily life.

Building upon the solid evidence showing that self-reported stress is associated with paranoid symptoms in daily life, the goal of this study was to investigate the temporality of the association between self-reported stress, physiological stress and paranoid experiences, and to identify the moderating value of ER. We expected that self-reported and physiological stress would predict the subsequent report of paranoia over the course of a day. Furthermore, we expected that paranoia would in turn predict the intensity of subsequent self-reported and physiological stress. Finally, we hypothesized that the habitual use of adaptive and maladaptive ER would moderate the path from daily stress to paranoia. Building on the continuum of psychotic symptoms and their associated risk factors [25], we used a community sample with elevated levels of psychotic-like experiences in order to gain insight into the development of subclinical symptoms prior to the full unfolding of the disorder.

## 2. Methods and materials

### 2.1. Participants

Participants from previous studies who had consented to be contacted for future projects and first semester psychology students were prescreened for the occurrence of psychotic-like experiences as measured by the Community Assessment for Psychotic Experiences (CAPE) [26]. Potential participants were invited to participate starting from those with highest value and ending with score of eight on the positive syndrome subscale of CAPE, which corresponds to the score of 50th percentile of the large community sample published in Schlier et al. [27]. The acquired sample consisted of 67 individuals (71.6% female,  $M_{age} = 23.01$ ,  $SD_{age} = 4.63$ ). Nine participants reported to have been given a diagnosis of a mental disorder in the past, and 18 participants reported a mental disorder of a family member. The majority of participants (80.6%) were students; 47.8% reported to be working six or more hours per week. Most participants (83.6%) were German nationals. All participants provided written informed consent and were compensated with 10€ per hour or granted credit points.

### 2.2. Procedure

Baseline assessment took place at Universität Hamburg in Germany. First, an electrocardiogram (ECG) sensor was attached to

the participants' chest and activated. The participants then completed paper-pencil questionnaires. Thereafter, they received Android smartphones that allowed the use of the movisensXS ESM application (Movisens GmbH) only. After activating the application, the participants left the laboratory and the ESM assessment phase began. As can be seen in Fig. 1a, the ECG recorded arousal continuously over 24 h. The smartphones were programmed to beep in approximately 20-min-intervals (between 9 AM and 10 PM) resulting in 38 samples over 24 h. The starting and ending time-point of the ESM assessment phase varied across participants, but no participant was subject to any assessments between 10 PM and 9 AM. Participants were instructed to behave as usual, with the restriction that they were not allowed to take a shower or exercise excessively. Fig. 1b illustrates the time references between any two ESM questionnaires. As can be seen, after the beep at any given time-point ( $t$ ), participants answered questions regarding their stress level since the previous beep (thus for the time-period between  $t-1$  and  $t$ ). Furthermore, participants answered questions on momentary paranoid symptoms referring to the time-point  $t$ .

### 2.3. Assessment

#### 2.3.1. Psychosis proneness assessment

Psychosis proneness was assessed with the CAPE [26] that captures lifetime psychotic-like experiences. The CAPE is a self-report questionnaire composed of the depressive, negative, and positive syndrome subscales. The scale is constructed with 42 items to be self-rated on a four-point Likert scales ranging from 0 = "never" to 3 = "nearly always". The CAPE has been found to be a valid and reliable measure of psychosis proneness [28] and to be sensitive in detecting individuals at ultra-high risk for psychosis [29]. The German version of the CAPE has good to excellent internal consistency [27].

#### 2.3.2. Emotion regulation assessment

**2.3.2.1. Adaptive ER.** In order to capture the comprehensive spectrum of strategies, adaptive ER was measured with a composite score derived from two questionnaires – German version of the emotion specific Emotion Regulation Skills Questionnaire (ERSQ-ES) [30] and the adaptive subscale of the German version of the Cognitive Emotion Regulation Questionnaire (CERQ) [31]. The ERSQ-ES assesses the following adaptive strategies: clarity, understanding, acceptance, tolerance, self-support, willingness to confront situations cuing undesired emotions when necessary to attain personally relevant goals, and modification. The ERSQ-ES measures the use of strategies differentially for stress, anxiety, anger, sadness and shame. The total score used for this study was the mean score of all items relative to the number of emotions. The psychometric properties of the ERSQ-ES were good to excellent in large clinical and non-clinical samples [30]. The following additional adaptive strategies were assessed with the CERQ: acceptance, positive refocusing, refocusing on planning, positive reappraisal and putting into perspective. The German version of the CERQ has acceptable to good psychometric properties [31]. The internal consistency of the composite adaptive ER scale consisting of the ERSQ-ES total mean score and CERQ adaptive ER subscale mean score was excellent on our sample, with Cronbach's  $\alpha = 0.92$ .

**2.3.2.2. Maladaptive ER.** Maladaptive ER was assessed with the mean score on the maladaptive ER subscale of CERQ, which includes the strategies self-blame, catastrophizing, rumination/focus on thought, and blaming others. The maladaptive strategies subscale in our sample showed an acceptable internal consistency with Cronbach's Alpha = 0.703.

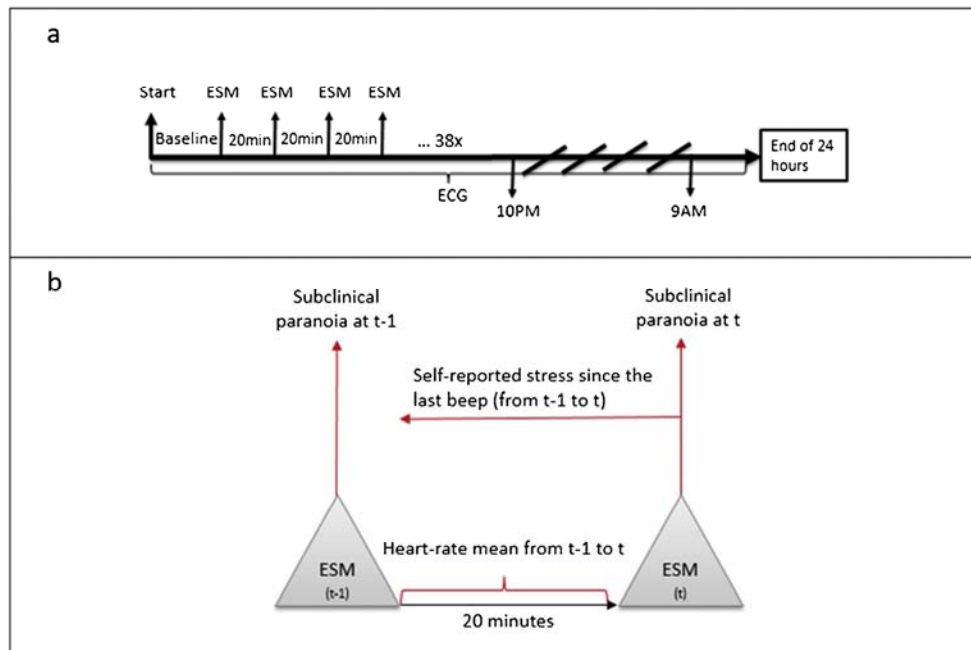


Fig. 1. Study procedure (1a) and time reference between any two experience sampling time-points (1b). ESM, Experience Sampling Method; t, time-point; t-1, time-point preceding.

### 2.3.3. Assessment of momentary physiological stress

Physiological arousal was measured as heart rate per minute using a sensor (Movisens ECGmove) designed for collecting ambulatory ECG. ECGmove is a small  $62.3 \times 38.6 \times 11.5$  mm sensor attached to electrodes that were placed on the left side of the chest. As presented in Fig. 1, heart rate was measured continuously and was subsequently analyzed as a mean heart rate between two beeps. The correction of artefacts that can emerge from physical activity during assessment or from the disturbances in electrode connection was performed automatically in DataAnalyzer (Movisens GmbH).

### 2.3.4. Assessment of momentary self-reported stress

The self-reported stress was assessed with 10 items referring to the previous 20 min (between t-1 and t, see Fig. 1b) rated on a 10-point scale. The scale included self-ratings of arousal, stress and control (subscale based on Gaab et al. [32]: "The situation was stressing me"; "I was able to control the situation"; "I was calm and relaxed"; "I was helpless in the situation") as well as self-ratings of fear, sadness, anger, shame, guilt, and (un-)happiness (subscale based on Stemmler et al. [33]). The pre-analysis showed that two subscales loaded on a common factor so that the score was computed as the mean of the 10 items at each time-point. Geldof, Preacher and Zyphur [34] propose to conduct the multilevel analysis of reliability when dealing with hierarchical data, since the single level Cronbach's  $\alpha$  yields untrustworthy parameters due to the confounding variance of the two measurement levels (between and within). For the momentary self-reported stress the within-subject-level internal consistency was acceptable to good with

Cronbach's  $\alpha = 0.82$  and good for the between-subject-level with Cronbach's  $\alpha = 0.88$ .

### 2.3.5. Assessment of momentary paranoia

Momentary subclinical paranoia was assessed with a three item version of the Paranoia Checklist containing items identified as sensitive to change in previous studies: "I need to be on my guard against others"; "Strangers and friends look at me critically"; and "People try to upset me" [35]. Participants were asked to which extent each of the statements applies to them at the moment of the beep (time-point t, see Fig. 1b) on a 10-point scale. The scores used were the mean scores of the three items at each time-point. The multilevel reliability was questionable to poor for the within-subject-level (Cronbach's  $\alpha = 0.62$ ), and excellent for the between-subject-level (Cronbach's  $\alpha = 0.92$ ).

### 2.4. Statistical analysis

Data was analyzed using IBM SPSS Statistics software (version 22) and R. First, correlations were computed between self-reported stress and heart rate. Second, in order to account for the hierarchical design of the study we applied multilevel analysis. As suggested in literature [36], independent variables that were repeated measures were centered around the subject-mean and baseline independent variables were grand-mean centered. The time-point was included as a control variable in all models. To test whether preceding stress (at time-point between t and t-1) predicted subsequent paranoia (at time-point t) we calculated linear mixed-effect models (Package 'lme4' for R) with random

intercept and random slope. The same analysis was done in order to test the reversed causation (preceding paranoia at time-point t-1 predicting subsequent stress at the time-point between t-1 and t). In the final model, we entered the adaptive and maladaptive ER as moderators. As proposed by Singer and Willet [37] we calculated pseudo- $R^2$  statistic as an indicator of the effect size and classified these as small, medium or large [38]. The pseudo- $R^2$  statistic expresses the proportion of variance explained between and within persons in comparison to the null model.

### 3. Results

#### 3.1. Descriptive statistics

The sum scores and standard deviations of the psychotic experiences measured with the CAPE, ER mean scores at baseline, as well as the ESM scores of self-reported stress, heart rate and paranoia symptoms are presented in Table 1. Regarding psychosis-proneness measured by CAPE, the mean sum-score on CAPE positive syndrome subscale in our sample with  $M = 16.16$  was higher than the mean sum-score found in psychosis samples ( $M = 14.50$ ) in Schlier et al. [27]. Specifically, all participants indicated to experience no less than one symptom of each CAPE subscale at least "sometimes". All participants indicated to experience at least one positive symptom "often" and 32% of participants indicated to experience at least one positive symptom "nearly always". The compliance rate for the ESM assessment was 81% in average, ranging from 8 to 100%, which is comparable with compliance rates usually found in ESM studies [39]. Only four participants had a compliance rate for the ESM assessments of less than 50%. Excluding these from the analysis under assumption of "non-random missing" did not alter the results, so that all participants

were included in all subsequent analyses. Four percent of heart rate data points were missing due to technical issues.

#### 3.2. The association between self-reported stress and heart rate

The two indicators of daily stress, self-reported stress and heart rate were significantly correlated across subjects and time-points, with  $r = 0.202$  ( $p < 0.05$ ). However, within-subject correlation as calculated with "psych" package in R, function "statsBy", was non-significant with  $r = -0.020$  ( $p = 0.390$ ). The same was found for the between-subject correlation with  $r = 0.180$  ( $p < 0.140$ ).

#### 3.3. Preceding stress indicators as predictors of subsequent paranoid symptoms

For all ESM measures, one part of their variance was explained by the variation at the within-subject level (fluctuations within one person over the time) and another part at a between-subject level (differences between individuals). This was expressed by intra-class-correlation coefficient ICC ( $\rho = 0.523$  indicating that 52.3% of the variance in paranoia was explained by fluctuations within individuals;  $\rho = 0.419$  for self-reported stress;  $\rho = 0.149$  for heart rate). After controlling for preceding paranoia at t-1 and for the time-point, both preceding self-reported stress and heart rate between t-1 and t were significant predictors of subsequent paranoia at t (see Table 2). In case of self-reported stress, the explained proportion of variance was  $R^2_{\text{within}} = 0.20$ , which means that 20% of the within person variance in paranoia was explained by self-reported stress, which corresponds to a medium to large effect. For heart-rate the explained proportion of variance was  $R^2_{\text{within}} = 0.04$  corresponding to a small effect.

**Table 1**  
Mean, standard deviation and range of baseline and experience sampling method variables.

	N	M	SD	Range
Baseline variables				
CAPE positive	67	16.16	4.46	8.00–30.00
CAPE negative	67	15.76	6.30	5.00–34.00
CAPE depressive	67	9.85	3.73	3.00–20.00
Adaptive ER (CERQ+ERSQ-ES)	66	3.26	0.86	1.28–6.02
Maladaptive ER (CERQ)	67	1.59	0.50	0.58–3.42
Experience sampling method variables				
Heart rate	2432	88.96	19.80	43.99–188.61
Self-reported stress	2042	2.25	1.34	0.00–10.00
Paranoia	2039	0.74	1.24	0.00–7.00

CAPE = Community Assessment of Psychotic Experiences; Adaptive ER = adaptive emotion regulation as composite score of adaptive emotion regulation subscale of Cognitive Emotion Regulation Questionnaire (CERQ) and Emotion Regulation Skills Questionnaire (ERSQ-ES); Maladaptive ER = maladaptive emotion regulation subscale of Cognitive Emotion Regulation Questionnaire.

**Table 2**  
Self-reported stress and heart rate as predictors of paranoia when controlled for time-point and paranoia on previous time-point, moderated by emotion regulation.

	b	SE	p	CI (95%)
Time-point	-0.004	0.003	0.207	-0.009–0.002
Paranoia t-1	0.209	0.035	<0.001	0.139–0.279
Self-reported stress between t-1 and t	0.238	0.037	<0.001	0.165–0.312
Heart rate between t-1 and t	0.004	0.002	0.033	0.000–0.006
Adaptive ER	-0.242	0.130	0.068	-0.502–0.018
Maladaptive ER	0.740	0.225	0.002	0.291–1.189
Self-reported Stress × Adaptive ER	-0.059	0.040	0.145	-0.140–0.021
Self-reported Stress × Maladaptive ER	0.188	0.071	0.011	0.045–0.331
Heart rate × Adaptive ER	<0.001	0.002	0.937	-0.004–0.004
Heart rate × Maladaptive ER	0.006	0.003	0.090	-0.001–0.012

ER = emotion regulation.

### 3.4. Reversed model: preceding paranoia as a predictor of subsequent stress

After controlling for preceding self-reported stress ( $b = 0.389$ ,  $SE = 0.032$ ,  $p < 0.001$ , 95% CI [0.323, 0.456]) and time-point ( $b = 0.0007$ ,  $SE = 0.003$ ,  $p = 0.980$ , 95% CI [-0.006, 0.006]), preceding paranoia at  $t-1$  was not a significant predictor of subsequent self-reported stress between  $t-1$  and  $t$  ( $b = -0.009$ ,  $SE = 0.029$ ,  $p = 0.779$ , 95% CI [-0.69, 0.052]).

Similarly, in a separate model, when controlled for the preceding heart rate ( $b = 0.532$ ,  $SE = 0.031$ ,  $p < 0.001$ , 95% CI [0.469, 0.595]) and time-point ( $b = 0.121$ ,  $SE = 0.033$ ,  $p < 0.001$ , 95% CI [0.055, 0.187]), preceding paranoia at  $t-1$  was not a significant predictor of subsequent heart rate between  $t-1$  and  $t$  ( $b = -0.231$ ,  $SE = 0.453$ ,  $p = 0.615$ , 95% CI [-0.704, 1.166]).

### 3.5. ER as moderator of the path from stress to paranoia

Maladaptive and adaptive ER were not significantly correlated ( $r = -0.066$ ,  $p = 0.592$ ). As can be seen in Table 2, higher values on the CERQ maladaptive subscale were associated with higher momentary paranoia. The score in adaptive ER was not related to momentary paranoia.

Maladaptive ER significantly moderated the path from preceding self-reported stress between  $t-1$  and  $t$  to subsequent momentary paranoia at  $t$  (Table 2). Specifically, the positive unstandardized estimate (see Table 2) indicated that the more maladaptive emotion regulation strategies an individual used, the more preceding self-reported stress predicted subsequent paranoia. The effect size was medium with  $R^2_{between} = 0.16$ , meaning that 16% of the variance in slopes for preceding self-reported stress as a predictor of paranoia was explained by maladaptive ER. As depicted in Table 2, maladaptive ER was not a significant moderator of the path from preceding heart rate to subsequent paranoia. Adaptive ER was neither a significant moderator for the path from self-reported stress to paranoia nor for the path from heart rate to paranoia.

## 4. Discussion

This study investigated temporal effects of self-reported as well as physiological stress on subclinical paranoia in the daily life of individuals with the elevated psychosis-proneness. Furthermore, we investigated the relevance of emotion regulation for the emergence of paranoid symptoms in the face of everyday stress.

Consistent with our first hypothesis, both preceding self-reported stress and heart rate were significantly associated with subsequent paranoia in daily life. This corroborates previous studies that found self-reported stress to predict paranoia [6,11,12] and extends them by showing that physiological stress is also relevant to understanding the emergence or exacerbation of paranoia. Although we had expected to find a vicious circle, in which paranoid symptoms would not only be predicted by previous stress but also be predictive of subsequent stress, this is not what we found. Rather, our results indicate that on the "micro" level of one day, the association between both self-reported and physiological stress and paranoia appears to be a "one-way street", where daily stress impacts the severity of paranoid symptoms, but not the other way around. Nevertheless, it remains possible and plausible that paranoid symptoms will lead to elevated levels of distress on the "macro" level over a period of days or even months. Only few longitudinal studies on the association of affect and symptoms in individuals with psychosis have tested for reverse pathways. In line with our findings, the few longitudinal studies that have investigated reverse pathways also do not support the notion that positive symptoms are followed by

increased anxiety [40], or depressed mood [40,41]. It needs noting that Van der Steen et al. [7] report a reversed path in a clinical high risk sample, but their analysis was based on correlations and thus – strictly speaking – only supports co-occurrence between symptoms and affect rather than temporal prediction.

Maladaptive ER was predictive of paranoia and also moderated the link between self-reported stress and paranoia. In particular, participants who reported more maladaptive strategies tended to report more paranoia after experiencing stress. This implies that maladaptive ER could be a risk factor for exacerbation of paranoid symptoms under stress. In contrast to our expectation and to a previous finding from our group showing adaptive ER to moderate the increase of paranoia following a stressor [24] – adaptive ER was not a significant moderator. The stronger relevance of maladaptive compared to adaptive ER are in line with findings of Aldao et al. [16] who found maladaptive strategies to be more strongly related to general psychopathology than adaptive strategies, as well as with findings of Westermann et al. [42] who reported only maladaptive ER to prospectively predict subclinical paranoia. Due to differences between studies in measures and in the methodological approach (experimental versus ESM), it might be premature to conclude that adaptive ER is not relevant to paranoia.

Surprisingly, the path from the physiological stress to paranoia was not moderated by ER. We can only speculate on the reasons for this; one possibility being that this pathway is influenced by different types of ER strategies (i.e. behavioral rather than cognitive strategies, such as changing the pace of breathing, or muscle relaxation) that were not assessed here.

The findings must be interpreted in light of several limitations. First, the sample primarily consisted of students limiting the generalizability of the findings to the general population where lower education and socio-economic status could be expected. Moreover, recruiting from the participant-database could have increased the risk of a selection-bias. For instance, it could be that the participants who already took part in psychological studies have a higher interest in psychological processes in general, or a higher introspection ability due to previous experiences in psychological testing. Another limitation is that due to technical features, the ambulatory physiological measurement was limited to a time period of one day. Furthermore, the intensive assessment with 38 assessment points in the course of only one day could have triggered reactivity in participants. Such a reactivity effect could become evident in a time effect (e.g. if participants pay more attention to symptoms and affective states over time resulting in higher ratings at later time-points or pay more attention in the beginning but get accustomed over time, resulting in lower ratings at later time-points). However, time-point of the assessment was included as a control variable in our analyses and showed no such effect on stress or paranoia. Also, an assessment over a longer period of time would have been beneficial to differentiate between rapid and slow changes in stress and symptoms as proposed in Jahng et al. [43] as well as to measure daily life more reliably by covering both workdays and weekends. Future studies should include a direct comparison of subclinical and clinical samples to test whether the moderating mechanism of maladaptive ER found in this study applies across the continuum of psychosis.

Despite these limitations, the findings corroborate previous work showing stress to precede paranoid symptoms in daily life and shed further light on the directionality of association between stress and paranoia. A central contribution of this study is in showing that this association is not limited to the self-reports as shown in previous work but also holds true for physiological indicators of stress, i.e. heart rate. This finding thus further corroborates the relevance of autonomic stress responses for the formation and exacerbation of psychotic symptoms long-since

postulated by vulnerability-stress-models [44] but seldom put to rigorous empirical tests. As a clinical implication we suggest that bio-feedback methods focusing on heart-rate monitoring [45,46] could potentially be used to regulate physiological stress and in turn prevent the emergence of psychotic experiences for those at risk. However, further research on such bio-feedback methods is necessary before implementing them in clinical practice.

Finally, our study highlights the importance of maladaptive ER as a significant risk factor related to the question why stress in daily life translates into psychotic symptoms. Considering that we found this pattern in a subclinical sample with only elevated psychosis-proneness, we suggest that maladaptive emotion regulation should be addressed in the earliest stage possible.

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#### Conflicts of interest

None.

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### Appendix C: Study III

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## An experience sampling study on the nature of the interaction between traumatic experiences, negative affect in everyday life, and threat beliefs☆

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## ABSTRACT

Research suggests that trauma is associated with the development of psychotic experiences, such as paranoia, via affective processes. However, the empirical evidence on the exact mechanism is limited and it is unclear which aspects of trauma are relevant. Here we tested whether self-reported frequency of trauma, recurring trauma, age, and type of trauma are predictive of later threat beliefs in daily life and which role affective processes (self-reported negative affect and autonomic arousal) play in this association. We tested two often postulated mechanisms: mediation, with affective processes in everyday life explaining the association between trauma and threat beliefs; and moderation, with trauma strengthening the association between affective processes and threat beliefs in everyday life. Trauma was assessed at baseline with the Trauma-History-Questionnaire in 67 individuals with attenuated symptoms of psychosis. We then applied the experience-sampling-method during 24 h to assess negative affect, heart rate and threat beliefs. Multilevel analysis showed that negative affect ( $p < 0.001$ ) and heart rate ( $p < 0.05$ ) were predictive of subsequent threat beliefs. There was no significant mediation effect from any trauma characteristic to threat beliefs via negative affect and heart rate. Trauma frequency ( $p < 0.001$ ), age at first trauma ( $p < 0.001$ ), as well as the presence of physical trauma ( $p < 0.001$ ) moderated the path from negative affect to subsequent threat beliefs. Our findings indicate that more frequent trauma, trauma at young age and physical trauma strengthen the association from negative affect to threat beliefs and could be relevant to determining the extent of vulnerability to psychosis.

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## 1. Introduction

Individuals who report traumatic experiences have been found to be 3 to 12 times more likely to develop psychotic symptoms or a psychotic disorder (Janssen et al., 2004; Spaauwen et al., 2006; Varese et al., 2012). Moreover, the association between psychotic experiences and trauma has been found across the psychosis continuum – from population samples (Arseneault et al., 2011; Lataster et al., 2006), over at-risk groups (Addington et al., 2013; Thompson et al., 2009), to individuals diagnosed with a psychotic disorder (Bailey et al., 2018; Shevlin et al., 2007). However, it remains unclear whether or not there are specific trauma characteristics that are especially important for this association and by which mechanisms trauma is associated with the formation of psychotic symptoms. Advancing our understanding of such mechanisms and narrowing down the relevant trauma characteristics is

relevant to both detection and prevention of psychosis and to improving the interventions offered to individuals with a history of trauma.

The findings on the relevance of different trauma aspects in relation to psychosis, such as frequency of trauma and recurring traumatic experiences, the age at which the trauma took place, and the type of trauma, are inconsistent: Among the few studies that reported results on frequency, some found a dose-response relation with more frequent trauma being associated with a higher risk for psychosis (Rubino et al., 2009; Shevlin et al., 2007) and more psychotic symptoms (De Loore et al., 2007), whereas others found no such association (Powers et al., 2016). The research on the relevance of the age at which trauma occurred is similarly equivocal. Although one study by Lataster et al. (2012) found that both early and recent trauma equally predicted an increased risk for psychosis over the following ten years, some more recent findings support the hypothesis that especially trauma at a young age predicts more severe psychopathology (Powers et al., 2016; Russo et al., 2014). Moreover, in a prospective study on an early psychosis sample, Alameda et al. (2016) have found patients who were traumatized before the age of 12 to develop higher levels of positive and negative symptoms of psychosis and affective symptoms over the course of three years, whereas those with a later trauma (from age 12 to 16)

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only developed more severe negative symptoms. Taken together, the existing research could indicate that being younger at the time of exposure will render it more likely to develop a range of symptoms including positive symptoms. Finally, it remains unclear whether different types of trauma are more or less relevant to psychosis. Specifically, whereas some studies found physical trauma to be the most robust predictor of psychotic disorders (Fisher et al., 2010; Rubino et al., 2009), others found only sexual trauma to be important (Thompson et al., 2014). Furthermore, Van Nierop et al. (2014) found interpersonal trauma with the intention to harm (comprising of physical, emotional, and sexual trauma) to be the crucial type of trauma in regard to psychosis. This was indirectly supported in a review by Gibson et al. (2016), who concluded the research on non-intentional trauma, such as injuries or natural disasters to be “limited and conflicting”. Thus, further research is needed to understand whether and to which extent specific characteristics of traumatic experiences are relevant to the risk of developing psychosis.

Another factor that makes the search for relevant trauma characteristics difficult is the existence of varying models that have been put forward to explain the mechanisms by which trauma translates into the psychosis. There is a strong evidence from longitudinal studies (Fowler et al., 2012; Jaya et al., 2017; Oliver et al., 2012), experimental studies (e.g., Freeman et al., 2015; Lincoln et al., 2010) and diary studies in everyday life (e.g., Ben-Zeev et al., 2011; Kramer et al., 2014; Thewissen et al., 2011) that negative affect, in form of depression, worry and anxiety, predicts the formation of psychotic symptoms and in particular the formation of paranoia. Building on these findings, a *mediation model*, in which the link between trauma and psychosis is mediated by negative affect, represents one plausible mechanism. The mechanism could be explained either by the finding that trauma induces increased and enduring negative affective states such as fear, anger, or sadness (Amstadter and Vernon, 2008) or because it impairs peoples’ ability to down-regulate negative affect (Gratz et al., 2007; Shields and Cicchetti, 1998), leading to more intense and enduring affective reactions to everyday stressors that could then lead on to psychotic symptoms. In line with this notion, Glaser et al. (2006) found people with a history of trauma to show increased affective reactivity and a recent study found impaired emotion regulation skills to partially mediate the prospective paths from childhood trauma to symptom distress (Lincoln et al., 2017). Moreover, Freeman and Fowler (2009) found anxiety (but not depression) to mediate between various types of trauma and paranoia in a cross-sectional study on a community sample. In a large-scale study, Bebbington et al. (2011) found especially the depressive states to act as a mediator. This was recently replicated by Marwaha and Bebbington (2015) who reported 30% of the link between sexual abuse and psychosis to be mediated by depression and 8.5% to be mediated by anxiety. Thus, there is sufficient evidence indicating that negative affect due to being traumatized could explain the link between trauma and psychosis.

A different perspective is to assume a moderating mechanism. Although negative affect tends to precede and predict the formation and persistence of paranoia, it is also evident that not everyone who experiences negative affect develops psychotic symptoms. Thus, there are likely to be further characteristics that make a person more vulnerable to respond to negative affect by experiencing symptoms. Trauma is a likely candidate in this regard because according to the emotional processing theory (Foa et al., 1989; Foa and Kozak, 1986), negative affective states are more likely to be closely linked to threatening experiences in those with a history of trauma, making the activation of fear structures, along with the physiological, cognitive and behavioral responses that serve to escape or avoid danger more likely. Following this line of reasoning, a history of trauma would constitute a vulnerability factor that determines whether negative affect in response to minor everyday stressors in later life is more or less likely to translate into psychotic symptoms. Such a vulnerability-stress mechanism would be reflected in a *moderation model*. Thus, both a mediation model and a moderation

model are theoretically founded, although to our knowledge, the moderation model has not yet been put to a comprehensive test and no study has tested for both mechanisms.

In the current study we hypothesized that more frequent trauma, recurring trauma, younger age at trauma, and the specific type of trauma would be predictive of threat beliefs in everyday life. Furthermore we hypothesized that the negative affect, including self-reported negative affect and arousal in the form of an increased heart rate in everyday life, would predict threat beliefs. Finally, we tested whether the interplay of trauma, self-reported negative affect, heart rate and threat beliefs fit the *mediation model* with trauma aspects leading to threat beliefs via increased negative affect in everyday life, or the *moderation model* with trauma aspects determining the strength of the link between negative affect and threat beliefs.

## 2. Methods

This study is a part of a larger project and the methods used here are the same as those presented in Krkovic et al. (2018) where we examined the role of emotion regulation for the affective path to psychosis.

### 2.1. Participants

We prescreened participants from former studies who had consented to be contacted for future projects and first semester psychology students for psychotic symptoms and subclinical psychotic experiences as measured by the Community Assessment for Psychic Experiences (CAPE; Stefanis et al., 2002). Individuals who scored eight or higher on the positive subscale of CAPE, which corresponds to the 50th percentile within a large community sample (Schlier et al., 2015), were invited to participate. Based on the recommendation by Krefelt and De Leeuw (1998) and Field (2005) we aimed to maximize the number of units on both between level (20 units have been discussed as minimum) and within level. The sample consisted of 67 individuals (71.6% female,  $M_{age} = 23.01$ ,  $SD_{age} = 4.63$ ). A diagnosis of a mental disorder in the past was reported by nine individuals, and 18 participants reported a mental disorder in a family member. Most participants (80.6%) were students. The project was approved by the local ethics committee. All participants provided written informed consent and were compensated with 10€ per hour or granted credit points if they were studying psychology.

### 2.2. Procedure

Baseline assessment was carried out at Universität Hamburg in Germany. Upon arrival, the ambulatory electrocardiogram (ECG) and an electro-dermal activity (EDA, not analyzed here) sensor were attached and activated. The participants then completed paper-pencil questionnaires, after which they received smartphones (for detailed information see the supplementary data). As depicted in Fig. 1a, the ECG recording was continuous over 24 h. The smartphones were programmed to beep in approximately 20-min-intervals (between 9 AM and 10 PM) resulting in 38 samples over 24 h. The starting and ending time-point of the ESM phase varied across participants. Participants were asked to behave as usual. The time references between any two ESM questionnaires is presented in Fig. 1. After the beep at any given time-point ( $t$ ), participants answered questions regarding their affect since the previous beep (thus for the time-period between  $t - 1$  and  $t$ ). Furthermore, participants answered questions on momentary threat beliefs referring to the time-point  $t$ .

### 2.3. Assessment instruments

#### 2.3.1. Screening assessment of psychosis proneness

The CAPE (Stefanis et al., 2002) measures lifetime psychotic symptoms and subclinical psychotic experiences. It is composed of 42 self-

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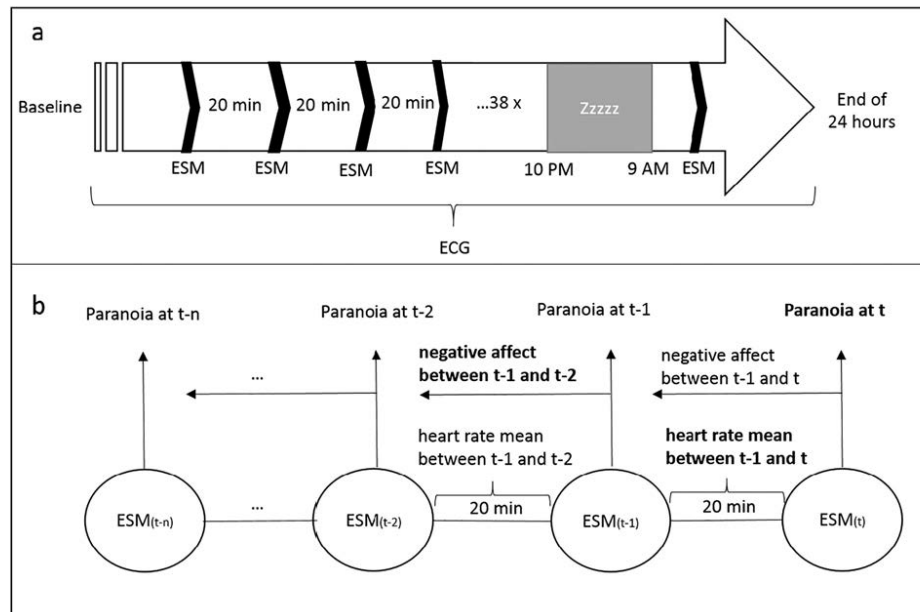


Fig. 1. Study procedure (1a) and time reference between any two experience sampling time-points (1b). ESM = Experience Sampling Method;  $t$  = time-point;  $t - 1$  = time-point preceding  $t$ ;  $t - 2$  = time-point preceding  $t - 1$ .

report items divided into the depressive, negative, and positive syndrome subscales. The items are rated on a four-point Likert scale ranging from 0 = “never” to 3 = “nearly always”. The CAPE has been found to be a valid and reliable measure of psychosis proneness (Konings et al., 2006) and to be sensitive in detecting individuals at ultra-high risk for psychosis (Mossaheb et al., 2012). The German version of the CAPE has good to excellent internal consistency (Schlier et al., 2015).

### 2.3.2. Baseline assessment of trauma

Trauma History Questionnaire (THQ; Hooper et al., 2011) is a 24-item self-report questionnaire that refers to potentially traumatic events including non-intentional (general) trauma such as natural disasters or accidents, and intentional trauma such as crime (specific), sexual and physical trauma. The questions are answered with “yes” or “no”. Trauma “frequency” is defined as the number of reported different traumas. Moreover, participants are asked to provide how often an event took place (if any event took place more than once, the variable “recurring trauma” is scored 1, otherwise 0) as well as the age at the time of the first traumatic experience. Types of trauma are scored as follows: if a participant answers “yes” to any of the items referring a one type of trauma (item 1 to 4 = crime related intentional; item 5 to 17 = non-intentional; item 18 to 20 = sexual; item 21 to 23 = physical), the respective trauma type variable is scored 1, otherwise 0. Item 24 refers to any other trauma that was not asked for and it is not included in the calculation. Additionally, participants are asked to provide a brief description of the event which was not analyzed in this study. The THQ has been found to have good psychometric properties (for a review see Hooper et al., 2011).

### 2.3.3. ESM assessment

Negative affect was assessed with six items referring to the previous 20 min (i.e., between  $t - 1$  and  $t$ , between  $t - 2$  and  $t - 1$ , etc., see

Fig. 1b). The scale included self-ratings of fear, sadness, anger, shame, guilt, and (un-)happiness, which were rated on a 10-point scale (based on Stemmler et al., 2001). The multilevel analysis of reliability (Geldhof et al., 2014) was acceptable with Cronbach's  $\alpha = 0.74$  for the within-subject level and Cronbach's  $\alpha = 0.86$  for the between-subject level.

We applied a sensor (Movisens ECGmove) designed for ambulatory electrocardiogram (ECG) to measure the heart rate. ECGmove is a small  $62.3 \times 38.6 \times 11.5$  mm sensor attached to electrodes that were placed on the left side of the chest. As presented in Fig. 1b, we used the mean heart rate between two beeps for the analyses (i.e., between  $t - 1$  and  $t$ , between  $t - 2$  and  $t - 1$ , etc.). The correction of artefacts was performed automatically in DataAnalyzer (Movisens GmbH).

State threat beliefs were assessed with the three items of the Paranoia Checklist (Freeman et al., 2005) that have been identified as sensitive to change (i.e., increase as well as decrease) in previous experimental studies (Schlier et al., 2016): “People are trying to make me upset”; “I need to be on my guard against others”; “Strangers and friends look at me critically”. In the same study, Schlier et al. (2016) also report first results of a pilot study showing that these items were sensitive to change in an ESM study. Participants answered on a 10-point Likert scale to which extent the statement applies to them at the moment of the beep (time-points  $t - 1$ ,  $t - 2$ , etc., see Fig. 1b).

### 2.4. Statistical analysis

Data was analyzed using SPSS Inc 22. We applied multilevel analysis and calculated linear mixed-effect models with random intercept and random slope for the mediation and moderation analyses. To analyze the effects of preceding negative affect on subsequent threat beliefs, we considered a lagged variable self-reported negative affect between  $t - 1$  and  $t - 2$  and mean heart rate between  $t$  and  $t - 1$  as a predictor of threat beliefs at  $t$  (see Fig. 1). We conducted separate mediation and

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moderation analyses for negative affect, heart rate, and all aspects of trauma (i.e. trauma frequency, recurring trauma, age of first trauma, and type of trauma).

To test the mediation model, we first examined whether different trauma aspects as independent variables predicted the outcome variable (i.e., threat beliefs; criterion 1). Next, any significant predictor of the outcome variable was further tested for a significant association with the hypothesized mediators (i.e. negative affect and heart rate) in a second set of regression models (criterion 2). If criterion 1 and 2 were fulfilled, we tested whether any of the two hypothesized mediators predicted the outcome variable (criterion 3) and whether the direct effect of the independent variable on the outcome variable was significantly reduced when the mediator was controlled for (criterion 4). This was performed by entering both the independent variable and the mediator as predictors in a third regression model.

To conduct the moderation analysis, independent variables that were repeated measures (negative affect and heart rate) were person-mean centered. All collected aspects of trauma (i.e. trauma frequency, recurring trauma, age at first trauma, intentional trauma, non-intentional trauma, sexual trauma, and physical trauma) were considered as the moderators and grand mean-centered. The moderation model was tested by entering the respective independent variable, the respective moderators and their interaction in a regression analysis predicting the outcome.

### 3. Results

#### 3.1. Descriptive statistics

The sum scores and standard deviations of the CAPE and the THQ at baseline, as well as the ESM scores of negative affect, heart rate and threat beliefs are summarized in Table 1. As reported in Krkovic et al. (2018), all participants indicated to experience at least one positive symptom in the CAPE “often” and 32% of participants indicated to experience at least one positive symptom “nearly always”. The compliance rate for the ESM assessment was 81% on average. Four percent of the heart rate data were missing due to technical issues. Intra-class-correlation coefficients were  $\rho = 0.523$  for threat beliefs,  $\rho = 0.470$  for self-reported negative affect, and  $\rho = 0.149$  for heart rate.

#### 3.2. Mediation models

Threat beliefs were significantly predicted by trauma frequency, recurring trauma, and by sexual trauma (criterion 1; see Table 2). Therefore, only these trauma aspects were further analyzed for potential mediation effects.

**Table 1**  
Mean, standard deviation and range of baseline and experience sampling method variables.

	N	M%	SD	Range
Baseline variables				
CAPE positive syndrome subscale	67	16.16	4.46	8.00–30.00
CAPE negative syndrome subscale	67	15.76	6.30	5.00–34.00
CAPE depressive syndrome subscale	67	9.85	3.73	3.00–20.00
Frequency of traumas	66	3.52	3.26	0–17
% reporting recurring trauma	67	53.73%	–	–
Age at first trauma	53	11.23	5.32	0–25
% reporting intentional trauma (crime)	67	36%	–	–
% reporting non-intentional trauma	67	82%	–	–
% reporting sexual trauma	67	24%	–	–
% reporting physical trauma	67	22%	–	–
Experience sampling method variables				
Heart rate	2432	88.96	19.80	43.99–188.61
Self-reported negative affect	2042	1.53	1.24	0.00–10.00
Paranoia	2039	0.74	1.24	0.00–7.00

Note. CAPE = Community Assessment of Psychic Experiences.

**Table 2**  
Direct effect of trauma aspects on paranoia in everyday life (criterion 1 of mediation analysis).

	b	SE	t	p	CI (95%)
Trauma frequency	0.137	0.031	4.406	<0.001	0.075; 0.200
Trauma recurrence	0.574	0.218	2.636	0.010	0.139; 1.009
Age at first trauma	−0.023	0.026	−0.892	0.377	−0.750; 0.029
Non-intentional trauma	0.461	0.294	1.570	0.121	−0.125; 1.047
Intentional trauma (crime)	0.169	0.238	0.712	0.479	−0.305; 0.644
Sexual trauma	0.545	0.260	2.095	0.040	0.026; 1.065
Physical trauma	0.391	0.269	1.454	0.151	−0.146; 0.929

Both tested predictors – negative affect, and heart rate – were significant predictors of subsequent threat beliefs (see Table 3).

Tests of criterion 2 showed that trauma frequency predicted negative affect ( $b = 0.129$ ,  $SE = 0.030$ ,  $t = 4.370$ ,  $p < 0.001$ ,  $CI\ 95\%[0.070; 0.189]$ ), and heart rate ( $b = 1.086$ ,  $SE = 0.440$ ,  $t = 2.472$ ,  $p = 0.016$ ,  $CI\ 95\%[0.208; 1.965]$ ), whereas recurring trauma neither predicted negative affect ( $b = 0.216$ ,  $SE = 0.216$ ,  $t = 0.999$ ,  $p = 0.322$ ,  $CI\ 95\%[−0.216; 0.647]$ ) nor heart rate ( $b = 2.585$ ,  $SE = 2.930$ ,  $t = 0.882$ ,  $p = 0.381$ ,  $CI\ 95\%[−3266; 8.436]$ ) so that a mediating model was not further considered. Sexual trauma only predicted negative affect ( $b = 0.709$ ,  $SE = 0.240$ ,  $t = 2.959$ ,  $p = 0.004$ ,  $CI\ 95\%[0.231; 1.188]$ ) but not heart rate ( $b = 5.894$ ,  $SE = 3.370$ ,  $t = 1.749$ ,  $p = 0.085$ ,  $CI\ 95\%[−0.836; 12.624]$ ).

When both trauma frequency and negative affect were entered to predict subsequent threat beliefs, negative affect was a significant predictor (criterion 3 fulfilled:  $b = 0.127$ ,  $SE = 0.035$ ,  $t = 3.604$ ,  $p = 0.001$ ,  $CI\ 95\%[0.056; 0.198]$ ), and the direct effect of trauma frequency was not reduced (criterion 4 not fulfilled:  $b = 0.141$ ,  $SE = 0.032$ ,  $t = 4.410$ ,  $p < 0.001$ ,  $CI\ 95\%[0.077; 0.205]$ ).

When both trauma frequency and heart rate were entered to predict subsequent threat beliefs, heart rate was a significant predictor (criterion 3 fulfilled:  $b = 0.006$ ,  $SE = 0.002$ ,  $t = 2.856$ ,  $p = 0.006$ ,  $CI\ 95\%[0.002; 0.010]$ ), but the direct effect of trauma frequency did not substantially change (criterion 4 not fulfilled:  $b = 0.141$ ,  $SE = 0.031$ ,  $t = 4.581$ ,  $p < 0.001$ ,  $CI\ 95\%[0.080; 0.203]$ ).

When both sexual trauma and negative affect were entered to predict subsequent threat beliefs, negative affect was a significant predictor (criterion 3 fulfilled:  $b = 0.121$ ,  $SE = 0.035$ ,  $t = 3.453$ ,  $p = 0.001$ ,  $CI\ 95\%[0.051; 0.191]$ ) but the direct effect of sexual trauma was not reduced (criterion 4 not fulfilled:  $b = 0.631$ ,  $SE = 0.267$ ,  $t = 2.364$ ,  $p = 0.021$ ,  $CI\ 95\%[0.098; 1.164]$ ).

#### 3.3. Moderation models

There was a significant interaction effect of trauma frequency  $\times$  negative affect, a significant interaction effect of age at first trauma  $\times$  negative affect, and a significant interaction effect of physical trauma  $\times$  negative affect on subsequent threat beliefs (see Table 3).

No interaction effect between any trauma variable and heart rate on subsequent threat beliefs was significant, except for a trend significant interaction effect of recurrence  $\times$  heart rate on subsequent threat beliefs (all moderation analyses are summarized in Table 3).

### 4. Discussion

As expected, given the strong evidence for a trauma history as a risk factor for psychosis (Janssen et al., 2004; Spauwen et al., 2006; Varese et al., 2012), we found trauma to be predictive of severity of threat beliefs in everyday life. Our results extend previous research by confirming the assumption that different aspects of trauma are differentially relevant and by contributing to a better understanding of the affective mechanism that links trauma to threat beliefs in daily life.

Specifically, we found that more frequent, recurring trauma and sexual trauma were predictive of experiencing more threat beliefs, whereas the age at which the trauma took place, non-intentional trauma,

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**Table 3**  
Moderation analyses with trauma aspects moderating the path from negative affect to paranoia.

	<i>b</i>	SE	<i>t</i>	<i>p</i>	CI 95%
NA between <i>t</i> and <i>t</i> – 1	0.313	0.042	7.519	<b>&lt;0.001</b>	0.230; 0.396
NA between <i>t</i> – 1 and <i>t</i> – 2	0.121	0.035	3.476	<b>0.001</b>	0.051; 0.190
Heart rate	0.005	0.002	2.470	<b>0.017</b>	0.001; 0.010
Frequency × NA between <i>t</i> and <i>t</i> – 1	0.023	0.011	2.083	<b>0.043</b>	0.001; 0.046
Recurring trauma × NA between <i>t</i> and <i>t</i> – 1	0.067	0.083	0.794	0.430	–0.101; 0.234
Age at first trauma × NA between <i>t</i> and <i>t</i> – 1	–0.015	0.009	–1.720	0.093	–0.033; 0.003
Intentional trauma × NA between <i>t</i> and <i>t</i> – 1	0.032	0.090	0.350	0.727	–0.148; 0.211
Non-intentional trauma × NA between <i>t</i> and <i>t</i> – 1	0.058	0.111	0.525	0.601	–0.163; 0.279
Sexual trauma × NA between <i>t</i> and <i>t</i> – 1	0.025	0.095	0.265	0.792	–0.166; 0.217
Physical trauma × NA between <i>t</i> and <i>t</i> – 1	0.174	0.093	1.875	0.066	–0.012; 0.360
Frequency × NA between <i>t</i> – 1 and <i>t</i> – 2	0.023	0.009	2.516	<b>0.016</b>	0.005; 0.041
Recurring trauma × NA between <i>t</i> – 1 and <i>t</i> – 2	–0.010	0.071	–0.143	0.887	–0.152; 0.132
Age at first trauma × NA between <i>t</i> – 1 and <i>t</i> – 2	–0.015	0.007	–2.144	<b>0.040</b>	–0.029; –0.001
Intentional trauma × NA between <i>t</i> – 1 and <i>t</i> – 2	–0.004	0.076	–0.046	0.963	–0.156; 0.149
Non-intentional trauma × NA between <i>t</i> – 1 and <i>t</i> – 2	0.051	0.095	0.542	0.590	–0.138; 0.240
Sexual trauma × NA between <i>t</i> – 1 and <i>t</i> – 2	–0.017	0.079	–0.216	0.830	–0.177; 0.142
Physical trauma × NA between <i>t</i> – 1 and <i>t</i> – 2	0.168	0.074	2.256	<b>0.029</b>	0.018; 0.317
Frequency × heart rate	<0.001	<0.001	0.613	0.544	–0.001; 0.001
Recurring trauma × heart rate	–0.008	0.004	–1.997	0.050	–0.017; 0.000
Age at first trauma × heart rate	–0.001	<0.001	–1.471	0.147	–0.002; 0.000
Intentional trauma × heart rate	0.001	0.004	0.114	0.886	–0.008; 0.009
Non-intentional trauma × heart rate	–0.002	0.006	–0.312	0.756	–0.013; 0.010
Sexual trauma × heart rate	<0.001	0.005	0.079	0.937	–0.010; 0.010
Physical trauma × heart rate	<0.001	0.005	0.006	0.995	–0.009; 0.009

Note. **Bold** = *p* < 0.05; NA = negative affect.

intentional trauma related to crime and physical trauma were not. The finding that sexual but not physical trauma was directly associated with threat beliefs contradicts studies by Fisher et al. (2010) and Rubino et al. (2009), who both found physical trauma to be the strongest and most robust predictor of psychosis. However, these studies investigated psychosis as a diagnostic category and not in form of specific symptoms as in the present study. Interestingly, Murphy et al. (2012) reported paranoia to act as a mediator between sexual abuse and psychosis, suggesting a direct relation between sexual abuse and paranoia that we also find in our study. In line with this, in the most recent meta-analysis, Bailey et al. (2018) found positive but not negative symptoms to be related to childhood abuse, whereby compared to other trauma types, childhood sexual abuse was most robustly related with delusions. Taken together, this suggests specific associations between certain types of trauma and specific symptoms of psychosis. Our results do not exclude the presence of an additive effect and interactions between different trauma characteristics, as was found by Fisher et al. (2010) who reported that physical abuse from the main mother figure before the age of 12 showed the most robust associations with psychosis. Due to the relatively small subsamples with specific types of trauma at different ages this could not be analyzed in the present study.

In regard to the mechanism that links trauma to negative affect and paranoia in daily life, our findings provide stronger support for the moderation than for the mediation model: Negative affect and heart rate did not mediate between different aspects of trauma and threat beliefs, although they both independently predicted threat beliefs. This does not support existing studies that found specific affective components to serve as mediators between trauma and paranoia (Freeman and Fowler, 2009; Lincoln et al., 2017; Marwaha and Bebbington, 2015). However, whereas previous studies refer to cross-sectional or longitudinal data including a relatively long time range of several months, our study reports on negative affect and threat beliefs on a “micro” level of one day. As discussed by Reininghaus et al. (2016), psychological mechanisms of psychosis formation could take place on both “macro” level (i.e., distal, life-span related) and “micro” level (i.e., proximal, over the short period of time). Hence, although this study does not support the notion that trauma predicts paranoia via negative affect in everyday life, this does not exclude the possibility that trauma is associated

with affective processes over longer time-periods, which then translate into threat beliefs.

Contrary to our expectations, in our sample, trauma did not moderate the association between physiological arousal and threat beliefs, with the exception of recurring trauma that was trend significant. On the one hand, this is surprising since physiological arousal triggered by the activation of the autonomic nervous system is assumed to be a biological counterpart of the affective response (Ekman, 1984; Friedman, 2010; Kreibig, 2010) so that similar patterns as those found for self-reported negative affect would have been expected. On the other hand, it is possible that heart rate is not a sufficiently specific indicator: whereas the subjective ratings in this study were tailored to assess negative rather than positive affect, elevated heart rate has been found to be indicative of both positive and negative arousal (compare a review by Kreibig (2010) that also suggests that different types of negative emotions appear to differ in the physiological response). Since this is the first study to examine the associations between different aspects of trauma, physiological and subjective stress and threat beliefs, future studies are needed for replication. Furthermore, it needs to be investigated whether the associations are the same for other physiological stress indicators that represent more specific indicators of negative affect and stress, such as salivary cortisol.

The findings should be viewed in the light of several limitations. One is that the ESM was limited to only 24 h, questioning the representativeness for everyday life. However, this short assessment period was a trade-off for including the ambulatory assessment of the heart rate that represents a more direct and objective measure of arousal. Moreover, the extent to which ESM methodology induces measurement-reactivity in participants that biases the data is still an open question (as discussed by Myin-Germeys et al., 2018). Our high-frequency assessment may be more prone to induce such measurement reactivity than one-week assessments with 90-min sampling intervals. Exploratory tests for time-effects (for detailed information, see the supplementary data), however, yielded no indication of reactivity in our sample. Another potential limitation is that the self-reported negative affect was assessed retrospectively for the last 20 min as this could have reduced the “momentary” nature of the ESM. However, considering the high frequency of questionnaires, a memory bias is unlikely. Moreover, we did not investigate the social context in which the

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negative affect and threat beliefs took place. Considering the complexity of people's lives, we argue that stressors cannot be assessed within an ESM study in a way that would help us to quantify the extent that threat beliefs are founded in social contexts. This requires observational studies that collect data on external stressors in a more objective manner or experimental studies that manipulate different types of stressors (e.g., urban environment, social-evaluation), which thus remain a necessary complement to ESM studies. The findings of the moderation analyses suggest that higher frequency, younger age and physical trauma, but not the other trauma characteristics, strengthen the path from negative affect to threat beliefs. Following the emotional processing theory (Foa and Kozak, 1986; Foa et al., 1989) it can be speculated that these trauma aspects could make a person vulnerable to respond to negative affect by an activation of fear structures that serve to escape or avoid danger, thereby evoking threat beliefs. Whether or not the activation of such fear structures takes place in this process remains to be investigated, however. Finding physical but not sexual trauma to be a significant moderator was surprising given that sexual trauma was more closely associated with threat beliefs than physical trauma. This suggests that different types of trauma are associated with symptoms in different manner, some directly and some by strengthening the path from negative affect to threat beliefs. It should be noted that in this study the assessment of trauma relied on self-report that is prone to memory bias and that the perceived severity of the trauma was not assessed, despite the fact that this has been considered important to determining the risk of psychosis (Kelleher et al., 2013; Spauwen et al., 2006). Moreover, we cannot exclude the possibility that trauma was confounded with other proximal factors (e.g., low current socioeconomic status, low social rank, a higher frequency of past and present non-traumatic stressors) that are also relevant to the etiology of psychosis. Finally, we did not assess the specificity of the associations by comparing the associations found for threat beliefs to other psychopathology, such as depression. Related to this, we assessed rather mild subclinical threat beliefs that can be placed at a lower position of the continuum of paranoia (factors of interpersonal sensitivity and mistrust, as found in Bebbington et al., 2013). Although this choice of items is advantageous in order to secure a sufficient variance in a general population sample, we cannot exclude the possibility that the associations may be different for more severe threat beliefs. Therefore, a replication of the study covering both healthy and clinical participants and including more severe threat belief items is advised.

Despite the limitations we argue that the results hold relevant clinical implications. Knowing which characteristics of trauma (i.e. sexual trauma, recurring trauma, trauma at young age) are more closely associated with threat beliefs than others could help to refine etiological models of positive symptoms. In particular, relying on the sociodevelopmental-cognitive model of schizophrenia by Howes and Murray (2014), it could now be tested if dopamine system (dys-)regulation that is often found in psychosis can be linked to specific aspects of trauma identified in our study and to examine how these relate to other etiological factors. For instance, it could be further examined whether specific trauma characteristics may explain the dysfunctional schemas that have been repeatedly found to be associated with paranoia (Bortolon et al., 2013; Sundag et al., 2016; Sundag et al., 2017), or whether the development of a healthy way of regulating emotions could alleviate the effect of specific trauma aspects on the affective path to symptoms as already suggested by Kim-Spoon et al. (2013) regarding general symptomatology in maltreated children. The findings of our study also point to potential targets in prevention: As trauma is hardly avoidable, trying to loosen the association between negative affect and threat beliefs may be worthwhile. One approach to this could be to provide adaptive emotion regulation skills trainings that have been found to be promising in various inpatient populations (Berking et al., 2008; Berking et al., 2013). Whether such interventions need to be adapted specifically for individuals prone to psychotic experiences or whether it is advantageous to use a non-specific approach at this

early and non-specific stage of risk (McGorry et al., 2010) is another issue for future research.

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#### Contributors

KK conceptualized the study design, analyzed the data and wrote the first draft of the manuscript. BS contributed to conceptualizing the study and edited the manuscript. TML contributed to interpreting and discussing the results, and substantially edited the manuscript. All authors contributed to and have approved the final manuscript.

#### Conflict of interest

The authors state that no potential conflicts of interest are associated to this manuscript.

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#### Appendix A. Supplementary data

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#### **Appendix D: Study IV**

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Emotions as predecessors, concomitants and consequences of paranoia. An experience sampling study

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## Abstract

**Objective:** Recent etiological models highlight the importance of emotions for the emergence of paranoia. To increase our understanding of the exacerbation of paranoid beliefs in daily life, we tested the hypothesis that negative emotions would precede paranoid beliefs, and that paranoid beliefs would trigger more negative affect – thus forming a vicious cycle.

**Methods:** The sample consisted of 30 individuals diagnosed with schizophrenia spectrum disorder. We applied the experience-sampling-method for one week to acquire repeated measures of sadness, anxiety, anger, guilt, shame, and paranoia in everyday life. We calculated multilevel models to test our hypotheses. **Results:** All negative emotions, except for sadness, were associated with paranoid beliefs measured at the same time-point ( $p < .05$ ). Only anxiety temporally predicted the subsequent paranoid beliefs ( $p < .05$ ). Anger was the only emotion that emerged as a consequence of paranoid beliefs ( $p < .05$ ). **Conclusion:** In contrast to our expectations, the results do not support a vicious cycle but showed that more anxiety led to more paranoia, while more paranoia led to more anger. Speculatively, paranoid beliefs could have a regulatory role, modifying anxiety into anger. We discuss short-term benefits and long-term detriment of this process by embedding our findings into a reinforcement learning model of symptom maintenance.

*Keywords:* Delusions; Negative affect; Psychosis

**Significant outcomes**

- We found paranoid beliefs to be accompanied by various negative emotions such as elevated anxiety, anger, guilt, and shame.
- Our results suggest that anxiety acts as a predecessor or trigger of paranoid beliefs and that anger represents a short-term emotional consequence of paranoid beliefs. This emphasizes the central role of emotional experiences for the formation, exacerbation and possibly maintenance of paranoid beliefs.
- Based on the findings, we propose a reinforcement learning model to explain the maintenance of paranoia, with short-term emotional benefits and long-term detriments of symptoms.

**Limitations**

- The findings are based on self-report. Physiological counterparts of emotional experiences should be included in future studies.
- Medication intake and its possible influences on affective experiences was not controlled for.

## 1. Introduction

Cognitive models (1–3) emphasize the central role of emotional experiences for the formation, exacerbation and maintenance of paranoid symptoms as one of the core symptoms of psychosis. These models propose that emotional distress leads to paranoia and accompany it, and that, in turn, paranoia leads to more emotional distress (3). This accords with the idea of a vicious cycle in which negative emotions drive the exacerbation of paranoid symptoms and vice versa. The first assumption, namely that emotional distress leads to symptoms and accompanies these, is supported by a large body of empirical research, including reviews and meta-analyses (4–7), experimental studies (8–11), and experience sampling studies (12–15).

In contrast, very few studies have investigated emotional processes that emerge as a time-delayed consequence of paranoid symptoms. In one such study, Van Rossum et al. (16) found a bi-directional dose-response association between depression scores and psychotic symptoms in a large scale prospective study on adolescents and young adults. In a qualitative interview study on the subjective experience of paranoia, Campbell and Morrison (17) reported that participants named anxiety, fear and anger as emotional consequences of paranoid symptoms. Furthermore, in a large scale general population study, Moritz and van Quaquebeke (18) reported a positive association between the conviction with which paranoid symptoms occurred with subsequent feelings of surprise, disbelief, anger, and helplessness. In contrast, a longitudinal study by Flower et al. (19) found only a one-directional path from depressed mood to paranoid symptoms in patients with psychosis. Also in our recent experience sampling method (ESM) study that focused on individuals with attenuated psychotic experiences, paranoid symptoms did not predict subsequent negative affect (20). In light of these sparse and to some extent contradictory findings,

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more research is required to test the notion of a vicious cycle, in which negative emotions do not only precede but also follow paranoid symptoms.

Finally, to gain better understanding of emotional processes related to paranoid symptoms, it would be advantageous if this research included a broad spectrum of emotions. So far, studies have strongly focused on anxiety and sadness as the most relevant emotions for paranoid symptoms (e.g., 10,14,15,21–23). However, there is also empirical evidence highlighting the importance of other emotional states, such as shame (24,25) or anger (26,27). In particular, the relevance of anger has been highlighted by Ullrich et al. (27) and Coid et al. (26) who both found that delusions predicted anger and that anger in turn predicted violent behavior in large-scale prospective studies. Hence, concurrently assessing and analyzing different emotional states is relevant in order to understand the emotional processes that take place before, during, and after paranoid symptoms and may contribute to their exacerbation and maintenance.

*Aims of the study*

We tested the notion of a vicious cycle of emotions and paranoid symptoms by investigating whether (a) negative emotions (anger, anxiety, sadness, shame, and guilt) temporally precede paranoid beliefs and (b) covary with the paranoid beliefs within a given time-point, and whether (c) paranoid beliefs temporally precede negative emotions in everyday life of individuals diagnosed with a schizophrenia spectrum disorder.

**2. Methods***2.1 Sample*

Participants were recruited via flyers distributed at the outpatient center for psychological therapy at the Universität Hamburg, as well as in outpatient facilities of general practitioners and psychiatrists. We recruited 33 participants who met following inclusion criteria: age between 18

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and 65, diagnosed with a schizophrenia spectrum disorder. Acute suicidality, dementia, severe organic diseases, bipolar disorder, substance abuse and insufficient knowledge of German language were classified as the exclusion criteria. Three participants dropped out of the study, resulting in a sample of 30 participants ( $M_{\text{age}} = 43.27$ ;  $SD_{\text{age}} = 11.46$ ; 33% female). Fifteen participants were diagnosed with a schizophrenia, twelve with a schizo-affective disorder and three with other schizophrenia spectrum disorder. Five participants fulfilled criteria for a comorbid disorder: two for major depression, one for a general anxiety disorder, one for post-traumatic stress disorder and one for panic disorder. The majority of the sample (83%) reported to take at least one antipsychotic medication on a regular basis.

### 2.2 Procedure

Baseline assessment took place at the Universität Hamburg, Germany. Participants were informed about the aims and procedure of the study and signed the informed consent form. First, a trained psychologist performed the diagnostic assessment with the participant. Depending on the participant's preference, the second part of the study was conducted after a short break or on another day within the following week. Here, participants were first asked to complete a computerized self-assessment battery. Thereafter, they received a smartphone equipped with the movisensXS ESM application (Movisens GmbH). After activating the application, the participants left the laboratory and the ESM assessment phase began. The smartphones were programmed to alert the participants ten times per day in approximately 90 minutes intervals between 9 AM and 10 PM for seven consecutive days. The starting and ending time-point of the ESM assessment phase varied across participants, but no participant was subject to any assessments between 10 PM and 9 AM. Participants were instructed to behave as usual. Following the alert at a given time-point ( $t$ ), participants answered questions regarding their



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momentary negative affect (thus for the time-point t). Furthermore, participants answered questions on paranoid beliefs referring to the time-period between two consecutive questionnaires (between t-1 and t). After completing the ESM phase, participants returned the smartphones and were debriefed. Participants were compensated with 10€ per hour for the baseline assessment (interviews and self-assessment questionnaires) and additional 30€ for completion of the ESM assessment.

*2.3 Assessment instruments**2.3.2 Baseline Assessment*

*Psychopathology.* The German Version of the Structured Clinical Interview for DSM-IV Axis I disorders (SCID-I) (28) was conducted to assess the diagnostic criteria of the schizophrenia spectrum disorders and possible comorbidities. The psychotic disorders section of the interview was updated with a preliminary translation of updated SCID-I based on the DSM V diagnostic criteria. A semi-structured interview, The Psychotic Symptom Rating Scales (PSYRATS) (29), was used to quantify the severity of delusions and hallucinations. The delusions subscale of the PSYRATS is comprised of six items: duration and frequency of preoccupation, intensity of distress, amount of distressing content, and conviction and disruption. The PSYRATS has been shown to have good concurrent validity in relation to the commonly used Positive and Negative Syndrome Scale (PANSS) (30) and good internal consistency and sensitivity to change (31).

*Questionnaire battery.* Demographic data, the emotion-specific Emotion Regulation Skills Questionnaire (32), the Cognitive Emotion Regulation Questionnaire (CERQ) (33), the Perceived Stress Questionnaire (34), and the Trauma History Questionnaire (THQ) (35) were administered. Except for demographic data, the questionnaire battery was not analyzed in this study and therefore we abstained from reporting the results of these scales.

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*2.3.3 ESM assessments*

Negative affect was assessed with six items rated on a 10-point Likert scale (based on Stemmler et al. (36)). Participants were instructed to describe the emotions they were experiencing momentarily (at time-point  $t$ ). The scale included self-ratings of anxiety, sadness, anger, shame and guilt. The scale has been used in previous studies to examine stress reactivity in laboratory and in daily life and has shown a good psychometric quality (12,37).

Paranoid beliefs were assessed with a five item, change-sensitive scale (38), based on the Paranoia Checklist (39). Participants were asked to which extent each of the statements applied to them within the time period since the last beep (time-point between  $t-1$  and  $t$ ) on a 10-point Likert scale. The multilevel reliability of the five-item scale was acceptable for the within-subject-level (Cronbach's  $\alpha = .72$ ), and excellent for the between-subject-level (Cronbach's  $\alpha = .94$ ).

Experience sampling included further questions regarding momentary stress level, momentary happiness and hallucinations, which were not analyzed in this study given the focus on negative affect and paranoia. Additionally, participants could click on a button presented on the display to report their paranoid or hallucinatory symptoms at any time, which was also not analyzed in the present study.

*2.4 Statistical Analysis*

Data was analyzed using IBM SPSS Statistics software (version 22). In order to account for the hierarchical design of the study with multiple observations nested within participants, we applied multilevel analyses. As suggested by Wang and Maxwell (40), independent variables that were repeated measures were centered around the subject-mean. To test whether negative affect and paranoid beliefs covaried, we calculated a linear mixed-effect model with random intercept

and fixed slope, including paranoid beliefs as dependent variable and anxiety, anger, sadness, shame, and guilt at the same time-point as independent variables. Variability of the intercept was modeled for participants and for days of assessment. To test whether negative affect measured at t-1 predicted paranoid beliefs measured at t, we applied time-lagged linear mixed-effect models with random intercept and the within-person autoregressive slope (i.e., paranoid beliefs measured at t-1 predicting paranoid beliefs measured at t was estimated at person-level and allowed to vary randomly across individuals). Due to collinearity issues leading to statistical artifacts, separate analyses were conducted for each negative emotion (i.e. anger, anxiety, sadness, shame, and guilt). Finally, to test whether paranoid beliefs measured at t-1 predicted negative affect at t, we again applied time-lagged linear mixed-effect models with random intercept and the within-person autoregressive slope (i.e. negative affect measured at t-1 predicting negative affect measured at t).

### 3. Results

#### 3.1 Descriptive statistics

Table 1 summarizes the means, standard deviations and range of the PSYRATS items as a baseline measure of paranoid beliefs and the ESM measures. Intraclass-correlation coefficient (ICC) for paranoid beliefs in the ESM was  $\rho = .554$ , and for emotions the ICCs ranged from  $\rho = .515$  to  $\rho = .735$ .

#### 3.3 Emotions preceding paranoid beliefs

Controlling for paranoid beliefs assessed at time t-1, only anxiety measured at t-1 predicted paranoid beliefs measured at t (see Table 2). Thus, the more anxiety participants reported at one time-point, the more paranoid beliefs they reported at the next time-point.

#### 3.2 Emotions accompanying paranoid beliefs

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All tested emotions were positively associated with paranoid beliefs measured at the same time-point (intercept:  $b = 2.048$ ,  $SE = 0.434$ ,  $t = 4.724$ ,  $p < .001$ ,  $CI\ 95\% [1.161, 2.935]$ ; anxiety:  $b = 0.051$ ,  $SE = 0.018$ ,  $t = 2.897$ ,  $p = .004$ ,  $CI\ 95\% [0.016, 0.086]$ ; anger:  $b = 0.151$ ,  $SE = 0.017$ ,  $t = 8.924$ ,  $p < .001$ ,  $CI\ 95\% [0.118, 0.184]$ ; shame:  $b = 0.131$ ,  $SE = 0.019$ ,  $t = 6.850$ ,  $p < .001$ ,  $CI\ 95\% [0.094, 0.169]$ ; guilt:  $b = 0.070$ ,  $SE = 0.019$ ,  $t = 3.711$ ,  $p < .001$ ,  $CI\ 95\% [0.033, 0.108]$ ), except for sadness that was marginally non-significant ( $b = 0.037$ ,  $SE = 0.019$ ,  $t = 1.888$ ,  $p = .059$ ,  $CI\ 95\% [-0.001, 0.075]$ ).

### *3.4 Emotions following paranoid beliefs*

When paranoid beliefs measured at t-1 were considered as a predictor of the negative affect measured at t, while controlling for the respective negative affect measured at t-1, only the model predicting anger was significant (Table 3). That is, the more paranoid beliefs were reported at t-1, the more anger was reported at t.

All results are graphically presented in Figure 1.

## **4. Discussion**

Aiming to understand the emotions involved in the formation, exacerbation, and maintenance of paranoid symptoms, the present study focused on emotions that precede, accompany, and emerge as a consequence of paranoid beliefs in the everyday life of individuals with psychosis. Whereas different emotions accompanied the paranoid beliefs, we found only anxiety to precede these, and only anger to be elevated as a consequence of paranoid beliefs.

The finding that anxiety was the only emotion temporally predictive of paranoid beliefs is in line with other studies showing that anxiety is a robust predictor of paranoid symptoms (8–11). This further corroborates the assumption postulated in the model of persecutory delusions by Freeman et al. (3) that anxiety and paranoid beliefs are closely related, possibly through their

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common content – “the anticipation of threat”. Somewhat surprisingly, sadness neither preceded nor followed paranoid beliefs. This is in contrast to some previous findings where sadness was found to be the best predictor of paranoid symptoms next to anxiety (5,41) but confirms findings of other studies that did not detect a predictive role of sadness for paranoid symptoms (e.g., 14,41). Furthermore, in contrast to the studies that found anger, guilt or shame to be relevant to paranoia (24–27), in this study we found no temporal associations between these emotions and paranoid beliefs. Hence, our findings further underline the specific importance of anxiety as a precedent of paranoid symptoms.

With regard to the emotions accompanying paranoid symptoms, anxiety, anger, guilt, and shame covaried with paranoid beliefs at the same time point. Hence, paranoid symptoms were accompanied with a diffuse negative emotional state. This mixture of emotions may imply that individuals have difficulties in differentiating and understanding their emotions during distressing paranoid episodes, which would be in line with studies showing deficits in awareness and understanding of emotions in individuals with psychosis (e.g., 43,44). This diffuse emotional state could also be interpreted as “feeling overwhelmed“ by emotions and paranoid explanations may represent an attempt to down-regulate this overwhelming state.

In contrast to the assumption postulated in the model of persecutory delusions by Freeman and colleagues (3), which suggests that anxiety precedes and follows from paranoid beliefs and therefore contributes to the exacerbation and maintenance of symptoms, we did not find anxiety to follow from paranoid beliefs. Similar to the study on a community sample with elevated psychosis proneness where we found a uni-directional, but no bi-directional, association between negative affect and subsequent paranoid symptoms (12), in the present clinical sample we found almost no time-lagged emotion eliciting effect of paranoid beliefs. The only emotion

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that paranoid beliefs were predictive of was anger. The path from anxiety, that preceded paranoid beliefs, and the diffuse negative emotional state during the paranoid episode to anger could be explained by attributional processes that emerge from “a search for meaning” in anxiety eliciting situations (3,45). Bentall and Kaney (45) proposed that negative self-representations (e.g., low self-esteem), that are common in individuals with psychosis (46), paired with threatening events, would trigger externalizing attributions (e.g., blaming others) that then take on the form of paranoid explanations. In contrast to internal attributions (self-blame), such externalizing attributions have been found to have a short-term positive effect on negative affect and self-esteem (47). Moreover, Gilbert and Miles (48) found specific associations with anger: In a study on the sensitivity to social put-down, they found that whereas anxiety was only related to self-blame, blaming others was associated with feeling angry in healthy participants. These attributional processes can be integrated in a *reinforcement learning model* to explain the exacerbation and maintenance of paranoid symptoms: By transforming anxiety, that is related to threat avoidance behavior, into anger, that instills agency and facilitates approach to the threat (49), paranoid attributions could be beneficial for the individual in short-term. Thus, paranoid attributions may emerge as an emotion regulatory process to reduce anxiety and overwhelming affective states and re-establish short-term control. In the long run, however, paranoid attributions may become increasingly dysfunctional as they consolidate the perception of a hostile environment and facilitate further development of paranoid beliefs (51). However, the long-term consequences and their contribution to the learning mechanism are speculative at present.

Several limitations need to be considered. First, this study relies solely on self-reported data. Although the subjective perception of participants is relevant, studies on emotional states

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should be complemented by further aspects of emotional processing, such as autonomic arousal or activation of the hypothalamic-pituitary-adrenal axis (52). This seems especially important considering that patients with psychosis have been found to have difficulties in emotion awareness (53,54), so that physiological data could offer additional, more accurate measures of negative affect. Moreover, as common in ESM research, the ESM assessments took place approximately every 90 minutes during seven consecutive days. Thus, the lagged associations refer only to 90 minutes intervals and it is possible that the associations would have been different if we had studied shorter or longer periods of time. Furthermore, the repeated measures design can evoke specific reactivity in individuals, such as changes in perception of own emotions or thoughts (55,56). Finally, our sample also included individuals with the diagnosis of a schizoaffective disorder who might show different patterns of affectivity than those with non-affective psychosis. Due to small subsamples we were not able to separately analyze diagnosis subgroups with sufficient power.

Despite these limitations, the study adds to gaining a clearer understanding of the association between emotions and paranoid symptoms in daily life of individuals with psychosis. Our results imply that interventions focusing on emotions and attributional processes could be indicated. For instance, the transdiagnostic affect regulation training by Berking and Whitley (57) could be a promising approach to regulate anxiety and prevent the development and exacerbation of paranoid symptoms in everyday life. This training provides strategies to become aware of, identify, and understand emotions with regard to triggering situations and underlying cognitions and teaches participants to use other strategies to adaptively cope with the anxiety. Furthermore reasoning trainings could be useful to reduce externalizing attributional processes (58,59). The study's most relevant contribution, however, is that it questions the notion of a

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vicious circle of paranoia and anxiety as postulated by previous researchers and points to the possibility of reinforcement learning mechanisms involved in the exacerbation and maintenance of paranoia. This is likely to provide a basis for further research on this mechanism, which may even result in novel interventions for paranoia.



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Table 1. Mean, standard deviation and range of the baseline and the experience sampling method variables

	<i>N</i>	<i>M</i>	<i>SD</i>	Range
<b>Baseline variables</b>				
PSYRATS Duration of preoccupation	30	1.93	0.907	0-4
PSYRATS Frequency of preoccupation	30	2.20	1.095	0-4
PSYRATS Intensity of distress	30	2.67	1.295	0-4
PSYRATS Amount of distressing content	30	2.40	1.499	0-4
PSYRATS Conviction	30	1.73	1.112	0-3
PSYRATS Disruption	30	1.67	0.884	0-3
<b>Experience sampling method variables</b>				
Anxiety	1287	2.21	2.756	0-10
Sadness	1286	2.59	2.792	0-10
Anger	1286	1.98	2.525	0-10
Shame	1284	2.05	2.994	0-10
Guilt	1282	2.76	3.225	0-10
Paranoid beliefs	1280	2.004	2.505	0-10

PSYRATS, The Psychotic Symptom Rating Scales



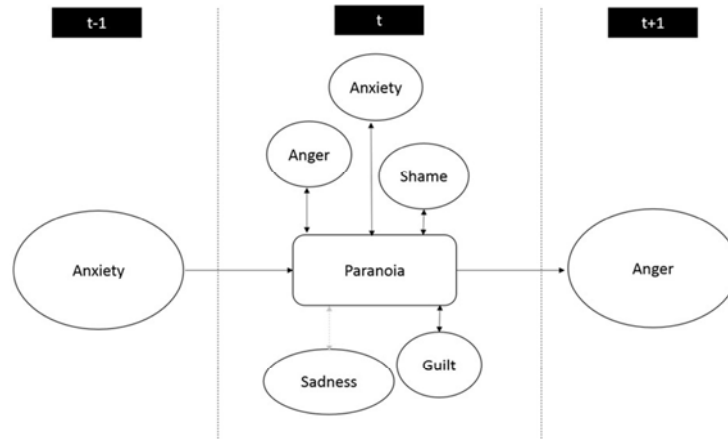


Figure 1. Graphic illustration of study results. Anxiety at preceding time-point t-1 predicting paranoia at t, paranoia and diffuse emotional state correlated at t, and paranoia at t predicting anger at t+1.

Graphic illustration of study results. Anxiety at preceding time-point t-1 predicting paranoia at t, paranoia and diffuse emotional state correlated at t, and paranoia at t predicting anger at t+1.

160x123mm (150 x 150 DPI)

## Appendix E: Curriculum vitae, publication list and statements

### Curriculum vitae

#### Work experience

Since 03/2017	Cognitive behavioral therapist in training, PTA Hamburg, Germany
Since 07/2015	Research assistant, Universität Hamburg, Germany
12/2012 - 06/2015	Research assistant, University of Luxembourg, Luxembourg
03/2012 - 06/2012	Psychology intern, General hospital, Clinic for children, Neuro-oncology, Vienna, Austria
08/2011 - 11/2011	Psychology intern, Kolpinghaus - gemeinsam leben, Gerontology, Vienna, Austria

#### Education

Since 10/2015	Postgraduate training for behavioral psychotherapy, PTA Hamburg, Germany
Since 07/2015	Doctoral studies in psychology, Universität Hamburg, Germany
Since 06/2014	Postgraduate training for clinical and health psychologists, AAP Austrian Academy for Psychology, Vienna, Austria
2013 - 2015	PhD studies in educational psychology, University of Luxembourg, Luxembourg
11/2012	Psychology degree – mag.rer.nat (with honours), University of Vienna, Austria
2007 - 2012	Diploma studies of psychology, University of Vienna, Austria
2003 - 2007	High school „Uros Predic“, Pancevo, Serbia
1995 - 2003	Primary school „Jovan Jovanovic Zmaj“, Pancevo, Serbia

#### Funding

Ca. 136 000€	Scientific grant AFR, Fondation National du Recherché Luxembourg (personal PhD-Stipendium 2013 - 2015)
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**Teaching**

Clinical diagnostics (Bachelor curriculum seminar in WS 2015, WS 2016 and WS2017)

Anxiety disorders (Bachelor curriculum seminar in SS 2017 and SS 2018)

Bachelor thesis seminar (Bachelor curriculum SS 2016)

First supervisor of various bachelor and master theses

**Languages**

Serbian – mother tongue

German – fluent

English – fluent

**Publication list****Book chapters**

**Krkovic, K., Mustafic, M.,** Wüstenberg, S., & Greiff, S. (2017). Shifts in the assessment of problem solving. In P. Griffin, B. McGaw, & E. Care (Eds.), *Assessment and teaching of 21<sup>st</sup> century skills*. Dordrecht: Springer.

**Peer-reviewed journal papers**

**Krkovic, K., Schlier, B., & Lincoln, T. M.** (2018). *Do paranoid beliefs have an emotion regulatory role? An analysis of the temporal interplay of emotions and paranoid symptoms in daily life*. Manuscript submitted for publication.

Schlier, B., **Krkovic, K., & Lincoln, T.** (2018). *An experience sampling study on the nature of the interaction between traumatic experiences, negative affect in everyday life, and paranoia*. Manuscript submitted for publication.

Pillny, M., **Krkovic, K., & Lincoln, T.** (in press). Development and initial validation of the Dysfunctional Belief Scale: A new scale to assess dysfunctional beliefs related to amotivation. *Cognitive Therapy and Research*.

**Krkovic, K., Schlier, B., & Lincoln, T. M.** (in press). An experience sampling study on the nature of the interaction between traumatic experiences, negative affect in everyday life, and threat beliefs. *Schizophrenia research*.

**Krkovic, K., Clamor, A., & Lincoln, T. M.** (2018). Emotion regulation as a predictor of the endocrine, autonomic, affective, and symptomatic stress response and recovery. *Psychoneuroendocrinology, 94*, 112-120.

**Krkovic, K., Krink, S., Lincoln, T. M.** (2018). Emotion regulation as a moderator of the

interplay between self-reported and physiological stress and paranoia. *European Psychiatry*, 49, 43-49.

Clamor, A. & **Krkovic, K.** (2018). Paranoid delusions as an adaptive response to social evaluative stress? *Zeitschrift für Psychologie*, 226, 191-196.

**Krkovic, K.**, Moritz, S., & Lincoln, T. M. (2017). Neurocognitive deficits or stress overload: Why do individuals with schizophrenia show poor performance in neurocognitive tests? *Schizophrenia Research*, 183, 151-156.

Hennig, T., **Krkovic, K.**, & Lincoln, T. (2017). What predicts self-reported inattention in adolescents? An experience-sampling study comparing chronotype, subjective and objective sleep parameters. *Sleep Medicine*, 38, 58-63.

Greiff, S., **Krkovic, K.**, & Hautamäki, J. (2016). The prediction of problem-solving assessed via microworlds: A study on the relative relevance of fluid reasoning and working memory. *European Journal of Psychological Assessment*, 32, 298-306.

**Krkovic, K.**, Wüstenberg, S., & Greiff, S. (2016). Assessing collaborative behaviour in students: An experiment-based assessment approach. *European Journal of Psychological Assessment*, 32, 52-60.

**Krkovic, K.**, Greiff, S., Kupiainen, S., Vainikainen, M.-P., & Hautamäki, J. (2014). Teacher evaluation of student ability: what roles do teacher gender, student gender, and their interaction play? *Educational Research*, 56, 243-256.

Greiff, S., **Krkovic, K.**, & Nagy, G. (2014). Systematic variation of task characteristics facilitates understanding of task difficulty. A Cognitive Diagnostic Modeling approach towards Complex Problem Solving. *Psychological Test and Assessment Modeling*, 56, 83-103.

Ras, E., **Krkovic, K.**, Greiff, S., Tobias, E., & Maquil, V. (2014). Moving towards the assessment of collaborative problem solving skills with a tangible user interface. *The Turkish Online Journal of Educational Technology*, 13, 95-104.

**Krkovic, K.**, Pásztor-Kovács, Molnár, G., & Greiff, S. (2013). New technologies in psychological assessment: The example of computer-based collaborative problem solving assessment. *Special Issue of the International Journal of e-Assessment*, 1, 1-13.

#### **Conference papers (as a first author)**

**Krkovic, K.**, Schlier, B., & Lincoln, T. (2018, May). *Emotions as triggers, companions and consequences of paranoia in everyday life.* [Emotionen als Auslöser, Begleiter und

*Folge von Paranoia im Alltag.*] Oral presentation at 36<sup>th</sup> symposium of Fachgruppe für Klinische Psychologie und Psychotherapie der Deutschen Gesellschaft für Psychologie, Landau, Germany.

**Krkovic, K., & Lincoln, T.** (2017, September). *The interplay between traumatic experiences, stress sensitivity, and subclinical paranoid symptoms in daily life.* Oral presentation at 6<sup>th</sup> European Conference on Schizophrenia Research, Berlin, Germany.

**Krkovic, K., & Lincoln, T.** (2017, May). *Paranoid due to stress? Emotion regulation as an important component of the stress reaction.* [*Paranoid durch Stress? Emotionsregulation ist ein wichtiger Baustein der Stressreaktion.*] Oral presentation at 35<sup>th</sup> symposium of Fachgruppe für Klinische Psychologie und Psychotherapie der Deutschen Gesellschaft für Psychologie, Chemnitz, Germany.

**Krkovic, K., Krink, S., & Lincoln, T. M.** (2016, May). *Does emotion regulation influence the path from daily hassles to psychosis? An experience sampling study.* [*Beeinflusst die Emotionsregulation den Weg vom Alltagsstress zur Psychose? Eine „Experience-Sampling“ Studie.*] Oral presentation at 34<sup>th</sup> symposium of Fachgruppe für Klinische Psychologie und Psychotherapie der Deutschen Gesellschaft für Psychologie, Bielefeld, Germany.

**Krkovic, K., Greiff, S., Kupiainen, S., Vainikainen, M.-P., & Hautamäki, J.** (2014, August). *How gender influences performance assessment. Teacher-student gender interaction in focus.* Paper presented at the EARLI SIG1 conference in Madrid, Spain.

**Krkovic, K. & Greiff, S.** (2014, July). *The assessment of collaborative problem solving: an experiment-based assessment of behavior approach.* Paper presented at the 9<sup>th</sup> Conference of the International Test Commission in San Sebastian, Spain.

**Krkovic, K. & Greiff, S.** (2014, April). *Collaborative problem solving. Concept, assessment, and first results.* Invited Speech at the 6<sup>th</sup> Szeged Workshop on Educational Evaluation, University of Szeged, Hungary.

**Krkovic, K. & Greiff, S.** (2013, September). *Geschlechterbias in der Bildung: Die Interaktion der Geschlechter als Ursache* [*Gender bias in education – gender interactions as a source*]. Paper presented at the 14<sup>th</sup> Conference on Educational Psychology of the German Psychological Association in Hildesheim, Germany.

**Krkovic, K., Rudolph, J., & Greiff, S.** (2013, April). *Collaborative Problem Solving: Current Assessment Possibilities and Issues.* Invited Speech at the 5<sup>th</sup> Szeged Workshop on Educational Evaluation, University of Szeged, Hungary.