



The influence of stress on social cognition in patients with borderline personality disorder



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Summary

Background: Borderline personality disorder (BPD) is characterized by severe difficulties in interpersonal relationships and emotional functioning. Theories of BPD suggest that individuals with BPD have heightened emotional sensitivity, increased stress reactivity, and problems in making sense of intentions of others. In this study we investigated stress reactivity in BPD and its interference with social cognition, and tested whether any differences are specific for BPD or are inherent to personality disorders in general.

Methods: We investigated 22 patients with BPD, 23 patients with Cluster C personality disorder (CPD), and 24 nonpatients on facial emotion recognition and social evaluation before and after stress induction based on the Trier Social Stress Test (TSST).

Results: The results show that stress increased subjective negative emotions in the BPD group to a larger extent than in the other groups, whereas physiological responses were attenuated.

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Importantly stress induction increased negative evaluations about others, but surprisingly to a similar extent in the BPD and CPD groups as in the nonpatient control group. In addition facial emotion recognition performance was higher after than before stress, but no significant group differences were observed.

Conclusion: These results suggest that heightened psychological reactivity in BPD co-occurs with attenuated physiological responses to psychosocial stress and that stress affects social cognition to a similar extent in BPD as in others.

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1. Introduction

Borderline personality disorder (BPD) is characterized by severe difficulties in interpersonal relationships and emotional functioning (APA, 2000). Relationships of individuals with BPD are chaotic, intense and marked with difficulties. Furthermore, it has been assumed that they are emotionally more susceptible to social cues that signal social threat or rejection (Domes et al., 2009). Linehan et al. (2007) suggested that individuals with BPD have intense reactions to emotional stimuli and an inability to regulate their intense physiological arousal. It is unclear how emotional reactivity is related to problems in social interaction. The theory of Fonagy and Bateman (2008) posits that, due to early trauma and disruption of the attachment relationships, the capacity to make sense of oneself and others (i.e. mentalization) becomes unstable during emotional arousal. This suggests that emotional reactivity might interfere with social cognition capacities. Emotional reactivity elicits the rapid release of the stress hormones adrenaline and noradrenaline via the autonomic nervous system and the slow release of cortisol via the hypothalamus–pituitary–adrenal (HPA) axis (Ulrich-Lai and Herman, 2009). Initial studies that investigated HPA axis functioning in BPD suggest that its negative feedback on the release of cortisol is reduced, though this appears to have little influence on basal cortisol levels (Zimmerman and Choi-Kain, 2009). More recent studies investigated emotional and physiological reactivity in social interaction by inducing social stress. One study found elevated cortisol levels during recovery after an interpersonal conflict task in patients with BPD compared to nonpatients (Walter et al., 2008). Other studies have used the well-established Trier Social Stress Test (TSST) and observed heightened subjective emotional distress and cortisol reactivity in BPD patients with severe dissociation, but not in those with low dissociation (Simeon et al., 2007). In contrast, later studies with larger samples found attenuated cortisol responses using the same TSST protocol even though the participants with BPD reported higher subjective emotional intensity (Nater et al., 2010; Scott et al., 2013). Although these stress induction studies show mixed results, they provide initial evidence for dysregulation of the stress system in BPD.

Another line of research has investigated social cognition in BPD. Social cognition refers to the capacity to understand ourselves and others as individuals with feelings, beliefs and a personality (Marton et al., 2005; Mitchell et al., 2004) and is considered to be essential to successful social adaptation (Arntz et al., 2009). One aspect of social cognition is the ability to correctly recognize facial expressions of others. Studies that investigated facial emotion recognition found

that patients with BPD recognize emotions of others either less accurately (Levine et al., 1997) or more accurately (Lynch et al., 2006; Domes et al., 2008; Schulze et al., 2013). Another aspect of social cognition is the capacity to understand perspectives and intentions of others, underlying their observable behavior (theory of mind or mentalizing; Arntz et al., 2009). Studies that investigated more complex social cognition found better performance on a theory of mind test in BPD compared to control groups (Fertuck et al., 2009; Arntz et al., 2009), but also found impaired social cognitive abilities in BPD (Preißler et al., 2010). Other studies found a stronger tendency in BPD to describe others in a more negative and dichotomous manner (Veen and Arntz, 2000; Arntz and Veen, 2001; Sieswerda et al., 2013; Arntz and Ten Haaf, 2012).

Although the results from these studies are mixed, they support the notion that BPD is associated with dysfunctional emotional as well as social processing. Daros and colleagues (2013) proposed a model to explain the divergent findings in facial emotion recognition, in which moderate levels of arousal enhance social cognitive performance but high levels of arousal deteriorate performance in BPD. Research in healthy individuals indeed suggests that arousal affects social cognition. For example, performance on a social cognition test after stress induction is improved in women with moderate cortisol responses, but performance is lower in women with higher cortisol responses. (Smeets et al., 2009). Furthermore, the induction of negative emotions influences social problem solving negatively in individuals with BPD traits (Dixon-Gordon et al., 2011). However, it remains unclear if stress affects social cognition in patients with BPD, which could be an explanation for their social interaction problems. To directly address whether dysregulation of the stress system affects social cognition in BPD, we assessed facial emotion recognition and social evaluation before and after social stress. Because none of the former stress studies examined whether stress regulation problems were specific for BPD, we compared a BPD patient group with a Cluster C personality disorder (CPD) patient group and a nonpatient control group. Before and after social stress induction based on the TSST, participants completed a facial emotion recognition task and a social evaluation task that assessed the participant's evaluation of the experimenter during social interaction. On the basis of recent TSST studies (Nater et al., 2010; Scott et al., 2013), we expected that the BPD group would show dysregulation of the stress system, as reflected by enhanced emotional but reduced physiological responses to stress. We did not already expect large differences in facial emotion recognition performance at baseline, as emotion recognition accuracy is not strongly affected in

BPD under normal circumstances (Daros et al., 2013), though social evaluations may already be slightly more negative (Arntz and Ten Haaf, 2012). Importantly, we expected that social stress would negatively affect social cognition to a larger extent in patients with BPD than in patients with CPD and nonpatients as reflected by more negative social evaluations and relatively lower facial emotion recognition accuracy after stress induction.

2. Methods

2.1. Participants

Twenty-three patients diagnosed with borderline personality disorder (BPD), 23 patients diagnosed with Cluster C personality disorder (CPD), and 24 non-patient comparison subjects were included. Patients between 18 and 46 years of age were recruited from inpatient and outpatient departments of Vincent van Gogh Institute for Psychiatry in Venray and Venlo, The Netherlands. The non-patients were recruited from the personal networks of the employees of the same institute. All procedures were carried out with the adequate understanding and written consent of the participants. Only female participants were recruited as female patients with BPD are overrepresented in the treatment population. The groups were matched on age and intelligence quotient (IQ) as measured with the Dutch version of the National Adult Reading Test (NART; Schmandt et al., 1992). Patients had to meet the criteria for BPD or one or more diagnoses of the Cluster C PDs as defined by the DSM-IV-TR (APA, 2000) and measured with the Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II; First et al., 1997). CPD patients with more than two traits of BPD were excluded from participation. Exclusion criteria for all the participants included psychotic and bipolar disorders and current anorexia as measured with the Mini-International Neuropsychiatric Interview (M.I.N.I.; Sheehan et al., 1998). Additional exclusion criteria for the nonpatients were a history of psychiatric treatment or current psychiatric treatment, significant impairment because of axis I (assessed with the M.I.N.I.) or axis II traits or more than two criteria of a personality disorder (assessed with the SCID-II). Exclusion criteria for all groups were the use of neuroleptics (with the exception of low dose quetiapine as sleep medication), acute substance related disorder, major medical illness, history of neurological treatment or current neurological treatment, abnormal (uncorrected) vision, use of corticosteroids <6 months before participation, pregnancy, breast feeding, irregular menstrual cycle, excessive physical exercise and use of corticosteroids or other medication which affect cortisol levels (i.e. medication for blood pressure control or respiratory tracts).

All participants used hormonal contraceptives at the time of the study or took part in the experiment during their luteal phase to minimize hormonal variations across the menstrual cycle. Participants were instructed to minimize physical exercise, not to use alcohol 24 h preceding the experiment, not to use illicit drugs 72 h preceding the experiment, not to use benzodiazepines at least 48 h or quetiapine 72 h preceding the experiment, to cease or lower smoking from the day before the experiment and at least not to

smoke 2 h preceding the experiment, and not to take large meals, coffee and drinks with low PH during the morning preceding the experiment, because these variables may affect cortisol levels (Dickerson and Kemeny, 2004; Kirschbaum et al., 1999). One participant of the BPD group used benzodiazepines the day before the experiment and was therefore excluded from the analyses. Demographic data are presented in Table 1. The study was approved by the local ethics committee and all participants signed informed consent before participation.

2.2. Materials

2.2.1. Diagnostic interviews

Axis II diagnoses were assessed by trained psychologists with the Dutch version of the Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II; First et al., 1997), which has good psychometric characteristics (Lobbestael et al., 2008). Comorbid axis I diagnoses were assessed by trained psychologists with the Dutch version of the Mini-International Neuropsychiatric Interview (M.I.N.I.) that has good psychometric characteristics (Sheehan et al., 1998).

2.2.2. Social stress induction

The protocol used to induce stress was a modified version of the Trier Social Stress Test (TSST), a well-established test for inducing social stress (Kirschbaum et al., 1993). The participants were requested to prepare (3 min) and deliver a speech (5 min) and perform difficult mental arithmetic (5 min) in front of a video camera and the experimenter. The experimenter of the same gender (female) was unknown to the participants before the experimental session. The participants were instructed to speak about why they would be suitable for a desired job and to include their positive and negative personality characteristics. The experimenter did not provide verbal or non-verbal feedback during the speech and asked confrontive questions thereafter (e.g. ‘‘what makes you think that you are suitable for this job and not somebody else?’’). The participants were told that their speech would be recorded and judged by a panel of experts. During debriefing after the experiment, participants were informed that this would not happen.

2.2.3. Emotional state measures

To assess changes in subjective emotional state during the experiment, visual analog scales (VAS) of the short form of the Profile of Mood States (POMS-SF; Shacham, 1983) were used. In line with Curran et al. (1995), we computed a total POMS score from the depression, tension, anger, fatigue, and inverted vigor subscales. High POMS scores reflected high levels of negative affective states. Psychometric characteristics of the POMS are good (Shacham, 1983; Curran et al., 1995).

The POMS was assessed before, immediately after TSST and 25–30 min after TSST (delayed measures). Continuous heart rate was measured using the POLAR RS800CX Heart Rate Monitor at a sampling rate of a 1000 Hz which was converted to beats per minute (BPM) and averaged for pre-TSST baseline (before TSST instructions and during the first two social cognition tasks), TSST-anticipation (during TSST instructions and speech preparation), TSST-performance

Table 1 Participant characteristics of patients with borderline personality disorder (BPD), cluster C personality disorder (CPD), and nonpatient controls (NP).

	BPD (<i>n</i> = 22)	CPD (<i>n</i> = 23)	NP (<i>n</i> = 24)	<i>p</i> -Value
Age (years)	31.43 (8.16)	31.91 (6.43)	28.63 (8.28)	0.29 ^d
Intelligence quotient (IQ)	93.55 (11.56)	93.61 (9.71)	96.83 (9.21)	0.45 ^d
Medication (<i>n</i>)				
Antidepressants	10	11	0	1.00 ^e
Hormonal contraceptives ^a	16	16	15	0.75 ^f
Other ^b	4	3	3	0.85 ^f
Smokers (>5 cigarettes/day)	15	7	2	0.00 ^f
Axis I comorbidity (<i>n</i>)				
Depressive disorder	7	6		
Dysthymic disorder	8	3		
Panic disorder	4	1		
Post-traumatic stress disorder	5	2		
Obsessive compulsive disorder	8	7		
Generalized anxiety disorder	5	3		
Substance related disorder ^c	3	0		
Axis II comorbidity (<i>n</i>)				
Cluster B				
Borderline PD	22	0		
Narcissistic PD	0	0		
Histrionic PD	0	0		
Antisocial PD	3	0		
Cluster C				
Avoidant PD	5	3		
Dependent PD	0	2		
Obsessive-compulsive PD	4	2		
Personality disorder NOS	0	17		
Cluster A				
Paranoid PD	3	1		
Schizotypal PD	0	0		
Schizoid PD	0	0		

Age and IQ present the mean (SD).

^a Participants who did not use hormonal contraceptives were tested in the luteal phase (in the period of the last two weeks of the menstrual cycle).

^b Including medication for somatic symptoms. Sedative medication was discontinued before the experiment (see Section 2).

^c Substance dependence or acute substance abuse.

^d One-way ANOVA.

^e χ^2 for two patient groups.

^f χ^2 for three groups.

(speech performance and arithmetic task of the TSST), and post-TSST (heart rate during the second two social cognition tasks). Saliva samples were obtained before, immediately after and 35–40 min after TSST using non-invasive Salivette devices (Sarstedt, Rommelsdorf, Germany). All samples were stored at -20°C until assaying. Biochemical analysis of free cortisol in saliva was performed using commercially available chemiluminescence immunoassay (CLIA, IBL, Hamburg, Germany). Cortisol data of one subject were excluded due to extreme values ($>50\text{ nmol/L}$). Changes in cortisol level were analyzed as the percentage change from pre-TSST baseline.

2.2.4. Social cognition

In order to measure social evaluation, a list with VAS items with opposing personality characteristics was used on which the participants judged the experimenter's

character. The list included three subscales: personality characteristics related to BPD specific views of self and others (BPD cognitions; e.g. reliable–unreliable, hostile–not hostile), personality characteristics with a negative–positive polarity which were not specifically related to BPD cognitions (negative cognitions; e.g. passive–active, dull–interesting), and personality characteristics without negative–positive polarity (neutral cognitions; rational–intuitive, introvert–expressive). The dimensional subscales have excellent interrater reliability (Veen and Arntz, 2000).

To assess emotion recognition accuracy, a facial emotion recognition task (ERT) was used. This task measures the recognition of six basic emotional expressions: anger, disgust, fear, happiness, sadness and surprise (Montagne et al., 2007). The stimuli used in the task are color pictures of present-day faces of two males and two females.

Video-clips were constructed that morphed the faces from neutral to emotional. The experiment started with the most difficult video-clips that morphed from 0% to 20% emotional intensity. The level of emotional intensity of the morphing stimuli increased throughout the task from 20% to 100% in steps of 10%, thereby successively decreasing task difficulty. Participants were instructed to identify the emotion of the face by selecting the appropriate emotion label presented next to the faces using a mouse. One hundred eight trials from one male and one female actor were presented before the stress induction (i.e. 2 actors \times 6 emotions \times 9 difficulty levels) and 108 trials from another male and female actor were presented after the stress induction. The order of tasks was counterbalanced across subjects. For data analysis, one emotion recognition accuracy score was defined for each of the emotions by summation of the correctly identified stimuli across all actors and difficulty levels. In addition, a total score was calculated by summation of all trials across the different emotions.

2.3. Procedure

All participants visited the lab twice. On the first day (diagnostic screening) a trained psychologist assessed the inclusion and exclusion criteria. On the second (test) day participants arrived between 12:30 and 16:00 in the afternoon to avoid high morning cortisol levels. The participants were administered a training session of the emotion recognition task and thereafter watched a relaxing movie to minimize variability in cortisol levels due to physical activity or stress related to novelty with the experiment. Afterwards the baseline social cognition tasks were administered in the absence of the experimenter, and baseline POMS scores and a saliva sample were collected. Thereafter, social stress was induced using the TSST protocol described above, after which the POMS and a saliva sample were obtained again. Subsequently, the second administration of the social cognition tasks took place. The ERT was performed in the presence of a video camera and the experimenter who took notes and observations and told the participant that her previous performance was not adequate and told the participant to do better this time. This procedure was used to ensure that the second ERT was performed in the context of continued negative social evaluation. The second social evaluation task that was administered thereafter was not obtained in presence of the experimenter because the participants had to judge the experimenter. Instead, participants were assured that the social evaluation task would be handled anonymously. In addition, delayed measures of the POMS (25–30 min after TSST) and a saliva sample (35–40 min after TSST) were obtained. Finally, participants were debriefed and informed about the nature of the study and care was taken to assure that participants did not go home upset.

2.4. Statistical analysis

Baseline measures were compared between groups (BPD, CPD, nonpatient (NP)) using one-way ANOVAs. Stress-induced changes in emotional state measures and facial emotion recognition accuracy were analyzed using mixed

model ANOVAs with the factors group and time (with corresponding number of levels). In addition we specifically assessed emotional reactivity as the change from pre-TSST to TSST and recovery as the change from pre-TSST to post-TSST. The influence of social stress on social evaluation was analyzed using mixed model ANOVAs with the levels group, time (pre-TSST, post-TSST), and cognition type (BPD cognitions, negative cognitions, neutral cognitions). Data of at least one measurement were missing for the following variables: POMS (1 BPD, 1 NP), heart rate (3 CPD, 2 NP), cortisol (4 BPD, 3 CPD, 9 NP), social evaluation (2 CPD). Huynh–Feldt correction of the degrees of freedom was applied when sphericity was violated, nonparametric tests were used when the normality assumption was violated (e.g. for emotion specific tests), and alpha was set at 5% throughout. Because no previous studies have investigated the influence of stress on social cognition in BPD and the effect size is therefore unknown, we planned our sample size such that we would at least have sufficient power to detect large effects. A power calculation in G*Power 3.1 showed that a total sample size of 64 subjects is required to obtain 80% power at 5% alpha to detect group (3) \times time (2) interactions with a large effect size ($f=0.4$) (Faul et al., 2007).

3. Results

3.1. Baseline results

To investigate whether there were differences between the groups before stress induction we analyzed emotional states and social cognition at baseline.

3.1.1. Emotional states

Negative subjective emotional state as measured with the POMS at baseline was significantly different between the groups ($F(2,65)=22.28$, $p<0.001$, $\eta^2=0.41$; see Fig. 1). POMS total scores were significantly higher in the BPD group ($M=1325.41$, $SD=532.65$) than in the nonpatient group ($M=489.52$, $SD=222.18$; $t(27.84)=-6.82$, $p<0.001$), and marginally different from the CPD group ($M=1044.17$, $SD=471.56$; $t(43)=1.88$, $p=0.07$). Subsequently we explored whether the differences in POMS scores were related to specific emotions. The BPD group scored higher than the nonpatient group on depression ($U=52$, $z=-4.66$, $p<0.001$), anger ($U=61.5$, $z=-4.35$, $p<0.001$) tension ($U=90.5$, $z=-3.82$, $p<0.001$) and fatigue ($U=36$, $z=-5.01$, $p<0.001$) and lower on vigor ($U=87.5$, $z=-3.88$, $p<0.001$), and higher than the CPD group on anger ($U=162$, $z=-2.07$, $p=0.039$) and fatigue ($U=158$, $z=-2.16$, $p=0.031$).

We assessed physiological states at baseline by measuring heart rate ($F(2,62)=0.21$, $p=0.818$) and cortisol levels ($F(2,58)=1.37$, $p=0.263$) but did not observe any significant differences between the groups, suggesting there were no differences in physiological states before stress induction (Fig. 1).

3.1.2. Social cognition

The social evaluation task at baseline showed significant differences between the groups in borderline cognitions

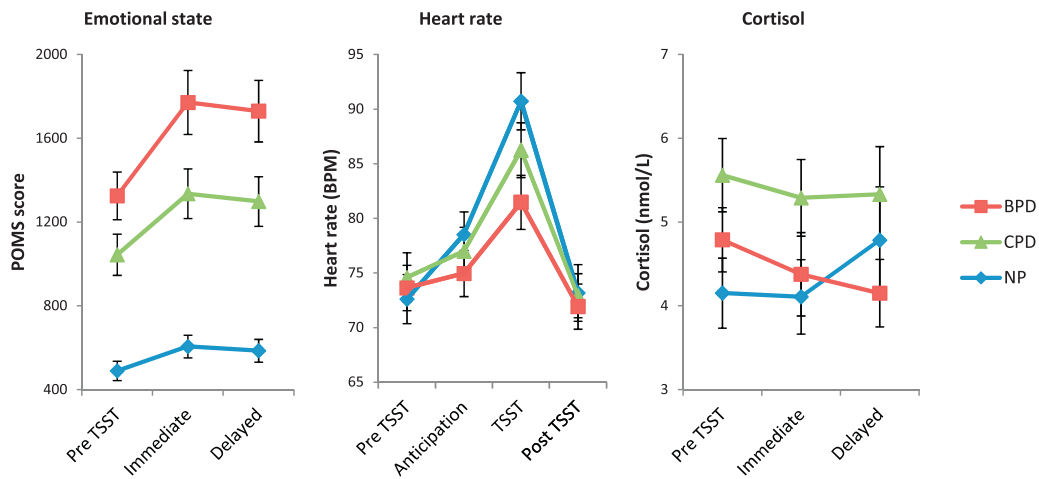


Figure 1 Emotional and physiological responses before and after stress induction in the borderline personality disorder (BPD) group, cluster C personality disorder (CPD) group and nonpatient control group (NP). Data represent the mean \pm SEM.

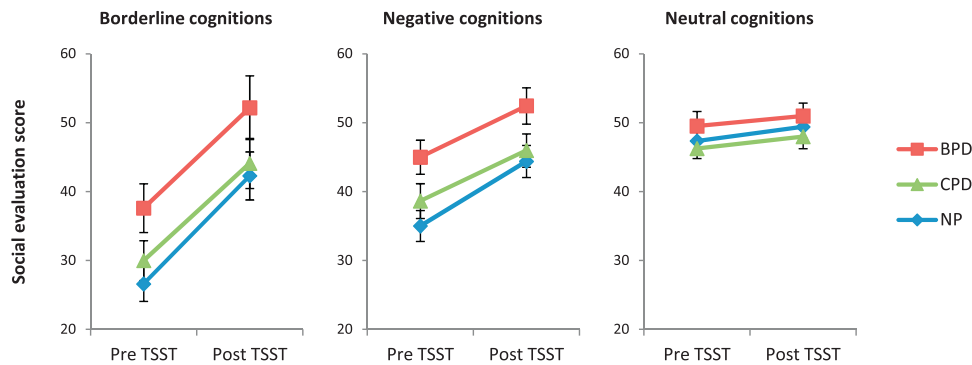


Figure 2 Social cognition scores before and after the stress induction for the borderline personality disorder (BPD) group, cluster C personality disorder (CPD) group and nonpatient control group (NP). Data represent the mean \pm SEM.

($F(2,66) = 3.52$, $p = 0.035$, $\eta^2 = 0.10$) and negative cognitions ($F(2,65) = 4.43$, $p = 0.016$, $\eta^2 = 0.12$), but not in neutral cognitions ($F(2,65) = 0.97$, $p = 0.385$, $\eta^2 = 0.03$; see Fig. 2), indicating that the groups differed in borderline and negative cognitions before stress induction.

The BPD group ($M = 37.59$, $SD = 16.62$) scored significantly higher on borderline cognitions than the nonpatient group ($M = 26.59$, $SD = 12.34$; $t(44) = -2.56$, $p = 0.014$), and marginally higher than the CPD group ($M = 29.98$, $SD = 13.87$; $t(43) = 1.67$, $p = 0.10$).

The BPD group ($M = 45.00$, $SD = 11.62$) also scored significantly higher on negative cognitions than the nonpatient group ($M = 35.00$, $SD = 11.02$; $t(44) = -2.96$, $p = 0.004$) and marginally higher than the CPD group ($M = 38.63$, $SD = 11.84$; $t(42) = 1.80$, $p = 0.079$).

Performance on the total scores of the facial emotion recognition task was not significantly different between the groups (BPD group: $M = 11.44$, $SD = 1.50$; nonpatient group: $M = 11.83$, $SD = 1.74$; CPD group: $M = 11.45$, $SD = 1.71$; $F(2,66) = 0.43$, $p = 0.65$, $\eta^2 = 0.013$). The groups did not differ either in performance on the individual emotions (Kruskal–Wallis; all p -values > 0.13 ; see Supplementary Fig. 1), which indicates that the BPD group performed at control level with respect to the labeling of sad, anxious, angry,

surprised, happy or disgusted emotional facial expressions before stress induction.

Supplementary Fig. 1 related to this article can be found, in the online version, at <http://dx.doi.org/10.1016/j.psyneuen.2014.11.003>.

3.2. Stress induction

3.2.1. Changes in subjective emotional state

The influence of stress on emotional states is presented in Fig. 1. To examine whether the subjective emotional state changed after the stress induction, a group \times time ANOVA was conducted. The results showed a main effect of time ($F(1,84,118.03) = 34.99$, $p < 0.001$, $\eta_p^2 = 0.35$) with higher POMS scores after than before stress induction, indicating that stress resulted in increased negative emotions. Importantly there was also a group \times time interaction ($F(3.69,118.03) = 3.63$, $p = 0.010$, $\eta_p^2 = 0.10$). The increase in POMS scores, assessed immediately after the stress induction, was larger in the BPD group ($M(\text{pre}) = 1359.48$, $SD = 520.67$; $M(\text{post}) = 1822$, $SD = 692.54$) than in the nonpatient group ($M(\text{pre}) = 489.52$, $SD = 222.18$; $M(\text{post}) = 617.70$, $SD = 263.30$; $t(31.82) = -2.86$, $p = 0.007$,

but did not differ from the CPD group $M(\text{pre})=1044.17$, $SD=471.58$, $M(\text{post})=1335.52$, $SD=569.64$; $t(43)=1.31$, $p=0.197$. Subsequently we explored whether the increase in negative emotional state was related to specific emotions. Increases in tension ($U=90.50$, $z=-3.81$, $p<0.001$), anger ($U=138.00$, $z=-2.61$, $p=0.009$) and depression ($U=171.00$, $z=-2.05$, $p=0.041$) were higher for the BPD group than for the nonpatient group. The BPD group differed from the CPD group in the increase in tension ($U=159.50$, $z=-2.12$, $p=0.034$), which was higher for the BPD group. Differences between the BPD group and CPD group reached a trend toward significance in the increase in anger, which was slightly higher in the BPD group ($U=176.50$, $z=-1.74$, $p=0.082$).

We subsequently assessed the decrease in subjective emotions 25–30 min after stress induction (delayed POMS scores). Across groups, subjective emotions tended to decrease after stress ($t(67)=1.62$, $p=0.110$), which did not differ significantly between the groups ($F(65)<1$). To assess whether the BPD group recovered from stress as much as the other groups, we tested if the delayed total POMS scores were higher with respect to the baseline. The subjective emotions of the BPD group remained higher ($M(\text{delayed})=1730.14$, $SD=674.41$) than the subjective emotions of the non-patient group ($M(\text{delayed})=596.17$, $SD=267.77$); $t(33.75)=-2.53$, $p=0.016$, but did not differ from the CPD group ($M(\text{delayed})=1298.70$, $SD=567.93$); $t(42)=1.14$, $p=0.262$. Together these results show that the overall change in experienced emotions, immediately after stress-induction, was higher in BPD patients than in nonpatients, and was higher compared to CPD patients specifically for tension and with a trend for anger. Furthermore after stress, there were no differences between the groups in the decrease of the subjective emotions, but the BPD group showed less recovery with respect to the baseline compared to the non-patient group.

3.2.2. Physiological changes

To examine whether heart rate (beats per minute (bpm)) changed during the stress induction, a group \times time ANOVA was conducted. The results showed a main effect of time ($F(1.47, 89.50)=133.79$, $p<0.001$, $\eta_p^2=0.69$), with higher heart rate during than before stress induction. Importantly there was also a group \times time interaction ($F(2.93, 89.5)=6.10$, $p=0.001$, $\eta_p^2=0.17$). The increase in heart rate was smaller in the BPD group ($M(\text{pre})=73.64$, $SD=9.76$; $M(\text{TSST})=81.48$, $SD=11.62$) compared to nonpatient group ($M(\text{pre})=72.61$, $SD=10.56$; $M(\text{TSST})=90.73$, $SD=12.28$; $t(28.88)=3.45$, $p=0.002$) and almost significantly smaller compared to the CPD group ($M(\text{pre})=74.65$, $SD=10.62$; $M(\text{TSST})=86.15$, $SD=12.25$, $t(41)=-1.99$, $p=0.054$).

Heart rate returned to baseline levels after stress (paired t -tests; post-TSST–pre-TSST); in the nonpatients ($M(\text{post})=73.18$, $SD=12.16$; $t(21)=-0.65$, $p=0.525$) and decreased even further in the BPD ($M(\text{post})=71.93$, $SD=9.71$; $t(21)=2.90$, $p=0.009$) and CPD group ($M(\text{post})=72.70$, $SD=9.41$; $t(19)=2.77$, $p=0.012$). The recovery of heart rate (post-TSST–pre-TSST) in the BPD group differed significantly from the nonpatient group ($t(42)=2.15$, $p=0.038$), but not from the CPD group ($t(40)=0.27$, $p=0.789$).

The percentage change in cortisol levels compared to baseline level was tested using a group \times time ANOVA. The results showed that the change in cortisol reached a trend toward significance between the groups ($F(2,50)=2.70$, $p=0.077$, $\eta_p^2=0.098$). Whereas cortisol levels increased in the nonpatient group ($M=9.87$, $SD=33.96$), cortisol levels decreased in the BPD group ($M=-10.04$, $SD=20.30$) and in the CPD group ($M=-4.39$, $SD=21.71$). To address our hypothesis about altered physiological responses in BPD directly, we compared the BPD group and the nonpatient group and found that the cortisol response was reduced in the BPD group ($t(31)=2.08$, $p=0.046$). There was no significant difference between the BPD and CPD group, $p=0.40$. Thus, the physiological response to stress as measured with heart rate and cortisol was attenuated in BPD and CPD.

3.2.3. Change in social cognition

The influence of stress on borderline, negative, and neutral cognitions on the social evaluation task is presented in Fig. 2. To examine whether social evaluations changed after the stress induction, a group \times time \times cognition type ANOVA was conducted. The results showed a main effect of time ($F(1, 64)=79.76$, $p<0.001$, $\eta_p^2=0.56$) with higher social evaluation scores after than before stress induction. As expected the results also showed a main effect of cognition ($F(1.63, 104.00)=20.92$, $p<0.001$, $\eta_p^2=0.246$), showing that, overall, the three cognition subscales were scored differently. Importantly there was a time \times cognition interaction ($F(1.66, 106.27)=31.03$, $p<0.001$, $\eta_p^2=0.33$). Across groups, contrast analysis revealed that the increase in the borderline cognitions ($M(\text{pre})=31.67$, $SD=14.74$; $M(\text{post})=46.49$, $SD=18.66$) was higher than the increase in negative cognitions ($M(\text{pre})=39.90$, $SD=11.43$; $M(\text{post})=47.81$, $SD=11.52$, $F(1,64)=21.38$, $p<0.001$, $\eta_p^2=0.25$) and that the increase in negative cognitions was higher than the increase in neutral cognitions ($M(\text{pre})=47.88$, $SD=7.82$; $M(\text{post})=49.64$, $SD=8.34$; $F(1,64)=37.78$, $p<0.001$, $\eta_p^2=0.37$). Surprisingly, the results showed no significant differences between the groups in the change in social evaluation scores (all $ps>0.7$).

To examine whether facial emotion recognition (ERT) changed after stress induction, a group \times time ANOVA was conducted for the total emotion recognition scores. The results showed a main effect of time ($F(1,66)=8.20$, $p=0.006$, $\eta_p^2=0.110$), with higher ERT scores after than before stress induction, indicating better performance after stress induction for all the groups. The groups did not differ in their performance over time ($F(2,66)=1.65$, $p=0.201$, $\eta_p^2=0.05$) and neither differed in recognition performance for the specific emotions (Kruskal–Wallis; all p -values >0.10 ; see Supplementary Fig. 1).

Together these results show that stress increased borderline and negative cognitions on the social evaluation task to a similar extent in all the groups. Facial emotion recognition accuracy also increased after stress, but again no group differences were observed.

3.3. Depression comorbidity

To explore whether depression comorbidity had an influence on emotional states and social cognition before and after

stress induction, we compared the results of BPD patients with and without major depressive disorder (MDD).

3.3.1. Emotional and physiological states at baseline and after stress induction

The BPD subgroups did not differ in emotional states (total POMS scores) at baseline ($t(20) = -0.34$, $p = 0.741$) or after stress induction ($F(2,38) = 0.26$, $p = 0.773$). Heart rate at baseline was significantly higher in the BPD group with MDD ($M = 81.07$, $SD = 7.57$) than in the BPD group without MDD ($M = 70.17$, $SD = 8.81$; $t(20) = -2.82$, $p = 0.01$, $\eta^2 = 0.28$). However, the change in heart rate during stress was not significantly different for the BPD subgroups (MDD \times time interaction; $F(1.95, 39.05) = 1.19$, $p = 0.32$). Cortisol levels were not significantly different between the BPD subgroups at baseline ($t(18) = -0.91$, $p = 0.374$) nor after stress induction ($F(1,16) = 1.24$, $p = 0.28$).

3.3.2. Social cognition at baseline and after stress induction

On the social evaluation task, the BPD subgroups did not differ in borderline ($t(20) = 0.04$, $p = 0.969$), negative ($t(20) = -0.35$, $p = 0.729$) or neutral cognitions ($t(20) = 0.20$, $p = 0.847$) at baseline, nor after stress induction ($F(1.86, 37.25) = 0.41$, $p = 0.651$).

Performance on the total scores of the facial emotion recognition task was not significantly different between the borderline subgroups at baseline ($t(20) = 0.02$, $p = 0.982$). However, the results showed a statistical trend for the change in performance over time ($F(1,20) = 4.18$, $p = 0.054$, $\eta_p^2 = 0.173$). Performance of the BPD group without MDD was enhanced after stress induction ($M(\text{pre}) = 11.44$, $SD = 1.71$; $M(\text{post}) = 12.08$, $SD = 1.68$), whereas performance of the BPD group with MDD deteriorated after stress induction ($M(\text{pre}) = 11.43$, $SD = 1.06$; $M(\text{post}) = 10.95$, $SD = 1.70$). When analyzed separately for the distinct emotions, this effect was also significant for fear ($U = 23.00$, $z = -2.10$, $p = 0.036$) and surprise ($U = 21.50$, $z = -2.19$, $p = 0.028$).

4. Discussion

The aim of this study was to investigate stress reactivity in Borderline personality disorder (BPD) and how this interferes with social cognition. To determine whether any differences are specific to BPD or are common to personality disorders, we compared patients with BPD to patients with Cluster C personality disorder (CPD) as well as nonpatients. The results show that stress increased negative subjective emotions in the BPD group to a larger extent than in the other groups, whereas physiological responses were attenuated. Importantly negative social evaluations increased during social stress, but surprisingly to a similar extent in the BPD and CPD groups as in the nonpatient group. This suggests that emotions have a similar impact on social evaluation in BPD patients and in CPD patients as in nonpatients.

4.1. Stress reactivity

Initial studies that investigated stress reactivity in small samples with BPD have found increased physiological

responses (Simeon et al., 2007; Walter et al., 2008), whereas our results are in line with later studies (Nater et al., 2010; Scott et al., 2013) with larger samples that reported increased subjective emotional responses combined with attenuated physiological responses. Our results partially contradict the theory of Linehan et al. (2007) which posits that emotional dysregulation in BPD is characterized by increased sensitivity for social cues enhanced reactivity to emotional stimuli and an inability to regulate heightened physiological arousal with a slow return to emotional baseline. Although the enhanced subjective emotional responses of patients with BPD largely confirm this hyperreactivity theory, the results suggest that it is not so much a slower return to baseline that creates longer periods of negative emotions in BPD, but the initially stronger response – which even with parallel slopes compared to nonpatients leads to longer periods of negative emotions. However, their attenuated physiological responses to stress are not in line with this theory, which predicted heightened physiological arousal. Moreover, their heart rate decreased even beyond the baseline level, which contrasts with the predicted slow return to baseline. Although it is not yet clear how to reconcile this divergent pattern of subjective emotional and physiological reactivity, it appears that subjective hyperreactivity is not directly associated with physiological hyperresponsivity. A prominent explanation for these paradoxical findings is that a genetic predisposition in combination with chronic early life stress leads to developmental changes of the HPA axis, leading to later hyporesponsivity of the HPA axis (Sanchez et al., 2005; Carpenter et al., 2007; Nater et al., 2010). Altered HPA axis function may in part be mediated by differential sensitivity of the receptors that bind cortisol such as the glucocorticoid receptor (GR) (Carpenter et al., 2009). Cortisol also binds to the mineralocorticoid receptor (MR), which has been hypothesized to play a role in BPD (Wingenfeld et al., 2014). The GR has low affinity for cortisol and mediates the slow effects of cortisol, whereas the MR has high affinity for cortisol and can exert rapid effects (De Kloet et al., 2005). The balance between MR and GR sensitivity may therefore also have an influence on HPA axis function in BPD, as has been suggested for depression (Young et al., 2003). Interestingly, our study shows that the attenuated cortisol reactivity in CPD is similar to that in BPD and even decreased with respect to baseline rather than increased as in nonpatients, which may also be caused by early life stress (Carpenter et al., 2007). However, the attenuated heart rate increase was more pronounced in the BPD than CPD group. Together, this suggests that the patient groups mainly differ in their sympathetic response, which may be the consequence of differences in biological vulnerability and more severe trauma exposure in the BPD group than the CPD group (Johnson et al., 1999). This relationship should be further investigated in future studies.

4.2. Social cognition

Emotional arousal due to stress was accompanied by increased social evaluation scores for all the groups. Participants showed the greatest increase in BPD-specific cognitions which are related to “BPD specific views” of others (i.e. unreliable, hostile), followed by negative cognitions

related to more general negative views of others such as unkindness, impatience and dullness. The stress induction had no significant influence on the neutral subscale. This suggests that stress influenced specific negative cognitions that are related to the social interaction with the experimenter rather than it induced a response bias leading to equally higher scores for all categories. The change in BPD specific cognitions occurred in all groups, suggesting that a social stress situation in which people have to perform unexpectedly and are judged on their performance leads all people to judge others as being hostile, aggressive and unreliable. This appears characteristic for patients with BPD as it was already higher in this group in the neutral situation before stress induction. Nevertheless, our results show that borderline cognitions were also present in nonpatients and patients with CPD after the social stress induction. It therefore seems that patients with BPD are anticipating potential threat already before a confrontive social interaction, but that their reaction to an actual stressor does not differ from nonpatients and patients with CPD.

It has been suggested that patients with BPD show disturbances in social cognition only in situations that reflect BPD-relevant themes such as abandonment and rejection (Arntz and Ten Haaf, 2012; Jennings et al., 2012; Fonagy and Bateman, 2008). Accordingly, our stress protocol was intended to induce social stress and contained social judgment and elements of rejection. Indeed, the protocol evoked stronger emotions in the BPD group than in the control groups, and anecdotally, the majority of BPD patients noted during debriefing after the experiment that they feared being judged negatively and felt vulnerable. However, it remains unclear whether this stress protocol elicited BPD specific beliefs, or whether it also applies to everyday social stress. Therefore, it would be interesting to investigate whether the increase in BPD cognitions is the same across general stressful situations, or if this is different in situations that specifically trigger BPD themes.

We also observed a higher accuracy on the emotion recognition task (ERT) after stress induction compared to baseline. Although this improvement might indeed be caused by stress, practice effects cannot be excluded because there was no control condition. Importantly, no significant group differences were observed before or after stress induction. Previous studies that investigated emotion recognition in BPD have observed mixed results (see, e.g. the recent meta-analysis of Daros et al., 2013). It is possible that studies with positive results have used tasks that were more sensitive to detect differences between groups than the ERT. Nevertheless, the ERT has been used successfully to detect small differences between males and females and decrements in various psychiatric patient groups with similar sample sizes (Montagne et al., 2005, 2006). This suggests that the differences in facial emotion recognition in BPD are at least smaller than in those patient groups, which may also explain why previous BPD studies could have observed mixed results. Indeed, Daros et al. (2013) demonstrated that while patients with BPD performed worse than controls on the recognition of negative emotions, effect sizes were in the small to medium range, requiring larger sample sizes to demonstrate significant group differences. Furthermore, the most pronounced deficit was identified in recognizing neutral expressions, which we did not assess in the present

study. Although Daros et al. (2013) did not identify effects of comorbid depression on emotion recognition in BPD, our results do suggest that comorbid major depressive disorder (MDD) determines the impact of stress on facial emotion recognition performance. Whereas recognition accuracy was enhanced after stress in the BPD group without comorbid MDD, accuracy deteriorated in the BPD group with comorbid MDD. This suggests that impaired facial emotion recognition in BPD may be related to comorbid depression. However, these results are based on a small subsample of our initial BPD group and need to be replicated in larger samples. Regardless, these results suggest that depression comorbidity can contribute to the mixed results in studies on BPD and emotion recognition.

4.3. Strengths and weaknesses

This study has to be considered in the light of several strengths and weaknesses. In particular, this is the first study that investigated the consequences of stress on social cognition in BPD patients. In addition we examined whether the previously observed differences in physiological responses were specific for BPD by comparing the BPD group with the CPD group. A first limitation is that we did not exclude patients who were current smokers, used medication, or used hormonal contraceptives. Smoking, as well as female sex and hormonal contraceptives, are factors that are known to attenuate cortisol responses to stress. In fact, the TSST does not lead to increased cortisol levels in healthy smoking women, which may explain the small changes in cortisol levels in our study (Kirschbaum et al., 1999; Rohleder and Kirschbaum, 2006). However, excluding these participants would have reduced the number of eligible participants heavily and reduced the external validity of our results, as smoking and medication use is common in the BPD population. To diminish the potential influence of these factors we excluded patients using neuroleptics, instructed the participants to reduce smoking the day prior to the experiment, cease smoking two hours preceding the experiment, and stop sedative medication at least two days before the experiment. Importantly, there were no significant differences in the use of antidepressant medication between the patient groups. Moreover, we expect that the effects of medication on physiological variables were negligible because Nater et al. (2010) investigated medication free BPD patients using the TSST and reported comparable results. Furthermore, the percentage of hormonal contraceptive use was similar between groups, suggesting that any influence of hormonal contraceptives on cortisol responses is unlikely to explain differences between groups. Second, we did not investigate the influence of PTSD or dissociative symptoms, which have been suggested to influence emotional reactivity in BPD (Dixon-Gordon et al., 2013; Simeon et al., 2007). However, the proportion of patients with PTSD ($N=5$) or dissociative symptoms ($N=2$) in our study was small, suggesting that the influence of these symptoms on our results was presumably limited. Third, the experimental design of our study with testing before and after stress induction has particular strengths and weaknesses. This repeated measures design allows the investigation of differences at baseline before stress induction and thereby also to control for baseline

effects using a within-subject comparison, and minimizes the amount of patients that have to be included. But the absence of a non-stress control condition limits the interpretation of the main effects of stress due to potential test–retest effects, and certain parameters such as the facial emotion recognition task may not be optimal to detect subtle differences in such a design. Future studies may consider using a randomized design in which subjects are allocated to a stress or non-stress condition.

4.4. Conclusion

In conclusion, this study shows that patients with BPD experience increased negative emotions and are anticipating social threat even in neutral social situations. In response to a social stress test they report more negative subjective emotional reactions even though their physiological responses are attenuated. This result partially contradicts the theory of Linehan et al. (2007), which posits that BPD is characterized by increased sensitivity for social cues, enhanced reactivity to emotional stimuli and inability to regulate heightened physiological arousal with a slow return to baseline. Although the subjective emotional responses of the BPD group were largely in line with this theory, their physiological responses to stress were lower than those of the controls and even decreased further over time with respect to emotional baseline in comparison to non-patients. Furthermore, this study shows that patients with BPD develop more negative evaluations of the other in reaction to a confronting social stress situation, but to a similar extent as the control groups. Fonagy and Bateman (2008) suggested that social evaluation in BPD is particularly affected under the influence of emotions and has been associated with negative social evaluations. The results of our study provide partial confirmation for this idea by demonstrating that a social stress situation, in which people have to perform unexpectedly and are judged on their performance, increases “borderline views” of others (BPD specific cognitions such as unreliable, hostile). However, this tendency does not appear to be specific for patients with BPD, as the same holds true for patients with CPD and nonpatients.

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Conflict of interest

None declared.

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