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It is Time for Context – Determinants of the Negative Bias in Emotion Recognition in Borderline Personality Disorder

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We do not describe the world we see, we see the world we can describe

René Descartes

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LIST OF ABBREVATIONS

ACC	Anterior cingulate cortex
BA	Brodmann Area
BPD	Borderline Personality Disorder
BSL	Borderline Symptom List
CIMH	Central Institute of Mental Health
CPZ	Chlorpromazine
DBT	Dialectical behavior therapy
DERS	Difficulties in Emotion Regulation Scale
DSS	Dissociative Symptoms Scale
ERQ	Emotion Regulation Questionnaire
FEP	First episode psychosis
HC	Healthy controls
IAPS	International Affective Picture System
IFG	Inferior frontal gyrus
IPDE	International Personality Disorder Examination
LPS	Leistungsprüfsystem
NAcc	Nucleus Accumbens
PANAS	Positive and Negative Affect Schedule
PANSS	Positive and Negative Syndrome Scale
PFC	Prefrontal cortex
PTSD	Post-traumatic stress disorder
ROI	Region of interest
SAM	Self-Assessment-Manikin
SANS	Scale for the Assessment of Negative Symptoms
SCID	Structured Clinical Interview for DSM-IV
SPQ	Schizotypal Personality Questionnaire
STS	Superior temporal sulcus
SZ	Schizophrenia
TAS	Toronto Alexithymia Scale
WST	Wortschatztest

1 INTRODUCTION

Positive social interactions are a prerequisite for interpersonal closeness, trust and social support. A mental disorder that is explicitly characterized by impairments in building such social bonds is borderline personality disorder (BPD). Patients with BPD suffer from difficulties in maintaining stable relationships and from frequently occurring interpersonal conflicts that may arise from misinterpretations of social signals (Lazarus, Cheavens, Festa, & Rosenthal, 2014; Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). Studies on emotion recognition in BPD, however, revealed heterogeneous results, ranging from deficits to even higher sensitivity for emotions (Mitchell, Dickens, & Picchioni, 2014). Therefore, deepening the understanding of emotion recognition deficits and specific response patterns, like the so called “negative bias” in BPD, is a research topic of high importance and the primary aim of this dissertation.

Successful social interactions require a comprehensive understanding of the interaction partner including his or her emotional and mental states, wishes and intentions (Frith & Frith, 1999; Gallese, Keysers, & Rizzolatti, 2004). These states can become apparent via different channels and are based on various sources. It is known that a combination of different sources can facilitate the understanding, but if taken separately the highest informational value is provided undoubtedly by the human face (Paulmann & Pell, 2011). Even if no other information is available, the facial expression can provide a large amount of valuable information about a person’s feelings, intentions and relations to the interaction partner immediately (Erickson & Schulkin, 2003; Frith, 2009; Horstmann, 2003). To understand a facial expression and use this information in social interaction, a number of information processing steps have to be completed. These steps are collectively described as „social cognition“ (Brothers, 1990, 1996). In a variety of mental disorders, social-cognitive functions are known to be impaired (Happé & Frith, 2014; Rokita, Dauvermann, & Donohoe, 2018) with extensive negative consequences on the social integration of affected patients (Fett et al., 2011; Lazarus et al., 2014).

This dissertation aimed at identifying specific factors influencing disturbed emotion recognition from facial expressions in a prominent clinical example of impairments in social interaction and interpersonal functioning. We investigated the negative bias in BPD as well as in a clinical comparison group of schizophrenia

patients. We tried to identify neural correlates of context dependent alterations in emotion recognition in healthy participants to further elucidate the mechanisms of disturbed social functioning in patients. This doctoral thesis will first give a brief introduction on emotion recognition and will present an overview on findings regarding this process in BPD. The main part will present empirical data on deficits in emotion recognition, the specificity of these deficits in BPD and potential neural correlates, investigated within the scope of this dissertation. The dissertation will conclude with a general discussion of a model of negatively biased perception in BPD.

1.1 Emotion recognition

Facial emotion recognition is a crucial component of social-cognitive processes, because it is an important ability allowing insight in the feelings and intentions of others. The correct identification of emotions in facial expressions of other people is a prerequisite of adequate social interaction (Herba & Phillips, 2004; Quintana, Guastella, Outhred, Hickie, & Kemp, 2012). When emotion recognition abilities are impaired, misunderstandings and social conflicts may occur frequently with far-reaching consequences for social integration and quality of life (Dyck et al., 2009; Hofer et al., 2009; Renneberg et al., 2012).

The neural correlates of face processing have been studied extensively and a clear model exists that describes the relevant brain regions. Haxby and colleagues proposed that facial emotion recognition relies on a core and an extended system (Haxby, Hoffman, & Gobbini, 2000). According to this model the core system, including the fusiform gyrus and superior temporal sulcus (STS), plays an important role for identifying changeable features in a face and integrating different sources of information. The extended system includes – besides other regions – amygdala and insula which are reciprocally connected to the STS, and thus, enable the recognition of emotions in facial expressions (Haxby et al., 2000).

It is assumed that emotion recognition is primarily driven by perceptual (i.e. bottom-up) processing, although it seems to be continuously modulated by activation of prior experiences and knowledge in a top-down fashion as well as in an indirect manner via simulation (Adolphs, 2002). In other words, emotion processing represents a complex interplay between bottom-up stimulus driven and top-down modulated cognitive processes (Ochsner & Gross, 2014). Gilbert and Sigman state that no definable starting point of these interactions exists, since perception is a

result of resonance between feedforward and top-down processes (Gilbert & Sigman, 2007). Each state of brain function can provide a context and influences upcoming information processing. Top-down modulation is defined as any kind of influence on basic processes through more complex information, for example, prior expectations, priming, memories, hypothesis testing, internal states and also trait variables. In dependence on the actual sensory stimulus input and contextual cues, the interplay of higher and lower order structures is reciprocally changed and adapted (Gilbert & Sigman, 2007). The interactions with contextual cues are important since top-down processes guide the selection of relevant information and influence the convergence between expectation and sensory reality (Gilbert & Sigman, 2007). This flexible adaptation might fail in patients due to alterations in specific brain structures, their functioning and their connectivity (Gilbert & Sigman, 2007).

1.2 Emotion recognition in borderline personality disorder

BPD is a mental disorder that is accompanied by severe functional impairments, high rates of comorbidity, and a high degree of psychological strain and burden for the patients concerned. This leads to a high rate of suicide as well as high utilization of the health care system with significant costs for the society (Soeteman, Hakkaart-van Roijen, Verheul, & Busschbach, 2008). The disorder is characterized by profound affective instability, difficulties in impulse control and an instable self-image as well as interpersonal relationships. Patients show difficulties in emotion regulation and experience frequent problems in social interactions (Lieb et al., 2004).

BPD – besides autism and schizophrenia (Couture, Penn, & Roberts, 2006; Sasson et al., 2007) – is one of the mental disorders explicitly characterized by substantial social impairments and frequently occurring interpersonal conflicts (Herpertz, Jeung, Mancke, & Bertsch, 2014). BPD patients often feel rejected and suffer from the fear of being abandoned. They tend to interpret the reactions of others as hostile, rejecting and negative (Arntz & Veen, 2001; Barnow et al., 2009; Sieswerda, Barnow, Verheul, & Arntz, 2013). In the sense of a self-fulfilling prophecy, this leads to increased social impairments, resulting in social isolation, enhanced experience of loneliness, a reduced quality of life, and a low level of social functioning (for an overview: Lazarus et al., 2014). One explanation why BPD patients come to such negative evaluations of others' emotions and intentions and encounter these social impairments is a misattribution of social signals due to deficits in social-cognitive functions.

As mentioned above, a basic social-cognitive function is the identification of emotions in other people's faces, which allows to correctly infer mental states, intentions and a prediction of action tendencies. In BPD, previous research has revealed heterogeneous results regarding facial emotion recognition performance ranging from severe and global deficits to an in general heightened sensitivity (Lynch et al., 2006; Mitchell et al., 2014). However, some evidence points towards deficits only in specific emotional categories or even performance comparable with healthy people (Daros, Zakzanis, & Ruocco, 2013; Mitchell et al., 2014). Recent meta-analytic findings show a significant negative response bias for neutral and ambiguous facial expressions in BPD patients (Mitchell et al., 2014). Although there is clear evidence for a negative bias in emotion recognition in BPD, not all studies support its existence (e.g. Daros, Uliaszek, & Ruocco, 2014). Reasons could be differences in study design, small sample size (i.e., reduced statistical power) and varying participant characteristics – especially applied diagnostic criteria. Furthermore, the stimulus material, the specific task and task instructions varied highly between individual studies (Mitchell et al., 2014). However, there might be further causes for the fact that not all studies have found a negative bias in BPD. Heterogeneity in different disorder-specific traits or mood states is a conceivable factor of variance. Situational aspects that sometimes are related to but often irrelevant for the subsequent evaluation process may also play an important role. Study 1a and 1b of this dissertation aimed at specifying some of these factors possibly influencing the occurrence of a negative bias in BPD.

1.2.1 Factors potentially contributing to the occurrence of a negative bias

The severe emotional dysregulation in BPD is characterized by heightened sensitivity to emotional stimuli, intense reactions even to stimuli of low intensity and difficulties in returning to baseline level (Linehan, 1993b). This vulnerability may influence the adequate recognition of emotions and could result in biased response patterns leading to interpersonal misunderstandings and severe conflicts. In daily life, we are confronted with a great number of incoming emotional information, much of which is not relevant to us or the situations we are actually in. However, flexibly and quickly adapting to new incoming emotional information while disregarding other emotional information is crucial to navigate social interaction. Due to emotion regulation deficits BPD patients may have difficulties to suppress reactions to previous emotional information and, thus, be more susceptible to interfering

information. This specific relationship between emotion dysregulation and a negative bias in BPD is discussed in the following.

1.2.1.1 Neural correlates of emotion recognition and emotional dysregulation

On the neural level, emotion dysregulation is reflected by amygdala hyperactivation and reduced prefrontal control (Banks, Eddy, Angstadt, Nathan, & Phan, 2007). Evidence for an association between amygdala hyperactivation and emotion dysregulation has even been found in healthy individuals (Drabant, McRae, Manuck, Hariri, & Gross, 2009). The same association and reduced control of the prefrontal cortex (PFC) over the limbic system have been shown in various mental disorders (Etkin, Prater, Hoeft, Menon, & Schatzberg, 2010; Fan et al., 2013; Foland et al., 2008), including BPD (Johnson, Hurley, Benkelfat, Herpertz, & Taber, 2003; Leichsenring, Leibing, Kruse, New, & Leweke, 2011; Silbersweig et al., 2007). Interestingly, the pattern of aberrant brain activation during emotion recognition in BPD resembles to a great extent the pattern that usually characterizes emotion dysregulation. Facial emotion processing in BPD was frequently associated with hyperreactivity of the amygdala (Donegan et al., 2003; Koenigsberg et al., 2009; Mier et al., 2013; Minzenberg, Fan, New, Tang, & Siever, 2007) as well as hypoactivation in the inferior frontal gyrus (IFG) and STS during more complex social-cognitive tasks (Dziobek et al., 2011; Mier et al., 2013). These repeated observations of altered patterns of brain activation led to the assumption that enhanced emotional processing of social stimuli in patients with BPD may provide the neural basis of social-cognitive impairments (Mier et al., 2013). Amygdala hyperactivation in BPD was further shown to occur in concert with reduced prefrontal regulation, especially of the anterior cingulate cortex (ACC; Cullen et al., 2011; Minzenberg et al., 2007; Ruocco, Amirthavasagam, Choi-Kain, & McMain, 2013). Additionally, a recent meta-analysis points towards hyperactivation of the insula in reaction to negative stimuli in BPD, which was assumed to underlie the increased experiences of negative emotions (Ruocco et al., 2013). Since the insula serves as a relay between frontal and subcortical regions, its hyperactivation supports the idea of a fronto-limbic dysregulation especially for negative emotions in BPD (Ruocco et al., 2013). Assuming the involvement of similar brain regions as in the face perception model by Haxby and colleagues (2000), Mitchell and colleagues (2014) propose a neural model of impaired facial emotion recognition in BPD based on their meta-analytic findings. Accordingly, as in the model by Haxby and colleagues (2000), the fusiform

gyrus is proposed to receive input from the visual cortex, leading to stimulus-driven amygdala activation. Regular top-down modulation of the amygdala may be reduced, due to altered connectivity to areas, such as the ACC or the IFG, that modulate the processing of face information in the limbic system. This abnormal modulation of the amygdala results in altered processing of emotional stimuli and in consequence yields altered activation in regions like the STS. The result may be a heightened sensitivity to emotional cues, a negative bias, as well as delusions that may occur during stress (Mitchell et al., 2014).

Overall, these findings emphasize the importance of gaining more information on the role of emotion dysregulation for emotion recognition processes in BPD and suggest that understanding emotion recognition deficits on a brain system level may enhance our knowledge on social-cognitive deficits in BPD.

1.2.1.2 Alexithymia

An important concept that is clearly linked to emotion dysregulation and that may be also important for social-cognitive functions, like emotion recognition, is alexithymia (Domes, Grabe, Czieschnek, Heinrichs, & Herpertz, 2011; Erkic et al., 2018; Laloyaux, Fantini, Lemaire, Luminet, & Laroi, 2015). Alexithymia is characterized by difficulties in identifying and describing feelings and to separate those from bodily sensations. Further it is associated with a lack of the ability of imagination and an external oriented mode of thinking (Hendryx, Haviland, & Shaw, 1991). It was shown that alexithymia is related to a frequent use of maladaptive emotion regulation strategies in healthy individuals as well as in patient samples (Erkic et al., 2018; Taylor, 2000). Since BPD patients suffer from severe emotion dysregulation, it is not surprising that BPD patients score higher on alexithymia scales compared to healthy controls (New et al., 2012). Furthermore, higher scores of alexithymic traits were linked to lower emotion recognition accuracy in healthy individuals (Lane et al., 1996) and alexithymia was also previously associated with difficulties in emotion recognition in BPD (Domes et al., 2011). Given this overlap with emotion regulation difficulties and emotion recognition deficits, it seems important to account for alexithymia in the assessment of the negative bias in BPD patients.

1.2.1.3 Emotional context information and processing time

In case of disturbed emotion regulation abilities as observed in BPD patients, a current affective state may have a strong influence on emotion recognition and the

occurrence of a negative bias. Even in healthy participants affective states and emotion identification thresholds are correlated (Coupland et al., 2004). A comparable interference as elicited by the current affective state may also be induced via external emotional information that is not related to the emotional cue of interest. Mobbs and colleagues (2006) showed that face-related emotional context information preceding the presentation of facial expressions shifted the evaluation of identical faces. This seemed to be dependent on the valence of the emotional context information. Further studies showed that context information influences judgments of facial expressions in healthy individuals (Carroll & Russell, 1996; Righart & de Gelder, 2006) as well as in clinical samples (Hooker et al., 2011; Kim et al., 2011). Priming by emotional facial expressions for example led to more negative judgments of pleasantness for neutral faces in euthymic patients with bipolar disorder than in healthy controls (Kim et al., 2011). Negative affective priming through pictures from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2005) resulted in reduced trustworthiness ratings for faces in patients with schizophrenia compared to healthy controls (Hooker et al., 2011). These findings point towards a higher susceptibility for negative emotional information and a negative bias in schizophrenia as well as bipolar patients compared to controls. This sensitivity may also be present in BPD. In daily life, individuals are faced with a large amount of emotionally significant information arising from different sources and not necessarily related to each other. This may cause interference of prior with subsequent emotional information and may result in shifted evaluations.

As basis for this dissertation, we assume that a negative bias can be provoked or enhanced by task-irrelevant, interfering emotional information, especially in BPD patients. In other words, we hypothesize that a negative bias during emotion recognition occurs because an independent context factor, such as incoming but unrelated emotional information (may it be a thought, or an affect elicited by a preceding situation (Lemerise & Arsenio, 2000)), may not be properly differentiated from information which is the actual target of emotion recognition.

Furthermore, we argue that another important factor influencing correct emotion recognition is the available processing time. Longer processing time, or in an experimental setting prolonged presentation time of target emotions, may alleviate the abovementioned altered processes. In healthy individuals, a longer presentation time can increase recognition performance of emotional facial expressions (Esteves

& Öhman, 1993; Kirouac & Doré, 1984; Neath & Itier, 2014). However, presentation times in studies on emotion recognition in BPD vary a lot and the influence of these differences has not been evaluated systematically. Only one study examined the influence of processing time on emotion recognition in BPD, which showed that emotion discrimination under time pressure was associated with higher arousal and resulted in a greater amount of errors in patients with BPD compared to healthy controls. Especially neutral facial expressions were more often evaluated as negative, suggesting time constraints to be a relevant moderator of a negative bias in BPD (Dyck et al., 2009).

In summary, several internal and external influencing factors are possibly impairing correct evaluations of facial expressions in BPD. While several studies showed that emotional context information can impair emotion recognition, no comparable study investigated the association of emotion regulation abilities, external emotional information and emotion recognition in BPD compared to healthy controls and other clinical samples, when we started our research on the negative bias in BPD. The primary aim of this dissertation was to identify determinants of the negative bias in BPD by investigating the influence of interfering contextual information and restrictions of processing time and to examine associations with relevant psychopathological measures. An additional aim was to assess the specificity of these factors for the negative bias in BPD by comparison to a clinical control group of schizophrenia patients. To examine the neurobiological mechanisms that may be disturbed in patients with BPD, brain responses to the described determinants were measured in a healthy group of participants.

In the first study (study 1a) of this doctoral thesis we investigated a) the influence of previously presented task-irrelevant social emotional information, and b) time constraints on subsequent emotion recognition. Further, we examined c) whether impairments in emotion recognition can be explained by a negative bias in BPD patients, and d) whether a negative bias in BPD patients is associated with emotion dysregulation.

2 STUDY 1A: INVESTIGATING THE NEGATIVE BIAS IN BORDERLINE PERSONALITY DISORDER[1]

2.1 Emotion recognition in borderline personality disorder: effects of emotional information on negative bias

2.1.1 Abstract

Background: Borderline Personality Disorder (BPD) is characterized by severe deficits in social interactions, which might be linked to deficits in emotion recognition. Research on emotion recognition abilities in BPD revealed heterogeneous results, ranging from deficits to heightened sensitivity. The most stable findings point to an impairment in the evaluation of neutral facial expressions as neutral, as well as to a negative bias in emotion recognition; that is the tendency to attribute negative emotions to neutral expressions, or in a broader sense to report a more negative emotion category than depicted. However, it remains unclear which contextual factors influence the occurrence of this negative bias. Previous studies suggest that priming by preceding emotional information and also constrained processing time might augment the emotion recognition deficit in BPD.

Methods: To test these assumptions, 32 female BPD patients and 31 healthy females, matched for age and education, participated in an emotion recognition study, in which every facial expression was preceded by either a positive, neutral or negative scene. Furthermore, time constraints for processing were varied by presenting the facial expressions with short (100 ms) or long duration (up to 3000 ms) in two separate blocks.

Results: BPD patients showed a significant deficit in emotion recognition for neutral and positive facial expression, associated with a significant negative bias. In BPD patients, this emotion recognition deficit was differentially affected by preceding emotional information and time constraints, with a greater influence of emotional information during long face presentations and a greater influence of neutral information during short face presentations.

[1] Published paper: Fenske, S., Lis, S., Liebke, L., Niedtfeld, I., Kirsch, P., & Mier, D. (2015). Emotion recognition in borderline personality disorder: effects of emotional information on negative bias. *Borderline Personal Disord Emot Dysregul, 2*, 10. doi:10.1186/s40479-015-0031-z

Conclusions: Our results are in line with previous findings supporting the existence of a negative bias in emotion recognition in BPD patients and provide further insights into biased social perceptions in BPD patients.

2.1.2 Background

Borderline Personality Disorder (BPD) can be characterized by severe emotional dysregulation and affective instability (Schmahl et al., 2014). Patients suffering from BPD show a significant fear of being abandoned and pervasive problems in interpersonal relationships (Lieb et al., 2004; Lis & Bohus, 2013). One possible cause for these frequently occurring interpersonal conflicts might be a misattribution of social signals: Patients with BPD demonstrate a more negative and hostile perception of social relationships (Benjamin & Wonderlich, 1994), are characterized by an anxious attachment style (Meyer, Pilkonis, & Beevers, 2004), and judge others as more negative, rejecting and aggressive (e.g. Arntz & Veen, 2001; Barnow et al., 2009; see also Lazarus et al., 2014 for a review; Sieswerda et al., 2013). However, previous studies directly investigating emotion recognition in BPD provide heterogeneous results, ranging from deficits to a heightened sensitivity for emotional expressions (Daros et al., 2013; Lazarus et al., 2014). Hence, it can be assumed that the emotion recognition performance in BPD is subject to influencing factors, such as the emotional context (Mobbs et al., 2006).

Most of the early studies on emotion recognition in BPD reported deficits in emotion recognition, particularly in the identification of negative emotions (Bland, Williams, Scharer, & Manning, 2004; Guitart-Masip et al., 2009; Levine, Marziali, & Hood, 1997; Merkl et al., 2010; Unoka, Fogd, Fuzy, & Csukly, 2011). A recent meta-analysis however (Daros et al., 2013), reported that BPD patients show an overall deficit in recognition accuracy (when including all basic emotions and neutral expressions in the analysis). Furthermore, this meta-analysis suggests that BPD patients are not impaired in the recognition of all negative emotions, but have a specific deficit in the recognition of disgust and anger. However interestingly, the largest deficit was revealed for the identification of neutral facial expressions, suggesting that BPD patients tend to misattribute emotions to faces that do not convey emotional information. In line with this meta-analysis of Daros and colleagues (2013), another recent meta-analysis of Mitchell and colleagues (2014) supports the assumption of a negative bias in BPD; i.e. that patients with BPD demonstrate a tendency to attribute negative emotions to neutral facial expressions.

In agreement with the idea that patients with BPD do not show profound deficits in the recognition of negative emotions, but rather a negative bias, there are several studies reporting either no significant emotion recognition deficit in BPD at all (Matzke, Herpertz, Berger, Fleischer, & Domes, 2014; Mier et al., 2013) or a deficit that occurs only under specific conditions. In some of these studies, difficulties in emotion recognition were only revealed by low intensity levels of emotional expression (Robin et al., 2012), or when a fast discrimination was required (Dyck et al., 2009). Furthermore, there are studies that demonstrate higher accuracy in the classification of emotional expressions in BPD (Lynch et al., 2006; Wagner & Linehan, 1999). Wagner and Linehan (Wagner & Linehan, 1999) for example, reported a heightened sensitivity in the recognition of fearful facial expressions only, and Lynch and colleagues (Lynch et al., 2006) showed that BPD patients tend to identify happy and angry faces at an earlier level of intensity. For male faces with an angry expression this was also true in a study by Veague and Hooley (2014). In addition, there are findings explicitly pointing to a response bias in BPD patients favoring negative emotion categories when confronted with ambiguous or neutral facial expressions (Domes et al., 2008; Domes et al., 2011; Domes, Schulze, & Herpertz, 2009; Dyck et al., 2009; Veague & Hooley, 2014). For ambiguous expressions (morphing from one emotion to another), BPD patients had a response bias, favoring anger over disgust and happiness (Domes et al., 2008). Among the studies using continuously morphed pictures (morphing from neutral to a full emotional display), several found no differences in recognition threshold between groups (Domes et al., 2008; Domes et al., 2011; Matzke et al., 2014), but higher error rates for fear and surprise in one of the studies (Domes et al., 2011). In addition, Veague and Hooley (2014), found not only that patients with BPD had a higher sensitivity for male faces that displayed anger, but also a response bias for anger in neutral faces and morphed faces that contained no anger-cues (happy and fearful). In contrast to these studies pointing to a negative bias, a recent study by Daros and colleagues (2014) suggested that a misattribution of emotional states may be linked to both a misinterpretation as negative as well as positive valent emotional states; i.e. a general tendency to attribute emotions to neutral facial expressions. Taken together, albeit not all individual studies found a significant negative bias in BPD (e.g. Daros et al., 2014), recent meta-analytic evidence suggest a negative response bias to neutral and ambiguous expressions (Mitchell et al., 2014) that might be

pronounced for the misattribution of anger (Domes et al., 2008; Veague & Hooley, 2014). However, it is not clear why this deficit in the recognition of neutral as well as emotional facial expressions and especially the negative bias is not found consistently across studies.

One explanation is that when asking for basic emotions, a statistical bias for negative emotions is inherent. Another explanation is that emotion recognition performance in BPD is depending on the context and modulated by the prominent emotion regulation deficits in this patient group (Levine et al., 1997).

Patients with BPD are known to experience frequent states of negative emotions and aversive tension (Ebner-Priemer et al., 2007; Reisch, Ebner-Priemer, Tschacher, Bohus, & Linehan, 2008). This affective instability seems to arise from a high susceptibility for emotional information in combination with a severe emotion regulation deficit (Putnam & Silk, 2005). It was shown in healthy participants that negative affect biases the processing of emotional information (Coupland et al., 2004). Mobbs and colleagues (2006) showed that preceding emotional information shifted ratings for identical faces in the direction of the preceding emotional information (see also Carroll & Russell, 1996; Wallbott, 1988). Moreover, studies using emotional contextual information found that emotion recognition performance was biased by this contextual information (e.g. Mobbs et al., 2006; Righart & de Gelder, 2006). Interestingly, in a study with euthymic bipolar patients, it was shown that priming with emotional facial expressions resulted in a negative shift of pleasantness judgments for neutral target faces (Kim et al., 2011). Furthermore, Hooker and colleagues (Hooker et al., 2011) demonstrated that negative affective priming with pictures from the International Affective Picture System (Lang et al., 2005) led to lower trustworthiness ratings of faces in schizophrenia patients than in healthy controls, indicating a higher susceptibility for negative emotional information in this patient group (Hooker et al., 2011). Hence, there is considerable evidence for an influence of emotional information on emotion perception in healthy people, as well as in clinical samples.

However, to our knowledge - despite the vast evidence of emotion regulation deficits in patients with BPD - until now no comparable study exists that investigates the influence of emotional information on emotion recognition, and/or the association between emotion recognition and emotion regulation in BPD.

Another factor that affects emotion recognition performance is the available time to perceive and process the incoming information. Several authors showed that longer presentation times increased discrimination performance in healthy samples (e.g. (Esteves & Öhman, 1993; Kirouac & Doré, 1984; Neath & Itier, 2014). However, studies on emotion recognition in BPD differ in regard to the given time constraints. So far there are no studies that systematically investigated the effect of this factor. The first study emphasizing the important role of processing time for emotion recognition in BPD patients was conducted by Dyck and colleagues (2009). The authors demonstrated that fast emotion discrimination leads to higher arousal levels and more errors in emotion recognition in BPD patients than in healthy controls. In this case, particularly, neutral facial expressions were more often identified as negative.

Therefore, the aim of the present study was to investigate the influence of emotional information on emotion recognition performance in BPD. We hypothesized that (1) patients with BPD show a deficit in emotion recognition compared to healthy participants. We further assumed (2) that this deficit is augmented when facial expressions are preceded by emotional information (i.e. that patients with BPD perform worse than healthy control participants when the preceding information is arousing and has an emotional valence in comparison to emotionally neutral preceding information). Since it was shown that time pressure causes an increase in arousal levels and results in stronger negative bias in BPD (Dyck et al., 2009), the influence of emotional information on emotion recognition in BPD was assessed with and without time pressure. It was hypothesized that (3) restricted presentation time of the facial expression leads to a pronounced influence of the emotional information on emotion recognition. Moreover, for neutral facial expressions, we expected that (4) the emotion recognition deficit in BPD is due to a negative bias. Lastly, it was hypothesized that (5) the negative bias is associated with self-reported deficits in emotion regulation.

2.1.3 Methods

2.1.3.1 Sample

Before participating in the study, participants were informed about study procedures and gave written informed consent. The study was approved by the local Ethics Board of the Medical Faculty Mannheim, University of Heidelberg.

The sample consisted of 32 females with BPD and 31 healthy female controls (table 1). All patients met DSM-IV criteria for BPD (APA, 2000). 93.75% of them also had a comorbid psychiatric diagnosis, and 75% received psychotropic medication (see supplementary table S1 for percentages of specific diagnoses and medication). Diagnoses were made by experienced clinicians (psychologists or psychiatrists) at the Outpatient Unit of the Clinic for Psychosomatics and Psychotherapeutic Medicine at the Central Institute of Mental Health (CIMH) by means of a German version of the SCID-I interview (Wittchen, Wunderlich, Gruschwitz, & Zaudig, 1997), and the International Personality Disorder Examination (IPDE; Loranger et al., 1994). Patients with a comorbid diagnosis of schizophrenia, bipolar disorder, or addiction (currently or within the last 3 years), as well as with neurological diseases were excluded. Sixteen of the patients were inpatients. Healthy controls were recruited via local databases of the CIMH and participated in the SCID-I interview and completed the SCID-II questionnaire (Fydrich, Renneberg, Schmitz, & Wittchen, 1997) to exclude participants with current or life-time psychiatric diagnosis. Moreover, healthy participants were excluded when reporting a neurological disorder. General inclusion criteria were the ability to give written informed consent and sufficient command of the German language to understand task instructions and to complete the questionnaires.

After participating in the experiment, all participants completed several questionnaires. Severity of borderline symptoms and emotion regulation deficits were assessed with the Borderline Symptom List-23 (BSL-23; Bohus et al., 2009; Bohus et al., 2001) and the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The current affective state was assessed before and after the experiment with the Positive and Negative Affect Schedule (PANAS; Krohne, Egloff, Kohlmann, & Tausch, 1996; Watson, Clark, & Tellegen, 1988). Concerning the PANAS and BSL-23, data of two patients and one healthy control is missing. Further, data of the DERS is missing for three patients and one healthy control (see table 1 for group averages).

Table 1. Sample characteristics.

	BPD *N = 32*	HC *N = 31*	*p*
Mean age in years	30.35 *(8.22)*	29.84 *(7.70)*	0.838
Mean years of education	11.03 *(1.64)*	11.52 *(1.57)*	0.235
DERS sum score	128.10 *(24.38)*	59.77 *(11.86)*	<0.001
BSL-23 sum score	2.24 *(0.79)*	0.15 *(0.19)*	<0.001
PANAS_positive_pre	2.43 *(0.64)*	2.95 *(0.60)*	0.002
PANAS_positive_post	2.07 *(0.62)*	2.51 *(0.71)*	0.012
PANAS_negative_pre	1.97 *(0.73)*	1.07 *(0.08)*	<0.001
PANAS_negative_post	2.06 *(0.79)*	1.10 *(0.19)*	<0.001

Note. Means and standard deviations (in parentheses) of DERS = Difficulties in Emotion Regulation Scale, BSL-23 = Borderline Symptom List-23, PANAS = Positive and Negative Affect Schedule, positive_pre = positive affect before the experiment, positive_post = positive affect after the experiment, negative_pre = negative affect before the experiment, negative_post = negative affect after the experiment.

2.1.3.2 Emotion recognition task

An emotion recognition task was applied, in which each facial expression was preceded by a picture varying in valence and arousal. The preceding pictures were taken from the International Affective Picture System (IAPS; Lang et al., 2005). The IAPS pictures either showed a scene with positive valence and high arousal (e.g. depicting sport scenes), negative valence and high arousal (e.g. depicting crime scenes) or neutral valence and low arousal (e.g. depicting daily conversational situations). Importantly, we explicitly avoided the selection of pictures with a sexual theme for the positive IAPS category to prevent adverse responses in the BPD patients that are due to a history of sexual traumatization. Valences of the positive and negative pictures were matched to be equally distant from the neutral pictures (positive: *M* = 7.06, *SD* = 0.52; neutral: *M* = 5.02, *SD* = 0.36; negative: *M* = 3.02, *SD* = 0.45). Positive and negative pictures were also matched for arousal extent (positive: *M* = 5.98, *SD* = 0.52; negative: *M* = 5.99, *SD* = 0.47) while neutral pictures had a lower arousal (*M* = 3.09, *SD* = 0.37; see supplementary materials, table S2 for a list of all IAPS pictures that were presented in the course of the experiment). The facial stimuli were taken from the “NimStim Set of Facial Expressions” (Tottenham et al., 2009) and consisted of 5 male and 5 female actors. The faces showed an emotional (happy or angry) or neutral expression. To avoid ceiling effects in emotion

recognition performance, emotional facial expressions with reduced emotion intensity were applied (60% emotion, 40% neutral). The morphed facial expressions were taken from Matzke and colleagues (2014). Participants were instructed to look at all pictures, but to rate the valence of the facial expressions only, and not the valence of the scenes, by selecting one of three buttons (positive, neutral, negative) on a standard computer keyboard. We decided using only three emotion categories that were presented with equal probability to avoid a statistical bias for the selection of a negative emotion that is merely due to the presence of more negative categories; i.e. as it naturally occurs when using all basic emotions.

The task was applied in two blocks, differing in the presentation time of the facial expression. In both blocks, IAPS pictures were shown for 3 seconds and were immediately followed by a picture with a facial expression (figure 1). In one of the blocks, the facial expressions were presented until one of the response buttons was pressed, but for 3 seconds at most (“self-paced” condition). In the other block, presentation time was restricted to 100 milliseconds (“timed” condition). In both blocks, participants had up to 3 seconds to rate the valence of the emotion, and the facial expression was followed by a mask (a grey rectangle) for 500 milliseconds. Trial order was pseudo-randomized and block order was counterbalanced across participants. Each block consisted of 90 trials, i.e. 10 combinations of each IAPS category (positive, neutral, negative) with each face category (happy, neutral, angry) and took about 11 minutes. While completing the emotion recognition task, galvanic skin response and heart rates were recorded. The results from this psychophysiological assessment will be reported elsewhere.

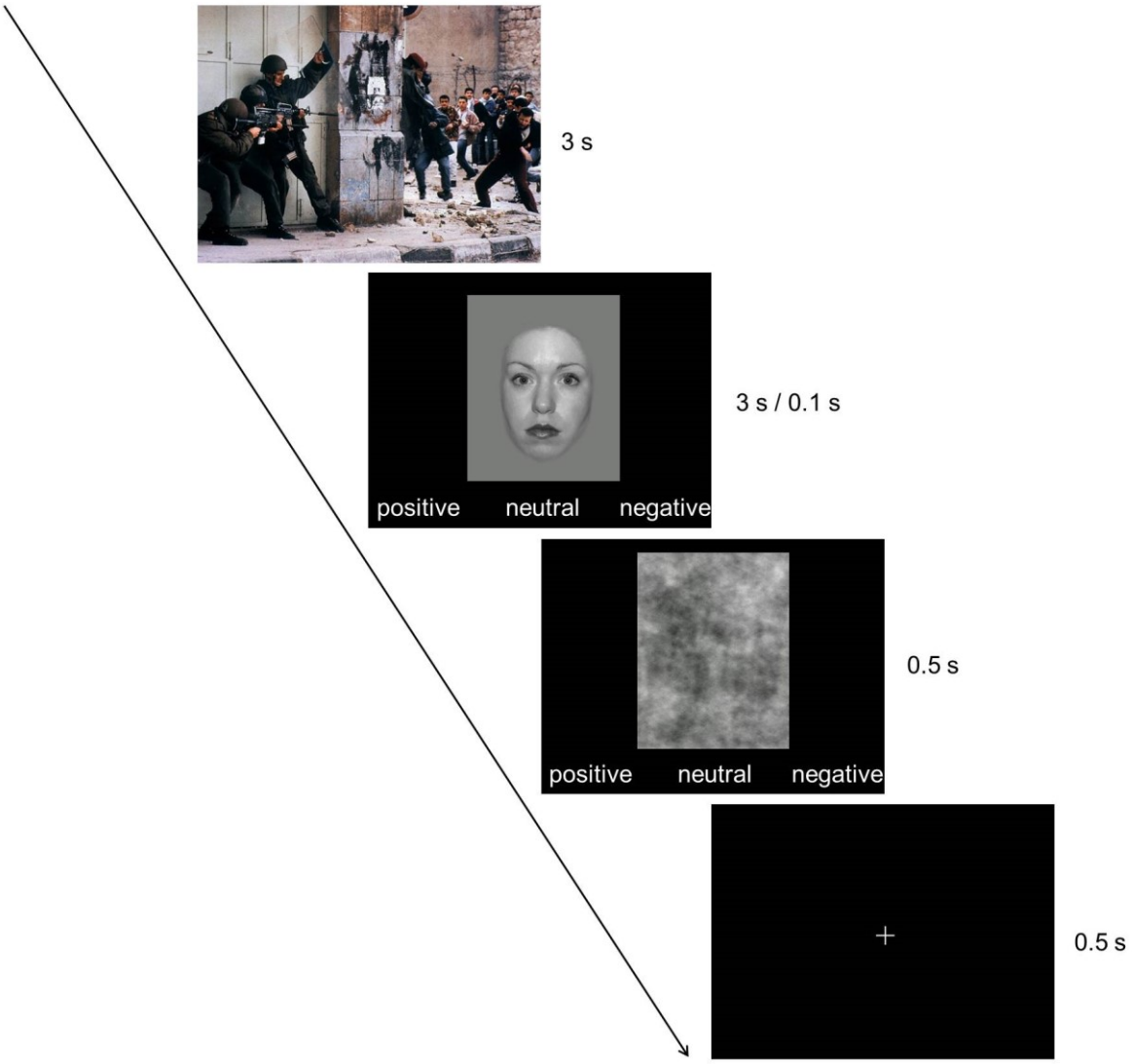

Figure 1. Experimental design with up to 3 seconds presentation of the facial expression in the “self-paced” condition and 100 milliseconds presentation of the facial expression in the “timed” condition.

2.1.3.3 Rating of experimental stimuli

Immediately after the emotion recognition task, the applied pictures were presented again for valence and arousal ratings. This additional evaluation of stimulus valence and arousal was completed to assess ratings without an influence of the experimental setup. For this purpose, faces and scenes were presented to the participants in two separate blocks, always starting with the faces block. Participants were asked to indicate the valence and arousal of each of the pictures using the Self-Assessment-Manikin (SAM; Hodes, Cook, & Lang, 1985; Lang, 1980) on a 5-point scale. This rating procedure was self-paced.

2.1.4 Results

Statistical analyses were performed with SPSS Statistics 21 (IBM Corporation, New York). Applying one-sample Kolmogorov-Smirnov-tests, no significant violations of the normal distribution were revealed (all *ps* > 0.11). In the case that Levene-tests for equality of variances revealed significant differences in variance between groups, the according *p*-statistics are reported with Greenhouse-Geisser correction. Effect sizes are specified as Cohen's *f* and *d*.

2.1.4.1 Emotion recognition task

Hypothesis 1

To investigate the first hypothesis, that patients with BPD show impaired emotion recognition performance, a 2 (group) x 3 (face valence) repeated measures ANOVA was conducted (table 2). There was a significant face valence x group interaction: Post hoc comparisons revealed that BPD patients identified both neutral and positive facial expressions less often correctly than healthy controls (neutral: $t(61) = 4.52$, $p < 0.001$, $d = 1.19$; positive: $t(61) = 2.80$, $p = 0.008$, $d = 0.79$), but not negative ones ($t(61) = 0.42$, $p = 0.678$, $d = 0.11$) (see figure 2). Due to the higher-order interaction effect, the interpretability of the main effect of group is restricted. However, there was also a main effect of face valence: Positive facial expressions were better recognized than neutral and negative facial expressions (neutral: $t(62) = 4.96$, $p < 0.001$, $d = 0.62$; negative: $t(62) = 9.86$, $p < 0.001$, $d = 1.24$). Further neutral facial expressions were more often recognized correctly than negative facial expressions ($t(62) = 4.19$, $p < 0.001$, $d = 0.53$).

Table 2. a) Statistical data of the group × face valence repeated measures ANOVA for emotion recognition performance, and b) descriptive values for the percentages of correctly recognized facial expressions.

a)

	df	*F*	*f*	*p*
Group	1,61	19.32	0.65	<0.001
Face valence	2,122	44.21	1.12	<0.001
Group x Face valence	2,122	4.27	0.27	0.023

b)

	BPD		HC	
Valence of facial expression	*M*	*SD*	*M*	*SD*
Positive	88.54	13.00	95.27	3.97
Neutral	78.28	13.44	90.32	6.74
Negative	73.85	12.26	75.00	9.30

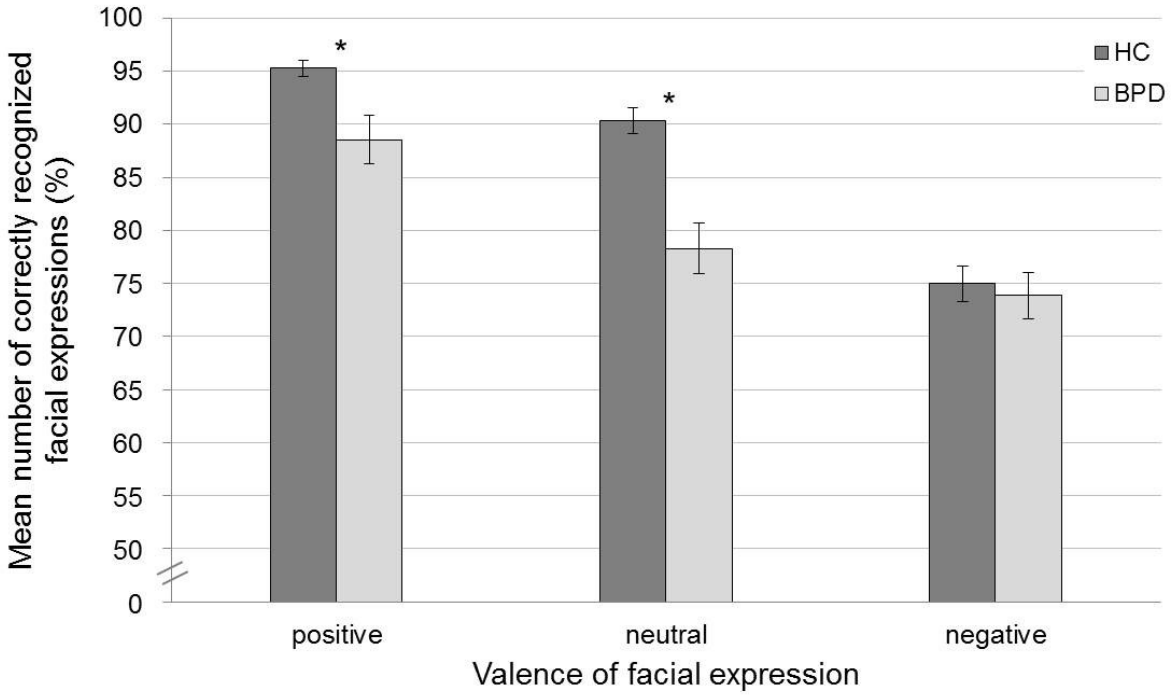

Figure 2. Mean numbers of correctly recognized facial expressions in percent correct, separated for group and face valence. Error bars display the standard errors, stars indicate significant group differences ($p < 0.05$).

Hypotheses 2 and 3

To further analyze whether the deficits in the perception of positive and neutral facial expressions are influenced by emotional information and time constraints, a group x face valence x IAPS valence x time repeated measures ANOVA was conducted (table 3). This analysis revealed a marginally significant four-way interaction, indicating that differences between groups were differentially affected for neutral and positive faces by the preceding IAPS picture and by the time constraints. Post-hoc comparisons to disentangle this interaction effect were conducted separately for the two presentation times, as well as for the comparison between the

presentation times. There was a stronger effect of preceding negative emotional information on the recognition of neutral versus positive facial expressions in the BPD group compared to healthy controls in the self-paced condition ($t(61)= -2.17$, $p = 0.034$, $d = -0.60$). Moreover, there was a trend for more incorrect responses for neutral compared to positive facial expressions in the BPD group compared to healthy controls when the facial expressions were preceded by positive emotional information in the self-paced condition ($t(61) = -1.70$, $p = 0.093$, $d = -0.44$). In the timed condition, there was a marginally significant higher error rate for neutral compared to positive facial expressions in the BPD group compared to the healthy controls when the preceding information was neutral ($t(61) = -1.99$, $p = 0.051$, $d = -0,54$). These difference values did not differ significantly between the two time conditions (figure 3). Due to the higher-order interaction effect, the interpretability of the main effects and lower-order interaction effects is restricted.

Table 3. a) Statistical data of the group × face valence x IAPS valence x time repeated measures ANOVA for emotion recognition performance, and b) descriptive values for the percentage of incorrectly recognized facial expressions, depending on the IAPS-category and the timing.

a)

	df	*F*	*f*	*p*
Group	1,61	18.80	0.64	<0.001
Face valence	1,61	26.49	0.79	<0.001
IAPS valence	2,122	3.54	0.25	0.032
Time	1,61	43.87	1.09	<0.001
Group x Face valence	1,61	3.32	0.24	0.073
Group x IAPS	2,122	0.59	0.1	0.557
Group x time	1,61	0.19	0.05	0.664
Face valence x IAPS valence	2,122	6.97	0.36	0.010
Face valence x time	1,61	1.02	0.13	0.317
IAPS valence x time	2,122	0.10	0.04	0.902
Group x face valence x IAPS valence	2,122	1.07	0.13	0.346
Group x face valence x time	1,61	0.053	0.03	0.819
Group x IAPS valence x time	2,122	0.32	0.07	0.727
Face valence x IAPS valence x time	2,122	2.35	0.20	0.099
Group x face valence x IAPS valence x time	2,122	2.49	0.21	0.087

b)

	BPD		HC	
Incorrect responses	*M*	*SD*	*M*	*SD*
Self-paced				
Positive IAPS				
Neutral face	15.31	16.06	5.81	9.58
Positive face	4.38	8.40	0.32	1.80
Neutral IAPS				
Neutral face	19.06	15.32	8.71	10.56
Positive face	7.19	15.08	0.65	2.50
Negative IAPS				
Neutral face	18.75	19.80	4.84	8.51
Positive face	6.88	13.06	1.29	4.28
Timed				
Positive IAPS				
Neutral face	20.94	19.73	11.61	9.69
Positive face	16.25	16.61	7.42	7.73
Neutral IAPS				
Neutral face	29.06	22.91	14.52	10.60
Positive face	11.88	14.47	8.06	9.46
Negative IAPS				
Neutral face	23.13	18.22	11.61	11.28
Positive face	17.50	19.84	9.68	11.40

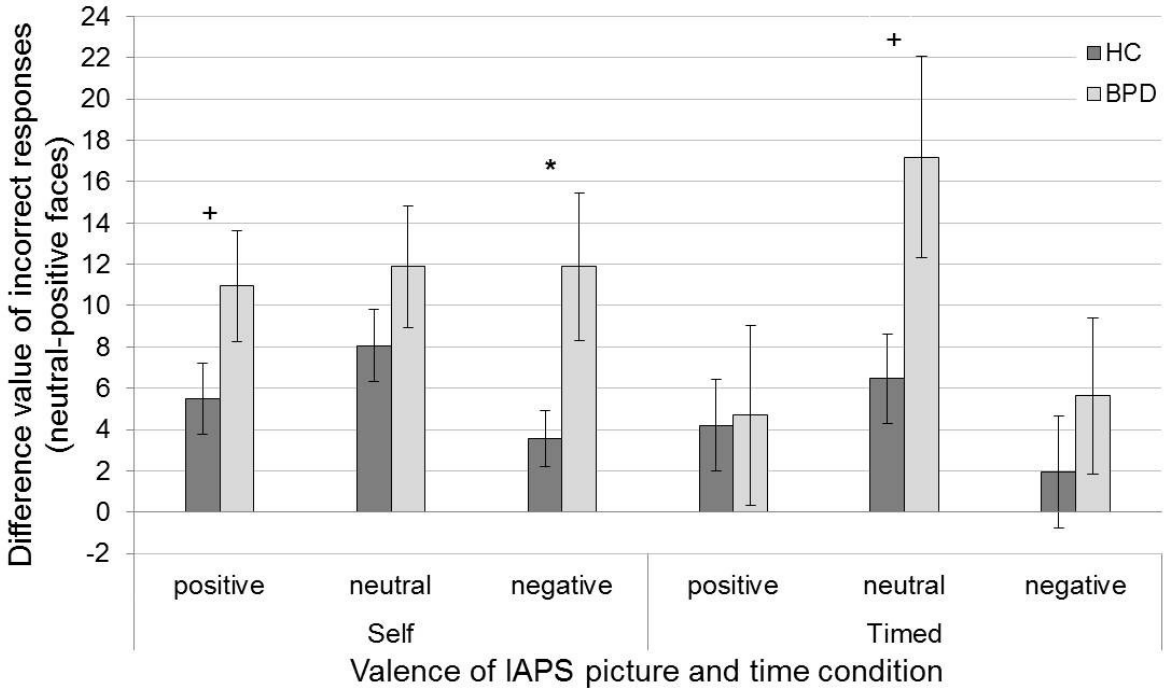

Figure 3. Difference values, showing percent of incorrectly recognized neutral minus incorrectly recognized positive facial expressions, separated for the preceding IAPS picture, time condition and group. Error bars display the standard errors, stars indicate significant group differences ($p < 0.05$), a plus indicates marginally significant group differences ($p < 0.1$).

Hypothesis 4

To examine whether the emotion recognition deficit for neutral facial expressions in the BPD patients was due to a negative bias, the incorrect answers in response to neutral facial expressions were sub-divided in negatively and positively biased responses, i.e. a misattribution of a positive or negative valence. A 2 (group) x 2 (bias valence) repeated measures ANOVA was conducted (table 4). There was a significant group x bias interaction: Post hoc comparisons revealed that BPD patients showed a stronger negative bias than healthy controls ($t(61) = -3.98$, $p < 0.001$, $d = -1.09$), while groups did not differ in the amount of positive bias ($t(61) = -0.882$, $p = 0.381$, $d = -0.22$) (see Figure 4). Due to this higher-order interaction effect, the interpretability of the main effects of group and bias valence is restricted.

Additional exploratory comparisons for the amount of negative bias between the three most common comorbidities in our BPD-sample, as well as between BPD-in- and BPD-outpatients were not significant (depression: $p = 0.615$, PTSD: $p = 0.700$, eating disorders, $p = 0.181$, inpatients: $p = 0.324$).

Table 4. a) Statistical data of the group × bias valence repeated measures ANOVA for the recognition of neutral facial expressions, and b) descriptive values for the percentages of biased responses.

a)

	df	*F*	*f*	*p*
Group	1,61	18.09	0.62	<0.001
Bias valence	1,61	22.39	0.71	<0.001
Group x Bias valence	1,61	11.17	0.47	0.001

b)

	BPD		HC	
Valence of bias	*M*	*SD*	*M*	*SD*
Positive	4.43	3.40	3.71	3.03
Negative	16.61	14.38	5.81	5.34

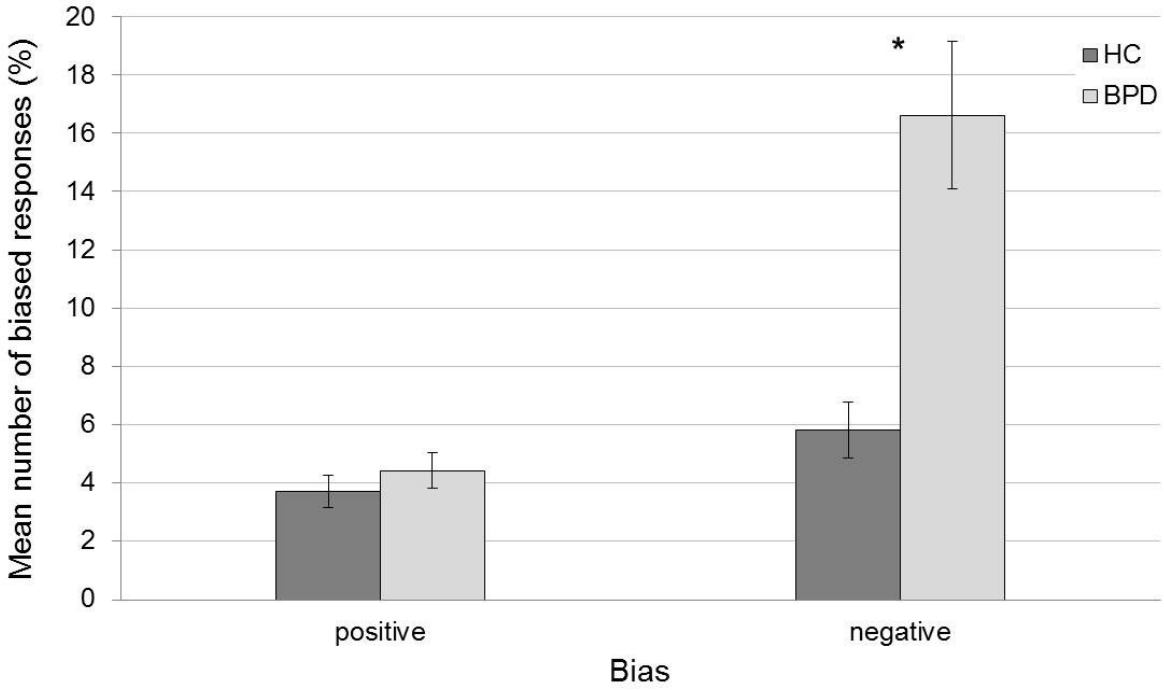

Figure 4. Bias x group interaction. Percentages of all responses to neutral facial expressions that were either positively or negatively biased, separated for the two groups. Error bars display the standard errors, stars indicate significant group differences ($p < 0.05$).

Hypothesis 5

To analyze hypothesis 5; i.e. the association between emotion regulation abilities and the negative bias, BSL- and DERS-scores were analyzed. Pearson

correlation coefficients of the questionnaire data and the amount of negative bias were calculated for all participants. There were significant correlations of the BSL-score (r = 0.545, p < 0.001) and the DERS-total score (r = 0.606, p < 0.001) with the negative bias across all participants. In the control group, BSL-scores correlated significantly with the amount of negative bias (r = 0.629, p < 0.001), while this correlation was not significant in the BPD group (r = 0.287, p = 0.125). Further, correlations were only trend-level significant in the control group between the negative bias and the DERS-sum score (r = 0.347, p = 0.060), while in the BPD group, there was a significant correlation between the negative bias and the DERS-sum score (r = 0.453, p = 0.014).

2.1.4.2 Ratings of the experimental stimuli

The analysis of the SAM ratings showed no group differences for valence ratings. All participants rated positive scenes and positive faces with higher and negative ones with lower valence than neutral pictures. Overall, negative IAPS pictures were rated with highest arousal. Positive IAPS pictures were rated with significantly lower arousal and neutral IAPS pictures with the lowest arousal. For neutral, negative and on a trend-level for positive facial expressions, higher arousal ratings were found in the BPD group. Arousal ratings were also higher in the BPD group than in the control group for the IAPS pictures. Arousal and valence ratings, as well as according analyses are reported in the supplementary materials.

2.1.5 Discussion

To investigate the influence of emotional information on emotion recognition in BPD, an emotion recognition task in which each facial expression was preceded by an IAPS picture, varying in valence and arousal, was applied. It was hypothesized that patients with BPD show an emotion recognition deficit and that this deficit is augmented when facial expressions are preceded by emotional information, and when processed under time constraints. Furthermore, it was assumed that the emotion recognition deficit for neutral faces in BPD patients is due to a negative bias, which in turn is associated with emotion regulation deficits.

In accordance to our hypothesis, BPD patients showed a clear emotion recognition deficit that was evident for neutral and positive facial expressions. Moreover, this deficit was accompanied by a negative bias in the perception of neutral faces. These results are in line with findings indicating that patients with BPD

have the most pronounced difficulties in the classification of neutral facial expressions (Daros et al., 2013), as well as a negative bias (Mitchell et al., 2014). Interestingly and in contrast to previously reported higher error rates for BPD patients in the identification of negative emotions (Bland et al., 2004; Guitart-Masip et al., 2009; Levine et al., 1997; Unoka et al., 2011), we found a comparable performance in the recognition of angry facial expressions as negative valent. Hence, our results suggest that BPD patients do not have a general emotion recognition deficit, but a deficit in the recognition of emotions without a negative valence. Alternatively, with regard to different negative emotions, especially the ability to recognize anger, might be spared in BPD. For example, Guitart-Masip and colleagues (2009) also showed no group differences for the recognition of angry facial expressions, as used in our paradigm as well, but for disgusted and fearful faces. From a neurobiological point of view, the often reported increased amygdala-activation in BPD patients in response to facial expressions (Donegan et al., 2003; Mier et al., 2013; Minzenberg et al., 2007) might elicit a higher vigilance per se and especially a higher vigilance for threatening information, making BPD patients even more sensitive for angry facial expressions. Hence, threatening information might be more salient and subjectively more likely to occur to patients with BPD, leading to a “more accurate” recognition when an angry face is presented, but to more false positive responses when an expression is not negative, particularly not angry (Unoka et al., 2011). This proneness to false positives might be enhanced by the severe emotion regulation deficits in BPD (Lieb et al., 2004; Linehan, 1993a). In BPD patients’ daily life, this might lead to the often occurring negative expectations concerning others (e.g. Arntz & Veen, 2001; Barnow et al., 2009; Sieswerda et al., 2013).

As mentioned before, on a neurobiological level, several authors (Donegan et al., 2003; Mier et al., 2013; Minzenberg et al., 2007) showed an enhanced amygdala-activation in patients with BPD in response to facial expressions. This limbic hyperactivation occurs in concert with deficits in the regulatory function of the prefrontal cortex (PFC, particularly the anterior cingulate cortex) (Minzenberg et al., 2007; Ruocco et al., 2013). Interestingly, a recent meta-analysis by Ruocco and colleagues (2013) additionally found evidence for insula hyperactivation in response to negative in comparison to neutral stimulus materials in BPD. The authors interpret this insula hyperactivation as possibly underlying the intensified subjective experience of negative emotions in BPD. With the insula as a connecting region

between frontal and subcortical brain regions, this hyperactivation also supports the assumption of impaired fronto-limbic regulation of negative emotions in BPD. To our knowledge, not explicitly in BPD (New et al., 2007), but in other disorders, this reduced control of the PFC over the limbic system has been repeatedly shown to be associated with deficits in emotion regulation (Etkin et al., 2010; Fan et al., 2013; Foland et al., 2008). In the context of social cognition, reduced control of the PFC over the amygdala might enhance the tendency to categorize neutral (and maybe also positive) stimuli as more negative (Domes et al., 2009). Hence, a deficit in emotion regulation should be associated with a more pronounced negative bias. Indeed, we found that the number of negatively biased responses was significantly correlated with emotion regulation deficits measured by the DERS across all participants, and within the BPD patient group. There was only a significant correlation with the strength of borderline symptoms measured by the borderline symptom list (BSL-23) in the healthy group, but not for the BPD patients. Accordingly, it can be concluded that deficits in emotion regulation are associated with the amount of negative bias in general, while the BSL (which serves as a more global measurement of emotion regulation deficits and borderline symptom severity) might only significantly explain variance below a specific cut-off (i.e. within a non-clinical range of occurrence).

In addition to deficits in emotion regulation, other factors might influence emotion recognition performance in BPD. Considering previous findings from studies investigating the influence of emotional context information (Carroll & Russell, 1996; Mobbs et al., 2006; Righart & de Gelder, 2006; Wallbott, 1988) and priming (Hooker et al., 2011; Kim et al., 2011) on emotion recognition, we hypothesized that emotional information that precedes emotion recognition should impair the performance in BPD patients. It was further assumed that time restriction should enhance this effect of emotional information in BPD patients. This assumption was based on the study by Dyck and colleagues (Dyck et al., 2009) who failed to show a general emotion recognition deficit in BPD, but reported a deficit in a fast emotion discrimination task. In particular, it was assumed that a brief presentation time of the faces forces more intuitive emotion recognition and in consequence might result in a stronger influence of the preceding IAPS picture.

We found a marginally significant interaction of IAPS valence, time condition, face valence and group. In the condition without time restriction, negative emotional

information was associated with more errors in the recognition of neutral facial expressions compared to positive expressions in the BPD group than in the control group. This was also (on a trend-level) true for positive emotional information. Hence, this provides first evidence for the assumption that emotional information influences emotion recognition performance in BPD to a higher extent than in healthy controls and that this is especially true for the recognition of neutral facial expressions. Interestingly, in the condition with time restriction, there were more errors for neutral in comparison to positive facial expressions in the BPD group compared to healthy controls when the preceding information was neutral. Thus, in the case of limited processing time of the facial information, especially neutral information seems to elicit false responses to neutral facial expressions. One explanation for that might be that neutral information is more ambiguous for patients with BPD, and in consequence is not perceived as neutral, especially when processed under time pressure. This perceived ambiguity could be augmented and results in misinterpretations when the target is also not showing an emotional valence. Considering that post hoc valence ratings of the IAPS pictures and the facial expressions did not differ significantly between the groups (see supplementary materials: rating of stimuli), it is remarkable that emotion recognition was more impaired and more negatively biased in the BPD group when it was combined with preceding information. Hence, it can be assumed that the experimental pairing of IAPS pictures with facial expressions fostered the emotion recognition deficit in BPD patients.

However, it has to be acknowledged that the four-way interaction including the IAPS valence and time constraints was only marginally significant. Not disregarding the reduced statistical power of this four-way ANOVA, an explanation might be the occurrence of carry over effects resulting from the pseudo-randomized presentation of the different emotional categories. Indeed, all participants showed less positive affect after the experimental task (see supplementary materials: affective state). In agreement with our assumption, there was a significant correlation between the increase of negative affect in the course of the experiment and the amount of negative bias (see supplementary materials: correlations of negative bias with affective state), which again emphasizes current mood as an influencing factor for emotion recognition. It is important to mention that the applied IAPS pictures were selected to be appropriate for a sample of female BPD patients. Hence, due to the high prevalence of sexual traumatization in BPD (Lobbestael, Arntz, & Bernstein,

2010; Zanarini, 2000), no pictures depicting sexual scenes were used for the IAPS category with positive valence. In consequence, to match for arousal in the positive and the negative category, only pictures of average arousal and thus also average valence levels could be applied in both categories, which might have reduced the influence of the preceding emotional information. Future studies might use a block wise presentation of the different preceding valences or a mood induction to investigate whether a stronger differential influence of emotional information is elicited when carry-over effects can be excluded. Furthermore, it would be interesting to disentangle stimulus valence and arousal to investigate the effect of these dimensions on emotion recognition in BPD. The dependency of valence and arousal in our study is due to the fact that they represent different parameters of motivational systems: While the valence dimension indicates which system is activated (appetitive or aversive), arousal shows to which degree the system is activated (Lang, Greenwald, Bradley, & Hamm, 1993). Hence, valence and arousal ratings are highly correlated for the IAPS pictures (Lang et al., 1993), and it would be interesting to develop novel paradigms with other stimulus materials / arousal induction methods that allow to investigate whether the activation of the aversive system or the degree of activation – independent of the system – is more important for social-cognitive performance in BPD.

A limitation of the current experimental design can be seen in the categorical response alternatives: False responses for positive facial expressions per se were negatively biased and for negative facial expressions per se were positively biased. Therefore, future studies might additionally include a response format that allows for shifting responses within one category by applying continuous response formats. Moreover, albeit we carefully matched the emotional IAPS pictures for the normative arousal levels provided with the IAPS database, participants in our study rated IAPS pictures with a negative valence with higher arousal levels than the ones with a positive valence. Hence, the potential influence of positive IAPS pictures was weaker than intended and has to be interpreted with care. However, Hooker and colleagues (Hooker et al., 2011) did not find a priming effect of positive IAPS pictures on trustworthiness ratings either, possibly suggesting a stronger influence of negative than positive emotional information on social cognition. Moreover, since the study was of an exploratory nature, to investigate the complex interaction between different emotional information, emotion recognition categories and processing time in BPD for

the first time, no experiment-wise error correction was applied. Thus, future studies are needed that replicate our findings, probably using paradigms with a more ecological experimental design.

2.1.6 Conclusions

In conclusion, the study replicated previous findings of an emotion recognition deficit for neutral and positive facial expressions in BPD patients. In addition, we could show a differential influence of valence of the preceding information and processing time: The emotion recognition deficit for neutral facial expressions was augmented in the BPD group when faces were presented after emotional information, when processing time of the preceding information was not restricted, and after neutral information, when processing time was restricted. While previous studies revealed heterogeneous results concerning the existence of a negative bias in emotion recognition in BPD, our findings provide clear evidence for a negative bias. We suggest that this negative bias in emotion recognition forms a basis for the more negative judgments of others in BPD (e.g. Arntz & Veen, 2001; Barnow et al., 2009; Sieswerda et al., 2013). Moreover, we propose that current mood states can influence the social perception of patients with BPD and with this might explain the misperceptions of social signals in social interactions. This negative bias can significantly impair the quality of social interactions and the stability of social bonds. Hence, psychotherapeutic interventions should focus on training patients with BPD in their ability to consciously perceive the influence of situational factors that could affect their current mood and arousal levels, and by this to enable them to reflect on the potential benevolence of interaction partners. Learning to differentiate between current feelings and newly incoming information could help BPD patients to establish more adequate interpretations and behavioral reactions in social interactions.

2.1.7 Supplementary materials

Table S1. Comorbid psychiatric diagnoses and psychotropic medication of BPD patients.

Comorbid psychiatric diagnosis	93.75%
Recurrent depressive disorder (of these remitted)	*66.7% (10.0%)*
Posttraumatic stress disorder	*46.7%*
Eating disorders	*43.3%*
Alcohol or Cannabis abuse	*20.0%*
Social phobia	*13.3%*
Attention deficit hyperactivity disorder	*10.0%*
Specific phobia	*6.7%*
Panic disorder	*6.7%*
Generalized anxiety disorder	*6.7%*
Adaptation disorder	*6.7%*
Dysthymia	*3.3%*
Obsessive-compulsive disorder	*3.3%*
Somatoform pain disorder	*3.3%*

Psychotropic medication	75.0%
Antidepressants	*91.7%*
Antipsychotics	*45.8%*
Sedativa	*20.8%*
Methylphenidate	*12.5%*
Anticonvulsants	*8.3%*
Anti-epileptics	*4.2%*
Opioid antagonists	*4.2%*

Note. Italic typed diagnoses and medication indicate the sub-proportion of patients having this specific diagnosis or taking this medication, of all patients having any comorbid diagnosis or taking any psychotropic medication.

Table S2. IAPS-codes for the pictures used as emotional information cues in the emotion recognition task.

Positive pictures	Neutral pictures	Negative pictures
1650	2393	2661
2303	2579	2683
5260	2870	2688
5480	5390	2691
5621	5731	3216
8031	7037	3500
8170	7038	6211
8260	7041	6213
8400	7234	8485
8500	9700	9925
2216	2191	1932
5622	2396	6244
5623	2440	6550
7501	2580	6821
7502	2595	6836
8034	2880	6940
8179	5120	8480
8191	5510	9050
8200	7000	9427
8496	7493	9520
2345	2038	5971
5626	2102	6250
5629	2235	6838
8210	2383	9160
8250	2480	9424
8300	5740	9429
8370	7034	9495
8467	7036	9621
8490	7130	9622
8499	7180	9630

2.1.7.1 Affective state

To explore the affective state before and after the experiment pre- and post-measurement ratings of the PANAS were analyzed by conducting a 2 (group) x 2 (time) x 2 (PANAS) repeated measures ANOVA. There was a significant group x PANAS interaction: BPD patients showed a lower positive affect ($t(61) = 3.22$, $p = 0.002$, $d = 0.82$), and a higher negative affect compared to healthy controls ($t(61) = -7.81$, $p < 0.001$, $d = -2{,}41$). Further there was a significant time x PANAS interaction: Post hoc comparisons revealed that participants had higher positive affect before the experiment (Pre: $M = 2.69$, $SD = 0.67$, Post: $M = 2.29$, $SD = 0.70$, $t(62) = 5.98$, $p < 0.001$, $d = 0.75$). Negative affect did not differ between the two assessment time points (Pre: $M = 1.53$, $SD = 0.69$, Post: $M = 1.59$, $SD = 0.75$, $t(62) = -0.89$, $p = 0.378$, $d = 0.11$). However, no group x time x PANAS interaction occurred, which indicates that patients with BPD were not more affected in their mood over the course of the experiment. Due to the higher-order interaction effects, the interpretability of the main effects is restricted (table S3).

Table S3. a) Statistical data of group × time x PANAS repeated measures ANOVA and b) descriptive values of the PANAS.

a)

	df	*F*	*f*	*p*
Group	1,61	5.61	0.32	0.021
Time	1,61	18.31	0.62	<0.001
PANAS	1,61	99.18	2.07	<0.001
Group x time	1,61	0.69	0.11	0.409
Group x PANAS	1,61	55.51	1.32	<0.001
Time x PANAS	1,61	17.73	0.61	<0.001
Group x time x PANAS	1,61	0.011	0.0	0.918

b)

	BPD		HC	
PANAS	*M*	*SD*	*M*	*SD*
Positive	2.25	0.56	2.73	0.61
Negative	2.02	0.67	1.08	0.10

2.1.7.2 Correlations of negative Bias with affective state

Pearson correlation coefficients of the current affective state and the amount of negative bias were calculated for all participants. There were significant correlations of the PANAS-score for negative affect before (r = 0.418, p = 0.001) and after the experiment with the negative bias (r = 0.673, p < 0.001), as well as of the difference between the time points (r = 0.397, p = 0.001), while there was no significant correlation of the negative bias and the PANAS-score for positive affect across the whole sample. In the BPD group there were significant correlations only for the PANAS-score for negative affect after the experiment (r = 0.609, p < 0.001) and for the difference between time points with the amount of negative bias (r = 0.486, p = 0.005). Interestingly, correlation analysis also revealed a negative significant correlation of the change in positive affect over the experiment with the amount of negative bias (r = -0.354, p = 0.047). In the control group there was only a significant correlation of the PANAS-score for negative affect before the experiment with the amount of negative bias (r = 0.413, p = 0.021).

2.1.7.3 Rating of stimuli

A 2 (group) x 3 (face valence) ANOVA for the face ratings revealed a highly significant main effect of face valence: As expected, positive facial expressions (M = 1.67, SD = 0.43) were rated with a higher valence than neutral (M = 3.07, SD = 0.35, t(62) = 25.03, p < 0.001, d = 3.13) and negative facial expressions (M = 4.13, SD = 0.43, t(62) = -34.81, p < 0.001, d = 4.39). Neutral facial expression had higher valence ratings than negative expressions (t(62) = -24.78, p < 0.001, d = 3.13). There was no significant main effect of group for the valence ratings of the facial expressions, as well as no significant interaction for group face valence (table S4).

Table S4. Statistical data of group x face valence repeated measures ANOVA.

	df	*F*	*f*	*p*
Group	1,61	0.16	0.0	0.901
Face valence	2,122	909.08	15.36	<0.001
Group x face valence	1,61	0.46	0.08	0.634

To explore arousal-ratings for the faces, a 2 (group) x 3 (face arousal) ANOVA was conducted. There was a significant main effect of group: BPD patients rated faces with a higher arousal than healthy controls (BPD: M = 2.52, SD = 0.61, HC: M

= 1.91, *SD* = 0.52; *t*(61) = -4.24, *p* < 0.001, *d* = 1.08). Moreover there was a significant group x face arousal interaction: Ratings for arousal were higher in the BPD group compared to the healthy controls for neutral (*t*(61) = -5.88, *p* < 0.001, *d* = 1.54), and negative faces (*t*(61) = -3.34, *p* = 0.001, *d* = 0.85), and on trend-level for positive faces (*t*(61) = -1.79, *p* = 0.078, *d* = 0.45). Due to this higher-order interaction effect, the interpretability of the main effect of face arousal is restricted (table S5).

Table S5. a) Statistical data of group x face arousal ANOVA and b) descriptive values of arousal ratings.

a)

	df	*F*	*f*	*p*
Group	1,61	17.99	0.62	<0.001
Face arousal	2,122	95.77	2.01	<0.001
Group x face arousal	2,122	4.02	0.27	0.023

b)

	BPD		HC	
Valence of facial expression	*M*	*SD*	*M*	*SD*
Positive	2.04	0.70	1.74	0.62
Neutral	2.17	0.65	1.41	0.33
Negative	3.34	0.88	2.58	0.91

For IAPS pictures, a 2 (group) x 3 (IAPS valence) ANOVA revealed a significant main effect of IAPS valence: Positive IAPS pictures were rated with a higher valence (*M* = 1.99, *SD* = 0.44) compared to neutral (*M* = 2.76, *SD* = 0.38; *t*(62) = 14.57, *p* < 0.001, *d* = 1.84) and negative IAPS pictures (*M* = 4.28, *SD* = 0.44; *t*(62) = -27.22, *p* < 0.001, *d* = 3.45), and neutral IAPS pictures with a higher valence than negatives ones (*t*(62) = -26.63, *p* < 0.001, *d* = 3,37). There was no significant main effect of group, as well as no significant interaction of group x IAPS valence (table S6).

Table S6. Statistical data of group x IAPS valence ANOVA.

	df	*F*	*f*	*p*
Group	1,61	0.58	0.08	0.450
IAPS valence	2,122	611.09	10.48	<0.001
Group x IAPS valence	2,122	0.72	0.03	0.931

To explore arousal-ratings for the IAPS pictures, a 2 (group) x 3 (IAPS arousal) ANOVA was conducted. There was a significant main effect of group with BPD patients having higher arousal ratings overall (BPD: $M = 2.67$, $SD = 0.68$; HC: $M = 2.30$, $SD = 0.64$; $t(61) = -2.21$, $p = 0.031$, $d = 0.56$), and a main effect of IAPS arousal: Negative IAPS scenes were rated with higher arousal ($M = 3.48$, $SD = 0.89$) compared to positive IAPS scenes ($M = 2.48$, $SD = 0.89$, $t(62) = -13.08$, $p < 0.001$, $d = 1.65$) and neutral IAPS scenes ($M = 1.52$, $SD = 0.57$, $t(62) = -19.58$, $p < 0.001$, $d = 2.47$). Positive IAPS scenes were rated with higher arousal than neutral IAPS scenes ($t(62) = -10.87$, $p < 0.001$, $d = 1.37$). No significant interaction for group x IAPS arousal occurred (table S7).

Table S7. Statistical data of group x IAPS arousal ANOVA.

	df	*F*	*f*	*p*
Group	1,61	4.88	0.29	0.031
IAPS arousal	2,122	246.69	4.52	<0.001
Group x IAPS arousal	2,122	0.72	0.03	0.931

3 STUDY 1B: SPECIFICITY OF THE NEGATIVE BIAS IN BORDERLINE PERSONALITY DISORDER

The results of the first study confirm the assumption of a negative bias in facial emotion recognition in BPD patients and provide some first evidence for its relationship with interfering emotional information as well as available processing time (Fenske et al., 2015). In Fenske and colleagues (2015), we conclude that this negative bias might cause impaired social interactions in BPD. Since reduced social functioning and interpersonal conflicts are also frequent in other mental disorders, the question arises whether this response pattern is specific for BPD. However, studies assessing the specificity of a negative bias in BPD are rare (e.g. Catalan et al., 2016; van Dijke, van 't Wout, Ford, & Aleman, 2016). One additional mental disorder – besides autism - that is well-known to be characterized by deficient social cognition is schizophrenia (Brüne, 2003; Green, Horan, & Lee, 2015; Pinkham, 2014). Deficits in social cognition have been constantly shown in these patients and are usually accompanied by reduced functional outcome and quality of life (Cohen, Forbes, Mann, & Blanchard, 2006; Couture et al., 2006; Hooker & Park, 2002; Sasson et al., 2007). In schizophrenia, social perception and social cognition were even found to predict social functioning to a higher degree than non-social cognition (Brüne, 2005; Green, Uhlhaas, & Coltheart, 2005), indicating the relevance of impaired social cognition in these patients, and making them a suitable comparison group to investigate the specificity of biased emotion recognition in BPD.

3.1 Schizophrenia as a clinical control group

Accordingly, the literature on facial emotion recognition in schizophrenia is vast and impairments are well-described (Kohler, Walker, Martin, Healey, & Moberg, 2010). Deficits were found across all emotional valences (Feingold et al., 2016; Morrison, Bellack, & Mueser, 1988) with the greatest degree of impairments in the recognition of negative emotions (Marwick & Hall, 2008), although some studies did not find reduced performance across all emotional categories (e.g. Weisgerber et al., 2015). However, differences in findings of emotion recognition deficits were associated with methodological variations between studies (Edwards, Jackson, & Pattison, 2002; Kohler et al., 2010; Mandal, Pandey, & Prasad, 1998). Meta-analytic findings point towards a task-independent deficit, but modulation by demographic

factors like age, sex, inpatient status, age of onset, number of positive and negative symptoms, and antipsychotic medication was observed (Kohler et al., 2010). As in BPD, it is likely that additional internal and external influence emotion recognition in schizophrenia. In BPD and in schizophrenia, disturbed affective processes are important factors of illness (Koenigsberg et al., 2002; Kring & Elis, 2013) and emotion dysregulation as well as alexithymia are apparent in both groups (Carpenter & Trull, 2013; Glenn & Klonsky, 2009; Lysaker et al., 2017; Moran, Culbreth, & Barch, 2018).

Another important aspect that suggests schizophrenia patients as a valuable clinical control group is the growing evidence for a negative bias in emotion recognition in both disorders (Kohler et al., 2003; Mier et al., 2014; Mitchell et al., 2014; Premkumar et al., 2008). This raises the question whether impairments and biased perceptions occur due to the same mechanism in both disorders, or have different sources of impairment. However, in schizophrenia findings of biased perception are mixed regarding its direction of effect, suggesting different mechanisms between the disorders. Daros and colleagues (2014) for example found differences in the direction of biased perception depending on the stage of illness and emotional valence of the target faces. Furthermore, in remitted patients with low levels of negative symptoms a negative bias for ambiguous stimuli (positive and negative pictures at one time point) and a positive bias in the recognition of neutral scene stimuli was found (Constant et al., 2011). A more recent study showed a generalized pattern of emotion recognition deficits and a higher tendency to misattribute emotions to neutral facial expressions in these patients that pointed to a general emotional bias in schizophrenia (Romero-Ferreiro et al., 2016).

Neuroimaging studies revealed abnormalities in structure and functioning of the amygdala with a hyperactivation in reaction to neutral facial expressions (Marwick & Hall, 2008) as well as a hyperactivation in the STS in schizophrenia (Mier et al., 2010), while other results are inconsistent (Li, Chan, McAlonan, & Gong, 2010). Activation in these regions was also altered in BPD patients in response to neutral facial expressions (Mier et al., 2013; Mitchell et al., 2014).

Only a few recent studies compared emotion recognition performance and error patterns directly between patients with BPD and schizophrenia. Van Dijke and colleagues (2016) showed that patients with schizophrenia and patients with BPD both are impaired in emotion recognition, but differ regarding the type of errors made in affect recognition. BPD patients tended to label neutral expressions more often as

fearful compared to schizophrenia patients and healthy controls, whereas schizophrenia patients misattributed neutral facial expressions more often as happy compared to BPD patients (van Dijke et al., 2016). Furthermore, Andreou and colleagues (2015) state that different mentalizing errors are characteristic for each of the disorders and that these errors are dependent on different predictors. Over-mentalizing was more characteristic for BPD patients, while schizophrenia patients exhibited more under-mentalizing errors (Andreou et al., 2015).

Catalan and colleagues (2016) found differences in the recognition of neutral faces between first episode psychosis (FEP) and BPD patients as well as healthy controls in a task with angry, happy, fearful and neutral facial expressions. Both patient groups had a deficit in the recognition of neutral faces. FEP patients further performed worse in the recognition of anger. Both patient groups more often attributed negative emotions to happy faces and BPD patients also tended to interpret neutral facial expressions as negative, supporting the assumption of a negative bias. Interestingly, FEP patients recognized fearful expressions more often as neutral or happy (Catalan et al., 2016), which leads to the hypothesis that these patients do not show specifically negative biased perceptions. This is in line with findings of a generalized pattern of emotion recognition deficits and a higher tendency to misattribute emotions to neutral facial expressions in these patients, which points to a general emotional but not necessarily to a negative bias (Romero-Ferreiro et al., 2016).

To assess the specificity of emotion recognition deficits as well as a negative bias after previously presented emotional information in BPD patients, we included an extended sample with schizophrenia patients as a clinical control group in study 1b.

3.2 Sex effects

The pattern of impairments however, could not only be specific for disorder but also for other interindividual factors, such as sex. Hall and Matsumoto (2004) for example found differences in emotion recognition based on sex in healthy participants. They showed that women performed better even when little information about the stimuli was available. Furthermore, women showed more intense ratings than men (Hall & Matsumoto, 2004). Another investigation of sex differences in emotion recognition revealed less accurate emotion labeling and lower sensitivity in men compared to women in a student sample (Montagne, Kessels, Frigerio, de

Haan, & Perrett, 2005). Meta-analytic findings also point towards advantages in emotion recognition for women (McClure, 2000). This was also replicated in a more recent study (Schmid & Schmid Mast, 2010). In a meta-analysis of neuroimaging findings, there was no evidence for higher activation in response to emotional stimuli in women compared to men, but men showed higher lateralization of activation (Wager, Phan, Liberzon, & Taylor, 2003). However, Li and colleagues (2008) found that women demonstrated a greater sensitivity to negative stimuli which had a low salience compared to men. As a neural correlate for that, the right prefrontal cortex was revealed and discussed (Li et al., 2008).

With a lifetime risk of about 1%, prevalence rates are comparable between BPD and schizophrenia (Paris, 2018). In both disorders, women and men are equally often affected (Coid, Yang, Tyrer, Roberts, & Ullrich, 2006; DGPPN, 2006; Lenzenweger, Lane, Loranger, & Kessler, 2007). Sex differences are present regarding age of onset in schizophrenia with an earlier onset in men (Stilo & Murray, 2010). In BPD, a difference occurs regarding use of treatment since a majority of patients in clinical settings are female (Gunderson & Links, 2014). These factors may influence the female to male ratio of samples and, thus, may influence findings.

In schizophrenia, there is also evidence for sex differences in overall emotion recognition performance (Vaskinn et al., 2007) as well as for differences in the patterns of biased perceptions (Weiss et al., 2007). Male patients with schizophrenia labeled neutral facial expressions more often as angry while female patients recognized the same stimuli more often as sad. Furthermore, both sexes performed better in recognizing fear in same sex faces and anger in faces of the opposite sex (Weiss et al., 2007). The authors state that malevolent ascriptions to neutral facial expressions in male patients may contribute to sex differences in aggressive behavior in schizophrenia patients (Weiss et al., 2007). Based on these findings, sex effects may also occur in our paradigm (Fenske et al., 2015) in schizophrenia patients.

Research on sex effects in emotion recognition and especially on the negative bias in BPD is completely missing. Studies investigating emotion recognition in BPD mostly focused on female or sampled very small numbers of males, resulting in underpowered studies to investigate sex differences (Daros et al., 2013; Domes et al., 2008; Unoka et al., 2011).

In this thesis, specificity of disturbed emotion recognition and the negative bias in BPD was assessed by a) adding patients with schizophrenia to the initial sample. Since we concentrated only on female participants in study 1a b) male participants were recruited for all groups to allow the investigation of sex-specific effects. In addition, c) associations between psychopathology, such as emotion dysregulation and in schizophrenia positive and negative symptomatology and biases in emotion recognition were investigated.

3.3 Methods

3.3.1 Sample

The extended sample consisted of 105 participants with 35 in each group. For BPD patients and healthy controls, the recruitment was identical to study 1a with diagnostic procedures and inclusion- and exclusion criteria being the same. Patients with schizophrenia were recruited from the inpatient and the outpatient units of the Clinic for Psychiatry at the CIMH. Diagnoses were made by experienced clinicians (psychologists or psychiatrists) at the CIMH who were not involved in the study, according to ICD-10-GM (Graubner, 2013). Before participating in the study, participants completed the SCID-II questionnaire (Fydrich et al., 1997) and the follow-up interview was conducted when indicated. Patients with comorbid diagnoses of a personality disorder, bipolar disorder or substance use disorder (currently or within the last 3 years) and neurological diseases were excluded. The current symptomatology of schizophrenia patients was assessed by the Positive and Negative Syndrome Scale (PANSS; Kay, Opler, & Fiszbein, 2000) and the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1989) prior to the experiment. Borderline symptomatology was assessed with the BSL-23 (Bohus et al., 2007; Bohus et al., 2001). Emotion dysregulation was measured using the DERS (Gratz & Roemer, 2004) and alexithymia was assessed with the Toronto Alexithymia Scale-20 (TAS-20; Bach, Bach, de Zwaan, Serim, & Böhmer, 1996). Sample characteristics are presented in table B1.

Table B1. Sample characteristics.

	HC	BPD	SZ	*F*	*p*
female/male	17/18	20/15	16/19	0.98[1]	0.612
age	32.09 (8.76)	32.26 (6.99)	32.51 (9.26)	0.02	0.977
education	11.34 (1.55)	10.89 (1.76)	11.14 (1.63)	0.68	0.511
in-/out-patient	-	22/13	20/15	0.24[2]	0.404
BSL-23	0.13 (0.17)	1.99 (0.82)	0.56 (0.55)	99.86	<0.001
DERS sum	60.91 (11.00)	126.09 (22.80)	85.00 (19.00)	101.23	<0.001
TAS-20	1.99 (0.37)	3.14 (0.55)	2.54 (0.52)	49.15	<0.001
PANSS-pos			12.52 (4.24)		
PANSS-neg			18.79 (5.05)		
PANSS-GPP			32.82 (6.40)		
SANS			38.82 (17.29)		
PANAS					
pos_pre	2.83 (0.58)	2.64 (0.70)	2.66 (0.78)		
pos_post	2.60 (0.56)	2.19 (0.69)	2.40 (0.80)		
neg_pre	1.09 (0.16)	1.79 (0.61)	1.48 (0.60)		
neg_post	1.12 (0.18)	2.00 (0.81)	1.49 (0.48)		
DSS pre	0.08 (0.19)	1.26 (1.29)	0.84 (0.91)		
DSS post	0.08 (0.25)	1.68 (1.45)	0.67 (0.96)		
Arousal pre	1.50 (0.62)	2.54 (0.78)	2.34 (1.00)		
Arousal post	1.76 (0.65)	2.77 (0.97)	2.49 (0.98)		

Note. HC = healthy controls, BPD = borderline personality disorder, SZ = schizophrenia, BSL-23 = Borderline Symptom List-23, DERS sum = sum score of the Difficulties in Emotion Regulation Scale, TAS-20 = 20-item Toronto Alexithymia Scale, PANSS-pos = positive scale of the Positive and Negative Syndrome Scale, PANSS-neg = negative scale of the Positive and Negative Syndrome Scale, PANSS-GPP = general psychopathology scale of the Positive and Negative Syndrome Scale, SANS = Scale for the Assessment of Negative Symptoms, PANAS = Positive and Negative Affect Schedule (pos = positive affect, neg = negative affect), DSS = Dissociative Symptoms Scale, Arousal = self-reported arousal in the Self-Assessment-Manikin, pre = before the experiment, post = after the experiment.
[1]Chi-square score (Kruskal-Wallis-Test), [2]Chi-square score (Chi-square-Test).

Schizophrenia patients were diagnosed with paranoid schizophrenia (F20.0; *n* = 31; 88.6%), disorganized schizophrenia (F20.1; *n* = 3; 8.6%) or catatonic schizophrenia (F20.2; *n* = 1; 2.8%). BPD patients were diagnosed with emotionally unstable personality disorder, either borderline type (F60.31; *n* = 30; 85.7%) or

impulsive type (F60.30; *n* = 2; 5.7%) or mixed personality disorder with clear presence and dominance of emotionally unstable criteria (F61; *n* = 3; 8.6%). BPD patients further met criteria for two comorbid diagnoses on average while schizophrenia showed fewer comorbidities (table B2).

Table B2. Comorbid psychiatric diagnoses in the patients.

Comorbid psychiatric diagnosis	*N* total	BPD	SZ
(Recurrent) depressive disorder	24	19	5
(of these remitted)	(3)	(3)	(0)
Anxiety disorder	11	10	1
Posttraumatic stress disorder	11	11	0
Eating disorder	9	9	0
Attention-deficit hyperactivity disorder	6	6	0
Alcohol or cannabis abuse/dependence	10	2[1]/2[2]	6[2]
Other disorder	10	7	3

Note. N total = total number of cases, BPD = borderline personality disorder, SZ = schizophrenia.
[1]cannabis or alcohol dependence, currently abstinent since ≥ 3 years, [2] cannabis or alcohol abuse, abstinent at least since beginning of treatment.

Psychotropic medication of the patient samples is presented in table B3. Chlorpromazine (CPZ) equivalent scores were calculated according to Andreasen and colleagues (2010) and Gardner and colleagues (2010). 25.7% of the BPD patients had antipsychotic medication (CPZ: *M* = 111.78 mg, *SD* = 67.02 mg), and 82.9% of the schizophrenia patients (CPZ: *M* = 505.28 mg, *SD* = 242.20 mg).

Table B3. Psychotropic medication of the patient samples.

Psychotropic medication	*N* total	BPD	SZ
Antipsychotics	42	11	31
Antidepressants	29	21	8
Antiepileptics	8	2	6
Methylphenidate	4	4	0
Sedativa[1]	4	1	3

Note. N total = total number of cases, BPD = borderline personality disorder, SZ = schizophrenia.
[1]patients had the last intake at least one day before participation.

3.3.2 Data collection and analysis

The experimental procedure was identical with study 1a (for a description of the task see 2.1.3.2). Statistical analyses were performed with SPSS Statistics 25 (IBM Corporation, New York).

3.4 Results

3.4.1 Emotional context and time pressure

To disentangle whether the patient groups show an emotion recognition deficit compared to healthy controls and whether this deficit is restricted to or pronounced in specific conditions, a 3 (group) x 2 (timing) x 3 (IAPS valence) x 3 (Face valence) repeated measures ANOVA was conducted (table B4). The analysis revealed a main effect of group. Bonferroni-adjusted post-hoc comparisons revealed that healthy controls showed the best overall performance (M = 85.21%, SD = 4.54%) and performed significantly better than BPD patients (M = 79.94%, SD = 6.81%, p = 0.016) and schizophrenia patients (M = 75.44%, SD = 10.67%, p < 0.001). BPD patients performed marginally better than schizophrenia patients (p = 0.052).

Table B4. Statistical data of the group x timing x IAPS valence x face valence repeated measures ANOVA for emotion recognition performance.

	df	*F*	η^2_p	*p*
Group	**2,102**	**13.86**	**0.214**	**<0.001**
Timing	**1,102**	**263.29**	**0.721**	**<0.001**
IAPS valence	**2,204**	**4.63**	**0.043**	**0.011**
Face valence	**2,204**	**53.68**	**0.345**	**<0.001**
Group x Timing	2,103	0.56	0.011	0.556
Group x IAPS valence	4,204	0.61	0.012	0.656
Group x Face valence	**4,204**	**3.61**	**0.066**	**0.007**
Timing x IAPS valence	2,204	2.01	0.019	0.137
Timing x Face valence	**2,204**	**60.64**	**0.373**	**<0.001**
IAPS x Face valence	**4,408**	**5.45**	**0.051**	**<0.001**
Group x IAPS valence x Face valence	8,408	0.21	0.004	0.989
Group x IAPS valence x Timing	4,204	1.41	0.027	0.233
Group x Face valence x Timing	4,204	0.49	0.009	0.746
IAPS valence x Face valence x Timing	4,408	2.12	0.020	0.077
Group x IAPS valence x Face valence x Timing	8,408	0.76	0.015	0.639

Note. Significant effects are displayed in bold.

There was a significant group by face valence interaction (figure B1). Bonferroni-adjusted post-hoc comparisons revealed that both patient groups performed worse than healthy controls for neutral and positive facial expressions. BPD patients showed significantly more correct responses for positive and marginally significant for negative expressions compared to schizophrenia patients (table B5). Despite these group differences regarding face valence, positive faces were recognized significantly better than neutral ($p \leq 0.014$) and negative expressions ($p < 0.001$) in all groups. In healthy controls, neutral expressions were also identified better than negative expressions ($p < 0.001$) while in both patient groups performance between neutral and negative expressions did not differ significantly ($p \geq 0.205$).

Table B5. Post-hoc pairwise comparisons for the interaction effect of group x face valence.

Facial expression	groups	Mean differences	Standard error	p_{adj}	95%-CI
Positive	HC vs. BPD	0.57	0.24	0.059	-0.02 – 1.16
	HC vs. SZ	**1.28**	**0.24**	**<0.001**	**0.69 – 1.87**
	BPD vs. SZ	**-0.71**	**0.24**	**0.012**	**-1.30 – -0.12**
Neutral	**HC vs. BPD**	**1.26**	**0.38**	**0.004**	**0.33 – 2.18**
	HC vs. SZ	**1.23**	**0.38**	**0.005**	**0.31 – 2.16**
	BPD vs. SZ	0.02	0.38	1.000	-0.90 – 0.95
Negative	HC vs. BPD	-0.25	0.28	1.000	-0.94 – 0.44
	HC vs. SZ	0.41	0.28	0.444	-0.28 – 1.11
	BPD vs. SZ	-0.66	0.28	0.066	-1.35 – 0.03

Note. Significant effects are displayed in bold. adj = Bonferroni-adjusted. HC = healthy controls, BPD = borderline personality disorder, SZ = schizophrenia.

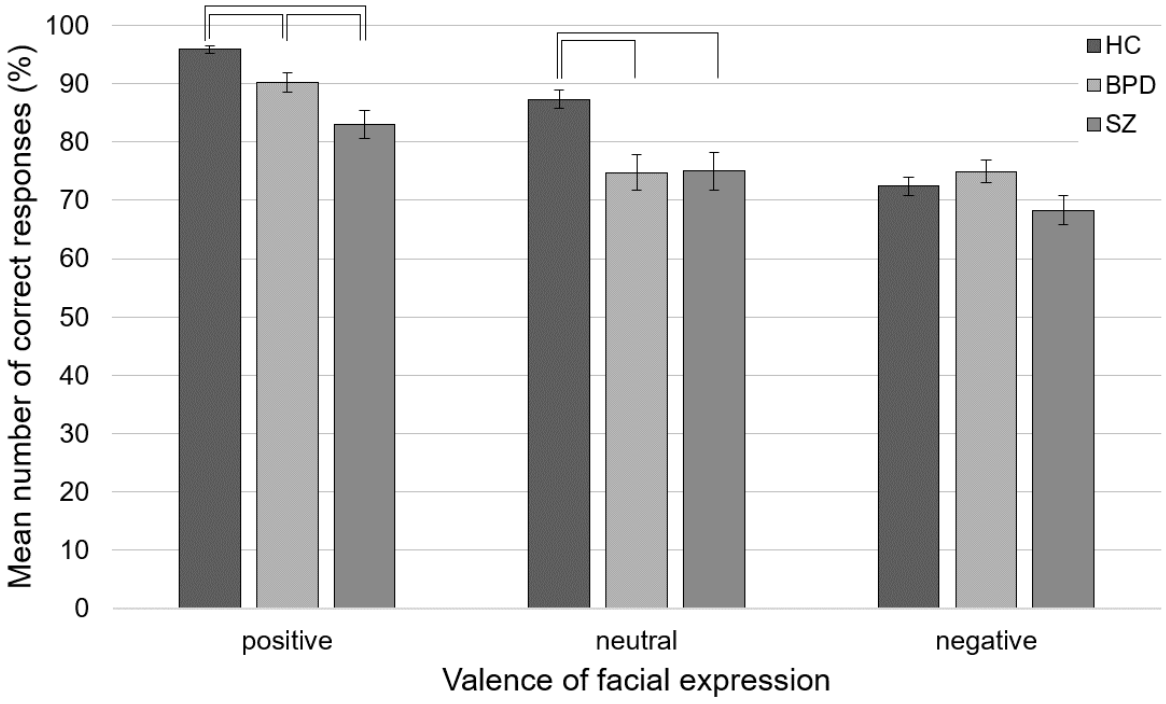

Figure B1. Interaction effect of group x face valence. Number of correctly identified facial expressions in percent separated by groups and face valences. Error bars display standard errors, parentheses indicate significant differences between groups ($p < 0.05$).

Furthermore, the analysis revealed significant main effects of timing, IAPS and face valence, a significant timing by face valence interaction as well as a significant IAPS by face valence interaction (Figure B2). The according three-way interaction of timing x IAPS valence x face valence reached marginal significance, indicating that time constraints as well as the valence of the scene and the facial expression had an influence on emotion recognition performance, but these influences did not differ significantly between groups and therefore according post-hoc comparisons are not reported in detail here, being beyond the scope of this thesis.

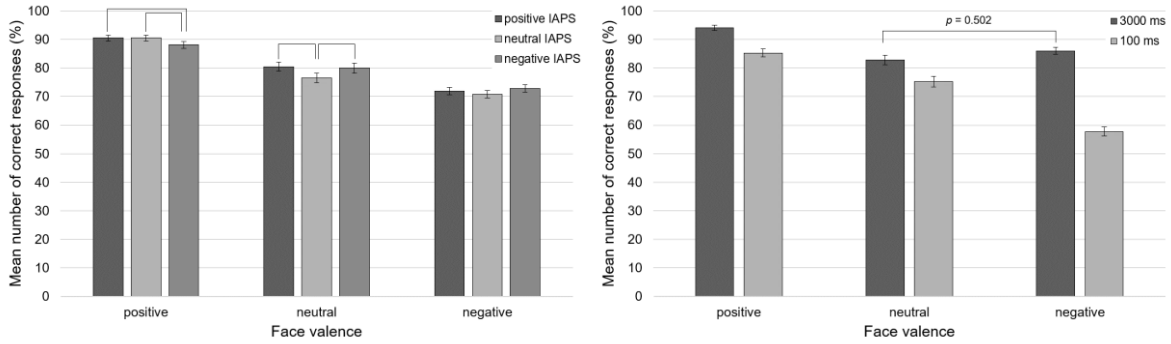

Figure B2. a) Interaction effect of face valence x IAPS valence. Percentages of correctly identified facial expressions separated for the different face valences and IAPS valences, parentheses indicate significant differences depending on the IAPS-pictures ($p < 0.05$). b) Interaction effect of timing x face valence. Percentages of correctly identified facial expressions separated for the different face valences and time conditions. All pairwise comparisons reached significance (all $ps < 0.001$), except that one displayed with parentheses. Error bars display standard errors.

Including sex as an additional factor in the analyses revealed no significant main or interaction effects. Since previous research consistently found an advantage for healthy women in emotion recognition tasks (e.g. Hall & Matsumoto, 2004; McClure, 2000), the general emotion recognition performance of healthy controls was analyzed separately in an independent samples t-test. The analysis revealed a significant difference in performance, with females showing a better performance ($t(33) = 2.32$, $p = 0.027$).

3.4.2 Analyses of response biases

To test the hypothesis that BPD patients and schizophrenia patients show a (negative) response bias for neutral facial expressions a bias valence x group ANOVA was conducted. The analysis revealed a significant group x bias valence interaction (table B6, figure B3). Adding sex as an additional factor to the analysis revealed no significant effect ($p = 0.231$).

Table B6. Statistical data of the group × bias repeated measures ANOVA for emotion recognition performance.

	df	*F*	η^2_p	*p*
Group	**2,102**	**5.84**	**0.103**	**0.004**
Bias valence	**2,204**	**46.20**	**0.312**	**<0.001**
Group x Bias valence	**2,102**	**5.07**	**0.090**	**0.008**

Note. Significant effects are displayed in bold.

Games-Howell post-hoc analysis showed a significantly higher negative bias in both patient groups compared to control subjects. There was no significant difference in negative bias between BPD and schizophrenia patients. The amount of positive biased responses did not differ significantly between groups (table B7). Although, there was a significant correlation between the negative and the positive bias in schizophrenia (r = 0.397, p = 0.018), which was neither significant in healthy controls (r = 0.172, p = 0.323), nor in BPD patients (r = 0.006, p = 0.974). Including sex as additional factor into the ANOVA revealed no significant main or interaction effects of sex.

Table B7. Interaction effect of group x bias valence: post-hoc pairwise comparisons.

Bias valence	Groups	Mean differences	Standard error	p_{adj}	95%-CI
Negative	**BPD > HC**	**11.05**	**3.19**	**0.004**	**3.28 – 18.82**
	SZ > HC	**7.52**	**3.19**	**0.013**	**-0.25 – 15.29**
	BPD > SZ	3.52	3.19	0.609	-4.25 – 11.29
Positive	BPD > HC	0.86	1.44	0.590	-4.37 – 2.65
	SZ > HC	2.76	1.44	0.231	-0.75 – 6.27
	SZ > BPD	1.90	1.44	0.485	-1.60 – 5.41

Note. Significant effects are displayed in bold. p_{adj} = Games-Howell-adjusted p-value because Levene's test showed that equal variances could not be assumed. HC = healthy controls, BPD = borderline personality disorder, SZ = schizophrenia.

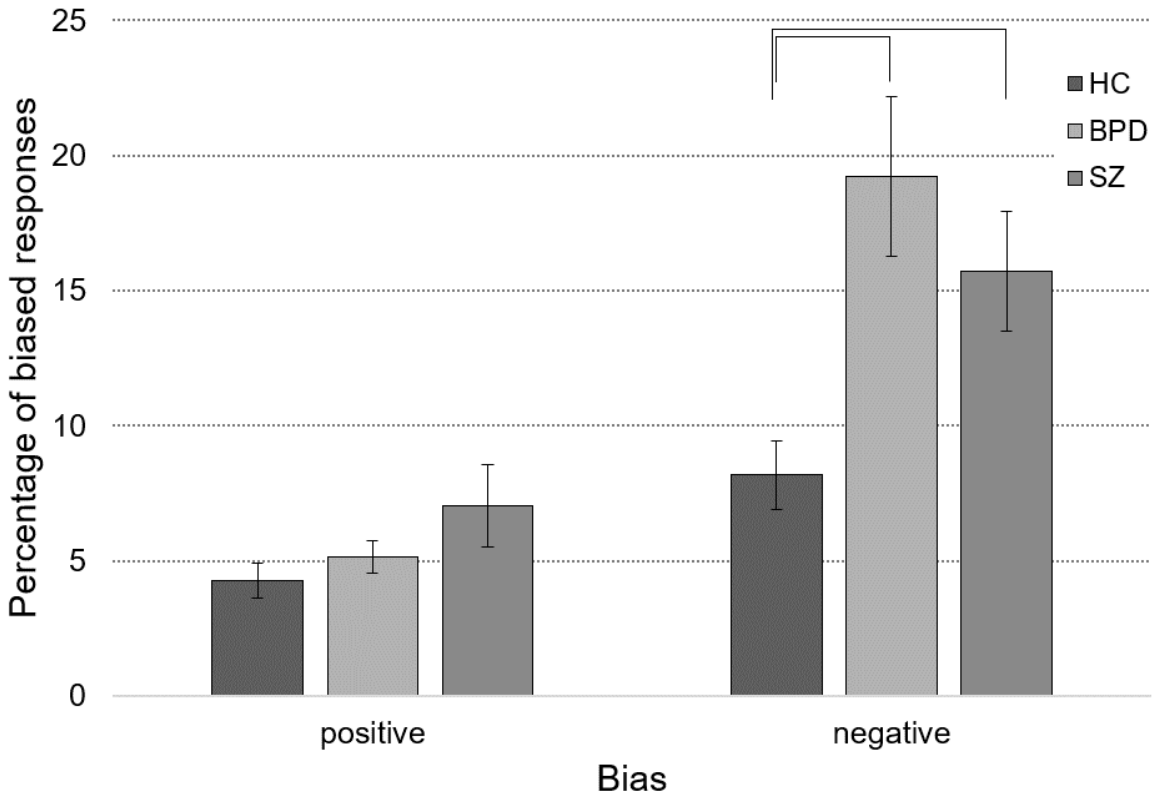

Figure B3. Bias x group interaction. Percentages of all responses to neutral facial expressions that were either positively or negatively biased, separated for the three groups. Error bars display standard errors and parentheses indicate significant group differences ($p < 0.05$).

3.4.3 Response bias and clinical scores

To investigate the association between emotion regulation abilities and biased perception, BSL-23- and DERS-scores were used. To explore correlations with alexithymia we further used TAS-20-scores. Associations between positive and negative symptoms in schizophrenia patients and biases in recognition of neutral facial expressions were assessed using PANSS-scores. Pearson correlation coefficients of the questionnaire and interview data and the amount of negative bias as well as positive bias were calculated for all participants. To differentiate between a specific negative bias and a general deficit in the recognition of neutral facial expressions a difference score was calculated for negative and positive bias which was also correlated with the variables reported in table B8 a – d. It has to be noted that some of the reported correlations were not significant after correction for multiple testing. There was no significant association between antipsychotic medication (CPZ equivalent scores) and the negative bias (BPD: $n = 9$, $r = 0.039$, $p = 0.920$; SZ: $n = 29$, $r = -0.233$, $p = 0.224$).

Table B8. Correlational analyses.

a)	Group (*n*)	Neg - Pos		Negative Bias		Positive Bias	
		r	*p*	*r*	*p*	*r*	*p*
BSL-score	All (104)	**0.295**	**0.002**	**0.279**	**0.004**	-0.033	0.736
	HC (35)	**0.409**	**0.015**	0.273	0.113	-0.302	0.078
	BPD (35)	0.146	0.401	0.164	0.346	0.075	0.668
	SZ (34)	-0.023	0.896	-0.115	0.519	-0.132	0.457

b)	Group (*n*)	Neg - Pos		Negative Bias		Positive Bias	
		r	*p*	*r*	*p*	*r*	*p*
DERS sum	All (103)	**0.376**	**<0.001**	**0.380**	**<0.001**	0.012	0.903
	HC (35)	*0.333*	*0.050*	0.245	0.157	-0.202	0.244
	BPD (35)	**0.373**	**0.028**	*0.316*	*0.064*	*-0.315*	*0.065*
	SZ (33)	-0.139	0.441	0.031	0.863	0.223	0.211

c)	Group (*n*)	Neg - Pos		Negative Bias		Positive Bias	
		r	*p*	*r*	*p*	*r*	*p*
TAS-20	All (103)	**0.427**	**<0.001**	**0.404**	**<0.001**	-0.049	0.623
	HC (35)	0.064	0.715	0.037	0.832	-0.058	0.739
	BPD (35)	**0.454**	**0.006**	**0.430**	**0.010**	-0.162	0.351
	SZ (33)	0.214	0.232	0.097	0.592	-0.150	0.406

Note: Significant correlations were also revealed for the TAS-20 scales "difficulty describing feelings" and "difficulty identifying feelings", but not for the scale "external thinking", in the whole sample, as well as in BPD patients.

d)	Group (*n*)	Neg - Pos		Negative Bias		Positive Bias	
		r	*p*	*r*	*p*	*r*	*p*
PANSS-pos	SZ (35)	0.096	0.582	**0.354**	**0.037**	**0.378**	**0.025**
PANSS-neg	SZ (35)	**0.348**	**0.040**	0.197	0.257	-0.200	0.248

Note. Significant effects are displayed in bold. All = all participants, HC = healthy controls, BPD = borderline personality disorder, SZ = schizophrenia, Neg – Pos = negatively minus positively biased responses, BSL = Borderline Symptom List, DERS sum = sum score of the Difficulties in Emotion Regulation Scale, TAS-20 = 20-item Toronto Alexithymia Scale, PANSS-pos = positive scale of the Positive and Negative Syndrome Scale, PANSS-neg = negative scale of the Positive and Negative Syndrome Scale.

Since we were also interested in sex specific correlations, the above reported analyses were also conducted separately for female and male participants in each group (table B9 a – d).

Table B9. Correlational analyses separated by sex.

a)	Group/Sex (*n*)	Neg - Pos		Negative Bias		Positive Bias	
		r/z	*p*	*r/z*	*p*	*r/z*	*p*
BSL-score	all/fem (53)	**0.407**	**0.003**	**0.431**	**0.001**	0.063	0.657
	all/male (52)	0.182	0.197	0.162	0.250	-0.031	0.827
	z-test	*1.24*	*0.109*	*1.48*	*0.069*	*0.47*	*0.320*
	HC/fem (17)	**0.764**	**<0.001**	**0.620**	**0.008**	*-0.477*	*0.053*
	HC/male (18)	0.134	0.597	0.056	0.824	-0.164	0.516
	z-test	*2.34*	*0.010*	*1.80*	*0.036*	*-0.95*	*0.171*
	BPD/fem (20)	0.366	0.112	0.395	0.084	0.003	0.988
	BPD/male (15)	-0.034	0.903	0.028	0.920	0.333	0.225
	z-test	*1.11*	*0.134*	*1.18*	*0.119*	*-0.91*	*0.181*
	SZ/fem (15)	0.005	0.986	0.108	0.702	0.237	0.395
	SZ/male (19)	-0.105	0.668	-0.245	0.312	-0.188	0.441
	z-test	*0.29*	*0.386*	*0.94*	*0.174*	*1.13*	*0.129*

b)	Group/Sex (*n*)	Neg - Pos		Negative Bias		Positive Bias	
		r/z	*p*	*r/z*	*p*	*r/z*	*p*
DERS sum	All/fem (53)	**0.535**	**<0.001**	**0.537**	**<0.001**	-0.049	0.735
	All/male (52)	0.239	0.088	**0.285**	**0.040**	0.108	0.447
	z-test	*1.76*	*0.039*	*1.53*	*0.063*	*-0.78*	*0.217*
	HC/fem (17)	**0.837**	**<0.001**	**0.614**	**0.009**	**-0.665**	**0.004**
	HC/male (18)	0.035	0.891	0.019	0.939	-0.033	0.897
	z-test	*3.17*	*0.001*	*1.87*	*0.030*	*-2.07*	*0.019*
	BPD/fem (20)	**0.454**	**0.044**	*0.423*	*0.063*	-0.316	0.175
	BPD/male (15)	0.357	0.191	0.310	0.261	-0.234	0.401
	z-test	*0.31*	*0.379*	*0.35*	*0.364*	*-0.24*	*0.407*
	SZ/fem (14)	0.206	0.479	0.270	0.351	0.103	0.726
	SZ/male (19)	-0.274	0.256	-0.043	0.862	0.274	0.257
	z-test	*1.25*	*0.105*	*0.82*	*0.207*	*-0.45*	*0.325*

c)	Group/Sex (*n*)	Neg - Pos		Negative Bias		Positive Bias	
		r/z	*p*	*r/z*	*p*	*r/z*	*p*
TAS-20	All/fem (53)	**0.599**	**<0.001**	**0.620**	**<0.001**	0.007	0.959
	All/male (52)	**0.294**	**0.034**	*0.262*	*0.061*	-0.051	0.719
	z-test	*1.93*	*0.027*	*2.27*	*0.012*	*0.29*	*0.386*
	HC/fem (17)	0.049	0.851	0.013	0.961	-0.090	0.731
	HC/male (18)	0.072	0.777	0.029	0.908	-0.090	0.723
	z-test	*-0.06*	*0.475*	*-0.04*	*0.483*	*0*	*0.5*
	BPD/fem (20)	**0.664**	**0.001**	**0.670**	**0.001**	-0.217	0.357
	BPD/male (15)	0.257	0.356	0.242	0.386	-0.066	0.814
	z-test	*1.45*	*0.074*	*1.52*	*0.065*	*-0.42*	*0.339*
	SZ/fem (14)	0.310	0.281	0.221	0.448	-0.182	0.532
	SZ/male (19)	0.119	0.628	0.020	0.937	-0.117	0.633
	z-test	*0.51*	*0.304*	*0.52*	*0.301*	*-0.17*	*0.433*

d)	Group/Sex (*n*)	Neg - Pos		Negative Bias		Positive Bias	
		r/z	*p*	*r/z*	*p*	*r/z*	*p*
PANSS pos	SZ/fem (16)	0.104	0.701	0.405	0.120	**0.733**	**0.001**
	SZ/male (19)	0.116	0.636	0.354	0.137	0.315	0.189
	z-test	*-0.03*	*0.487*	*0.16*	*0.437*	*1.63*	*0.051*
PANSS neg	SZ/fem (16)	0.228	0.279	0.217	0.419	-0.147	0.586
	SZ/male (19)	*0.452*	*0.052*	0.225	0.354	-0.252	0.298
	z-test	*-0.68*	*0.247*	*-0.02*	*0.491*	*0.29*	*0.385*

Note. Comparisons of correlation coefficients between females and males are reported with z-statistics in italics. All = all participants, HC = healthy controls, BPD = borderline personality disorder, SZ = schizophrenia, fem = females, male = males, Neg – Pos = negatively minus positively biased responses, BSL = Borderline Symptom List, DERS sum = sum score of the Difficulties in Emotion Regulation Scale, TAS-20 = 20-item Toronto Alexithymia Scale, PANSS-pos = positive scale of the Positive and Negative Syndrome Scale, PANSS-neg = negative scale of the Positive and Negative Syndrome Scale.

3.4.4 Rating of experimental stimuli

After the experiment all participants rated the experimental stimuli for valence and arousal separately for the stimulus category (faces vs. IAPS-pictures). A 3 (group) x 3 (face valence) repeated measures ANOVA on valence ratings for the faces revealed a main effect of face valence ($F(2,204) = 1082.32$, $p < 0.001$, $\eta^2_p = 0.914$), but no significant group differences ($F(2,102) = 0.22$, $p = 0.803$, $\eta^2_p = 0.004$)

or interaction ($F(4,204) = 0.52$, $p = 0.720$, $\eta^2_p = 0.010$). Bonferroni-corrected post-hoc comparisons showed that positive facial expressions were rated with highest, neutral with moderate and negative facial expressions with lowest valence (all ps < 0.001).

A 3 (group) x 3 (face valence) repeated measures ANOVA on arousal ratings for the faces showed a main effect of group ($F(2,102) = 3.77$, $p = 0.026$, $\eta^2_p = 0.069$), a main effect of face valence ($F(2,204) = 150.90$, $p < 0.001$, $\eta^2_p = 0.597$) and a marginally significant interaction ($F(4,204) = 2.27$, $p = 0.069$, $\eta^2_p = 0.043$) (figure B4).

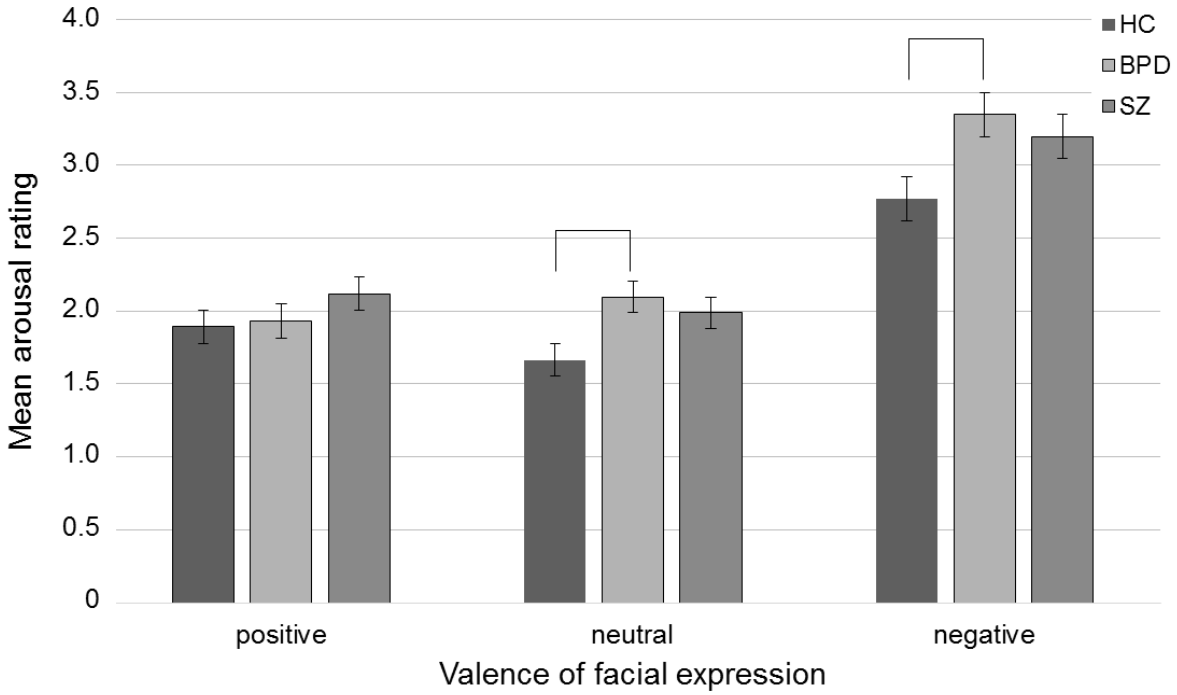

Figure B4. Marginally significant group x face valence interaction regarding arousal ratings. Error bars display standard errors and parentheses indicate significant group differences ($p < 0.05$).

A 3 (group) x 3 (IAPS valence) repeated measures ANOVA on valence ratings for the IAPS-scenes was conducted. It revealed a main effect of IAPS valence ($F(2,204) = 804.55$, $p < 0.001$, $\eta^2_p = 0.887$). No significant group differences ($F(2,102) = 0.24$, $p = 0.786$, $\eta^2_p = 0.005$) or interaction effects were found ($F(4,204) = 0.79$, $p = 0.507$, $\eta^2_p = 0.015$). Bonferroni-corrected post-hoc comparisons showed that positive scenes were rated with highest, neutral with moderate and negative facial expressions with lowest valence (all ps < 0.001).

A 3 (group) x 3 (IAPS valence) repeated measures ANOVA of arousal ratings for the IAPS-scenes showed a main effect of IAPS valence ($F(2,204) = 370.70$, $p < 0.001$, $\eta^2_p = 0.784$), a marginally significant main effect of group ($F(2,204) = 2.59$, $p < 0.080$, $\eta^2_p = 0.048$) and a marginally significant interaction ($F(4,204) = 2.16$, $p <$

0.080, η^2_p = 0.041). Bonferroni-corrected post-hoc comparisons revealed that negative scenes were rated with higher arousal than positive and neutral scenes. Neutral scenes were rated with lower arousal than positive scenes (all *p*s < 0.001).

3.4.5 Ratings of affective state, aversive tension and arousal

Ratings of positive affect differed significantly before and after the experiment (table B10): Positive affect was significantly lower after the experiment (p < 0.001). Ratings of negative affect were not affected significantly by the experimental procedure, but differed significantly between groups (table B11). BPD patients showed higher negative affect than schizophrenia patients (p = 0.001) and healthy controls (p < 0.001). Schizophrenia patients also showed higher negative affect than healthy controls (p = 0.002).

Table B10. Statistical data of the group × time point repeated measures ANOVA for positive affect (PANAS pos).

	df	*F*	η^2_p	*p*
Group	2,101	2.02	0.039	0.138
time point	**1,101**	**33.03**	**0.246**	**<0.001**
Group x time point	2,101	1.56	0.030	0.216

Note. Significant effects are displayed in bold.

Table B11. Statistical data of the group × time point repeated measures ANOVA for negative affect (PANAS neg).

	df	*F*	η^2_p	*p*
Group	**2,101**	**26.57**	**0.345**	**<0.001**
time point	1,101	2.19	0.021	0.142
Group x time point	2,101	1.32	0.025	0.271

Note. Significant effects are displayed in bold.

Self-reported arousal differed between groups and before and after the experiment (table B12). BPD patients and schizophrenia patients reported significantly higher arousal levels than healthy controls (p < 0.001). The patient groups did not differ in arousal (p = 0.530). Arousal was lower before than after the experiment (p = 0.009).

Table B12. Statistical data of the group × time point repeated measures ANOVA for self-reported arousal.

	df	*F*	η^2_p	*p*
Group	**2,101**	**17.68**	**0.259**	**<0.001**
time point	**1,101**	**7.04**	**0.065**	**0.009**
Group x time point	2,101	0.20	0.004	0.816

Note. Significant effects are displayed in bold.

Self-reported dissociative symptoms measured by DSS varied dependent on group and time point (table B13). BPD patients showed higher values than schizophrenia patients (p = 0.005) and healthy controls (p < 0.001). Schizophrenia patients also reported higher values than healthy controls (p = 0.008; figure B5).

Table B13. Statistical data of the group × time point repeated measures ANOVA for self-reported dissociative symptoms (DSS).

	df	*F*	η^2_p	*p*
Group	**2,102**	**19.92**	**0.281**	**<0.001**
time point	1,102	2.07	0.020	0.153
Group x time point	**2,102**	**9.61**	**0.159**	**<0.001**

Note. Significant effects are displayed in bold.

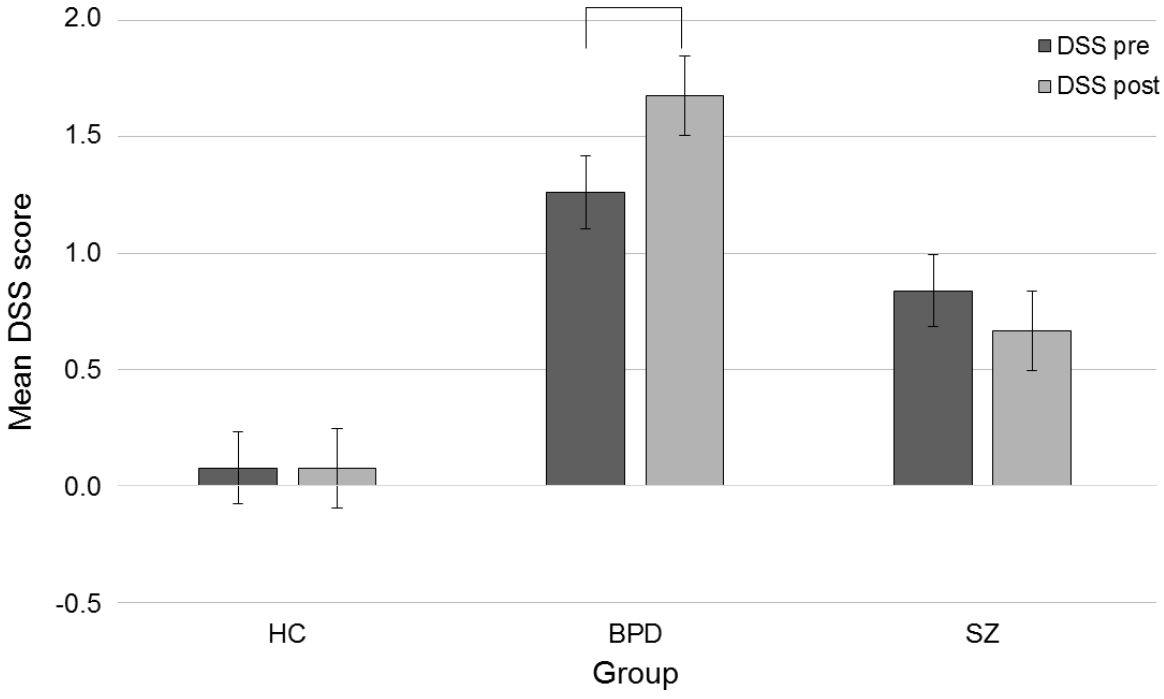

Figure B5. Group x time point interaction regarding DSS ratings. Error bars display the standard errors, parentheses indicate significant time point differences (p < 0.05).

3.5 General discussion of study 1

The first study of this dissertation was conducted to explore the nature of emotion recognition deficits and to specify factors influencing a negative bias in BPD. We assumed that preceding emotional information as well as time constraints reduce emotion recognition performance in BPD and provoke a negative bias. Further, we hypothesized that this negative bias is associated with emotion dysregulation and increased arousal. To address the question whether the negative bias is specific for BPD, a clinical comparison group with schizophrenia patients was included and additionally sex effects were explored. We assumed that emotion recognition deficits in schizophrenia would be less specific in a negative direction, suggesting a more general emotional bias. The previously reported emotion recognition advantage of healthy females may also exist in female patients. However, also the opposite could be true, since most of the previous studies reporting a negative bias predominantly investigated women. Thus, it was an explorative question for our study whether the negative bias in BPD is dependent on sex.

In the first part of this study (1a) we investigated a female sample of BPD patients and matched female controls. The analyses revealed an emotion recognition deficit for facial expression without a negative valence in women with BPD compared to healthy female controls. This finding was also revealed in another recent study on emotion recognition performance in BPD (Anupama, Bhola, Thirthalli, & Mehta, 2018). We found the most pronounced deficit in BPD patients for neutral facial expressions. This deficit in emotion recognition was due to a negative bias, which is in line with existing meta-analytic findings (Mitchell et al., 2014). The deficit seemed to be susceptible to the influence of preceding visual scene information as well as time constraints. In the condition with longer presentation times of the facial expressions, female BPD patients (study 1a) showed a higher number of incorrectly recognized neutral compared to positive faces when preceded by an emotional and especially negative IAPS picture. When time constraints were strong female BPD patients more often showed incorrect classifications of neutral compared to positive faces when preceded by non-emotional scenes. However, these interaction effects have to be interpreted cautiously because they only marginally reached significance. When processing time was not restricted at all, no differences in the evaluation of the valence of facial expressions occurred between healthy subjects and BPD patients.

In conclusion, available processing time has an important influence on emotion recognition performance in females with BPD.

In the second part of the first study (1b), we focused on investigating the specificity of emotion recognition deficits and the negative bias by comparing BPD patients not only to a healthy control group, but also to a clinical control group of schizophrenia patients. The same pattern of emotion recognition deficits was observed in the extended BPD sample with male subjects and schizophrenia patients. This supports the negative bias in both patient groups while no group differences in the amount of positive biased evaluations were present. Contrary to our hypothesis, the patient groups did not differ in the amount of negatively biased responses and showed a comparable negative bias in the recognition of neutral facial expressions in comparison to the healthy controls. However, schizophrenia patients showed the lowest performance in emotion recognition overall, indicating the most pronounced difficulties in social-cognitive processing in this group. Further, our findings show again that BPD patients are most impaired in the evaluation of neutral facial expressions and to a lesser extent in recognizing positive expressions, while schizophrenia patients exhibit also clear deficits in recognizing positive faces. In contrast to study 1a, no 4-way interaction between group, time, face and IAPS valence was revealed. This may be due to the small effect size and the fact that the interaction reached only marginally significance even in study 1a. Hence, our primary hypothesis that preceding negative context information would pronounce the negative bias in BPD patients compared to healthy controls was not consistently supported.

Interestingly, in the healthy control group, the amount of negative bias was associated with borderline symptomatology and emotion dysregulation pointing towards the general relevance of emotion regulation abilities for accurate emotion recognition. In agreement, higher emotion dysregulation scores were accompanied by a predominance of the negatively biased over positively biased responses in BPD patients, supporting the high relevance of emotion dysregulation for the negative bias in BPD. In schizophrenia, biased perception was correlated with positive and negative symptomatology instead. Negative and also positive biases were highly correlated with positive symptoms while a predominance of negatively biased responses was associated with a higher number of negative symptoms. These correlations suggest different factors influencing the negative bias in BPD and

schizophrenia. One preliminary conclusion from our results may be that in comparison to patients with schizophrenia, patients with BPD have a specific negative bias without any further emotion recognition deficits.

3.5.1 Available processing time

Presentation time of the facial expressions substantially influenced overall recognition performance in all groups. In the condition with short processing time of the facial expressions, there was a reduced performance for all valence categories compared to longer presentation times of facial expressions. However, no specific enhancement of the negative bias in the patient groups due to higher time pressure was observed.

At first glance, this seems to contradict the existing results by Dyck and colleagues (2009), showing impaired emotion recognition performance and a negative bias in BPD patients compared to healthy controls only under conditions of time pressure. In their study a stimulus presentation time of 2 s in the fast discrimination experiment and no time limit in the emotion recognition task was applied. Against this background and to be precise, processing time was restricted in both of our experimental conditions (100 ms vs. 3000 ms). We showed that patients exhibited a higher negative bias compared to healthy participants in both conditions but no further increase of negative biased responses due to more severe restriction of processing time. Interestingly, as mentioned before, valence ratings of the experimental stimuli after our experiment without any time restriction differed not between groups. Comparing the two different experimental timing conditions with the self-paced ratings after the experiment may be more suitable to evaluate the consistency between previous results on restricted processing time (see Dyck et al., 2009) and the present investigation. We add to these findings by Dyck and colleagues (2009) that further reduction of processing time results in an increased overall impairment of emotion recognition, but not in a group specific enhancement of the negative bias. The amplified overall impairment could be simply a result of increased task difficulty and reduced processing capacity as well as a need of more cognitive effort (Derntl, Seidel, Kainz, & Carbon, 2009).

In line with the findings by Derntl and colleagues (2009), an explanation why the negative bias is not differentially affected and linearly increased with the enhanced restriction of processing time could be that it is subject to disturbed top-down modulation processes that can only be compensated during conscious and

effortful information processing. Whether a facial expression is presented for 3000 ms or only 100 ms may not have a crucial impact since both conditions do not allow the necessary regulation processes. Successful emotion recognition under circumstances of unlimited processing time may involve other processes that compensate for impaired automatic top-down regulation processes. Conscious, controlled emotion regulation (e.g. reappraisal) has been shown to be effective in modulating the reaction to negative emotional stimuli (McRae et al., 2010) and was associated with downregulation of activation in the amygdala (Buhle et al., 2014). This kind of effect may diminish negative shifts in evaluation of emotional stimuli. However, studies investigating such emotion regulation strategies, usually allow for more time (usually ≥ 8 s) then it was the case in our experiment (e.g. Eippert et al., 2007; Schulze et al., 2011). If conscious and effortful regulation processes generally require more time to be executed, patients may not be able to engage these strategies when processing capacities are restricted. Thus, the presentation times in both of our conditions may have equally impeded the use of compensation strategies for impaired automatic top-down regulation in patients. In daily life, the absence of conscious emotion regulation in the context of emotion recognition is the norm rather than the exception, explaining the occurrence of negative biases in social-cognitive processing. Consequently, evidence-based therapy approaches for BPD often emphasize mindful perception and identification as well as awareness of emotions (Byrne & Egan, 2018). Future research, therefore, should determine if these strategies may also enhance automatic top-down control to maintain performance even in situations of low processing capacities.

3.5.2 Emotional interference

Another interpretation of the data is that emotionally interfering information is a relevant factor for the emergence of a negative response bias in BPD additionally to mere time restriction. Our results showed a complex interaction of emotional information, time constraints and group in study 1a. In BPD compared to healthy controls, longer presentation time was associated with more incorrect evaluations of neutral compared to positive faces when they were preceded by emotional, especially negative information. This supported our hypothesis that interfering negative social information leads to impaired recognition of neutral facial expressions, resulting in a negative bias. In contrast, under highly limited processing time, BPD patients showed more incorrect evaluations of neutral compared to

positive faces after preceding neutral information compared to healthy controls. That could be explained by increased ambiguity that results in higher error rates especially under time pressure.

It is important to note that these rather small effects were not significant in the second part of the study. We could not clearly confirm our prior hypothesis of an increased negative bias in BPD patients due to negative preceding information. The absence of group differences depending on prime valence is in line with an affective priming study in BPD, that was published concurrently with study 1a (Donges, Dukalski, Kersting, & Suslow, 2015). In the study by Donges and colleagues (2015) happy, neutral, and angry facial expressions (stimulus material from Ekman & Friesen, 1976) were used as subliminal primes. Prime faces were shown for 33 ms followed by a 467 ms presentation of the neutral mask faces. Subjects were instructed to evaluate the expressions (neutral masks) as negative or positive on a six-point rating scale. The authors found an effect of the prime face but no group differences or interaction between group and prime category. Angry primes led to negative shifts in the evaluation of the neutral masks. Happy primes did not result in significant evaluative shifts (Donges et al., 2015). Importantly and in contrast to our experiment, primes were presented without conscious awareness of the participants (subliminal priming) and priming effects were only examined within the same stimulus category and identity (i.e. emotional differences in the same faces). While we did not observe differential influence of preceding emotional information between groups in all of our subsamples the negative bias occurred consistently in all patient groups during our experiment.

Especially when considering that the ratings of the stimulus materials after the experiment revealed no negative bias in the patient groups, different explanations are possible for the occurrence of the negative bias within the experimental setting: First, it could have occurred exclusively due to restricted processing time as discussed above. Second, the experimental setting itself (additionally) created a more negative context for patients including increased arousal and changes in affective state. In addition to influences of the preceding information and in the light of habitual emotion dysregulation thereby altered perception might have occurred and led to negatively biased evaluations.

Increased negativity could have resulted from the preceding negative emotional information that could be even more salient to the patients than to the

healthy controls. As known from literature on emotional stroop effects (McKenna & Sharma, 2004; Phaf & Kan, 2007), there might have been carry-over effects between trials due to the negative information. Such carry-over effects are also known as “slow stroop” and describe interference of emotional information that is transferred from one trial to the following (McKenna & Sharma, 2004; Phaf & Kan, 2007). According to Waters and colleagues (2005) such effects are representations of attentional processes and result from difficulties in disregarding salient stimuli. BPD patients might be more prone to carry-over effects of negative emotional information than healthy participants due to increased emotional sensitivity, as well as slower habituation (Linehan, 1993a). BPD patients in our sample reported higher negative affect and arousal at baseline, which could reflect a pronounced sensitivity for negative information and heightened emotionality.

Interference of negative emotional information could have also occurred via different pathway, if the transferred emotional information resulted in an induction of negative affect and thereby shifted response tendencies in patients. Assessment of current affect before and after the experiment could support the assumption of an overall influence of the negative preceding information. However, it should be noted, that reduced positive affect after the experiment could also be due to negative effects of participation itself (e.g. annoyance of the participant due to duration of the experiment or boredom).

Since self-reported arousal levels increased during the experiment in all participants, increased perceived arousal could also play a role in determining the negative bias. As mentioned before, patients reported higher baseline arousal levels. Due to higher starting values and insufficient regulation strategies, patients might be less able to down-regulate their arousal level and distinguish the sources of it. This was also described by Linehan as “slow return to baseline level” (Linehan, 1993a), which is reflected on the neural level by a slow habituation of amygdala responding (Hazlett et al., 2012). Difficulties in down-regulation of arousal level might be also reflected in the increase of dissociative symptoms in our BPD sample during the experimental procedure, which could be a reaction to the accumulation of aversive inner tension due to arousal (Korzekwa, Dell, & Pain, 2009; Stiglmayr et al., 2008; Stiglmayr, Shapiro, Stieglitz, Limberger, & Bohus, 2001). In summary, several factors could contribute to a negative bias in BPD, ranging from specific circumstances, such as restricted processing time, up to general negative affectivity that biases

perception. Furthermore, the assumed carry-over effects are difficult to disentangle from the proposed slow habituation. Thus, future studies should attempt to design experimental paradigms that allow separating these factors to investigate the influence of each of them on disturbed emotion processing in BPD.

A model explicitly explaining hyper-mentalization in BPD (Bo, Sharp, Fonagy, & Kongerslev, 2017; referring to Sharp, 2014) might be also beneficial for the understanding of the negative bias. Hyper-mentalizing describes an over-attribution of mental states to others while there is no objective support for it (Sharp et al., 2013; Sharp et al., 2011). The negative bias seems to be a comparable phenomenon reflecting the over-attribution of emotions to faces not containing and representing this information. The authors argue that deficits in mentalizing occur in BPD patients only when different co-occurring processes, such as emotional and cognitive mentalizing must be integrated. Different factors, however, determine why in specific situations integration or selection of the correct interpretation fails. One important influencing factor on this is the available capacity to integrate different sources and processes (Sharp et al., 2013; Sharp et al., 2011). Following this argumentation, reduced processing time is again a promising candidate enhancing the probability for a negative bias. However, processing capacity can also be reduced by high states of arousal, intense emotions and context factors (Pessoa, 2009). It might not only be necessary to consciously perceive that there are different sensory inputs driving one reaction, but to regulate the emotional reactions triggered by them. Therefore, one has to differentiate between the competing sources and decide which one should be considered for an evaluation of the present situation (Lemerise & Arsenio, 2000). Due to difficulties in top-down modulation, this regulation could take much longer in patients compared to healthy people and can therefore only be successful when time is unlimited. Time restriction might additionally increase arousal level, which in turn reduces the ability to regulate emotional interference.

The negative bias was further associated with emotional dysregulation in BPD, which points to the relevance of successful top-down control in unbiased emotion recognition. This complex interplay between heightened negative affect and arousal, increased sensitivity to emotional stimuli resulting from hyperactivation in the amygdala (Donegan et al., 2003; Herpertz et al., 2001), and slow return to baseline (Linehan, 1993a) as well as reduced prefrontal control resulting in persistent dysregulation of emotions (Silbersweig et al., 2007) might result in negative biased

perception of neutral facial expressions when interfering emotional information is not discriminated from the target.

3.5.3 Specificity of emotion recognition deficits and a negative bias for borderline personality disorder – comparison with schizophrenia

Although schizophrenia patients exhibited a tendency for lower overall performance in emotion recognition compared to patients with BPD, both patient groups showed comparable response patterns. That is a significant negative bias in the recognition of neutral facial expressions. Although there was previous research that rather pointed to a general emotional bias instead of a preference for negative evaluations in schizophrenia (Catalan et al., 2016; Romero-Ferreiro et al., 2016), our sample was characterized by a significant negative response bias. This provides further support for a number of studies, indicating that besides a general emotion recognition impairment (Kohler et al., 2010), these patients also exhibit a negative bias (Kohler et al., 2003; Mier et al., 2014). Further, in agreement with our results, previous findings by Catalan and colleagues (2016) showed that overall emotion recognition performance was worse for schizophrenia patients, although not significantly reduced compared to BPD, and that BPD as well as schizophrenia subjects showed an impaired recognition for neutral faces. In a further recent study, it was shown that there were no significant differences in affect labeling between schizophrenia and BPD in an emotion discrimination task, but a higher negative bias in BPD (van Dijke et al., 2016).

BPD patients and schizophrenia patients did not significantly differ in the amount of negatively biased responses. However, in BPD the negative bias was clearly associated with emotion dysregulation. Interestingly, although schizophrenia is characterized by reduced top-down control and emotional dysregulation as well (Gilbert & Sigman, 2007; van der Meer, van't Wout, & Aleman, 2009), there was no significant correlation between the negative bias and emotion regulation deficits in this group. This points to a specificity of the negative bias in BPD regarding its underlying causes which might differ from that in schizophrenia patients. Schizophrenia patients by contrast showed a correlation between the negative bias (and also positive bias) with positive symptoms. This is in line with the assumption that aberrant salience and a reduced top-down modulation (Cook, Barbalat, & Blakemore, 2012; Laviolette, 2007; Mier & Kirsch, 2017) are involved in the emergence of a negative bias in schizophrenia. Fretland and colleagues (2015)

found an association between positive symptoms and over-mentalizing in a theory of mind task in schizophrenia patients, which could also be an explanation for negatively biased responses, since emotions are attributed to faces that do not contain emotional information.

Another significant correlation was revealed in schizophrenia: The more negative symptoms were reported, the greater was the difference between negative and positive biases favoring the negative bias. This might point to an association between disturbed or flattened emotional responding and the negative bias. In schizophrenia different symptom constellations could contribute to the negative bias which fits to the interpretation that patients with schizophrenia exhibit more under-mentalizing errors in social cognitive tasks compared to BPD (Andreou et al., 2015). While the biased responses in schizophrenia increase in general with positive symptoms and could be due to a tendency to over-mentalize, an excess of the negative bias over positively biased response may be more likely when negative symptoms are pronounced. The underlying basis of a predominance of negatively biased responses might be enhanced negative affect. In line with that, there is a great overlap between negative symptoms and depressive symptom as well as frequent co-occurrence of these symptom-patterns (An der Heiden, Leber, & Hafner, 2016; Krynicki, Upthegrove, Deakin, & Barnes, 2018). A more general influence of positive and negative symptoms on impairments in emotion recognition was also shown before (Kohler, Bilker, Hagendoorn, Gur, & Gur, 2000).

3.5.4 Sex specificity

Most studies on emotion recognition and biased perception in BPD only included female patients or a very low number of male patients, making conclusions about male BPD patients hard and a comparison of sex specific alterations nearly impossible. In contrast to our hypothesis of an emotion recognition advantage in females, there was no significant effect of sex regarding performance or error patterns across all participants. Since some authors have found such an advantage especially in healthy groups (e.g. Hall & Matsumoto, 2004; Hampson, van Anders, & Mullin, 2006; McClure, 2000; Schmid & Schmid Mast, 2010), this group was analyzed separately. This analysis indeed revealed that healthy females performed superior to men. Therefore, we assume our healthy control group as being representative for healthy populations. Within the patient groups there were no sex specific differences in emotion recognition performance or amount of negatively biased responses,

suggesting emotion recognition deficits to occur independently of sex in these disorders. This is remarkable, since previous research at least in schizophrenia pointed to sex effects in emotion recognition favoring female patients (Scholten, Aleman, Montagne, & Kahn, 2005). However, when going into study details, some revealed no overall sex difference. For example, male patients performed only worse for auditory stimuli (Vaskinn et al., 2007), or sex differences occurred regarding the accuracy in differentiating between different negative emotions (Weiss et al., 2007). In the study by Weiss and colleagues (2007) for example, female patients more often evaluated neutral faces as sad, while male subjects interpreted the same faces more often as angry. The authors conclude that this tendency might be important in the context of pronounced aggressive behavior in male schizophrenia patients (Weiss et al., 2007). In the present study we focused on visual stimuli and response format included only a classification of different valences not a differentiation between negative emotions. These could be possible reasons why we did not find an advantage in emotion recognition for female schizophrenia patients. We conclude from our data that in overt behavior, when the mere classification of valence is required in a visual task, female and male schizophrenia and BPD patients show comparable deficits and biases.

Interestingly, in the healthy control group, the correlation between the amount of negatively biased responses and higher borderline symptomatology as well as higher degree of difficulties in emotion regulation was driven by female subjects and differed significantly between females and males. However, a comparable pattern was not revealed in the BPD group. Since we found a trend for higher emotion dysregulation in BPD females than BPD males, it seems possible that an advantage by female sex is confounded with higher emotion dysregulation, resulting in equal performance between sexes.

3.5.5 Limitations

A major reason for conducting the study was the existing heterogeneity of findings regarding emotion recognition and a negative bias in BPD. Some of the previous studies might have facilitated or reduced the occurrence of a negative bias due to stimulus composition or response formats (e.g. comparison of forced choice discrimination tasks with different number of response alternatives in the study by Dyck and colleagues (2009)).

We explicitly used a valence evaluation to provide a balanced design neither favoring specific positive nor negative emotions. A limitation that arises from this is of course that we do not gain information about advantages or biases for specific emotions and it might be argued that labeling of valence does not reflect emotion recognition in a narrower sense when referring to discrimination between specific emotions. However, to determine a direction of biased perception it seems rather sufficient and economic. To get a more sensitive measure for shifts in response patterns (also for positive and negative expressions) we used a continuous rating scale in our follow-up fMRI study. Another limitation of the first study was the possibility of carry over effects of negative emotional information on the other conditions due to the pseudorandomized trial order as mentioned above. A potential solution for that is provided within the following fMRI study and described in the next section in more detail.

3.6 Summary

In summary, the results of study 1 point to a sex and disorder unspecific occurrence of emotion recognition deficits and a negative bias in BPD and schizophrenia. However, the occurrence of an overtly similar negative bias might be driven to a different extent by distinct underlying mechanisms. In BPD emotion dysregulation should be emphasized, while in schizophrenia severity of positive and negative symptomatology seems to play an important role regarding biased perception. Disorder-specific features are important moderators of a negative bias which was evoked in both patient groups when processing time was restricted. While those disorder-specific mechanisms might not be observable on the behavioral level, they might be evident when looking on the neurobiological level. Therefore, neuroimaging studies should uncover specific mechanistic impairments in mental disorders. The next study presents such a neuroimaging approach in healthy participants, allowing a deeper understanding of the influence of emotional context information on emotion recognition.

4 STUDY 2: IDENTIFICATION OF NEUROBIOLOGICAL CORRELATES OF NEGATIVELY PRIMED EMOTION RECOGNITION IN HEALTHY INDIVIDUALS AND IMPLICATIONS FOR DISTURBED MECHANISMS IN PATIENTS

The results of study 1 are in line with previous findings of a negative bias in facial emotion recognition in patients with BPD as well as schizophrenia (e.g. Kohler et al., 2003; Mitchell et al., 2014). Albeit, the influence of preceding emotional information was less differential than hypothesized, we conclude that indeed *there is an influence* of previously presented social information on subsequent emotion recognition processes and that this influence might differ between patients and healthy subjects. This was also indicated by evaluations of the experimental stimuli after the experiment which did not differ significantly between groups regarding valence ratings.

As already described in the introduction as well as in study 1, contextual influences on emotion recognition have previously been shown also in healthy participants (Mobbs et al., 2006). It was shown that perceptions of subsequent situations and emotional displays were altered by situational context and prior experiences (Barbalat, Bazargani, & Blakemore, 2013). Most of the literature investigating shifts in the evaluation of emotions refers to affective priming procedures. That is, they either used subliminal presentation of emotional faces (e.g. Dannlowski et al., 2007; Hietanen & Astikainen, 2013; Nomura et al., 2004) or contextual information as primes (e.g Kim et al., 2004; Mobbs et al., 2006; Schwarz, Wieser, Gerdes, Mühlberger, & Pauli, 2013). Although context primes were either presented simultaneously or were even explicitly associated with the target, enhancing the probability of being considered for the evaluation, these tasks provided insights in the neural processing of emotional primes. Emotional contexts (positive and negative sentences) provoked heightened amygdala activation in response to neutral facial expressions compared to baseline (Schwarz et al., 2013). Increased activation in this region could be associated with negative shifts in evaluation, emphasizing the role of the amygdala in biasing response tendencies (Dannlowski et al., 2007; Kim et al., 2004; Schwarz et al., 2013; Suslow et al., 2013). During emotion recognition stronger activation after negative emotional priming has also been found in the temporal pole, STS, insula and ACC (Mobbs et al., 2006).

Moreover, a negative correlation between amygdala activation and the IFG (ventral PFC) could be shown during the evaluation of facial expressions when primed by angry faces (Nomura et al., 2004). These findings point to a top-down modulated process of facial emotion perception with influences due to prior expectations and focus of attention rather than mere bottom-up sensory processing (Cook et al., 2012).

In study 2, we hypothesized that during evaluation of facial expressions consciously perceived, negative, task-irrelevant priming would produce increased activation in a network for emotion and intention recognition. This network was shown to be altered in BPD patients in response to social stimuli per se (Mitchell et al., 2014) and during social-cognitive tasks in particular (e.g. Mier et al., 2013). Such altered brain activation patterns might also be involved in the mechanisms driving the negative bias in emotion recognition as it has been observed under various conditions in BPD (e.g. Dyck et al., 2009; Fenske et al., 2015).

In a previous study we found that even marginal variations of a social-cognitive task (emotional vs. motoric imitation of facial expressions) resulted in differential activation of the network, especially the amygdala in healthy subjects (Fenske, 2012). Emotional imitation compared to a motoric imitation produced a neural response pattern that seems to reflect an enhanced emotional processing of social stimuli. As described above a similar pattern was also found partly in BPD patients in reaction to facial emotional stimuli (Mier et al., 2013).

To investigate neural processes underlying the negatively biased emotion recognition shown in the previous study we adapted our experimental paradigm. The rather small effects between prime categories could have resulted from the study design: First of all, scenes had an only moderate intensity, which might have reduced their capability to produce emotional interference. Even more importantly, the pseudorandomized order of the different priming valences could have produced carry over effects across trials and thereby obscured the effects of the different categories. Negative emotional information might have been more salient during the course of the experiment and could have influenced not only the perception of subsequent facial expressions but also preceding information and faces in other trials. Another restriction could be the categorial response alternatives used in study 1. False responses for positive faces resulted automatically in a negative bias, since there was no alternative for that. The same is true for negative faces vice versa. This might skew the resulting picture of responses.

To overcome these limitations, we implemented several changes in the experimental paradigm. For one thing, the presentation of neutral and negative preceding social information was moved to separate sessions. Task-irrelevant information was presented as before, but the possibility of carry over effects between prime emotions was excluded. Further, we decided to increase the difference in emotional valence between negative and neutral scenes. We also dropped the positive scenes that only served as an additional control condition in the initial paradigm. Response format was changed into a continuous rating scale allowing for positive shifts also when positive faces were presented and negative shifts of already negative facial expressions. Applying the improved paradigm, we intended to examine the neural mechanisms underlying shifted responses in emotion recognition.

We hypothesized that our adapted paradigm would not only activate the described network in a differential fashion, but also help to understand which parts of the network can be related to the negative shifts in emotion recognition elicited by task-irrelevant emotional information. This should in turn provide insights into the underlying mechanisms of disturbed emotion recognition and a negative bias by provoking response patterns similar to these occurring in patients in reaction to facial stimuli.

To clarify associations between brain functioning and biases in emotion recognition performance as well as BPD-traits, correlations between these measures will be explored.

4.1 The influence of negative affective priming on neural mechanisms of emotion recognition[2]

4.1.1 Abstract

The correct identification of emotional facial expressions is essential for successful social interactions. A negative bias in emotion recognition however, is characteristic for several mental disorders and can impair social functioning. The present study aimed at investigating the neural mechanism of such biased emotion recognition with an affective priming task in a healthy sample.

In our functional magnetic resonance imaging study, we investigated in 31 healthy participants whether task-unrelated negative social scene-pictures produce a negative bias in the recognition of facial expressions. In this paradigm emotional facial expressions that were preceded by either neutral or negative social scenes (which were not related to the faces) had to be classified as positive, neutral or negative.

A negative response shift occurred only for happy facial expressions that were preceded by negative social scenes. Analyses of brain activation showed enhanced recruitment of amygdala, nucleus accumbens, and superior temporal sulcus for neutral facial expressions under negative priming compared to neutral priming. Higher activation in nucleus accumbens was revealed for angry facial expressions and enhanced activation in the superior temporal sulcus for happy faces when preceded by a negative social scene.

These findings support the assumption that task-irrelevant negative contextual information biases the perception of facial expressions and leads to specific alterations in brain activation depending on face valence. We conclude that even in healthy participants, brain functioning is modulated by task-irrelevant negative contextual information which could result in biased emotion recognition.

[2] Manuscript submitted: Fenske, S. C., Stößel, G., Kirsch, P., & Mier, D. (submitted). The influence of negative affective priming on neural mechanisms of emotion recognition.

4.1.2 Introduction

Accurate emotion recognition is essential for social functioning. Deficits in this domain cause biased perceptions which are characteristic for a variety of mental disorders. Of special interest in this context is a negative bias in response to (i.e. the attribution of negative emotions to) neutral or ambiguous facial expressions, because it may lead to heightened hostility and social conflicts. Interestingly, in the context of affective priming, negative response shifts can also be provoked in healthy participants. In the present study, we aimed on investigating the neural basis of a negative bias in the context of affective priming in healthy participants.

Mental disorders that are often characterized by severe social impairments are – besides autism – schizophrenia and Borderline Personality Disorder (BPD; Green et al., 2012; Lieb et al., 2004). Both patient groups were found to have a negative bias in emotion recognition (Daros et al., 2013; Fenske et al., 2015; Kohler et al., 2003; Mier et al., 2014; Mitchell et al., 2014; Premkumar et al., 2008), and can be characterized by aberrant amygdala, superior temporal sulcus (STS) and inferior frontal gyrus (IFG) activation in response to facial expressions (Donegan et al., 2003; Marwick & Hall, 2008; Mier et al., 2013; Mier et al., 2010; Minzenberg et al., 2007; Seiferth et al., 2008). Importantly, in schizophrenia there is first evidence for a link between the negative bias for neutral facial expressions and amygdala activity (Mier et al., 2014). Further, schizophrenia is linked to aberrant salience attribution and changes in functioning of regions of the dopaminergic system, such as the nucleus accumbens (NAcc; Heinz & Schlagenhauf, 2010; Howes & Kapur, 2009; Kapur, 2003). In both disorders reduced connectivity between the amygdala and the prefrontal cortex was shown (Minzenberg et al., 2007; Ruocco et al., 2013; Williams et al., 2004), suggesting low control of the prefrontal cortex over the limbic system (Meyer-Lindenberg & Tost, 2012; Stein et al., 2007). Altered amygdala and amygdala-prefrontal circuit functioning in combination with impairments in dopamine transmission might affect the integration of motivational and emotional information in the NAcc, resulting in aberrant processing of emotionally salient sensory information (Laviolette, 2007). In consequence for schizophrenia, it has been assumed that reduced prefrontal control of the NAcc (Cook et al., 2012) in concert with amygdala hyperactivity might contribute to the negative bias for neutral facial expressions (Mier & Kirsch, 2017).

In line with findings of reduced prefrontal-limbic connectivity in schizophrenia and BPD, one mechanism that could explain biased perceptions is an atypical top-down modulation of social signal perception (Cook et al., 2012). Such top-down modulation of affective information can be investigated by affective priming tasks (Zajonc, 1980). Per definition, the valence of a prime alters subsequent judgments by shifting them in a congruent direction (Herr, Sherman, & Fazio, 1983; Klauer, Mierke, & Musch, 2003). Literature on affective priming effects in healthy samples often refers to subliminal, that is, masked affective priming by emotional faces (e.g. Dannlowski et al., 2007; Hietanen & Astikainen, 2013; Nomura et al., 2004), and several studies used contextual information as primes (e.g. Kim et al., 2004; Mobbs et al., 2006; Schwarz et al., 2013). In principle however, an affective prime can be any preceding emotional information that influences subsequent recognition of emotions like contextual cues, a foregoing mood or other previous information. In agreement, there is evidence for alterations in emotion perception due to the current affective state (Dunn & Schweitzer, 2005), and it has been shown that especially negative affect biases emotion processing (Coupland et al., 2004). In daily life, situational context and prior experiences can also influence the perception of subsequent situations including emotional displays (Barbalat et al., 2013).

On a neurobiological level there is evidence for heightened amygdala activation in response to emotional primes (Schwarz et al., 2013), and an association between a negative bias, due to affective priming, and amygdala activation was found repeatedly (Dannlowski et al., 2007; Kim et al., 2004; Schwarz et al., 2013; Suslow et al., 2013). Hence, the amygdala might contribute to biased perception by automatically shifting response tendencies (Dannlowski et al., 2007). Additionally, greater activation was found in BA 6 when faces were preceded by negative compared to positive contextual cues (Schwarz et al., 2013) as well as in the temporal pole, STS, insula and ACC for faces paired with emotional compared to neutral contexts (Mobbs et al., 2006). In a study by Nomura and colleagues (2004) a negative correlation between activation in the amygdala and the IFG (ventral PFC) was revealed during emotion recognition after priming with angry faces compared to neutral primes. This result supports the assumption that emotion recognition is not only influenced by bottom-up sensory, in this case visual processing, but is also modulated by top-down processes, which involve the focus of attention and influences due to prior expectations (Cook et al., 2012).

Further, the influence of the affective prime might not only depend on the valence or salience of the emotional stimulus, but also on factors within the considered persons. Few studies investigated affective priming of social information in schizophrenia and BPD. In schizophrenia, it is assumed that the underlying basis for alteration in top-down modulation is caused in general by an abnormal integration of new evidence into prior expectations (Blackwood, Howard, Bentall, & Murray, 2001; Freeman, 2007; Moritz & Woodward, 2006). This inflexibility to take new incoming information into account (or to differentiate between prior and newly incoming information) might explain the high susceptibility for affective priming in schizophrenia (Höschel & Irle, 2001), which has been shown to result in a negative bias for the evaluation of neutral facial expressions (Höschel & Irle, 2001; Suslow, Roestel, & Arolt, 2003). Hooker and colleagues (2011) further showed that preceding affective information alters subsequent trait judgments in schizophrenia: Negative affective primes in the form of pictures of the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1999) resulted in lower trustworthiness ratings of consecutively judged persons and this effect was associated with severity of positive symptoms (Hooker et al., 2011). To our knowledge, in BPD there is only one affective priming study examining evaluative shifts in the recognition of neutral faces. The authors used masked happy or angry facial expressions (Donges et al., 2015), and revealed that BPD patients show valence-congruent shifts in the perception of neutral faces. However, there were no differences in the amount of biased reactions between patients and healthy participants (Donges et al., 2015). From a dimensional approach, factors that are psychopathologically relevant in patients like severe emotion dysregulation or difficulties in top-down modulation of information processing might also affect the influence of prior emotional information on emotion recognition in healthy people. Therefore, specific personality features, or emotion regulation abilities should result in specific sensitivities or biases in non-clinical samples as well.

The aim of the current study was to assess whether task-unrelated negative social scene-pictures produce a negative bias in the recognition of facial expressions in healthy participants on the behavioral as well as on the neural level. Furthermore, we were interested in the link between schizophrenia and BPD related traits and the negative bias. We applied an adapted affective priming emotion recognition task (Fenske et al., 2015; Hooker et al., 2011; Mobbs et al., 2006) and hypothesized that preceding negative emotional information results in a negative shift of facial emotion

ratings; i.e. a negative bias. Further, we hypothesized that recognition of facial expressions after preceding negative social scenes compared to neutral social scenes would be associated with heightened limbic activation, especially of amygdala and NAcc as well as enhanced activation in IFG (BA44) and STS. Additionally, amygdala activation was assumed to correlate with the amount of negative bias. Furthermore, we suppose that shifts in the evaluation of facial expressions as well as its neural correlates are associated with traits that occur in schizophrenia and BPD, and are linked to reduced top-down control, such as emotion dysregulation (Petrovic & Castellanos, 2016) and schizotypy (Koychev, Deakin, Haenschel, & El-Deredy, 2011; Papousek et al., 2014).

4.1.3 Methods

4.1.3.1 Sample

The study was approved by the local ethics board of the Medical Faculty Mannheim, University of Heidelberg and was conducted in accordance to the declaration of Helsinki. Prior to attendance participants received detailed information about the study procedure and gave written informed consent. Five of 36 initially recruited participants had to be excluded due to high movement (> 3mm translation, or 3° rotation), headache, or low compliance during scanning. The final sample consisted of 31 healthy participants (for sample characteristics see table 1). Participants were recruited via local databases of the CIMH and were right-handed. All of them negated having a current or lifetime history of neurological disease. To exclude current or life-time psychiatric diagnoses, participants were screened with a German version of the SCID-I interview and completed the SCID-II questionnaire and interview when indicated (Fydrich et al., 1997; Wittchen et al., 1997). General inclusion criteria were sufficient command of the German language, MR inclusion criteria, as well as the ability to give written informed consent.

Before the experiment, fluid and crystallized intelligence were assessed by means of 5 subtests of the Leistungsprüfsystem 2 (LPS-2; subtests: 1) Allgemeinwissen, 2) Anagramme, 3) Figurenfolgen, 4) Zahlenfolgen, 5) Buchstabenfolgen; Kreuzpointner, Lukesch, & Horn, 2013) and the Wortschatztest (WST; Schmidt & Metzler, 1992). After scanning, participants completed several questionnaires, including the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), the emotion regulation questionnaire (ERQ; Abler & Kessler, 2009),

as well as the schizotypal personality questionnaire (SPQ; Klein, Andresen, & Jahn, 1997).

Table 1. Sample characteristics.

	N = 31
Sex	16 females, 15 males
Age (in years)	33.61 *(12.28)*
Education (in school-years)	11.26 *(1.44)*
WST-IQ	104.71 *(11.42)*
LPS-2 CI	107.19 *(17.75)*
LPS-2 FI	99.39 *(12.44)*
DERS sum	65.94 *(10.97)*
ERQ-CR	29.23 *(6.83)*
ERQ-ES	14.10 *(4.62)*
SPQ	8.00 *(7.24)*

Note. Means and standard deviations (in parentheses) of WST-IQ = Wortschatztest IQ, LPS-2 = Leistungsprüfsystem 2, CI = crystallized intelligence, FI = fluid intelligence, DERS sum =Difficulties in Emotion Regulation Scale sum score, ERQ = emotion regulation questionnaire (subscales: CR = cognitive reappraisal, ES = expressive suppression), SPQ = Schizotypal Personality Questionnaire.

4.1.3.2 Emotion recognition task

Functional MRI data was collected during an affective priming emotion recognition task (modified from Fenske et al., 2015), which was applied in two separate sessions representing two priming conditions. In both sessions facial expressions were preceded by a scene picture, which served as prime. These preceding pictures were taken from the IAPS (Lang et al., 1999) and all depicted humans. In the first session IAPS pictures showed scenes with neutral valence (*M* = 5.46, *SD* = 1.34) and low arousal (*M* = 3.50, *SD* = 1.97; e.g. depicting daily conversations) while in the second session negative valent IAPS pictures (*M* = 2.01, *SD* = 1.36) with high arousal (*M* = 6.02, *SD* = 2.20; e.g. depicting interpersonal violence) were shown (see supplementary materials for a list of the presented IAPS pictures, table S1). The separate sessions were used to avoid carry over effects between the prime categories. In comparison to our former study, we did not use positive primes, since our current research question was focused on the effect of negative preceding information and a negative bias. Facial stimuli consisted of 5 male (20, 25, 26, 34, 36) and 5 female actors (1, 6, 7, 8, 9; ethnicity: all european-american) and were taken from the NimStim Set of Facial Expressions (Tottenham et

al., 2009). Facial expressions were either emotional (happy or angry) or contained no emotion (neutral). Morphed facial expressions with reduced emotion intensity were applied (60% emotion, 40% neutral; (Matzke et al., 2014)) to avoid ceiling effects in emotion recognition performance. For each face valence category, a set of 10 different primes was selected that was matched for content, valence and arousal in each session (see supplementary materials for a comparison of the picture sets, table S2). Participants were instructed to look at all pictures, but to rate the valence of the facial expressions only (the left end of the scale was indicated as “negative” and was coded with -100, the middle position as “neutral”, coded with 0 and the right end of the scale was named as “positive” and coded with 100 in data analyses). After determining the valence of the faces, participants had to rate how certain they were with their decision from “very uncertain” (0) to very certain (100). For both ratings visual analogue scales were used.

In both sessions IAPS pictures were shown for 1 s and were followed after a variable ISI of 1-3 s by a picture with a facial expression that was also presented for 1 s (figure 1). Rating scales were presented for 5 seconds each. Each trial had a mean duration of 18.5 s including a variable ITI (2 - 4 s) in which zero-events were defined that had the same duration as the picture presentations. Ratings were given by button presses on a 4-Button Diamond Fiber Optic Response Pad (Current Designs Inc.). By the first button press, participants could decide whether they want to jump to one of the two ends, or to the middle of the scale. By continuously pressing a button after that first decision, participants were able to additionally adjust their initial answer. Each session consisted of 30 trials, i.e. 10 combinations of the primes with each facial category (happy, neutral, angry) and took about 9.5 minutes.

In addition, the current affective state was assessed before and after each session with the Positive and Negative Affect Schedule (PANAS; Krohne et al., 1996; Watson et al., 1988). The task as well as the PANAS were presented via monitor and mirror using Presentation software (Version 18.1, Neurobehavioral Systems). Due to an additional imitation task, participants were videotaped during the whole MRI-session.

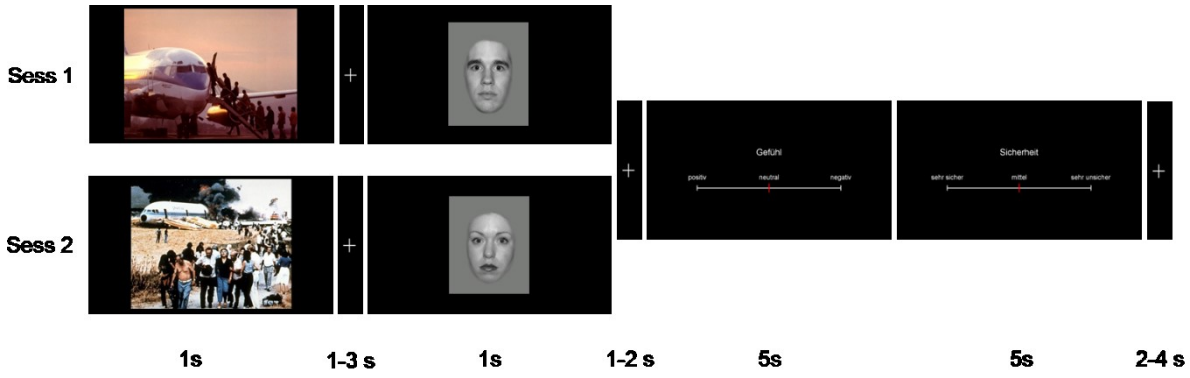

Figure 1. Experimental design: exemplary course of one trial per session with neutral facial expressions.

4.1.3.3 Rating of experimental stimuli

After the scanning, the applied pictures were presented again outside of the scanner on a computer screen to allow valence and arousal ratings, independent of the priming context. Faces and scenes were presented to the participants in two separate blocks, always starting with the faces block. Participants were asked to indicate the valence and arousal of each of the pictures using the Self-Assessment-Manikin (SAM; Hodes et al., 1985; Lang, 1980) on a 9-point scale. This rating procedure was self-paced.

4.1.3.4 Data acquisition and data analysis

A 3 T Siemens TIM TRIO (Siemens Medical Systems, Erlangen) scanner with a 12-channel head coil was used for fMRI data acquisition. Prior to the experiment, a 3D-T1-weighted MPRage was acquired (192 slices, slice thickness 1x1x1 mm, 256x256 matrix). 278 volumes were collected in each experimental session in a descending order with an T2*-weighted echo-planar sequence (28 slices, slice thickness 3x3x4 mm, TR 2000, TE 30, alpha 80°, FoV 192, 64x64 matrix). Slides were adjusted to AC-PC and tilted with -25°.

Data was pre-processed and analyzed using SPM12 (The FIL methods group, 2011). Pre-processing consisted of slice time correction, realignment with unwarping, normalization to the MNI standard template with coregistration to the MPRage and smoothing (8 mm full width at half maximum, Gaussian isotropic kernel). For statistical analyses, data was entered into a first level analysis. Both sessions were modulated together in one model using the onsets for face categories, scenes, control condition, and ratings as events, convolved with the hemodynamic response function (HRF). The resulting regressors were entered as predictors into a general

linear model. Regressors for the six movement parameters as well as for cerebrospinal fluid (CSF) and white matter (WM) were included as covariates. After model estimation, linear contrasts were defined for comparisons of the experimental conditions and the control condition (zero-events), as well as for comparisons between the experimental conditions. Second-level analyses were conducted, using T-tests and full factorial designs. Whole-brain activations are reported at an exploratory significance level of $p < 0.001$ uncorrected, $k = 10$. Region of interest (ROI) analyses were conducted for the STS, the inferior frontal gyrus (BA 44), the amygdala and the NAcc with a significance level of $p < 0.05$ small volume corrected (svc), $k = 10$. For correlation purposes, first eigenvariates of these ROIs were extracted (without applying a significance or cluster size threshold) for the neutral face events.

Behavioral and questionnaire data were analyzed using SPSS Statistics Version 24 (IBM Corporation). The assumptions of normality were assessed by graphical means and Kolmogorov-Smirnov-tests. The outcome variables were all approximately normally distributed. Repeated measures ANOVAs and post hoc t-tests were conducted to test the hypotheses related to the behavioral data. In the case of a significant Mauchly test of sphericity, the according *p*-values are reported with Greenhouse-Geisser correction. Effect sizes are specified as Cohen's *f* and *d*. Pearson's correlation coefficients were calculated to investigate the association between the ratings, questionnaire data and activation within the ROIs.

4.1.4 Results

4.1.4.1 Behavioral data

Effects of negative priming on valence ratings

To investigate the first hypothesis, that participants show a negative priming effect (negative bias) in emotion recognition, a 2 (prime valence) x 3 (face valence) repeated measures ANOVA was conducted. A significant priming by face valence interaction ($F(2;60) = 11.90$, $p < 0.001$, $f = 0.86$) was revealed (figure 2). Bonferroni-adjusted post-hoc comparisons revealed that participants rated positive facial expressions less positive when they were preceded by negative ($M = 44.90$, $SD = 22.46$) compared to neutral priming scenes ($M = 52.84$, $SD = 18.94$). For neutral and negative facial expressions no significant negative priming effect was revealed (according pairwise comparisons are reported in the supplementary materials, table

S3). Due to this interaction, the interpretability of the main effect of face valence ($F(2;60) = 191.41$, $p < 0.001$, $f = 2.63$) might be restricted. However, Bonferroni-adjusted post-hoc analysis revealed significant differences between all levels of face valence. Positive facial expressions were rated with highest valence ($M = 48.87$, $SD = 19.93$) and negative faces with the lowest ($M = -46.94$, $SD = 22.07$), while neutral faces received ratings in between ($M = -8.79$, $SD = 7.94$; according pairwise comparisons are reported in the supplementary materials, table S4). Exploratory analyses of certainty ratings and reaction times can also be found in the supplementary results (table S5-S7).

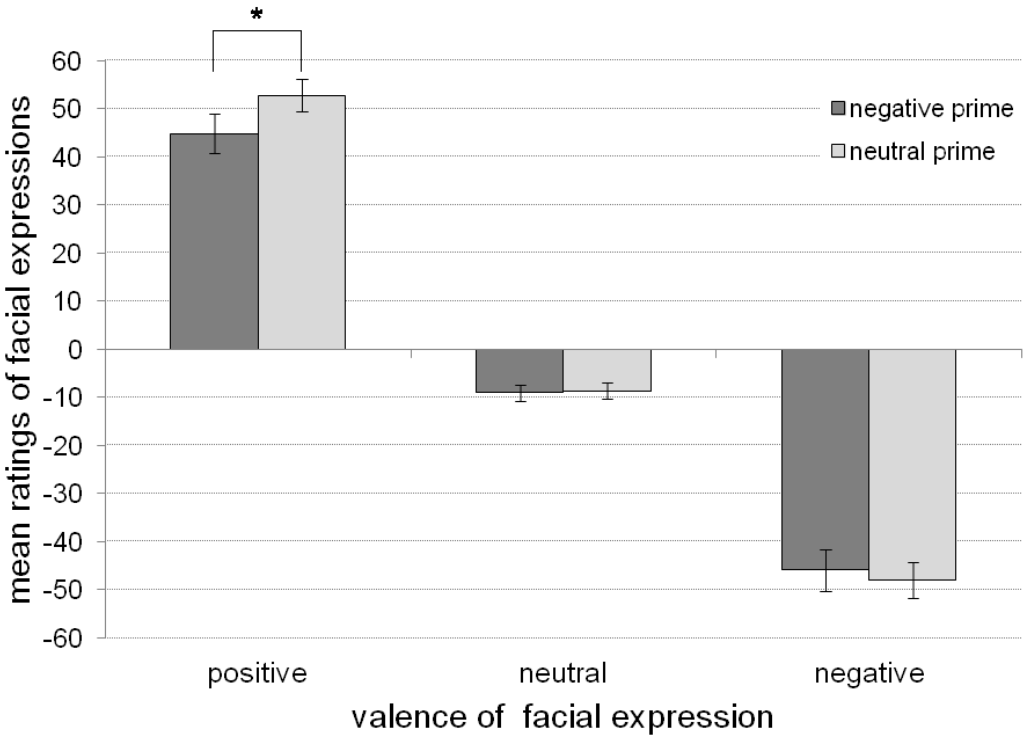

Figure 2. Prime valence by face valence interaction. Mean ratings of facial expressions separated for face valence and prime valence. Error bars display the standard errors, stars indicate significant differences ($p < 0.05$).

The ratings of facial expressions and prime pictures after the experiment served as a manipulation check and revealed that happy facial expressions were rated with the highest and angry facial expressions with the lowest valence, while ratings for neutral faces lay in between (table S8, S9). Arousal was highest for angry facial expressions followed by happy and neutral expressions. Neutral primes were rated with higher valence and lower arousal compared to negative primes (table S10).

4.1.4.2 Functional brain imaging

Manipulation check

As a manipulation check differences in brain activation in reaction to negative compared to neutral scenes were analyzed. In agreement with previous studies, using IAPS-scenes (Britton, Taylor, Sudheimer, & Liberzon, 2006; Lang et al., 1998), whole-brain analyses revealed that participants showed greater activation in visual association cortex as well as in fusiform gyrus in reaction to negative scenes (table S11). Applying ROI-analyses for this contrast additionally showed stronger activation in the left amygdala (peak voxel -24 -1 -19, cluster size 16, T = 3.16, p = 0.036 svc).

Negative priming effects

Effects of prime valence on brain activation during emotion recognition were assessed by means of whole-brain analyses with a full-factorial model and revealed a main effect of prime valence in putamen, dorsal striatum, thalamus, primary visual and visual association cortex, superior temporal gyrus, inferior frontal gyrus, supramarginal gyrus and anterior cingulate cortex (table S12). Further, whole-brain analyses revealed an interaction between prime valence and face valence in primary somatosensory cortex, anterior and medial prefrontal cortex, inferior and middle temporal gyrus, fusiform gyrus and amygdala (table S13). To disentangle the interaction and to investigate our specific hypotheses, post hoc t-tests were conducted for our ROIs.

For negatively primed neutral faces in comparison to neutral faces that were presented after neutral scenes, stronger activation was found in amygdala bilaterally (left: peak voxel -24 -4 -16, cluster size 37, T = 4.41, p = 0.002 svc; right: peak voxel 27 -7 -16, cluster size 38, T = 4.40, p = 0.002 svc), STS bilaterally (left: peak voxel -60 -49 11, cluster size 231, T = 4.23, p = 0.018 svc; right: peak voxel 48 -61 17, cluster size 117, T = 4.61, p = 0.003 svc) and NAcc bilaterally (left: peak voxel -21 8 -4, cluster size 31, T = 3.42, p = 0.043 svc; right: peak voxel 21 5 -7, cluster size 27, T = 4.44, p = 0.004 svc; figure 3). No significant difference depending on prime valence occurred for BA 44.

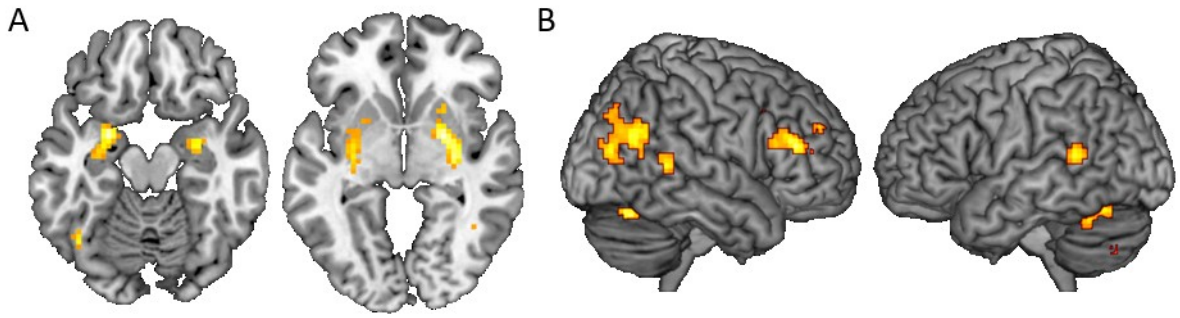

Figure 3. Enhanced activation for negatively primed compared to neutrally primed neutral facial expressions. A) horizontal view, B) lateral view. Note: significance level for display purposes is p = 0.001 uncorrected, k = 10.

Negatively primed happy faces compared to happy faces that were preceded by a neutral picture revealed stronger activation in right STS (peak voxel 51 -61 20, cluster size 107, T = 4.33, p = 0.007 svc), only. Angry faces that were preceded by negative compared to neutral prime stimuli revealed stronger activation in left NAcc (peak voxel -21 11 -1, cluster size 13, T = 3.62, p = 0.030 svc), only.

Correlational analyses

To test the assumption that amygdala activation is positively correlated with the amount of negative bias, Pearson correlation coefficients of first eigenvariates of amygdala activation for neutral faces preceded by negative compared to neutral scenes and the amount of negatively biased responses were calculated. The analysis revealed no significant association.

The supposed associations between shifts in the ratings of facial expressions and its neural correlates with emotion regulation capabilities and schizotypy were tested by calculating Pearson correlation coefficients of the questionnaire data as well as the behavioral ratings and differences in brain activation in the ROIs for neutral faces depending on the priming condition. Only one correlation survived the correction for multiple testing: A higher difference in activation in right BA 44 in reaction to neutral facial expressions between neutral and negative priming condition was associated with less cognitive reappraisal (r = -0.562, p < 0.001).

4.1.5 Discussion

To investigate the influence of negative affective information on subsequent emotion recognition in healthy participants, we conducted an emotion recognition task in which each facial display was preceded by either a neutral or a negative prime. It was hypothesized that ratings of face valence will be shifted in a negative

direction when a negative picture was shown before (i.e. that a negative bias occurs). Further, it was assumed that emotion recognition in a negative context will provoke heightened activation in the limbic system as well as in the IFG and the STS. The findings of our study suggest a dissociation between brain activation and overt behavior, which we assume to be due to different mechanisms that drive the specific response patterns.

On the behavioral level, it was shown that preceding negative information resulted in a negative shift in the valence evaluation of happy, but not neutral or angry facial expressions. Schmid and Schmid Mast (Schmid & Schmid Mast, 2010) applied movie scenes as mood priming and showed that emotion recognition was shifted in the direction of the prime when the target expressions were mood-incongruent. The authors state that incongruity drives this effect and that it only occurs on a level of general valence and not for specific emotions. In accordance with mood congruity theories (Bower, 1981; Schwarz, 1989), as well as the findings of Schmid and Schmid Mast (Schmid & Schmid Mast, 2010), we assume that dissonance between the negative prime and the positive valence of the facial expression is reduced by evaluating the facial expression as less positive. Since dissonance between negative primes and negative or neutral facial expressions was lower, healthy participants might be able to compensate the effect of the negative preceding pictures in these trials without a process of dissonance reduction. Since we did not explicitly aim on mood induction, but on creating an emotional context or frame for the emotion recognition task, an alternative label for the mechanism in our study might be “emotional interference reduction”, which accounts for the competing emotional information that is resolved by a negative shift in emotional ratings.

While in schizophrenia patients, one explanation for the high susceptibility for affective priming is an atypical top-down modulation due to the abnormal integration of new information in prior expectations (Blackwood et al., 2001; Freeman, 2007; Moritz & Woodward, 2006), healthy participants in our study showed this seeming inflexibility only for newly incoming positive information that result in incongruence to the prior negative picture. It is important to note that we did not use a positive prime, which might have led to a positive bias in the evaluation of negative facial expressions by implication of the abovementioned mechanism. Further, we conducted a former version of this experimental design with BPD patients who showed a negative bias for positive and neutral facial expressions following negative

primes (Fenske et al., 2015). In an extended sample including BPD and schizophrenia patients as well as healthy subjects, all participants showed a negative shift in the evaluation of positive faces when preceded by negative scenes (Fenske et al., unpublished data). Thus, this susceptibility to emotional inference seems only to be reflected in overt behavior when the dissonance is high, but manifests regardless of group.

Although we did not find a general negative bias in the rating of facial expressions due to the affective priming, there was a clear effect on brain activation, pointing to a higher sensitivity of neural reactions than of overt behavior to negative primes. Meaux and Vuilleumier (2016) state that different parts of the face processing networks are involved and modulated by the demands of a specific task. Such individual requirements might be evoked by the identification of neutrality or different emotional valences in a face that is preceded by namely irrelevant, but negative scenes resulting in the specific activation patterns. In particular, for neutral facial expressions that were preceded by negative IAPS pictures we found heightened activation in the limbic system, especially in amygdala and NAcc, as well as in the STS, a pattern that was indeed expected also for emotional facial expressions preceded by negative primes. Neutral facial expressions contain a high amount of ambiguity, which might require a more elaborated information processing. Negative preceding information could further increase this ambiguity, which might be reflected by the enhanced activation for neutral facial expressions that were preceded by negative emotional information. Mobbs and colleagues (2006) found - amongst other regions - enhanced STS activation due to emotional compared to neutral contexts in healthy participants, too. This region was revealed in our experiment not only for neutral facial expressions under negative priming conditions, but also for happy facial expressions. The STS is known to be associated with face processing in general and processing of variable aspects in a face like its expression in particular (Haxby et al., 2000). Meaux and Vuilleumier (2016) showed that the STS and prefrontal areas were recruited for the evaluation of independent features when a full face was presented under conditions of incongruence. Thus, we suggest that for happy faces following negative scenes a process of incongruence reduction occurred in our healthy sample. We do assume that this specific emotional interference reduction, which results in less positive evaluations of happy faces, differs substantially from the negative bias in the recognition of neutral faces that occurs in BPD patients. We

further argue that this interference reduction would conversely and regardless of group result in positively shifted responses for angry faces, when preceded by positive primes. However, since we did not use positive primes, we do not have empirical support for this assumption. In a mixed sample including BPD patients, schizophrenia patients and healthy subjects, we have found a similar effect for positive faces that were preceded by negative IAPS pictures (Fenske et al., unpublished data). All participants showed a negative shift in the recognition of positive faces after a negative scene picture. This effect did not differ between groups, indicating that this negative shift occurs in all participants including healthy participants. The negative bias in the evaluation of neutral facial expressions that was found in our previous studies in BPD and schizophrenia patients, but not in healthy participants (Fenske et al., 2015; Fenske et al., unpublished data) therefore might be a separate mechanism that is due to enhanced amygdala activation. In line with that, in our current study, increased amygdala activation was only revealed for negatively primed neutral, but not positive faces.

Aberrant amygdala activation, as it occurred in response to neutral faces, after negative priming, was also found for patients in reaction to facial expressions in general (Donegan et al., 2003; Marwick & Hall, 2008; Mier et al., 2013; Mier et al., 2010; Minzenberg et al., 2007; Seiferth et al., 2008) and in association with a negative bias (Donegan et al., 2003; Mier et al., 2014; Minzenberg et al., 2007). Further, for neutral, as well as for angry facial expressions that were preceded by a negative scene left NAcc activation was enhanced by negative preceding information, although there was no behavioral correlate for that. The enhanced NAcc activation might reflect the heightened salience that is elicited by threat and negative information (Vuilleumier, 2005). The shorter reaction times for angry facial expressions support this assumption of heightened salience of negative information. The generally faster responses in the session with negative primes seem also in agreement (see supplementary table S6), but it cannot be ruled out that this was due to learning effects caused by session order. Since our healthy sample showed no biased evaluations for neutral and angry facial expressions on the behavioral level, the results might point to an effectively working regulation mechanism in healthy participants, although brain activation is altered by irrelevant emotional information prior to the target emotion. More intense alterations in brain response might be

necessary to evoke impairments on the behavioral level, as we would expect them to occur in BPD patients.

A higher difference in activation in right BA 44 in reaction to neutral facial expression between neutral and negative priming conditions was associated with less cognitive reappraisal. In other words, the higher the difference in BA 44 activation in response to neutral faces between negative and neutral primed neutral faces, the lower was the habitual emotion regulation ability via cognitive reappraisal. This result is in line with the assumed associations between emotion regulation abilities and the negative bias. Further, these results are in agreement with our earlier findings in a behavioral study with BPD-patients that showed an association between emotion dysregulation and a negative bias for neutral faces (Fenske et al., 2015). BA 44 is part of the mirror neuron system and assumed to enable embodied simulation (Iacoboni & Dapretto, 2006). Thus, our results might suggest a tendency to over-attribute mental states to neutral faces when preceded by a negative situation. Since emotion regulation has been linked to top-down control (Braunstein, Gross, & Ochsner, 2017), we assume that a reduction of top-down control, as reflected in less cognitive reappraisal abilities might be causal to the negative bias in BPD. In our healthy participants however, this enhanced vulnerability on the neural level did not lead to behavioral deficits.

Except for the association with cognitive reappraisal, there were no further associations between assessed personality features and traits that are linked to reduced top-down control and the negative bias in our sample that survived correction for multiple testing. However, this might be attributable to the rather low values and variance of schizotypy and emotion regulation difficulties in our healthy participants. Since the SPQ-scores in our sample are comparable with earlier findings for healthy participants of Moritz and Andresen (2002) we assume our sample to reflect a representative picture of this population. Hence, further research to elucidate the relationship between emotion regulation and schizotypy by investigating groups with higher variance in the clinical measures, or directly by assessing patient samples is needed.

In contrast to our expectation, we found a response shift on the behavioral level, only for positive faces. An additional explanation to the presented incongruency hypothesis why there was no general priming effect due to the negative scenes could also be our study design. Short SOAs and non-consciously perceived (subliminal)

primes have been shown to elicit target-unspecific priming effects in healthy participants (e.g. Murphy & Zajonc, 1993), and evaluations of time courses further proved that short prime presentation times elicit the strongest priming effects in the sense of congruent affective evaluations. Primes that were presented for 1000 ms or longer did not result in such general shifts (e.g. Hermans, De Houwer, & Eelen, 2001). A longer presentation allows a higher degree of cognitive processing and provides more information about specific features of the prime that might cancel the priming effect on behavioral level. This conscious evaluation might be the reason for more accurate emotion recognition and reduced automatic priming effects in our study. Hence, we speculate that in healthy participants, mood or valence incongruence can cause a shift in emotion recognition especially under conditions of consciously perceived presentation of the irrelevant context information and the target.

In summary, we could show that in healthy people the neural level seems to be more sensitive to negative preceding information than the behavioral level and that negatively primed facial emotion recognition reveals resembling neural correlates in healthy participants that are known to be associated with a negative bias in response to facial expressions in patients with schizophrenia and borderline personality disorder (Donegan et al., 2003; Mier et al., 2014; Minzenberg et al., 2007), especially enhanced amygdala, NAcc and STS activation. Further, our results suggest that incongruence may lead to negative shifts in the evaluation of positive facial expressions. We conclude that emotion recognition in healthy people is affected by preceding context, albeit the effect seems to be less pronounced than it has been found in previous studies in patients (Fenske et al., 2015; Hooker et al., 2011). Further, we assume that these context driven biases in emotion recognition present on a continuum ranging from aberrations in brain responses to aberrations in overt behavior.

4.1.6 Supplementary materials

Table S1. List of presented IAPS-pictures separated in sets for each face category.

	neutral		negative	
	name	picture-code	name	picture-code
SET 1 - primes prior to happy facial expressions	Man	2512	Toddler	2095
	Casino	7506	SadChildren	2703
	Chess	2580	Attack	3500
	NeuWoman	2038	OpenGrave	3005,1
	Man	2357	DyingMan	3230
	Woman	2025	Mutilation	3225
	Couple	4605	BeatenFem	6315
	Woman	2513	Infant	3350
	Tourist	2850	Police	6838
	Golfer	8311	ManOnFire	9635,1
SET 2 - primes prior to angry facial expressions	NeutralMale	2499	SadChild	2800
	Market	2597	CarAccident	9910
	Couple	2396	Attack	3530
	Butcher	2235	Suicide	6570
	NeuMan	2102	Hospital	3220
	Office	7550	BurntFace	3101
	Woman	2305	BatteredFem	3180
	Men	2593	Kids	9520
	Mom/Son	2435	Soldier	9410
	Girl	2381	Fire	9921
SET 3 - primes prior to neutral facial expressions	Medicalworker	2394	InjuredChild	3301
	Jet	7620	PlaneCrash	9050
	Bakers	2579	Attack	6350
	Man	7493	Gun	2811
	Factoryworker	2393	Hospital	2205
	Farmer	2191	Mutilation	3051
	Man	2485	BatteredFem	3181
	Shopping	2745,1	BabyTumor	3170
	Woman	2620	Soldier	6212
	Teenager	2870	StarvingChild	9040

Table S2. One-factorial ANOVAs for the comparison of IAPS-picture-Sets separated for valence and arousal.

	SET 1	SET 2	SET 3	*F*	*p*	total
negative						
valence	1.99 (1.36)	2.02 (1.33)	2.02 (1.40)	0.023	0.977	2.01 (1.36)
arousal	6.00 (2.30)	6.06 (2.13)	6.00 (2.17)	0.026	0.975	6.02 (2.20)
neutral						
valence	5.47 (1.36)	5.42 (1.23)	5.48 (1.42)	0.096	0.908	5.46 (1.34)
arousal	3.48 (1.97)	3.49 (1.91)	3.53 (2.02)	0.031	0.970	3.50 (1.97)

Note. Means and standard deviations (in parentheses) of the matched picture sets on the basis of norm data of ratings for valence and arousal from Lang, P.J., Bradley, M.M., & Cuthbert, B.N. (2008). International affective picture system (IAPS): Affective ratings of pictures and instruction manual. Technical Report A-8. University of Florida, Gainesville, FL.

Table S3. Interaction effect of face valence x prime valence: post-hoc pairwise comparisons.

Neg – Neu	Mean differences	Standard error	p_{adj}	95%-CI
happy	-7.94	2.11	< 0.001	-12.24 – -3.63
neutral	-0.36	1.74	0.840	-3.91 – 3.21
angry	2.13	1.56	0.182	-1.06 – 5.31

Note. Neg = negative priming, Neu = neutral priming, adj = Bonferroni-adjusted.

Table S4. Main effect of face valence on valence ratings: post-hoc pairwise comparisons.

	Mean differences	Standard error	p_{adj}	95%-CI
happy - neutral	57.66	4.25	< 0.001	46.90 – 68.43
happy - angry	95.81	6.66	< 0.001	78.91 – 112.70
neutral - angry	38.15	3.24	< 0.001	29.92 – 46.37

Note. adj = Bonferroni-adjusted.

4.1.6.1 Effects of negative priming on certainty ratings

A 2 (prime valence) x 3 (face valence) repeated measures ANOVA was conducted to explore priming effects on certainty ratings. There was no significant interaction between prime valence and face valence, as well as no main effect of prime valence in this analysis. However, a main effect of face valence ($F(2;60) = 13.28$, $p < 0.001$, $f = 0.89$) was revealed. Post-hoc comparisons are shown in table S5: Participants' ratings of certainty differed between the face conditions with the highest reported certainty for happy faces ($M = 81.07$, $SD = 14.02$) followed by negative facial expressions ($M = 76.60$, $SD = 14.94$). For neutral

facial expressions they reported the lowest certainty (*M* = 70.99, *SD* = 17.81) compared to happy and negative faces. Negative primes did not affect certainty ratings significantly.

Table S5. Main effect of face valence on certainty ratings: post-hoc pairwise comparisons.

	Mean differences	Standard error	p_{adj}	95%-CI
happy - neutral	10.07	2.43	0.001	3.91 – 16.24
happy - angry	4.46	1.41	0.010	0.89 – 8.02
angry - neutral	5.61	1.90	0.018	0.79 – 10.44

Note. adj = Bonferroni-adjusted.

4.1.6.2 Effects of negative priming on reaction times

A 2 (prime valence) x 3 (face valence) repeated measures ANOVA was conducted to explore priming effects on reaction times. In this analysis no significant prime valence x face valence interaction between occurred. However, a main effect of priming was revealed (*F(1;30) = 8.78, p = 0.006, f* = 0.78). Bonferroni-adjusted post hoc comparison showed that reaction times for the valence ratings were shorter after preceding negative (*M = 687.18, SD = 222.86*) compared to neutral preceding pictures (*M = 751.86, SD = 230.77*) (see table S6).

Table S6. Main effect of prime valence on reaction times for valence ratings: post-hoc pairwise comparison.

	Mean differences	Standard error	p_{adj}	95%-CI
negative - neutral	-64.68	21.83	0.006	-171.25 – 24.73

Note. adj = Bonferroni-adjusted.

Furthermore, a main effect of face valence was shown (*F*(2;60) = 12.22, *p* < 0.001, *f* = 0.87). Bonferroni-adjusted post-hoc comparisons revealed that reaction times for angry faces *(M = 646.17, SD = 208.80)* were shorter than for happy facial expressions *(M = 719.57, SD = 212.13)* and neutral facial expressions (*M = 792.83, SD = 286.32*). Differences between happy and neutral faces were not significant (table S7).

Table S7. Main effect of face valence on reaction times: post-hoc pairwise comparisons.

	Mean differences	Standard error	p_{adj}	95%-CI
happy - neutral	-73.26	38.64	0.203	-171.25 – 24.73
happy - angry	73.39	20.79	0.004	20.68 – 126.11
neutral - angry	146.65	26.73	< 0.001	78.87– 214.43

Note. adj = Bonferroni-adjusted.

4.1.6.3 Valence and arousal ratings of experimental stimuli after the experiment

Ratings of experimental stimuli were analyzed conducting one factorial repeated measures ANOVAs separately for valence and arousal ratings of the facial expressions (see table S8, S9) and paired t-tests for ratings of the prime pictures (see table S10).

Table S8. Repeated measures ANOVAs for ratings of face stimuli: main effects.

	happy	neutral	angry	*F*	*p*
Valence	*M = 6.60*	*M = 4.46*	*M = 3.15*	162.74	< 0.001
	(SD = 0.64)	(*SD* = 0.39)	*(SD = .96)*		
Arousal	*M = 2.73*	*M = 2.44*	*M = 3.57*	12.13	< 0.001
	(SD = 1.53)	*(SD = 1.24)*	*(SD = 1.92)*		

Table S9. Repeated measures ANOVAs for ratings of face stimuli: post-hoc pairwise comparisons for a) valence ratings and b) arousal ratings.

a)

valence	Mean differences	Standard error	p_{adj}	95%-CI
happy - neutral	2.15	0.15	< 0.001	1.77 – 2.53
happy - angry	3.46	0.26	< 0.001	2.80 – 4.11
neutral - angry	1.31	0.15	< 0.001	0.93 – 1.70

adj = Bonferroni-adjusted.

b)

arousal	Mean differences	Standard error	p_{adj}	95%-CI
happy - neutral	0.29	0.23	0.679	-0.30 – 0.88
angry - happy	0.84	0.27	0.011	0.16 – 1.52
angry - neutral	1.23	0.21	< 0.001	0.59 – 1.66

Note. adj = Bonferroni-adjusted.

Table S10. T-tests for the comparison of ratings for prime pictures.

	neutral prime	negative prime	*T*	*p*
Valence	*M = 5.79 (SD* =0.68*)*	*M* = 2.31 (*SD* =0.*72*)	15.86	<0.001
Arousal	*M* = 2.55 (*SD* =1.19)	*M = 6.26 (SD* =1.51*)*	-14.10	<0.001

Table S11. Activation during presentation of negative scenes compared to the presentation of neutral scenes, p = 0.001 uncorrected, k = 10.

Negative IAPS > neutral IAPS			MNI			
Area	BA	k	x	y	z	T-value
Fusiform Gyrus	37	50	-42	-64	-7	4.77
Visual association cortex	19	25	48	-58	-7	4.70

Note. BA = Brodmann area, coordinates = MNI (Montreal Neurological Institute) coordinates of the peak voxel in the cluster, k = cluster-size.

Table S12. Main-effect of prime valence, p = 0.001, k = 10.

			MNI			
Area	BA	k	x	y	z	F-value
Lentiform Nucleus (Putamen)		164	30	-13	-1	33.54
Lentiform Nucleus			30	-4	-4	29.35
Lentiform Nucleus			27	5	11	14.21
Caudate		29	6	2	11	25.87
Lentiform Nucleus		181	-30	-4	2	21.48
Lentiform Nucleus			-27	-7	14	21.42
Lentiform Nucleus			-30	-13	-4	20.19
Thalamus (Pulvinar)		13	15	-37	14	18.87
Inferior Frontal Gyrus	BA 45	24	60	26	20	18.62
Supramarginal Gyrus	BA 40	87	60	-58	26	17.64
Anterior cingulate	BA 33	15	6	5	23	16.72
Culmen		26	9	-46	-7	15.18
Lingual Gyrus	BA 18	15	0	-85	-16	14.73
Inferior Occipital Gyrus	BA 17		-9	-94	-16	12.98
Superior Temporal Gyrus	BA 22	11	69	-43	5	14.52
Superior Temporal Gyrus	BA 38	15	42	14	-19	14.28

Note. BA = Brodmann area, coordinates = MNI (Montreal Neurological Institute) coordinates of the peak voxel in the cluster, k = cluster-size.

Table S13. Interaction prime valence x face valence $p = 0.001$, $k = 10$.

			MNI			
Area	BA	k	x	y	z	F-value
Medial Frontal Gyrus	10	181	9	53	20	11.43
Medial Frontal Gyrus	10		-3	56	17	10.61
Superior Frontal Gyrus	10		-6	62	29	8.97
Postcentral Gyrus	2	20	36	-31	38	11.32
Subcallosal Gyrus	25	21	-3	14	-13	11.30
Fusiform Gyrus	37	10	42	-52	-7	10.82
Parahippocampal Gyrus/Amygdala		39	-24	-4	-16	10.47
Culmen		16	9	-28	-34	9.73
Middle Temporal Gyrus	21	33	66	-25	-10	9.35
Middle Temporal Gyrus	21		54	-28	-7	8.54
Inferior Temporal Gyrus	21	11	57	-13	-25	9.29
Inferior Temporal Gyrus	20	10	60	-34	-22	9.26
Amygdala/Parahippocampal Gyrus		17	18	-1	-19	9.17
Amygdala/Uncus			27	-1	-25	7.51
Superior Frontal Gyrus	9	11	-6	56	41	8.96
Inferior Temporal Gyrus	20	10	-57	-22	-22	8.34
Middle Temporal Gyrus	21	10	45	11	-34	8.11
Superior Temporal Gyrus	38		36	14	-34	7.92

Note. BA = Brodmann area, coordinates = MNI (Montreal Neurological Institute) coordinates of the peak voxel in the cluster, k = cluster-size.

5 GENERAL DISCUSSION

The present dissertation aimed at investigating the nature of emotion recognition deficits in BPD patients in general and the negative bias in particular, as well as its specificity for BPD, and its dependence on influencing factors. Addressing this issue seems highly relevant as many studies found heterogeneous results with respect to emotion recognition deficits in BPD (Mitchell et al., 2014). A deeper understanding of these impairments is crucial, since a negative perceptual bias might explain the interpersonal difficulties in BPD. It could thereby strongly contribute to the psychological strain of the disorder as well as the resulting general healthcare costs. The paradigm we used to disentangle influences on emotion recognition assessed the ability to suppress reactions to irrelevant stimuli, and the recognition performance as well as the resulting error patterns under time constraints. To realize this aim within one task, a combined affective priming emotion recognition task was developed and applied to patients with BPD and patients with schizophrenia as well as to healthy participants. Emotion recognition performance and the negative bias were assessed via behavioral measures and during neuroimaging. Additionally, associations between these measures and potentially relevant symptomatology were examined. In the first study, the influences of task-irrelevant, social-emotional information and time pressure on the occurrence of the negative bias were investigated in a sample of female BPD patients and female healthy controls (study 1a). These factors were also explored in an extended sample including a clinical comparison group of schizophrenia patients as well as male participants (study 1b). Another aim of this dissertation was to assess the neurobiological mechanisms of biased emotion recognition. Therefore, the initial paradigm was adapted and applied during fMRI in a healthy sample (study 2). Emotion regulation abilities and other psychopathological indices were collected in all samples to gain further insight into associated states and traits.

5.1 Summary of study results

BPD as well as schizophrenia patients both showed clear emotion recognition deficits. Although, especially in schizophrenia previous studies reported an overall impairment in emotion recognition (Kohler et al., 2003) we found deficits in both patient groups only when processing time was restricted and not independent of

interfering emotional information. In these particular instances, deficits were obtained especially for neutral facial expressions and partly also for positive faces, but not for negative facial expressions. This points to an intact or referring to previous literature regarding BPD even heightened sensitivity for negative information (e.g. Lynch et al., 2006; Wagner & Linehan, 1999), even under conditions of reduced processing capacity and interfering information. The deficits in the recognition not only of neutral facial expressions, but also of positive facial expressions might increase difficulties in social interaction. Possible explanations might be the attribution of negative emotions to others, but also preventing the perception of cues signaling affiliation and safety (Bertsch, Hillmann, & Herpertz, 2018).

The negative bias in emotion recognition for neutral facial expressions in BPD and schizophrenia is in line with previous findings (Daros et al., 2013; Kohler et al., 2003; Mitchell et al., 2014). It did not differ between patient groups, although schizophrenia patients were more impaired in emotion recognition than BPD patients. This difference in general impairment could be due to the fact that schizophrenia patients suffer from considerably stronger cognitive disturbances (Javitt, 2009; Volk & Lewis, 2014) than BPD patients. Due to this overall impaired functioning, the experimental task might be more demanding for schizophrenia patients than for BPD patients resulting in pronounced deficits in emotion recognition performance. This assumption is supported by correlations of neurocognitive performance with emotion recognition in schizophrenia (Kohler et al., 2000).

Interestingly, these deficits disappeared when the evaluation of facial expressions was separated from other possible interfering information and conducted without any time constraints. That is, in the self-paced valence ratings for facial expressions, which were obtained after the main experiment in which faces were not preceded by emotional scenes, patients and healthy controls performed comparably well in emotion classification. This is in line with previous studies that did not find an emotion recognition deficit and a negative bias in BPD at all (Fertuck et al., 2009; Lynch et al., 2006) or only under specific circumstances, such as time constraints (Dyck et al., 2009).

It is important to mention that valence and arousal of the stimuli were rated separately after the experiment and while valence ratings of facial expressions did not differ between groups, the elicited arousal by facial stimuli was evaluated with higher values in the patient groups (especially in BPD patients for neutral and

negative faces). Attributing a higher arousal to facial expressions, especially to facial expressions which do not contain emotional information, points to a fundamental difference in the perception of facial expressions in BPD, which could be caused by the hyperreactive amygdala (e.g. Donegan et al., 2003; Mier et al., 2013), higher negative affect in general as well as higher aversive tension (as measured in study 1 by PANAS and DSS). This might represent a vulnerability that does not per se lead to a negative bias in facial emotion recognition, but causes it when other factors reduce processing capacity and top-down modulated interference reduction. We suggest that higher arousal in the course of the experiment, as well as higher arousal values ascribed to the stimuli might therefore also contribute to the negative bias.

Although error patterns in the two patient groups looked almost identical, there were distinctive associations between clinical measures and negatively biased responses. While in BPD in comparison to schizophrenia a stronger relation to emotion dysregulation was found, the level of positive and negative symptoms seemed to be closely related to the occurrence of a negative bias in schizophrenia suggesting differences in the mechanisms of developing negatively biased perceptions.

Comparing female and male patients did not reveal a differential pattern in emotion recognition performance in general or regarding the negative bias in particular. That is, female and male participants of both clinical groups showed comparable error rates in recognizing emotions in facial expressions independent of the emotional valence they judged. In contrast, female healthy controls performed better than male healthy subjects.

Study 2 used an adapted version of the paradigm to investigate neural correlates of negatively primed emotion recognition in healthy participants. Negative priming increased activation in amygdala, STS, and NAcc. These activation patterns point to a rather emotion-driven processing state – a pattern one would expect to find in BPD patients. This supports the idea of increased emotional processing of non-emotional information in patients with BPD.

Based on previous theories and current research findings, a theoretical model of the occurrence of the negative bias will be proposed in the following section. Based on this theoretical model, future research perspectives will be discussed. Furthermore, clinical and psychotherapeutic implications derived from the insights into preconditions promoting the occurrence of the negative bias will be presented.

5.2 Models of a negative bias in borderline personality disorder and schizophrenia

The proposed model (figure 1) summarizes the current state of existing literature as well as the findings of the studies derived from the present dissertation. Neurobiological models for facial emotion recognition processes assign the amygdala a crucial role in disturbed emotionality in BPD patients (e.g. Mitchell et al., 2014). It is assumed that hyperactivation of the amygdala in concert with reduced modulatory control by the PFC might provide a framework based on which subsequent disturbances in social cognition are composed. Emotional hypersensitivity and emotion dysregulation end up in negatively biased perceptions, especially under conditions of frequently changing and therefore interfering emotional information and short processing time. The model of a negative bias in schizophrenia (adapted from Mier & Kirsch, 2017) shows a substantial overlap with the model for BPD with regard to neurobiological findings of a hyperactivation in the amygdala as well as reduced coupling between prefrontal and subcortical regions. In contrast to BPD, reduced prefrontal control of the NAcc (Cook et al., 2012) in addition to hyperactivity of the amygdala is supposed to contribute to the negative bias for neutral facial expressions (Mier & Kirsch, 2017). Schizophrenia patients consistently showed functional alterations in the ventral striatum (i.e. the NAcc) which are associated with aberrant salience processing (Heinz & Schlagenhauf, 2010; Kapur, 2003; Winton-Brown, Fusar-Poli, Ungless, & Howes, 2014). In contrast to that, the functioning of the NAcc in BPD remains rather unexamined. An influence of alterations in salience processing might be also possible in BPD given the heightened emotional sensitivity. There were further hypotheses of dopamine dysfunction contributing to emotion dysregulation, impulse control and cognitive-perceptual disturbances in BPD, but clear empirical evidence for that is pending (Friedel, 2004). Since successful social interactions can be highly rewarding (Krach, Paulus, Bodden, & Kircher, 2010), negatively biased perception in BPD might also entail a loss of potential reward, which could also have a neural correlate in the reward system.

The role of the STS also seems to differ between the two patient groups. While in BPD aberrations of STS functioning are more heterogeneous regarding the direction of either hyper- or hypoactivation (Koenigsberg et al., 2009; Mier et al., 2013) and were rather associated to higher social-cognitive processes like theory of mind (Mier et al., 2013), its hyperactivation in schizophrenia was associated with

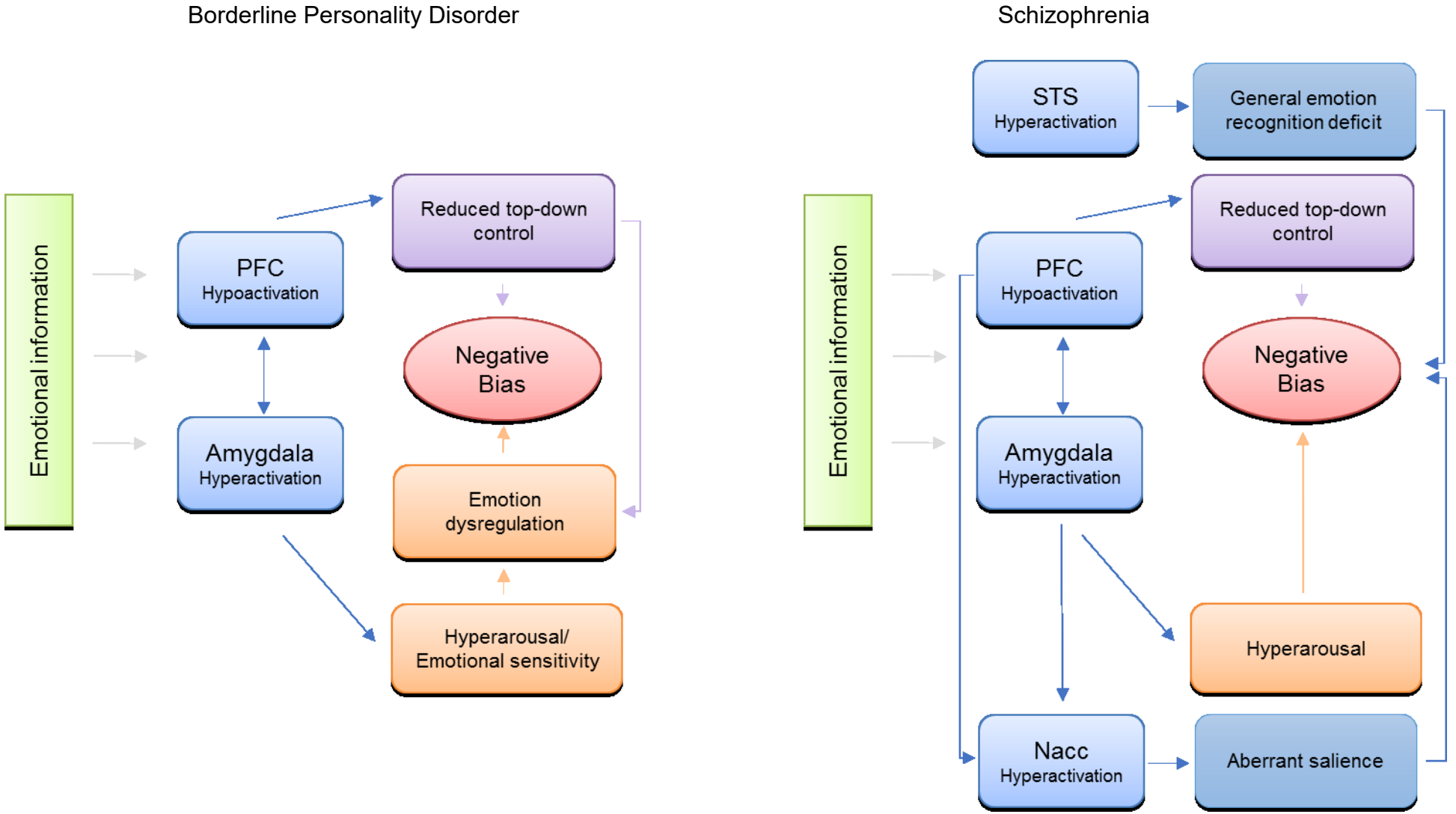

Figure 1. Neurobiological models of a negative bias in Borderline Personality Disorder and schizophrenia

processing of neutral faces and emotion recognition (Mier et al., 2010; Pinkham, Hopfinger, Pelphrey, Piven, & Penn, 2008).

Modulation of the increased emotional processing seems to be impaired due to abnormal connectivity of the amygdala with the PFC/ACC resulting in reduced top-down control in cortico-limbic circuits. The reduced inhibition of the emotionally driven hyperarousal might provide the neural underpinning of the less adequate and effective regulation strategies frequently observed in BPD patients. In BPD there are several hints for a disturbed top-down modulation in the context of emotional processing involving these areas (e.g. Bertsch et al., 2018; Koenigsberg et al., 2009). This is for example indicated by reduced connectivity between the amygdala and the prefrontal cortex (Minzenberg et al., 2007; Ruocco et al., 2013). Hypoactivation in frontal regions in concert with limbic hyperactivation was also revealed during explicit emotion regulation (Schulze et al., 2011), which reflects a typical top-down process. A deficit in emotion regulation should therefore also be associated with the negative bias. We found such a correlation between difficulties in emotion regulation and the negative bias in BPD. Schizophrenia patients, just like BPD patients, reported higher negative affect and enhanced arousal levels compared to healthy subjects. Moreover, they also showed significantly pronounced emotion dysregulation compared to healthy controls. However, in contrast to BPD, emotion dysregulation seemed to play a minor role in the occurrence of a negative bias in this disorder. This remarkable difference between BPD and schizophrenia cannot be sufficiently explained by the amount or intensity of emotion dysregulation, since there was also a significant association of emotion dysregulation with the negative bias in healthy subjects, i.e. the group with the lowest emotion regulation difficulties at all.

Also, our findings are in line with another study that revealed no association between social cognition and emotion regulation in schizophrenia (Rowland et al., 2013). Instead, symptom severity and especially positive symptoms appear to be more relevant for the occurrence of negatively biased responses. Since the development of positive symptoms is explained by aberrant salience processing and difficulties in the integration of newly incoming stimuli to previously processed information (Blackwood et al., 2001; Freeman, 2007; Moritz & Woodward, 2006), alterations in NAcc functioning might contribute to the emergence of the negative bias. As mentioned before, not much is known about the role of the NAcc in BPD. However, there is some evidence in BPD for interactions between disturbed

emotional processing and altered activation in the reward system (Enzi et al., 2013). It could be an interesting research question to investigate whether there is aberrant salience attribution also in BPD, or whether the disturbance in salience attribution differentiates between BPD and schizophrenia. These questions would have to be answered before conclusions regarding the specificity of the correlation between negative bias and positive symptoms in schizophrenia can be finally drawn.

Since BPD patients often present with negative prior experiences and corresponding assumptions, these preconditions might also contribute to more negative evaluations. If the hyperreactivity of the emotion processing brain network is primarily a result of previous experiences or rather the prerequisite for the development of the prominent emotion dysregulation in response to negative experiences, cannot be concluded from the present findings. According to the biosocial theory, BPD develops as a result of the interaction between biological vulnerabilities and environmental risk-factors (Crowell, Beauchaine, & Linehan, 2009). The previously presented results provide clear evidence for the proposed relation of impaired emotion processing and difficulties in emotion regulation. Neuroimaging data points to the role of the described network as a neural mechanism mediating emotional interference and disturbed evaluation of emotional stimuli.

In daily life people are confronted with a huge amount of emotional information that changes with high frequency whereby a large part of this information is actually not related to previous information. For BPD patients who have a hyperreactive amygdala in combination with a prolonged habituation and a reduced top-down modulation this might result in interference of subsequently processed emotional information. That is, processing of one emotional input has not yet finished when the next stimulus encounters the social cognitive processing network. Emotional stimuli of different valences being processed at the same time and in absence of intact inhibitory processes might yield mixed-up emotions that can hardly be discriminated and cannot be attributed correctly to their original source. In summary, amygdala hyperreactivity could provide the neural basis for emotional dysregulation that comprises a high sensitivity for emotional cues, intense reactions to them and a long persistence of emotional reactions. Reduced top-down modulation adds to it by lacking adequate regulation strategies. The result is a highly emotion-driven subjective experience and behavior characterized by negatively biased perceptions.

Compensation for these disturbances requires a setting that provides enough time and capacity for conscious evaluation and reappraisal of automatically shifted responses.

5.3 Future research

Since the present sample of study 2 only consisted of healthy participants, patients with BPD as well as schizophrenia should also be included in the fMRI experiment to study the neural correlates and to test the previously described model assumptions. The inclusion of patient samples should further provide detailed information about group differences in the adapted task. We would assume that patients with BPD will show a greater susceptibility to the negative preceding emotional information leading to an augmented negative bias as well as corresponding amygdala hyperreactivity in concert with reduced PFC modulation. Connectivity analyses should be performed to further elucidate the interplay of relevant brain areas and the assumption of reduced top-down control. Originally, the inclusion of the patient samples was also planned for the current thesis. However, unfortunately it was not possible to acquire a sufficient sample size in the patient groups within the realms of time limitations for a doctoral thesis.

As mentioned in the meta-analysis of Mitchell and colleagues, there is a great diversity of tasks, stimuli, sample characteristics and methods in the research field of emotion recognition and biased perception in BPD (Mitchell et al., 2014). This dissertation tried to explain some of the contradicting results by integrating different influencing factors in one task. Although, we were able to replicate previous findings and add further insights to the existing knowledge, future research would clearly benefit from further integration of existing evidence and comparison of inconsistent findings through direct experimental manipulation. A direct comparison of response formats in emotion recognition paradigms would be necessary to rule out that differences in response patterns are solely attributable to differing assessments of emotion recognition. A classification of valence might require other capacities than a differentiation between specific emotional categories. Forced choice between alternatives could also be different from continuous ratings. Such clarification might further be important for administered stimulus material, such as static or dynamic facial expressions or information stemming from different modalities. However, only few studies already addressed this particular issue, for example by using multi-modal information (Minzenberg, Poole, & Vinogradov, 2006; Niedtfeld et al., 2017) or by

comparing different processing steps in reaction to a changing stimulus (Lowyck et al., 2016). In our study, interfering emotional information was not investigated without time constraints. Therefore, potential confounding between emotional interference and timing could be seen as a limiting factor that hampers the interpretation of the observed effects. One important adaptation directly suggested by the findings of this dissertation could be, to separate the influence of time constraints and interfering emotional information. Since it is possible that context effects only occur given a restricted processing, emotional interference should be investigated in a completely self-paced design. Furthermore, the investigation of the neural correlates of these influencing factors on the negative bias in patients would be the next consecutive approach. In this context it would also be interesting to test the hypothesis that slow amygdala habituation (Hazlett et al., 2012) directly contributes to the occurrence of a negative bias in BPD. Another region of interest could be the NAcc and the question whether altered salience attribution directly influences the negative bias specifically in schizophrenia, but also in BPD. There are first hints for altered salience also in BPD patients (Catalan et al., 2018; Winter, Koplin, & Lis, 2015). For this purpose and to clarify whether the association of positive symptoms with biased perception is specific for schizophrenia, the analysis of correlations with stress related psychotic symptoms in BPD could be helpful. Connecting of all these loose ends could finally complete the fragmentary and to some extent heterogeneous picture of emotion recognition deficits in BPD.

5.4 Implications for psychotherapeutic interventions

BPD patients often report difficulties in describing and labeling their own current feelings and what they derive from (Derks, Westerhof, & Bohlmeijer, 2017). This was supported by the significant higher alexithymia scores in BPD patients compared to healthy controls found in our sample. Furthermore, they face a lot of interpersonal conflicts and disturbances in social relationships. An implication of the recent findings for psychotherapeutic interventions in BPD would be to put a stronger focus on situational factors that could influence current affective states of BPD patients and subsequent evaluations. Although, emotional information might be perceived consciously, it could often not be assigned to the right source and interfering emotions might result in negatively biased perception. BPD patients might tend to be overwhelmed by this variety of incoming information and not be able to discriminate irrelevant emotions from their current affective state and the present situation. To

some extent existing cognitive-behavioral interventions already account for this issue. For example, the SORKC-model (Kanfer & Saslow, 1974) that is widely used in cognitive behavioral therapy does include the organism-variable that explains interactions between a person's traits and the perception of a certain situation. Dialectical behavior therapy (DBT) that has been developed to particularly address problems that frequently occur in BPD comprises even more elaborated methods to target these interactions (Linehan, 1993a). However, none of these interventions specifically aims at reducing interference of subsequently perceived events. That is, most DBT interventions rather focus on interferences arising from biographical experiences and prior knowledge (Bohus & Wolf-Arehult, 2013). Thus, existing interventions may not be sufficiently explicit in helping patients to become more aware of situational and irrelevant emotional interferences. To train them to differentiate between a previous situation and new emotional information that is independent from the earlier perception (e.g. by extending the analysis of the organism-variable) could help to establish more adequate evaluations. There is first evidence for effects of DBT on neural responsiveness to negative stimuli and arousal in the amygdala (Schnell & Herpertz, 2007) as well as for better amygdala habituation after treatment (Goodman et al., 2014). However, which specific interventions led to the effect is not yet clearly described. Further research on the effect of well-defined psychotherapeutic interventions on interference reduction is needed to evaluate whether existing interventions really address this phenomenon sufficiently or whether adapted interventions are necessary to address the disturbed top-down modulation in the case of emotional interference. Andreou and colleagues (2015) argued that in the treatment of social-cognitive deficits in BPD and schizophrenia, there is no "one-size fits all approach". First promising evidence however for an effective reduction of contextual influences in schizophrenia was provided by Chung and Barch (2011). An enhancement of attention to contextual cues evoked by a specific categorization resulted in less interference (Chung & Barch, 2011). Combined with an emotion regulation approach this might also be a promising intervention for BPD patients.

5.5 Conclusion

Even though in the past decades a growing number of studies have been conducted on emotion recognition in BPD, still many questions remain open. Especially the numerous possible interdependencies between neurobiological,

behavioral and situational factors contributing to the social-cognitive deficits occurring in daily life of the patients appear to be barely understood. Since social misunderstandings and interpersonal disturbances are core problems of patients with BPD, extending the existing knowledge about these impairments and their basis is crucial for progress in treatment development.

We claim that emotion recognition based on facial expressions is an important ability for successful social interaction. Facial expressions frequently serve as the main or even only source of information and impairments in processing facial expressions can initiate a cascade of misattributions that substantially disturb social interactions. Therefore, we applied a paradigm that provides the possibility to not only look at different factors influencing this process but also to describe the nature of the impairments in more detail.

Given the data presented and discussed above we conclude that BPD patients do not show a general emotion recognition deficit, but a specific impairment for emotional expressions that are not negative. This deficit seems to be pronounced especially for neutral facial expressions, where obvious indicators for evaluations are missing. It is worth noting that the stimulus material and response format may considerably account for heterogeneous findings in previous studies.

Moreover, BPD patients exhibit a negative bias in the recognition of neutral expressions only under specific circumstances. We have shown that restricted processing time is an essential factor for the bias to occur. Additionally, preceding emotional contextual information, which is not directly associated to the actual content, but interferes with the subsequent emotion recognition, has an influence on negatively biased perception. This could be caused by an interaction of aberrant bottom-up (hyperarousal) and top-down processing (emotion dysregulation), resulting in an aggravated negative bias.

We could further show that processing of non-emotional facial expressions that were preceded by negative emotional information resulted in responses in brain regions linked with emotional processing, even in healthy participants. This response pattern might not be strong enough to result in disturbances in emotion recognition behavior in healthy people, but could be much more pronounced in BPD patients and thereby explaining the negatively biased behavioral responses.

However, such a negative bias in the evaluation of neutral faces emerges not uniquely in BPD patients but also in schizophrenia. Nevertheless, there seem to be

disorder-specific mechanisms, such as differential associations with emotion dysregulation, underlying comparable overt behavior. These specificities should be further clarified in additional experiments and might inform adaptations of currently applied psychotherapeutic interventions.

This dissertation was the first to investigate the influence of task-irrelevant preceding information and time pressure on facial emotion recognition by directly comparing BPD with schizophrenia patients. It provides valuable insights into neurobiological and behavioral factors, influencing the occurrence of a negative bias in emotion recognition in BPD and can be indicatory for adaptations in psychotherapeutic treatment of core symptoms in BPD.

6 SUMMARY

Patients with Borderline Personality Disorder (BPD) suffer from severe emotion dysregulation, instable relationships, and tend to perceive their interaction partners as hostile and rejecting. Causal to these negative perceptions of others might be deficits in social cognition, in particular a negative bias in emotion recognition (i.e. the attribution of negative emotions to neutral or ambiguous facial expressions). However, until now, findings regarding such a negative bias are heterogeneous, and influencing factors of its occurrence are neither well-described, nor systematically examined. The aim of this dissertation was to investigate internal and external determinants, as well as the specificity of the negative bias in BPD.

A behavioral study combining affective priming, emotion recognition and a manipulation of the available processing time in one paradigm was conducted with BPD patients, a healthy and a clinical control group of schizophrenia patients. The results support the existence of a negative bias in patients with BPD, and suggest that preceding emotional information, as well as available processing time are relevant factors for the occurrence of the negative bias. In addition, an association between the negative bias and emotion dysregulation was revealed in BPD. While schizophrenia patients showed a similar error pattern, the extent of negatively biased responses was not associated with emotion dysregulation, pointing to distinct mechanisms underlying the disturbed processing of facial expressions.

Further, an adapted task of affective priming combined with emotion recognition was applied to healthy participants in a functional magnetic resonance imaging study. Increased activation due to negative preceding information was revealed in brain regions such as the amygdala, the superior temporal sulcus, and the nucleus accumbens. These areas have been previously found to be disturbed in patients with BPD and also in patients with schizophrenia. The results of this study suggest that the brain’s response to facial expressions is sensitive to interfering negative emotional information, possibly reflecting a vulnerability factor for the emergence of the negative bias.

The findings of this dissertation fit well into existing literature of a negative bias in BPD and provide new insights into the mechanisms of disturbed emotion recognition. It was shown that processing time as well as context information

influence emotion recognition. Further, the results indicate a specific association between emotion dysregulation and the negative bias in BPD.

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