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Facial mimicry, facial emotion recognition and alexithymia in post-traumatic stress disorder

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ABSTRACT

Individuals with post-traumatic stress disorder (PTSD) show abnormalities in higher-order emotional processes, including emotion regulation and recognition. However, automatic facial responses to observed facial emotion (facial mimicry) has not yet been investigated in PTSD. Furthermore, whereas deficits in facial emotion recognition have been reported, little is known about contributing factors. We thus investigated facial mimicry and potential effects of alexithymia and expressive suppression on facial emotion recognition in PTSD. Thirty-eight PTSD participants, 43 traumatized and 33 non-traumatized healthy controls completed questionnaires assessing alexithymia and expressive suppression. Facial electromyography was measured from the muscles *zygomaticus major* and *corrugator supercilii* during a facial emotion recognition task. Corrugator activity was increased in response to negative emotional expressions compared to zygomaticus activity and vice versa for positive emotions, but no significant group differences emerged. Individuals with PTSD reported greater expressive suppression and alexithymia than controls, but only levels of alexithymia predicted lower recognition of negative facial expressions. While automatic facial responses to observed facial emotion seem to be intact in PTSD, alexithymia, but not expressive suppression, plays an important role in facial emotion recognition of negative emotions. If replicated, future research should evaluate whether successful interventions for alexithymia improve facial emotion recognition abilities.

1. Introduction

Processing of emotional stimuli entails both automatic components and more deliberate cognitive processes. The measurement of facial muscle activity using electromyography (EMG) can provide valuable information on automatic components of emotion processing. Research using facial EMG has shown that after seeing others' facial emotional expressions, automatic facial muscle activations congruent with the emotional valence of the expressions can occur in the observer (Dimberg, 1982). Precisely, increased muscle activity in the *zygomaticus major* muscle (associated with smiling) can be seen when observing facial expressions of positive valence, and increased muscle activity in the *corrugator supercilii* (associated with frowning) when observing facial expressions of negative valence (literature review by Hess & Fischer, 2013). This increase in muscular activity can be seen as early as 200 ms after stimulus onset in the *corrugator* and after 500 ms in the *zygomaticus* (Achaibou, Pourtois, Schwartz, & Vuilleumier, 2008). These responses occur automatically (Dimberg, Thunberg, & Grunedal, 2002), generally without the awareness of the expresser (Dimberg, Thunberg, & Elmehed, 2000). Based on such findings, this phenomenon has been termed rapid and automatic 'facial mimicry'.

Investigation of facial mimicry can thus provide information on whether there are abnormalities in automatic emotion processing. Deficits and abnormalities in automatic facial mimicry have been reported for several clinical populations, e.g. autism spectrum disorders (McIntosh, Reichmann-Decker, Winkielman, & Wilbarger, 2006), borderline personality disorder (Matzke, Herpertz, Berger, Fleischer, & Domes, 2014), depression (Zwick & Wolkenstein, 2017), and schizophrenia (Varcin, Bailey, & Henry, 2010). While there is evidence that individuals with PTSD show reduced facial expressivity (Fujiwara, Mizuki, Miki, & Chemtob, 2015; Kirsch, Krause, Spang, & Sachsse, 2008) and that facial expressivity increases over the course of a trauma

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therapy (Kirsch et al., 2018), the authors are unaware of published research investigating facial mimicry in PTSD.

There are, however, findings on facial mimicry from PTSD-related populations. For example, there is evidence from research on maltreated street boys that early adverse experiences are associated with reduced facial mimicry in response to positive and negative facial emotional expressions (Ardizzi et al., 2013). Furthermore, in female individuals with borderline personality disorder who show high rates of childhood traumatization (Battle et al., 2004), facial mimicry in response to negative valence emotional expressions was enhanced whereas facial mimicry in response to positive valence emotional expressions was decreased (Matzke et al., 2014). However, women who experienced childhood sexual or physical abuse by close others showed increased facial mimicry to positive valence facial emotional expressions and less facial mimicry to negative valence facial emotional expressions than women without (or low) childhood abuse (Reichmann-Decker, DePrince, & McIntosh, 2009). These mixed findings indicate that research investigating automatic facial mimicry directly in individuals with PTSD is needed.

Perception of emotional cues can also make their decoding necessary as is the case in facial emotion recognition. Akin to facial mimicry, facial emotion recognition deficits are commonly reported from clinical populations, e.g. autism spectrum disorders (Harms, Martin, & Wallace, 2010), borderline personality disorders (Domes, Schulze, & Herpertz, 2009), and antisocial populations (Marsh & Blair, 2008) and corresponding neural deficits have been identified (Harms et al., 2010; Mitchell, Dickens, & Picchioni, 2014). The published literature on facial emotion recognition in PTSD is sparse. However, research has shown deficits in facial emotion recognition in PTSD for recognition of the negative valence emotions fear and sadness (Poljac, Montagne, & de Haan, 2011), whereas other studies showed reduced recognition of positive valence emotional expression (Passardi et al., 2018), and a negative bias for the interpretations of neutral facial expressions (Passardi, Peyk, Fares-Otero, Schnyder, & Pfaltz, revise and resubmit). Although the findings are not conclusive in regard to the emotions' valence, it seems that there are facial emotion recognition deficits present in PTSD. However, factors underlying these deficits are vastly unknown.

A cognitive style related to emotion processing and emotion recognition in particular is alexithymia. Alexithymia is defined as a difficulty describing, identifying, and differentiating one's own feelings, a tendency to focus attention externally rather than internally, and having a poor imaginary inner life (Sifneos, 1973). Alexithymia has been linked to poor emotion regulation abilities and cognitive processing of emotions (Taylor, 2000). Importantly, not only has alexithymia been associated with deficits in facial emotion recognition in healthy individuals and individuals with eating disorders, somatoform disorders, and Asperger syndrome (Grynberg et al., 2012; Ihme et al., 2014), but research also shows that alexithymia might be a core factor in explaining emotion recognition deficits in clinical populations (Brewer, Cook, Cardi, Treasure, & Bird, 2015; Cook, Brewer, Shah, & Bird., 2013). Accordingly, the 'alexithymia hypothesis' suggests that emotion recognition deficits in individuals with autism spectrum disorder might be due to alexithymia in the subgroup of individuals with strong alexithymia (Bird & Cook, 2013; Brewer, Happe, Cook, & Bird, 2015; Kinnaird, Stewart, & Tchanturia, 2019). There is, in fact, evidence in individuals with autism spectrum disorders and eating disorders that comorbid alexithymia but not the primary disorder is linked to emotion recognition deficits (Brewer, Cook et al., 2015; Cook, Brewer, Shah, & Bird, 2013; Oakley, Brewer, Bird, & Catmur, 2016). Since the prevalence of alexithymia in PTSD is high (meta-analysis by Frewen, Dozois, Neufeld, & Lanius, 2008), it is plausible that difficulties in recognition of facial expressions could be linked to increased alexithymia in PTSD. To date, no published research exists on facial emotion recognition in relation to alexithymia in PTSD.

Related to emotion processing *styles* are emotion regulation *strategies*. Expressive suppression is an emotion regulation strategy that requires cognitive control and entails the deliberate inhibition of the ways an experienced emotion is usually expressed, e.g. by restraining facial expressions (Gross & Levenson, 1993). This phenomenon is more commonly observed in individuals with PTSD compared to controls (Roemer, Litz, Orsillo, & Wagner, 2001) and research has shown that application of emotion suppression is positively associated with PSTD symptomatology and severity (Amstadter & Vernon, 2008; Boden et al., 2013; Chukwuorji, Ifeagwazi, & Eze, 2017; Moore, Zoellner, & Mollenholt, 2008; Shepherd & Wild, 2014), which is further evidenced in a meta-analyis (Seligowski, Lee, Bardeen, & Orcutt, 2015). In addition, alexithymic individuals tend to use expressive suppression as an emotion regulation strategy, possibly stemming from a lack of understanding of their emotions (Laloyaux, Fantini, Lemaire, Luminet, & Larøi, 2015; Preece, Becerra, Robinson, & Gross, 2019). Expressive suppression is an emotion regulation strategy that is effortful, increases psycho-physiological arousal and disturbs memory (Cutuli, 2014; Gross, 2015). It is possible that an inward focus on suppressing the expression of experienced emotions affects the ability to recognise emotions from others' faces where a focus on the cues displayed by the other person is required. This potential association has yet to be investigated.

Aim of the current study was to investigate facial mimicry as well as more higher-order levels of emotion processing in PTSD to gain more information on emotion processing deficits and potential underlying factors in this population. To this end, facial EMG was recorded from the zygomaticus and corrugator muscles while participants watched videos of facial emotional expressions of positive and negative valence that they were asked to recognize by assigning labels. It was hypothesized that individuals with PTSD would show reduced facial mimicry in both muscles compared to traumatized (TC) and a non-traumatized healthy controls (HC). We furthermore hypothesized that alexithymia scores as well as expressive suppression during processing of facial expressions would be higher in the PTSD group than in TC and HC and predict decreased facial emotion recognition in PTSD.

2. Method

2.1. Participants

Recruitment took place via advertisements, online platforms, mailing lists, postings, the clinical information system of the University Hospital of Zurich (USZ), participant pools of former studies, and via therapists at the USZ or other out-patient clinics. For inclusion in the study, participants had to be aged 18–65 years, German native speakers (or equivalent proficiency) with a verbal IQ > 70 (according to a German multiple-choice vocabulary test, WST; Schmidt & Metzler, 1992) and have normal (or corrected to normal) visual functions. Exclusion criteria were lifetime psychotic symptoms, acute suicidality, medications affecting the autonomous nervous system (e.g. tricyclic antidepressants, benzodiazepines, beta-blockers, antiepileptics or antipsychotics), medical conditions biasing psychophysiological measurements (e.g. epilepsy, cardio-vascular diseases), and substance dependency/abuse during the past year.

Data of thirty-eight PTSD participants, 43 TC, and 33 HC, a subset of the sample in Passardi et al. (2018), were analysed. Four participants were excluded (PTSD = 1, TC = 1, HC = 2) from the original sample, due to missing EMG data or missing stimuli triggers.

Table 1 shows the demographic characteristics by group. In line with the criteria for study participation, PTSD individuals fulfilled the disorder-specific criteria according to the German version of the Clinician-Administered PTSD Scale for DSM V (CAPS, Müller-Engelmann et al., 2018). HC participants had never experienced a trauma as defined in the DSM V (American Psychiatric Association, 2013), whereas participants in the TC group had experienced at least one traumatic event but never met criteria for lifetime PTSD according to the CAPS (Müller-Engelmann et al., 2018). Individuals in the PTSD group

Table 1
Participants' demographic characteristics.

	PTSD (n = 38)	TC (n = 43)	HC (n = 33)	Group comparisons[a]
	n (%)	n (%)	n (%)	
Female gender	29 (76%)	28 (65%)	24 (73%)	ns ($\chi^2(2) = 1.29$, p = .524)
	Mean (SD)	Mean (SD)	Mean (SD)	
School years	11.4 (2.7)	11.9 (3.2)	12.1 (3.2)	ns (*p*'s > .279)
Age (years)	38.8 (12.9)	36.7 (12.1)	36.2 (10.3)	ns (*p*'s > .340)
PDS[b]	36.1 (8.8)	8.8 (7.3)	–	PTSD > TC (t(75) = 14.91, p < .001)
BDI[c]	24.4 (8.9)	3.2 (3.1)	2.8 (5.2)	PTSD > TC (t(76) = 13.84, p < .001), PTSD > HC(t(68) = 12.63, p < .001)

[a] PTSD: Post-traumatic stress disorder; TC: Traumatized healthy controls; HC: Non-traumatized healthy controls; ns: not significant.
[b] PDS: Post-traumatic Diagnostic Scale (modified according to DSM-5).
[c] BDI: Beck Depression Inventory.

Table 2
Trauma types by group.

Trauma types	PTSD n (%)	TC n (%)
sexual assault	16 (42.1)	4 (9.3)
non-sexual assault	9 (23.7)	8 (18.6)
accidents	7 (18.4)	18 (41.9)
life-threatening illness	1 (2.6)	2 (4.7)
natural disasters	0 (0)	3 (7.0)
work-related trauma	0 (0)	2 (4.7)
mix of different trauma types	1 (2.6)	2 (4.7)
other traumatic experiences	4 (10.5)	4 (9.3)

experienced on average 3.5 different trauma types (*SD* = 1.6) and individuals in the TC group 2 different trauma types (*SD* = 1.0); see Table 2 for the trauma types per group. A long-lasting or repeated trauma (i.e. type II trauma) was experienced by 63% of the participants in the PTSD group (*n* = 24) and 9% in the TC group (*n* = 4). A deliberately caused trauma was experienced by 74% participants in the PTSD group (*n* = 28) and 33% (*n* = 14) in the TC group.

HC and TC participants did not meet the criteria for a current psychological disorder according to the Mini International Neuropsychiatric Interview (M.I.N.I.; Ackenheil, Stotz-Ingenlath, Dietz-Bauer, & Vossen, 1999). Comorbid disorders in PTSD participants comprised agoraphobia (*n* = 15), bulimia nervosa (*n* = 1), dysthymia (*n* = 10), generalized anxiety disorder (*n* = 6), major depression (*n* = 22), obsessive-compulsive disorder (*n* = 2), panic disorder (*n* = 10), and social phobia (*n* = 7).

2.2. Facial EMG

Psychophysiological data were recorded using Biopac MP150 Systems (Biopac Systems, Inc., Goleta, CA) and AcqKnowledge (AcqKnowledge Software Palo Alto, CA) at a 1000Hz sampling rate. Facial EMG was recorded from the left *corrugator* and *zygomaticus* muscle regions using Ag/AgCl miniature electrodes filled with electrolyte gel, according to the guidelines published by Fridlund and Cacioppo (1986). Skin conductance, electrocardiogram, and respiratory rate were also recorded, the data of which will not be reported here.

2.3. Facial emotion recognition task

Video stimuli were taken from the Amsterdam Dynamic Facial Expression Set–Bath Intensity Variations (ADFES-BIV) (Wingenbach, Ashwin, & Brosnan, 2016); an adaptation of the ADFES (Van Der Schalk, Hawk, Fischer, & Doosje, 2011). From the full stimulus set, 310 stimuli were selected for the facial emotion recognition labelling task. This selection included 10 facial expressions (anger, disgust, sadness, fear, surprise, happiness, pride, embarrassment, contempt, and neutral) displayed by 10 encoders (5 male, 5 female) at 3 levels of expression intensity (low, intermediate and high). An additional 10 videos were selected for practise trials.

The facial emotion recognition task[2] thus included 310 trials in total. Each trial started with a fixation cross in the middle of the screen for either 1000 ms, 1500 ms, 2000 ms, 2500 ms, or 3000 ms (at random) before stimulus presentation. Each stimulus had a duration of 1040 ms showing a neutral face that either remained neutral or developed into one of the 9 emotional expressions. A blank screen appeared for 500 ms after stimulus offset. Thereafter, an answer screen appeared presenting 10 possible answer choices (i.e. one per emotion category).

2.4. Alexithymia

The German version of the Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994) was used to assess alexithymia traits. The TAS-20 is a widely used self-report instrument with good test-retest reliability (r = 0.77), containing 20 items distributed over 3 subscales: difficulty identifying feelings (7 items), difficulty describing feelings (5 items), and externally-oriented thinking (8 items). The answer format is a 5-point Likert-scale ranging from 1 (strongly disagree) to 5 (strongly agree) resulting in a possible minimum score of 20 and a maximum of 100. Scores from 52 to 60 represent possible alexithymia, scores < 52 can be interpreted as no alexithymia and scores > 60 suggest presence of alexithymia. Internal consistency in our study was high ($\alpha = 0.92$).

2.5. Expressive suppression

Participants' tendency to apply emotional suppression during the emotion recognition task was assessed with the widely used German version of the Emotion Regulation Questionnaire (ERQ; Abler & Kessler, 2009) with the 4 items of the expressive suppression subscale. The answering format is rating agreement with each statement from 1 (strongly disagree) to 7 (strongly agree) resulting in a possible minimum score for expressive suppression of 4 and a maximum of 28. An example item is: "When I am feeling positive emotions, I am careful not to express them". The internal consistency in our study was high ($\alpha = 0.83$). Abler & Kessler (2009) reported a test-retest reliability of 0.69 for 3 months.

2.6. Procedure

The study was approved by the Cantonal Ethics Committee of Zurich. All participants gave written informed consent prior to participation and received financial compensation (CHF 130 for HC, 140 for TC, and 150 for PTSD participants) upon completion of the study.

A two-step assessment of inclusion and exclusion criteria was

[2] Group differences in facial emotion recognition between PTSD, trauma controls, and healthy controls were investigated and are reported in Passardi et al. (2018); biases in interpreting neutral facial expressions will be reported in Passardi et al., (revise and resubmit).

performed. First, possible participants were screened by phone-interviews conducted by the first author (SP), the last author (MCP) or by graduate psychology students trained by SP or MCP. For individuals who had experienced at least one traumatic event, the Posttraumatic Diagnostic Scale (PDS), part III (Ehlers, Steil, Winter, & Foa, 1996; Foa, 1995), was administered to assess possible PTSD symptoms and symptom severity. Because the PDS-5 was not yet available during data collection, the PDS was slightly modified to meet DSM V diagnostic criteria. Second, appointments for a diagnostic interview were arranged, which was conducted by trained and supervised psychology students using the M.I.N.I. and the CAPS. The CTQ and PDS were also administered.

The experimental part of the study took part either immediately after the interview or on a second appointment within one week if participants were suitable for participation. Participants first filled out (at home or in the laboratory) online versions of the TAS-20 (Bagby, Ayearst, Morariu, Watters, & Taylor, 2014), the Beck Depression Inventory (Hautzinger, Bailer, Worall, & Keller, 1994) and other questionnaires on anxiety, dissociation and distress; the results of the dissociation questionnaires are reported elsewhere (Passardi et al., 2018).

When participants came to the laboratory for the experimental testing session, they were informed about the study procedure, provided with a participant information sheet to read, and asked to fill out a consent form. Participants then filled out the WST (Schmidt & Metzler, 1992). Thereafter, electrodes for the psychophysiological measurements were attached. These remained attached until the end of the testing session. After a 5-min baseline period, participants performed the emotion recognition task. E-Prime 2.0 was used as stimulus presentation software (Psychological Software Tools Inc.). Participants had to click as quickly as possible on the correct response field using the mouse device. A list with definitions and explanation for each emotion was handed out before the experiment.

After completion of the emotion recognition task, the ERQ and questionnaires on dissociation that are reported elsewhere (Passardi et al., 2018) were administered. The questions of the ERQ were slightly modified so that they were related to the emotion recognition task. That is, participants had to rate how much they used expressive suppression as emotion regulation strategies during the emotion recognition task.

2.7. Data preparation and statistical analyses

EMG data preparation was performed using ANSLAB (Blechert, Peyk, Liedlgruber, & Wilhelm, 2016; www.anslab.net) within MATLAB (Release 2012a, The MathWorks, Inc., Natick, Massachusetts, USA). The EMG signal was 28-Hz high pass filtered, rectified, smoothed using a 50 ms moving average and down sampled to 100Hz. The data of both EMG channels were *z*-transformed within each subject. Data epochs of 2500 ms from 1000 ms before to 1500 ms after stimulus onset were extracted per participant and separately for each EMG channel and emotional valence. The expressions included in the facial emotion recognition task were divided in two valence categories: positive (joy, pride), and negative (anger, fear, sadness, disgust, contempt, and embarrassment). Epochs were baseline-corrected by subtracting the mean of the 1000 ms before movie onset from the entire epoch.

To identify the most relevant time ranges in the EMG responses while correcting for multiple comparisons, nonparametric cluster level statistics as suggested by Maris and Oostenveld (2007) were calculated on the *F*-values of repeated measures ANOVAs that were conducted for each 10 ms sample of the 2500 ms EMG epochs (comprising the 1000 ms baseline window and the 1500 ms response window; resulting in a total of 250 ANOVAs per EMG channel). Clustering was performed on 1000 random permutations of the original participant dataset, separately for the two main effects (group and valence) and the interaction effect (group*valence), using a first level (sample level) error probability of .01 and a cluster level error probability of .01. The mass values presented in the results section represent the sum of significant *F*-values in a cluster. To obtain a measure of effect size, partial eta-squared values were calculated for each cluster based on a repeated measures ANOVA of the averaged EMG signal across all samples in a cluster.

Further analyses were performed in SPSS (IBM SPSS statistics, version 23). The percentages of correct responses averaged across emotions per valence category were calculated for each participant. Independent samples *t*-tests were conducted to investigate group differences on scores of alexithymia, PTSD symptom severity (between the PTSD and TC group), and expression suppression. Bonferroni-Holm correction of the *p*-values was applied to control for multiple testing; the *p*-values presented in the results section are after correction. Stepwise linear regressions were conducted with alexithymia and expression suppression as predictors and recognition accuracy of positive and negative valence emotions as dependent variables. These regressions were conducted for each group. Symptom severity (PDS score) was included as predictor in the linear regression models for the PTSD group and the TC group.

3. Results

3.1. Facial EMG

The cluster level analysis of the *zygomaticus* data revealed one significant cluster for the main effect of 'valence' from 790 ms to 1500 ms (mass = 716.1, $p < .001$, $\eta_p^2 = 0.089$). That is, the activity in the zygomaticus was significantly higher during observation of positive valence emotions than negative valence emotions; Fig. 1. No significant clusters were found for the main effect of 'group' or the 'valence*group' interaction. The cluster level analysis of the *corrugator* data showed one significant cluster for the main effect of 'valence' from 310 ms to 360 ms (mass = 68.3, $p = < .001$, $\eta_p^2 = 0.101$) and another significant cluster from 380 ms to 1500 ms (mass = 10761.9, $p < .001$, $\eta_p^2 = 0.556$). That is, the activity in the corrugator was significantly higher during observation of negative valence emotions than positive valence emotions; Fig. 1. No significant clusters were found for the main effect of 'group' or the 'valence*group' interaction.

3.2. Alexithymia, PTSD symptom severity, and expressive suppression

The PTSD group reported significantly more alexithymia ($M = 56.46$, $SD = 12.47$) than the HC group ($M = 37.91$, $SD = 8.65$, $t(64.28) = 7.29$, $p = .003$) and the TC group ($M = 37.05$, $SD = 8.19$, $t(61.14) = 8.03$, $p = .003$). There was no significant difference in alexithymia scores between the HC and the TC group ($t(72) = 0.44$, $p = .663$).

The PTSD group reported significantly higher PTSD symptom severity ($M = 36.08$, $SD = 8.76$) than the TC group ($M = 8.75$, $SD = 7.27$, $t(75) = 14.91$, $p < .001$).

The PTSD group reported significantly more expressive suppression ($M = 4.57$, $SD = 1.10$) than the HC group ($M = 3.02$, $SD = 1.27$, $t(68) = 5.48$, $p = .003$) and the TC group ($M = 3.09$, $SD = 1.10$, $t(76) = 5.97$, $p = .003$). There was no significant difference in expressive suppression scores between the HC and the TC group ($t(72) = -0.23$, $p = .821$).

3.3. Predicting facial emotion recognition

The model for the PTSD group with alexithymia, symptom severity, and expressive suppression as predictors for recognition of negative valence emotions was significant ($F(1, 35) = 10.46$, $p = .003$) and explained 20.8% of the variance. Alexithymia was a significant predictor for recognizing negative valence emotions (standardized $\beta = -0.480$, $t(35) = -3.23$, $p = .003$), Fig. 2; symptom severity ($\beta = -0.025$, $t(35) = -1.16$, $p = .878$) and expression suppression were excluded from the model ($\beta = 0.004$, $t(35) = 0.02$, $p = .981$).[3] The predictive

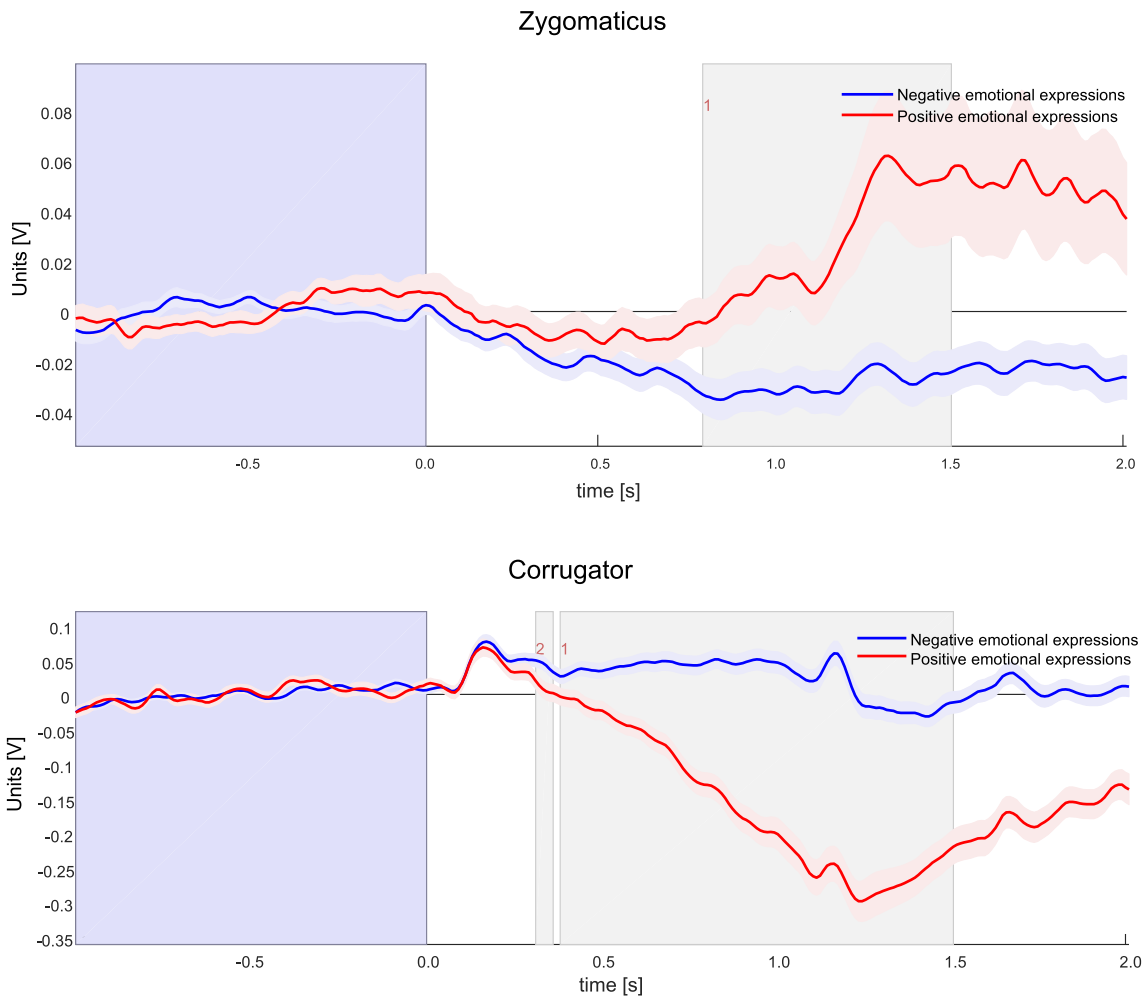

Fig. 1. *Zygomaticus major* and *corrugator supercilii* activations in response to watching facial emotional expressions of positive and negative valence. The grey shaded area represents the clusters with significant differences between the EMG activity from positive vs negative valence stimuli.

model was: recognition of negative valence = 82.37 + (−0.400*TAS-20). The models for the TC group and the HC group when predicting recognition of negative valence emotions were not computed. The models on predicting recognition of positive valence emotions were not computed for any of the 3 groups tested. That is, there was no discernable linear relationship between the predictors and recognition of positive valence emotions.

To follow up on the effect of alexithymia on recognition of negative valence emotion in PTSD,[4] another regression model was run including the 3 subscales of the TAS-20 as individual predictors. The model was highly significant ($F(1, 35) = 17.32$, $p < .001$) and explained 31.2% of the variance. Only the subscale 'externally-oriented thinking' was a significant predictor for recognizing negative valence emotions (standardized $\beta = -0.575$, $t(35) = -4.16$, $p < .001$); 'difficulty

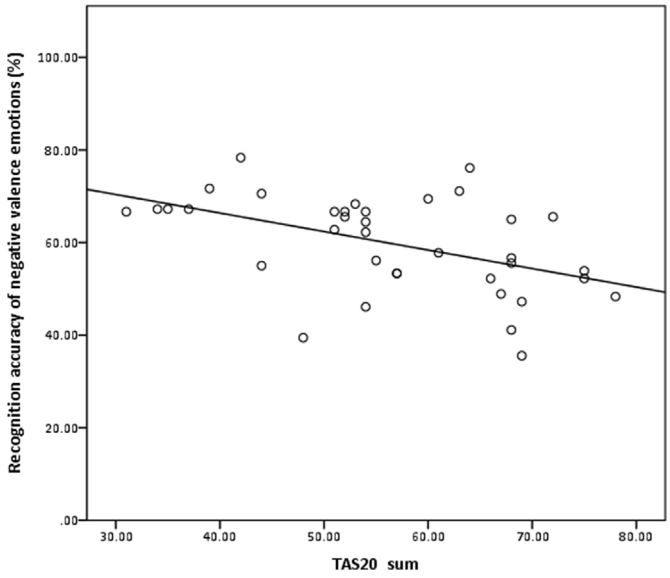

Fig. 2. Alexithymia predicting recognition accuracy of negative valence emotions in PTSD. Higher scores on the TAS-20 were associated with less accurate recognition of facial expressions displaying negative valence emotions.

[3] It was explored whether the results were specific to certain expression intensities and thus regression analyses were conducted for each intensity level of the negative valence emotion category for the PTSD group. Results were similar for each intensity level (low intensity: $F(1, 35) = 7.31$, $p = .011$, standardized $\beta = -0.416$, $t(35) = -2.70$, $p = .011$; intermediate intensity: $F(1, 35) = 8.59$, $p = .006$ standardized $\beta = -0.444$, $t(35) = -2.93$, $p = .006$; high intensity: $F(1, 35) = 7.35$, $p = .010$ standardized $\beta = -0.417$, $t(35) = -2.71$, $p = .010$).

[4] To check for a possible influence of time, since some participants participated split over several days and others did all tasks the same day, we calculated Mann–Whitney U tests for each group to check whether participants' performance on recognition of negative valence emotion differed depending on whether they performed the second study part on the same day or another day than part 1, which turned out not to be the case (PTSD group: $U = 93.50$, $p = .342$, TC: $U = 200.00$, $p = .799$, HC: $U = 80.50$, $p = .644$).

identifying feelings' ($\beta = -0.020$, $t(35) = 0.12$, $p = .902$) and 'difficulty describing feelings' ($\beta = -0.113$, $t(35) = 0.65$, $p = .521$) were excluded from the model.

4. Discussion

The present study assessed automatic and more higher-order aspects of emotion processing in individuals with PTSD compared to healthy traumatized and healthy non-traumatized controls. Whereas the automatic facial responses while observing others' facial emotional expressions did not differ significantly between groups, there were significant group differences in more complex emotion processing styles (alexithymia) and effortful emotion regulation strategies (expressive suppression).

The current study is the first to investigate automatic facial muscle responses when observing facial emotional expressions in individuals with PTSD compared to healthy traumatized and non-traumatized controls. Results showed that corrugator activity was significantly increased in response to observing others' negative valence emotional expressions compared to observing positive valence expressions. Further, zygomaticus activity was significantly increased while and after watching positive valence emotional expressions compared to such of negative valence. These findings replicate previous reports on automatic facial responses to negative and positive valence emotional expressions (e.g. Achaibou et al., 2008; Dimberg, 1982; Dimberg et al., 2002; Likowski et al., 2012; Sato & Yoshikawa, 2007) and suggest facial mimicry to have occurred. Against our hypothesis, the current study did, however, not reveal any group differences in facial mimicry. The findings of the current study thus suggest that automatic facial muscle activations to observed facial emotional expressions are intact in PTSD.

However, it is possible that individuals with PTSD are impaired on higher-order levels of emotion processing, which may provide more opportunity for intervention. When considering such higher-order levels of emotion processing, atypicality is often reported in PTSD (meta-analysis by Plana, Lavoie, Battaglia, & Achim, 2014). In line with previous reports (e.g. Frewen et al., 2008; Söndergaard & Theorell, 2004; Yehuda et al., 1997), alexithymia scores were significantly increased in the PTSD group compared to the other two groups in the current study. That is, individuals with PTSD seem to have more difficulties identifying and describing their own emotions and higher externally-oriented thinking than healthy controls with and without trauma experiences.

The current study was the first study to investigate the relationship between alexithymia and facial emotion recognition in PTSD. Alexithymia has been associated with deficits in emotion recognition in healthy individuals (Frewen et al., 2008) and in individuals with different psychiatric disorders (Grynberg et al., 2012). Moreover, comorbid alexithymia but not the primary disorder seems to account for emotion recognition deficits in autism (Cook et al., 2013; Oakley et al., 2016) and eating disorders (Brewer, Cook et al., 2015). Our results showed that increased alexithymia serves as predictor for less accurate recognition of negative valence emotional facial expressions in PTSD but was not found to be a significant predictor in healthy individuals and such with experienced trauma. In addition, symptom severity of PTSD was not found as predictor for facial emotion recognition. This result suggests that alexithymia has a specific effect on facial emotion recognition in PTSD. Furthermore, it aligns with the 'alexithymia hypothesis' when applied to clinical populations, which states that emotion recognition deficits are due to co-occurring alexithymia but not the primary disorder itself (Bird & Cook, 2013; Brewer, Happé et al., 2015). However, the effect of alexithymia on facial emotion recognition was only present for negative but not positive valence facial emotional expressions. These results align with reports that the recognition of negative valence emotions is more affected in alexithymia in general (Grynberg et al., 2012).

Analyses further showed that the results of alexithymia as significant predictor were driven by the subscale 'externally-oriented thinking'. That is, an external focus of attention during facial emotion recognition might diminish recognition of negative valence emotions in PTSD. This interpretation is in line with reports from an eye-tracking study where 'externally-oriented thinking' was found to be associated with an avoidance of negative valence emotional stimuli in healthy individuals (Wiebe, Kersting, & Suslow, 2017). Our results further align with the wider literature showing that increased scores on the 'externally-oriented thinking' subscale are associated with reduced physiological reactivity when watching sadness-inducing films (Davydov, Luminet, & Zech, 2013). Together, these results suggest that high levels of 'externally-oriented thinking' reduce attention towards negative valence stimuli. Whereas this cognitive style might serve as protection from negative emotional information (and, in PTSD, from being overwhelmed by negative affective states related to trauma reminders), it can lead to difficulties within social interactions, which, in individuals with PTSD, frequently occur (MacDonald, Chamberlain, Long, & Flett, 1999).

One explanation for the effect of externally-oriented thinking to only occur for negative and not positive valence emotions is that positive valence emotions are generally much easier to recognise than negative valence emotions (e.g. Calvo, Gutiérrez-García, Fernández-Martín, & Nummenmaa, 2014; Wingenbach et al., 2016). Even with less attention directed towards the emotional cues presented in the observed faces, recognition of positive valence emotional expressions can be high, because they are so easy to recognise. The positive valence emotions included in the current study entailed smiling, which itself is of high saliency and thus facilitates recognition (Leppänen & Hietanen, 2007). In contrast, the negative valence emotions included in the task had varying identifying facial features (e.g. nose wrinkled in disgust expressions, eyebrows pulled up and drawn together during fear), requiring more attention for recognition. High externally-oriented thinking might thus not have had a diminishing effect on recognition of positive valence emotions. Alternatively, externally-oriented thinking might be applied more in the context of negative emotional stimuli than positive ones, as there is less needing to protect oneself from positive emotional experiences than from negative ones (see, however, Roemer et al., 2001).

In line with previous research (Shepherd & Wild, 2014), expressive suppression was significantly increased in the PTSD group compared to the TC and HC group. Engaging in expressive suppression as an emotion regulation strategy can have negative effects. Research in the typical population has shown that it can lower positive affect while increasing negative affect (Brans, Koval, Verduyn, Lim, & Kuppens, 2013), interfere with memory processes (Richards & Gross, 2006), and exhaust executive cognitive resources (Ben-Naim, Hirschberger, Ein-Dor, & Mikulincer, 2013; Brans et al., 2013; Goldin, McRae, Ramel, & Gross, 2008; Gross, 2015; Johns, Inzlicht, & Schmader, 2008; Richards & Gross, 2006). Expressive suppression is further associated with difficulties in intimate relationships (Richards, Butler, & Gross, 2003) and has been shown to increase cardiovascular arousal and negative affect, even affecting the romantic partner (Ben-Naim et al., 2013). However, despite these potential negative effects, the current study did not reveal any effect of expressive suppression on facial emotion recognition. The results suggest that within a context of low emotional experience triggered by the stimuli (i.e. observing facial emotional expressions), expressive suppression is unrelated to facial emotion recognition.

4.1. Limitations and conclusion

A limitation of the current study relates to the different trauma characteristics of the TC group and the PTSD group. The PTSD group experienced more type II and more deliberately caused trauma than the TC group. However, the risk of PTSD is higher after type II and deliberately caused trauma, accounting for the differences in trauma types between the two groups and making it difficult to match a PTSD group with a TC group on trauma types (Terr, 1991). It should further be

noted that we measured facial mimicry based on facial muscle activations in line with the valence of the observed facial emotional expressions and the two main muscles associated with positive and negative valence. Whereas facial EMG has the advantage to discern even slightest facial muscle activations, it does not provide information on whether the muscle activation was indeed based on facial mimicry or an affective response to the stimuli. Furthermore, participants in the current study merely watched facial emotional expressions which are unlikely to induce strong emotions in observers. It should be considered that expressive suppression as an emotion regulation strategy is usually applied during emotion experience. Future research should thus investigate the effect of expressive suppression on facial emotion recognition with stimuli of more intense emotional content or after experimentally inducing emotional states. In addition, we used a self-report instrument to assess alexithymia, which despite its good psychometric properties, has the disadvantage of answer tendencies and provides a less differentiated picture of alexithymia than observer-rated instruments, for example the Toronto Structured Interview for Alexithymia (Bagby, Taylor, Parker, & Dickens, 2006; Rosenberg et al., 2016). Future research should use self-report as well as observer-rated instrument to assess alexithymia. Finally, interviews were conducted by psychology students who were trained and closely supervised by an experienced clinician (last author). Uncertainties regarding diagnoses were thoroughly discussed. However, the interviews were not repeated by a second examiner; therefore, we cannot provide reliability values.

Summing up, the current study supports previous findings of increased expressive suppression and alexithymia in individuals with PTSD and demonstrates that alexithymia, but not expressive suppression, plays an important role in facial emotion recognition of negative valence emotions in individuals with PTSD. Noteworthily, there is preliminary evidence that reducing alexithymia within psychotherapy may lead to better health outcomes (Beresnevaite, 2000; Tulipani et al., 2010). Since alexithymia explained a large amount of the variance for decreased recognition of negative valence emotions from others' faces in the current study, it is possible that decreasing alexithymia will improve facial emotion recognition. Therefore, the effects of treating alexithymia in psychotherapy on facial emotion recognition abilities should be addressed by future research. This seems important, given that relationship problems are common in individuals with PTSD (MacDonald et al., 1999) and that emotion recognition is a basic social skill that is essential for positive social relationships (Fischer & Manstead, 2008), which, in turn, are associated with well-being and mental health (e.g. Whisman & Baucom, 2012). Furthermore, positive social relationships are a resilience factor after traumatic experiences (DuMont, Widom, & Czaja, 2007) whereas a lack of social support is the strongest predictor for PTSD (meta-analysis by Brewin, Andrews, & Valentine, 2000). To address and prevent relationship problems in individuals with PTSD, it will thus be crucial to further explore factors underlying emotion recognition deficits in future research.

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