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ORIGINAL PAPER

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Keep smiling!

Facial reactions to emotional stimuli and their relationship to emotional contagion in patients with schizophrenia

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Abstract *Introduction* Emotional contagion is a common phenomenon in verbal and nonverbal communication between individuals. Perception and mimicry of facial movements play an important role in this process. Several studies have demonstrated impaired facial expression recognition in patients with schizophrenia and differences in their facial behavior compared to healthy subjects, but so far, the relationship between facial mimicry and emotional contagion has not been studied in this group. *Methods* Seventeen schizophrenic patients and an equal number of matched healthy controls were presented with digital versions of happy, sad and neutral faces from the "Pictures of facial affect" (Ekman and Friesen, Consulting Psychologists Press, Palo Alto, 1976) and were asked to pull their lip corners up or down (like in smiling or showing a sad face) according to the direction of two arrows that were presented simultaneously. In healthy subjects, congruous movements (i.e. pulling the lip corners up when seeing a happy face or pulling them down when seeing a sad face) are facilitated and dissonant movements are inhibited; these tendencies were considered as indicators of emotional contagion. *Results* In schizophrenic patients, these tendencies were significantly diminished. The patients were more apt to display a smile as a reaction to a sad face. We found a positive correlation between these effects and the PANSS—Scores for General Psychopathology. *Discussion* Patients' tendencies towards positive reactions even when a negative stimulus was presented could function as a protective mechanism against overwhelming emotional experiences.

Key words social interaction · nonverbal communication · facial expression · social cognition · emotional expression

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Introduction

The experience and processing of emotions in patients with schizophrenia are characteristically altered. Inappropriate or flattened affects are some of the core psychopathological symptoms of the disease and give rise to difficulties in social and emotional situations. Moreover, schizophrenic patients, as compared to healthy subjects, seem to interpret social clues differently. A multitude of social and emotional clues are conveyed by human facial expressions, and the extraction of such information is impaired in these patients. It remains unclear whether this is a generalized deficit in facial recognition [3, 27], or a more specific deficit—either in the recognition of facial expressions [20, 21, 36] in the visual processing of faces per se, in the correct "labelling" of facial expressions [42] or in a global deficit in attention.

In general, the emotional facial expressions of schizophrenic patients differ from those of healthy controls. Patients' facial displays of emotion are significantly reduced, and their patterns of nonverbal communication are different from those of healthy subjects [40]. Mattes et al. [32] found reduced zygomaticus activity measured by EMG in an experiment eliciting positive emotions, and suggested a reduced ability of schizophrenic patients to express happy facial expressions, such as smiling. Moreover, these reduced facial actions themselves have special features that have been described as "disintegration of

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facial actions" by Heimann et al. [24]. Thus, the observer gets the impression of a bizarre and asymmetrical behavior.

Because of these factors, the interaction between schizophrenic patients and non-patients is often characterized by uncertainty about the other person's present emotional state. Even therapists sometimes find it difficult to understand the patient's emotional experience, as patients do not seem to be able to convey what they feel.

The ability to communicate a certain mood on the one hand, and to be empathic with other people on the other hand—in other words the ability to "get in tune" with those around us, is called "emotional contagion". According to Hatfield et al. [23], the process begins with the perception of other people's movements, facial expression, postures or vocalisations—consciously and, as Morris et al. [34] and Whalen et al. [48] proved, unconsciously. The perception of these signals is followed by their imitation ("mimicry"), leading to a synchronization of movements between the object and the observer. This mimicry does not have to appear visibly and can sometimes only be detected by EMG activity [10, 15]. The afferent feedback from such mimicry is known to influence one's subjective emotional experience and to induce the perceived emotion in the observer [17]. The observer gets "infected" by the emotion he or she perceives.

Emotional contagion:

Perception → Mimicry → Feedback → Emotion

FMRI investigations have revealed that the perception of emotional facial expressions leads to an activation in the same regions of the brain that are activated when that particular emotion is experienced [1, 50]. Moreover, it is very likely that the ability to "infect" others with an emotion or to "catch" the emotions of others depends on the functioning of the mirror neuron system [13].

In sum, these findings raise the question as to whether patients' difficulties in emotional interaction could be explained by a deficit in patient's abilities to induce emotional contagion or to experience emotional contagion themselves. It could be argued that their difficulties in processing other people's facial expressions and the special features of their own facial actions prevent the cascade of emotional contagion.

The present study focuses on the initial part of this cascade. It aims at investigating the special features of the transition from the *perception* of an emotional stimulus to its *imitation* in patients with schizophrenia compared to healthy subjects. In an fMRI investigation of this process in healthy subjects by Wild et al. [50], medial basotemporal activation was found to be correlated with the execution of fast and congruent facial expressions in reaction to happy faces. This finding suggests that this region modulates facial movements. Clinical relevance comes from the finding that in schizophrenic subjects, the temporal lobe is the brain region in which abnormalities are documented most consistently [33, 37]. Numerous structural and functional abnormalities in the amygdala, the hippocampus and the parahippocampal region have been identified [4]. In patients with schizophrenia there is often a lack of spontaneous facial reactions even when medication-free [42] and even when they experience adequate emotional reactions [30]. Connections among these findings seem likely so an examination of the modulation of facial expressions by perception in patients with schizophrenia seemed appropriate.

In order to provide a standardized representation of the process of emotional contagion we chose pictures of emotionally expressive faces that have been shown to evoke facial and emotional reactions in the observer [49] and instructed subjects (schizophrenic or healthy) to either imitate the emotion displayed on the picture by moving the corners of the mouth in the same way, or, in the second condition, to display facial movement dissonant to the one presented on the picture. This allowed us to study the subjects' reaction times for the evoked facial movement as well as the interaction between the subjects' facial expressions and the facial expressions the subjects saw. We then used this design to measure the beginning of the emotional contagion process. Additionally, we performed a number of emotion recognition tasks and assessed the subjects' susceptibility to emotional contagion. Our hypothesis was that, according to the results of previous studies, the patients would perform more poorly than healthy controls in the emotion recognition tasks. Moreover, we expected schizophrenic patients to be less susceptible to emotional contagion than the healthy subjects. We expected the congruous conditions to facilitate the lip movement and the dissonant condition to inhibit the lip movement—and we expected that these effects would be weaker in the patient group than in the control group. We also expected the patients to display inappropriate facial reactions, e.g. bizarre expressions or reactions different to the ones requested in the instruction.

Methods

Subjects

The study was approved by the ethics committee of the medical faculty of Tübingen and is in accordance with the Declaration of Helsinki. After obtaining written informed consent of the subjects, 17 hospitalized schizophrenic patients (6 females, 11 males, mean age 28.2 years, SD 7.4 years) diagnosed by trained psychiatrists according to the ICD-10 criteria [14] with F20.0, and 17 non-hospitalized healthy controls (6 females, 11 males, mean age 27.6 years, SD 5.4 years) were included in this study. The age difference between patients and controls was not statistically significant. All subjects were right handed, handedness determination being carried out by the Edinburgh Handedness Inventory [38]. All patients

Table 1 Patients' mean duration of illness and age of onset

	Mean duration of illness (years)	Mean age of onset (years)	Mean dose of neuroleptic medication (chlorpromazine equivalents, mg/day)
Female patients (n = 6)	0.5 (SD 0.84)	26.5 SD 9.01	492.9 (SD 218.76)
Male patients (n = 11)	0.5 (SD 0.84)	27.09. SD 6.73	664.5 (SD 527.66)

were hospitalized at the University Hospital of Tübingen, Germany, Department of Psychiatry and Psychotherapy. Nine patients had had no previous stays in hospital, four patients had had one previous stay, and five patients had had three stays or more. Control subjects included University students, members of the hospital staff and other local people recruited by pamphlets or addressed personally. Using a semi-structured interview with DSM IV criteria, subjects with relevant previous or present psychiatric or neurological disorders (e.g. subjects reporting preoccupation with delusions or frequent auditory hallucinations) or with first-degree relatives with depressive or psychotic disorders, were excluded. All subjects were native German speakers and had normal or corrected to normal vision.

The groups were matched with respect to sex, age and handedness but differed significantly in their educational levels. The mean year of education was 11.79 (SD 1.4) for the patients and 12.76 (SD 0.8) for the controls. In a pre-test, however, we were able to show that emotion recognition and facial reactions were not influenced by educational levels [29].

At the time of testing, all patients were on medication consisting of both typical and atypical neuroleptics and sometimes a combination of both, so it was not possible to control for drug effects. Exclusion criteria of the patients were severe organic diseases, substance abuse and a diagnosis of schizoaffective disorder. Tables 1 and 2 show the patients' most relevant clinical characteristics.

One patient received treatment with 30 mg Flupentixole every 2 weeks so that the mean daily dose cannot be provided in his case, and one female patient also took part in a double-blind medication study, either receiving Risperidone or Haloperidole in an unknown dosage.

Test materials and procedure

Before testing, the procedure was fully explained to all subjects. The "Positive and Negative Syndrome Scale" (PANSS [25]) and the Abnormal Involuntary Movement Scale (AIMS [22]) were applied to the schizophrenic patients by trained raters. The AIMS was used to determine the degree of extrapyramidal symptoms should they arise, but none of the patients had any of these symptoms. All participants were seated in front of a computer screen. The stimuli utilized were taken from the *Pictures of facial affect* by Ekman and Friesen [18] in a digitized and morphed version, as described by Benson and Perret [5], Perret et al. [39] and Calder et al. [11], and the procedure as described below had also been employed in a previous study by Wild et al. [50]. The stimuli included six different black-and-white pictures of faces (3 males and 3 females) with happy, sad or neutral expressions, i.e. 18 different pictures. The pictures had been digitally altered so that only the faces, and no other body parts such as hair, were visible. Each face was combined with arrows pointing up- or downward on the left and the right-hand side of the picture, leading to a total number of 36 stimuli. The arrows indicated whether the subject should pull her/his lips up or down. Additionally, two arrows pointing up- or downward on a grey background were presented nine times each (Fig. 1). Every stimulus was presented on the computer screen in randomized order for 3 s. This led to a total of 54 stimuli. A digital video camera (Sony DHR-1000VC, 25 frames/s) recorded the subjects' facial expressions and the respective stimulus simultaneously. The subjects were asked to look at the stimulus holistically (i.e. not to concentrate on either the arrows or the faces only) and then to pull their lip-corners up- or downward, according to the direction of the arrows as quickly as possible. This instruction was given in order to prevent the subjects from simply concentrating on the arrows and not paying attention to the faces, which might influence the initiation of their lip movements.The stimuli were labelled as follows (Table 3).

Table 2 Overview of the patients' medication

Substance	Number of patients	Mean daily dose (mg)	Mean chlorpromazine equivalents (mg/day)
Typical neuroleptics			
Perphenazine	1	24	360
Haloperidole	6	15.5	775
Atypical neuroleptics			
Risperidone	3	4.17	166.67
Olanzapine	8	13.13	656.25
Others			
Biperidene	8	2.63	
Promethazine	1	37.5	
Lorazepame	7	1.7	
Chlorprothixene	1	37.5	
Zopiclone	2	2.25	

When comparing the average speed of the initiated movement in patients and controls, we found that both groups initiated their movements significantly faster to NEUup-stimuli than to arrows pointing up alone and significantly slower to NEUdown-stimuli than to arrows pointing down alone. Both groups had difficulties pulling their lip corners down especially when being presented with a face simultaneously, even if this face was supposed to be a neutral one. Apparently, the neutral faces also contained some emotional information and were not neutral to our subjects.

In addition to the PC-task, the subjects were given several questionnaires: the "Eysenck Personality Inventory" (EPI [19]), "the Emotional Contagion Scale" (assessment of susceptibility to emotional contagion [16]), and took the D2-test of attention [8].

Furthermore, the emotion–recognition test (EMO-Test, part I/II), developed by our research group [9], was carried out, in order to estimate the subjects' ability to correctly recognize and verbally label facial expressions. This test is based on Ekman's *Pictures of facial affect* and consists of three parts and two versions. We used the first two parts of version 2. The first part (EMO I "Matching") consists of 18 pictures with 10 female and 8 male faces. Three pictures display the emotions "joy", "sadness", "anger", "fear", "disgust" or "surprise", respectively. The subjects were sequentially given these pictures in a randomized order and were asked to compare them with a model on which the six emotions are displayed by only one male face, and then were asked to choose the picture on the model with the same facial expression. In the second part (EMO II "Labelling") three additional pictures with a neutral expression were given; this led to a total of 21 pictures, and the subjects were asked to assign verbal labels from a list to the facial expression on the photograph.

Data analysis

The time until the subjects made the correct movement according to the direction of the arrows was measured by counting the frames on the video tape from the instant when the stimulus occurred to the time at which the subjects' reaction began, i.e. when the first movement of the lip corners was visible [50]. The rater was trained in a pre-test with videos of non-patients but was not blind to the

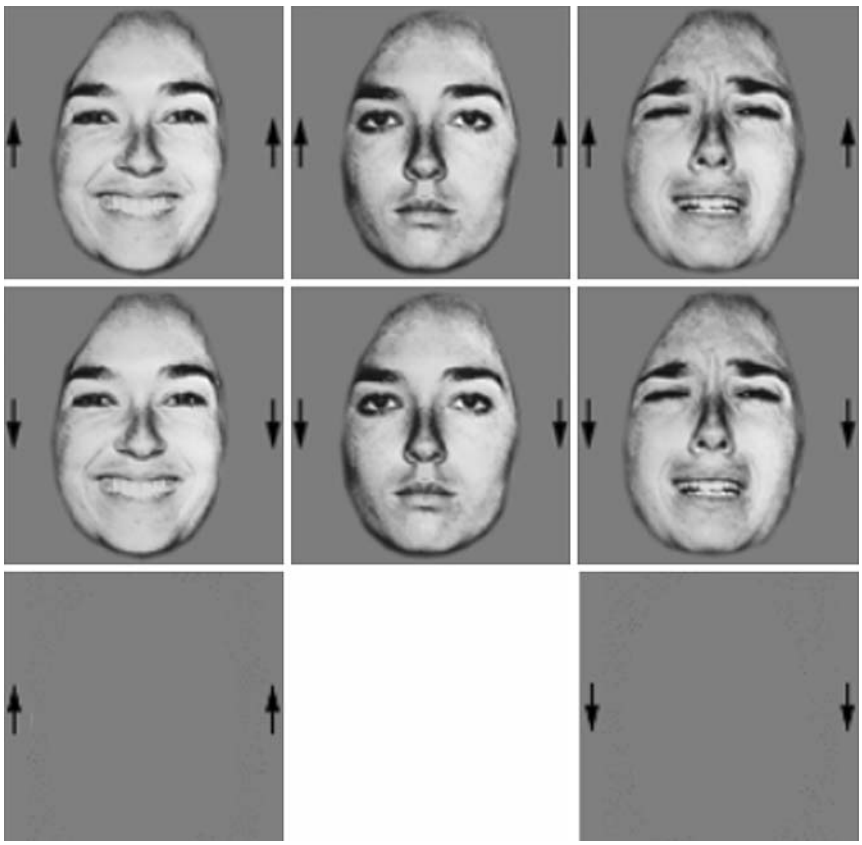

Fig. 1 Examples of different stimuli, happy expressions on the *left*, neutral expressions in the *middle* and sad expressions on the *right*, baseline stimuli in the *lower row*, each combined with the two different signs for moving the lip corners up (*upper row*) or down (*lower row*)

diagnostic status of the person in the video. The trial was not rated if no correct movement occurred. In trials where the wrong movement occurred first and was then corrected, time was measured until the correct movement began.

Statistical evaluation was performed using the SPSS® software package, Version 11.0. The Kolmogorow–Smirnow test was used to test for normality. All variables, except for "Educational level", were normally distributed. Correlations were tested using Pearson's correlation coefficient, or, if normal distribution was not present, Spearman's coefficient. Statistical significance of group differences was tested with a Univariate Analysis of Variance. Due to the rather preliminary character of the study undertaken to determine and document the project's viability the results were not corrected for multiple comparisons. $p \leq 0.05$ was considered statistically significant. Significances are for two-sided hypotheses if not otherwise stated.

Results

Questionnaires

EPI: The patients' average extraversion-score was 11.5 (SD 3.3), in the controls the score was 13.9 (SD 2.6; $t = -2.303$, $df = 28.\ 8$, $p = 0.029$).

D2-test of attention: The average number of symbols processed (patients: 409.4, SD 92.6, controls: 527.2, SD 92.5; $t = -3.71$, $df = 32$, $p = 0.001$) and the concentration-score (patients: 169.1, SD 38.4, controls: 207.2, SD 57.0; $t = -2.29$, $df = 28$, $p = 0.029$) differed significantly between patients and controls.

ECS-D: Susceptibility to emotional contagion in general was calculated by adding the susceptibility-scores for "joy", "love", "fear", "anger" and "sadness". No significant difference could be found between the two groups in their susceptibility to emotional contagion in general and to "joy" and "sadness". The differences between the two groups in their susceptibility to "love" (lower susceptibility in patients: patients' mean 10.9, SD 2.8, controls' mean 12.5, SD 1.5; $t = -2.10$, $df = 24.4$, $p = 0.04$) and to "anger" (stronger susceptibility in patients: patients' mean 9.2, SD 2.3, controls' mean 7.5, SD 1.7; $t = 2.48$, $df = 29.2$, $p = 0.02$) proved to be significant, and we also found a tendency in our patients to be more susceptible to "fear" (patients' mean 9.8, SD 2.2, controls' mean: 8.5, SD 2.5; $t = 1.69$, $df = 31.3$, $p = 0.1$).

PANSS: The PANSS could only be carried out in 16 out of 17 patients. Our patients' average score on the

Table 3 Labels of the different stimuli used in the experiment

HAPup	↑Happy face↑
HAPdown	↓Happy face↓
SADup	↑Sad face↑
SADdown	↓Sad face↓
NEUup	↑Neutral face↑
NEUdown	↓Neutral face↓
ARRup	↑Grey background↑
ARRdown	↓Grey background↓

scale for general psychopathology was 30.4 (SD 10.7), 14.3 (SD 5.2) for positive symptoms and 15.8 (SD 6.8) for negative symptoms. This represents a partially remitted sample.

EMO-test: In the first part of the facial recognition test ("Matching") the patients assigned a mean number of 13.8 (SD 1.4) out of 18 pictures correctly, in the controls the mean number was 14.7 (SD 1.5; $t = -1.86$, $df = 30.7$, $p = 0.072$). There was a tendency towards significantly poorer performance in assigning pictures with angry faces correctly between the patient group and the control group (patients' mean 1.7, SD 0.5, controls' mean 2.1, SD 0.7 out of three given pictures, $t = -1.99$, $df = 27$, $p = 0.056$). In the second part ("Labelling"), an average of 15.5 (SD 3.2) out of 21 emotional faces was labelled correctly by the patients (controls 17.0, SD 2.0, NS). Similar to the first part of the test, there was a significant difference regarding faces with angry expressions. The patients labelled 2.1 (SD 0.9) out of three pictures correctly, whereas the controls labelled 2.9 pictures correctly (SD 0.3; $t = -3.37$, $df = 21$, $p = 0.003$). Apparently, the patients had particular difficulties in the processing of angry faces.

Table 4 Initiation time for all presentation conditions in patients

Patient or control?	*N*	Mean	SD	*t*	Significance (two-tailed)
HAPup (ms)					
Patient	17	492.5	98.4	2.3 ($df = 31$)	0.03*
Control	17	421.0	84.8		
HAPdown (ms)					
Patient	17	732.7	209.9	2.6 ($df = 29$)	0.01*
Control	17	571.4	147.6		
SADdown (ms)					
Patient	17	628.7	187.0	2.7 ($df = 29$)	0.01*
Control	17	479.2	132.4		
SADup					
Patient	17	575.2	160.2	0.2 ($df = 29$)	0.84
Control	17	562.4	212.9		
NEUup (ms)					
Patient	17	536.9	131.1	1.4 ($df = 32$)	0.17
Control	17	472.9	132.9		
NEUdown (ms)					
Patient	17	613.8	192.4	1.9 ($df = 29$)	0.06*
Control	17	502.3	136.4		
ARRup					
Patient	17	572.4	139.4	1.7 ($df = 32$)	0.09*
Control	17	492.9	129.2		
ARRdown					
Patient	17	604.8	123.4	3.2 ($df = 32$)	0.00*
Control	17	473.6	115.8		
Total average initiation time (all conditions)					
Patient	17	594.6	121.7	2.4 ($df = 32$)	0.02*
Control	17	497.0	111.52		

PC-task

As expected, the patients initiated lip movements slower than the controls. The average initiation time per stimulus was 594.6 ms in patients (SD 121.7) and 497.0 in controls (SD 111.5; $t = 2.44$, $df = 31.8$, $p = 0.02$). Table 4 displays the performance data (i.e. initiation time in ms) for all presentation conditions.

In order to avoid a bias on the initiation of lip movements that were of interest to us, i.e. the ones triggered by happy or sad faces, due to the fact that subjects potentially attributed an emotional content to the neutral faces, we calculated a measure for the mimicry induced by emotional faces by determining each individual subject's average initiation times for the respective stimuli and subtracting them from their total average initiation time [50]. The result was used to quantify the effects of an accelerated or, if it had a negative sign, the effects of a decelerated emotional contagion (e.g. "acceleration" for happy stimuli = average reaction time of all movements minus average reaction time of upward movements during happy face perception). The differences in the speed of the initiation are strong indication that the effects are not due to the fact that the subjects only concentrated on the direction of the arrows, but carried out the given instructions correctly.

In both patients and controls an accelerating effect for the "HAPup"-condition, i.e. a happy face combined with an arrow pointing upwards, occurred (Table 5). A significant difference was found in the dissonant condition "SADup" though, with the patients displaying an accelerated reaction and controls displaying a decelerated reaction ($t = 2.17$, $df = 30.1$, $p = 0.037$).

Correlations

"Acceleration" and "deceleration" were not found to be correlated with any of the questionnaires' results. In particular, neither one was influenced by concentration, measured in the D2-test. Consequently, patients' weaker "acceleration" effects are not due to a lack of concentration. Contrary to our expectations, "acceleration" was influenced by neither personality traits (EPI) nor the ECS-D scores either. Furthermore, a correlation between medication and "acceleration" or "deceleration" could not be detected, indicating that the differences between patients and controls in the initiation of lip movements cannot be ascribed to medication effects.

The only significant correlation was found between "acceleration" and the patients' PANSS scores. Patients who scored high on general psychopathology had stronger "acceleration" reactions induced by the "SADup" stimulus (Fig. 2; rs $= 0.53$ $p = 0.015$), i.e. the more symptoms the patients had, the faster they pulled their lip corners up after a sad stimulus. Patients with higher psychopathology scores presented lower "acceleration" effects induced by the "SADdown" stimulus (Fig. 3; $r = -0.58$, $p = 0.018$), i.e. the

Table 5 "Acceleration"/"deceleration" in patients and controls

HAPup	HAPdown	SADup	SADdown
Patients $n = 17$ Acceleration: 104.7; SD 75.9	Deceleration: −135.4; SD = 132.4	Acceleration: 22.0; SD = 104.1	Deceleration: −31.5; SD = 101.1
Controls $n = 17$ Acceleration: 75.9; SD = 78.4	Deceleration: −74.4; SD = 90.1	Deceleration: −65.4; SD = 129.3	Acceleration: 17.8; SD 101.1
Significant difference? $t = 1.1$, $df = 32$ $P = 0.285$ NS	$t = -1.6$, $df = 28$ $P = 0.126$ NS	$t = 2.2$, $df = 31$ $P = 0.037$*	$F = -1.4$, $df = 32$ $P = 0.165$ NS

"Accelerating" (positive sign) and "decelerating" (negative sign) effects calculated as the difference between the subjects' total average initiation time minus the average initiation time for the respective stimulus
* $P \leq 0.05$

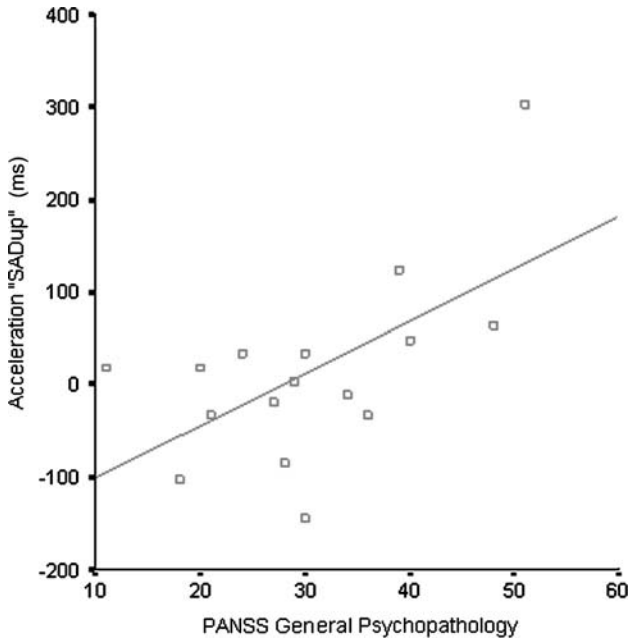

Fig. 2 The more symptoms the patients had, the faster they pulled their lip corners up after a sad stimulus ($n = 16$)

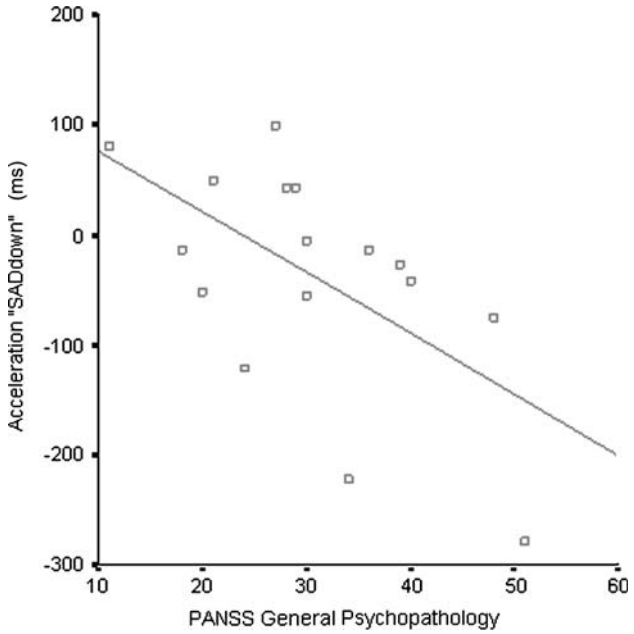

Fig. 3 The more symptoms the patients had, the slower they pulled their lip corners down after a sad stimulus ($n = 16$)

more symptoms the patients had, the slower they pulled their lip corners down after a sad stimulus.

Lower "acceleration" scores for the "SADdown" stimulus were also found in combination with higher scores for negative symptoms (Fig. 4; $r = -0.54$, $p = 0.03$) and higher scores for positive symptoms (Fig. 5; $r = -0.55$, $p = 0.029$), i.e. the more negative symptoms the patients had, the slower they pulled their lip corners down after a sad stimulus and the more positive symptoms they had, the slower they pulled their lip corners down after a sad stimulus.

Discussion

Three issues merit discussion: (1) patients' abilities to recognize and label facial expressions correctly, measured by the EMO-tests; (2) their similarity in subjective susceptibility to emotional contagion in general; and (3) the differences in the initiation of the emotional contagion cascade between schizophrenic patients and healthy controls.

Concerning the first issue, it was unclear as to whether the patients were able to recognize emotions correctly, but had difficulties in labelling them correctly. To distinguish between these two possible deficits, we applied the EMO-test in which patients and controls had to assign either similar facial expressions on photographs or labels to emotionally expressive faces. In our sample, there was no overall deficit in assigning or labelling emotions correctly. Only in labelling and later in assigning pictures with angry expressions correctly, the patients performed significantly poorer. This result is in accordance with previous studies of emotion recognition deficits in schizophrenia, which showed greater difficulties in patients' recognition of negative emotions [41]. As opposed to the results of Kohler et al. [28] this deficit in recognizing and labelling "anger" correctly did not

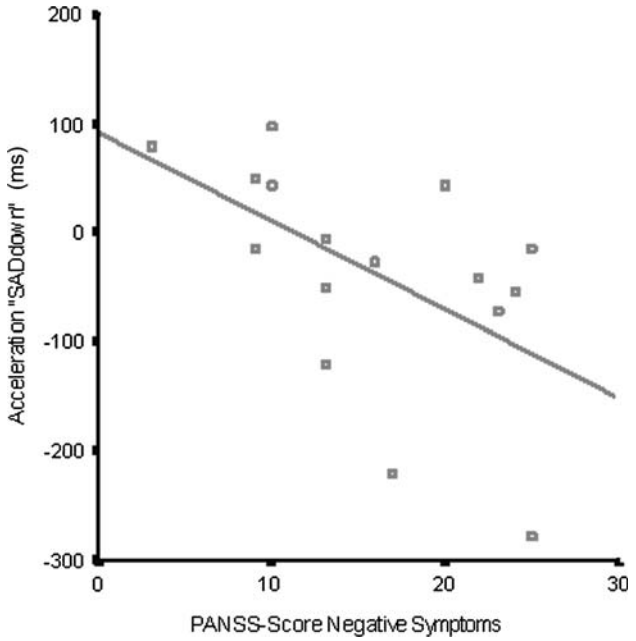

Fig. 4 The more negative symptoms the patients had, the slower they pulled their lip corners down after a sad stimulus (n = 16)

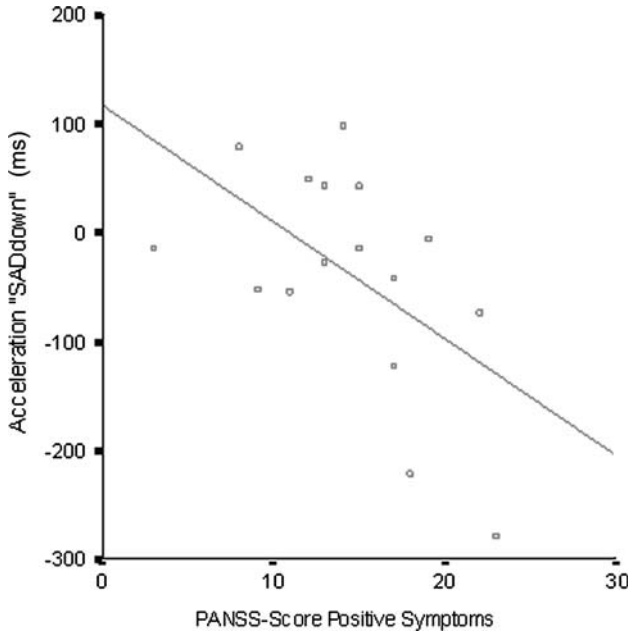

Fig. 5 The more positive symptoms the patients had, the slower they pulled their lip corners down after a sad stimulus (n = 16)

correlate with attention as measured by the performance in the D2-test of attention.

The next important result was that patients and controls did not differ significantly in their subjective ratings of susceptibility to emotional contagion in general. When comparing their susceptibility to different emotions we found that patients regarded themselves significantly less susceptible to "love" as an emotion and significantly more susceptible to "fear" and "anger". In the EPI, the patients scored significantly lower for "extraversion". Therefore, and in accordance with the results of Kring et al. [30], it could be argued that, despite their seemingly flattened affect, patients' subjective experiences of emotions in general are not altered—at least not in their subjective ratings—but, as they tend to be less extraverted, they might not show their emotions as overtly as healthy subjects. The fact that the patients were seen to be more susceptible to contagion with anger, but that in the EMO-test both their recognition and labelling of anger were disturbed, makes it easier to understand their irritation and their sometimes inappropriate reactions in emotional situations.

When comparing the initial part of the contagion cascade in patients and controls, the most striking result was a tendency in patients' responses towards "positive" reactions, i.e. to show a smile even if the stimulus was a sad face. Previous studies investigating emotional facial recognition [7, 12, 35, 45] showed that schizophrenic patients have difficulties in recognizing negative emotions or tend to rate negative emotions as positive. Schneider et al. [43] considered this an evidence for patients' lack of empathy. Studies investigating the production of emotional facial expressions [6, 31, 47] stated a dissociation between spontaneous and posed facial reactions: the patients displayed negative affect more frequently, but only under spontaneous conditions for example in interviews. In a more recent study by Tremeau et al. [44], however, the schizophrenic patients exhibited deficits in both posed and spontaneous emotional expressiveness, which is in accordance with our results. The use of congruent and dissonant conditions allowed us to examine facial reactions with both spontaneous (congruent) and posed (dissonant) components. Our patient group was impaired under both the congruent and the dissonant condition, but only if a sad facial expression was involved. Therefore, this effect cannot be accounted for by differences of spontaneous versus posed expressions, but rather represents the automatic and unconscious part of emotional contagion, probably in combination with the patients' difficulties in recognizing negative emotions correctly. The patients' overall tendencies to display positive facial expressions as reactions to the negative expressions perceived could therefore be interpreted as a strategy to counteract potential uncertainty about the actual content of the expression.

This assumption is supported by the positive correlation we found between the PANSS-scores for *General Psychopathology* and the "acceleration" reaction in the "SADup" condition, and the negative correlation between *General Psychopathology* and the "deceleration" reaction in the "SADdown" condition. Obviously, the severity of the symptoms influenced the reaction to the emotional stimuli in the sense that—although they were given clear instructions—the more symptoms the patients had, the more often they displayed a positive reaction, and the less

often they displayed a negative reaction to a sad stimulus. Additionally, the missing correlations between medication and "acceleration" and "deceleration" as well as the fact that none of the patients had any extra-pyramidal symptoms indicate that our results are not due to any drug-induced alternations in the patients' motor system.

The incongruous stimuli used in our experiment resemble those used in the Stroop task. Schizophrenic patients usually perform poorer than controls in this task [26], so this might also be considered an explanation for our results. But unlike in the Stroop task, our patients did not perform poorer in all incongruous conditions; they even displayed an "acceleration" reaction in the incongruent condition "SADup".

As the patients only had difficulties in recognizing angry facial expressions but not in recognizing other facially displayed emotions, their altered mimic reactions in this trial cannot only be attributed to a disturbed perception of faces.

The patients' tendencies to prefer positive reactions (smiling) over negative ones as a response to communication clues in the widest sense could be ascribed to the fact that positive emotions are experienced more often and can facilitate communication. Moreover, studies on "expressed emotions" found evidence that a high level of expressed (negative) emotions in patients' families can influence the course of the disease. In his social-cognitive theory, Walker [46] argues that patients tend to act and react positively in communication, in order to avoid social interaction and to protect themselves against irritating or confusing stimuli, so this might be the strategy the patients also applied in our experiment.

Besides, the process of emotional contagion is related to the functioning of certain brain structures. We used the same paradigm in an fMRI-investigation and found that a facilitating effect of congruent stimuli correlated with an activation of both medial basotemporal lobes (in particular the parahippocampal gyri) in healthy subjects [50]. This is a brain region consistently identified in many studies to have structural anomalies in schizophrenic patients [2].

Obviously, the difficulties that schizophrenic patients have in dealing with emotions have a wide range of sources. It is possible that patients' reduced ability to recognize and to rate emotions correctly leads to irritation, which would already be enough to make it difficult for them to react appropriately. The additional deficit in their own facial behavior impedes mutual communication even more, as it causes irritation in the communication partner as well thus augmenting the unfortunate situation. The special features we found in the patients' initiations of the emotional contagion cascade might explain why "getting in tune" with them is often so difficult.

It should, however, not be ignored that the patients still have emotional abilities that might be open to external influences or even improvement, e.g. by means of social training. Moreover, therapists or other persons in contact with the patients should always be aware of the various difficulties patients have and should not assume a reduced emotionality that might in fact only be a reduced expressiveness.

Limitations of the study: The number of patients tested is comparatively small and the data would probably not have survived an alpha-adjustment for multiple comparisons. The results should therefore be regarded as preliminary findings that could form the basis for further research. In order to obtain a more complete notion of the particularities of the emotional contagion process in schizophrenic patients, the actual emotional reactions to the emotional faces should also be taken into account in further investigations.

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