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Empathic deficits in schizophrenia: The potential role of rapid facial mimicry

KANDICE J. VARCIN, PHOEBE E. BAILEY, AND JULIE D. HENRY
School of Psychology, University of New South Wales, Sydney, New South Wales, Australia.

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Abstract

Emotional facial expressions evoke rapid, involuntary, and covert facial reactions in the perceiver that are consistent with the emotional valence of the observed expression. These responses are believed to be an important low-level mechanism contributing to the experience of empathy, which some have argued rely on a simulation mechanism subserved by the human mirror neuron system (MNS). Because schizophrenia is associated with pervasive social cognitive difficulties which have been linked to structural abnormalities in the MNS network, the aim of the present study was to provide the first assessment of how rapid facial mimicry reactions (within 1000 ms poststimulus onset) are affected in schizophrenia. Activity in the *corrugator supercilii* and *zygomaticus major* muscle regions was quantified using electromyography while individuals with schizophrenia (n = 25) and controls (n = 25) viewed images of happy and angry facial expressions. In contrast to controls, individuals with schizophrenia demonstrated atypical facial mimicry reactions which were not associated with any clinical features of the disorder. These data provide evidence for a low-level disruption that may be contributing to empathizing deficits in schizophrenia and are discussed in relation to neuropsychological models of empathy and schizophrenia. (*JINS*, 2010, *16,* 621–629.)

Keywords: Empathy, Emotions, Facial muscles, Facial expression, Electromyography, Psychopathology

INTRODUCTION

Social functioning deficits are more pronounced in schizophrenia than in any other psychiatric illness (Mueser & Bellack, 1998), with social cognitive impairment consistently identified as a key predictor of social function outcomes (Brekke, Hoe, Long, & Green, 2007; Brüne, 2005; Pijnenborg, Withaar, Evans, van den Bosch, Timmerman, & Brouwer, 2009). Much of this social cognitive research has focused on higher-level aspects of empathic behavior, and in particular emotion recognition (decoding of affective cues) and cognitive empathy (the understanding of another's beliefs and desires), with impairments consistently identified in both of these components (Brüne, 2005; Edwards, Jackson, & Pattison, 2002; Langdon, Coltheart, & Ward, 2006). However, empathy is a multifaceted construct that is considered to involve not only higher-level facets, but also lower-level facets such as the emotional experience of a perceived affective state, often referred to as affective empathy (Baron-Cohen, 2005; Decety & Meyer, 2008; Leiberg & Anders, 2006; Preston & de Waal, 2002; Singer & Lamm, 2009). Preliminary evidence indicates that, as with the other facets of empathy, affective empathy is disrupted in schizophrenia (Derntl et al., 2009; Shamay-Tsoory, Shur, Harari, & Levkovitz, 2007).

Although empathic deficits are well documented in schizophrenia, the precise mechanisms contributing to these difficulties have not yet been identified. Specifically, there is a relative dearth of research examining early or low-level processes that are involved in the experience of empathy in this population. As such, it is unclear as to whether a disruption in the low-level processes that are associated with empathic responding, may be underlying any empathic difficulties in this group. One such low-level mechanism that has been identified as an important contributor to an empathic response is rapid facial mimicry (for a review, see Singer & Lamm, 2009). This response refers to the tendency for individuals to involuntarily and rapidly synchronize their facial expressions with observed expressions (Dimberg, 1982; Dimberg & Lundqvist, 1988, 1990; Dimberg, Thunberg, & Elmehed, 2000). This form of motor mimetic responding has been considered as contributing to the experience of empathy (e.g., see Singer &

Correspondence and reprint requests to: Kandice J. Varcin, School of Psychology, University of New South Wales, Sydney, New South Wales 2052 Australia. E-mail: kvarcin@psy.unsw.edu.au

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Lamm, 2009) as well as a form of empathy in its own right (Blair, 2005).

Involuntary facial mimicry has been implicated in, and thought to facilitate, empathy *via* a process of simulation (Decety & Jackson, 2004; Oberman & Ramachandran, 2007). According to the simulation account, we come to empathize with others by involuntarily and subconsciously mimicking (i.e., "mirroring") the observed emotional expressions of others; this mimicry, or embodied representation, facilitates emotion understanding and serves to induce an appropriate empathic response (Adolphs, 2002; Decety & Jackson, 2004; Preston & deWaal, 2002). Rapid facial mimicry in response to the observation of others' emotional facial expressions has been interpreted as evidence for this simulation account of empathy (Oberman & Ramachandran, 2007). Consistent with this notion, functional magnetic resonance imaging (fMRI) indicates that in nonclinical volunteers the observation and imitation of emotional facial expressions activates a similar neural network (Carr, Iacobone, Dubearu, Mazziotta, & Lenzi, 2003).

The simulationist view of empathy is linked to perception-action models of empathic responding putatively argued to be supported by the human mirror neuron system (MNS; Preston & deWaal, 2002). Originally identified in macaque monkeys, mirror neurons refer to cortical brain cells that are named due to their tendency to activate both when an individual performs an action *and* when an individual observes the same action performed by another (Buccino & Amore, 2008; di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992). Although ongoing controversy remains surrounding the precise specificity and brain regions involved, in humans, neuroimaging studies have provided evidence for a network that appears to subserve such perception-action coupling—the human MNS (Decety, Chaminade, Grezes, & Meltzoff, 2002; Iacoboni, Woods, Brass, Bekkering, Mazziotta, & Rizzolatti, 1999; Iacoboni et al., 2001). Some key components of this network are considered to be the superior temporal sulcus, inferior parietal lobe, and inferior frontal gyrus (Iacoboni & Dapretto, 2006; Rizzolatti & Craighero, 2004).

Importantly, social cognitive difficulties in first episode psychosis have been shown to be related to structural abnormalities in the MNS network, and it has been argued that these, ". . . may account for the social cognitive deficits present in some psychiatric disorders, such as schizophrenia." (p. 79, Bertrand, Achim, Harvey, Sutton, Malla, & Lepage, 2008). Moreover, in a recent transcranial magnetic stimulation experiment, Enticott, Hoy, Herring, Johnston, Daskalakis, and Fitzgerald (2008) provided preliminary evidence that individuals with schizophrenia demonstrated reduced mirror neuron activation in premotor cortices of the brain compared with nonclinical individuals. Taken together, there are compelling reasons for assessing whether rapid facial mimicry responses are disrupted in this population.

Rapid facial mimicry responses are generally detected using facial electromyography (EMG; measurement of changes in the electrical activity of muscles), a technique which is sensitive to even subtle changes in facial muscle activity. Numerous EMG studies have shown that distinct facial reactions are reliably elicited by the mere perception of emotional facial expressions (Dimberg, 1982; Dimberg & Thunberg, 1998; Dimberg et al., 2000). That is, images of angry facial expressions evoke increased *corrugator supercilii* (i.e., brow) activity relative to happy facial expressions, and images of happy facial expressions evoke increased *zygomaticus major* (i.e., cheek) activity relative to angry (Dimberg, 1982; Dimberg & Petterson, 2000; Dimberg & Thunberg, 1998; Dimberg et al., 2000; Dimberg, Thunberg, & Grunedal, 2002). Facial mimicry reactions in nonclinical individuals reliably occur within one second of stimulus presentation (Bailey, Henry, & Nangle, 2009; Dimberg et al., 2000; Moody, McIntosh, Mann, & Weisser, 2007). Furthermore, attesting to the robust nature of this response, facial mimicry has been shown to occur within one second of stimulus exposure following explicit instructions *not* to react (Dimberg et al., 2002) and when images are presented subliminally (Bailey & Henry, 2009; Dimberg et al., 2000).

The primary aim of the present study was to provide the first assessment of whether low-level rapid facial mimicry reactions are, as with other empathic behaviors, disrupted in schizophrenia. The few EMG studies that have been conducted in this group have predominantly examined facial reactions in response to nonfacial stimuli (Mattes, Schneider, Heimann, & Birbaumer, 1995; Wolf et al., 2004; Wolf, Mass, Kiefer, Wiedemann, & Naber, 2006). Furthermore, the one EMG study that has presented images of facial stimuli examined facial responding over an extended timeframe (collapsed across seven seconds poststimulus exposure; Kring, Kerr, Earnst, 1999). The results indicated that *zygomaticus* activity in the schizophrenia group was comparable to that of the control group when exposed to positive facial expressions. Unexpectedly, however, the schizophrenia group exhibited greater *corrugator* activity when exposed to negative facial expressions, and Kring et al. suggested that this may not have been a "mimicry" reaction *per se*, but instead may have reflected processes related to confusion.

Importantly, facial EMG responses that occur within 1 s poststimulus exposure are less susceptible to the influences of puzzlement and confusion due to their relative rapidity, spontaneity, and subconscious nature (Dimberg et al., 2000; Moody & McIntosh, 2006). Thus, although Kring et al. (1999) identified an intact mimicry response in those with schizophrenia over an extended timeframe, it was predicted that because empathic behaviors are disrupted in this group (and have been argued to reflect structural abnormalities in the human MNS; Bertrand et al., 2008) schizophrenia would be associated with abnormalities in the empathy-related *rapid* facial mimicry response. A secondary aim was to assess whether any observed abnormalities in facial muscle activity are related to clinical symptoms of the disorder, and in particular, blunted affect.

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METHOD

Participants

Twenty-five clinical participants (10 males) and 25 controls (11 males) completed this study. All clinical participants were recruited from outpatient clinics in Sydney, and had received diagnoses of schizophrenia (n = 15) or schizoaffective disorder (n = 10) by treating psychiatrists according to the *DSM-IV* (American Psychiatric Association, 1994). All participants were aged over 18 and in a stable phase of illness. No significant differences were observed between schizoaffective or schizophrenia participants on any of the experimental variables of interest. The control participants were recruited from the general community *via* advertisements. All participants received approximately AUD$10 per hour (≈ US$9) for participation, and exclusion criteria included a neurological disorder, presence of motor abnormalities, alcohol/drug abuse, or any sensory impairment that would interfere with testing. Control participants were additionally screened to ensure that they had no personal history of psychopathology.

Background information for the two groups is reported in Table 1. In addition to being closely matched for gender (40% *vs*. 44% male for the schizophrenia and control groups, respectively), it can be seen that the two groups did not differ in age or years of education. The two groups were also matched with regard to current intelligence as indexed by the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999), as well as premorbid intelligence as indexed by prorated Full Scale IQ on the National Adult Reading Test (NART; Nelson & Willison, 1991). However, greater negative affect, as indexed by the Depression Anxiety Stress Scales-21 (DASS-21; Lovibond & Lovibond, 1995), was reported by the schizophrenia group relative to the control group.

Twenty-four clinical participants were receiving antipsychotic medication (21 atypical, 2 typical, 1 combined typical and atypical). It can be seen in Table 1 that the mean dose of antipsychotic medication received was very comparable to the mean dosage levels reported by a large-scale audit of outpatient antipsychotic usage (360 ± 253 mg/day chlorpromazine equivalents; Humberstone, Wheeler, & Lambert, 2004). It can also be seen that, while the average age of onset and duration of illness indicated the clinical sample were chronic (20.2 and 22.7 years, respectively), scores on the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1984a) and the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984b), indicated a relatively mild level of symptom presentation (consistent with all participants presenting in a stable phase of illness).

Procedure and Measures

Passive viewing facial electromyography paradigm

The current study closely followed the procedures set out by Bailey et al. (2009). Consistent with prior research demonstrating greater expressivity in the muscle regions of the left side of the face (see, e.g., Dimberg & Petterson, 2000), surface EMG was used to record changes in the levels of muscle activity over the left *corrugator* and *zygomaticus* regions. Recording over these regions to index responding to angry and happy expressions is considered standard in facial EMG research, as *corrugator* activity has been shown to be a sensitive marker of angry expressions and *zygomaticus* activity

Table 1. Characteristics of the schizophrenia and control participants

	Schizophrenia		Control		*Inferential statistics*			
Measure	*M*	*SD*	*M*	*SD*	*t*	*df*	*p*	*d*
Demographics								
Age	42.9	9.43	39.2	10.85	1.27	48	.211	.36
Education	13.9	2.79	13.0	1.89	1.28	48	.208	.38
Cognitive function								
WASI	101.9	16.17	107.8	15.67	1.29	47	.202	–.37
NART	105.8	11.80	108.6	12.48	0.81	47	.423	–.23
Negative Affect								
DASS-21	47.0	26.78	23.0	18.49	3.70	48	.001	1.04
Clinical characteristics								
CPZ Equivalents[a]	400.0	275.25	—	—	—	—	—	—
Age of onset	20.2	5.98	—	—	—	—	—	—
Duration of illness	22.7	10.92	—	—	—	—	—	—
SANS	9.4	3.01	—	—	—	—	—	—
SAPS	5.5	3.72	—	—	—	—	—	—

Note. Data for the NART, WASI, and SAPS/SANS are missing for one clinical participant. WASI refers to Wechsler Abbreviated Scale of Intelligence; NART refers to National Adult Reading Test; SAPS refers to the Scale for the Assessment of Positive Symptoms; SANS refers to the Scale for the Assessment of Negative Symptoms; CPZ refers to chlorpromazine; DASS-21 refers to the Depression Anxiety Stress Scales.

[a]Although this conversion is controversial as applied to atypical antipsychotics, it is nevertheless useful for assessing broad trends across patient groups. For details on the conversion procedure used, see Henry, Green, de Lucia, Restuccia, McDonald, and O'Donnell (2007).

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is particularly responsive to expressions of happiness (Tassinary & Cacioppo, 2000). Site preparation and electrode placement adhered to the standard procedure set out by Tassinary and Cacioppo (2000). The skin over the *zygomaticus*, *corrugator,* and center of the forehead was cleansed with an alcohol wipe and then gently abraded with NuPrep gel (Weaver and Co., Aurora, CO). Four gold-plated bipolar surface electrodes, with hat-shaped discs and 9-mm housings, were placed in pairs over the left *zygomaticus* and *corrugator* muscle regions, roughly parallel to the length of the muscle, with an interelectrode distance of approximately 1.25 cm; an additional electrode was placed approximately in the center of the forehead, acting as a ground. The electrodes were attached with Ten20 conductive paste (Weaver and Co.) and fastened with medical tape. Muscle activity was continuously recorded with a PowerLab 8/30 Data Acquisition System (AD Instruments, Castle Hill, Australia) at a sampling rate of 2000 Hz with a 10- to 500-Hz bandpass filter, a 60-Hz notch filter, and an amplification factor of 20,000. The PowerLab was triggered by DMDX (Version 3.2.3.0), software that precisely synchronized timing of the facial expression presentations with the recording of the data acquisition system.

Participants were told that they would be watching a series of stimuli on a monitor and that they should try to remain still throughout the procedure. To disguise the true purpose of the study, participants were also told that the EMG sensors were measuring changes in sweat gland activity (see Bailey et al., 2009). The stimuli were 16 × 24 cm black and white pictures of happy and angry facial expressions (Ekman & Friesen, 1976) that were presented approximately 60 cm in front of the participant. Each trial commenced with a 50 ms soft orienting tone which was followed by the presentation of a facial expression 1 s later. Each face was displayed on the monitor for 5 s, followed by a blank screen for 6 s. Consistent with several previous studies (Bailey et al., 2009; Dimberg & Thunberg, 1998; McIntosh, Reichmann-Decker, Winkielman, & Wilbarger, 2006), pictures of eight different people (four male, four female) were presented to participants. Happy and angry expressions were presented in nonmixed randomized blocks of eight trials (e.g., eight happy images, followed by eight angry images). Again, this procedure was consistent with prior research examining facial EMG reactions to facial stimuli (see Bailey et al., 2009; Dimberg, 1982; Dimberg & Lundqvist, 1990; Dimberg & Petterson, 2000; Dimberg & Thunberg, 1998; McIntosh et al., 2006). Each participant viewed a total of two blocks (one happy, one angry), and these blocks were counterbalanced across participants.

Raw EMG signals were screened for electrical noise and movement artifacts. In addition, a coder who was blind to condition watched videotapes of each participant and noted when they were not watching the stimulus or when they made extraneous movements such as yawning. These amounted to less than 5% of trials, which were deleted from subsequent analyses. An orienting tone, rather than a fixation cross, was used because it alerted the participant to the presentation of the stimulus, and indicated to the rater the time at which they should expect the participant to be looking at the monitor.

The average EMG signal was calculated using the root-mean-square (RMS) method, which represents the square root of the average power of the EMG signal over a given period of time (Tassinary & Cacioppo, 2000). The baseline for each trial was calculated as the average RMS EMG activity 500 ms before each stimulus presentation. The average of each 100 ms period was calculated separately for each expression valence (eight happy, eight angry) to reduce potential variability of responding across trials. Subsequently, the average RMS EMG percentage change from baseline (normalized EMG), averaged across individual trials within each block, was calculated for the window 0–1000 ms poststimulus onset. Consistent with research suggesting that in nonclinical individuals rapid facial mimicry reactions occur within one second of stimulus onset (Dimberg et al., 2002; McIntosh et al., 2006), facial muscle activity in the period 0–1000 ms poststimulus onset was considered for examination of rapid facial mimicry reactions.

Administration of facial EMG and the measures of cognitive and clinical functioning were counterbalanced. All testing procedures received ethical approval from the South Eastern Sydney Area Health Service in conjunction with the University of New South Wales.

RESULTS

EMG Response to Facial Emotion Stimuli

For the EMG analyses, the first step was to examine whether any group differences in average *corrugator* and *zygomaticus* EMG activity emerged for the 500 ms before presentation of the facial stimuli. Averaged across the eight stimulus presentations for each block, there were no group differences in either *corrugator* or *zygomaticus* baseline activity (all *p*s > .05).

The next step was to ascertain whether there were overall differences between the two groups in magnitude of responding to the facial stimuli. Independent samples *t* tests indicated that there were no differences in the level of *corrugator* or *zygomaticus* activity between the schizophrenia and control groups, averaged across angry and happy emotional stimuli ($p = .365$ and $p = .263$, respectively). In addition, differences in facial responding were examined between the schizophrenia and schizoaffective groups. Independent samples *t* tests indicated comparable responding in the schizophrenia and schizoaffective groups in both *corrugator* and *zygomaticus* regions to happy and angry images (all *ps* > .05). There were also no differences in the level of *corrugator* or *zygomaticus* activity between the schizophrenia and schizoaffective groups, averaged across angry and happy emotional stimuli (all *ps* > .05).

The *pattern* of facial responding was then assessed—and specifically, whether appropriate mimicry responses were shown by each group (i.e., whether *zygomaticus* activity was greater in response to happy relative to angry facial

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expressions, and whether *corrugator* activity was greater in response to angry relative to happy expressions). A 2 (group: control, schizophrenia) × 2 (target expression: happy, angry) × 10 (time poststimulus onset: 0–100, 100–200, 200–300, 300–400, 400–500, 500–600, 600–700, 700–800, 800–900, 900–1000 ms) repeated-measures mixed-design analysis of variance was conducted separately for the *zygomaticus* and *corrugator* muscle regions. The statistical assumptions for conventional repeated measures procedures were met. Data for the *zygomaticus* and *corrugator* responses for each group are shown in Figure 1.

For the *zygomaticus* region, there was no main effect of emotion, $F(1,48) = 1.00$; $p = .321$; $\eta_p^2 = .02$, or of group, $F(1,48) = 1.28$; $p = .263$; $\eta_p^2 = .02$. There was also no interaction between emotion and time, $F(9,432) = 1.53$; $p = .134$; $\eta_p^2 = .03$, time and group, $F(9,432) = 0.69$; $p = .720$; $\eta_p^2 = .014$ and no three-way interaction between emotion, time and group, $F(9,432) = 1.20$; $p = .296$, $\eta_p^2 = .02$. However, there was a main effect of time, $F(9, 432) = 4.31$, $p < .001$; $\eta_p^2 = .08$, and most importantly, an interaction between emotion and group, $F(1,48) = 5.16$; $p = .028$; $\eta_p^2 = .09$.

To follow up the interaction between emotion and group, independent samples *t* tests were conducted. These indicated that the two groups exhibited comparable *zygomaticus* activity in response to facial stimuli depicting anger, $t(48) = 0.86$; $p = .394$, but that *zygomaticus* muscle activity was lower in the schizophrenia group when viewing facial stimuli depicting happiness, $t(48) = 2.07$; $p = .044$. Within-subject comparisons also indicated that there was a trend for *zygomaticus* activity in response to happy (relative to angry) facial expressions to be increased in the control group ($p = .069$). However, this was not the case for the schizophrenia group ($p = .224$), for whom the pattern of responding was in the opposite direction to that which would be congruent with a mimicry response [i.e., it can be seen in Figure 1 that there was a trend for greater cheek activity to angry (relative to happy) facial expressions]. The main effect of time was not followed up formally, but indicated that there was increased *zygomaticus* activity as a function of time.

For the *corrugator* region, there was no main effect of emotion, $F(1,48) = 1.93$; $p = .171$; $\eta_p^2 = .03$, or of group, $F(1,48) = 0.84$; $p = .365$; $\eta_p^2 = .01$. There was also no interaction between emotion and time, $F(9,432) = 1.133$; $p = .338$; $\eta_p^2 = .02$, nor time and group, $F(9,432) = .685$; $p = .723$; $\eta_p^2 = .01$, and no three-way interaction between emotion, time and group, $F(9,432) = 1.58$; $p = .119$; $\eta_p^2 = .03$. However, there was a main effect of time, $F(9,432) = 6.54$; $p < .001$; $\eta_p^2 = .12$, and again, most importantly, an interaction between emotion and group, $F(1,48) = 4.54$; $p = .038$; $\eta_p^2 = .09$. The main effect of time was again not followed up formally, but indicated that over time there was a general trend for *corrugator* muscle activity to decrease.

Independent samples *t* tests to follow up the interaction between emotion and group indicated that the two groups exhibited comparable *corrugator* activity in response to facial stimuli depicting happiness, $t(48) = 0.63$; $p = .533$, but that *corrugator* muscle activity was lower in the schizophrenia group when viewing facial stimuli depicting anger, $t(48) = 2.19$; $p = .034$. Within-subject comparisons also indicated that the control group exhibited greater *corrugator* activity in response to angry (relative to happy) facial expressions ($p = .043$); however, this was not the case for the schizophrenia participants ($p = .521$).

Correlates of Facial Mimicry Response

The final analyses focused on investigating the correlates of *zygomaticus* muscle activity to happy facial stimuli, and

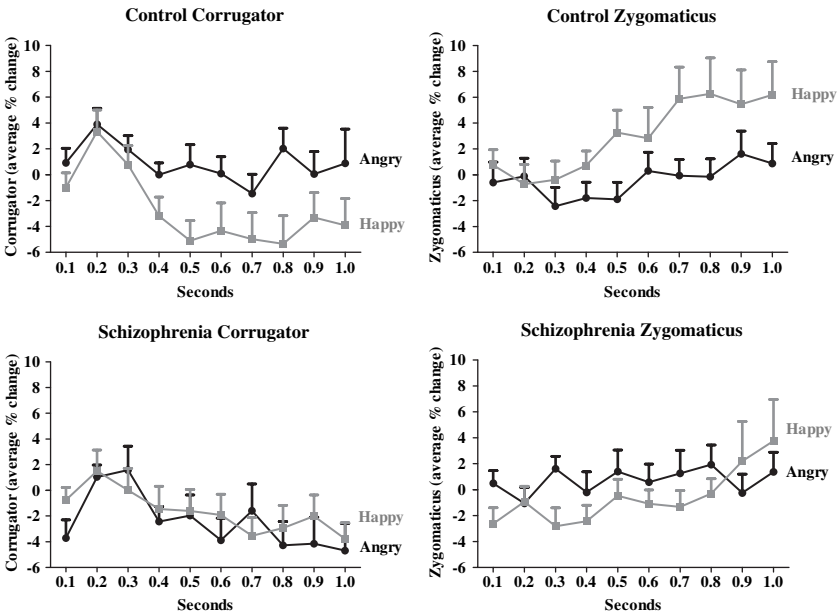

Fig. 1. Mean *corrugator* and *zygomaticus* electromyographic response as percentage change from baseline (+*SE*) to angry and happy stimuli for control and schizophrenia participants.

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corrugator muscle activity to angry facial stimuli (i.e., patterns of facial responding consistent with intact happy and angry mimicry responses, respectively). Results are reported in Table 2. In both groups, neither type of facial muscle activity was related to current or premorbid intelligence, or to negative affect (all *p*s > .05). Furthermore, facial muscle activity was not related to any clinical characteristics in the schizophrenia group as indexed by the SANS and the SAPS, duration of illness and chlorpromazine equivalents (all *p*s > .05).

DISCUSSION

The nonclinical control group displayed a distinct pattern of EMG responding consistent with a typical "mimicry" response (i.e., greater *zygomaticus* activity in response to happy faces relative to angry, and greater *corrugator* activity to angry faces relative to happy). That is, the mere perception of an emotional facial expression led control participants to rapidly and involuntarily produce a corresponding facial reaction within one second of stimulus exposure. It should be noted that the pattern of controls' *corrugator* activity in response to angry facial expressions was not an absolute increase in relation to the prestimulus (baseline) levels. A comparable pattern of facial EMG activity, however, has been observed in other studies examining this rapid facial mimicry response and has been attributed to preparatory activity of the brow (Dimberg, Hansson, & Thunberg, 1998; Dimberg et al., 2000). The critical finding was that *corrugator* activity was greater to angry facial expressions than happy facial expressions in nonclinical controls, psychophysiologically constituting a facial mimicry response (Dimberg et al., 2000). These findings are therefore consistent with considerable prior research demonstrating that emotional facial expressions are sufficient to elicit the most rapid of facial mimicry reactions (Bailey et al., 2009; Dimberg et al., 2000, 2002; McIntosh et al., 2006).

However, because impairments in empathic responding are well documented in schizophrenia (Derntl et al., 2009; Shamay-Tsoory et al., 2007), and have been argued to reflect structural abnormalities in the human MNS (Bertrand et al., 2008), it was predicted that this group would exhibit a disruption in this empathy-related rapid facial mimicry response. Consistent with predictions, the schizophrenia group failed to produce involuntary facial reactions congruent with observed emotional facial expressions. These group differences could not be attributed to demographic factors, as the two groups were equated in age, years of education, gender, as well as premorbid and current intellectual abilities. Furthermore, the failure to identify any association between facial muscle activity and clinical or cognitive parameters suggests that the absence of a typical rapid facial mimicry response is not secondary to specific clinical features of the disorder. However, this does not preclude a specific association between social functioning and facial muscle activity. This is particularly salient following research noting that clinical symptoms and general cognition cannot wholly account for the variance in social functioning in schizophrenia (Milev, Ho, Arndt, & Andreassen, 2005; Pijnenborg et al., 2009). Future research is needed to ascertain whether rapid facial movements relate to social functioning in this group. It should also be noted that the size of the schizophrenia sample was comparable to, or larger than, all previous EMG studies that have been conducted with this group (Median *n* = 17.5; Kring et al., 1999; Mattes et al., 1995; Wolf et al., 2004, 2006).

The group differences also cannot be easily attributed to attentional or motivational factors. This is because rapid facial mimicry responses are involuntary, preconscious reactions, with their elicitation only requiring the mere

Table 2. Pearson correlations between zygomaticus activity to happy facial stimuli and corrugator facial muscle activity to angry facial stimuli with measures of cognitive function, negative affect, and psychopathology

	Schizophrenia group		Control group	
Measure	*Zygomaticus*	*Corrugator*	*Zygomaticus*	*Corrugator*
Cognitive Function				
WASI IQ	.08	−.21	.31	.13
NART IQ	−.03	−.38	.28	.08
Negative Affect				
DASS-21	.15	.37	−.27	−.16
Psychopathology				
SAPS	−.16	.13	—	—
SANS	−.31	.04	—	—
Blunted Affect	−.15	−.11	—	—
CPZ Equivalents	−.22	−.08	—	—
Duration of Illness	.27	−.04	—	—

Note. Additional partial correlations (controlling for medication) revealed no associations between pattern of responding and key clinical variables (SAPS, SANS, blunted affect, duration of illness; *p*s > 0.5). WASI refers to Wechsler Abbreviated Scale of Intelligence; NART refers to National Adult Reading Test; DASS-21 refers to the Depression Anxiety Stress Scales; SAPS refers to the Scale for the Assessment of Positive Symptoms; SANS refers to the Scale for the Assessment of Negative Symptoms; CPZ refers to chlorpromazine.

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observance of an emotional facial expression. Indeed, Dimberg et al. (2002) demonstrated that, even when explicitly instructed to inhibit facial muscle activity, participants still involuntarily produced rapid facial mimicry reactions. As the current study involved examination of videotaped recordings to ensure participants were watching the presented facial expressions, it is unlikely that clinical participants would have been able to consciously or unconsciously inhibit the evocation of these reactions as a result of diminished motivation.

Moreover, the group differences in early, rapid facial responding in the current study seem unlikely to reflect medication effects. First, as noted, no baseline differences in facial EMG activity were observed between the schizophrenia and control groups. This suggests that medication was not having any impact on prestimulus facial muscle tension. Second, no group differences were observed in the overall level of facial EMG activity in either the *corrugator* or *zygomaticus* muscle regions during stimulus exposure. This indicates that it was only the *pattern* of responding that differed between the two groups—not their overall reactivity. Third, no associations were found between chlorpromazine equivalents and facial muscle activity, nor were any associations identified between key clinical variables and facial responding after controlling for medication level. Finally, although Kring et al. (1999) examined facial EMG responding over a longer timeframe, no differences in EMG activity among individuals with schizophrenia receiving neuroleptic medication and those not taking medication were identified.

Involuntary facial mimicry has been implicated in, and thought to facilitate, empathy *via* processes of simulation and perception-action coupling subserved by the MNS (Gallese, 2001; Oberman & Ramachandran, 2007; Preston & deWaal, 2002). Thus, involuntary facial mimicry has been regarded as an important low-level mechanism contributing to the experience of empathy (for a review, see Singer & Lamm, 2009). Consistent with this assumption, Sonnby-Borgstrom, Jonsoon, and Svensson (2003) found that nonclinical volunteers psychometrically identified as being high in empathy produce more rapid, involuntary mimicry reactions compared with low-empathic individuals. Furthermore, atypical rapid facial mimicry reactions have been identified in autism spectrum disorders, a group also characterized by marked empathizing deficits and abnormal MNS activity (Beall, Moody, McIntosh, Hepburn, & Reed, 2008; Cattaneo et al., 2007; Dapretto et al., 2006; Iacoboni & Dapretto, 2006; McIntosh et al., 2006; Oberman, Winkielman, & Ramachandran, 2009). Having now also demonstrated a disruption in the elicitation of rapid facial mimicry reactions in schizophrenia, the current study points to a need for further research that directly maps how aberrant facial mimicry responses in this group relates to other lower- and higher-level empathic processes.

An important caveat, however, is that while rapid facial mimicry reactions have been linked to perception-action neural links mediated by the MNS (e.g., Niedenthal, Barsalou, Winkielman, Krauth-Gruber, & Ric, 2005; Williams, Whiten, Suddendorf, & Perrett, 2001), others have argued that such reactions might instead reflect an emotional response (e.g., Moody et al., 2007). This view stems from the finding that rapid facial reactions to affective stimuli can be modulated through emotion induction (Moody et al., 2007). Thus, although the present data have been interpreted within the context of potential human MNS abnormalities in schizophrenia, an important limitation of these data is the absence of specific information relating to the neurological profile of the schizophrenia participants tested. Further research incorporating neuroimaging techniques, such as fMRI, is therefore required to delineate the precise mechanisms underlying this empathy-related rapid facial response. At present, it is not clear whether an impaired involuntary mimicry response in schizophrenia (or autism) is due solely to a disrupted perception-action neural link or early emotional processing deficits (or a contribution of each). Future research should also incorporate emotional nonfacial stimuli to clarify whether an atypical involuntary mimicry response in schizophrenia reflects a face-specific processing deficit in this group (at a visual scanning or neural level) or a more general motor mimetic impairment. For example, in a voluntary imitation paradigm, Park, Matthews, and Gibson (2008) found that individuals with schizophrenia demonstrated a range of impairments in motor imitation ability, from hand actions to voluntary imitation of emotional facial expressions.

To conclude, the present study provides the first evidence that individuals with schizophrenia demonstrate an atypical rapid facial mimicry response. Given that rapid facial mimicry has been conceptualized as an important mechanism in the experience of empathy, the current findings map onto the broader schizophrenia literature which has documented impairment in empathic behaviors, and more recently, abnormalities in the MNS. At present, the neural mechanisms and component processes that underlie the pervasive empathic and social cognitive deficits seen in schizophrenia remain poorly understood (Pinkham, Hopfinger, Pelphrey, Piven, & Penn, 2008). These data highlight abnormalities in rapid facial mimicry responses, and their neural underpinnings, as one potential area that warrants further consideration.

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