

Deficits in Early Stages of Face Processing in Schizophrenia: A Systematic Review of the P100 Component

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Introduction. Schizophrenia is associated with deficits in face and affect recognition, which contribute to broader social functioning deficits. The present aim was to conduct a meta-analysis of early face processing in schizophrenia, as indexed by the P100 event-related potential component. **Methods.** Twelve studies ($n = 328$ patients with schizophrenia, $n = 330$ healthy controls) of the P100 component during face processing were evaluated by calculating Cohen's d for each study and overall weighted mean effect size (ES). In additional exploratory analyses, moderating influences of method and design were investigated, and the P100 component during face processing was evaluated based on valence: 5 studies ($n = 225$ patients, $n = 225$ controls) included neutral stimuli, 5 studies ($n = 225$ patients, $n = 225$ controls) included happy stimuli, and 4 studies ($n = 209$ patients, $n = 209$ controls) included fearful stimuli. **Results.** The amplitude of the P100 to face stimuli was smaller in patients relative to controls ($ES = .41$, $P < .01$). Methodological or design differences did not account for heterogeneity in ES. When split by valence, results indicate smaller P100 in patients relative to control subjects in response to neutral ($ES = .32$, $P < .001$) and happy ($ES = .21$, $P < .05$) stimuli, whereas there was no difference in response to fearful faces ($ES = .09$, $P > .05$). **Discussion.** The results indicate that P100 amplitude in response to faces is smaller in patients with schizophrenia, showing that socially relevant visual processing deficits begin earlier in processing than previously suggested. Additionally, the exploratory analyses suggest emotional specificity in these deficits. Ramifications for our understanding of face processing deficits and treatment development are discussed.

Key words: P100 component/emotion/ERP

Schizophrenia is a disorder that affects 1% of the population, and is characterized by social deficits that contribute to the marked impairment and related disability.¹ Face

processing is a key aspect of these social deficits, and has been the focus of many studies of schizophrenia.² In order to assess the underlying mechanisms, many studies have examined the neural correlates of face and affect processing deficits in this population. In this regard, the use of electroencephalography (EEG) to examine event-related potentials (ERP) in response to faces is a particularly powerful investigative methodology as the high temporal resolution can give insight into underlying mechanisms and differential stages of processing. The majority of ERP studies of face processing in schizophrenia have focused on the N170 component, which is reflective of holistic processing of objects of expertise, such as faces.³ However, deficits may stem from earlier visual processing stages, such as those indexed by the P100 component, and to date, there has been no systematic review focusing on this component. Furthermore, while some of the related studies have shown significant differences between patients with schizophrenia relative to healthy controls in several key components that are associated with the processing of faces,^{4–8} findings reported in the critical early processing stages have been mostly inconsistent.^{9,10} The present review examines whether there are discrepancies in early visual processing, as indexed by the P100 ERP component, between patients with schizophrenia and healthy controls and aims to determine if differences in methodology and emotional valence of facial stimuli used between studies contribute to discrepant findings in this area.

A recent meta-analysis was conducted to examine discrepancies in amplitude between patients with schizophrenia and healthy controls for 2 event-related potentials that have been associated with face processing;¹¹ the N170, a negative deflection occurring approximately 170ms after stimulus onset, and the N250, a negative deflection occurring approximately 250ms after stimulus onset. The amplitude of the N170 component at lateral electrode sites has been shown to be

larger for human faces relative to nonhuman faces or novel categories that are not objects of visual expertise¹² and is thought to reflect basic structural encoding.^{13,14} The N250 has been associated with face¹⁵ and object¹⁶ familiarity. In an analysis of 21 (N170: mean weighted effect size (ES) = .64) and 6 (N250: ES = .49) studies of face processing, McCleery and colleagues¹¹ observed that both of these components were shown to differ between patients and healthy controls, giving clear evidence of discrepancies related to face processing in patients.

Although McCleery and colleagues¹¹ demonstrates that there are facial processing discrepancies between patients and controls, the meta-analysis was limited to the N170 and N250 components. This approach provides an important perspective of the psychophysiological correlates of facial processing, but to date, our understanding of the body of literature focusing on the integral early sensory processing function remains very limited. As decreased activation in brain regions associated with early visual processing of faces, such as the occipital and fusiform gyri, has been shown for patients with schizophrenia relative to healthy controls,¹⁷ the earliest stages of face processing are important to consider. Indeed, it has been shown that meaningful face-specific categorization begins at around 100ms after stimulus onset, as indexed by the P100 component.^{18,19} Originally thought to reflect only basic visual processing,²⁰ and early visual attention,^{21–23} an emerging literature suggests that the P100 is modulated during face processing. Specifically, studies have shown greater P100 amplitude during visual processing of faces relative to nonface objects,^{19,24,25} and P100 modulation by emotional expression of faces.^{5,26–31} For example, Pourtois and colleagues²⁹ found enhanced P100 amplitude to fearful faces in the general population, and Kolassa and Miltner³¹ found enhanced P100 to angry and happy faces relative to neutral in both people with social anxiety and healthy controls. Therefore, the P100 component may provide important information about underlying mechanisms in the deficits in face and affect recognition in psychiatric disorders such as schizophrenia. Given the broader literature which suggests that patients with schizophrenia exhibit deficits in processing specific types of faces,³² and that underlying abnormalities appear to be present at several stages in affective recognition,³³ synthesizing the available P100 literature is an important first step in both understanding core deficits and initiating targeted treatment approaches.

As noted, several individual studies have shown deficits in early visual processing in patients with schizophrenia, as indexed by the P100 component.^{4–8} In fact, 1 study has found discrepancies in P100 amplitude between individuals at-risk for schizophrenia and healthy controls, suggesting that these early processing deficits may be indicative of vulnerability to psychosis.³⁴

However, these results have been variable, as many studies have found no differences in P100 amplitude between patients and controls.^{9,10,35–42} Methodological differences, such as the use of emotional vs nonemotional stimuli, may contribute to these discrepancies in the literature. It is important to address whether social processing deficits in schizophrenia, specifically face and affect processing, begin from atypicalities in early perceptual processes. Furthermore, understanding how methodological or sampling differences (eg, method of P100 extraction, inclusion of emotional faces) may contribute to discrepant findings is a vital step for moving forward. The current quantitative review aims to examine (1) whether the overall mean ES for the P100 in response to face stimuli show differential activation for patients with schizophrenia relative to healthy controls and (2) whether there are any potential moderators of this effect (eg, examining mean vs peak P100 amplitude, use of emotional vs nonemotional stimuli). Further, in a set of exploratory analyses we aimed to examine independent emotional expressions, to see whether the overall mean ES for P100 in response to specific emotions (happiness, fear, and neutral) show differential activation for patients relative to healthy controls.

Methods

Eligibility Criteria

Inclusion criteria for the analysis were as follows: (1) the study included a sample of patients meeting criteria for schizophrenia (other disorders, including schizoaffective disorder, were not included) and a healthy control group, (2) the study stimuli included faces, (3) EEG was recorded while participants viewed faces, (4) statistics were reported that allowed calculation of the P100 component, defined as a positive deflection peaking 50–170ms post stimulus onset in the occipital region,⁴³ for both the patient and control group, and (5) the manuscript was written in English and published in a peer-reviewed journal. The cutoff date for the literature search was December 1, 2014.

Study Selection

The literature search was conducted using PubMed and Google Scholar with the following search terms: Schiz* AND P100 OR P1 AND fac*, which resulted in 375 articles. These articles were examined for eligibility and the citations were cross-referenced. Fifteen of the articles met eligibility criteria. Three articles did not provide sufficient information to calculate ES and attempts to obtain the data from the corresponding authors were unsuccessful.^{36,38,41} Thus, 12 ($n = 328$ patients, $n = 330$ controls) full-text articles were included in the current analyses. For the study characteristics, see [table 1](#). For the mean characteristics of the patient samples in each study, see [table 2](#).

Table 1. Study Characteristics

Study	<i>n</i> SZ:Ctrl	Face Stimuli	Expressions	Stimuli Color	Task	ERP
Brennan et al ⁴	108:108	NR	Angry, disgust, fearful, happy, neutral, sad	NR	No response required	Peak
Caharel et al ⁵	18:18	Self-face, 1 familiar face, 2 unknown faces	Disgust, happy, neutral	Grey scale	Respond to familiarity	Peak
Campanella et al ⁶	14:7	Ekman and Friesen	Happy, fearful, neutral, sad	NR	Respond to deviant face	Peak
Herrmann et al ¹⁰	24:28	12 different faces	Neutral	Grey scale	Silently categorize face or building	Peak
Jetha et al ³⁷	40:39	Ekman and Friesen	Angry, happy, fearful, neutral	Grey scale	No response required	Peak
Jung et al ³⁹	23:24	Chaelee face	Fearful, happy, neutral	Color	Respond to emotional face	Peak
Lee et al ⁴⁰	38:38	Chaelee face	Fearful, happy, neutral	NR	Respond to happy or fearful face	Peak
Müller et al ⁷	15:15	Facial emotions for brain activation inventory	Fearful, happy, neutral	Color	Rate expression	Mean
Obayashi et al ³⁵	16:23	JACFEE and JACNeuF	Angry, happy, fearful, neutral	Grey scale	Respond to image of shoes	Peak
Thoma et al ⁸	14:15	NimStim	Happy, fearful, neutral, sad	Grey scale	Respond to congruency of emotional valence (body/face)	Peak
Turetsky et al ⁴²	16:16	Penn facial emotion stimuli	Happy, neutral, somewhat sad, very happy, very sad	Grey scale	No response required (NR)	Mean
Wynn et al ⁹	26:27	Ekman and Friesen	Afraid, angry, ashamed, happy, sad, surprised	Grey scale	Classification (gender, emotion, or building)	Mean

Note: ERP, Event-Related Potentials; Ctrl, Healthy Controls; NR, Not Reported; SZ, Schizophrenia Patients; JACFEE, Japanese and Caucasian Facial Expressions of Emotion; JACNeuF, Japanese and Caucasian Neutral Faces.

Analyses

The primary variable of interest was the amplitude of the P100 ERP component in response to face stimuli for patients with schizophrenia compared with healthy control subjects (although definitions of the P100 differed by study, all studies included a positive deflection within the range of 50–170ms poststimulus onset). For each study, pooled ES (Cohen's *d*) was calculated to define the differences in P100 amplitude for the patient and control groups. Cohen's *d* was defined as the difference between group amplitudes divided by pooled within-group *SD* of both groups.⁴⁴ For the primary meta-analysis, each independent study was represented by 1 ES. When studies reported nonindependent results (eg, separated by hemisphere, multiple patient groups), conservative methods were used to calculate an independent standardized ES for each study.^{45,46} Specifically, 1 study reported results based on 1 control sample against multiple patient samples (patients high on symptom ratings, *n* = 7, and low on symptom ratings, *n* = 7),⁶ so separate ES were calculated for each patient group and the weighted mean was used for analysis.⁴⁵ Most studies reported P100 data at the average of occipital electrodes. To avoid duplication of analyses, when studies reported

the amplitude separately by hemisphere,^{4,8,37,40} we entered the mean between the hemispheres.^{45,46} The standardized ES were analyzed using random effects meta-analysis. For comparison of moderator variables in the primary meta-analysis, the *Q* test on heterogeneity between groups was used. Publication bias (the increased probability of statistically significant results to be published) was assessed using Egger's test as well as the graphical funnel plot method. An asymmetrical funnel plot would suggest publication bias due to smaller negative studies not appearing in the literature.⁴⁷ In all analyses, a positive ES is indicative of greater amplitude in healthy controls relative to patients.

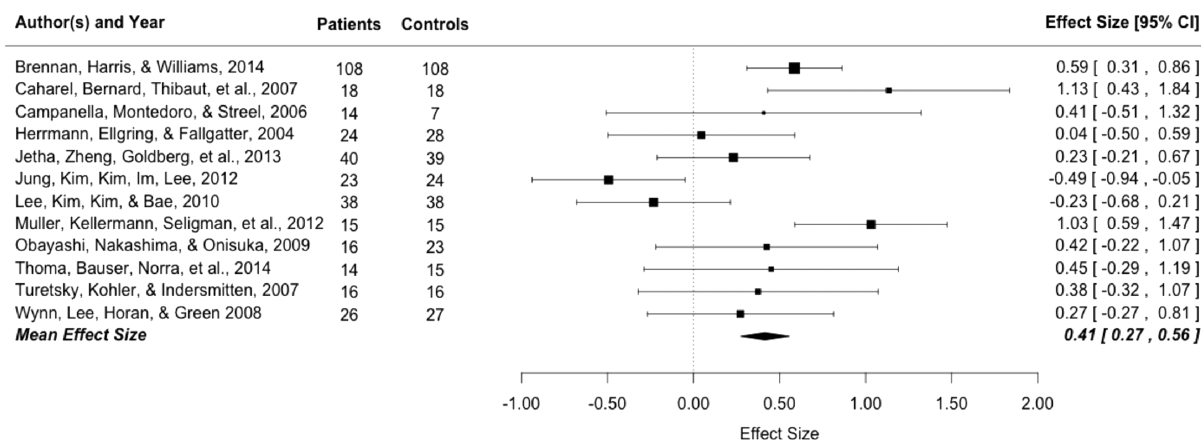
Results

This analysis is based on 328 patients and 330 healthy controls over 12 studies. The mean weighted ES of the 12 studies was of small to medium magnitude (ES = .41, *SE* = .07, 95% CI: .27, .56) with the P100 amplitude of patients being smaller than that of healthy controls (figure 1). This weighted mean ES differed significantly from zero (*z* = 5.72, *P* ≤ .01). The distribution of the ES indicated heterogeneity (*Q*₁₁ = 28.04, *P* ≤ .01), therefore the dispersion of ES is greater than expected

Table 2. Mean Characteristics of Patient Samples

Study	Age	Gender M:F	Clinical Status	Symptom Ratings	Illness Duration (y)	Medication Dosing (mg)
Brennan et al ⁴	20.7 (2.9)	70:38	Outpatient	PANSS total: 61.14 (18.25)	26.73 (55.34) (wk)	390.31 (193.58) CPZ equiv
Caharel et al ⁵	37.7 (8.29)	10:8	Inpatient and outpatient	NR	13.6 (9.97)	363.9 (297.44) CPZ equiv
Campanella et al ⁶	47.7 (11.9)	9:5	Inpatient	PANSS total: 88 (15) for low; 115 (17) for high	NR	9.01 (10.3) haloperidol equiv
Herrmann et al ¹⁰	32.3 (10)	19:5	NR	PANSS total: 25.3 (7.1)	NR	666 (430) CPZ equiv
Jetha et al ³⁷	42.4 (6.4)	28:12	Outpatient	PANSS total: 26 (5.5)	22.63 (5.9)	NR
Jung et al ³⁹	32.2 (10.1)	12:11	NR	PANSS total: 81.8 (25.8)	5.2 (4.9)	NR
Lee et al ⁴⁰	30.2 (10.3)	16:22	NR	PANSS 83.5 (22.8)	4.3 (3.3)	398 (103) M; 387 (120) F
Müller et al ⁷	35.1 (9.26)	11:4	Outpatient	SAPS total: 29.80 (21.6); SANS total: 37.8 (13.3)	14.33 (9.12)	NR
Obayashi et al ³⁵	32.9 (10)	16:0	NR	SAPS total: 7.6 SANS total: 12.5 (<i>SDs</i> NR)	9.9 (7.3)	487 (319) CPZ equiv
Thoma et al ⁸	34.1 (11.2)	8:6	Inpatient and outpatient	NR	NR	619.4 (500.5) CPZ equiv
Turetsky et al ⁴²	30.5 (6)	12:4	Outpatient	SAPS total: 1.4 (0.7) M; 0.3 (0.3) F; SANS total: 1.8 (0.7) M; 0.9 (0.3) F	7.5 (5.5) M; 10.5 (8.3) F	NR
Wynn et al ⁹	43.9 (10.2)	21:5	Outpatient	BPRS total 44.3 (8.9)	NR	NR

Note: BPRS, Brief Psychiatric Rating Scale; CPZ equiv, chlorpromazine equivalent; F, Female; M, Male; NR, Not Reported; PANSS, Positive and Negative Syndrome Scale; SANS, Scale for the Assessment of Negative Symptoms; SAPS, Scale for the Assessment of Positive Symptoms.

**Fig. 1.** P100 Effect Sizes and forest plot.

from sampling error. The funnel plot was symmetrical and Egger's regression test of funnel plot asymmetry was not significant (intercept = -0.17 , $SE = 1.8$, $P = .93$, $CI: -1.52, 2.68$), which does not suggest publication bias.

Moderating Variables

Moderating variables were tested using a mixed-effects model which showed that the ratio of males to females in

the patient sample, average age of patients, average medication dosage, nor average illness duration were significant moderators (all $P > .4$). The Q statistic indicated that the distribution of P100 ES departed from homogeneity. Therefore, we conducted follow-up analyses to examine whether specific methodological differences impacted the results. Method of P100 extraction (mean or peak) did not account for significant heterogeneity in ES [ES_{peak} ($n = 9$) = $.35$, $SE = .08$, ES_{mean} ($n = 3$) = $.65$, $SE = .16$,

$Q_{\text{between1}} = 1.16, P > .2]$. Additionally, task (passive or active response) did not account for significant heterogeneity in ES [$ES_{\text{pass}} (n = 4) = .41, SE = .1, ES_{\text{act}} (n = 8) = .33, SE = .1, Q_{\text{between1}} = 0.36, P > .5]$. Finally, differences in design that involved the inclusion of emotional faces (rather than only using neutral faces) did not account for significant heterogeneity in ES [$ES_{\text{emo}} (n = 10) = .39, SE = .09, ES_{\text{non}} (n = 2) = .46, SE = .12, Q_{\text{between1}} = 0.01, P > .9]$. However, only 2 articles did not involve the inclusion of emotional faces. For the other 10 articles, the standardized mean ES is reflective of the average of different emotional expressions. As emotion-specific deficits have been found in schizophrenia,^{48–50} it is important to examine whether deficits in this processing stage differ by the valence of face stimuli. Thus, further analyses were conducted to assess the impact of specific emotions when information was available. A small but sufficient amount of studies reported independent information for happy,^{4,37,39,40,42} fearful,^{4,37,39,40} and neutral^{4,10,37,39,42} stimuli.

Valence of Face Stimuli

As previously stated, we were interested in conducting exploratory analyses to examine whether differences in P100 amplitude between patients and controls were dependent upon the emotional valence of the presented faces. Therefore, standardized mean ES were calculated independently for happy, neutral, and fearful faces when information was provided to do so. To prevent duplication of analyses or reporting on nonindependent samples, 3 separate small meta-analyses were conducted. The results are as follows and can be seen in figure 2.

Neutral. The analysis of neutral faces is based on 225 patients and 225 healthy controls over 5 studies.^{4,10,37,39,42}

The mean weighted ES was of medium magnitude ($ES = .32, SE = .09, 95\% \text{ CI}: .13, .52$) and differed significantly from zero ($z = 3.72, P \leq .001$). P100 amplitude while viewing neutral faces was greater for healthy controls relative to patients.

Happy. The analysis of happy faces is based on 225 patients and 225 healthy controls over 5 studies.^{4,37,39,40,42} The mean weighted ES was of small magnitude ($ES = .21, SE = .09, 95\% \text{ CI}: .03, .40$), and differed significantly from zero ($z = 2.27, P \leq .05$). P100 amplitude was greater for controls relative to patients when viewing happy faces.

Fearful. The analysis of fearful faces is based on 209 patients and 209 healthy controls over 4 studies.^{4,37,39,40} The mean weighted ES was of small magnitude ($ES = .09, SE = .1, 95\% \text{ CI}: -.11, .28$) and did not differ significantly from zero ($z = 0.87, P > .05$), therefore there was no difference in P100 amplitude in response to viewing fearful faces for patients relative to controls.

Discussion

Efficient early perceptual encoding of faces is an important step for later recognition. The current review is the first comprehensive study to suggest that deficits in early visual processing are present in schizophrenia. More specifically, the results of the current meta-analysis show that there are consistent deficits in early visual processing in patients with schizophrenia, as indexed by the P100 component. This suggests that deficits in face processing in patients may begin earlier in processing than previously suggested and that an early sensory processing deficit may precede higher-order processing deficits as indexed by the N170 component. Importantly, our

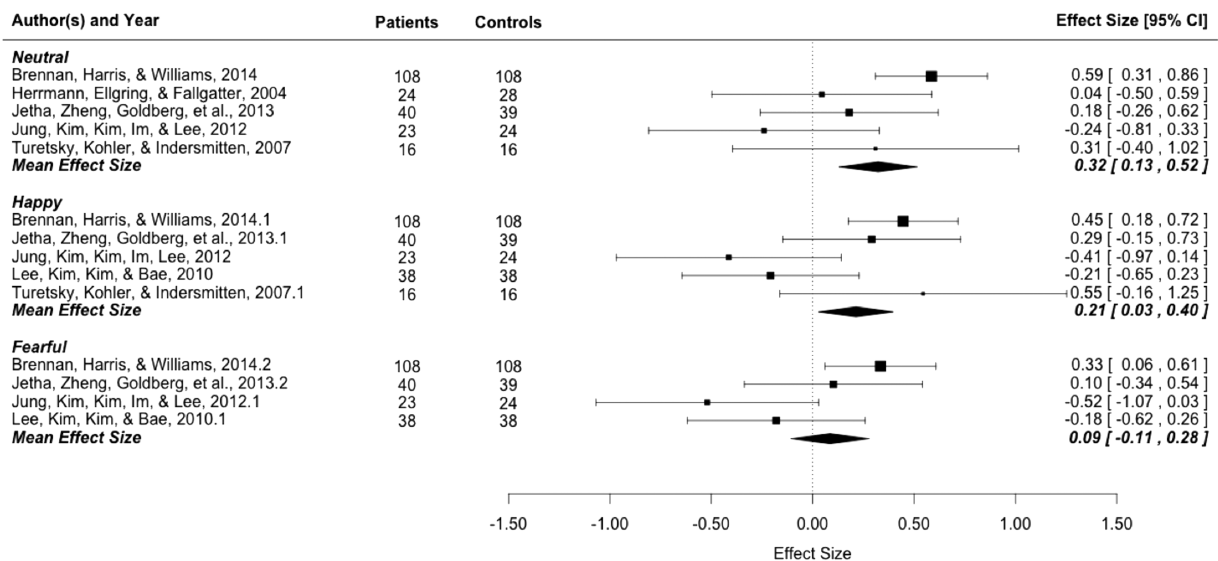


Fig. 2. P100 Effect Sizes and forest plot by meta-analyses based on emotional valence.

moderator analyses suggest that these perceptual deficits may depend on facial expression.

Much previous research has shown that patients with schizophrenia exhibit deficits in processing faces and facial affect,³² which has been associated with social functioning deficits.^{51,52} The present analysis also provides some evidence to suggest that early perceptual processing deficits are specific to neutral faces and faces portraying certain emotional expressions. In our 3 exploratory meta-analyses, it was found that patients show lower P100 amplitude to faces exhibiting neutral or happy expressions, but there is no difference between patients and controls for fearful expressions.

Our findings of emotional specificity in the processing deficits found in patients with schizophrenia are consistent with previous literature.^{48–50} For example, studies have found differential neural patterns of activation for negative vs neutral or positive facial expressions, suggesting that deficits in face and affect processing may be unique to specific emotions. Gur and colleagues⁵³ demonstrated differential limbic region activation for patients with schizophrenia relative to controls when viewing angry and fearful faces. Differential amygdalar responses to happy but not to fearful expressions have also been demonstrated in patients with schizophrenia,⁵⁴ although other studies have shown reduced amygdalar responses to fearful expressions.^{55,56} Importantly, Holt and colleagues⁵⁷ found reduced hippocampal habituation to fearful faces in patients relative to controls. However, no differences in activation to fearful faces between patients and controls were found in initial responses. Specifically, during 80-s blocks, initial hippocampal activation (blood oxygenation level-dependent [BOLD] signal during the first 40 s of repeated presentation) did not differ between patients and controls, whereas later activation (BOLD signal during last 40 s) decreased for controls but did not decrease for patients. The authors concluded that deficits in fearful face processing in patients with schizophrenia are not associated with a reduction in initial activation. Together with the current analysis, these findings suggest that deficits in fearful face processing in schizophrenia are due to later processing stages. Initial stages of face perception may only be impaired in patients when viewing neutral or positive valence faces, and may indeed be intact when viewing negative valence faces.

The primary limitation of the literature in this area relates to the small number of studies that report on the P100 component, and the smaller number of studies that report based on independent emotional expressions. As social processing deficits are robust in schizophrenia, it is important to examine the early perceptual processes associated with these deficits. As evidenced by our exploratory analysis, the literature would benefit from a detailed exploration of the deficits in recognition and processing of specific emotions, and the underlying neural impairments associated with these. Additionally, our analyses on specific emotions were limited to happy, fearful, and

neutral faces. This was due to an underrepresentation of other emotional stimuli (eg, angry, sad, surprised) in the literature. The increased use of multimodal approaches, as well as a greater representation of multiple emotional categories, would aid in our understanding of the detrimental social processing deficits in schizophrenia.

As noted previously, the P100 component has been associated with basic visual processing^{20,58} and spatial attention,^{21–23} and has only recently been shown to be modulated by factors such as emotional expression in face processing. This is important in that the findings of this review may extend beyond face processing and reflect deficits in basic visual encoding in schizophrenia. Indeed, studies have shown deficits in P100 amplitude in schizophrenia when encoding nonface stimuli, such as fragmented images^{59,60} and simple visual stimuli differing in luminance and contrast.⁶¹ It has been suggested that early sensory processing are associated with early dorsal, rather than ventral, visual stream processing,⁶² and that these deficits may be associated with poor working memory performance.⁶³ In fact, the P100 component has been successfully (80%–90% accuracy) used as a classifier for schizophrenia while patients and controls viewed basic visual images.⁶⁴ It has been suggested that these deficits are a function of impairments in visual attention in patients relative to healthy controls⁶⁵ and/or general deficits in early-stage visual processing.⁶¹ As all of the studies and stimuli types used in the fearful analysis were also included in the neutral and happy analyses, the differences between groups based on emotion are unlikely to be the result of differences in lower-level visual cues, such as contrast or color. However, it has been suggested that negative emotions are less discriminable⁶⁶ and this is exacerbated in patients due to generalized perceptual deficits.⁶⁷ The current findings may also be indicative of a decrease in visual attention to faces in general in patients with schizophrenia. Indeed, there has been evidence of an attentional advantage of emotional relative to neutral stimuli in healthy controls but not in patients.⁶⁸ As our moderator analyses show the largest difference between patients and healthy controls when viewing neutral faces, it is possible that patients initially process all faces, regardless of valence, in a manner similar to the processing of fearful faces in healthy controls. Future research is needed to examine these possibilities.

When developing potential remediations, the findings of the current analysis highlight important aspects to consider. First, face processing deficits occur earlier in visual processing than previously suggested, and therefore, social functioning remediations should focus on early stages of processing as well. Second, there may be emotional specificity of deficits in face processing in schizophrenia at different stages of processing. Given that neutral and positively valence faces are affected by early perceptual stages of processing, whereas negatively valence faces may be affected by later processing, different

treatments or training strategies are necessary to improve face processing in schizophrenia. Specifically, neutral and positive expressions may benefit from basic perceptual face training paradigms, whereas a focus on higher order cognition may improve processing and recognition involving negative expressions. Indeed, improvements in emotion recognition, as demonstrated both behaviorally and with ERP responses, has been shown following cognitive interventions.⁶⁹ Importantly, Training on Affect Recognition (TAR) has been successful in patients with schizophrenia.^{70–75} Interestingly, TAR differentially impacts certain emotions, such that training improves recognition of happiness and sadness, but not fear, anger, and disgust.⁷³ Use of training has also been successful in increasing face recognition accuracy in children⁷⁶ and adults⁷⁷ with autism spectrum disorders, and training in this population has been shown to modulate the P100 but not the N170 component.⁷⁸ Therefore, individuals with schizophrenia may also benefit from perceptual expertise training, but this may be limited to specific emotions.

The current analysis examined whether early perceptual deficits, as indexed by the ERP P100 component, are present in patients with schizophrenia relative to healthy controls. We have found that P100 amplitude to faces is reliably decreased in patients, suggesting that deficits in face processing in this population begin earlier than previously expected. Additionally, there is some evidence that these deficits are emotion specific. We believe this is an important step towards understanding at what stage face processing deficits occur. Future directions should aim to examine how deficits in face processing may relate to other generalized perceptual deficits, and how these deficits, as indexed by the P100, relate to later stages of processing, as indexed by components such as the N170 and N250. Finally, it would be of importance to examine whether these deficits are specific to schizophrenia or extend to other clinical populations.

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