REVIEW PAPER

Electrophysiological Studies of Reception of Facial Communication in Autism Spectrum Disorder and Schizophrenia

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Abstract

Autism spectrum disorder (ASD) and schizophrenia spectrum disorders (SZ) are characterized by difficulty with social cognition and atypical reception of facial communication—a key area in the Research Domain Criteria framework. To identify areas of overlap and dissociation between ASD and SZ, we review studies of event-related potentials (ERP) to faces across ASD and SZ populations, focusing on ERPs implicated in social perception: P100, N170, N250, and P300. There were many inconsistent findings across studies; however, replication was strongest for delayed N170 latency in ASD and attenuated N170 amplitude in SZ. These results highlight the challenges of replicating research findings in heterogeneous clinical populations and the need for transdiagnostic research that continuously quantifies behavior and neural activity across neurodevelopmental disorders.

Keywords Autism spectrum disorder · Schizophrenia · Electrophysiology · Face · Emotion · Research Domain Criteria

Autism spectrum disorder (ASD) and schizophrenia spectrum disorders (SZ) are highly prevalent neurodevelopmental disorders (ND) with considerable public health impact, costing the USA a combined $423 billion per year (Cloutier et al., 2016). Though these disorders are classified in two separate diagnostic taxonomies, social deficits are diagnostic hallmarks of both, and diagnostic confusion is common, with ASD originally conceptualized as a form of childhood schizophrenia (Wolff, 2004). Social dysfunction is a core symptom of both ASD and SZ, evident in decreased social motivation, reduced social reward, impaired mentalizing, and decreased likelihood of social engagement (Dawson et al., 2005; Dowd & Barch, 2010; Schultz, 2005). Moreover, parallel lines of research indicate commonalities in behavior, genetic pathways, and neural processes, suggesting shared neuropathology (Cristino et al., 2014; Fernandes et al., 2018; Mitchell, 2011; Trevisan et al., 2020; Zheng et al., 2018). These findings are consistent with the hypothesis that common mechanisms contribute to social communication deficits across behaviorally defined categories of ND.

Despite these commonalities, few studies have directly compared distinct diagnostic groups or conceptualized these diagnostic classes as heterogeneous manifestations of shared dysfunction in overlapping neural substrates. For this reason, little is known about whether atypical social perception across NDs reflects common or distinct neural underpinnings. In this review, we focus on literature related to face and emotion processing across ASD and SZ, highlighting what is known, where the disorders converge and diverge, and what work remains to be done to understand social processes related to perception of facial communication across disorders.

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Decreased attention to human faces is evident throughout the lifespan in ASD (Dawson et al., 2005; Maestro et al., 2002; Osterling & Dawson, 1994). Though SZ is not usually diagnosed until adolescence or early adulthood, infants later diagnosed with psychosis also show decreased attention to faces (Massie, 1978). Eye-tracking studies of perception of facial stimuli demonstrate atypical face scanning in individuals with ASD and SZ, with both clinical groups spending less time fixating on the eyes and more time fixating on other parts of the face (ASD: Joseph & Tanaka, 2003; Klin et al., 2002; Pelphrey et al., 2002; SZ: Gordon et al., 1992; Phillips & David, 1997). Individuals with ASD and SZ also show deficits in accurately identifying emotions, sorting emotional faces, and matching faces based on emotion (ASD: Baron-Cohen et al., 2001; Hobson, 1986; Ami Klin et al., 1999; Law Smith et al., 2010; SZ: Bellack et al., 1996; Morrison et al., 1988; Mueser et al., 1996). Atypical sensitivity to emotional expressions is a stable feature of multiple NDs and has been shown to associate with chronicity, severity, and type of active symptomatology (Addington & Addington, 1998; Davis & Gibson, 2000; Gaebel & Wolwer, 1992; Kington et al., 2000; Lewis & Garver, 1995). Difficulties in emotion recognition and discrimination in SZ and ASD are associated with specific disruption in social process systems, such as increased social withdrawal and impaired social cognition.

Face and emotion processing rely upon a network of specialized brain regions. Neuroimaging research has clarified the role of specific brain areas, including the fusiform gyrus, superior temporal sulcus, and amygdala, in face perception (Kanwisher, 2001; Kesler-West et al., 2001). Event-related potential (ERP) studies reveal a specific time course for related but distinct stages of face processing in this network (Luo et al., 2009; Wong et al., 2009). This sequence of ERPs is distinguished by scalp topography and chronology and shows sensitivity across perceptual processes, from detecting faces to interpreting differences in facial identity, direction of gaze, and displays of emotion (Bentin et al., 1996; Conty et al., 2007; Eimer & Holmes, 2002; Gosling & Eimer, 2011). These ERPs provide temporally precise indices of function across the regions involved in face perception, or, as conceptualized in the NIMH's Research Domain Criteria (RDoC) framework, the subdomain *reception of facial communication* within the broader category of *social process systems* (Kozak & Cuthbert, 2016). Prior research has demonstrated the relevance of a subset of these components across neurological and psychiatric disorders (Feuerriegel et al., 2015). Here, we focus on their manifestation in ASD and SZ, reviewing a broad, temporally sequential set of ERP components associated with face processing: P100, N170, N250, and P300.

Measured over the occipital cortex as a positive deflection approximately 100 ms after the onset of a visual stimulus, the P100 reflects low-level processing in the visual cortex (V1) that is modulated by age and may be affected by the social nature of stimuli (Key et al., 2005; Mangun, 1995). In typical development (TD), faces elicit a larger (Herrmann et al., 2005; Itier & Taylor, 2004c) and faster (Kuefner et al., 2009; Taylor et al., 2001) P100 than objects, and in a number of studies, inverted faces elicit a larger (Roxane J Itier & Taylor, 2004a, 2004c) but slower (Itier & Taylor, 2004c; Taylor et al., 2001) P100 than upright faces. P100 amplitude can be modulated by emotion (Batty & Taylor, 2003).

The N170 is a negative-going component, recorded over the occipito-temporal scalp approximately 170 ms after viewing a face, which indexes the earliest stages of face processing (i.e., structural encoding). Neural generators of the N170 have been localized to occipito-temporal sites, including the fusiform gyrus (Itier & Taylor, 2002, 2004b. Rossion et al., 2003; Sadeh et al., 2010), superior temporal sulcus (Itier & Taylor, 2004d; Yovel et al., 2008), lingual gyrus (Shibata et al., 2002), posterior inferotemporal gyrus (Schweinberger et al., 2002; Shibata et al., 2002), and inferior occipital gyrus (Jacques et al., 2018). Like the P100, in TD the N170 exhibits enhanced amplitude (more negative) and faster latency to faces relative to objects (Bentin et al., 1996; Itier & Taylor, 2004c) and larger amplitude but slower latency to inverted than to upright faces (Eimer, 2000; Itier & Taylor, 2004b, 2004c, 2004d; Rossion et al., 2000). For the N170, this "face inversion effect" is not found for upright vs. inverted objects, consistent with the interpretation that the N170 indexes face-selective processing (Eimer, 2000; Itier & Taylor, 2004c). N170 latency tends to be faster (Blau et al., 2007) with greater amplitude (Itier & Taylor, 2004b, 2004c; Rossion et al., 2003) over the right hemisphere than the left. Finally, N170 also may reflect an early indicator of emotion processing. Happy faces elicit faster N170 latencies than faces displaying negative affect (Batty & Taylor, 2003), and fearful faces elicit more negative N170 amplitude than neutral (Batty & Taylor, 2003; Blau et al., 2007) and happy (Brenner et al., 2014) faces.

The N250 is a negative-going component occurring approximately 250 ms post-stimulus (Schweinberger et al., 2002). Though less well characterized than the P100 and N170 components and rarely studied in typically developing populations, the N250 demonstrates increased amplitude in response to emotional expressiveness relative to neutral faces (Balconi & Pozzoli, 2008; Carretié et al., 2001; Labuschagne et al., 2010; Sato et al., 2001; Streit et al., 2001). As such, the N250 is considered a marker of higher-order face processing, such as affect decoding and emotion processing, and is presumed to reflect the modulatory influence of subcortical structures, including the amygdala (Streit et al., 1999). Research on face learning and repetition suggests the N250 is modulated

by the identity of a face; familiar or repeated faces evoke a more negative N250 than unfamiliar or novel faces (Gosling & Eimer, 2011; Kaufmann et al., 2009; Tanaka et al., 2006). N250 latency delays to inverted faces have been observed in a face repetition task (Itier & Taylor, 2004b), and, as with the N170, the N250 has been shown to be right lateralized (Kaufmann et al., 2009). There is variation in published studies in terms of scalp electrodes selected to capture the N250 component. For the purposes of the current review, we included all topographies, permitting that the designated component was the N250 and in the context of experimental investigation of facial emotion perception.

The P300 is a large-amplitude positive component measured at central, frontal, and occipital locations approximately 300 ms after stimulus onset. It is associated with detecting novel and significant events (Ferrari et al., 2010; Picton, 1995; Picton, 1992; Polich et al., 1995), as well as with context updating (Donchin, 2008; Donchin & Coles, 1988, 1998). In the context of face perception, the P300 reflects processing of self-relevant stimuli, such as one's own face (Ninomiya et al., 1998; Onitsuka et al., 2001). Emotional stimuli elicit a greater P300 amplitude than neutral stimuli (Johnston et al., 1986), and salient or attended stimuli elicit a greater P300 response than passively viewed stimuli (Carretié et al., 1997). P300 amplitude is larger to upright than to inverted faces and may be modified by emotional valence (Kestenbaum & Nelson, 1992), though the evidence is mixed (Conroy & Polich, 2007). As with the N250, there has been variance across studies in scalp electrodes selected to capture the P300; as above, we included all studies referencing a P300 in the context of experimental investigation of emotional face perception.

In this review, we highlight findings related to the amplitude and latency of these four ERP components, as elicited in response to face and emotion stimuli in individuals with ASD and SZ. This work expands on prior reviews focused solely on the N170 in ASD (Kang et al., 2018), a review on all four components in SZ (Murashko & Shmukler, 2019), and other disorders (Feuerriegel et al., 2015) by reviewing points of convergence and divergence for additional neural components associated with face processing transdiagnotically.
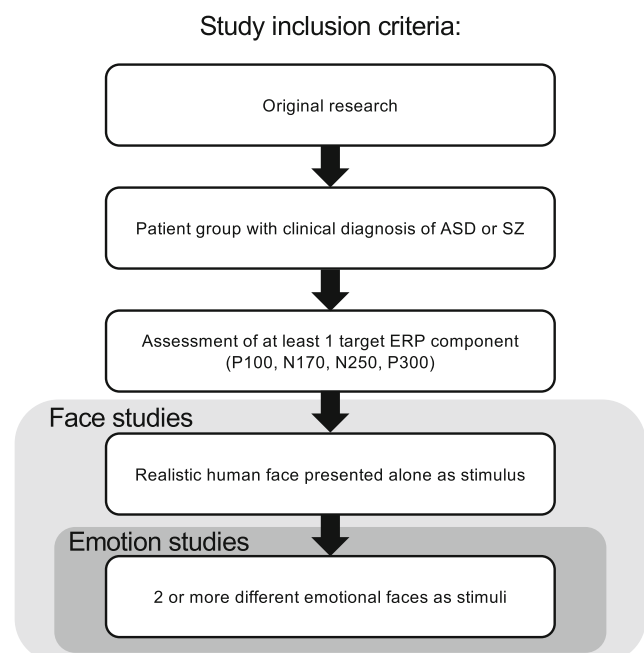
Methods

Search Criteria

Scopus, Google Scholar, and PubMed were queried using the following search criteria: (ASD OR autis* OR "pervasive developmental disorder" OR PDD-NOS OR Asperger* OR SZ OR schizo*) AND (ERP OR EEG OR electrophys* OR event-related potential OR P100 OR N170 OR N250 OR P300) AND (face OR facial OR emotion*). Reference lists, review articles, and meta-analyses were scanned to identify additional peer-review articles. Searches reflect updated content through December 31, 2020.

Inclusion Criteria: Methods and Components

These searches yielded 74 eligible studies (38 ASD, 36 SZ); all included articles were peer-reviewed. Eligible studies were required to investigate at least one of the four key ERP components most commonly investigated in neuroscientific studies of face processing in typical and atypical development: P100, N170, N250, and P300. Studies that did not include at least one of these components were excluded (e.g., McMahon & Henderson, 2015). Included studies were required to use images of human faces as stimuli. Studies in which more than one face or object was simultaneously presented were excluded (e.g., Maher et al., 2016), as were studies in which all visual stimuli were combined with auditory stimuli (Müller et al., 2012). Studies that compared ERPs to faces with two or more emotions were designated as "emotion" studies. "Face" studies were categorized as either (1) studies that included only non-emotionally valenced faces or (2) emotion studies that collapsed across different emotions to examine main effects of diagnostic group. As a result, any particular study could be both a face and an emotion study, just a face study, or just an emotion study. Finally, studies whose results were not possible to interpret in our framework were excluded (e.g., Yang et al., 2017). See Fig. 1 for a flowchart of our inclusion criteria.

Study inclusion criteria:

Original research

↓

Patient group with clinical diagnosis of ASD or SZ

↓

Assessment of at least 1 target ERP component (P100, N170, N250, P300)

↓

Face studies

Realistic human face presented alone as stimulus

↓

Emotion studies

2 or more different emotional faces as stimulus

Fig. 1 Flowchart of study inclusion criteria. Studies that fell within the lighter gray shading were included as face studies, and studies that fell within the darker gray shading were included as emotion studies only

Inclusion Criteria: Subjects

Details about population demographics, subject characterization, study methodology, and stimuli are presented in Table 1. Studies were required to examine individuals diagnosed with either ASD or SZ, and those studying only high-risk individuals or relatives were excluded (Wolwer et al., 2012). No studies included both diagnostic groups. Studies were required to report their method for confirming patients' diagnostic status. In over half of the studies of ASD (21 of 38), diagnosis was confirmed with a gold-standard clinical assessment, the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2012). For the remaining ASD studies, participants entered the study with a prior diagnosis according to DSM-IV or DSM-5 criteria (American Psychiatric Association, 1994; Association, 2013). The DSM-5 diagnosis of ASD removed subtypes, reconfigured the DSM-IV triad of impairments to a dyad, and added atypical sensory responses to the diagnostic criteria; though not identical, both sets of criteria were deemed appropriate for this review in that they both focus on social communication (American Psychiatric Association, 1994). Similarly, the DSM-5 diagnosis of SZ removed subtypes (e.g., paranoid) but was otherwise very similar to the DSM-IV diagnosis (American Psychiatric Association, 1994). In 31 of 36 SZ studies, clinicians confirmed diagnosis based on DSM-IV or ICD-10 criteria. The studies that did not specify these criteria for schizophrenia completed a Schedule for Affective Disorders and Schizophrenia (Spitzer & Endicott, 1979) or the combination of the Structured Clinical Interview and Rating Criteria (SCID) and the Positive and Negative Syndrome Scale (PANSS) (Kay et al., 1991). Study samples targeting individuals under three years of age were excluded, given the developmental emergence of some of the ERP components at this point (Itier & Taylor, 2004b). Studies were otherwise included regardless of target age group; however, all SZ studies targeted adults or late adolescents, whereas two-thirds of ASD studies exclusively included children. Studies that included both a child and an adult group analyzed separately were reported on as two separate studies in the "Results" section. Studies without a typically developing (TD) comparison group were excluded. For studies that examined additional clinical groups besides ASD or SZ (Tye et al., 2014; Tye et al., 2013), we focused only on results regarding ASD and SZ.

Results

Details on all study results described below can be found in Table 2. A graphical overview of findings can be found in Fig. 2.

P100 Amplitude

P100 Amplitude in ASD: Face

The majority of ERP literature reported no significant differences in P100 amplitude to faces between individuals with ASD and TD. Twenty-four studies of face processing in ASD reported on P100, 17 of which reported comparable P100 amplitudes in ASD and TD. In these 17 studies, no significant group differences in P100 amplitude to faces were identified across a range of task demands (attending to non-social target images to actively attending to facial stimuli) and in both child and adult samples (Akechi et al., 2010; Apicella et al., 2013; Luyster et al., 2017 [child and adult]; McPartland et al., 2011; Neuhaus et al., 2016; O'Connor et al., 2005 [child and adult]; 2007; Senju et al., 2005; Shen et al., 2017; Sysoeva et al., 2018; Tye et al., 2013; Wagner et al., 2013; Webb et al., 2010; Wong et al., 2008; Yamasaki et al., 2017). Studies reporting group differences (7 of 24) in P100 amplitude to faces revealed inconsistent directionality of effects. One study of adults found increased P100 amplitude to faces in ASD versus TD (Cygan et al., 2014). One child study and one adult study reported inversion effects (greater P100 amplitude to inverted versus upright faces) in the TD group, but not in the ASD group (Hileman et al., 2011; Webb et al., 2012). Poorer face memory in the study of Webb et al. (2012) was correlated with greater P100 response to inverted versus upright faces in adults with ASD; however, there were no significant differences between groups in processing upright faces or objects. One study of children reported attenuated P100 amplitude to faces in ASD compared to TD, but lacked control stimuli (Batty et al., 2011), and a second study replicated this effect in young adults when using photographs of emotional faces, but reported a slightly larger P100 amplitude in ASD when using realistic drawings of emotional faces (Tseng et al., 2015). An additional study of children reported reduced P100 amplitude in ASD for repeated versus novel faces and objects whereas no difference was found in TD (Key & Corbett, 2014). A final study found reduced P100 lateralization in the ASD group as compared to controls (Luckhardt et al., 2017). Of these seven studies, three found effects across face and objects, and four did not include non-face stimuli. As such, no studies found a face-stimulus specific effect.

P100 Amplitude in ASD: Emotion

There were 13 studies of emotion processing in ASD that reported on P100 amplitude, distributed across childhood, adolescence, and adulthood. Across all studies, P100 was modulated similarly between diagnostic groups in response to emotional faces (Akechi et al., 2010; Apicella et al., 2013; Batty et al., 2011; Hileman et al., 2011; Luckhardt et al., 2017; Luyster et al., 2017 [child and adult]; O'Connor et al.,

Table 1 Participant characterization and ERP methods for all studies reviewed

Author, year	Task	Stimuli	ASD/SZ group: mean age (years), range, SD, n	Characterization and behavioral measures	Control group matched by	Medications
ASD: face processing						
Churches et al., 2012a	Active: find target	N faces. Target: face identity	31.8; SD = 6.9; n = 11	Clinician diagnosis, IQ test	Age, IQ	–
Churches et al., 2010	Active: find target	N faces; chairs. Target: repeated stimuli	31.4; SD = 6.7; n = 13	Clinician diagnosis, WAIS-III	Age, IQ	–
Churches et al., 2012b	Active: find target	N faces; objects; face-like objects. Target: flowers	30.6; SD = 6.2; n = 10	Clinician diagnosis, IQ test	Age, IQ	–
Cygan et al., 2014	Active: respond to all stimuli	Own/close other's/celebrity/ unfamiliar faces. Own/close other's/celebrity/unfamiliar names	20.4; r = 17–27; SD = 2.8; n = 23	ADOS, ADI-R, WAIS-R	Age, IQ, handedness	–
Grice et al., 2005	Passive	N faces with direct or averted gaze	5.08; r = 42–87; SD = 1.0; n = 10	Clinician diagnosis, CARS; ADOS and ADI-R if needed	Age, sex	–
Groom et al., 2017	Passive	Face w/ left or right gaze shift (center gazing image followed by left or right gazing image)	12.52; SD = 2.36; r = 8–15; n = 10	DAWBA, DSM-IV, ADOS, SCQ, WASI,	Age, IQ, sex	No non-stimulant medication
Gunji et al., 2009	Passive	Familiar, unfamiliar, self, scrambled, object	10.8; SD = 2.9; n = 8	DSM-IV, WISC	Age, sex, handedness	–
Gunji et al., 2013	Active: find target	Own/mother's/unfamiliar faces; objects. Block 1 target: own face. Block 2 target: mother's face. Block 3 target: unfamiliar face. Block 4 target: object	8.0; SD = 1.2; n = 9	Clinician diagnosis, WISC-III	Age, sex, handedness, IQ	–
Jones et al., 2018	Passive	Time 1: mother's face, stranger's face. Time 2: face, toy	Age 2: r = 1.5–2.5, n = 18; age 4: r = 4–6.5, n = 27 (only age 4 results included)	ADOS, ADI-R, Mullen, DSM-IV	Age, sex	None
Key & Corbett, 2014	Active: find target	Test 1: N faces (1 repeating, 50 novel); houses (1 repeating, 50 novel). Target: cartoon faceRe-test: 3 weeks later	10.76; SD = 1.5; n = 13	ADOS, WASI, SCQ	Age	None
Khorrami et al., 2013	Passive	Upright/inverted faces; upright/inverted cars	14.8; r = 9–17; SD = 3.1; n = 14	Clinician diagnosis, CARS, ASDS, Raven Progressive Matrices	Age, sex	No antipsychotic meds
McPartland et al., 2011	Active: find target	Upright/inverted faces; upright/ inverted houses; letters/ pseudoletters. Target: repeated stimuli	11.2; SD = 3.4; n = 32	ADOS, ADI-R, WASI, Benton	Age, sex, IQ, handedness	–
McPartland et al., 2004	Active: find target	Upright/inverted faces; upright/ inverted furniture. Target: butterflies	21.2; r = 15–42; SD = 8.3; n = 9	Clinician diagnosis, ADI-R, IQ test, faces subtest of Wechsler Memory Scale	Age, sex, IQ, handedness, ethnicity	–
Neuhaus et al., 2016	Active: find target	Upright and inverted faces and houses	11.3; SD = 4.4; n = 28 for P1; n = 25 for N170	ADOS, ADI-R, WASI (DAS if < 6)	Age, all twins/triplets	No medications known to affect EEG
Senju et al., 2005	Active: find target	N faces with direct/down/ laterally averted gaze. Block 1 target: direct gaze. Block 2 target: averted gaze	12.1; r = 9.9–14.1; n = 13	Clinician diagnosis, NVIQ	Age	–
	Active: identify target		6.93; SD = 1.11; n = 12	DSM-IV, BRSA	Age, sex, IQ	–

Table 1 (continued)

Author, year	Task	Stimuli	ASD/SZ group: mean age (years), range, SD, n	Characterization and behavioral measures	Control group matched by	Medications
Shen et al., 2017		Block 1: N faces, cars. Block 2: mother's face, only eyes of N and mother, only mouth of N and mother, favorite toy, unfamiliar toy, target butterfly				
Sysoeva et al., 2018	Active: find target	Upright/inverted faces; upright/inverted houses. Target: scrambled faces	15.2; SD = 2.7; r = 12–21; n = 59	ADI-R, SRS, DSM-IV	Age, sex, handedness	-
Tye et al., 2013	Active: count target	Upright/inverted faces with direct/indirect gaze. Target: flags in fixation stimuli	ASD: 11.7; r = 8–13; SD = 1.7; n = 19. ASD + ADHD: 10.5; r = 8–13; SD = 1.7; n = 29	ADOS, ADI-R, SCQ, WASI	Age, sex	None
Webb et al., 2006	Passive	Mother's/unfamiliar faces; familiar/unfamiliar objects	3.8; r = 2.8–4.5; SD = 0.3; n = 27	ADOS, ADI-R, Mullen	Age	–
Webb et al., 2010	Active: find target	Familiar/repeating-unfamiliar/ novel-unfamiliar faces. Target: houses	22.4; r = 18–44; SD = 6.1; n = 29	ADOS, ADI-R, Wechsler IQ test, Wechsler Memory Scale, Faces subtest	Age	No anti-convulsants, barbiturates
Webb et al., 2012	Active: find target	Upright/inverted faces; upright/inverted houses. Target: scrambled faces	23.1; r = 18–44; SD = 6.9; n = 32	ADOS, ADI-R, Wechsler IQ test, Benton, AQ, SADS, SCQ, Wechsler Memory Scale: Faces subtest, Woodcock Johnson: object recognition, House memory test	Age, IQ	No anti-convulsants, barbiturates
ASD: emotion processing						
Akechi et al., 2010	Active: identify emotion and find target	F/A faces with averted or direct gaze. Target: asterisk	13.7; r = 10.2–17.8; SD = 2.3; n = 14	WISC-III, clinical diagnosis, ASD	Age, sex, IQ	–
Apicella et al., 2013	Active: find target	H/F/N faces; trees. Target: cartoons	10.2; r = 6–13; n = 10	ADOS-G and ADI-R	–	–
Batty et al., 2011	Active: find target	H/F/A/S/D/R/N faces. Target: cars/ butterflies/planes	10.5; r = 5.25–16.5; SD = 3.31; n = 15	Clinician diagnosis, CARS, VIQ, NVIQ	Age, VIQ	–
Faja et al., 2016	Active: find target	H/F/N faces	23.3; SD = 7.7; r = 18–44; n = 27	ADOS, DSM-IV, ADI-R, WAIS-III, Woodcock Johnson	Age, IQ, sex	No anti-convulsants, barbiturates
Fujita et al., 2013	Passive	F/N upright/ inverted faces, upright/ inverted objects	n = 31.5; r = 23–39; 10	DSM-IV, PARS, WAIS	Age, sex	2 ASD on antidepressants, stable
Hileman et al., 2011	Active: find targets	H/F/A/N upright/inverted faces; upright/inverted cars. Target: female faces; cars facing left	13.3; r = 9.4–17.4; SD = 2.8; n = 27	ADOS, WISC-IV, SCQ, RMET (children's), strange stories (mentalizing)	Age, sex, IQ	–
Luckhardt et al., 2017	Active: identify blonde hair (implicit) or angry face (explicit)	D/S/A faces	12.5; SD = 2.2; n = 21	ICD-10 (Kinder-DIPS), ADI-R, ADOS	Age, sex, handedness, IQ	No psychotropic meds
Luckhardt et al., 2018	Active: part 1 = identify hair color; part 2 = identify if emotion is anger	A/S/D faces	12.7; r = 8–20; SD = 3; n = 33	ICD-10 diagnosis, ADOS, ADI-R,	Age, IQ, sex, handedness	No medication changes

Table 1 (continued)

Author, year	Task	Stimuli	ASD/SZ group: mean age (years), range, SD, n	Characterization and behavioral measures	Control group matched by	Medications
Luyster et al., 2017	Active, sort by emotion	N/H/F/A faces	Group 1: 12.44, SD = .25, n = 17 12 y/o; group 2: 21.1, SD = 1.24, n = 12 18–22 y/o	Kinder-DIPS, WISC-IV/WAIS-III ADOS, IQ	Age, IQ, sex	–
Magnee et al., 2011	Active: find target	Block 1: H/F faces. Target: white dotBlock 2: H/F auditory stimuli. Target: tone. Block 3: H/F faces with H/F auditory stimuli. Target: white dot/tone	22.7; SD = 3.8; n = 23	ADOS, ADI-R, WAIS-III	Age, IQ	–
O'Connor et al., 2007	Active: find target	Block 1: S/N eyes/mouths. Target: N faces. Block 2: S/N faces; objects. Target: N faces	23.5; r = 18–41; SD = 5.2; n = 15	Clinician diagnosis	Age	3 ASD on SRIs
O'Connor et al., 2005	Active: identify emotion	H/F/A/S/N faces	Adult: 24.6; r = 18–45; SD = 8.8; n = 15. Child: 11.6, r = 11–15; SD = 1.9; n = 15	Clinician diagnosis	Age, sex	4 ASD adults, 7 ASD children on SRIs
Tseng et al., 2015	Active: rate emotionality of face	A/N/H photo and drawn face	19.6; SD = 1.96, n = 10	Gillberg, DSM-IV, ICD-10, WAIS-III, WASI	Age, sex, IQ	2 ASD on therapy medication
Tye et al., 2014	Active: find target	H/F/A/D/N faces. Target: blue circle around stimulus, delayed onset	11.7; r = 8–13; SD = 1.7; n = 19	ADI-R, ADOS, Connor's, SCQ, PACS, WASI	Age, IQ, handedness	No meds, stimulants interrupted 48 h prior
Wagner et al., 2013	Active: find target	F/A/N faces; houses. Target: repeated stimuli	17.0; r = 13.6–21.1; SD = 2.2; n = 18	Clinician diagnosis, ADOS, SCQ, IQ test (Kaufman brief IQ test)	Age	11 ASD on stimulants, SRIs, antipsychotics
Wong et al., 2008	Active: identify gender, emotion	H/F/A/S/N faces	8.5; r = 6–10; SD = 1.5; n = 10	ADOS, ADI-R, Raven's Progressive Matrices	Age, NVIQ	–
Yamasaki et al., 2017	Passive	N/H/A faces	29.9; SD = 7.1; n = 14	DSM-IV, clinical Dx	Age, sex, IQ	–
SZ: face processing						
Herrmann et al., 2004	Active: identify stimulus type	N faces; buildings	32.3; r = 18–57; SD = 10; n = 24	PANSS, DSM-IV diagnosis	Age, handedness, education	17 SZ on antipsychotics
Liu et al., 2016	Active: press button when monkey face appears	N human and monkey faces, some presented concurrently with sound	44.6; SD = 7.94, r = 18–55; n = 18	DSM-IV, SCID, PANSS, Verbal IQ	Age, handedness, parental SES, normal hearing	–
Onitsuka et al., 2006	Active: find target	N faces; hands; cars. Target: butterflies	42.7; r = 20–55; SD = 10; n = 20	DSM-IV diagnosis, PANSS, VIQ	Age, sex, handedness	All SZ on antipsychotics
Onitsuka et al., 2009	Active: find target	N faces. Target: N female face	42.4; r = 20–55; SD = 10.8; n = 17	DSM-IV diagnosis, PANSS, WAIS-R	Age, sex, handedness, parent SES	All SZ on antipsychotics
Salisbury et al., 2019	Active: find target	N faces, cars. Target: butterflies	Chronic: 38.8; SD = 10.0; n = 32. Acute: 24.1; SD = 6.8; n = 31	SCID, PANSS, SAPS, SANS	Age, sex, handedness, IQ, SES	Chronic: 30 on antipsychotics;Acute: 16 on antipsychotics
Tsunoda et al., 2012	Active: find target	Upright/inverted faces; upright/inverted cars. Target: butterflies	30.4; r = 25–35; SD = 3.2; n = 15	DSM-IV diagnosis, SAPS, SANS, SFS	Age, sex, handedness, SES, parent SES	1 typical, 14 atypical psychotics (all SZ)

Table 1 (continued)

Author, year	Task	Stimuli	ASD/SZ group: mean age (years), range, SD, n	Characterization and behavioral measures	Control group matched by	Medications
Zheng et al., 2016	Mental count of flowers and ignore other stims	Upright faces, upright tables, inverted faces, interverted tables. Target: flowers	32.3, SD = 11.2 n = 24	DSM-IV, PNSS, Personal and Social Performance w/ psychiatrist or psychologist	Age	–
SZ: emotion processing						
Akbarfahimi et al., 2013	Active: find target	H/F/A/S/N faces. Target: houses	33.3; r = 20–45; SD = 5.7; n = 28	PANSS, SADS with psychiatrist and case files	Age, handedness, education	–
Bediou et al., 2007	Active: count R faces, gender	H/F/D/R/N faces	27.8; SD = 8.4; n = 10	DSM-IV diagnosis, MINI, PANSS	Age	Chloropromazine equivalent 346 mg
Brenner et al., 2015	Active: match emotion	H/VH/F/A/S/N faces	31; SD = 8.65; n = 38	SCID, PANSS	Age, sex	Chloropromazine equivalent 327 mg
Caharel et al., 2007	Active: identify familiarity	H/D/N faces of self/familiar/unfamiliar faces	37.7; r = 25–56; SD = 8.3; n = 18	DSM-IV diagnosis	Age, handedness	All SZ on antipsychotics
Campanella et al., 2006	Active: find target	Block 1: N faces same identity. Target: N face deviant identity. Block 2: H/F/S/N faces. Target: H/F/S faces	47.7; SD = 11.9; n = 14	DSM-IV diagnosis, PANSS	Age, handedness, Beck Depression Scale	Haloperidol equivalent 9 mg
Fukuta et al., 2014	Passive	N, pos, neg, low emo (dynamic faces NR here)	33.3; SD = 6.2; n = 13	DSM-IV diagnosis, PANSS	Age, sex, handedness	Risperidone equivalent 7.8 mg
Ibanez et al., 2012	Active: identify emotion	H/A faces; positive/negative words	38.6; SD = 12.0; n = 13	DSM-IV Diagnosis, PANSS, SCAN, Raven's Test, Trail Making Test - Part A	Age, sex, education, intellectual capabilities/processing speed	All SZ on antipsychotics
Jetha et al., 2013	Passive	H/F/A/N faces	42.2; SD = 6.4; n = 40	PANSS, DSM-IV diagnosis, Cheek and Buss Shyness Scale	Age, sex	39/40 SZ on antipsychotics
Johnston et al., 2005	Active: find target	H/F/A/S/D/R/N faces. Target: R faces, male/female	33.4; r = 19–44; SD = 8.3; n = 11	ICD-10 diagnosis, Emotion recognition behavioral task	–	All SZ on antipsychotics
Jung et al., 2012	Active: find target	H/F/N faces. Target: H/F faces	32.2; SD = 10.1; n = 23	PANSS, DSM-IV diagnosis	Age, sex, handedness, education	All SZ on atypical antipsychotics
Kim et al., 2015	Active: find target	Block 1: F/N faces, broad spatial freq (BSF). Block 2: F/N faces, low spatial freq (LSF). Block 3: F/N faces, high spatial freq (HSF). Target: chairs	37.6; SD = 11.4; n = 21	SCID, PANSS	Age, sex, education	All SZ on atypical antipsychotics
Kirihara et al., 2012	Active: find target	H/F/A/S/D/R/N faces; cars. Target: butterflies	34.5; SD = 6.8; n = 15	DSM-IV diagnosis (SCID), PANSS, National Adult Reading Test, NEO-Five Factor Inventory, Global Assessment of Functioning (GFA)	Age, handedness	All SZ on antipsychotics
Komlosi et al., 2013	Active: identify emotion	F/N faces; scrambled faces	34.2; SD = 10.3; n = 24	DSM-IV diagnosis, PANSS, Symptom Checklist-90	Age, sex, education	All SZ on antipsychotics
Lee et al., 2007	Active: find target	H/F/N faces. Target: H/F faces	30.2; SD = 10.3; n = 11	DSM-IV diagnosis, PANSS	Age, sex, handedness, education	All SZ on atypical antipsychotics

Table 1 (continued)

Author, year	Task	Stimuli	ASD/SZ group: mean age (years), range, SD, n	Characterization and behavioral measures	Control group matched by	Medications
Lee et al., 2010	Active: find target	H/F/N faces. Target: H/F faces	30.2; SD = 10.3; n = 38	DSM-IV diagnosis, PANSS	Age, sex, handedness, education	All SZ on atypical antipsychotics
Lynn & Salisbury, 2008	Active: find target	Block 1: N faces; cars. Target: butterflies. Block 2: H/F/A/S/D/N faces. Target: N faces	35.5; SD = 9.4; n = 24	DSM-IV diagnosis, PANSS, VIQ, WAIS-II, SAPS, SANS	Age, sex, IQ, handedness, education, parent SES	Chlorpromazine equivalent 409
Mori et al., 2012	Active: identify and count emotion	Block 1: H/N infant faces. Block 2: S/N infant faces. Target: H/S infant faces. Session 1 (S1): med naïve; S2: 3 mo post-med; S3: 12 mo post-med	27.1; SD = 8.9; n = 30	ICD-10 diagnosis	Age, sex	None at S1
Obayashi et al., 2009	Active: find target	H/F/N faces; blurred faces; houses. Target: shoe	32.9; r = 18–47; SD = 10.0; n = 16	DSM-IV diagnosis, SAPS, SANS, GAF	Age, handedness, parent SES	All SZ on antipsychotics
Onitsuka et al., 2020	Active: find target	N/S/H/F faces	44.9; r = 20–55; SD = 9.2; n = 20	DSM-IV diagnosis, PANSS, VIQ	Age, sex, handedness	All SZ on antipsychotics
Ramos-Loyo et al., 2009	Active: find target	H/F/A/S/D/R/N faces. Block 1 target: H faces. Block 2 target: F faces. Block 3 target: face identity	30.2; r = 18–45; SD = 7.3; n = 9	DSM-IV diagnosis, PANSS	Age, sex, handedness, education	All SZ on typical antipsychotics
Shah et al., 2018	Active: identify emotion	H/A/F/S/N faces, chairs	46.39; r = 18–60; SD = 12.39; n = 23	DSM-IV, SCID, GAF	Age, sex, education	All SZ on antipsychotics
Thoma et al., 2013	Active: find target	H/F/S/N face/body pairs. Target: same emotion in both stimuli	34.1; r = 21–59; SD = 11.2; n = 14	ICD-10 diagnosis, Beck Depression Inventory, vocabulary test	Age, education	All SZ on antipsychotics
Tso et al., 2015	Active: identify gaze direction	F/N faces with direct/rotated orientation and direct/averted gaze	41.4; r = 18–60; SD = 13.3; n = 28	DSM-IV diagnosis, SAPS, SANS, reading ability (subtest of WRAT3-R), cognition (BACS)	Age, sex, handedness, parent education	Most medicated
Turetsky et al., 2007	Active: identify emotion	H/S/N faces	30.5; r = 22–38; SD = 6.0; n = 16	DSM-IV diagnosis, DIGS, BPRS, SAPS, SANS, Hamilton Depression Rating Scale	Age, sex, handedness	10/16 SZ on atypical antipsychotics; 6 unmedicated.
Ueno et al., 2004	Active: find target	Block 1: H infant faces; flowers. Target: H faces. Block 2: S infant faces; flowers. Target: S faces. Block 3: N infant faces; flowers. Target: N faces	26.5; r = 16–34, SD = 4.7; n = 32	DSM-IV diagnosis, PANSS	Age	All SZ on antipsychotics
Wang et al., 2020	Active: identify emotion	H/S/N faces	34.0; SD = 7.2; n = 20	DSM-IV diagnosis, PANSS	Age	18/20 SZ on antipsychotics
Wynn et al., 2008	Active: identify gender, emotion, object	H/F/A/S/R/M faces; buildings. Block 1 target: identify gender. Block 2 target: identify emotion.	43.9; SD = 10.2; n = 26	DSM-IV diagnosis, SANS, BPRS	Age, sex, education	All SZ on antipsychotics

Table 1 (continued)

Author, year	Task	Stimuli	ASD/SZ group: mean age (years), range, SD, n	Characterization and behavioral measures	Control group matched by	Medications
Wynn et al., 2013	Active: identify gender, emotion, object	Block 3 target: count stories of building; H/F/A/S/R/M faces; buildings. Block 1 target: identify gender. Block 2 target: identify emotion. Block 3 target: count stories of building	45.3; SD = 9.4; n = 32	DSM-IV diagnosis, PBRS	Age, sex, parent education	30/32 SZ on antipsychotics
Zhang et al., 2016	Active: identify duration	H/F/N faces. Target: H/F/N faces with altered duration	31.3; r = 18–42; n = 47	SCID, PANSS	Age, sex, handedness, IQ, education	All SZ on antipsychotics

Notes: "Targets" were not included in the analyses unless repeated in cell. We use the term sex instead of gender because sex was used in the original studies

A angry, ADHD attention deficit-hyperactivity disorder group, amp amplitude, ASD autism spectrum disorder group, D disgusted, emo emotion, F fear, fo face-only effect (study does not include non-face stimuli), H happy, lat latency, M ashamed, N neutral, neg negative, nfs not face-specific effect (effect across face and non-face stimuli), obj object, pos positive, R surprised, S sad, SZ schizophrenia group, TD typically developing group

2005 [child and adult]; 2007; Wagner et al., 2013; Wong et al., 2008; Yamasaki et al., 2017).

P100 Amplitude in SZ: Face

Most studies of face processing (14 of 18) reported comparable P100 amplitudes to face stimuli between SZ and TD groups across a variety of methodologies and sample compositions (Bediou et al., 2007; Brenner et al., 2015; Fukuta et al., 2014; Herrmann et al., 2004; Jetha et al., 2013; P. J. Johnston et al., 2005; Jung et al., 2012; Kim et al., 2015; Lee et al., 2010; Liu et al., 2016; Obayashi et al., 2009; Thoma et al., 2013; Turetsky et al., 2007; Wynn et al., 2008). Among studies reporting P100 amplitude differences, all found attenuated P100 to faces in SZ groups (Caharel et al., 2007; Campanella et al., 2006; Shah et al., 2018; Zhang et al., 2016), though Campanella et al. (2006) found that this effect was specific to a group displaying high SZ symptomatology (compared to TD and low-symptom SZ groups). In all 4 of the studies that found significant differences, participants were actively attending to facial stimuli; 12 of the 14 studies that reported null results also included active attention to stimuli. Importantly, only 3 of 18 studies included control (i.e., non-face) stimuli, and only one of these reported differences between SZ and TD (Shah et al., 2018) while two did not (Obayashi et al., 2009; Wynn et al., 2008).

P100 Amplitude in SZ: Emotion

Out of 16 emotion-processing studies reporting P100 amplitude in SZ, 13 studies reported no significant group differences in P100 amplitude as a function of the emotional content of presented faces (Brenner et al., 2015; Caharel et al., 2007; Campanella et al., 2006; Fukuta et al., 2014; Johnston et al., 2005; Jung et al., 2012; Kim et al., 2015; Lee et al., 2010; Obayashi et al., 2009; Thoma et al., 2013; Turetsky et al., 2007; Wynn et al., 2008; Zhang et al., 2016). One study reported amplitude modulation by emotion in the TD group but not in the SZ group (Bediou et al., 2007). Jetha et al. (2013) reported that moderate shyness in SZ was associated with less emotional reactivity as indicated by P100 amplitude. This finding suggests that differences in P100 amplitude may mark alterations in emotion processing associated with specific symptom dimensions. Finally, Shah et al. (2018) found amplitude responses in the SZ group to be smaller than those of the TD group in sad, angry, and fearful faces occipitally and sad faces parietally. Taken together, these results suggest that P100 amplitude does not specifically index alterations in face or emotion processing.

Table 2 ERP results from all reviewed studies

Author, year	Correlations	P100		N170		N250	P300		
		Amplitude	Latency	Amplitude	Latency	Amplitude	Lat	Amplitude	Latency
ASD: face processing									
Churches et al., 2012a	—	—	—	ASD > TD, fo	ns	Target face: ASD > TD, fonontarget face: ns	—	—	—
Churches et al., 2010	—	—	—	TD: active < passive, fo. ASD: ns	ns	—	—	—	—
Churches et al., 2012b	—	—	—	Face, face-like obj: nsObj: ASD > TD	ns	—	—	—	—
Cygan et al., 2014	—	Faces: ASD > TD. Names: ns	ns	All stim: LH, TD < ASD, nfs. TD: names, LH < RH; faces, ns. Fam vs. Unfam: ns	ns	—	—	—	—
Grice et al., 2005	—	—	—	ns	ns	—	—	—	—
Groom et al., 2017	—	—	—			—	—	—	—
Gunji et al., 2009	—	—	—	ASD: face < non-face	Adult TD < PDD and Child TD	—	—	TD: self > familiar; ASD: ns	—
Gunji et al., 2013	—	—	—	—	—	—	—	TD: fam > unfam, fo. ASD: ns	ns
Jones et al., 2018	P1 amplitude to faces w/ age 4 social approach (+)	-	-	-	-	-	-	-	-
Key & Corbett, 2014	N170 amp w/ parietal P3 amp (+)	All stim: TD: LH re-test, repeated > novel, nfs. ASD: LH re-test, repeated < novel, nfs	—	All stim:ASD: LH re-test, repeated < novel, nfs. TD: ns	—	—	—	All stim: Repeated: ASD > TD, nfs. Novel: ns. ASD: repeated>novel, nfs. TD: ns	—
Khorrami et al., 2013	ns	—	—	ns	All stim: ASD > TD, nfs. Face vs. Obj: ns. Up vs. Inv: TD: up obj < inv obj	—	—	—	—
McPartland et al., 2011	N170 lat and ASD amp w/ face recognition (+)	ns	ns	All stim: TD: RH < LH, nfs ASD: ns	Face vs. Obj; Face, RH; ASD > TD	—	—	—	—

Table 2 (continued)

Author, year	Correlations	P100		N170		N250		P300	
		Amplitude	Latency	Amplitude	Latency	Amplitude	Lat	Amplitude	Latency
				Face vs. obj; obj: ASD > TD Faces: ns Up vs. Inv: TD: inv < up, fo ASD: up < inv, fo	House: ns Up vs. Inv: ns				
McPartland et al., 2004	ASD N170 lat w/ face memory (-)	—		ns	Face vs. Obj: Face: ASD > TD Obj: ns Up vs. Inv: ASD > TD, fo TD: inv > up ASD: ns				
Neuhaus et al., 2016	TD and ASD lat and peak amp: older participants < younger; MZ twins highly correlated (+) on latencies and amplitudes for both P1 and N170, also for DZ except N170 amp	Houses > faces	TD < ASD; TD upright < inverted faces and houses; ASD: upright < inverted faces, inverted < upright houses; TD and ASD: older participants < younger	—					
Senju et al., 2005	—	ns	ns	ns	ns	—	—	ns	ns
Shen et al., 2017	—	Objects > faces	N faces < objects; N faces vs. objects ASD: left hemi < right	Faces and eyes < mouths and objects	ASD: LHI < RH	—			
Sysoeva et al., 2018	—	ns	ns	Up vs. Inv at vertex e-: ASD < TD (nfs)	Facs vs. Obj:TD: face < objASD: ns	—			
Tye et al., 2013	N170 amp lateralization w/ social communication (+)	ns	All stim: TD: RH > LH, fo. ASD/ASD + ADHD: LH > RH, fo. ASD: ns. Up vs. Inv: ns. Gaze:TD,ADHD: direct >	All stim: TD: LH > RH, fo. ASD + ADHD: LH < RH, fo. ASD: ns. Up vs. Inv: ns. Gaze: ns	ns				

Table 2 (continued)

Author, year	Correlations	P100 Amplitude	P100 Latency	N170 Amplitude	N170 Latency	N250 Amplitude	N250 Latency	P300 Lat	P300 Amplitude	P300 Latency
Webb et al., 2006	—	—	—	All stim: TD: RH < LH, nfsASD: ns. Face vs. Obj: TD: ns. ASD: face < obj. Fam vs. Unfam: ns	Face vs. Obj: TD: face < obj. ASD: face>obj. Fam vs. Unfam: ns	—	avert. ASD: avert > direct	—	—	—
Webb et al., 2010	N250 amp w/ VIQ, social behavior and social communication (+)	ns	ns	ns	All stim: TD: ns ASD: lat < med, fo Fam vs. Unfam: ns	All stim: ASD < TD, fo Fam vs. Unfam: ASD, fam-unfam: RH < LH, fo TD, fam-unfam: ns	—	—	—	—
Webb et al., 2012	ASD N170 amp, N170 lat, ASD P1 amp and lat w/ social anxiety, social behavior and face memory	Face vs. Object: ns. Up vs. Inv: TD: inv > up, nfs. ASD: ns	ns	Face vs. Obj: nsUp vs. Inv:Up: RH med, TD < ASD, fo. Inv: RH lat, TD < ASD, fo	All stim: TD: lat > med, nfs. ASD: nsFace vs. Obj: ns. Up vs. Inv: ns	—	—	—	—	—
ASD: emotion processing										
Akechi et al., 2010	—	ns	TD: RH < LH, fo. ASD: ns. Emo: ns	Emo/Gaze:TD: congruent > incongruent, fo. ASD: ns. Emo: ns	ns	—	—	—	—	—
Apicella et al., 2013	—	ns	ns	ns	ns	—	—	—	—	—
Batty et al., 2011	—	All stim: ASD < TD, fo. Emo: ns	All stim: ASD > TD, fo. Emo:TD: D > H,S,R ASD: ns	ns	All stim: ASD > TD, fo. Emo: ns	—	—	—	—	—
Faja et al., 2016	TD N170 influenced by emotion			ASD: F < N						
Fujita et al., 2013	—	ns	ns		ns	—		—	ns	ns
Hileman et al., 2011	TD P1 and N170 amp w/ social	Face vs. Obj: ns. Up vs. Inv: TD: inv >	ns	ns	All stim: ASD > TD,	—		—	—	—

Table 2 (continued)

Author, year	Correlations	P100		N170		N250		P300		
		Amplitude	Latency	Amplitude	Latency	Amplitude	Latency	Lat	Amplitude	Latency
	behavior and cognition (− and +, respectively)	up, fo. ASD: ns. Emo: ns				—	nfs. Face vs. Obj: ns. Up vs. Inv: ns. Emo: ns	—	—	—
Luckhardt et al., 2017	—	TD:Explicit condition > implicit; ASD: ns; TD: stronger lateralization	ns	Explicit < implicit	ns	—	ns	—	—	—
Luckhardt et al., 2018	—	ns	ns	ns	ns	—	ns	—	—	—
Luyster et al., 2017	—	12 y/o > 18–22 y/o	12 y/o > 18–22 y/o; in 18–22 group, TD < ASD	12 y/o < 18–22 y/o		—	TD < ASD	—	—	—
Magnee et al., 2011	—	—	—	ns	ns	—	ns	—	—	—
O'Connor et al., 2007	—	ns	ns	ns	ns	—	Face vs. Obj: Face: ASD > TDObj: nsEmo: ns	—	—	—
O'Connor et al., 2005	ns	ns	Adult: All stim: ASD > TD, fo Emo: ns Child: ns	Adult: All stim: ASD > TD, TD, fo Emo: ns Child: ns		—	Adult: All stim: ASD > TD, fo Emo: ns Child: ns	—	—	—
Tseng et al., 2015	ASD slower RT to evaluate A/H faces, shorter for N faces (photo and drawing)	Photo: ASD < TD; Drawing: TD < ASD	—		ns	—	ns	—	—	—
Tye et al., 2014	N170 amp emo diff w/ social communication (+)	—	—	All stim: ASD/ASD + ADHD>TD, nfsEmo: ASD/ASD + ADHD: N < FTD: ns	—	—	TD: nsASD: A > N; F > HASD + ADHD: A < N; F < H	—	—	—
Wagner et al., 2013	TD P1 lat w/ pupil diameter (-); N170 lat w/ eye gaze (+)	All stim: TD: ns. ASD: mid > RH, nfs. Face vs. Obj: Faces: ns. Obj: ASD > TD Emo: ns	ns	Face vs. Obj: ns. Emo:TD: F > A. ASD: ns	ns	—	ns	—	—	—
Wong et al., 2008	—	ns	ns	ns	ns	—	ns	—	—	—
Yamasaki et al., 2017	—	ns	ASD < TD	—	TD < ASD	—	TD < ASD	—	—	—

SZ: face processing

Table 2 (continued)

Author, year	Correlations	P100 Amplitude	P100 Latency	N170 Amplitude	N170 Latency	N250 Amplitude	N250 Lat	P300 Amplitude	P300 Latency
Herrmann et al., 2004	ns	ns	ns	Face vs. obj: Face: SZ > TD Obj: ns Face-obj diff: SZ < TD	ns	–	–	–	–
Liu et al., 2016	ns	ns	ns	TD < SZ	ns	Sound + face > face	–	–	–
Onitsuka et al., 2006	SZ N170 amp w/ L and R posterior fusiform (+)	–	–	Face vs. Obj: Face: SZ > TD. Obj: ns. TD: face<hand,obj. SZ: ns	–	–	–	–	–
Onitsuka et al., 2009	ns	–	–	SZ > TD, fo	–	–	–	–	–
Salisbury et al., 2019	Long term: ns. 1st hosp: ns	–	–	Chronic: Obj > face. Obj and face: SZ > TD. Acute: Obj > face. Obj and face: SZ > TD	–	–	–	–	–
Tsunoda et al., 2012	SZ N170 amp w/ social behavior (+)	–	–	Face vs. Obj: TD: face < obj. SZ: ns. Face: SZ > ns. TD. Obj: Up vs. Inv: TD: face, inv < up; obj, ns. SZ: ns	ns	–	–	–	–
Zheng et al., 2016	–	–	–	SZ > TD for inverted faces	SZ > TD for upright and inverted faces	–	–	–	–
SZ: emotion processing									
Akbarfahimi et al., 2013	ns	–	–	ns	All stim, LH: SZ > TD, fo Emo: F face: SZ > TD	–	–	–	–
Bediou et al., 2007	–	TD: emo modulated.SZ: ns	–	Emo task: SZ > TD, fo. Gender task: ns. Emo: ns	–	–	–	TD, emo task: F > NTD, gender task: ns SZ: ns	–
Brenner et al., 2015	ns	ns	–	ns	–	ns	–	–	–
Caharel et al., 2007	P1 lat w/ N170 lat (+)	All stim: SZ < TD, fo. Fam vs. Unfam: ns. Emo: ns	All stim: SZ > TD, fo. Fam vs. Unfam: ns. Emo: ns	Fam vs. Unfam: own < fam, unfam, fo. SZ: unfam < own, fam, fo. Emo: TD: D <	All stim: SZ > TD, fo. Fam vs. Unfam: ns. Emo: ns	ns	–	–	–

Table 2 (continued)

Author, year	Correlations	P100		N170		N250		P300	
		Amplitude	Latency	Amplitude	Latency	Amplitude	Lat	Amplitude	Latency
Campanella et al., 2006	SZ N170 amp w/ pos SZ symptom (-)	All stim : Hi-SZ < Low-SZ, TD, fo. Emo: ns	ns	H, N, SZ, LH: D > H,N, D: TD < SZ Hi-SZ > TD, fo. Emo:TD: F < H,S. SZ: ns	ns	—	—	—	—
Fukuta et al., 2014	ns	ns	ns	—	—	—	—	—	—
Ibanez et al., 2012	—	—	—	Emo: H face: SZ > TD. TD: pos face < neg face. SZ: ns. Face vs. word: TD: face<word. SZ: ns	—	—	—	—	—
Jetha et al., 2013	P1 and TD N170 amp w/ shyness (+ is -)	ns	—	All stim: SZ > TD, fo Emo: ns	—	—	—	—	—
Johnston et al., 2005	P3 amp w/ R fusiform and L superior temporal gyrus (+)	ns	ns	—	ns	—	ns	ns	ns
Jung et al., 2012	ns	ns	—	All stim: SZ > TD, fo. Emo:H, F: SZ > TD. N: ns	—	All stim: TD: ns. SZ: male<female, fo. Emo: ns	—	ns	—
Kim et al., 2015	—	TD, F: LSF > HSF, fo. TD, N: ns. SZ, F: BSF > HSF, LSF, fo. SZ, N: BSF > HSF; LSF > HSF, fo	ns	TD, LH: BSF < HSF,LSF, fo. TD, RH: BSF < HSF, fo. SZ, LH: BSF < HSF,LSF, fo. SZ, RH: LSF < HSF, fo. Emo: ns	ns	ns	ns	ns	ns
Kirihara et al., 2012	SZ N170 amp w/ extraversion (+)	—	—	Face vs. Obj:Face, RH: SZ > TD. Obj: NR. Emo:TD: emo < N. SZ: ns	—	—	—	—	—
Komlosi et al., 2013	SZ P3 amp emo diff w/ pos (+) and neg (-) SZ symptom and med dose (+)	—	—	ns	—	—	—	—	—
Lee et al., 2007	SZ N170 lat w/ SZ symptom (+)	—	—	ns	ns	—	—	—	—
Lee et al., 2010	P1 and N170 lat w/ neg SZ symptom (-)	ns	Emo: Female LH: SZ > TD to H	SZ > TD, fo Emo: ns	All stim: SZ > TD, fo	ns	ns	All stim: TD: ns	All stim: SZ > TD, fo Emo: ns

Table 2 (continued)

Author, year	Correlations	P100		N170		N250	P300		
		Amplitude	Latency	Amplitude	Latency	Amplitude	Lat	Amplitude	Latency
		TD RH: female < male to H			Female, RH: H/F face, SZ > TD			SZ: female > male, fo Emo: ns	
Lynn & Salisbury, 2008	SZ N170 amp to emo w/ N170 amp to face/object (+)	—		All stim: SZ > TD, nfs. Face vs. Obj: ns. Emo: TD: S < H/F/A/D. SZ: ns	ns	—	—	—	—
Mori et al., 2012	-	—		—		—	—	H: SZ@S3 < TDSZ, S: S1 < S2,S3, foTD, SZ@S2 and S3: S > HSZ@S1: ns	H: SZ@S3 > TDSZ: S3 > S1,S2, foSZ@S3: S < H
Obayashi et al., 2009	N170 amp w/ global functioning (+)	ns	All stim: SZ > TD, nfs. Emo: ns	Face vs. Obj:Face: SZ > TD. Obj: ns. Emo: ns	ns	—	—		
Onitsuka et al., 2020	ns	—		S/H/F: SZ > TD		—	—	F/N: TD > SZ	
Ramos-Loyo et al., 2009	—	—		ns	—	—	—	All stim: ID face task, LH: SZ < TD, fo. H face target: SZ < TD, fo. Emo: ns	
Shah et al., 2018	ns	Parietal Sad: SZ < TD; Occipital S, A, F, SZ < TD; All face: SZ < TD	—	All stim: SZ > TD Emo: TD: N,F > H, N,F > A SZ: ns	—	—	—	All stim: SZ < TD Emo: ns	
Thoma et al., 2013	—	ns	ns	Emo: ns. Face and body: TD: incongruous<congruous, nfs. SZ: ns	ns	—	—		
Tso et al., 2015	N170 amp emo diff w/ delusions (+)	—		F-N face, averted gaze, LH: SZ > TD. TD: forward face>rotated face. SZ: ns	ns	—	—		
Turetsky et al., 2007	SZ N170 w/ pos SZ symptom (−); N170 amp w/ P3 amp; P3 amp w/ depression (−) and task (+)	ns		All stim: SZ > TD, fo. Emo: S face: SZ > TD	—	ns	—	All stim: SZ < TD, fo. Emo: ns	
Ueno et al., 2004	SZ P3 amp w/ neg SZ symptom (−)	—		—		—	—	All stim: SZ < TD, nfs. Emo:TD,	All stim: SZ > TD, nfs. non-- paranoid SZ >

Table 2 (continued)

Author, year	Correlations	P100 Amplitude	P100 Latency	N170 Amplitude	N170 Latency	N250 Amplitude	N250 Latency	P300 Lat	P300 Amplitude	P300 Latency
									paranoid SZ: H < N < S. Non-paranoid SZ: S < N < H. SZ: ns	TD,paranoid SZ, nfs. Emo: ns
Wang et al., 2020	SZ N170 amp w/ pos SZ symptom (+) and Personal and Social Performance Scale (−)	—	—	All stim: SZ > TD	—	—	—	—	—	—
Wynn et al., 2008	—	ns	—	All stim: SZ > TD, nfs Emo: ns	All stim: SZ > TD, Emo: ns	All stim: SZ > TD, nfs Face vs. Obj: ns Emo: ns	ns	ns	—	—
Wynn et al., 2013	—	—	—	All stim: SZ > TD, nfs. Face vs. Obj: ns. Emo: ns	—	All stim: SZ > TD, nfs. Face vs. Obj: ns. Emo: ns	All stim: SZ > TD, nfs. Face vs. Obj: ns. Emo: ns	ns	—	—
Zhang et al., 2016	SZ N170 amp w/ neg SZ symptom (−)	All stim: SZ < TD, fo. Emo: ns	—	All stim: SZ > TD, fo. Emo:TD: H,F < N. SZ: H < F,N	—	—	—	—	—	—

Note: for negative-going components, smaller amplitude indicates greater magnitude (e.g., SZ > TD for the N170 indicates that the TD group's N170 component was more negative than the SZ group's)

A angry, *ADHD* attention deficit-hyperactivity disorder group, *amp* amplitude, *ASD* autism spectrum disorder group, *DZ* dizygotic, *emo* emotion, *F* fear, *fo* face-only effect (study does not include non-face stimuli), *H* happy, *lat* latency, *M* ashamed, *MZ* monozygotic, *N* neutral, *neg* negative, *nfs* not face-specific effect (effect across face and non-face stimuli), *obj* object, *pos* positive, *R* surprised, *S* sad, *SZ* schizophrenia group, *TD* typically developing group, + positive correlation (larger magnitude or faster latency correlated with greater performance or fewer symptoms), − negative correlation (larger magnitude or faster latency correlated with poorer performance or more symptoms)
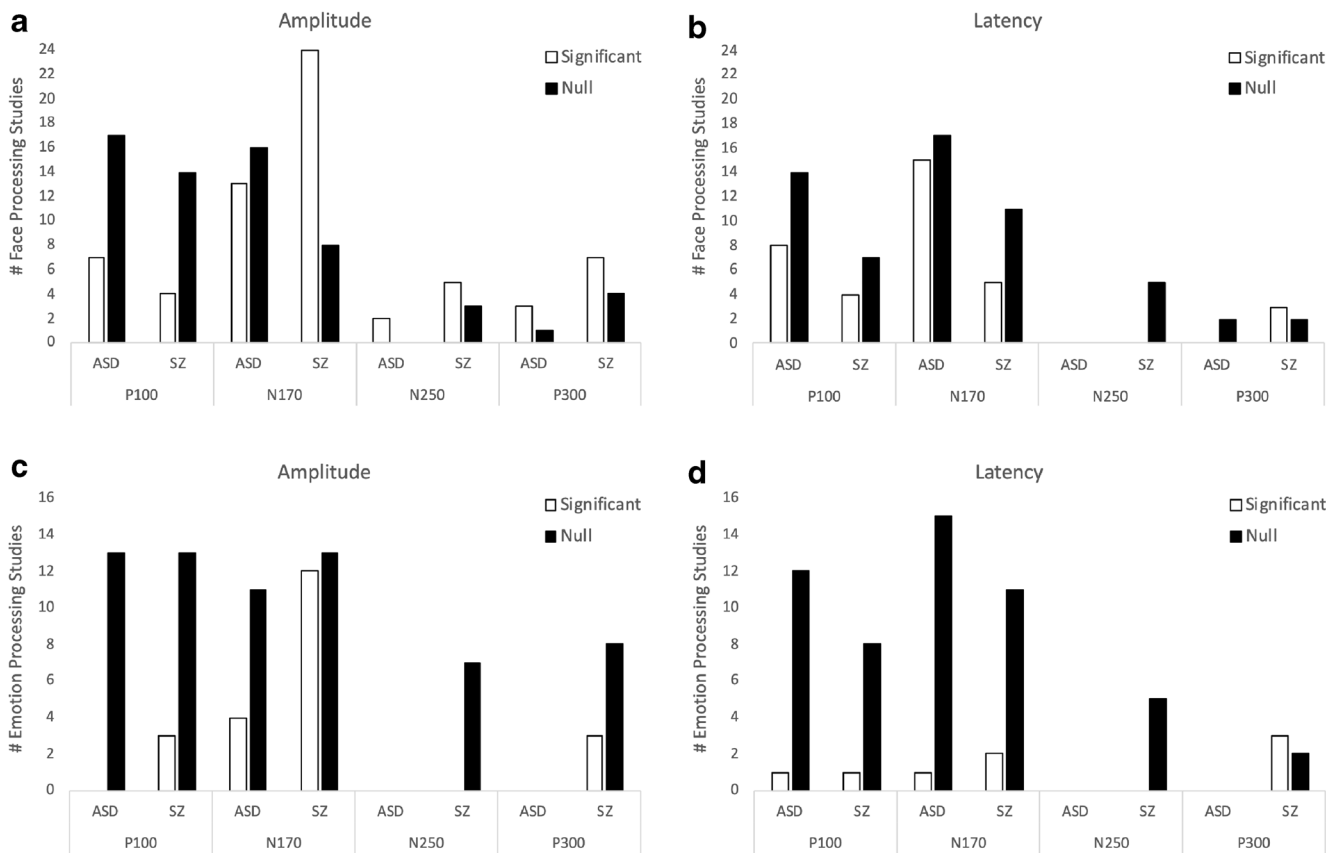
Fig. 2 Number of studies reporting significant versus null findings. **a** Face processing studies reporting amplitude. **b** Face processing studies reporting latency. **c** Emotion processing studies reporting amplitude. **d** Emotion processing studies reporting latency

P100 Latency

P100 Latency in ASD: Face

Almost two-thirds of studies (14 of 22) did not find significant differences between ASD and TD groups regarding P100 latency to faces (Apicella et al., 2013; Cygan et al., 2014; Hileman et al., 2011; Luckhardt et al., 2017; Luyster et al., 2017 [child]; McPartland et al., 2011; O'Connor et al., 2005 [child]; 2007; Senju et al., 2005; Sysoeva et al., 2018; Wagner et al., 2013; Webb et al., 2010; Webb et al., 2012; Wong et al., 2008). Eight studies reported group differences in response to facial stimuli. Two child and two adult studies reported increased latency to faces in ASD groups compared to the TD group (Batty et al., 2011; Luyster et al., 2017 [adult]; Neuhaus et al., 2016; O'Connor et al., 2005 [adult]). Of note, both Luyster et al. (2017) and O'Connor et al.'s (2005) child samples did not show differences between groups despite using the same methods as in their adult samples, where P100 latency delays were identified. A single study found shorter P100 latencies to faces in adults with ASD versus TD (Yamasaki et al., 2017). Two other child studies reported group differences in lateralization, wherein P100 latency

was faster in the right versus left hemisphere in TD, but this effect was absent in ASD (Akechi et al., 2010; Tye et al., 2013). A final study showed shorter left hemisphere P100 latencies present only in the ASD group (Shen et al., 2017).

P100 Latency in ASD: Emotion

Twelve of 13 studies of emotion processing in ASD did not report significant group differences between TD and ASD groups in P100 latency sensitivity to emotion (Akechi et al., 2010; Apicella et al., 2013; Hileman et al., 2011; Luckhardt et al., 2017; Luyster et al., 2017 [child and adult]; O'Connor et al., 2005 [child and adult]; 2007; Wagner et al., 2013; Wong et al., 2008; Yamasaki et al., 2017). However, Batty et al. (2011) reported that P100 was modulated by emotion in TD children, but not in ASD.

P100 Latency in SZ: Face

Seven of 11 studies reporting P100 latency to face stimuli reported no significant differences between SZ and TD groups (Campanella et al., 2006; Fukuta et al., 2014; Herrmann et al., 2004; Johnston et al., 2005; Kim et al., 2015; Liu et al., 2016;

Thoma et al., 2013). The remaining studies reported longer P100 latency in SZ groups compared to TD groups (Caharel et al., 2007; Lee et al., 2010; Obayashi et al., 2009; Wynn et al., 2008). None of these results are clearly face-specific, however; two studies did not include control stimuli (Caharel et al., 2007; Lee et al., 2010), and two found P100 latency delays in SZ across both face and control stimuli (Obayashi et al., 2009; Wynn et al., 2008).

P100 Latency in SZ: Emotion

Out of nine studies, eight reported no difference between P100 latency in SZ and TD groups in response to emotional faces (Caharel et al., 2007; Campanella et al., 2006; Fukuta et al., 2014; Johnston et al., 2005; Kim et al., 2015; Obayashi et al., 2009; Thoma et al., 2013; Wynn et al., 2008). One study reported sex-specific diagnostic differences in emotion processing: in females, P100 in the left hemisphere was delayed in SZ compared to TD when attending to happy faces (Lee et al., 2010). Moreover, longer latency to happy faces correlated with more negative symptomatology in SZ females. In SZ males, there was no overall delay in P100 compared to the TD group, and longer latency correlated with better performance on a behavioral detection task for happy versus fear faces.

N170 Amplitude

N170 Amplitude in ASD: Face

Since the initial report of N170 as a marker of atypical social perception in ASD (McPartland et al., 2004), 29 studies have examined N170 amplitude to faces in individuals with ASD, with 13 reporting differences in face processing by diagnosis. Of those 13, 6 studies (2 in children and 4 in adults) reported attenuated amplitude in ASD compared to TD (Churches et al., 2012b; Cygan et al., 2014; Groom et al., 2017; O'Connor et al., 2005 [adult]; Tye et al., 2014; Webb et al., 2012). Seven other studies reported differences in N170 response to faces between ASD and TD groups, but these differences were not always evident in direct statistical comparisons between groups (e.g., absence of lateralization in one group without demonstration of a group by lateralization interaction). One such study of children reported a typical face inversion effect in TD but the opposite effect in ASD (McPartland et al., 2011); conversely, another study found a stronger inversion effect in ASD, though this finding was not face-specific (Sysoeva et al., 2018). Three studies reported right hemisphere lateralization in TD but lack of lateralization in ASD (J. C. McPartland et al., 2011; Tye et al., 2013; Webb et al., 2006). Three studies reported findings of N170 amplitude differentiation between face stimulus types in TD but not ASD (Akechi et al., 2010), active versus passive tasks in TD

but not ASD (Churches et al., 2010), and repeated versus novel faces and objects in ASD but not TD (Key & Corbett, 2014).

Most studies reporting correlative analyses demonstrated associations between N170 amplitude and autistic symptomatology or social behavior. More negative N170 amplitude to inverted faces correlated with better face recognition in children with ASD (McPartland et al., 2011) and better social skills in adults with ASD (Webb et al., 2012). Stronger overall N170 amplitude to faces was associated with more typical social behavior in TD children (though not in the ASD group; Hileman et al., 2011), and increased right lateralization was associated with better social communication across both TD and ASD children (Tye et al., 2013). Sixteen of the 29 studies of N170 amplitude to faces in ASD did not find differences between diagnostic groups (1 of children, 5 distributed across childhood into adolescence, 2 of adolescents, 2 spanning adolescence into early adulthood, and 6 of adults) (Apicella et al., 2013; Batty et al., 2011; Churches et al., 2012a; Faja et al., 2016; Grice et al., 2005; Hileman et al., 2011; Khorrami et al., 2013; Luckhardt et al., 2017; Magnée et al., 2011; McPartland et al., 2004; O'Connor et al., 2005 [child]; 2007; Senju et al., 2005; Wagner et al., 2013; Webb et al., 2010; Wong et al., 2008). Approximately half of these 16 studies did, however, find differences in N170 latency, reported below. Of note, most of these studies did not instruct participants to attend to the faces specifically but included non-face targets to monitor attention. Only 4 of 16 studies with null results required participants to attend to faces. In contrast, all but one study (Webb et al., 2010) that reported differences between ASD and TD groups required participants to actively attend to stimuli. Given evidence that N170 amplitude is enhanced by attention (Churches et al., 2010; Mohamed et al., 2009), this finding suggests that differentiation of N170 amplitude emerges when the task demands are most face-specific.

N170 Amplitude in ASD: Emotion

Eleven of 15 studies reported no significant difference in N170 amplitude between ASD and TD groups during emotional face processing (Apicella et al., 2013; Batty et al., 2011; Hileman et al., 2011; Luckhardt et al., 2017; Luyster et al., 2017 [child and adult]; Magnée et al., 2011; O'Connor et al., 2005 [child and adult]; 2007; Wong et al., 2008). The remaining four studies reported group differences. In two of these studies, N170 amplitude was modulated by emotion in TD groups but not in ASD groups (Akechi et al., 2010; Wagner et al., 2013). One study found the opposite effect, showing N170 amplitude modulation by emotion only in the ASD group (Faja et al., 2016). This last finding is consistent with one other finding in which ASD groups with and without

comorbid ADHD showed increased N170 amplitude to neutral relative to fearful faces, whereas amplitude in the TD group was not modulated by emotional valence (Tye et al., 2014). More negative N170 amplitude overall, as well as more negative amplitude to fearful compared to neutral faces, correlated with better social communication skills across both TD and ASD children (Tye et al., 2014). Contrary to patterns detected among face processing findings, only one of these four studies yielding group differences required participants to attend to the emotional valence of the stimuli; the other two required participants to attend to non-social target stimuli. Likewise, four studies with null results tasked participants with identifying the emotional valence of stimuli (O'Connor et al., 2005 [child and adult]; 2007; Wong et al., 2008). Thus, the degree to which explicit demands for attention to emotion may contribute to N170 amplitude alteration in ASD for emotion processing is less clear than for face stimuli.

N170 Amplitude in SZ: Face

Twenty-four out of 32 studies investigating N170 amplitude to faces in adults with SZ reported significant differences in face processing between diagnostic groups. In four of these studies, amplitude to faces was smaller (less negative) in SZ relative to TD, whereas amplitude to objects was not significantly different between groups (Herrmann et al., 2004; Obayashi et al., 2009; Onitsuka et al., 2006; Tsunoda et al., 2012). One study observed N170 attenuation in SZ across inverted face and non-face images (Zheng et al., 2016). Another study showed right lateralization of N170 to low spatial frequency compared to high spatial frequency of fearful faces in SZ but no differentiation in TD groups (Kim et al., 2015). Eighteen additional studies report attenuated N170 amplitude to faces in SZ relative to TD; however, across all these studies, findings were not necessarily face-specific, as several lacked non-face control stimuli (Bediou et al., 2007; Caharel et al., 2007; Campanella et al., 2006; Jetha et al., 2013; Jung et al., 2012; Kirihara et al., 2012; Lee et al., 2010; Onitsuka et al., 2009; Onitsuka et al., 2020; Turetsky et al., 2007; Wang et al., 2020; Zhang et al., 2016) and the remainder showed main effects of group where N170 amplitude attenuation spanned both face and non-face stimuli (Ibanez et al., 2012; Liu et al., 2016; Lynn & Salisbury, 2008; Salisbury et al., 2019; Shah et al., 2018; Wynn et al., 2013). Of note, Salisbury et al. found this effect in both a group of patients presenting with chronic SZ and a group presenting with their first SZ-related hospitalization.   A handful of studies reported correlations between greater N170 amplitude to faces and higher global functioning (Obayashi et al., 2009), less positive SZ symptomatology (Campanella et al., 2006), more positive SZ symptomatology and lower personal and social performance scores (Wang et al., 2020), decreased shyness (Jetha et al., 2013), higher social competency (Tsunoda et al., 2012),

and increased extraversion (Kirihara et al., 2012). Together, these studies provide strong evidence for an association between more robust N170 response to faces and better social and psychiatric functioning across both TD and SZ populations. Eight studies reported no significant differences in N170 amplitude to faces between diagnostic groups (Akbarfahimi et al., 2013; Brenner et al., 2015; Komlosi et al., 2013; Lee et al., 2007; Ramos-Loyo et al., 2009; Thoma et al., 2013; Tso et al., 2015; Wynn et al., 2008). However, one found emotion-specific results that are detailed in the following section (Thoma et al., 2013), and the second found decreased N170 differentiation to facial angle as compared to TD controls (Tso et al., 2015). These eight studies have comparable methodologies and group demographics to those studies that do detect group differences.

N170 Amplitude in SZ: Emotion

A number of studies (12 of 25) suggest altered N170 amplitude during emotion discrimination in SZ compared to TD (Caharel et al., 2007; Campanella et al., 2006; Ibanez et al., 2012; Jung et al., 2012; Kirihara et al., 2012; Lynn & Salisbury, 2008; Onitsuka et al., 2020; Shah et al., 2018; Thoma et al., 2013; Tso et al., 2015; Turetsky et al., 2007; Zhang et al., 2016). Four of these studies reported that TD groups showed modulation of N170 amplitude based on emotional valence, whereas SZ groups did not (Campanella et al., 2006; Ibanez et al., 2012; Kirihara et al., 2012; Lynn & Salisbury, 2008). Four studies reported attenuated N170 amplitude in SZ compared to TD to specific emotions, such as disgust, sadness, and happiness (Caharel et al., 2007; Jung et al., 2012; Onitsuka et al., 2020; Turetsky et al., 2007). One found that N170 amplitude was differentially modulated by emotion in SZ relative to TD: whereas in TD it was larger for happy and fearful faces than for neutral faces, in SZ it was larger for happy faces than for neutral or fearful (Zhang et al., 2016). Another found emotion modulation, with neutral and fearful larger in amplitude than happy and angry faces, in the controls but on the SZ group (Shah et al., 2018). Additionally, reduced left hemisphere N170 amplitude to fearful faces in particular was correlated with severity of blunted affect in patients. Two additional studies found group differences as a function of more nuanced influences on emotion perception, including larger left lateralized N170 amplitude in fearful vs. neutral averted-gaze faces in SZ (Tso et al., 2015), and enhanced N170 amplitude in TD, but not SZ, when the emotion displayed in a stimulus differed from the stimulus emotion proceeding it (Thoma et al., 2013).   Thirteen studies investigating N170 amplitude reported no significant differences between SZ and TD in this ERP component during emotion processing (Akbarfahimi et al., 2013; Bediou et al., 2007; Colleen A Brenner et al., 2015; Jetha et al., 2013; Kim et al., 2015; Komlosi et al., 2013; S. Lee et al., 2007; S.-H. Lee et al.,

2010; Obayashi et al., 2009; Ramos-Loyo et al., 2009; Wang et al., 2020; Wynn et al., 2013; Wynn et al., 2008). These studies have comparable population demographics and methodologies to the studies above where differences were detected.

N170 Latency

There were 32 studies of ASD that reported N170 latency. Nineteen of these studies were performed in children, and 13 were in adults.

N170 Latency in ASD: Face

Of the 19 studies investigating N170 latency during face processing in children with ASD, eight reported atypical temporal processing in the ASD group. One study reported face-specific delays in the ASD group compared to the TD group (McPartland et al., 2011); another reported delayed latency to faces compared to objects in the ASD group, contrasted with faster latency to faces than objects in the TD group (Webb et al., 2006); and four studies found slower N170 latency in ASD compared to TD groups across both face and non-face stimuli (M. Batty et al., 2011; Hileman et al., 2011; Khorrami et al., 2013; Luyster et al., 2017 [child]). A study by Gunji et al. (2009) found delayed latency when comparing a child ASD group to adult controls but not to child controls. One study observed shorter N170 latencies in the left hemisphere only in the ASD group across both social and nonsocial stimuli (Shen et al., 2017); this finding is in the opposite direction than others, indicating faster N170 latency in ASD. Eleven studies in children did not find significant between-group differences in N170 latency to faces (Akechi et al., 2010; Apicella et al., 2013; Grice et al., 2005; Luckhardt et al., 2017; O'Connor et al., 2005 [child]; Senju et al., 2005; Sysoeva et al., 2018; Tye et al., 2014; Tye et al., 2013; Wagner et al., 2013; Wong et al., 2008). There were no observable differences in methodology or group characteristics to account for variations in study findings (Table 1). In adults, seven of 13 studies indicated atypical N170 latency in ASD groups. Across two studies, faces elicited a slower N170 latency in ASD compared to TD, while between-group differences in N170 latency to object images were not significant (McPartland et al., 2004; O'Connor et al., 2007); an additional three studies also found N170 delays to faces, but there were no non-social control stimuli in these experiments (Luyster et al., 2017 [adult]; O'Connor et al., 2005 [adult]; Yamasaki et al., 2017). Two studies suggest a relation between N170 latency and clinical symptoms. McPartland et al. (2004) found that, in ASD but not TD, slower N170 latency to both upright and inverted faces associated with better face memory, suggesting that longer processing times may confer improved behavioral performance. Though Webb

et al. (2012) did not find deficits in N170 latency to upright faces relative to either houses or inverted faces or replicate McPartland et al.'s (2004) association between N170 latency with face memory, faster N170 to upright versus inverted faces was related to both better social skills and better face memory. Two studies reported topographic differences in N170 latency between ASD and TD. N170 latency to faces was faster medially than laterally in TD (Webb et al., 2012), but trending toward faster laterally than medially in ASD (Webb et al., 2010). Finally, six studies of adults with ASD found no significant between-group differences or differences in lateralization in N170 latency to face stimuli (Churches et al., 2012a; Churches et al., 2012b; Owen Churches et al., 2010; Cygan et al., 2014; Magnée et al., 2011; Tseng et al., 2015). A recent meta-analysis of face processing studies in ASD indicated that, on average, N170 latency was delayed in ASD relative to TD (Kang et al., 2018).

N170 Latency in ASD: Emotion

Fifteen of sixteen studies of children and adults converge in reporting no emotion-dependent differences in N170 latency between individuals with ASD and TD (Akechi et al., 2010; Apicella et al., 2013; Batty et al., 2011; Hileman et al., 2011; Luckhardt et al., 2017; Luyster et al., 2017 [child and adult]; Magnée et al., 2011; O'Connor et al., 2005 [child and adult]; 2007; Tseng et al., 2015; Wagner et al., 2013; T. K. Wong et al., 2008; Yamasaki et al., 2017). Seven of these studies were of children only, two studies included both child and adult groups, and four studies only included adults. Just one study reported significant differences in emotion perception: when taking ADHD diagnosis into account, Tye et al. (2014) found a group by emotion interaction in which children with ASD showed a longer N170 latency to angry faces than to neutral faces and a shorter latency to happy faces than to fearful faces, whereas children with ASD and comorbid ADHD showed the opposite pattern and TD children did not show any differences in N170 latency across emotions. This finding suggests that a cross-diagnostic approach to identifying neural indices of emotion processing may be more informative than studies restricted to one clinical group.

N170 Latency in SZ: Face

Eleven of 16 studies reported no difference in N170 latency between diagnostic groups in response to face stimuli (Campanella et al., 2006; Herrmann et al., 2004; Johnston et al., 2005; Kim et al., 2015; Lee et al., 2007; Lynn & Salisbury, 2008; Obayashi et al., 2009; Thoma et al., 2013; Tso et al., 2015; Tsunoda et al., 2012; Wynn et al., 2008). The remaining five studies found adults with SZ showed delayed N170 latency to faces compared to TD controls. One of these studies found these results for both upright and inverted faces

(Zheng et al., 2016) and showed that N170 latency correlated with both negative and general psychiatric symptoms in SZ patients. However, because the other four studies either had no control stimuli (Caharel et al., 2007; Lee et al., 2010; Wynn et al., 2013) or saw main effects across both face and non-face stimuli (Akbarfahimi et al., 2013; Caharel et al., 2007; Lee et al., 2010), N170 delays in SZ are not necessarily face-specific.

N170 Latency in SZ: Emotion

Eleven of 13 studies of emotion processing reported no significant differences between SZ and TD in N170 latency modulation to emotional faces (Caharel et al., 2007; Campanella et al., 2006; Johnston et al., 2005; Kim et al., 2015; Lee et al., 2007; Lynn & Salisbury, 2008; Obayashi et al., 2009; Thoma et al., 2013; Tso et al., 2015; Wynn et al., 2013; Wynn et al., 2008). Two studies reported a differential effect of emotional faces on N170 latency in SZ versus TD. In a passive viewing study, adults with SZ showed a delayed N170 to fearful faces relative to other emotional faces, whereas controls displayed the shortest latencies to fearful faces (Akbarfahimi et al., 2013). Investigating gender differences, Lee et al. (2010) reported emotion-specific differences between females, but not males, with SZ and TD: in the right hemisphere, happy and fearful faces elicited a delayed N170 in females with SZ compared to those with TD. Slower N170 latency to happy faces in females with SZ was also correlated with increased expression of negative SZ symptomatology. An earlier study from the same group reported faster N170 latency to happy and neutral faces was associated with increased positive and negative SZ symptomatology, but found no significant differences between groups (Lee et al., 2007); thus, examining gender-specific effects may clarify the nature of processing deficits and relations with clinical symptoms.

N250 Amplitude

N250 Amplitude in ASD: Face

Two studies in adults with ASD reported on N250 amplitude, and both found significant, yet conflicting, group differences. When viewing a series of neutral faces and instructed to attend to a target face, relative to TD adults, adults with ASD showed reduced N250 amplitude to targets (Churches et al., 2012a). Groups did not differ in their N250 response to non-target faces. In contrast, Webb et al. (2010) reported enhanced N250 amplitude to both familiar and unfamiliar face stimuli in the ASD group relative to TD adults. In the same study, N250 amplitude difference between familiar versus unfamiliar faces was right lateralized in ASD, while it was uniform across hemispheres in the TD group. Differing methodologies across these two studies may have elicited different main effects. The first used unfamiliar target faces in an active viewing task demanding attention and facial memory; the second used familiar faces in a passive viewing task. Of note, Webb et al. (2010) also reported that increased right lateralization of N250 to familiar versus unfamiliar faces in the ASD group correlated with lower ADOS scores in communication and social domains.

N250 Amplitude in ASD: Emotion

There were no studies of emotion processing in ASD that reported N250 amplitude.

N250 Amplitude in SZ: Face

Three of eight studies of SZ measuring N250 amplitude to faces reported group differences. Two of these studies reported attenuated N250 in SZ groups relative to TD groups to both face and non-face stimuli (Wynn et al., 2013; Wynn et al., 2008). These studies used the same methodology, and the authors noted that while their TD groups were different across the two studies, their SZ groups were highly overlapping. A third investigated sex differences and reported larger N250 amplitudes in males versus females with SZ when attending to faces; this sex difference was absent in TD (Jung et al., 2012). Overall, however, no between-group (SZ versus TD) differences were significant. The remaining five studies did not find significant differences in N250 amplitude between groups (Brenner et al., 2015; Kim et al., 2015; Lee et al., 2010; Liu et al., 2016; Turetsky et al., 2007).

N250 Amplitude in SZ: Emotion

Seven of the eight studies reported above used emotional stimuli, but none found group differences in N250 amplitude as a function of the emotional valence of faces.

N250 Latency

N250 Latency in ASD There were no studies of face or emotion processing in individuals with ASD that reported N250 latency.

N250 Latency in SZ

There were five studies of face and emotion processing in SZ that reported N250 latency; however, none found significant group differences, either to faces overall or to emotional faces specifically (Kim et al., 2015; Lee et al., 2010; Liu et al., 2016; Wynn et al., 2013; Wynn et al., 2008).

P300 Amplitude

P300 Amplitude in ASD: Face

Three of four studies of ASD reporting on P300 amplitude detected differences between diagnostic groups, albeit in conflicting directions. In two studies, familiar faces elicited greater P300 than unfamiliar faces in TD children, whereas this effect was absent in children with ASD (Gunji et al., 2013; Gunji et al., 2009). In contrast, Key and Corbett (2014) found enhanced P300 amplitude to repeated faces in children with ASD relative to the TD group. P300 amplitude was also greater to repeated faces than to neutral stimuli in ASD but not TD. However, in this study, the same pattern was seen for repeated objects, suggesting the enhanced P300 effect during stimulus repetition was not necessarily face-specific. When measuring neural response to averted and direct eye gaze, there were no significant differences in P300 amplitude between children with ASD and TD to either stimulus type (Senju et al., 2005).

P300 Amplitude in ASD: Emotion

There were no studies of emotion processing in ASD that reported P300 amplitude.

P300 Amplitude in SZ: Face

Seven of 11 studies of SZ reporting on P300 amplitude detected diagnostic differences during reception of facial stimuli. In five of these studies, P300 amplitude was attenuated in SZ relative to TD, although these results were either found across both faces and control stimuli (Shah et al., 2018; Ueno et al., 2004) or had no control stimuli (Onitsuka et al., 2020; Ramos-Loyo et al., 2009; Turetsky et al., 2007). However, greater P300 amplitude was correlated with lower negative SZ symptomatology (Ueno et al., 2004) and better emotion identification (Turetsky et al., 2007), suggesting its relevance for social symptomatology specifically. In a study of sex differences, while P300 amplitude was equivalent in TD males and females, females with SZ showed a larger P300 amplitude to faces than males with SZ; overall, there were no between-group differences with regard to P300 amplitude (Lee et al., 2010). Finally, a study investigating effects of antipsychotic medication reported that, relative to TD controls, the SZ group showed attenuated P300 amplitude to faces, but only after exposure to 12 months of medication (Mori et al., 2012). Four studies reported no group differences in P300 amplitude to faces (Bediou et al., 2007; Johnston et al., 2005; Jung et al., 2012; Kim et al., 2015). There were no clear patterns among results to indicate methodological or sample differences accounting for this variability.

P300 Amplitude in SZ: Emotion

Three of 11 studies of emotion processing reported significant group differences in P300 amplitude to emotional faces in SZ. When observing images of infant faces and objects, TD adults showed the largest P300 amplitude to a crying infant face and the smallest amplitude to a happy face (Ueno et al., 2004), suggesting that an infant's distress is most salient to TD adults. In the SZ group as a whole, P300 amplitude was not modulated by the emotional valence of the infant face; however, when the SZ group was divided by subtype, adults with paranoid SZ showed the same pattern as the TD group, while those with non-paranoid SZ showed an inverted effect, with larger P300 amplitude to happy than to crying faces. In a second study, fearful adult faces elicited greater P300 amplitude than neutral faces in TD adults tasked with attending to emotional valence. There was no modulation of the P300 response by emotion in the SZ group (Bediou et al., 2007). A third study found that TD adults had higher P300 amplitudes to fearful and neutral faces than the SZ group, but the groups responded similarly to happy and sad faces (Onitsuka et al., 2020). The remaining eight studies found no significant group differences in emotion processing as measured by P300 amplitude (Johnston et al., 2005; Jung et al., 2012; Kim et al., 2015; Lee et al., 2010; Mori et al., 2012; Ramos-Loyo et al., 2009; Shah et al., 2018; Turetsky et al., 2007).

P300 Latency

P300 Latency in ASD

Two face processing studies in ASD reported on P300 latency; neither found group differences (Gunji et al., 2013; Senju et al., 2005). No emotion processing studies in ASD reported on P300 latency.

P300 Latency in SZ

Three of five studies of face and emotion processing that reported on P300 latency in SZ found group differences. Two studies found delayed P300 in SZ compared to TD in response to all stimuli, regardless of whether stimuli were faces or objects, or of the emotional valence of face stimuli (Lee et al., 2010; Ueno et al., 2004). Latency differences were, however, modulated by paranoia. Specifically, adults with non-paranoid SZ showed longer P300 latency to faces than TD adults, whereas individuals with paranoid SZ did not differ from the TD group (Ueno et al., 2004). This pattern parallels the P300 amplitude finding from the same study. In a third study, adults with SZ who had been medicated for 12 months showed delayed P300 latency to happy—but not neutral or sad—faces relative to TD adults (Mori et al., 2012). In the same study, a slowing effect of medication on P300 latency in SZ was

apparent: adults with SZ showed slower P300 latency after 12 months of medication compared to their responses while medication-naïve and medicated for a shorter, 3-month interval. Two studies did not report group differences in P300 latency (Johnston et al., 2005; Kim et al., 2015).

Discussion

Here we have reviewed the results of studies reporting on P100, N170, N250, and P300 ERP components in response to neutral and emotional face stimuli in individuals with autism spectrum disorders and schizophrenia. Our review sheds light on patterns of replicability in the literature and highlights multiple areas marked by inconsistent findings and in need of further research.

For the P100, which reflects early stages of visual processing, there is little evidence of amplitude or latency alterations in ASD across face or emotion processing. Scattered findings suggest that P100 amplitude may index neural processes relevant to ASD, but primary P100 deficits are unlikely. As with face processing, P100 latency does not seem to be a reliable indicator of emotion processing differences between ASD and TD groups. While P100 latency may index variability in speed of low-level visual processing across groups, it does not capture behavioral differences in face or emotion processing between ASD and TD groups. In schizophrenia, there is similarly little evidence for P100 amplitude or latency alterations being effective indices of face or emotion processing deficits.

The N170, a well-studied index of face processing, was the component most consistently demonstrating atypical neural response across the two neurodevelopmental disorders. In ASD studies, approximately 40% of all findings reported diminished amplitude and delayed latency to faces in ASD compared to TD; however, several of these studies did not include non-face control stimuli or found effects that were not specific to faces. Two-thirds of the studies that found significant differences required active attention to stimuli, whereas three-quarters of the studies that found no significant differences were passive viewing tasks. This distinction suggests that N170 deficits in ASD are particularly prominent when attentional demands are involved. There was little evidence of N170 amplitude or latency reliably indexing deficits in emotion processing in ASD. In studies of SZ, evidence for N170 latency delays to faces and differential N170 latency by emotion was mixed. However, there was strong evidence of diminished N170 amplitude to both face and non-face stimuli, indicating differences by diagnosis in early visual—but not necessarily face-specific—processing. There was also strong evidence of diminished N170 amplitude in SZ from studies of emotion processing. In general, N170 amplitude was differentially responsive to neutral versus emotionally valenced faces in TD groups, whereas SZ groups did not tend to show this specialization. This pattern may suggest that processing the emotional content of faces, rather than just faces themselves, may be most specifically atypical in SZ. Moreover, a recent systematic review on the N170 in SZ noted that correlations with clinical features were strongest in older individuals and that abnormalities in electrophysiological findings are more associated with more severe symptomatology (Murashko & Shmukler, 2019). In contrast, early structural processing of faces in general, more so than differentiation of particular emotions, may be more consistently impacted in ASD. However, the utility and reliability of the N170 as a biomarker in ASD remain debated and experiment features such as sample size and effect size must be considered closely (Kang et al., 2019; Vettori et al., 2019).

Across the N250 and P300 components, there are too few studies in ASD to draw conclusions despite promising group differences. Further research comparing N250 and P300 amplitude in ASD and TD, along with re-analysis of previously published studies that did not include these components in their original analysis, may help elucidate the neural underpinnings of atypical face and emotion perception. Many studies that measured N250 and P300 amplitude found group differences in face perception, indicating that both components may be understudied but promising leads for indexing social cognition deficits.

In SZ, there is a slightly larger literature, but evidence for diminished N250 and P300 amplitude to face stimuli is mixed. N250 amplitude was attenuated to face, but not emotion, stimuli in approximately 40% of SZ studies, but N250 latency appears to be unaffected. P300 amplitude to face stimuli appear to be more reliably affected, and P300 amplitude may both mark symptom levels and be affected by medication treatments. Though findings from a small sample of studies suggest P300 latency may reliably index cognitive processing deficits for both face and emotion stimuli in SZ, the relative paucity of literature measuring P300 latency makes it difficult to identify this component as a sensitive neural marker of social cognition differences.

To expand both ASD and SZ literatures, given that ongoing, continuous EEG is recorded before segmentation for ERP analyses, it is likely that pre-existing data from studies reporting on earlier components (e.g., P100 and/or N170) could provide an opportunity to analyze N250 and P300 components to test for group differences during face and emotion processing. Such re-analysis of existing data may provide a quick and efficient mechanism to address outstanding questions of whether later, attentional neural responses are impacted in the context of reception of facial communication, particularly in ASD where existing data is more limited.

Accounting for Variability Across Studies

Variation in methodologies—such as stimulus design and subject factors—may account for some of the inconsistencies

among studies. Differences in low-level visual characteristics, such as luminance, contrast, and color, can produce significantly different waveforms (Rossion & Caharel, 2011; Rousselet & Pernet, 2011), yet most studies did not provide specific details on the low-level visual aspects of their stimulus set. Likewise, there were many different types of stimuli across studies. For example, while all studies included face stimuli, the nature of these faces varied (i.e., upright vs. inverted, familiar vs. unfamiliar, neutral vs. emotional, child vs. adult). Moreover, faces were sometimes the sole stimuli within a study paradigm, whereas other studies included non-face conditions. Study design, such as the use of blocking by stimulus type (e.g., face vs. non-face; neutral vs. emotional faces) vs. randomly interspersing all stimuli, can affect ERPs due to repetition and habituation effects (Bentin & Peled, 1990; Ravden & Polich, 1999), which few studies examined. Importantly, many studies claiming group differences in face processing did not include non-face control stimuli, preventing conclusions regarding the specificity of reported findings to faces.

Participant tasks varied widely across studies, from passive viewing, to providing button-press responses to occasional attentional control trials not included in later analyses, to providing task-relevant verbal responses to each trial. Variability in task design yields inconsistencies in the nature and burden of the cognitive load placed on the participant and may manipulate how intensely and/or consistently the participant is attending to the stimuli. Unfortunately, there were very few instances in which clear patterns of replicable vs. less consistent findings were revealed as a function of particular aspects of stimulus type, task design, or demands for participant attention and task engagement. Finally, no studies in this review monitored participant gaze with eye tracking; thus, while participants were ostensibly looking toward the screen (based on experimenter observations or attention monitoring tasks), gaze was not directly measured.

Subject factors also may have contributed to variability across studies. For example, not all studies matched clinical and control groups by IQ, making it difficult to ascertain which observed differences were due to diagnosis versus may have been driven by differences in cognitive functioning. Moreover, most studies did not account for sex when matching controls, and this variable may drive certain effects or correlations in ASD and SZ groups, as highlighted by several studies (Jung et al., 2012; Lee et al., 2010). ERP components indexing face perception change in amplitude and latency over the lifespan (Batty et al., 2011; Itier & Taylor, 2004b), yet, when comparing studies of ASD and SZ, we are limited by the fact that most studies of ASD are in children, while all studies of SZ are in adults. Participants in clinical groups, especially SZ, were often medicated with psychoactive agents. In their longitudinal investigation of the effects of antipsychotic use on ERP waveforms, Mori and colleagues

(Mori et al., 2012) reported changes in patients' P300 amplitude and latency between drug-naïve and medicated time points, and another study reported a positive correlation between medication dosage and P300 amplitude to emotional compared to neutral faces (Komlosi et al., 2013). Many studies did not report on the medication status of patients, and still others reported medication status but did not discuss testing for correlations between ERP waveforms and medication dose or controlling for medication status. Thus, effects of medication differences among participants and between clinical and control groups could also contribute to variability in findings across studies.

Variability in clinical profiles within participant samples could also have affected results. With respect to subject characterization, many studies did not report confirming ASD or SZ diagnoses in the context of the study with an ADOS (for ASD; Lord et al., 2012) or SCID (SCID, for SZ; First, 1995). Lack of rigorous characterization of participants may mean that some clinical groups were not composed entirely of participants with gold-standard ASD or SZ diagnoses and/or may have included individuals falling short of diagnostic threshold. Many studies did not report level or severity or particular profiles of symptomatology within patient samples, despite the fact that ASD and SZ samples tend to be phenotypically heterogeneous. With disorders that are behaviorally defined (and which likely lump individuals with multiple different etiological origins based on their symptoms; Happé et al., 2006; Insel, 2010), re-grouping participants by subtypes or symptom profile can be a helpful way of finding patterns. Conducting correlations and regressions accounting for symptom levels on well-validated measures like the ADOS (for ASD; Lord et al., 2012) and Positive and Negative Syndrome Scale (PANSS, for SZ; Kay et al., 1987), as well as broader measures of symptoms that cut across diagnoses, may help further clarify the association between clinical profiles and neural processing abnormalities. For example, when dividing their SZ group into paranoid and non-paranoid subgroups, Ueno et al. (2004) found that the paranoid subgroup showed similar P300 amplitude and latency to the TD group, whereas the non-paranoid subgroup differed in P300 amplitude to emotional faces and in latency to all stimuli. When looking at the SZ group as a whole, however, these differences were obscured.

Related to the need to carefully characterize and account for variation in symptom profiles among participants is the need to recruit large study samples in order to capture heterogeneity and enable statistical analyses that are adequately powered to detect effects using covariates and regressions. Among the studies highlighted in this review, 50% of studies in ASD (18 of 38) and approximately 25% of those in schizophrenia (8 of 36) had fewer than 15 participants per group. Moreover, whereas nearly 25% of SZ studies (8 of 36) included 30 or more participants per group, only approximately 10%

of ASD studies (4 of 38) had sample sizes this large. Small samples are particularly vulnerable to spurious or non-generalizable findings (Button et al., 2013). Thus, it may be the case that some of the inconsistencies across studies relate to the relatively small sample sizes, particularly in the ASD literature. Moving forward, recruiting large samples to enable examination of more homogenous subsets of participants and to allow for evaluation of the association between ERP components and clinical traits measured in a continuous fashion might help to explain differences among studies where samples can be quite variable. Performing meta-analyses will also help to disentangle type two error due to small sample size from biologically real effects (e.g., Fernandes et al., 2018; Kang et al., 2018; Zheng et al., 2018).

Considerations for Future Research

This review of electrophysiological studies of face perception in ASD and SZ indicates that the N170, and possibly the N250 and P300, hold promise as biomarkers indexing the integrity of social perceptual systems across neurodevelopmental disorders and for providing clues about underlying neural dysfunction within and across disorders. Several caveats for future research emerge from these findings.

First, this review makes clear that alterations in the highlighted ERP indices are not diagnostically specific. Similar patterns of atypical processing at identical components were observed in both ASD and SZ. This suggests that, consistent with the RDoC framework, it may be most useful to evaluate the underlying neural bases of neurodevelopmental disorders in terms of specific, dimensionally measured processes related to function and dysfunction transdiagnostically, rather than designing studies around a particular diagnostic category. Although the reviewed evidence does not make a reliable case that any one particular ERP component may be a consistent diagnostic biomarker, correlations with symptomatology and key associated features (e.g., emotion recognition) indicate promise with respect to other areas of biomarker development. These include classifying individuals into subgroups relevant to treatment selection (stratification biomarkers), demonstrating the influence of an intervention on a targeted neural system (target engagement biomarkers), or offering short-term indication of intervention effects on symptoms or underlying neural systems (early efficacy biomarkers; McPartland, 2016). Our review focused specifically on ASD and schizophrenia; however, social cognition and these ERP components are germane to multiple other psychiatric diagnoses (Feuerriegel et al., 2015). Increased focus on transdiagnostic samples can provide greater clarity regarding the information these ERP indices provide about the neural processes underlying neurodevelopmental and psychiatric disorders.

As the variability in findings across existing studies makes clear, it is necessary to carefully consider individual differences in participant characteristics, such as age, both within and across diagnostic groups. Differences in findings reported within ASD between child and adult samples and cross-diagnostically between studies of ASD and SZ may reflect differences in age-related characteristics of particular samples. For example, some alterations in neural responses may be more prominent in adults, reflecting later-onset difficulties or premature developmental plateaus, whereas some may be most notable in young children and resolve over time. Future research on markers of social perception must either constrain age ranges to reduce variability, conduct studies with sufficient statistical power to co-analyze age effects, or examine participant samples longitudinally to study the divergence of particular neural responses as they relate to emergence or amelioration of symptoms over time. Though age effects are less germane to schizophrenia, a disorder most commonly occurring in adulthood, whether alterations in ERP response to face stimuli are present in prodromal phases is an important question. Moreover, as other factors such as cognitive ability are also demonstrated to influence ERP indices, these characteristics must be considered and controlled, either statistically or through reduction of sample heterogeneity. Finally, prioritization of exploring hypotheses in large participant samples, either within individual studies or by harnessing "big data" sources, such as the National Institute of Mental Health's RDoC or NDAR data repositories (data-archive.nimh.nih.gov), will maximize the probability of detecting robust, clinically meaningful, and replicable findings.

Studies will benefit from increased consistency and methodological rigor in both experimental design and equipment. Based on existing data, it cannot be determined to what degree differences in EEG data recording and processing methods (e.g., high versus low impedance EEG systems, electrode density, choice of scalp topography for component extraction) contributed to the diversity of results. Similarly, research on social perception must include appropriate control stimuli. In the research reviewed here, in some cases, inference was limited by omission of non-face stimuli, stimuli matched for low-level visual features, and, in studies of emotion, both neutral/natural and emotional faces. Because many studies focused exclusively on social stimuli, it was often impossible to determine whether reported anomalies reflected specific difficulties in social brain circuitry or more general problems with object perception.

Many of these principles have been recognized by researchers and funding agencies, and within-disorder studies incorporating these features are in progress. Though not in the context of electrophysiological markers, the schizophrenia field recognized more than a decade ago with the MATRICS initiative the importance of identifying reliable markers and

developing standardized batteries of cognitive tests for use in diagnosis and assessing treatment outcomes (Marder & Fenton, 2004). The autism field has more recently begun promoting a similar agenda, with increased focus on the potential utility of electrophysiological biomarkers for capturing heterogeneity and predicting treatment. To this end, the Autism Biomarkers Consortium for Clinical Trials (www. asdbiomarkers.org) is evaluating a battery of EEG and eye-tracking social-communicative biomarkers in a large, rigorously characterized sample of children with ASD, with careful attention to participant characteristics, meticulous control of stimulus, task, and EEG recording parameters across sites, and a longitudinal component to provide information about developmental effects. Likewise, the European Autism Interventions — A Multicentre Study for Developing New Medications (EU-AIMS; Murphy & Spooren, 2012) is investigating similar markers, as well as those from additional biomarker domains, such as functional magnetic resonance imaging (fMRI). Although these initiatives are focused on single disorders, their scope and thoroughness are likely to advance understanding of the practical utility of ERP indices of face perception for developing novel interventions and guiding clinical trials.

Finally, the scarcity of studies including multiple diagnostic categories within the same project highlights the urgent need for transdiagnostic work if we are to better understand the specificity versus universality of particular alterations in brain responses to individuals with neurodevelopmental disorders. Thus, moving forward, it is imperative to include both individuals with ASD and SZ—and potentially those with other neurodevelopmental or psychiatric disorders or subthreshold traits—within a single study sample and to measure dimensional traits associated with both disorders across all participants. Studying children or adults at biological risk of ASD or SZ (i.e., siblings, parents, children of affected individuals) to see whether those at risk show altered neural response and/or behavior would also provide important information about intermediate phenotypes and risk states as they relate to brain functioning and clinical profiles. In addition, studies with a longitudinal design and an intervention (targeting either face or emotion processing circuits) may help better parse out causally relevant effects across diagnostic categories.

In summary, both the inconsistencies in findings within ASD and SZ samples and the overlap between findings across ASD and SZ samples make a clear case for the need to consider symptomatology in a dimensional fashion when designing studies and analyzing results in search of biologically meaningful and behaviorally relevant markers of face and emotion processing. Taking a cross-diagnostic, the RDoC approach allows for both direct comparison across diagnoses and more detailed sub-grouping or correlations featuring diagnostically cross-cutting, categorically agnostic phenotypic

traits. In addition, this type of study would also allow for clinical characterization to be made along a continuum, rather than imposing an artificial dichotomy (i.e., clinical vs. TD). By taking this approach, future research can address whether and how each categorical disorder (or subset of individuals within a disorder) shows a unique neural signature of altered reception of facial communication, versus whether some alterations in ERP markers of face and emotion processing are reflective of broad clinical impairment in social functioning versus diagnosis or trait-specific pathology. In so doing, research will begin to make more effective headway toward understanding the complexity of distinct versus overlapping etiologies of associated neurodevelopmental disorders, which will in turn position the field to accumulate the precision and replicability of findings needed to develop more targeted treatment approaches.

Declarations

Conflict of Interest   The authors declare no competing interests.

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