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Face Processing in ADHD: A Review of the N170 Event-Related Potential

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Attention-deficit hyperactivity disorder (ADHD) is associated with deficits in social functioning, including peer difficulties and poor relationship quality. Little is known, however, about the integrity of foundational sociocognitive abilities that support interpersonal interactions in ADHD. Face processing—a fundamental component of social cognition—has been a popular topic of recent investigations in this area. Researchers have attempted to delineate face processing mechanisms in ADHD to elucidate social deficits often seen in the disorder. Investigating the N170 event-related potential, a neural marker of face processing, has been a popular approach in this endeavour. Here, we present two accounts that offer competing views of how social deficits might arise in those with ADHD. Next, we systematically review and synthesise the literature on the N170 in ADHD to identify whether atypicalities in sociocognitive domains like face processing occur in this patient population. Gaps in the literature are identified and concrete solutions are offered to improve future research in this area. We end by discussing immediate implications for treatment approaches designed to address widely observed social deficits in individuals with ADHD.

Public Significance Statement
Attention-deficit hyperactivity disorder (ADHD) is a common psychological disorder characterised by inattention, impulsivity, and hyperactivity. On top of the attention deficits that are common with this disorder, research has also highlighted poor social outcomes for individuals with ADHD. This systematic review considers evidence for face processing deficits as one potential cause of these impoverished social outcomes.

Keywords: attention-deficit hyperactivity disorder, face processing, N170 event-related potential, social cognition, systematic review

Attention-deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterised by inattention, impulsivity, and hyperactivity (American Psychiatric Association, 2013). It is prevalent and hindering, with symptoms emerging at a young age and frequently persisting into adulthood (American Psychiatric Association, 2013; Faraone et al., 2006). It is well understood that ADHD impairs functioning in a variety of domains, including cognitive, academic, and occupational functioning (Barkley, 2003). Additionally, social impairments have been well-documented in ADHD. A sizeable literature devoted to characterising social functioning in ADHD consistently demonstrates that children with ADHD are rated as less popular by peers, have fewer reciprocated friendships, and are prone to more frequent peer victimisation than their typically developing (TD) counterparts. Adults with ADHD report poorer relationship quality, are less likely to marry, and experience higher divorce rates (Bagwell et al., 2001; Barkley & Murphy, 2010; Blachman & Hinshaw, 2002; Hoza, 2007). Despite the clear consequences of social impairment in ADHD, there is uncertainty surrounding the underlying mechanisms causing these social deficits to emerge (see Uekermann et al., 2010, for a review). To provide context for the systematic review to follow, we next briefly summarise two different accounts that vary in the degree to which social deficits in ADHD are thought to stem from characteristic impairments in executive functioning or atypical sociocognitive factors like face processing. We present these accounts not as formal theories but rather as summaries of approaches that prior researchers have taken in explaining why those with ADHD tend to experience poor social outcomes.

Consequential Social Deficit Account

The "consequential social deficit" account refers to the notion that those with ADHD experience social deficits as a consequence of core ADHD symptoms (i.e., inattention, hyperactivity, impulsivity). This theory posits that inattention, hyperactivity, impulsivity, and/or working memory deficits in childhood trigger a series of downstream social consequences that result in children with ADHD being less popular among classmates and often alienated by their peers (Hoza et al., 2005; see Nijmeijer et al., 2008, for a review). This perspective has been reflected in a variety of ways in the literature but ultimately suggests that hallmark symptoms of ADHD are the root cause of poor social outcomes for these individuals (e.g., Kofler et al., 2019). Put succinctly, Kofler et al. (2011, p. 1) write, "impaired social interactions

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in children with ADHD reflect, to a significant extent, the behavioural outcome of being unable to maintain a focus of attention on information within working memory."

As one example of this account at work, children with ADHD are indeed able to demonstrate appropriate social behaviours when prompted to do so in controlled settings (Merrell & Boelter, 2001; Saunders & Chambers, 1996), suggesting that they are capable of such behaviours, and that it is perhaps inattention symptomology leading to poor social behaviours in other, more distracting environments (e.g., classrooms). Along the same lines, inattention in ADHD has been found to correlate negatively with perception of emotion in others (Miller et al., 2011; Sinzig et al., 2008; see Shaw et al., 2014, for a review). In sum, the consequential social deficit account contends that poor social outcomes in those with ADHD are resultant from core ADHD symptomology leading to in appropriate social behaviours in demanding environments which can negatively influence how peers view and judge these individuals.

Inherent Social Deficit Account

Another possible account for why those with ADHD could suffer from poor social outcomes is based more so in patients' underlying neurobiological makeup. This idea, which we will call the "inherent social deficit" account, refers to the notion that those with ADHD may experience social malfunctions due to inherent neural deficits in sociocognitive information processing specifically. Unlike the consequential social deficit account which argues that poor social outcomes are a downstream consequence of core ADHD symptoms, this inherent social deficit account instead contends that social deficits in ADHD arise because of differences in the way the brain processes sociocognitive information. The inherent social deficit account would predict that those with ADHD may display neural markers of impoverished sociocognitive processing in relevant domains such as face processing. Critically, these markers would be distinct from underlying differences in neurobiology related to executive function impairments, which are thought to be the cause of poor social outcomes in the aformentioned consequential social deficit account (see Cubillo et al., 2012; Krain & Castellanos, 2006, for reviews).

As a recent meta-analysis by Bora and Pantelis (2016) suggests, adolescents with ADHD demonstrate significantly impaired theory of mind, as well as attenuated facial and vocal emotion recognition abilities (especially to angry and fearful emotions). Rapport et al. (2002) and Da Fonseca et al. (2009) have both posited that impairments in emotional information processing stem from low-level sociocognitive deficits (e.g., affect recognition) rather than hallmark executive functioning symptoms. Indeed, a review by Collin et al. (2013) included ADHD in a group of developmental disorders that demonstrate emotion recognition deficits. Other studies confirmed that ADHD patients are unencumbered when performing nonsocial control tasks (Buitelaar et al., 1999; Da Fonseca et al., 2009), which should not be the case if broader attention mechanisms are to blame for poor social perception. Thus, there has been mounting evidence in recent years that those with ADHD experience atypical sociocognitive functioning including in the domains of face perception and affect recognition, among others. What remains uncertain is whether these deficits are resultant from the same mechanisms that also underlie characteristic executive function impairments.

Face Processing and the N170 Event-Related Potential

Faces are among the most salient social stimuli for humans (Itier & Batty, 2009) and are thought to serve as sources of rich social-cognitive information, including emotion, identification of others, and gaze, just to name a few (Schwiedrzik et al., 2015). Distinct networks for face processing are thought to emerge quickly in childhood and undergo fine-tuning for years to follow (Cohen Kadosh et al., 2011). Within the realm of evolutionary psychology, face-specific neural regions have even been discovered in other social creatures, such as macaque monkeys (Schwiedrzik et al., 2015) and dogs (Dilks et al., 2015). Given their great importance, an entire field has emerged that is devoted to understanding how faces are processed (e.g., Itier et al., 2006; Richler & Gauthier, 2014). This domain of research has largely suggested that face processing is a foundational component of social cognition, with real-world consequences when it is impaired in humans (Freiwald et al., 2016). Therefore, it is clear that face processing represents a core feature of social cognition that helps to form the building blocks of everyday social interactions. But how might we measure it?

Neuroimaging techniques are among the most common methodologies used to study face processing because they allow for measurement of cognitive mechanisms at the neuroanatomical level. Electroencephalography (EEG) is one such neuroimaging tool that is often employed to measure event-related potentials (ERPs) as markers of ongoing cognitive processes, including those relevant for viewing faces. Specifically, the N170 ERP is a face-sensitive, negative-going waveform that can be observed approximately 130–200 ms after presentation of a face stimulus (Itier & Taylor, 2004). The N170 has been demonstrated as sensitive to holistic face recognition (Itier & Taylor, 2004), as well as to individually presented components of faces such as mouths and eyes (Taylor, Edmonds, et al., 2001). Critically for our purposes, the N170 seems to be an automatic response that is agnostic to attention (Cauquil et al., 2000) and is similarly uninfluenced by the familiarity of a face (Eimer, 2000). In sum, the N170 ERP has long been a hallmark of face processing due to its distinct sensitivity to faces, its automatic initiation, and its ambivalence toward individual difference factors such as learning ability.

Because the N170 has a reliable pattern of activation when participants view images of faces, any alteration to its predictable form is thought to indicate atypical processing of faces in the brain (Naumann et al., 2018). Abnormalities in amplitude (i.e., how strong the signal is) and/or latency (i.e., the speed of the signal following stimulus presentation) of the N170 ERP are two of the most common signs that an individual is not processing faces in a typical fashion. Impairment of the N170 is evident in certain neurodevelopmental disorders, including schizophrenia (Billeke & Aboitiz, 2013), bipolar disorder (Ibáñez et al., 2014), and autism spectrum disorder (ASD; Kang et al., 2018), possibly contributing to real-world social functioning deficits in these disorders (see Feuerriegel et al., 2015, for a review). However, abnormal N170 responses have yet to be well-characterised in other disorders that feature outward behavioural deficits in social functioning, such as ADHD.

As discussed earlier, those with ADHD are characterised by well-documented deficits in social outcomes. A growing area of research has highlighted diminished emotional face processing as a potential cause for these social impairments. In a recent review, Romani et al. (2018) found that the vast majority of research on face processing in ADHD investigated emotion recognition of some sort, whereas investigations of pure facial recognition outside of emotional

contexts were found in only a handful of studies. In two behavioural studies (Kibby & Cohen, 2008; Lee et al., 2016), those with ADHD showed normal facial recognition performance. In a third study by Demirci and Erdogan (2016), however, those with ADHD scored significantly lower on the Benton face recognition test (Benton et al., 1983). Therefore, it remains uncertain whether ADHD patients have intact face processing but still struggle in social situations due to other factors (e.g., inattention), or if their face processing is inherently flawed. By reviewing whether face processing elicits a typical N170 ERP response in ADHD patients, we can infer an answer to this critical research question.

Systematic Review of N170 ERP in ADHD

Face processing has only recently begun to be acknowledged as a potential component of socioemotional deficits in ADHD. Throughout the past decade, to determine whether abnormal face processing serves as a mechanism of social deficits in the disorder, several studies have attempted to identify signs of atypical face processing in ADHD at the neural level. However, mixed results have emerged, making it unclear whether irregularity in neural markers of face processing—specifically the N170 ERP—characterise ADHD. The development of targeted interventions to alleviate consequences associated with social deficits in ADHD requires identification of the root cause of such deficits before effecitve treatments can be designed. Thus, the goal of this review is to synthesise existing results to ascertain whether face processing, as measured by the N170 ERP, is indeed atypical in individuals with ADHD relative to TD controls (i.e., whether the inherent social deficit account has merit). Recall that an alternative explanation is that core ADHD symptoms (e.g., inattention) themselves are leading to poor social development (i.e., the consequential social deficit account). Here, we chose to focus our review on differences in N170 amplitude in particular as it is thought to provide a direct measure of variation in neural face processing mechanisms and most studies reviewed here reported their analyses based on this measure. Put simply, the central goal of this review is to determine if those with ADHD have inherently atypical face processing leading to poor social outcomes, or if face processing is intact but it is instead their ADHD symptoms that lead to downstream sociodevelopmental consequences.

Method

To ensure that our review would be comprehensive, we used a systematic procedure to identify relevant studies. Database searches were conducted using APA PsycInfo, Pubmed, and Web of Science databases on October 2, 2020. In all cases, the following Boolean search string was used: ((ADHD AND ERP AND (fac* OR express*))). No restrictions were made on the date of publication. Additionally, reference lists from relevant reviews and meta-analyses were mined for any articles that had not been identified via database searches. The identification and study selection procedures reported here follow the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (Moher et al., 2009; see Figure 1).

Initial searches yielded 206 total articles, and an additional 10 articles were identified from reference list searches. After removal of duplicates, 163 studies remained. Titles of these articles were assessed for relevance, revealing several articles that did not focus on social cognition (but rather often studied other neurocognitive processes), did not use visual stimuli, and/or did not focus on ADHD, resulting in the removal of 117 studies. Both authors independently reviewed the remaining 46 articles for relevance based on the content of their abstracts. Specifically, three key aspects were considered: inclusion of an ADHD group, ERP data, and use of social/facial stimuli. After this second filtering pass, 24 studies were excluded, leaving 22 highly relevant studies that were deemed eligible for in-depth review. Finally, after excluding articles that either did not present original N170 data or used irregular approaches to our current topic of interest, 14 comparable studies remained and will serve as focal pieces of evidence considered in this review. We begin now by reviewing the overall N170 signal as compared between ADHD and TD participants.

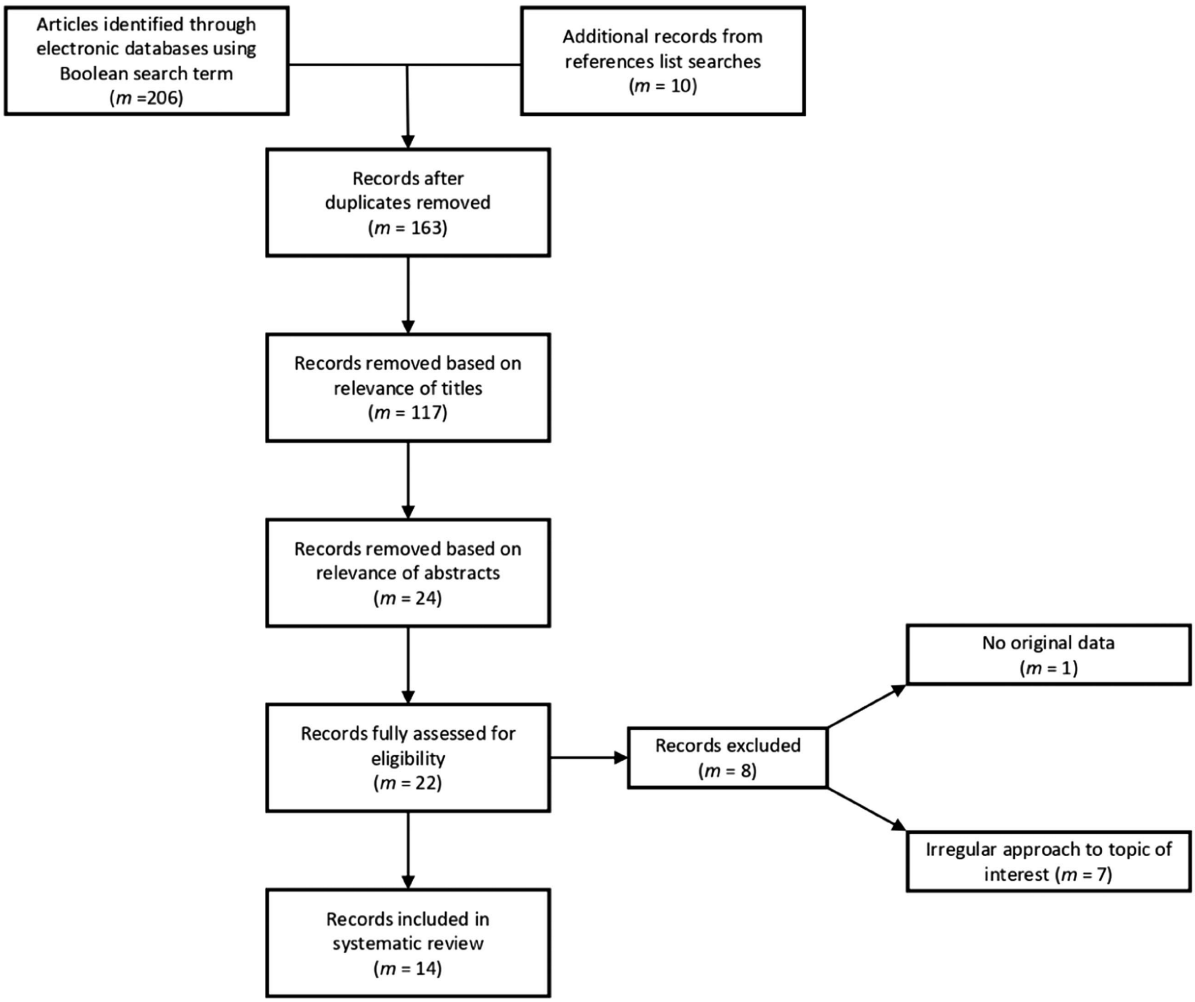
Group-Level Differences in N170 Response

As an initial step, we first review whether there have been consistent group-level differences in N170 responses between ADHD groups and neurotypical control groups. To do so, we considered the prevalence of significant main effects of experimental group on the N170 across all face stimuli types, regardless of participant age, N170 localization, encoding tasks, or the emotional expressions of facial images. By starting with high-level comparisons such as this, we attempt to first answer the most basic fundamental question for this topic: Are there any primary research article examples of face processing differences between ADHD and TD controls as measured by the N170? The answer to this question is seemingly the most direct way of lending initial support to a consequential or inherent social deficit account for social cognition abnormalities apparent in ADHD patients: the former would predict equivalent N170 responses between groups, whereas the latter would not.

After reviewing all relevant articles, the majority of studies did not detect significant main effects of group, indicating that the N170 responses to face stimuli were *not* different overall in ADHD participants relative to TD controls (although interactions with facial emotional expression were occasionally observed; Alperin et al., 2017; Flegenheimer et al., 2018; Groom et al., 2017; Karalunas et al., 2020; Raz & Dan, 2015a, 2015b; Rinke et al., 2017; Tye et al., 2013, 2014; Zhao et al., 2020). Within these articles, there were no clear third variables that could explain such consistent null results. There are examples of null group effects in studies that observed adults (e.g., Raz & Dan, 2015a) and children (e.g., Flegenheimer et al., 2018), experiments that employed active (e.g., Alperin et al., 2017) and passive (e.g., Tye et al., 2013) tasks, as well as in neutral (e.g., Tye et al., 2014) and emotional (e.g., Groom et al., 2017) face stimuli.

A handful of studies did, however, detect significant main effects of group on N170 responses to face stimuli (Ibáñez et al., 2014; Meier et al., 2012; Williams et al., 2008). Even in this latter set, though, different results emerged regarding the *direction* of group differences. Whereas one article identified an enhanced N170 to faces in ADHD relative to control participants, two reported dampened N170 responses in ADHD participants. Specifically, Williams et al. (2008) detected increased N170 amplitude (i.e., more negative) in response to the face stimuli among adolescents with ADHD relative to their TD peers. However, Ibáñez et al. (2014) reported reduced N170 amplitude to faces among several clinical groups—including ADHD participants—relative to neurotypical controls. Further, using traditional localised ERP analytic techniques, Meier et al. (2012) detected a significant attenuation of overall N170 responses in the patient group, but only when comparing a group of "delinquent" ADHD participants to healthy controls; no difference was detected

Figure 1
PRISMA Flowchart for the Systematic Review



Note. PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

between controls and a "nondelinquent" ADHD group. Within the same article by Meier et al. (2012), when data were instead analysed by way of global field power across all recording electrodes in the N170 epoch, both "delinquent" and "nondelinquent" ADHD groups demonstrated reduced N170 amplitude relative to controls.

Ultimately, there seems to be no systematic pattern of results in terms of overall group differences in N170 amplitude or latency between ADHD and controls. While the majority of studies (10 out of 13) demonstrated no overall group difference in N170 responses to faces, others did detect group differences but were inconsistent in the directionality of the effect such that two articles reported diminished N170 responses in ADHD patients, whereas one article reported the opposite.

In relation to our two accounts for social deficits in ADHD, this initial, broad look at the literature generally supports the consequential social deficit account due to the lack of consistent group-level differences in N170 responses. The inherent social deficit account on the other hand receives little initial support from this overview of the literature insofar as differences in overall face processing are concerned. The takeaway for this portion of the review should be twofold: (1) there are no obvious group-level differences between ADHD and controls in terms of overall face processing, and (2) point one is moot if we do not drill down further. Subtle nuances of participant age, hemispheric localization of the ERPs, encoding task demands, and stimuli valence may all indeed play critical roles in moderating N170 responses. Subtle nuances of participant age, hemispheric localization of the ERPs, encoding task demands, and stimuli valence may all indeed play critical roles in moderating N170 responses. With the possibility of moderators in mind, we now turn to participant age as the first potential factor that could influence how the N170 manifests in ADHD participants across their lifespan.

Effects of Sample Age

As a next step in the review, studies were divided by sample age (i.e., youth under 18 years of age vs. adults over 18 years of age) and results were reexamined separately to determine whether ADHD and TD participants might display differing patterns of N170

responses at certain stages of development but not at others. Age was selected as a moderator of interest because both ADHD symptom presentation and the N170 ERP response display well-established age-related changes. Specifically, ADHD symptoms in general—and hyperactivity subtypes in particular—typically decrease with advancing age, while the N170 ERP increases in amplitude and decreases in latency over the lifespan (Döpfner et al., 2015; Taylor et al., 1999). Thus, it seems reasonable that the variability in sample age across studies in this review may contribute to the inconsistent effects detailed above. Table 1 presents demographic characteristics of participant samples within the reviewed studies, including the average age of participants.

Among studies that collected youth samples (<18 years of age), two reported significant group differences in N170 responses between ADHD and control participants (Karalunas et al., 2020; Williams et al., 2008), whereas six found no main effect of ADHD status on N170 amplitude (Alperin et al., 2017; Flegenheimer et al., 2018; Groom et al., 2017; Tye et al., 2013, 2014; Zhao et al., 2020), once again providing inconsistent results. Within adult samples (≥18 years of age), similar inconsistencies arose. Ibáñez et al. (2014) identified group differences in N170 amplitude between ADHD participants and controls when using an adult sample, while other studies (even from the same researchers) did not (Ibáñez et al., 2011; Raz & Dan, 2015b).

Taken together, no systematic differences were identified based on age of participant samples, suggesting this factor does not moderate N170 responses in ADHD. This pattern of findings is in line with the consequential account in that no systematic deficits in face processing are evident at the neural level in ADHD, even when age groups are considered independently. Overall, there does not seem to be a particular developmental period wherein ADHD participants demonstrate consistent differences in N170 amplitude from TD controls.

Hemispheric Lateralization

Hemispheric lateralization of the N170 ERP is one of the most consistent patterns of results observed for the signal in neurotypical populations. Normally, N170 amplitude is much more pronounced in the right brain hemisphere relative to the left (e.g., Dundas et al., 2014; Itier & Taylor, 2002; Sagiv & Bentin, 2001; Taylor, Edmonds, et al., 2001; for reviews see Chung & Thomson, 1995; Taylor, Itier, et al., 2001). Furthermore, this hemispheric asymmetry is thought to be consistent across the lifespan (Chung & Thomson, 1995; A. Young, 1983; A. W. Young et al., 1985; cf. Taylor, Itier, et al., 2001).

Divergence from this traditional pattern of right-dominant lateralization characterises other neurodevelopmental disorders. For instance, patients with ASD often display diffused N170 amplitude across both hemispheres, rather than the right-side dominant electrophysiological responses that are observed in neurotypicals (e.g., Jemel et al., 2006). Because our current question concerns atypical sociocognitive functioning in those with ADHD, right-dominant hemispheric lateralization of the N170 ERP may serve as a key indicator of normal face processing in this patient group.

Of the 14 reviewed articles, normal right-dominant hemispheric lateralization occurred in the ADHD groups of three studies (Ibáñez et al., 2011; Tye et al., 2013, 2014), whereas only one article reported diffused hemispheric responses (Alperin et al., 2017). The remaining 10 articles did not provide enough statistical information to assess the interaction, or used incompatible methodologies (e.g., global field power analyses) such that we could not determine clearly if there were any group by hemisphere interactions present (Flegenheimer et al., 2018; Groom et al., 2017; Ibáñez et al., 2014; Karalunas et al., 2020; Meier et al., 2012; Raz & Dan, 2015a, 2015b; Rinke et al., 2017; Williams et al., 2008; Zhao et al., 2020).

Ibáñez et al. (2011) reported no significant difference in hemispheric lateralization between their ADHD and neurotypical control groups. Meaning, both groups demonstrated greater N170 responses in the right hemisphere. Tye et al. (2014) found a similar effect whereby there was a statistically marginal trend such that all N170 responses were higher in the right than the left hemisphere, but there was no indication of this effect differing between ADHD and control groups. While Tye et al. (2013) did observe a group by

Table 1
Demographic Characteristics of Studies Addressing N170 Responses in ADHD

	Experimental group demographics							
	ADHD				Typically developing controls			
Article citation	*N*	% male	Age *M* (*SD*)	% right-handed	*N*	% male	Age *M* (*SD*)	% right-handed
Alperin et al. (2017)	49	85.71	13.70 (1.48)		60	63.33	13.87 (1.08)	
Flegenheimer et al. (2018)	19	73.68	6.49 (0.79)[a]		28	64.29	6.49 (0.79)[a]	
Groom et al. (2017)	12	53	11.94 (2.35)		20	80	12.58 (1.92)	
Ibáñez et al. (2011)	10	90	33.10 (3.60)	70	10	90	33.00 (3.80)	80
Ibáñez et al. (2014)	16	87.5	34.60 (11.1)	100	41	63.41	38.30 (11.40)	97.56
Karalunas et al. (2020)	61	83.3	13.9 (1.50)		69	58.8	13.8 (1.10)	
Meier et al. (2012)	13	100	31.30 (9.73)		13	100	28.50 (5.49)	
Raz and Dan (2015a)	17	17.65	24.07 (1.73)		20	30	24.52 (2.87)	
Raz and Dan (2015b)	21	23.8	25.42 (2.11)		19	21.05	24.72 (2.72)	
Rinke et al. (2017)	29	82.76	12.09 (2.76)	82.76	21	42.86	12.08 (3.00)	90.48
Tye et al. (2013)[b]	18	100	10.48 (1.91)	94.44	26	100	10.56 (1.79)	88.46
Tye et al. (2014)[b]	18	100	10.48 (1.91)	94.44	26	100	10.56 (1.79)	88.46
Williams et al. (2008)[c]	51	100	13.79 (2.33)		51	100	13.09 (2.39)	
Zhao et al. (2020)	29	79	8.48 (3.50)		29	62	8.22 (3.40)	

Note. Blank spaces indicate factors that were not reported in the original article. ADHD = attention-deficit hyperactivity disorder.
[a] Mean age of sample not reported separately by group. [b] Tye et al. (2013) and Tye et al. (2014) used the same participant sample. [c] Study employed a pre–post design; we only present data from preintervention.

hemisphere interaction of N170 amplitude, their study included three groups: ADHD patients, controls, and ASD patients, the latter of which appeared to be driving the interaction effect due to their diffused N170 responses across both hemispheres.

Alperin et al. (2017) was the sole article that reported a significant group by hemisphere interaction of the N170 response, such that the ADHD group demonstrated diffused N170 ERP responses across both hemispheres. However, this result should be interpreted with caution as the control group in their study demonstrated an abnormal pattern of N170 lateralization as well, such that the signal was actually strongest in the *left* hemisphere. Therefore, the unique pattern of N170 activation observed in Alperin et al. (2017) may be resultant of some unidentified experimental factor that would cause a deviation from the typical lateralized neural response to face stimuli.

Hemispheric lateralization of the N170 is undoubtedly a key indicator of neurotypical function. In related patient groups such as those with ASD, face processing is thought to follow an abnormal pattern such that the signal is diffused across both hemispheres. In the case of face processing in ADHD, the story is less clear. From the four articles that present sufficient statistical or descriptive information to draw conclusions about hemispheric asymmetry of the N170 in ADHD, three studies support the notion that individuals with ADHD follow the typical pattern of results, while one reports contrasting results. Overall, the evidence available right now does tentatively suggest that ADHD patients elicit typical N170 hemispheric lateralization. The immediate implication is weakened support for an inherent account of social cognition deficits in ADHD populations. The observed typical lateralization of N170 responses in ADHD does serves as another neural indicator that face processing—representing a critical aspect of social cognition—is intact in ADHD.

Effects of Cognitive Load

Although face processing is a quick and highly automatic process, the N170 appears to remain susceptible to attentional modulation under certain task demands (Aranda et al., 2010). A number of studies have identified that the amplitude of the N170 response to faces can be modulated by characteristics of the task being done while ERPs are measured (e.g., Morgan et al., 2008). Specifically, it has been demonstrated that cognitive load—or the amount of working memory resources required for a certain task—is an important consideration when looking at neural correlates of early visual processing (Biehl et al., 2013). Because a review of this type necessitates aggregating data collected while participants complete tasks inducing different levels of cognitive load, as a next step we examine whether differences in task demands between studies differentially affects the strength of the N170 signal in participants with and without ADHD.

In order to address this question, we reviewed whether differences in task demands across studies accounted for the aforementioned inconsistency of results. To do so, we used task descriptions provided by each study to determine whether the task performed by participants while the N170 was measured was a "low load" (defined as a task requiring few working memory resources; e.g., passive face viewing) or "high load" task (defined as a task requiring intense working memory resources; e.g., N-back). We then compared results from studies using low and high load tasks to examine whether this categorization moderated the relationship between ADHD status and N170 integrity.

Of the 14 studies considered in the current review, eight were deemed to use "high load" tasks (Alperin et al., 2017; Groom et al., 2017; Karalunas et al., 2020; Meier et al., 2012; Raz & Dan, 2015a, 2015b; Rinke et al., 2017; Zhao et al., 2020). The specific tasks varied between experiment but included tasks such as go/no-go tasks, oddball tasks, N-back tasks, and continuous performance tasks. While the nature of each of these tasks is somewhat different, they all place demands on the executive function skills of participants and thus require high levels of cognitive load. After these studies were identified as the "high load" subset, results from each article were examined in order to determine whether they reported a significant main effect of group (i.e., ADHD vs. TD) on N170 amplitude to face stimuli.

Of these eight high load studies, the vast majority (six out of eight) did not identify differences in N170 amplitude between ADHD and TD participants. That is, most studies employing high cognitive load tasks found no statistical difference in N170 amplitude to faces between participants with ADHD and TD controls. One article (Karalunas et al., 2020) reported an interaction between group and emotional valence of the face presented as part of the task such that TD participants displayed similar N170 amplitude to all faces, but participants with ADHD showed reduced N170 amplitude to positive relative to neutral faces. However, this study does not report statistics on the main effect of diagnostic group, so it is thus undetermined whether ADHD status influences N170 amplitude independently in this particular case. Another study in the "high load" group (Meier et al., 2012) found that participants with ADHD did exhibit reduced N170 amplitude to faces relative to controls while completing a go/no-go task, but this effect was isolated to the subset of participants with ADHD with "delinquent" traits and did not extend to ADHD participants without such traits. Overall, the majority of studies identified no differences between ADHD and TD participants' N170 amplitude in response to faces while participants completed highly demanding tasks.

Next, six of the 14 articles were identified as using "low load" tasks that did not demand high levels of cognitive load or use of executive functions while participants completed them (Flegenheimer et al., 2018; Ibáñez et al., 2011, 2014; Tye et al., 2013, 2014; Williams et al., 2008). These tasks included passive face viewing, emotion recognition tasks, and dual valence tasks. After reviewing results of these articles in accordance with the process described above, it was determined that four of the six studies found no significant difference in N170 amplitude between ADHD and TD participants.

Work by Williams et al. (2008) found *higher* N170 amplitude among ADHD participants relative to TD controls during an emotion recognition task. On the other hand, Ibáñez et al. (2014) found *reduced* N170 amplitude in ADHD relative to TD participants while completing a dual valence classification task. Taken together, a majority of studies employing tasks with low cognitive demands found no differences in N170 amplitude to faces between participants with ADHD and TD controls. While two studies did identify group differences, they reported effects in opposite directions to one another.

In summary, dividing studies by task difficulty did not reveal systematic differences in the integrity of the N170 response to faces between participants with and without ADHD. While some studies using more cognitively demanding tasks did identify N170 alterations in ADHD participants (e.g., Meier et al., 2012), others using similarly demanding tasks did not (e.g., Raz & Dan, 2015b). Moreover, inconsistencies in N170 amplitude also emerged during

low load tasks, although again the majority of studies identified no group differences.

Effects of Emotional Valence

Humans are inherently social creatures, with processing of social cues serving as one of the most critical aspects of our day-to-day interactions. While faces serve as a fundamental social cue to interpret, they are often much more dynamic than the static black and white images of neutral faces that researchers frequently display on computer screens when studying face processing. Due to the immense availability of social cues that can be gleaned from faces, humans have become highly accurate at interpreting even the most minor changes in facial emotional valence (Durand et al., 2007; Orgeta & Phillips, 2007). For our major research question then, it is of paramount importance to consider how other aspects of social cognition, such as emotion perception, may interact with face processing in providing a more nuanced view of how sociocognitive processes unfold at the neural level in ADHD.

Although the N170 is consistently and clearly linked with early face processing (Itier & Taylor, 2004), more recent debate has surrounded its relevance for the detection of emotional facial expressions (Hinojosa et al., 2015). One model of face recognition proposed by Bruce and Young (1986) suggests that there are two steps involved in face processing: structural encoding of facial features, and later processing of identity and emotional expression. In accordance with this model, if the N170 only reflects processing of low-level facial features (i.e., step one in Bruce and Young's model), then it should not be modulated by emotional expression (i.e., Step 2). However, more recent evidence has suggested that processing of facial features can inform emotion processing at very early stages, indicating a possible role for emotion in the N170 response (Martens et al., 2010). For instance, neurotypical participants often display an increase in N170 response for threat-related emotional expressions such as fearful (e.g., Blau et al., 2007) or angry faces (e.g., Bediou et al., 2009) relative to neutral expressions. Overall, while debate persists over whether the N170 is modulated by emotion, a recent meta-analysis supported the notion that the N170 can indeed be modulated by emotional facial expressions (Hinojosa et al., 2015).

According to a consequential account of social deficits in ADHD, there should be no difference whatsoever between control and ADHD groups in terms of sociocognitive processes such as those marked by the N170 face response. How might we then assess whether the N170 is modulated by emotion in ADHD? Well, it has been hypothesised that ADHD is characterised by a deficit in threat-related emotion processing (e.g., Manassis et al., 2007). Evidence of reduced amygdala volume in ADHD patients—a brain area known to modulate processing of threat cues—is consistent with the notion of threat-related information processing deficits in this group (Frodl et al., 2010; Hoogman et al., 2017; Vuilleumier, 2015; Vuilleumier & Driver, 2007). Therefore, one effective way to evaluate whether sociocognitive processes are impacted in ADHD is by looking for differential ERP responses to threat-related face stimuli. Table 2 offers an overview of N170 amplitude to various emotional face stimuli in the reviewed studies, including articles that used threat-related face stimuli.

Much like in the previous sections of this review, we chose to assess atypicality of facial expression processing in ADHD by comparing each patient group to their respective neurotypical control groups (i.e., the critical result is a group by emotion interaction within an experiment), rather than compare the reported N170 response of ADHD groups to that of the broader literature (i.e., comparing them to the notion that the N170 response should be greater for threat-related stimuli). Of the 14 reviewed articles, five studies identified differential N170 responses in the ADHD group based on emotional valence of face stimuli (Alperin et al., 2017; Flegenheimer et al., 2018; Ibáñez et al., 2011; Raz & Dan, 2015a; Williams et al., 2008); four studies found no modulation by emotion (Ibáñez et al., 2014; Karalunas et al., 2020; Rinke et al., 2017; Tye et al., 2014); two studies were unclear from the provided statistics (Meier et al., 2012; Raz & Dan, 2015b), and finally three studies did not investigate emotional facial expressions in their studies at all (Groom et al., 2017; Tye et al., 2013; Zhao et al., 2020).

While a wide variety of emotional expressions have been employed within the N170 ADHD literature (neutral, anger, happiness, sadness, disgust, fear, joy, and surprise), we chose to restrict our current discussion to only those that are considered threat-related (anger and fear), in comparison to those that are neutral or positive (happiness). With a threat-related hypothesis and related evaluative criteria in-hand, we can examine whether ADHD participants tend to elicit a neurotypical threat-related boost in N170 amplitude.

Of the five articles that reported emotion by group interactions of the N170, results were split such that three studies reported further *enhanced* threat-related > neutral N170 responses in ADHD relative to the TD group (Ibáñez et al., 2011; Raz & Dan, 2015a; Williams et al., 2008), whereas two studies showed significant atypical *reductions* in N170 responses to threat-related stimuli in ADHD patients relative to the TD group (Alperin et al., 2017; Flegenheimer et al., 2018). Finally, it is worth remembering that an additional three studies did employ emotional face stimuli (including threat-related emotional faces), yet reported no interaction of group and emotion whatsoever (Ibáñez et al., 2014; Karalunas et al., 2020; Rinke et al., 2017; Tye et al., 2014), which could lend evidence to the notion that emotional face processing is indeed normal in ADHD.

In summary, four studies reported no differential modulation of threat-related emotion processing in ADHD as measured by the N170, whereas five did. However, of the five articles that reported interactions, three suggested there is enhanced processing of threat-related stimuli in ADHD that goes beyond the usual N170 enhancement seen in TD individuals. Two other studies suggested that N170 responses to threat-related faces could instead be attenuated in ADHD populations. Thus, some studies may point toward differential emotion processing in ADHD as measured by the N170, but the fact that the *direction* of these differences are inconsistent may be indicative of fluctuating statistical power or an unidentified moderating factor. Nonetheless, that many studies reviewed here indicate some sort of differential N170 response to emotional stimuli provides support for an inherent social deficit account. That is, the evidence indicates that those with ADHD may have atypical processing of emotion which may lead to worsened social outcomes, rather than the hallmark symptoms of ADHD themselves (e.g., hyperactivity) affecting social-cognitive development.

General Discussion

ADHD is characterised by a core set of symptoms that includes hyperactivity, inattention, and impulsivity. Individuals with this disorder often experience diminished social outcomes relative to their

Table 2
Effects of Emotional Face Modulation on N170 Responses in ADHD

Article citation	Behavioural task when viewing faces	Valence of faces	Effect of face valence on N170 Amplitude within ADHD groups			
			Neutral	Happy	Angry	Fear
Alperin et al. (2017)	Emotional go/no-go	N, H, F	>H, F	<N		<N
Flegenheimer et al. (2018)	Passive face viewing	N, A, H, F, Sa, Su	>F	=N	=N	<N
Groom et al. (2017)	Visuospatial attention cueing task	N	=N in TD			
Ibáñez et al. (2011)	Dual valence classification task	A, H		=A (<H in TD)	=H	
Ibáñez et al. (2014)	Dual valence classification task	A, H		<H in TD[a]	<A in TD[a]	
Karalunas et al. (2020)	Emotional go/no-go	N, H, F	=F, >H	<N		=N
Meier et al. (2012)	Modified visual emotional go/no-go task	N, A, H	<N in TD[a,b]	<H in TD[a,b]	<A in TD[a,b]	
Raz and Dan (2015a)	Visual-emotional oddball task	N, A, H	—	<A	>H	
Raz and Dan (2015b)	Visual-emotional oddball task	N, A, H	=A, H	=A, N	=H, N	
Rinke et al. (2017)	Emotional continuous performance test	N, A, H	=A, H			
Tye et al. (2013)[c]	Passive viewing	N	=N in TD[a]			
Tye et al. (2014)[c]	Passive viewing	N, A, F, D, J	=F		—	=N
Williams et al. (2008)[d]	Emotion recognition	N, A, H, F, Sa, D	—	—	>A in TD	>F in TD
Zhao et al. (2020)	Face recognition (S1–S2 paradigm)	N	=N in TD			

Note. All contrasts are within the examined ADHD groups, except when specified as a between-group comparison to the control group ("in TD"). Blank spaces indicate factors that were not mentioned in the original article. Dashes (—) indicate factors that were included in the original article but not discussed in the results. TD = typically developing; N = neutral; A = angry; H = happy; F = fear; Sa = sadness; Su = surprise; J = joy; D = disgust; ADHD = attention-deficit hyperactivity disorder.
[a] Main effect reported but specific contrasts by emotion were not provided in the original article. [b] Both the ADHD and ADHD-delinquent groups differed from controls when analysing used global field potentials. [c] Same participant sample used in both studies. [d] Study employed a pre–post design; we only present data from preintervention.

peers. While much work has been done to investigate these social deficits in ADHD, questions remain as to how these attenuations come about. In this review, we offer two viable competing accounts in an attempt to summarise the work done to-date and offer insight into where the evidence stands in relation to these two accounts.

The consequential social deficit account posits that impoverished social outcomes in ADHD are simply a consequence of poor social interactions and learning stemming from inattentive behaviours (or other factors like reduced working memory capacity) that are hallmark symptoms of the disorder. In other words, individuals with ADHD may find it hard to pay attention or inhibit impulsive behaviours when in highly demanding situations—like interacting with others in the classroom—leading to poor interactions with their peers. The inherent social deficit account on the other hand contends that individuals with ADHD suffer from atypical sociocognitive functioning in facets like face processing, all of which are separable from definitional symptoms of the disorder. That is, those with ADHD experience diminished social outcomes because of their atypical sociocognitive functioning, rather than downstream consequences stemming from inattention, hyperactivity, etc.

After filtering through hundreds of articles on the subject, we narrowed down our data set to 14 highly similar reports. Each study in our qualitative analysis included an ADHD group and a control group, used facial stimuli, and recorded N170 activity during one or more tasks. By comparing these 14 studies, we provide an overview of the literature as it stands today. Moderating factors such as participant age, hemispheric lateralization, cognitive load, and emotional valence of the face stimuli were considered in this endeavour.

Ultimately, no consistent evidence was found to support either of the two accounts. Because most of the moderating factors we considered would be hypothesised to alter the N170 in ADHD if the inherent social deficit account is correct, one might conclude that the lack of consistent group differences instead offers support for a consequential account. Of course, absence of evidence is not evidence of absence, and there are a few reasons why the studies we reviewed here may have provided inconsistent findings.

A Path Toward Progress

Over a decade ago, Uekermann et al. (2010) identified the need for further research into the nature of social cognition in ADHD. Since that time, several articles have investigated the integrity of the N170 in ADHD patients; however, the current review makes very clear the fact that questions still remain. Specifically, this review underscores the need for future investigations exploring N170 integrity in ADHD. The architects of any such study would be wise to consider a priori analyses of statistical power, adoption of best practices in EEG research, and harmonised methods/data reporting.

Sample Size Calculations

Several of the studies reviewed here employed relatively small samples. For example, Ibáñez et al. (2011) have only 10 participants in each group, while seven additional studies each present data from less than 20 ADHD participants (Flegenheimer et al., 2018; Groom et al., 2017; Ibáñez et al., 2014; Meier et al., 2012; Raz & Dan, 2015a; Tye et al., 2013, 2014). Group differences in N170 response may be subtle, and between-subjects studies such as these may be underpowered to detect statistically significant differences even if true effects exist.

Performing an a priori power analysis is a common and effective way to ensure sufficient statistical power for a given design (Perugini et al., 2018), yet only two of the 14 studies reviewed here reported a formal power analysis (Flegenheimer et al., 2018; Groom et al., 2017). Free software such as GPower (Faul et al., 2007) can be used for sample size determination in simple research designs, whereas

more complex designs can take advantage of simulated power analyses offered by the Superpower package for *R* (or its more user-friendly web-based Shiny app; Lakens & Caldwell, 2021).

Another effective way to gain higher power is via a multisite registered report ("many-labs" reports). These collaborative efforts take advantage of vast sample sizes, harmonised data collection, and preregistered statistical analyses to perform conclusive investigations. They have been successful in cognitive psychology (e.g., Morey et al., 2022), social psychology (e.g., Hagger et al., 2016) and have recently been launched in the realm of EEG research as well (the "EEGManyLabs" project; Pavlov et al., 2021).

One further way to mitigate issues of statistical power is by turning to contemporary single-subject ERP analyses. There are now numerous methods available for single-subject analyses of ERP data (Amin et al., 2023; Kallionpää et al., 2019; Zhang et al., 2023), but the basic objective is to treat the effect size of the difference between two conditions (e.g., viewing faces vs. houses) in an individual as the dependent measure of interest before comparing this metric across groups (e.g., ADHD vs. neurotypical). Doing so cuts down on extraneous statistical noise and can result in higher statistical power.

It is worth mentioning that limitations in sample size are to be expected in the present case. Working with special population groups poses many difficulties, one of which is simply finding enough participants. Even then, the use of electrode caps and EEG systems may be frightening to some volunteers which could deter participation. Therefore, no acrimony is intended in this review; the organic perils of working with patients and neuroimaging techniques pose substantial barriers to conducting high-powered, easily replicable studies and are not to be taken lightly.

Implementation of Best Practices in EEG Research

Beyond subject-level statistical power, noisy data are a well-known complication of EEG (Cohen, 2017; Mikhail et al., 2010) and can further exacerbate issues due to data attrition and increased trial-level variance. We therefore recommend a broad adoption of "best practices" in EEG research, many of which have been honed in recent years. Niso et al. (2022) provide an excellent, up-to-date review of this subject in the realm of EEG research specifically, offering a pipeline of best practices beginning with preregistration and extending through statistical analyses.

Aside from experimental practices, steps can be taken to improve replicability at the writing stage as well: Consistency in reporting between studies will facilitate later meta-analyses that will surely follow once there is sufficient data in this area. For instance, when reporting N170 amplitude it is important to mention whether the reported values represent mean amplitude or peak amplitude. Similarly for latency, one must specify how the measure was defined (e.g., beginning with stimulus onset/offset, at peak amplitude, or a percent-area latency measure; see Liesefeld, 2018). Clarifying measures such as these provides just one actionable step that can be taken to increase replicability in this area and benefit future meta-analysts. Similar guidelines for best practices have been outlined in related fields recently as well, such as using EEG to study those with autism (Webb et al., 2015).

Moving forward, harmonisation of methods and data reporting across studies, as well as increases in statistical power and/or effect sizes will help determine whether face processing is disrupted at the neural level in those with ADHD. Additionally, combining N170 ERP studies with results from other neuroimaging methods (e.g., functional magnetic resonance imaging, magnetoencephalography) in search of converging evidence will help bridge the gap in order to further our understanding of the nature of social functioning in ADHD and address areas in which EEG research is currently limited.

Limitations and Implications of the Current Review

While we hope to speak to social cognition in those with ADHD more generally, we recognise that face processing is only one related facet (Arioli et al., 2018). That is, when trying to discern whether atypical sociocognitive functioning exists in ADHD, face processing is just one marker that we can assess for irregularity. Therefore, while the current review is narrow enough to highlight face processing in detail, broader conclusions regarding sociocognitive deficits in ADHD are tentative. For example, it could very well be the case that face processing is intact in ADHD, yet theory of mind is impaired—something that is not detectable by the N170 ERP. In recent review, Dan (2020) claim that ADHD patients perform similarly to their neurotypical peers on emotion discrimination tasks, yet their neural activity (as measured by functional magnetic resonance imaging and EEG) still differ. Thus, it is possible that there are different, or perhaps compensatory mechanisms at play that lead to distinct sociocognitive processing that can be detected with modern neuroimaging methods, even in individuals that do not exhibit attenuated performance on social tasks.

While we presented two broad accounts based in consequential and inherent social functioning deficits, it is worth highlighting that the former is much more susceptible to impacts from confounding variables. Because the consequential social deficit account rests entirely on the notion that impoverished social outcomes in ADHD are resultant from core ADHD symptoms leading to poor social interactions at a young age, it stands to reason that individual differences can impact social functioning tremendously. Factors such as participant gender, ADHD subtype, and severity of core symptomology can all have downstream consequences on social standing. For instance, males with ADHD are perceived by the public and school teachers to be more likely to exhibit behavioural problems than their female ADHD patient counterparts (Quinn & Wigal, 2004). In addition, females with ADHD are thought to exhibit inattention symptoms rather than the more typical hyperactivity subtype seen in males (Quinn & Wigal, 2004), and this mismatch in behaviour could be a leading factor contributing to underdiagnosing of ADHD in women (S. Young et al., 2020). The public's perception of ADHD and the associated stigmas surrounding its manifestation could lead to differences in the supports available and peoples' willingness to accommodate abnormal behaviours. Although sex differences, age, and symptom presentation considerations are not exclusive to the consequential social deficit account (as they are also potentially linked to sociocognitive functions), it is essential to recognise that these factors in particular may significantly influence social development due to varying societal expectations. Therefore, they should be a central focus of future research in this domain.

The broad relevance of these research questions is perhaps most obvious when considering the immediate clinical implications of this review. Specifically, while the need for efficacious interventions to address social deficits in ADHD is clear, evidence-based

treatments to remedy social deficits have yet to be developed. In a recent meta-analysis of 45 studies examining social skills training in ADHD, Storebø et al. (2019) found no reliable evidence to support that such training programs are efficacious.

While it is unclear exactly why social skills interventions work so poorly for those with ADHD, it is widely believed that the lack of understanding of the nature behind social deficits limits the efficacy of interventions because they are not designed in a targeted fashion to address the core mechanisms driving social impairments. Thus, identifying the precise locus of social deficits in ADHD is arguably a *prerequisite* for designing effective interventions. The present review demonstrates that face processing is one foundational social facet where confusion remains about whether deficits in those with ADHD exist. Despite some limited evidence that individuals with ADHD may demonstrate atypical face processing at the neural level (e.g., in response to emotional faces), the majority of the literature reviewed here did not find differences in face processing between people with ADHD and their TD peers. Our review suggests that interventions targeted to address shortcomings of face processing in ADHD would not be warranted until further evidence demonstrates a need to compensate for a deficit in this area.

Conclusion

This review began by outlining two possible accounts that could explain social decrements in ADHD. The consequential account argues that poor socialisation is a result of hallmark ADHD symptoms, while the inherent account contends that diminished social outcomes stem from sociocognitive impairments that are separable from these hallmark symptoms. After reviewing 14 studies, we failed to find any consistent evidence to support either of these accounts. The variation of N170 responses between groups was not reliably affected by participant age, hemispheric lateralization, cognitive load, or emotional valence of the face stimuli. It is likely that these inconsistent findings are due to low statistical power and high variation in experimental methodology. Nonetheless this area of research offers a fruitful avenue to investigate social functioning in ADHD and should continue to be improved upon. We suggest a wide adoption of best practices in this area, including sample size calculations, preregistered neuroimaging pipelines, and further consideration of individual differences and societal impacts. In the meantime, caution is advised when designing ADHD interventions that aim to remedy deficits in social processing separate from core ADHD symptomology. Fortunately, our understanding of social cognition and the techniques used to measure it are rapidly improving which will facilitate identification of the root cause of social deficits in ADHD and development of more effective interventions.

Résumé

Le trouble du déficit de l'attention avec hyperactivité (TDAH) est associé à des déficits de fonctionnement social, notamment à des difficultés avec les pairs et à une mauvaise qualité des relations. Cependant, l'on sait peu de choses sur l'intégrité des capacités sociocognitives fondamentales qui soutiennent les interactions interpersonnelles dans le TDAH. Le traitement des visages, une composante fondamentale de la cognition sociale, a fait l'objet de recherches récentes dans ce domaine. Des chercheurs ont tenté de définir les mécanismes de traitement des visages dans le TDAH afin d'élucider les déficits sociaux souvent observés dans ce trouble. L'étude du potentiel lié à l'événement N170, un marqueur neuronal du traitement des visages, a été une approche populaire dans cette initiative. Nous présentons ici deux comptes rendus qui offrent des vues divergentes sur la façon dont les déficits sociaux peuvent survenir chez les personnes atteintes de TDAH. Ensuite, nous examinons et résumons systématiquement la littérature sur le N170 dans le TDAH afin d'identifier si des atypies dans les domaines sociocognitifs tels que le traitement des visages se produisent dans cette population de patients. Des lacunes de la littérature sont identifiées et des solutions concrètes sont proposées pour améliorer la recherche future dans ce domaine. Nous terminons en discutant des implications immédiates pour les approches thérapeutiques conçues pour traiter les déficits sociaux largement observés chez les personnes atteintes de TDAH.

Mots-clés : trouble du déficit de l'attention avec hyperactivité, traitement des visages, potentiel lié à l'événement N170, cognition sociale, examen systématique

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