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Alpha oscillatory activity during attentional control in children with Autism Spectrum Disorder (ASD), Attention-Deficit/Hyperactivity Disorder (ADHD), and ASD+ADHD

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Background: Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) share impairments in top-down and bottom-up modulation of attention. However, it is not yet well understood if co-occurrence of ASD and ADHD reflects a distinct or additive profile of attention deficits. We aimed to characterise alpha oscillatory activity (stimulus-locked alpha desynchronisation and prestimulus alpha) as an index of integration of top-down and bottom-up attentional processes in ASD and ADHD. **Methods:** Children with ASD, ADHD, comorbid ASD+ADHD, and typically-developing children completed a fixed-choice reaction-time task ('Fast task') while neurophysiological activity was recorded. Outcome measures were derived from source-decomposed neurophysiological data. Main measures of interest were prestimulus alpha power and alpha desynchronisation (difference between poststimulus and prestimulus alpha). Poststimulus activity linked to attention allocation (P1, P3), attentional control (N2), and cognitive control (theta synchronisation, 100–600 ms) was also examined. ANOVA was used to test differences across diagnostics groups on these measures. Spearman's correlations were used to investigate the relationship between attentional control processes (alpha oscillations), central executive functions (theta synchronisation), early visual processing (P1), and behavioural performance. **Results:** Children with ADHD (ADHD and ASD+ADHD) showed attenuated alpha desynchronisation, indicating poor integration of top-down and bottom-up attentional processes. Children with ADHD showed reduced N2 and P3 amplitudes, while children with ASD (ASD and ASD+ADHD) showed greater N2 amplitude, indicating atypical attentional control and attention allocation across ASD and ADHD. In the ASD group, prestimulus alpha and theta synchronisation were negatively correlated, and alpha desynchronisation and theta synchronisation were positively correlated, suggesting an atypical association between attentional control processes and executive functions. **Conclusions:** ASD and ADHD are associated with disorder-specific impairments, while children with ASD+ADHD overall presented an additive profile with attentional deficits of both disorders. Importantly, these findings may inform the improvement of transdiagnostic procedures and optimisation of personalised intervention approaches. **Keywords:** Autism Spectrum Disorder; ADHD; attention; comorbidity.

Introduction

Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) are two of the most common neurodevelopmental disorders. ASD is characterised by impaired communication and social interaction, as well as restricted interests and repetitive patterns of behaviour, whereas ADHD is defined by persistent patterns of inattention, hyperactivity, and impulsivity (*Diagnostic and Statistical Manual of Mental Disorders 5th Ed.*, 2013). These disorders frequently co-occur (Grzadzinski, Dick, Lord, & Bishop, 2016; Simonoff et al., 2008) and it has been shown that they share some behavioural features, neurocognitive impairments, and genetic mechanisms (Rommelse, Geurts, Franke, Buitelaar, & Hartman, 2011; Ronald, Happé, & Plomin, 2008; Van Der Meer et al., 2012). However, it is not well understood if co-occurrence of ASD and ADHD reflects a distinct clinical condition or an additive comorbidity with unique deficits of both disorders (Johnson, Gliga, Jones, & Charman, 2015; Taurines et al., 2012). One way to investigate this question is to characterise overlapping and distinct neurocognitive atypicalities in ASD and ADHD, as well as their manifestation in individuals with a diagnosis of both ASD+ADHD. Importantly, this can help improve the stratification of ASD and ADHD in diagnostic procedures, and optimise the selection of personalised intervention strategies (Jeste, Frohlich, & Loo, 2015).

ASD and ADHD share impairments across a variety of cognitive domains, including attention

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(Rommelse et al., 2011). Posner and Petersen (1990) originally conceptualised attention as three sets of cognitive processes: an alerting system, responsible for the production and maintenance of optimal vigilance states; an orienting system, responsible for attending to different sensory input exogenously (involuntarily) and endogenously (voluntarily); and an executive control system, responsible for monitoring and resolving conflict. This model provides a useful framework to identify differences and commonalities in attention modulation between ASD and ADHD. For instance, previous work has shown that ASD is characterised by impairments in the exogenous orienting system (Renner, Grofer Klinger, & Klinger, 2006; but see Grubb et al., 2013; Pruett et al., 2011 for conflicting findings) and in the executive control system, with mixed evidence for the alerting system (Keehn, Müller, & Townsend, 2013). Instead, ADHD is characterised by deficits in the alerting and executive control systems (Mullane, Corkum, Klein, McLaughlin, & Lawrence, 2011), with some evidence showing impairments in endogenous orienting (Caldani et al., 2020; but see Roberts, Ashinoff, Castellanos, & Carrasco, 2018 for conflicting findings). However, while such attention deficits have been widely investigated in ASD and ADHD separately (Keehn et al., 2013; Schoechlin & Engel, 2005), to date only a few studies include a direct comparison of both groups or a separate comorbid group.

For instance, based on the same cohort used in this study, Tye et al. (2016) examined attentional impairments in children with ASD, ADHD, and ASD+ADHD (compared with typically developing children, TD) by measuring their reaction times during a fixed choice reaction time task ('Fast task'; Cheung et al., 2016; Kuntsi, Andreou, Ma, Börger, & van der Meere, 2005). In line with previous research (Karalunas, Geurts, Konrad, Bender, & Nigg, 2014), they found that responses of children with ADHD (ADHD and ASD+ADHD) were slower and more variable than those of ASD-only and TD children. The finding that children with ADHD (ADHD and ASD+ADHD) show more variable responses is particularly relevant, since intraindividual variability in reaction time is thought to reflect suboptimal arousal and attention lapses (Karalunas et al., 2014).

In another study, Tye et al. (2014) used a flankered cued-continuous performance task (CPT-OX; McLoughlin et al., 2011) to examine event-related potential (ERP) markers of attentional control. Consistent with Tye et al. (2016), results showed that children with ADHD (ADHD and ASD+ADHD) displayed increased reaction time variability compared with ASD-only and TD children. Children with ADHD (ADHD and ASD+ADHD) also showed reduced amplitude for the cue-P3 and no-go-P3 components, which have been linked to difficulties in attentional orienting and inhibitory control (Banaschewski et al., 2004; Doehnert, Brandeis, Imhof, Drechsler, & Steinhausen, 2010; Fallgatter et al., 2004; Valko et al., 2009), as well as reduced N2 amplitude for target and nontarget trials, indicating deficits in stimulus categorisation and conflict monitoring (Albrecht, Banaschewski, Brandeis, Heinrich, & Rothenberger, 2005; Albrecht et al., 2008; McLoughlin et al., 2009; Satterfield, Schell, & Nicholas, 1994; Senderecka, Grabowska, Gerc, Szewczyk, & Chmylak, 2012). Moreover, children with ASD (ASD-only group) exhibited attenuated N2 amplitude from target to nontarget trials, indicating deficits in attentional shifting and conflict monitoring (Hill, 2004; Sanders, Johnson, Garavan, Gill, & Gallagher, 2008). Note that previous studies have also reported increased N2 amplitude in ASD, which may reflect more effortful processing during conflict monitoring (Faja, Clarkson, & Webb, 2016; Høyland et al., 2017).

The findings by Tye et al. (2014, 2016) suggest that, while ASD and ADHD are associated with disorder-specific impairments, children with co-occurring ASD+ADHD have an additive profile of atypical attention. However, it is not yet well understood to what extent the distinct attentional impairments manifested in ASD and ADHD share common neurocognitive mechanisms, and if these mechanisms are shared with the comorbid group. Importantly, both ASD and ADHD have been associated with impaired top-down and bottom-up modulation of attention (Greenaway & Plaisted, 2005; Hasler et al., 2016; Keehn & Joseph, 2008; Keehn, Nair, Lincoln, Townsend, & Müller, 2016; Schneidt, Jusyte, Rauss, & Schönenberg, 2018), which indicates a potential shared mechanism between these disorders.

Electroencephalography (EEG) is particularly pertinent to study the neurocognitive mechanisms of attention, since it allows for time-sensitive measurements of overt and covert cognitive processing. Neurophysiological indices derived from EEG include oscillatory brain activity in different frequency bands, that is delta (1–3 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta (12–30 Hz), and gamma (30–70 Hz). Oscillatory activity in each of these frequency bands has been linked to different cognitive processes and, equally, distinct alterations in these oscillatory patterns during task and rest have been associated to ASD and ADHD (Adamou, Fullen, & Jones, 2020; Barry, Clarke, & Johnstone, 2003; Kessler, Seymour, & Rippon, 2016; Lau-Zhu, Fritz, & McLoughlin, 2019; Shephard et al., 2018). Alpha oscillatory activity in the occipito-parietal cortex is of particular interest in the context of attention because it is the only frequency band that shows a decrease in power during visual stimulation (or other cognitive activity) and an increase in power in the absence of visual input (Klimesch, 1999, 2012). Importantly, early studies showed that alpha power also decreases when a visual target is anticipated

(i.e. before it is presented; Foxe, Simpson, & Ahlfors, 1998; Klimesch, Doppelmayr, Russegger, Pachinger, & Schwaiger, 1998), supporting the notion that alpha band activity is related to the alerting system of attention (Fan et al., 2007). Successive studies using attention selection tasks have found that alpha power increases (more synchronised) in hemispheres processing the unattended visual hemifield, but decreases (more desynchronised) in hemispheres processing the attended visual hemifield, both in adults (Händel, Haarmeier, & Jensen, 2011; Kelly, Lalor, Reilly, & Foxe, 2006; Sauseng, Klimesch, Stadler, et al., 2005; Thut, Nietzel, Brandt, & Pascual-Leone, 2006; Worden, Foxe, Wang, & Simpson, 2000) and children (Vollebregt et al., 2015). These findings suggest that alpha band activity might be linked to the integration of top-down and bottom-up attentional processes, where alpha synchronisation is associated with active inhibition of information flow and reduced task engagement, and alpha desynchronisation is related to facilitation of information flow and increased task engagement (i.e. release from inhibition; Klimesch, 2012). However, to our knowledge, no previous study has directly investigated alpha oscillatory activity as a shared mechanism underlying attentional impairments across ASD and ADHD.

Notably, alpha band activity is desynchronised during anticipatory attention (i.e. before sensory stimulation) and it has been found that prestimulus alpha power predicts forthcoming stimulus perception (Romei, Gross, & Thut, 2010). For instance, greater prestimulus alpha power is linked to poorer performance in visual discrimination tasks (van Dijk, Schoffelen, Oostenveld, & Jensen, 2008; Ergenoglu et al., 2004; Hanslmayr et al., 2007; Thut et al., 2006) and poorer inhibition of motor responses to a visual stimulus (Bengson, Mangun, & Mazaheri, 2012; Mazaheri, Nieuwenhuis, Van Dijk, & Jensen, 2009). Prestimulus alpha also modulates the latency and amplitude of poststimulus ERPs linked to early visual processing, such as the P1 or N1 components (Brandt & Jansen, 1991; Fellinger, Klimesch, Gruber, Freunberger, & Doppelmayr, 2011; Klimesch, 2011; Klimesch, Schabus, Doppelmayr, Gruber, & Sauseng, 2004; Lou, Li, Philiastides, & Sajda, 2014). Moreover, it has been shown that pre- and poststimulus alpha rhythms are coupled with poststimulus frontocentral theta synchronisation (i.e. activity in the 4–8 Hz band; Kawasaki, Kitajo, & Yamaguchi, 2010; Keller, Payne, & Sekuler, 2017; Klimesch, 2012; Sauseng, Klimesch, Schabus, & Doppelmayr, 2005; Williams, Kappen, Hassall, Wright, & Krigolson, 2019). These studies find that, while alpha band activity is related to attentional processes, frontocentral theta band activity is linked to cognitive control and working memory, and suggest that alpha-theta coupling indicates integration of task-relevant local information processing with long-range functional networks underlying central executive functions. Overall, this suggests that alpha oscillatory activity is important for how we process and respond to incoming stimuli, and raises the question of whether prestimulus alpha rhythms represent a shared mechanism for differing attentional deficits in ASD and ADHD.

Previous studies have found that both ASD and ADHD are linked to atypical modulation of alpha band activity in attentional tasks. Compared with TDs, children with ASD do not show alpha desynchronisation when presented with relevant targets (Keehn, Westerfield, Müller, & Townsend, 2017), nor an increase in prestimulus alpha power when presented with irrelevant stimuli (Murphy, Foxe, Peters, & Molholm, 2014). Similarly, children and adults with ADHD do not present lateralisation of alpha synchronisation/desynchronisation during visuospatial attention tasks (ter Huurne et al., 2013; Vollebregt, Zumer, ter Huurne, Buitelaar, & Jensen, 2016), do not show correlations between degree of alpha lateralisation and performance (ter Huurne et al., 2013; Vollebregt et al., 2016), and show reduced alpha desynchronisation in preparation and response to targets (Hasler et al., 2016; Mazaheri et al., 2014). This suggests that abnormal alpha band activity associated with failures in selective attention (i.e. orienting to salient stimuli and inhibiting task-irrelevant information) may indicate a common mechanism underlying attentional deficits in ASD and ADHD. However, these studies do not directly compare alpha rhythms between ASD and ADHD, and do not include the possibility of co-occurring symptoms. Interestingly, a resting state study on the same cohort used in the present investigation (Shephard et al., 2018), found that children with ASD (ASD and ASD+ADHD) showed reduced alpha power compared with ADHD-only and TD children. They also found that no differences were uniquely associated with the ASD+ADHD group, suggesting an additive profile of atypical alpha power in the comorbid group. Yet, patterns of alpha power during resting state reflect generalised neural processing and do not inform about task-specific effects (e.g. related to attention). Thus, it remains unknown whether atypical alpha rhythms during attention tasks are shared between ASD and ADHD, or if these atypicalities follow an additive or unique profile in the comorbid group. Moreover, no previous studies have directly compared theta synchronisation and P1 amplitude patterns across these diagnostic groups, nor have investigated the relationship between prestimulus oscillatory activity and poststimulus neurophysiological activity in ASD and ADHD (and the comorbid group) in the context of attention. These investigations are crucial to better understand the mechanisms underlying attentional impairments in ASD and ADHD, and could in turn contribute to the identification of transdiagnostic subgroups.

This study aimed to characterise the profile of alpha rhythm patterns (prestimulus alpha power and stimulus-locked alpha desynchronisation) during attention in children with ASD, ADHD, ASD+ADHD, and a group of TD controls. Note, we were specifically interested in stimulus-related effects on alpha oscillations, rather than response-related effects or ongoing modulations on alpha. We also aimed to describe the patterns of poststimulus neurophysiological activity linked to attention (P1, N2, P3) and executive functions (theta synchronisation) across diagnostic groups. Further, we aimed to investigate the relationship between alpha rhythms and poststimulus activity (theta synchronisation and P1), as well as between alpha rhythms and behavioural performance (note that findings on behavioural performance for this same study have been previously reported in Tye et al. (2016)). Participants completed the 'Fast task' (Cheung et al., 2016; Kuntsi et al., 2005), a fixed choice reaction time task that allows us to measure brain activity related to attentional control. We tested the following hypotheses. First, increased prestimulus alpha power and attenuated alpha desynchronisation would be found for ASD and ADHD compared with TD, reflecting less attentional engagement with the stimulus and, by extension, the task. In line with previous studies, we also expected that the comorbid group would present an additive profile with both ASD- and ADHD-related atypicalities. Second, we expected a similar pattern of findings as those Tye et al. (2014) reported in the CPT-OX paradigm, as they directly compared TD, ASD, ADHD, and ASD+ADHD groups: in the context of our task, we hypothesised that ADHD and ASD would be associated with attenuated N2 amplitude (reflecting impairments in attentional control), ADHD would be related to reduced P3 amplitude (reflecting reduced attention allocation; Cheung et al., 2017), and comorbid ASD+ADHD would manifest an additive profile. No previous studies have directly compared theta synchronisation and P1 patterns across ASD, ADHD, and ASD+ADHD, so these analyses were exploratory. Finally, we hypothesised that, in TD children, lower prestimulus alpha power and greater alpha desynchronisation would be related to greater theta synchronisation and P1 amplitude, as well as to better task performance (i.e. faster and less variable reaction times). Due to the lack of previous studies investigating these correlations in ASD and ADHD, our analysis for the ASD, ADHD, and ASD+ADHD groups was exploratory.

Materials and methods

Participants

A priori power calculation to detect medium to large effects (alpha set at .05) required a minimum of 19 participants in each group. Because the research diagnosis process (see below) led to unequal group sizes, our final sample included 25 TD participants, 19 participants with ASD, 17 participants with ADHD, and 27 participants with ASD+ADHD. Post hoc power analyses showed that, with this sample, effects related to diagnostic group and ASD/ADHD diagnosis had moderate to excellent power (79–99%). To reduce sample heterogeneity, and given the higher ratio of ASD diagnoses among males than among women (Elsabbagh et al., 2012; Polanczyk, Silva de Lima, Lessa Horta, Biederman, & Augusto Rohde, 2007), all participants were males. Groups were matched on age (Table 1) and the age range was 8–13 years. Participants included in the study had IQ scores in the normal range (>70 on the Wechsler Abbreviated Scale of Intelligence; Wechsler, 1999) and normal or corrected-to-normal vision. Participants were excluded from the study if they were taking medications other than stimulants. In line with other studies (Andreou et al., 2007; Hasler et al., 2016), stimulant medication was interrupted 48 hr before the testing session to secure wash-out (Kolar, Keller, Cumyn, Syer, & Hechtman, 2008). Six participants with ADHD and six participants with ASD+ADHD were receiving stimulant medication (ADHD: one Equasym, three Concerta, one Equasym plus Concerta, one unspecified; ASD+ADHD: five Concerta, one dexamphetamine). A further one participant with ADHD and one participant with ASD+ADHD had previously received stimulants (both unspecified) but were not taking any medication at the time of the study. Participants were also excluded if they had neurological disorders or comorbid psychiatric conditions other than ASD and ADHD (excluding oppositional defiant disorder).

Participants with ASD and/or ADHD were recruited from outpatient neurodevelopmental clinics and local parent support groups in South London. All participants had a clinical diagnosis for ASD and/or ADHD according to the ICD-10 criteria (WHO, 1993). Trained researchers then confirmed single-diagnosis and comorbid groups with a comprehensive research diagnosis. Participants were initially screened for ASD symptoms with the Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003), and for ADHD symptoms with Conners' 3rd Edition Parent Rating Scale short form (Conners, 2008; Table 1). ASD cases were further assessed with the Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter, & Lecouteur, 1994) and the Autism Diagnostic Observation Schedule (ADOS-G; Lord et al., 2000), and ADHD cases were assessed with the Parent Account of Childhood Symptoms (PACS; Taylor et al., 1986). Comorbid ASD and ADHD met full diagnostic criteria for ASD using the ADI-R/ADOS and full diagnostic criteria for ADHD using the PACS. When clinical diagnosis and research diagnosis were different, participants were reallocated on the basis of their research diagnosis. In particular, 16 participants clinically diagnosed with ASD were reallocated to the ASD+ADHD group, 1 participant clinically diagnosed with ASD+ADHD was reallocated to the ASD group, and 3 participants clinically diagnosed with ADHD were reallocated to the ASD+ADHD group. For the TD group, participants were recruited through local schools and forums and were included if they had no neurodevelopmental or psychiatric diagnoses, and no siblings with ASD or ADHD. TD participants were assessed for subclinical symptoms with the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997), SCQ and Conners' questionnaires.

The study was granted ethical approval by a medical ethics committee, and written parental consent was obtained before completion of the study.

Task

To measure brain activity related to attentional control, we used the 'Fast task', which has been previously employed as a measure of reaction time (RT) variability in ADHD (Andreou et al., 2007; Kuntsi et al., 2005). Participants first saw a warning signal on the screen (four empty circles arranged side-

Table 1 Clinical and demographic characteristics

	TD (n = 25)		ASD (n = 19)		ADHD (n = 17)		ASD+ADHD (n = 27)				
	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	*F*	*p*	Post hoc
Age	10.51	1.81	11.69	1.70	10.47	1.97	10.51	1.75	2.17	.097	n.s.d.
Full-scale IQ	120.0	13.69	115.7	15.73	104.3	14.64	110.0	12.86	4.68	.004	TD > ADHD
SCQ	3.92	3.60	19.89	6.49	10.71	5.46	24.93	5.88	74.4	<.001	ASD+ADHD > ASD > ADHD > TD
Conners DSM-Inattentive	56.32	11.20	67.10	14.13	83.59	7.47	80.11	11.98	26.4	<.001	ADHD, ASD+ADHD > ASD > TD
Conners DSM-Hyperactive	59.28	17.24	66.10	12.99	88.06	3.27	84.59	7.05	31.3	<.001	ADHD, ASD+ADHD > TD, ASD

n.s.d., nonsignificant difference; *SD*, standard deviation.

by-side). After a fore-period (presentation interval for the warning signal), the circle designated as the target signal for that trial was filled (coloured) in. The child was instructed to press the response key that matched the location of the target stimulus. After the response, the stimuli disappeared from the screen and a fixed intertrial interval of 2.5 s followed. Speed and accuracy were emphasised equally, and if the child did not respond within 10 s, the trial was terminated.

The 'Fast task' consisted of three sections. First, participants completed a practice session during which they had to respond correctly to five consecutive trials. Second, participants completed a slow-baseline condition, which consisted of 172 trials with an 8-second fore-period. Finally, participants completed a fast-incentive condition (80 trials with a 1-second fore-period) where children could win smiley faces depending on their performance and earn real prizes at the end. Note that the fast-incentive condition was excluded from the analysis reported in the present paper: since it involves faster responses and more eye movements (children gaze back and forth to the smiley faces) the neurophysiological data is much noisier with a large amount of artifacts making it unsuitable for the current analysis.

Behavioural performance

A detailed description of behavioural performance in this cohort has been previously reported in Tye et al. (2016). Behavioural measures relevant for the present paper include mean reaction time to respond after target onset (RTM, in ms) and three measures of within-subject variability in reaction time (RTV, all in ms): standard deviation (RTV-SD), coefficient of variation (RTV-CV; computed as RT-SD/RTM), and Tau (RTV-Tau; ex-Gaussian parameter corresponding to the exponential tail of the distribution; Karalunas et al., 2014).

EEG acquisition and processing

Electroencephalograms were recorded using a 62 active electrode recording system (ActiCap, Brain Products, Germany; extended 10–20 montage). The recording reference electrode was positioned at FCz. Vertical and horizontal electrooculograms (EOGs) were recorded simultaneously from electrodes above and below the left eye and at the outer canthi. The signal was digitised at a 500-Hz sampling rate, stored, and analysed offline.

Data processing and analysis was performed using the EEGLAB toolbox (v11.0.3.1b; Delorme & Makeig, 2004) for MATLAB (R2012a; The Mathworks, Inc., Natick, Massachusetts). Before processing, the channel signals were re-referenced to average reference (helps to remove line noise when using ICA). We then applied a band-pass filter between 0.5 Hz and 40 Hz. Time points with any channel value larger than 200 μV in absolute value were rejected from the data and excluded from further analysis (note 200 μV is used to ensure that eye blinks are not removed, as these are removed later with ICA).

We used adaptive mixture ICA (AMICA; Delorme, Palmer, Onton, Oostenveld, & Makeig, 2012; Palmer, Kreutz-Delgado, & Makeig, 2006) to separate the channel data into maximally instantaneous independent components (IC) processes. We computed equivalent dipole models for each IC scalp topography using a template four-layer adult boundary element method head model implemented in the DIPFIT toolbox for MATLAB (Oostenveld & Oostendorp, 2002). Eye blink components were removed using Eyecatch (EEGLAB). Individual participant ICA decompositions were examined, and components with nonlocalised scalp maps, dipole locations outside the scalp, and atypical power spectral density were rejected. The EEG data were back projected, and channels were normalised to 10 uV standard deviation to standardise the data power across subjects to normal EEG range. Data were epoched into segments from −6 s to +4 s, time-locked to stimulus presentation, to ensure good estimates of spectral power. For each of the following electrophysiological indices, electrodes were based on the location of maximum source projection on the scalp.

Alpha power corresponds to the total power in the band 8–12 Hz, and was computed at channel Oz for two separate epochs: prestimulus alpha power was computed in the period from −4 s to 0 s (relative to stimulus presentation) and poststimulus alpha power was computed from the period 0 s to 2 s. Note that, although the poststimulus period contains response times, in computing alpha power, the low-frequency fluctuations related to the response should not enter into the higher frequency alpha band related to the stimulus. Moreover, interfering fluctuations would be expected to degrade the significance of alpha desynchronisation findings, and therefore should not invalidate the significance of the present findings. The power spectral density (PSD) was computed on the respective data segments, using the Welch method, with a Fast Fourier Transform (FFT) analysis window of 512 time points (1024 ms). The FFT window overlap was 500 time points and a Hamming window was used. Spectral measures were computed by averaging over the stated time and frequency range in the computed spectrogram. The PSD was then converted to dB by normalising the PSD and setting dB = 10*log10(PSD) (this allows for context in relation to other studies of power). Alpha desynchronisation was computed as the difference between alpha poststimulus and alpha prestimulus.

Theta synchronisation corresponds to the total power in the band 4–8 Hz, and was computed at channel FCz over the period 100 ms to 600 ms. This period was determined from the average spectrogram, where theta synchronisation was observed in the interval 100 ms to 600 ms poststimulus; note that 500 ms contains approximately three theta cycles, which

is sufficient to obtain an estimate of theta power. The power spectral density (PSD) was computed on the respective data segments, using the Welch method, with an FFT analysis window of 512 time points (1,024 ms). The FFT window overlap was 500 time points and a Hamming window was used. Spectral measures were computed by averaging over the stated time and frequency range in the computed spectrogram. The baseline PSD was computed by averaging the spectrogram over the time period −1 s to 0 s (before stimulus presentation) and this was removed from the poststimulus power spectra.

The P1 was computed at channel Oz, as the maximum amplitude in the period from 100 ms to 250 ms after target stimulus presentation. The N2 was computed at channel FCz, as the minimum amplitude between 200 ms and 300 ms after target stimulus presentation. The P3 was computed at channel Pz, as the maximum amplitude between 300 ms and 800 ms after target stimulus presentation. Peak selection was automatic. To ensure that no peaks fell at the edge of the time range, any peak that co-ocurred in the last time point before the boundary was rejected. The time windows used produced edge maxima or minima in fewer than 5 trials on average per participant.

Statistical analyses

Groups were compared on six different neurophysiological measures: prestimulus alpha power, alpha power desynchronisation, poststimulus P1 amplitude, poststimulus N2 amplitude, and post-timulus P3 amplitude for attentional control; poststimulus theta synchronisation for executive functions. To evaluate differences between single-diagnosis and comorbid groups, for each neurophysiological parameter, we fitted a one-way ANOVA with diagnostic group (TD, ASD, ADHD, ASD+ADHD) as between-subject factor. To further assess attentional profiles in the comorbid group (i.e. unique conditions or additive model), we fitted a 2 × 2 ANOVA with ASD diagnosis (ASD− or ASD+) and ADHD diagnosis (ADHD− or ADHD+) as between-subject factors, where a nonsignificant interaction between disorders is compatible with an additive model of comorbidity. Post hoc pairwise comparisons using Bonferroni-adjusted *p*-values were also computed. Note that age and IQ were nonsignificant covariates for all parameters, so they were not included for any analysis.

Because the absence of an interaction effect cannot be interpreted as evidence for an absence of this effect, we used equivalence testing to robustly reject interaction effects and in turn support the null hypothesis of an additive model of comorbidity. Equivalence testing allows researchers to test if a difference between two groups is at least as extreme as the smallest effect size of interest (SESOI), which defines the equivalence bounds: if the observed difference falls within the interval indicated by the equivalence bounds, the two groups are considered statistically equivalent (Lakens, 2017; Lakens, Scheel, & Isager, 2018). To run the equivalence test on the interaction effect, we first defined the interaction effect in our model in terms of a *t*-test, that is comparing the ASD and ADHD groups (group 1) versus the TD and ASD+ADHD groups (group 2). We then defined the SESOI by running a sensitivity analysis in G*Power (Faul, Erdfelder, Buchner, & Lang, 2009; Faul, Erdfelder, Lang, & Buchner, 2007): with 36 participants in group 1, 52 participants in group 2, a desired power of at least 80%, and an α of .05, the SESOI (i.e. Cohen's *d*) was .614 so equivalence bounds of ±.614 were used. Finally, we performed equivalence tests for the interaction effect of all six neurophysiological measures with the R-based TOSTER package (Lakens et al., 2018), where a significant equivalence test indicates that the two groups are statistically equivalent.

Spearman's correlations were conducted between all six neurophysiological parameters and symptom scores across the whole sample controlling for diagnostic group. To account for the significant association between ASD and ADHD symptoms (SCQ-inattention: $\rho = .367$, $p < .001$; SCQ-hyperactivity/impulsivity: $\rho = .386$, $p < .001$), these correlations were conducted on symptom scale corrected scores (i.e. we run correlations between ASD symptoms and neurophysiological measures controlling for ADHD symptoms, and correlations between ADHD symptoms and neurophysiological measures controlling for ASD symptoms). To correct for multiple correlations Bonferroni-corrected alpha levels were used.

We also investigated the relationship between alpha neurophysiological measures (i.e. prestimulus alpha power and alpha power desynchronisation) and poststimulus neurophysiological measures (based on previous evidence we focused on theta synchronisation and P1 amplitude), as well as between alpha neurophysiological measures and task performance (RTM, RTV-SD, RTV-CV, RTV-Tau). For this, Spearman's correlations were conducted between the parameters of interest for each diagnostic group, and Bonferroni-corrected alpha levels were used to correct for multiple correlations within each diagnostic group. Differences in associations between neurocognitive measures across diagnostic groups were further investigated using Fisher's transformations, and Bonferroni-corrected alpha levels were used to correct for multiple comparisons within each correlation (i.e. for each correlation there were 6 possible comparisons: TD–ASD, TD–ADHD, TD–ASD+ADHD, ASD–ADHD, ASD–ASD+ADHD, and ADHD–ASD+ADHD).

Results

Behavioural performance

A detailed description of behavioural performance in this cohort has been previously reported in Tye et al. (2016). Briefly, it was found that responses of children with ADHD (ADHD and ASD+ADHD) were slower (higher RTM) and more variable (higher RTV-SD, RTV-CV, and RTV-Tau) than those of ASD-only and TD children (see Table S1).

Group differences in neurophysiological measures

Table 2 shows descriptives of neurophysiological measures for each diagnostic group.

For alpha prestimulus (Figure 1A–E), there was no main effect of diagnostic group, $F(3, 84) = .401$, $p = .477$, $n_p^2 = .014$. Moreover, there was no main effect of ASD or ADHD diagnosis (ASD: $F(1, 84) = .672$, $p = .415$, $n_p^2 = .008$; ADHD: $F(1, 84) = .000$, $p = .998$, $n_p^2 = .000$), and no interaction effect between ASD and ADHD ($F(1, 84) = .495$, $p = .484$, $n_p^2 = .006$). Equivalence testing further supported the absence of an interaction effect ($t(71.04) = 2.106$, $p = .019$).

For alpha desynchronisation (Figure 1A–D,F), there was a main effect of diagnostic group, $F(3, 84) = 11.03$, $p = .001$, $n_p^2 = .168$. Post hoc pairwise comparisons further showed that children with ADHD and ASD+ADHD showed attenuated alpha desynchronisation compared with TD children (ADHD: $t(40) = 3.64$, $p = .003$, $d_z = .562$; ASD+ADHD: $F(50) = 2.97$, $p = .023$, $d_z = .412$). Moreover, the factorial analysis yielded a main effect of ADHD, where ADHD diganosis (ADHD and ASD+ADHD) was associated with attenuated alpha desynchronisation, *F*

Table 2 Descriptives for neurophysiological measures

	TD (n = 25)		ASD (n = 19)		ADHD (n = 17)		ASD+ADHD (n = 27)	
	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
Alpha prestimulus	13.64	5.36	12.04	5.08	12.90	5.07	12.78	3.83
Alpha desync	−2.71	1.58	−2.33	1.28	−1.12	1.06	−1.57	1.46
Theta sync	1.32	0.849	1.31	0.970	1.29	0.607	1.24	0.554
P1 amplitude	5.48	1.84	5.20	1.94	5.54	2.53	5.35	2.15
N2 amplitude	−3.43	0.803	−3.89	1.51	−2.24	1.02	−3.08	1.03
P3 amplitude	4.73	1.26	4.62	2.07	3.25	1.46	3.79	1.40

SD, standard deviation.

(1,84) = 15.18, $p < .001$, $n_p^2 = .153$. There was no main effect of ASD ($F(1, 84) = .010$, $p = .919$, $n_p^2 = .000$) and no interaction effect between ASD and ADHD ($F(1, 84) = 1.91$, $p = .171$, $n_p^2 = .022$). Equivalence testing further supported the absence of an interaction effect ($t(83.64) = 1.727$, $p = .044$).

For theta synchronisation (Figure 2), there was no main effect of diagnostic group, $F(3,84) = .053$, $p = .984$, $n_p^2 = .002$. Moreover, there was no main effect of ASD or ADHD diagnosis (ASD: $F(1, 84) = .022$, $p = .883$, $n_p^2 = .000$; ADHD: $F(1, 84) = .102$, $p = .750$, $n_p^2 = .001$), and no interaction effect between ASD and ADHD ($F(1, 84) = .011$, $p = .918$, $n_p^2 = .000$). Equivalence testing further supported the absence of an interaction effect ($t(71.84) = -2.696$, $p = .004$).

For P1 amplitude (Figure 3A,B), there was no main effect of diagnostic group, $F(3, 84) = .100$, $p = .960$, $n_p^2 = .004$. Moreover, there was no main effect of ASD or ADHD diagnosis (ASD: $F(1, 84) = .270$, $p = .605$, $n_p^2 = .003$; ADHD: $F(1, 84) = .053$, $p = .818$, $n_p^2 = .001$), and no interaction effect between ASD and ADHD ($F(1, 84) = .009$, $p = .925$, $n_p^2 = .000$). Equivalence testing further supported the absence of an interaction effect ($t(70.05) = 2.696$, $p = .004$).

For N2 amplitude (Figure 3C,D), there was a main effect of diagnostic group, $F(3, 84) = 7.41$, $p < .001$, $n_p^2 = .209$. Post hoc pairwise comparisons further showed that children with ADHD showed attenuated N2 amplitude compared with TD children ($t(40) = 3.48$, $p = .005$, $d_z = .55$) and ASD children ($F(34) = 4.53$, $p < .001$, $d_z = .776$). Moreover, the factorial analysis yielded a main effect of ADHD ($F(1, 84) = 17.84$, $p < .001$, $n_p^2 = .175$), where ADHD diagnosis (ADHD and ASD+ADHD) was associated with attenuated N2 amplitude. There was also a main effect of ASD ($F(1, 84) = 7.53$, $p = .007$, $n_p^2 = .082$), where ASD diagnosis (ASD and ASD+ADHD) was associated with greater N2 amplitude. There was no interaction effect between ASD and ADHD ($F(1, 84) = .657$, $p = .420$, $n_p^2 = .008$). Equivalence testing further supported the absence of an interaction effect ($t(52.92) = -2.232$, $p = .015$).

For P3 amplitude (Figure 3E,F), there was a main effect of diagnostic group, $F(3,84) = 4.18$, $p = .008$, $n_p^2 = .130$. Post hoc pairwise comparisons further showed that children with ADHD showed attenuated P3 amplitude compared with TD children ($t(40) = 3.05$, $p = .018$, $d_z = .471$). Moreover, the factorial analysis yielded a main effect of ADHD ($F(1, 84) = 11.91$, $p = .001$, $n_p^2 = .124$), where ADHD diagnosis (ADHD and ASD+ADHD) was associated with attenuated P3 amplitude. There was no main effect of ASD ($F(1, 84) = .397$, $p = .530$, $n_p^2 = .005$) and no interaction effect between ASD and ADHD ($F(1, 84) = .944$, $p = .334$, $n_p^2 = .011$). Equivalence testing further supported the absence of an interaction effect ($t(60.41) = 2.037$, $p = .023$).

Correlations between neurophysiological measures and symptom scores

Table 3 shows correlations between neurophysiological measures and symptom scores, across the whole sample. No correlations survived the Bonferroni correction.

Correlations between alpha measures, poststimulus neurophysiological measures, and behavioural performance

Table 4 shows correlations between alpha measures (prestimulus alpha and alpha desynchronisation) and poststimulus neurophysiological measures, as well as between alpha neurophysiological measures and behavioural performance, for each diagnostic group. It also shows differences in correlations between diagnostic groups. Figure S1 shows scatterplots for correlations that are significant for at least one diagnostic group.

For the ASD group, there was a significant negative correlation between prestimulus alpha power and theta synchronisation ($\rho = -.628$, $p = .004$) and a significant positive correlation between alpha desynchronisation and theta synchronisation ($\rho = .721$, $p < .001$). Fisher's transformations indicated no differences in these correlations between groups.

Fisher's transformations indicated a difference between ADHD and ASD+ADHD groups in the correlation between alpha desynchronisation and P1 amplitude ($p = .005$; but note these correlations were not significant).

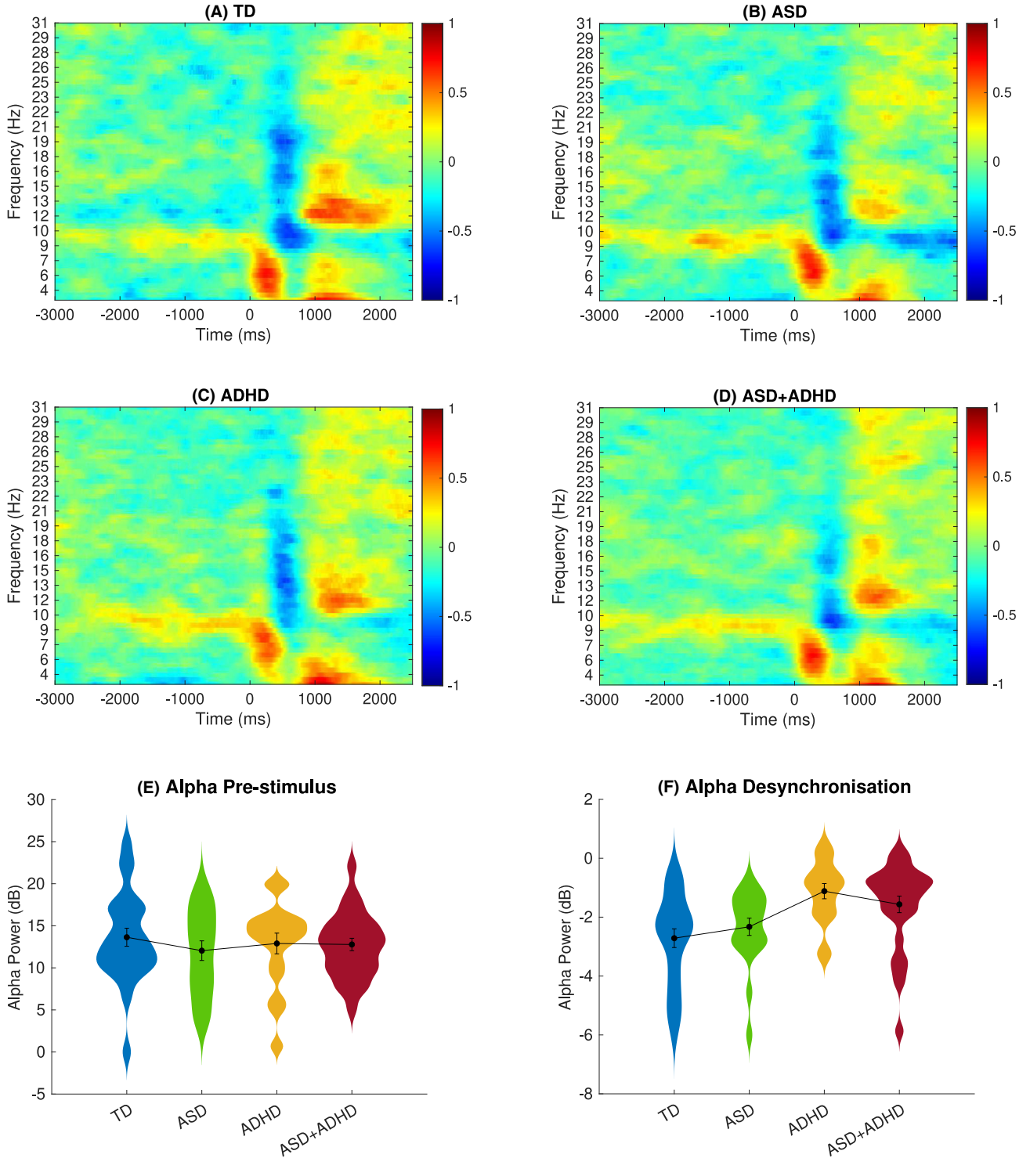

Figure 1 Time-frequency plots of alpha band activity locked to stimulus presentation for each diagnostic group (A–D), and corresponding violin plots showing mean (filled circle), *SE* (error bars) and frequency of values (width of the distribution) of alpha prestimulus (E) and alpha desynchronisation (F) for each diagnostic group

No other correlations between measures, or differences in correlations between diagnostic groups, survived the Bonferroni correction.

Discussion

Here, we investigated the neurophysiological profile of children with ASD, ADHD, ASD+ADHD, and a group of TD controls during attentional control, particularly focusing on alpha rhythms (prestimulus alpha power and alpha desynchronisation). We found that children with ADHD (ADHD and ASD+ADHD groups) showed attenuated alpha desynchronisation compared with children without ADHD (TD and ASD groups), suggesting impaired attentional engagement. Consistent with previous

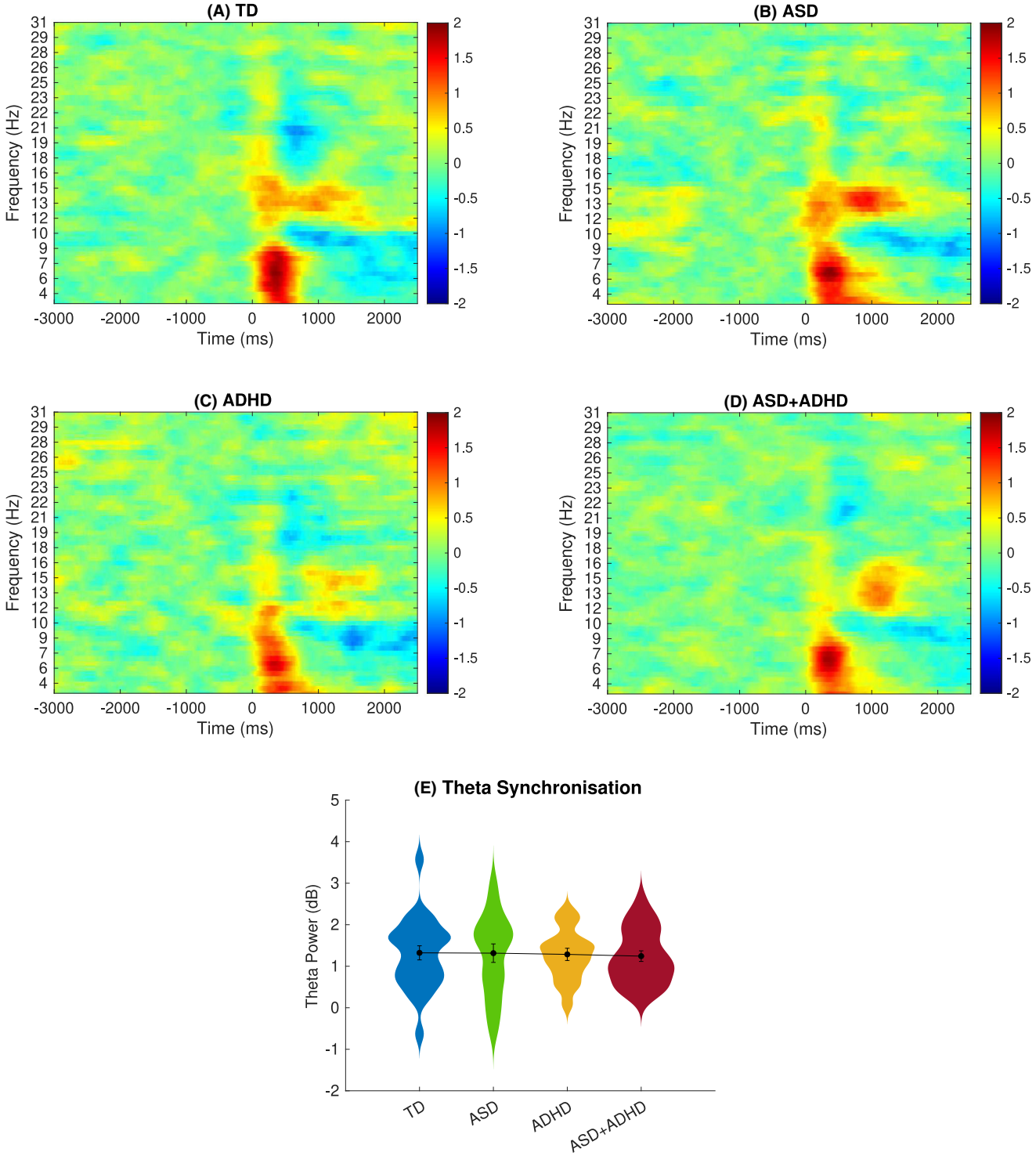

Figure 2 Time-frequency plots of theta band activity locked to stimulus presentation for each diagnostic group (A–D), and corresponding violin plot showing mean (filled circle), *SE* (error bars), and frequency of values (width of the distribution) of theta power for each diagnostic group (E)

studies, children with ADHD (ADHD and ASD+ADHD groups) also showed attenuated N2 and P3 amplitude, although children with ASD (ASD and ASD+ADHD groups) showed greater N2 amplitude than children without ASD (TD and ADHD groups). Exploratory correlations indicated that alpha rhythms were strongly associated with theta synchronisation in the ASD group. Overall, children with comorbid ASD+ADHD showed characteristics of both ASD and ADHD groups, supporting an additive profile of atypical attentional control when these disorders co-occur.

Results showed that alpha desynchronisation was attenuated in children with ADHD (ADHD and ASD+ADHD groups) compared with children without ADHD (TD and ASD groups), indicating reduced

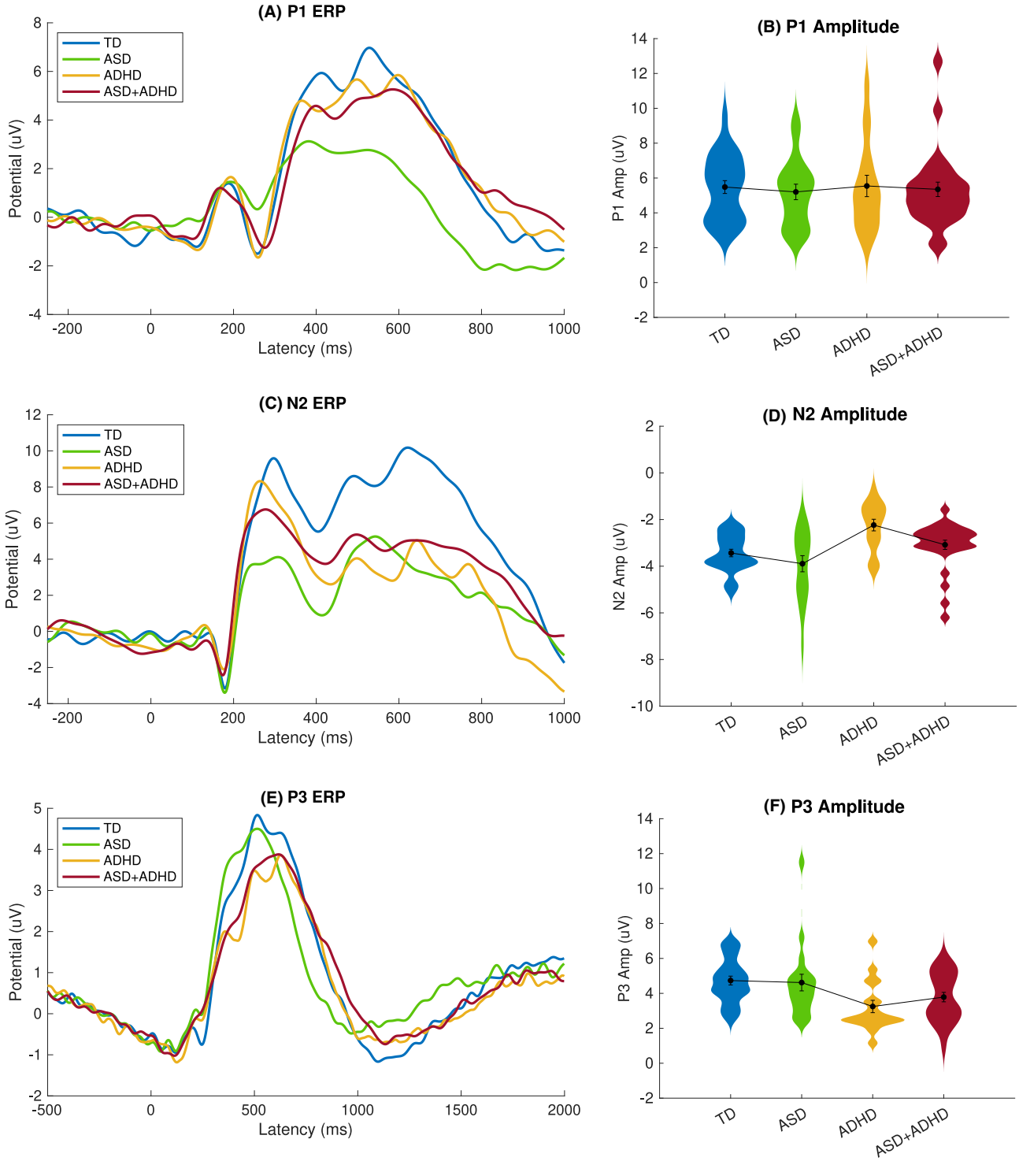

Figure 3 Grand mean ERPs locked to stimulus presentation for each diagnostic group (left column), and corresponding violin plots (right column) showing mean (filled circle), *SE* (error bars), and frequency of values (width of the distribution) for P1 (A–B), N2 (C–D), and P3 (E–F)

attentional engagement to the task at hand. This is in line with previous studies showing that children and adults with ADHD present reduced alpha desynchronisation during selective attention tasks (Hasler et al., 2016; ter Huurne et al., 2013; Mazaheri et al., 2014; Vollebregt et al., 2016). Moreover, the fact that there were group differences for alpha desynchronisation, but not for alpha prestimulus power, further suggests that attentional impairments in ADHD are linked to poor integration of top-down and bottom-up attentional processes rather than a specific deficit in anticipatory attention (Kelly et al., 2006; Klimesch, 2012; Thut et al., 2006). Contrary to previous studies (Keehn et al., 2017), there was no reduction in alpha desynchronisation for children with ASD (ASD and ASD+ADHD

Table 3 Correlations (ρ) between neurophysiological measures and symptom scores

	Autism-SCQ	ADHD-Conner's inattention	ADHD-Conner's hyperactivity/impulsivity
Alpha prestimulus	−.142	−.005	−.057
Alpha desync	−.031	.029	.115
Theta sync	−.066	.034	−.030
P1 amplitude	.008	−.130	−.030
N2 amplitude	−.239*	.173	.227*
P3 amplitude	.040	−.123	−.279**

Asterisk signify difference at $p < .05$*, $p < .01$**, $p < .001$***; bold indicates that survives Bonferroni correction for multiple testing (none survived); Bonferroni-corrected alpha level (18 comparisons) = .0027.

groups) compared with children without ASD (TD and ADHD groups). Thus, the direct comparison of ASD and ADHD groups in this study suggests that impairments in attentional control are more strongly associated with ADHD. Given the association between alpha band activity and the alerting system of attention (Fan et al., 2007), these findings are also in line with previous studies showing an impairment of the alerting system in ADHD (Mullane et al., 2011), and further suggest no deficits in this system in ASD.

Poststimulus N2 and P3 amplitude were attenuated in children with ADHD (ADHD and ASD+ADHD groups) compared with children without ADHD (TD and ASD groups), suggesting deficits in attentional control and attention allocation. Previous studies have found a similar pattern of results when comparing ADHD and TD groups, both for N2 (Albrecht et al., 2005, 2008; McLoughlin et al., 2009; Satterfield et al., 1994; Senderecka et al., 2012; Tye et al., 2014) and P3 (Banaschewski et al., 2004; Doehnert et al., 2010; Fallgatter et al., 2004; Valko et al., 2009). We also found that children with ASD (ASD and ASD+ADHD groups) presented greater N2 amplitude compared with children without ASD (TD and ADHD group). These findings contrast with those reported by Tye et al. (2014) where children with ASD showed attenuated N2 amplitude from target to nontarget trials compared with ADHD-only and TD groups. However, other studies have found a similar pattern of increased N2 amplitude in ASD during conflict monitoring (Faja et al., 2016; Høyland et al., 2017), which may reflect more effortful processing in ASD (Faja et al., 2016). Overall, these findings support previous evidence showing deficits in the alerting and executive control systems of attention in ADHD (Mullane et al., 2011), as well as atypicalities in the executive control system of attention in ASD (Keehn et al., 2013). Note that there were no group effects for the exploratory analyses on P1 amplitude and theta synchronisation. This is in line with previous studies suggesting that attentional impairments in ADHD may not affect early stages of attention allocation as indexed by P1 (Chmielewski et al., 2018; López et al., 2006). Moreover, a limitation of the 'Fast task' is that it does not require much cognitive control: this likely elicits weaker theta synchronisation which, in turn, makes it hard to detect differences between diagnostic groups.

Importantly, the findings on alpha desynchronisation and poststimulus N2 and P3 amplitude are generally consistent with an additive profile of atypical attention for the comorbid ASD+ADHD group. Previous studies have found attenuated alpha desynchronisation in both ASD and ADHD during attention tasks (Hasler et al., 2016; ter Huurne et al., 2013; Keehn et al., 2017; Mazaheri et al., 2014; Murphy et al., 2014; Vollebregt et al., 2016), and it

Table 4 Correlations between alpha neurophysiological measures, poststimulus neurophysiological measures and behavioural performance

	Alpha prestimulus						Alpha desynchronisation					
	Theta sync	P1 amp	RTM	RTV-SD	RTV-CV	RTV-Tau	Theta sync	P1 amp	RTM	RTV-SD	RTV-CV	RTV-Tau
Correlations (ρ)												
TD	−.117	.435*	−.267	−.413*	−.245	−.430*	.318	−.049	−.049	.318	.146	.103
ASD	**−.628****	.240	.354	.302	.130	.251	**.721*****	−.232	−.235	−.253	−.187	−.230
ADHD	−.544*	.598*	.240	.152	.092	.164	.218	−.520*	.137	−.125	−.357	−.252
ASD+ADHD	−.418*	−.129	.138	.124	−.031	.123	.305	.367	−.112	−.102	-.042	−.114
Differences across groups (*p*-value)												
TD – ASD	.067	n.s.d	.057	.026*	n.s.d	.034*	.086	n.s.d	n.s.d	.082	n.s.d	n.s.d
TD - ADHD	n.s.d	n.s.d	n.s.d	.092	n.s.d	.076	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d
TD - ASD+ADHD	n.s.d	.050*	n.s.d	.063	n.s.d	.055	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d
ASD – ADHD	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d	.068	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d
ASD - ASD+ADHD	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d	n.s.d	.061	n.s.d	n.s.d	n.s.d	n.s.d
ADHD - ASD+ADHD	n.s.d	.018*	n.s.d	n.s.d	n.s.d	n.s.d	.073	**.005****	n.s.d	n.s.d	n.s.d	n.s.d

Correlations: asterisk signify difference at $p < .05$*, $p < .01$**, $p < .001$***; bold indicates that survives Bonferroni correction for multiple testing; Bonferroni Bonferroni-corrected alpha level (12 comparisons/group) = .0041.
Differences in correlations across groups: asterisk signify difference at $p < .05$*, $p < .01$**, $p < .001$***; bold indicates that survives Bonferroni correction for multiple testing; Bonferroni-corrected alpha level (6 comparisons/correlation) = .0083.
n.s.d., nonsignificant difference.

has also been reported that children with ASD+ADHD show an additive profile of atypical alpha power during resting state (Shephard et al., 2018). Here, we show for the first time that, during an attention task, comorbid ASD+ADHD is also linked to the additive co-occurrence of the profile found in both disorders: children with ADHD-only and children with ASD+ADHD showed similar attenuation of alpha desynchronisation, indicating impaired integration of top-down and bottom-up attentional processes in both groups. Consistent with previous studies (Tye et al., 2014), we found that atypical modulation of poststimulus neurophysiological measures was also present in the ASD+ADHD group. In particular, children with ASD+ADHD showed attenuated N2 and P3 amplitude associated with ADHD, as well as greater N2 amplitude associated with ASD. Crucially, the additive profile of atypical attention found in the ASD+ADHD group is in line with findings from twin studies showing genetic overlap between ASD and ADHD traits (Ronald, Simonoff, Kuntsi, Asherson, & Plomin, 2008), and in particular for ASD/ADHD traits linked to attention problems (Polderman et al., 2013).

An interesting question is the relationship between alpha oscillatory activity, poststimulus neurophysiological activity and behavioural performance: to our knowledge, no previous study has directly tested these relationships across ASD, ADHD, and ASD+ADHD. Our exploratory analyses yielded a strong negative correlation between prestimulus alpha power and theta synchronisation in the ASD group. Similarly, there was a strong positive correlation between alpha desynchronisation and theta synchronisation in the ASD group. Previous studies have shown that ASD is linked to atypical alpha and theta synchrony during resting state, attention tasks, and social cognition tasks (Dickinson, DiStefano, Senturk, & Jeste, 2018; Ghuman, van den Honert, Huppert, Wallace, & Martin, 2017; Shephard et al., 2018, 2019; Stavropoulos & Carver, 2018; Ye, Leung, Schäfer, Taylor, & Doesburg, 2014). Our preliminary results extend this previous evidence by showing a strong coupling of alpha band activity and poststimulus theta band activity during attentional control. One possibility is that in ASD there is atypical functional connection of attentional brain networks (Doesburg, Vidal, & Taylor, 2013; Han & Chan, 2017; Shephard et al., 2019; Ye et al., 2014), which may result in poor integration of attentional control processes and central executive functions (Kawasaki et al., 2010; Keller et al., 2017; Klimesch, 2012; Sauseng, Klimesch, Schabus, et al., 2005; Williams et al., 2019). Another possibility is that, since there are no behavioural performance differences between ASD and TD groups (Tye et al., 2016), the stronger coupling between alpha and theta band activity in ASD reflects a compensatory mechanism in ASD to support typical-like performance (Livingston & Happé, 2017). However, note that these correlations were not significantly different across diagnostic groups (see Table 4) and future studies with larger sample sizes will be needed to clarify these preliminary findings. There was also an association between prestimulus alpha power and theta synchronisation for the ADHD and ASD+ADHD groups, between prestimulus alpha power and P1 amplitude for the TD and ADHD groups, as well as between prestimulus alpha power and variability in performance (RTV-SD and RTV-Tau) for the TD group. However, the evidence for these associations was weaker (the tests did not pass the correction for multiple comparisons) and warrants further investigation with larger sample sizes.

Some limitations and future directions should also be considered. First, the sample size in this study was relatively small, which limits our ability to draw firm interpretations from the data due to low power. Particularly for the correlations between alpha rhythms and poststimulus activity, as well as between alpha rhythms and behavioural performance, future studies with larger sample sizes will be needed to clarify the patterns and strength of our findings. Second, children with ADHD-only had lower IQ compared with TD children, which could be a potential confound for some of the findings. Although our analyses did not yield any effect of IQ when included as a covariate, and the pattern of results was largely retained when IQ was included, future studies may investigate the impact of intellectual ability across the different diagnostic groups. Third, it is important to note that there may be different subgroups within the ASD+ADHD group. For instance an additive-effects model and a distinct-condition model are not exclusive of each other, both at the group and individual level: indeed, previous research has shown that neurocognitive atypicalities in ASD+ADHD follow both additive-like and distinct-like patterns depending on the neurocognitive component of study (Tye et al., 2013, 2014, 2016). Moreover, individuals in the ASD+ADHD group may be differently loaded towards an ASD or ADHD phenotype. Having access to information about when participants received each diagnosis (e.g. simultaneously or successively) could aid the identification of subgroups. Similarly, longitudinal studies that combine large samples with machine learning methods would allow for the study of these effects at the individual level. Fourth, this study directly compared attentional profiles in ASD and ADHD, and found that ASD is characterised by atypicalities in the executive control system of attention, while ADHD is characterised by deficits in the alerting and executive control systems of attention. This suggests that, while alerting processes may be a distinct cognitive marker between ASD and ADHD, executive control processes may constitute a common cognitive marker between both conditions. Future studies will be needed to further

clarify this possibility. Finally, future research will need to investigate the potential use of attentional neurophysiological activity (such as alpha rhythms, N2 amplitude, or P3 amplitude), to elucidate overlapping and distinct etiological pathways to ASD and ADHD. Interestingly, it has been found that behavioural performance during the 'Fast task' has moderate-to-good test-retest reliability, which warrants its suitability for studies investigating familial or genetic effects (Kuntsi et al., 2005). Studies that investigate underlying neurophysiological activity within genetically sensitive designs, together with validation of test-retest reliability of these measures, will be crucial to identify transdiagnostic subgroups across ASD and ADHD.

Conclusion

This is the first study to directly investigate the profile of alpha rhythm patterns during attention in children with ASD, ADHD, and ASD+ADHD. The findings show attenuated alpha desynchronisation in children with ADHD (ADHD and ASD+ADHD), indicating that attentional impairments linked to ADHD may particularly stem from poor integration of top-down and bottom-up attentional processes. Consistent with previous studies, children with ADHD (ADHD and ASD+ADHD) showed atypical poststimulus N2 and P3 amplitudes, suggesting atypical attentional control and allocation. In contrast, children with ASD (ASD and ASD+ADHD) showed greater poststimulus N2 amplitude, indicating more effortful attentional control. We also report for the first time a strong correlation between alpha rhythms and theta synchronisation in ASD, which may indicate atypical functional integration between attentional control processes and central executive functions. Overall, our findings suggest that while ASD and ADHD are associated with disorder-specific impairments in alpha rhythms and poststimulus neurophysiological activity, children with comorbid ASD+ADHD have an additive profile of atypical attention. These findings contribute towards a better understanding of common and distinct causal mechanisms underlying ASD and ADHD, which will aid the identification of transdiagnostic subgroups and personalised intervention strategies.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Table S1. Descriptives for behavioural measures.
Figure S1. Scatterplots showing correlations between alpha measures.

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Key points

- ASD and ADHD are characterised by atypical attention, but it is not well understood if they share a common neurocognitive mechanism, and if a dual diagnosis (ASD+ADHD) is associated with a distinct or additive profile of attention deficits.
- Children with ADHD (ADHD and ASD+ADHD) show attenuated alpha desynchronisation, indicating poor integration of top-down and bottom-up attentional processes in ADHD.
- Children with ADHD (ADHD and ASD+ADHD) show atypical attentional control and attention allocation (reduced N2 and P3 amplitude), while children with ASD (ASD and ASD+ADHD) show more effortful attentional control (greater N2 amplitude).
- Children with ASD+ADHD present an additive profile with attentional deficits of both disorders.
- These findings will aid the identification of transdiagnostic subgroups and personalised intervention strategies.

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