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## Special Issue "Neurodevelopmental Neurodiversity": Research Report

# Relationship between autonomic arousal and attention orienting in children and adolescents with ADHD, autism and co-occurring ADHD and autism



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## ABSTRACT

**Introduction:** Attention-Deficit/Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) may be characterized by different profiles of visual attention orienting. However, there are also many inconsistent findings emerging from the literature, probably due to the fact that the potential effect of autonomic arousal (which has been proposed to be dysregulated in these conditions) on oculomotor performance has not been investigated before. Moreover, it is not known how visual attention orienting is affected by the co-occurrence of ADHD and autism in people with a double diagnosis.

**Methods:** 99 children/adolescents with or without ADHD and/or autism (age  $10.79 \pm 2.05$  years, 65% boys) completed an adapted version of the gap-overlap task (with baseline and overlap trials only). The social salience and modality of stimuli were manipulated between trials. Eye movements and pupil size were recorded. We compared saccadic reaction times (SRTs) between diagnostic groups and investigated if a trial-by-trial association existed between pre-saccadic pupil size and SRTs.

**Results:** Faster orienting (shorter SRT) was found for baseline compared to overlap trials, faces compared to non-face stimuli and—more evidently in children without ADHD and/or autism—for multi-modal compared to uni-modal stimuli. We also found a linear negative association between pre-saccadic pupil size and SRTs, in autistic participants (without

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ADHD), and a quadratic association in children with ADHD (without autism), for which SRTs were slower when intra-individual pre-saccadic pupil size was smallest or largest.

**Conclusion:** Our findings are in line with previous literature and indicate a possible effect of dysregulated autonomic arousal on oculomotor mechanisms in autism and ADHD, which should be further investigated in future research studies with larger samples, to reliably investigate possible differences between children with single and dual diagnoses.

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## 1. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD, from hereon, autism) are neurodevelopmental conditions characterized by different patterns of behaviors. ADHD is characterized by inattention and hyperactivity/impulsivity, while Autism is characterized by social communication/interaction difficulties and restricted and repetitive behaviors, interests or activities ([American Psychiatric Association, 2013](#)). Nevertheless, socio-emotional and communication difficulties are often present as co-occurring symptoms in ADHD ([Leitner, 2014](#); [Rommelse et al., 2010, 2011](#)) and inattention and hyperactivity/impulsivity co-occur with autistic traits at clinical and subclinical level ([Krakowski et al., 2022](#); [Reiersen et al., 2007](#); [Ronald et al., 2008](#)).

This symptomatologic overlap may reflect the fact that ADHD and autism arise from similar etiologic factors (genetic and/or environmental; [Hoogman et al., 2022](#)), which could also explain why infants later diagnosed with either ADHD or autism show similar behavioral profiles during early infancy, i.e., inattention and reduced joint attention, high negative affect and emotionality, and difficulties with effortful control ([Johnson et al., 2015](#); [Visser et al., 2016](#)). The high co-occurrence and symptomatologic overlap between ADHD and autism led several authors to speculate that they might be different phenotypic expressions of one overarching condition; specifically, ADHD could be a less severe form of an overarching condition than ASD (see, for example, [Rommelse et al., 2016, 2018](#); and [van der Meer et al., 2012](#)). Furthermore, research has shown that ADHD and ASD are characterized by both condition-specific and shared brain alterations at the structural and functional levels (e.g., [Hoogman et al., 2022](#); [Lukito et al., 2020](#)), indicating potential overlap in their etiological pathways.

It has been proposed that altered development of oculomotor mechanisms in ADHD and autism, early in life, could affect information gathering/processing and self-regulation skills ([Johnson et al., 2015](#)). Some of the mechanisms that naturally facilitate attention orienting in neurotypicals—e.g., preference for faces ([Farroni et al., 2002](#); [Morand et al., 2010](#)) and increased responsivity to objects presented in multiple sensory modalities ([Steenken et al., 2008](#))—are likely to differ in individuals with ADHD and/or autism (e.g., difficulties in maintaining fixations on salient objects or disengaging visual

attention from distressing stimuli; [Frazier et al., 2017](#); [Kawakami & Otsuka, 2021](#); [Riddiford et al., 2022](#); [Uekermann et al., 2010](#)). Moreover, in neurotypicals, worsening of oculomotor performance has been theorized to arise due to both drowsiness and excessive arousal ([Aston-Jones & Cohen, 2005](#); [Yerkes & Dodson, 1908](#)). Considering that previous research has found signs of autonomic dysregulation in autism and ADHD ([Arora et al., 2021](#); [Bellato et al., 2020](#); [Cheng et al., 2020](#)), it could be speculated that dysregulated arousal could particularly affect oculomotor performance in individuals with these conditions. Amongst the several methods that are used to investigate visual attention and arousal mechanisms, eye-tracking has proven particularly helpful, since it provides markers of oculomotor performance (e.g., Saccadic Response Times, SRTs) and autonomic arousal (e.g., pupil size) recorded simultaneously.

In the first 3–4 months of life, oculomotor mechanisms are predominantly under exogenous control, i.e., infants automatically shift their attention to any object entering and moving within the visual field ([Johnson, Posner, & Rothbart, 1991](#)). It is only around 5–6 months that infants begin to ‘take control’ of visual attention (endogenously). This is supported by the maturation of cortical systems responsible for controlling eye muscles, a process that continues until at least late childhood ([Bucci et al., 2012](#)). In adults, exogenous and endogenous mechanisms of visual attention orienting are mediated by overlapping but anatomically distinct brain systems ([Corbetta & Shulman, 2002](#); [Petersen & Posner, 2012](#); [Posner & Petersen, 1990](#)). The fronto-parietal ventral attentional network, including right superior parietal cortex, temporal–parietal junction, ventromedial PFC, anterior insula, pulvinar nucleus of the thalamus and superior colliculi, underlie reflexive orienting of attention. Conversely, endogenous orienting (which requires voluntary disengagement of attention from a certain object and re-orienting towards another) is supported by the dorsal attentional network, which is comprised of anterior cingulate cortex, basal ganglia, temporoparietal junction, superior parietal lobe and frontal eye fields ([Corbetta & Shulman, 2002](#); [Petersen & Posner, 2012](#); [Posner & Petersen, 1990](#)).

Research has shown altered development and functioning of cortical systems involved in visual attention and oculomotor control in both ADHD ([Shaw et al., 2014](#)) and autism ([Zwaigenbaum & Penner, 2018](#)), suggesting that oculomotor alterations may characterize both conditions; however, differences between ADHD and autism have also been reported.

For example, there is evidence of early problems in visual attention orienting in autistic people,<sup>1</sup> from early in life; including more fragmented saccadic pathways (Keehn et al., 2013), slower disengagement and re-orienting of visual attention (Elsabbagh et al., 2009, 2013; Keehn et al., 2021; Landry & Bryson, 2004; Sacrey et al., 2014; Zwaigenbaum et al., 2007), less accurate and slower attention orienting towards objects presented in the visual periphery (Townsend et al., 2001; Wainwright & Bryson, 2002). Oculomotor difficulties in autism have been attributed to altered functioning of the ventral attentional network and cerebellar systems (Keehn et al., 2016). However, it should be noted that many contradictory findings (either non-significant or suggesting more efficient oculomotor mechanisms in autism) have also been reported (see, for example, Fischer et al., 2016; Kelly et al., 2013; Skripkauskaitė et al., 2021; van der Geest et al., 2001; Zalla et al., 2018), making it difficult to reach a conclusion on the nature of oculomotor difficulties in autism.

In relation to ADHD, recently published systematic reviews and meta-analyses (Chamorro et al., 2022; Maron et al., 2021; Sherigar et al., 2022) found evidence of less accurate and slower saccades during anti-saccade tasks, and more intrusive saccades during fixation tasks, in people with ADHD compared to neurotypical (NT) controls, but no group differences on reflexive saccades. The authors of these meta-analytic studies—in line with the findings of a previous systematic review of the literature (Huang-Pollock & Nigg, 2003) —concluded that oculomotor alterations in ADHD are likely to arise from altered functioning of brain systems responsible for inhibitory oculomotor control, vigilance and working memory (Amso & Scerif, 2015; Cortese et al., 2012; Hart et al., 2013). Increased prevalence of disorders of vision, weaker contrast and color discrimination abilities have also been reported in people with ADHD compared to neurotypical controls (Bellato, Perna et al., 2022). Visual search and social attention have only been investigated by a few studies in people with co-occurring ADHD + autism (see, for example, Seernani, Damania, et al., 2021; 2021b). In these studies, children with ADHD + autism showed worse visual search performance and a different visual processing style (in relation to processing of social and non-social cues) compared to autistic children (without co-occurring ADHD), children with ADHD-only and neurotypicals. Nevertheless, to our knowledge no study has previously compared oculomotor performance during a gap-overlap task between children and adolescents with ADHD, autistic children and adolescents, and children with a dual diagnosis of ADHD + autism.

Amongst the several studies that have been designed to study oculomotor mechanisms in humans, the gap-overlap task (Reulen, 1984; Reuter-Lorenz et al., 1991; Saslow, 1967) —which we adapted and implemented in the current study—is a

simple pro-saccade experimental paradigm that separately triggers reflexive and voluntary mechanisms of visual attention orienting. It usually comprises three conditions: (1) baseline, where a central fixation stimulus is presented and a second visual object appears in the visual periphery as soon as the central stimulus disappears from the screen; (b) gap, where there is a temporal gap between the offset of the central fixation stimulus and the onset of the peripheral; and (c) overlap, where the peripheral stimulus is presented while the central is still on the screen. Baseline and gap conditions trigger reflexive orienting of attention, since disengagement of visual attention from the central object happens automatically due to its disappearance before or with the onset of the peripheral. Conversely, in the overlap condition, voluntary disengagement of visual attention from the central stimulus is required in order to orient attention towards the peripheral stimulus. Considering that endogenous visual attention orienting requires more time compared to exogenous orienting (Kingstone & Klein, 1993; Reuter-Lorenz et al., 1991), SRTs (i.e., the time between the onset of a certain object in the visual periphery and start of an eye movement towards such object) are slower during overlap trials, compared to baseline and gap trials (this is usually referred to as the ‘gap effect’).

The first aim of our study was to investigate reflexive and voluntary mechanisms of visual attention orienting during an adapted gap-overlap task (comprising only baseline and overlap conditions) in children and adolescents with ADHD, autism, co-occurring ADHD + autism, and neurotypicals. Our hypotheses are summarized in Table 2. We expected to find faster SRTs for baseline compared to overlap trials (Hypothesis 1); and slower SRTs for overlap trials in both autistic children and children with ADHD, compared to neurotypicals (Hypothesis 2). Due to the scarcity of previous studies investigating oculomotor performance in children with co-occurring ADHD + autism, we could not specify precise hypotheses for this group. We therefore investigated whether children with ADHD + autism displayed an additive profile of alterations reported in the two conditions or a unique profile compared to children with a single diagnosis.

Humans orient their visual attention more quickly towards face-like stimuli; this is reported in literature as a ‘face salience effect’ that facilitates attention orienting towards faces (Morand et al., 2010) and is evident from the first days of life (Farroni et al., 2002). Furthermore, visual attention orienting is faster when the target stimuli are multi-sensory, e.g., they include visual and auditory information, compared to uni-sensory, e.g., visual only (Steenken et al., 2008). Therefore, our second aim was to test whether SRTs during the gap-overlap task were affected by the salience of the target stimuli, in relation to their social (face compared to non-face) and sensory (uni-sensory compared to multi-sensory) features, and whether these effects differed across diagnostic groups (children with ADHD, autistic children, children with a dual diagnosis of ADHD + autism, and neurotypicals).

Several studies have previously reported that autistic children, adolescents, and adults demonstrate reduced attention allocation to face-like visual stimuli, or at least to certain parts of the face (Frazier et al., 2017; Riddiford et al., 2022). Although there is not much literature on face processing in ADHD, there is some evidence of altered face processing

<sup>1</sup> We acknowledge that there have been some debates, within the scientific community, about the use of ‘person-first’ or ‘identity-first’ language to refer to people who receive a clinical diagnosis of Autism Spectrum Disorder. In this paper, we decided to use ‘autistic people’ since this is what the majority of a sample of autistic adults, family members or friends and parents of an autistic person have reported to prefer in an online survey conducted in the UK (Kenny et al., 2006). We are aware, though, that this may not be the case for other countries.

(Uekermann et al., 2010) but also evidence suggesting that altered orienting of attention to faces is specifically associated with autistic traits, not ADHD (Groom et al., 2017). Autism has been associated with difficulties in multi-sensory integration, especially in relation to audio-visual integration (Kawakami & Otsuka, 2021). Conversely, difficulties in multi-sensory integration have not been reported in individuals with ADHD, who could instead benefit from presentation of information in multiple compared to single modalities if this increases the novelty or salience of those stimuli (McCracken et al., 2020).

Overall, we expected to find longer SRTs to orient attention towards non-face stimuli, especially in the overlap condition (Hypothesis 3); and faster orienting of attention towards stimuli presented multi-modally compared to uni-modally (Hypothesis 4). Based on the literature, we expected the face salience effect (faster orienting of attention to faces than shapes) to be stronger in non-autistic children (with a single diagnosis of ADHD or neurotypicals), and to be reduced or absent in autistic children (Hypothesis 5). In relation to modality, we predicted that multi-modal stimuli would elicit faster SRTs in children with ADHD, compared to without ADHD (i.e., autistic children with a single diagnosis and neurotypicals) (Hypothesis 6). Lastly, we predicted slower attention orienting to multi-modal stimuli in autistic children (with and without ADHD), but not in non-autistic children (i.e., neurotypicals and children with ADHD) (Hypothesis 7).

Previous research has shown an inverted U-shaped relationship between arousal and attentional performance (Aston-Jones & Cohen, 2005; Yerkes & Dodson, 1908) in manual motor tasks (Gilzenrat et al., 2010; Murphy et al., 2011). This could, at least theoretically, explain why motor responses (including oculomotor) are likely to be slower during periods of hypo-arousal and drowsiness, while more impulsive and less accurate responses seem to characterize hyper-arousal (Howells et al., 2012). However, previous research investigating the role of arousal in oculomotor mechanisms has not found evidence in support of the Aston-Jones & Cohen's model. Conversely, a linear relationship between pre-saccadic pupil size and saccadic latencies has been proposed, with smaller pupil size predicting slower SRTs suggesting that temporary drops in arousal (e.g., due to boredom or drowsiness) are associated with slower oculomotor performance (Jainta et al., 2011; Yamagishi & Furukawa, 2020).

Previous research has also identified a role for altered autonomic arousal regulation in explaining cognitive differences in ADHD (Bellato et al., 2020) and autism (Arora et al., 2021; Cheng et al., 2020), suggesting that an association between dysregulated arousal and oculomotor performance could be especially evident in individuals with ADHD and/or autism, compared to neurotypicals. For example, it has been found that a group of autistic children who displayed hyper-arousal (i.e., larger resting-state pupil diameter) had slower SRTs during overlap trials of the gap-overlap task (Keehn et al., 2021), while in children with ADHD temporary increases in arousal (reflected in pupil dilations) were found to elicit shorter SRTs (Kleberg et al., 2020).

We therefore aimed to test whether oculomotor performance (i.e., SRTs) was predicted by an index of arousal (pupil size measured before the saccade) (Hypothesis 8). We modelled linear and non-linear (quadratic) equations between

pre-saccadic pupil size and SRTs to verify if there was (1) a quadratic relationship between the two variables, with smallest and largest pupil sizes associated with longer SRTs (in support of the Aston-Jones & Cohen's model); (2) a negative or (3) positive linear relationship between the two variables; or (4) no relationship between pre-saccadic pupil size and SRTs. Moreover, we took an exploratory approach to understand if the association between pre-saccadic pupil size and SRTs differed between children with a diagnosis of ADHD and/or autism, compared to neurotypicals (Hypothesis 9).

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## 2. Materials and methods

### 2.1. Sample characteristics, recruitment and ethical approval

The present paper is based on data collected for the SAAND study (Studying Attention and Arousal in children and adolescents with Neurodevelopmental Disorders), carried out at the University of Nottingham (UK) between September 2017 and May 2020. Ethical approval for the study was obtained from the UK National Research Ethics Committee and the Health Research Authority.

Children between 7 and 15 years of age diagnosed with or under clinical assessment for autism and/or ADHD, and neurotypical children from the local community, were recruited (see Table 1 for a summary). Children under pharmacological treatment for ADHD with stimulants were required to withdraw their medication for at least 24 h before the testing session. Potential participants were excluded if they had any neurological conditions; if they or their parent/legal guardians were unhappy with stimulant medication being withdrawn for 24 h; if they were on non-stimulant medication (for example, atomoxetine, guanfacine or clonidine), for which temporarily withdrawal was not possible; or if they did not speak fluent English. Children were not excluded if they had a co-occurring diagnosis of mental health conditions (including Anxiety, Depression, Oppositional Defiant or Conduct Disorder), or intellectual disability (children with IQ < 70 were not excluded from the study). Children recruited as neurotypical comparison participants were not included if they were siblings of a child with a clinical diagnosis or if they exhibited clinically significant symptoms on any measures.

### 2.2. Clinical assessment

Symptoms of ADHD and autism were evaluated using parent- and teacher-report Conners' Rating Scales (CRS-3; Conners, 2008) and Social Communication Questionnaire (SCQ; Berument et al., 1999; Rutter et al., 2003), respectively. Further, the Autism Diagnostic Observation Schedule, Second Edition (ADOS-2; Lord et al., 2012) was administered by IA and PK (both accredited with research reliability), and a proportion of reports of ADOS conducted by IA were checked for consistency by a Consultant Child & Adolescent Psychiatrist (PK). Parents also completed the Development and Well-Being Assessment (DAWBA; Goodman et al., 2000) and the Strengths and Difficulties Questionnaire (SDQ; Goodman, 2001), which gave a computer-generated profile of children

Table 1 – Main socio-demographic and clinical characteristics of the sample.

	NT	Autism-only	ADHD-only	ADHD + Autism	Group differences
N	31	18	24	33	–
% Males	58%	61%	67%	76%	None <sup>a</sup>
Age (years) [SD]	10.89 [2.45]	10.91 [2.09]	10.57 [2.25]	10.86 [1.51]	None <sup>b</sup>
WASI – FSIQ [SD]	116.26 [13.09]	104.61 [15.64]	108.12 [11.65]	101.85 [19.02]	ADHD + autism < NT <sup>b</sup>
SCQ – Total score [SD]	5.10 [7.64]	19.11 [5.98]	15.29 [6.83]	21.06 [6.16]	NT < (ADHD-only, autism-only, ADHD + autism) <sup>b</sup>
CRS-3 – ADHD Global Index (T score) [SD]	47.97 [8.36]	79.44 [12.59]	87.96 [4.18]	87.21 [5.26]	ADHD + autism > ADHD-only <sup>b</sup>
Co-occurring diagnoses (N per group)					NT < autism-only < (ADHD-only, ADHD + autism) <sup>b</sup>
Anxiety	–	10 [56%]	6 [25%]	15 [45%]	
Depression	–	3 [17%]	1 [4%]	6 [18%]	
CD/ODD	–	11 [61%]	17 [71%]	22 [67%]	
Tics	–	1 [6%]	2 [8%]	4 [12%]	

Abbreviations. CD/ODD: Conduct and Oppositional Defiant Disorder. CRS: Conner's Rating Scales. FSIQ: Full Scale Intelligence Quotient. NT: Neurotypicals. SCQ: Social Communication Questionnaire. SD: Standard Deviation. WASI: Wechsler Abbreviated Scale of Intelligence.

<sup>a</sup> Chi-Squared Test.

<sup>b</sup> Univariate ANOVA (pairwise comparisons).

and adolescents' behavioural and emotional characteristics, prosocial behaviours and predicted clinical diagnoses. Parents also completed the Child Sensory Profile 2 (Dunn, 2014) to provide a measure of their child's sensory processing profile. The Wechsler Abbreviated Scale of Intelligence (WASI-II; Wechsler, 2011) was administered to obtain a measure of intellectual functioning. Using all available information (CRS-3, SCQ, ADOS, DAWBA, SDQ, Sensory Profile, and WASI), a consensus diagnosis of autism and/or ADHD was confirmed by two clinicians (CH, PK).

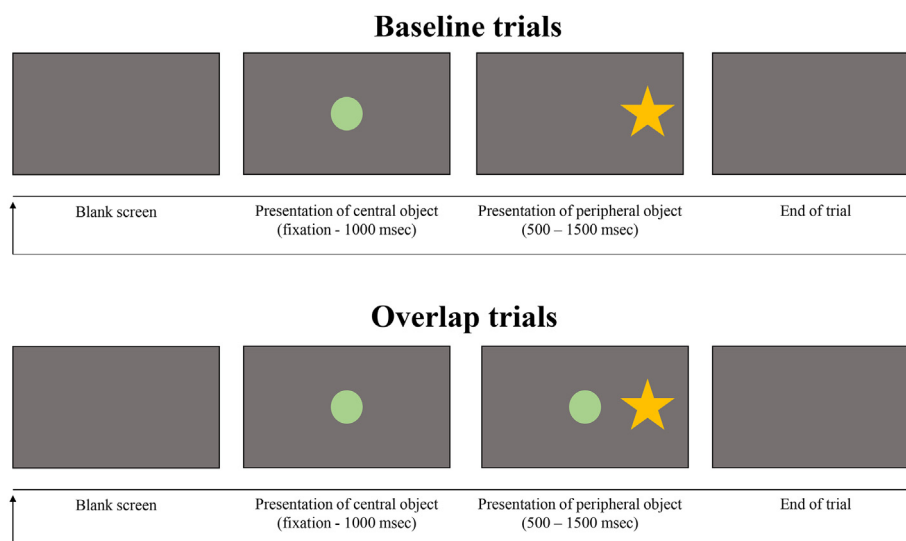
### 2.3. Oculomotor task

The testing session of the SAAND study included a battery of EEG and eye-tracking tasks. In this paper, we report results from the gap-overlap task, which is part of the eye-tracking battery. Results from other tasks—included in the eye-tracking and the EEG battery—have been published elsewhere (Arora et al., 2022; Bellato, Arora et al., 2022; Bellato et al., 2021). The oculomotor task design was based on a review of methods used in the autistic and ADHD literature. The choice of using social and non-social stimuli (presented in single or multiple modalities) was in line with studies conducted on autistic children or siblings of autistic children (e.g., Elsabbagh et al., 2009).

In our adapted version of the gap-overlap task (see Fig. 1), the central stimulus was a color-filled circle with a white cross in the middle, positioned at the center of a uniform dark grey background. The central stimulus expanded and contracted at regular intervals (expanding for the first 500 msec, contracting for another 500 msec, and so on), until the eye tracker detected that the participant had fixated the central stimulus continuously for 1000 msec. At that point, a peripheral stimulus appeared for a variable duration of 500- to 1500-msec before a blank screen was presented and a new trial started. We manipulated three main variables of the task: (a) Condition (baseline, overlap), (b) Stimulus (face, non-face) and (c) Modality (uni-modal and multi-modal).

In the baseline condition, the peripheral visual stimulus appeared immediately after the disappearance of the central object, while in the overlap condition the central visual stimulus remained onscreen after the presentation of the peripheral object (thus, there was a temporal overlap of both stimuli presented on the screen). The presentation of visual stimuli in the baseline condition has been found to elicit a quick reflexive orienting response, with eye movement latencies in the range 100–200 msec (Bekkering et al., 1996; Fischer & Ramsperger, 1984), reflecting the involvement of the ventral attentional network. Conversely, in the overlap condition the dorsal attentional network is primarily involved in facilitating the voluntary disengagement of attention from the central object and initiating a saccade towards the peripheral visual object. This results in longer eye movements' latencies during the overlap condition, usually in the range of 200–250 msec.

Peripheral stimuli were either human faces or geometrical shapes. Face-stimuli were selected and adapted from the UvA-NEMO Smile Database (Dibeklioglu et al., 2012). This database was selected over other databases because it includes videos which were useable both as a static picture and as a video) and the videos are recorded at high resolution and in a controlled



**Fig. 1 – Gap-overlap task diagram.**

environment with an artificially illuminated background. Twelve different video stimuli were selected from the UvA-NEMO Smile Database representing different ages and genders. Six three-dimensional shapes following different rotation patterns were artificially created with CINEMA 4D (Maxon Computer) to provide a control condition against which to compare the faces.

In the uni-modal condition, the central and peripheral stimuli were presented as static pictures without any sounds, while in the multi-modal condition custom sounds were presented together with the visual objects, which were also dynamic (i.e., the faces moved, and the shapes rotated). Non-speech human sounds, defined as ‘social’, (e.g., laughing), and non-human sounds defined as ‘non-social’ (e.g., tweeting birds), were downloaded from an online database of sound effects (<http://soundbible.com>) and balanced in terms of duration and volume, to create multi-modal stimuli. Specifically, ‘social’ sounds accompanied face-stimuli, while ‘non-social’ sounds accompanied 3D shapes. Some short creative commons cartoons, downloaded from [www.google.co.uk](http://www.google.co.uk), were used to create 30-sec video breaks. These were presented to participants once every 7 trials (see next paragraph for full details); participants were instructed to take a break from the task and watch the short cartoon.

#### 2.4. Procedure, data processing and outcome measures

A nine-points-of-gaze calibration was initially carried out by presenting an attractive colorful stimulus in the center and eight peripheral points of the computer screen. Participants’ eye movements were recorded through an Eyelink® 1000 (SR Research) eye-tracking system. Participants’ distance from the screen was measured and kept within the range recommended by Eyelink using stickers placed on the participants’ forehead (a chinrest was not used for this task, but we could monitor, via the Eyelink software, whether the participant was too close or too far away from the screen). Eye movements

from both eyes were recorded at 500 Hz through a 25-mm lens, without the use of any chinrest, from an average distance of 60 cm and with an estimated accuracy of  $.25^{\circ}$ – $.5^{\circ}$ . The gap-overlap task was delivered on a 21.5’ LCD screen with 60 Hz refresh rate, placed behind the eye-tracking device. Luminance was kept constant across the entire sample of participants and, in parallel, screen brightness was kept constant as well. The gap-overlap task was comprised of 12 blocks of 7 trials each, divided by video breaks of 6-s duration, leading to a total of 84 task trials. Initially, the task was designed to include 144 trials. However, pilot-testing with a neurotypical sample revealed that the task was too long for children in our target age range. We therefore reduced the length to 12 blocks of 7 trials. This, however, led to a slight imprecision in counterbalancing of the eight task conditions (i.e., some conditions were presented 10 times during the overall task, and some 11, but in total, the number of trials was equivalent between the gap and overlap conditions, and the social and non-social conditions). The order of presentation of trials was randomized. The eye-tracking session, including calibration and gap-overlap task, lasted between 15 and 20 min.

The following exclusion criteria were used to discard invalid trials (the procedure for defining trials as valid/invalid was applied in Excel after exporting raw data from the Eyelink data analysis software): 1) anticipation, i.e., the saccade towards the peripheral stimulus location occurred before stimulus-onset; 2) absence of a saccade towards the peripheral stimulus, or in the direction opposite to the peripheral stimulus; 3) SRTs shorter than 80 msec, which are likely to characterize eye motor reflexes, instead of eye movements (Hess et al., 1946); 4) data loss due to technical problems. The number of valid trials did not differ between groups,  $p = .335$ ; ADHD: 90% valid trials; Autism: 90%, ADHD + Autism: 93%, Neurotypicals: 93%.

The outcome measures extracted from the raw eye-tracking data were SRTs and pupil size. SRTs were calculated for each valid trial, as the time (in milliseconds) between

the onset of a peripheral stimulus and the start of an eye movement (i.e., a saccade) from the fixated central object towards the peripheral stimulus (Johnson et al., 1991). We used the EyeLink data analysis software to obtain pre-saccadic right-eye pupil size data in the temporal period between the onset of the central fixation stimulus and the start of the saccade towards the peripheral visual object (for overlap trials, this included the time between the onset of peripheral stimulus and start of saccade). The EyeLink software detects blinks and applies an algorithm to interpolate data around the time period of the blink, ensuring that the blink does not confound data analysis, and that data loss due to blinks is minimized.

## 2.5. Analysis plan and statistical tests

Main effects of ADHD and autism were investigated using binomial between-subjects factors of ADHD and Autism (0 = absent; 1 = present) reflecting the presence (or absence) of a diagnosis of ADHD or autism. In this way, we could compare children with and without ADHD (0: NT and autism-only; 1: ADHD-only and ADHD + autism), and with or without autism (0: NT and ADHD-only; 1: autism-only and ADHD + autism), to test condition-specific effects on the outcome measures (see Table 2 for a summary of specific hypotheses). In this way, we could also investigate the impact of co-occurring autism and ADHD, and compare the group of children with co-occurring ADHD and autism to the other groups. When following-up significant interactions between ADHD and Autism factors, we calculated *P*-values adjusted for multiple comparisons using the Benjamini-Hochberg (BH) method (Benjamini & Hochberg, 1995). Greenhouse-Geisser adjusted degrees of freedom are reported for those variables for which we found violation of sphericity, which was evaluated through Mauchly's tests.

We analyzed SRTs through a repeated measures ANOVA, with Condition (2-levels; baseline, overlap), type of peripheral Stimulus (2-levels; face, non-face) and Modality of presentation (2-levels; uni-modal, multi-modal) which were added to the model as within-subjects factors, while ADHD and ASD (2-levels: yes/no) were included as between-subjects factors. Considering we did not expect any effect of Condition, Stimulus or Modality on pupil size, we used a univariate ANOVA to analyze pupil size averaged across trials for each participant, with ADHD and Autism (2-levels: yes/no) included as between-subjects factors. In all analyses, sample-mean-centered age, sex, verbal and performance IQ were included as covariates. Results were considered statistically significant if a *p* value  $\leq .05$  was reported. Partial eta-squared ( $\eta_p^2$ ) was used to calculate *f* as a measure of effect size. *f* is usually interpreted as follows: .10 indicates a small effect, .15 a medium effect and .20 a large effect (Cohen, 1988).

To investigate the single trial association between pre-saccadic pupil size and SRTs, we standardized pupil size and SRTs for each participant based on individual's average and SD (considering that absolute values differed between participants; this is in line with what was done in Jainta et al., 2011) and investigated linear and non-linear associations between these two variables. Specifically, we used the Curve Fitting function in SPSS to investigate if a linear or non-linear

(i.e., quadratic) function better fitted the data; this was done for all trials, initially, and secondly, by calculating equations for each diagnostic groups. A summary of a-priori defined hypotheses and statistical tests used to investigate such hypotheses, is reported in Table 2.

## 2.6. Pre-registration and data availability

No part of the study procedures or analyses was pre-registered prior to the research being conducted. We reported, above, how we determined our sample size, all data exclusions, all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all measures in the study. Data analyzed in this manuscript (including trial-by-trial outputs, per participant), task stimuli and full SPSS outputs, are available at <https://osf.io/5jshw/>.

## 3. Results

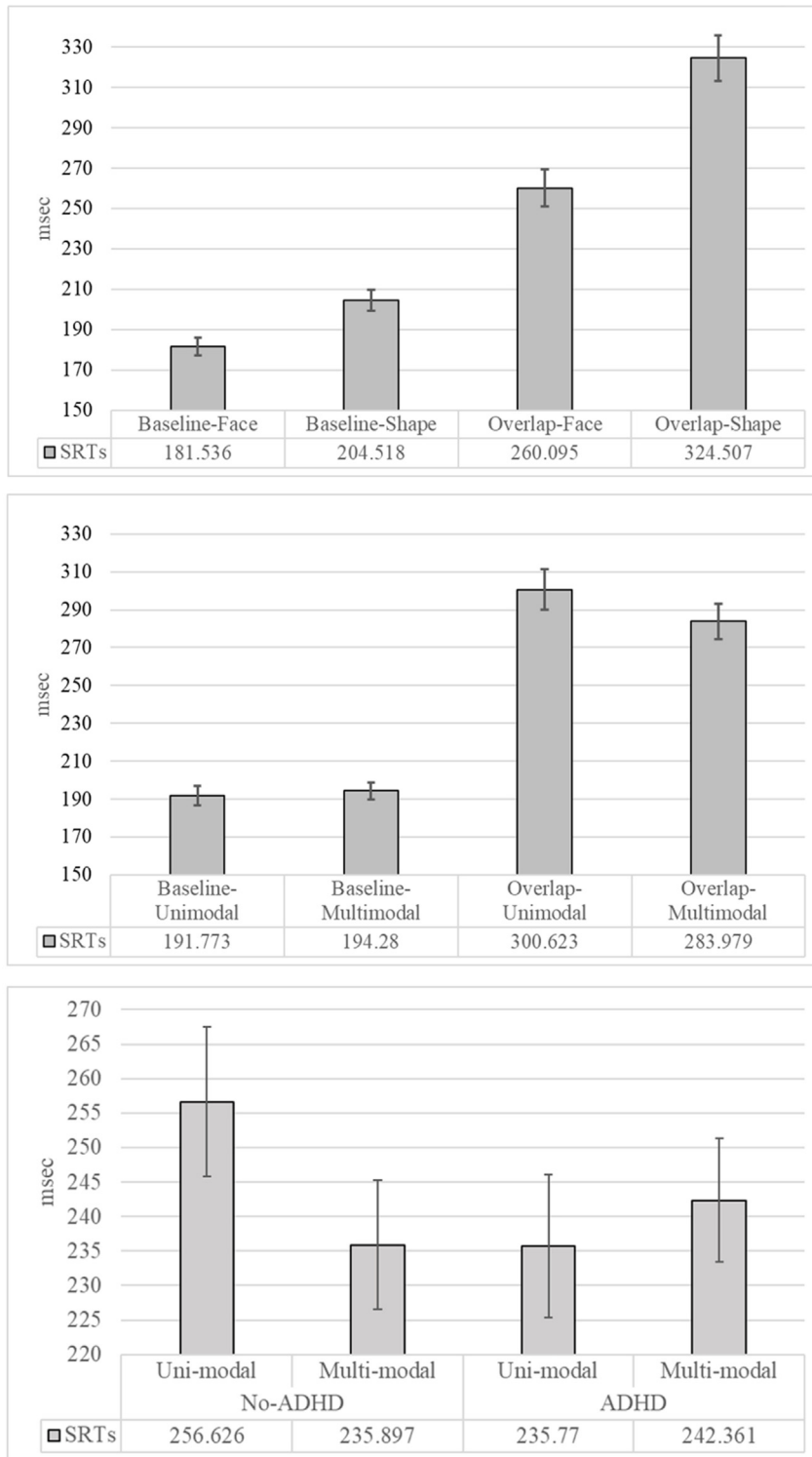
### 3.1. Sample characteristics

Table 1 summarizes the main characteristics of the final sample (106 children and adolescents: mean age = 10.81 years, SD = 2.06 years; 66% males). For the present study, data was available and valid for 99 participants (mean age = 10.79 years, SD = 2.05 years; 65% males; no statistically significant differences between overall sample and sample analyzed here). Twenty-nine children were neurotypicals (NT); 21 were diagnosed with ADHD (but not autism); 18 were autistic but did not have ADHD; and 31 met criteria for co-occurring ADHD + autism.

### 3.2. Saccadic reaction times

SRTs were shorter, indicating faster orienting of attention, on baseline than overlap trials ( $F_{1,86} = 194.35$ ;  $p < .001$ ;  $f = 1.49$ ; mean difference = 99.27 msec), and for face stimuli compared to shapes (main effect of Stimulus:  $F_{1,86} = 68.24$ ;  $p < .001$ ;  $f = .89$ ; mean difference = 43.697). We also found a statistically significant interaction between Condition and Stimulus ( $F_{1,86} = 17.644$ ;  $p < .001$ ;  $f = .45$ ) and between Condition and Modality ( $F_{1,86} = 6.697$ ;  $p = .011$ ;  $f = .28$ ). Orienting of attention was fastest towards faces during baseline trials and slowest for non-face stimuli during overlap trials (Fig. 2). Furthermore, an effect of Modality (i.e., faster SRTs during multi-modal compared to uni-modal trials) was detected for overlap trials but not for baseline trials (baseline: mean difference = 2.51 msec,  $p = .528$ ; overlap: mean difference = 16.644 msec,  $p = .035$ ) (Fig. 2). These findings support Hypotheses 1, 3 and 4 (Table 2). Hypothesis 2 was not supported by our data, since the interaction between Condition, Autism and ADHD was non-significant ( $F_{1,86} = 1.552$ ;  $p = .216$ ;  $f = .14$ ). Similarly, Hypothesis 5 was not confirmed, since the interaction Stimulus  $\times$  Autism was non-significant ( $F_{1,86} = 1.636$ ;  $p = .204$ ;  $f = .14$ ).

In relation to Hypothesis 6, we found a statistically significant interaction between Modality and ADHD ( $F_{1,86} = 7.575$ ;  $p = .007$ ;  $f = .30$ ). Nevertheless, upon further exploration our initial hypothesis was not confirmed. In fact, although in



**Fig. 2 – Average SRTs for: (a) face and non-face stimuli, during baseline and overlap trials (top); (b) uni-modal and multi-modal stimuli, during gap and overlap trials (middle); (c) uni-modal and multi-modal stimuli, for ADHD (ADHD-only and ADHD + autism) and no-ADHD (neurotypicals and autism-only) participants (bottom). Error bars represent S.E.**

**Table 2 – Summary of statistical hypotheses of the study.**

Hypothesis	Statistical test used and effect investigated	Hypothesis confirmed	Main study findings
1 Shorter SRTs during Gap compared to Overlap trials	Repeated measures ANOVA on SRTs: main effect of Condition	Yes	Faster attention orienting during Gap compared to Overlap trials
2 Shorter SRTs for Overlap trials in neurotypicals, compared to autistic children and children with ADHD	Repeated measures ANOVA on SRTs: interaction Condition x Autism x ADHD	No	No differences between neurotypicals and children with a diagnosis of ADHD and/or Autism on attention orienting during Overlap trials
3 Shorter SRTs towards Face-stimuli compared with shapes, with this difference being larger in the overlap than baseline condition	Repeated measures ANOVA on SRTs: interaction Condition x Stimulus	Yes	Faster attention orienting towards Face-stimuli compared to Shape-stimuli. This effect was more evident in Overlap trials compared to Baseline trials.
4 Shorter SRTs during Multi-modal compared to Uni-modal trials	Repeated measures ANOVA on SRTs: main effect of Modality	Partly	Faster attention orienting during Multi-modal compared to Uni-modal trials in Overlap but not Gap trials
5 Stronger face salience effect in non-autistic children, compared to autistic children	Repeated measures ANOVA on SRTs: interaction Stimulus x Autism	No	No differences between autistic and non-autistic participants on attention orienting to Face or Shapes
6 Shorter SRTs during Multi-modal trials in children with ADHD, compared to without	Repeated measures ANOVA on SRTs: interaction Modality x ADHD	No	Although an interaction Modality x ADHD was significant, no differences between children with and without ADHD on SRTs during Multi-modal trials.
7 Longer SRTs during Multi-modal trials in autistic children compared to non-autistic	Repeated measures ANOVA on SRTs: interaction Modality x Autism	No	No differences between autistic and non-autistic children on SRTs during Multi-modal trials.
8 Linear or quadratic relationships between SRTs and pre-saccadic pupil size	Curve fitting analysis: statistically significant linear and/or quadratic relationship between pre-saccadic pupil size and SRTs (exploratory analyses aimed at investigating effects of diagnostic group)	Partly	Negative linear association between pre-saccadic pupil size and SRTs, but only in children with a single diagnosis of ADHD and autism.
9 Different relationship between pre-saccadic pupil size and SRTs in children with a diagnosis of ADHD and/or autism compared to neurotypicals			

children without ADHD (neurotypical and Autism groups) we detected an effect of Modality, with faster attention orienting towards multi-modal compared to uni-modal stimuli (mean difference = 20.729 msec;  $p = .004$ ), and this effect was not statistically significant in children with ADHD (ADHD and ADHD + Autism groups) (mean difference = 6.591 msec;  $p = .329$ ); there were no differences between children with and without ADHD on SRTs during multi-modal trials (mean difference = 6.464 msec,  $p = .628$ ) (Fig. 2). The interaction between Modality and Autism was non-significant ( $F_{1,86} = 2.109$ ;  $p = .150$ ;  $f = .16$ ), as the interaction between Condition, Modality, and Autism ( $F_{1,86} = 3.838$ ;  $p = .053$ ;  $f = .21$ ), suggesting that Hypothesis 7 was not supported by data.

### 3.3. Relationship between pre-saccadic pupil size and SRTs

We did not find any statistically significant effect of either ADHD ( $F_{1,89} = .02$ ;  $p = .89$ ;  $f < .03$ ), Autism ( $F_{1,89} = .76$ ;  $p = .39$ ;  $f < .03$ ), or a statistically significant interaction between ADHD and Autism ( $F_{1,89} = .11$ ;  $p = .75$ ;  $f < .03$ ) on average pre-saccadic pupil size, suggesting that groups did not differ on this variable.

Curve fitting analysis highlighted statistically significant linear ( $F_{1,7636} = 17.125$ ,  $R^2 = .002$ ,  $p < .001$ ) and quadratic ( $F_{1,7636} = 8.660$ ,  $R^2 = .002$ ,  $p < .001$ ) associations between pre-saccadic pupil size and SRTs (Hypothesis 8), and this was not detected in all groups (Hypothesis 9). For neurotypicals and those with a dual diagnosis of ADHD + Autism, none of the models (either linear or quadratic) significantly fit the data. For autistic children (single diagnosis), a linear model significantly explained the relationship between pre-saccadic pupil-size and SRTs (negative linear coefficient: smaller pupil size associated with slower SRTs). Lastly, for those with ADHD (single diagnosis), both a linear and a quadratic model significantly fit the data; there was a negative linear association between pre-saccadic pupil size and SRTs and, in line with the Aston–Jones & Cohen inverted U-shaped model, a significant quadratic relationship whereby SRTs were slowest when pupil size was either the smallest or the largest (see Table 3 and Fig. 3 for full results).

## 4. Discussion

We conducted a study to investigate oculomotor mechanisms during a gap-overlap task in children and adolescents with ADHD, autism, co-occurring ADHD + autism, and neurotypicals. We tested whether visual attention orienting was affected by the social and/or sensory nature of the stimuli, and we explored the association between pre-saccadic pupil size (an index of autonomic arousal and vigilance) and saccadic reaction times (SRTs; a measure of oculomotor performance).

We tested several hypotheses, summarized in Table 2. Overall, our task-related hypotheses were all confirmed, but none of those in relation to ADHD and/or autism were supported. We found faster attention orienting during baseline trials (Hypothesis 1), with slowest orienting of attention towards non-faces during overlap trials and fastest orienting to faces during baseline trials (Hypothesis 3). These findings are

in line with the existing literature and confirm, once again, the existence of a ‘gap effect’ (Kingstone & Klein, 1993; Reuter-Lorenz et al., 1991) and a ‘face salience effect’ (Morand et al., 2010). We also found evidence in support of the idea that multi-sensory information triggers faster attention orienting compared to uni-sensory information (Steenken et al., 2008) (Hypothesis 4); however, this was only found for overlap trials.

Contrary to our expectations, we did not find evidence of altered oculomotor mechanisms in children with a diagnosis of ADHD and/or autism. Surprisingly, but in line with most recent literature, we did not find any evidence of slower attention orienting during overlap trials (which would have suggested difficulties in attentional disengagement) in children with ADHD or autistic children (Hypothesis 2). Similarly, we did not find evidence of altered orienting to faces in autistic children (Hypothesis 5), or different oculomotor performance during multi-modal trials in children with ADHD (Hypothesis 6) or autistic children (Hypothesis 7).

As suggested by our research notes/observation and by previous literature (e.g., Bellato et al., 2020), and as reported for another task of the EEG battery in the same sample (Bellato et al., 2021), we speculate that children with ADHD were in a state of general hypo-arousal during the gap-overlap task, reflecting generally poor engagement with the task. Children with ADHD may have focused on just completing the activity (for which, the only instruction was—literally—to ‘look at any object you see on the screen’) very quickly and were therefore less influenced by the physical differences between the visual stimuli presented. Nevertheless, this is only a speculation, considering that no group differences were found on the only measure of autonomic arousal that was collected during the gap-overlap task, i.e., pupil size.

These non-significant effects might reflect our use of a gap-overlap task which was possibly too simple and passive to sufficiently challenge visual attention and arousal regulation in ADHD and autism (see, for example, Chamorro et al., 2022; Maron et al., 2021; Elsabbagh et al., 2009, 2013; Huang-Pollock & Nigg, 2003; Keehn et al., 2013, 2021; Sherigar et al., 2022; Zwaigenbaum et al., 2007). Effects of ADHD and autism on oculomotor mechanisms may be context- and task-dependent, possibly becoming more evident in naturalistic settings and when more complex tasks, e.g., visual search or habituation tasks, or tasks challenging response inhibition, e.g., anti-saccade task, are implemented. This idea is partly supported by the fact that, in another task from the same eye-tracking battery of the SAAND study (Arora et al., 2022), we found evidence of increased difficulty in processing complex or unpredictable visual information associated with autism. In future studies, it will be important to utilize other measures of engagement and visual processing (e.g., event-related potentials), besides eye-tracking measures, to further clarify at what stage alterations in visual processing emerge (if any) in individuals with a diagnosis of ADHD and/or autism.

We analyzed the trial-by-trial association between pre-saccadic pupil size and saccadic latencies: a negative linear relationship between pre-saccadic pupil size and SRTs was found, but this was not detected in all groups. In those with ADHD, we found evidence of a quadratic relationship in support of the Aston–Jones & Cohen model, with longer SRTs

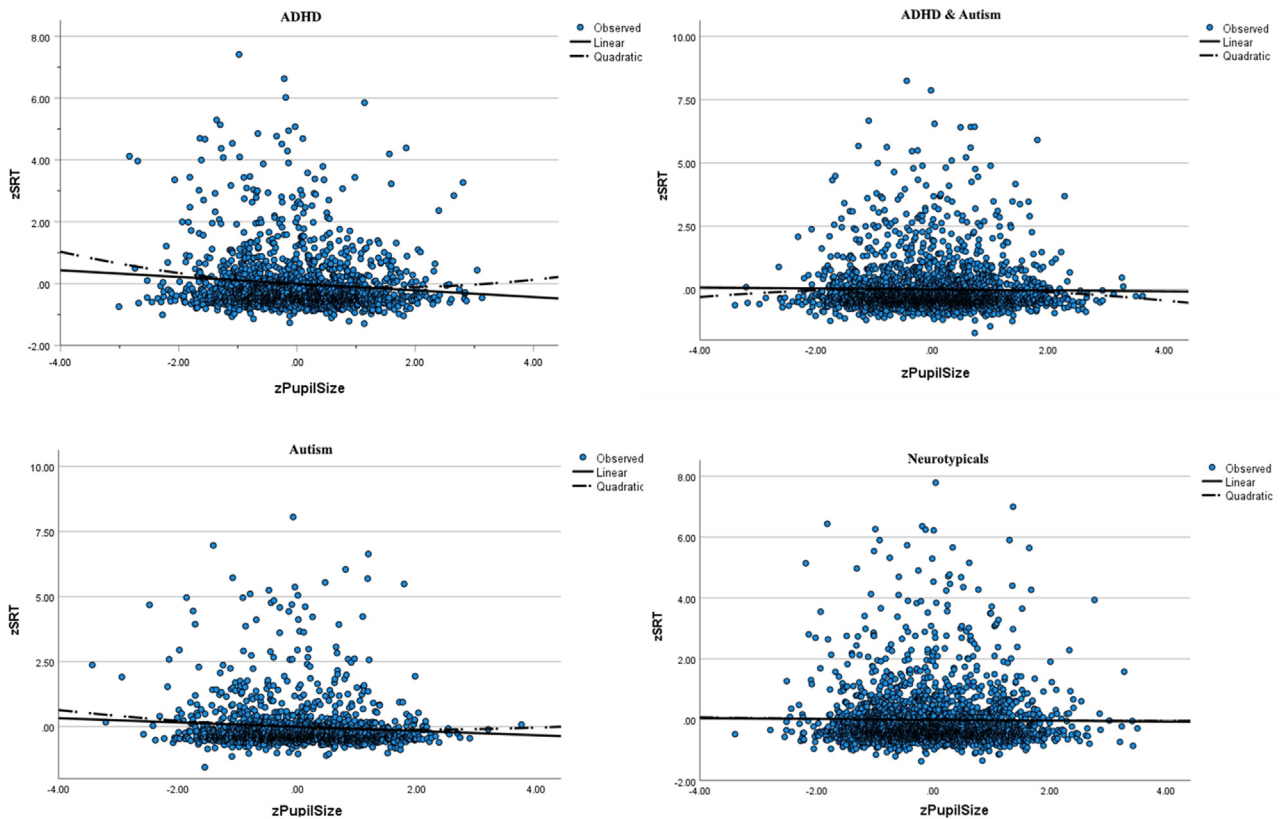
**Table 3 – Summary of models and parameter estimates for the curve fitting analyses.**

Group	R	R2	Adjusted R2	Standard Error Estimate	Coefficient	B	Standard Error B	Beta	t	p
<b>Linear model</b>										
ADHD	.107	.012	.011	.988	Linear (x)	-.108	.025	-.107	-4.303	<.001 <sup>a</sup>
ASD	.082	.007	.006	.991	Linear (x)	-.082	.027	-.082	-3.028	.003 <sup>a</sup>
ASD&ADHD	.019	<.001	.000	.994	Linear (x)	-.019	.020	-.019	-.951	.342
Neurotypicals	.015	<.001	.000	.994	Linear (x)	-.015	.021	-.015	-.696	.487
<b>Quadratic model</b>										
ADHD	.119	.014	.013	.987	Linear (x)	-.113	.025	-.112	-4.488	<.001 <sup>a</sup>
					Quadratic (x2)	.039	.019	.051	2.047	.041 <sup>a</sup>
ASD	.086	.007	.006	.991	Linear (x)	-.084	.027	-.084	-3.112	.002 <sup>a</sup>
					Quadratic (x2)	.02	.02	.027	.986	0.324
ASD&ADHD	.039	.002	.001	.993	Linear (x)	-.017	.02	-.017	-.814	.416
					Quadratic (x2)	-.025	.015	-.034	-1.664	.096
Neurotypicals	.015	0	-.001	.994	Linear (x)	-.015	0.021	-.015	-.7	.484
					Quadratic (x2)	0.001	0.016	.002	.086	.932

Dependent Variable: Standardized SRTs; Independent Variable: Standardized Pupil Size.  
 Beta: standardized regression coefficients.  
<sup>a</sup> Statistically significant at  $p < .05$ .

being predicted by either small or large pre-saccadic pupil size. In autistic participants, we found that slower oculomotor performance was predicted by smaller pupil size, while no association between pre-saccadic pupil size and SRTs was found in neurotypical children and adolescents.

These findings are challenging to interpret, but there is some ground to derive preliminary conclusions. It may be that the Aston–Jones & Cohen's model is only valid in some experimental or daily life situations, i.e., only when arousal and vigilance mechanisms are truly challenged, and



**Fig. 3 – Graphical representation of the linear and quadratic associations between standardized pre-saccadic pupil size (zPupilSize) and SRTs (zSRT), for each diagnostic group.**

behavioral/cognitive strategies for regulating arousal should be implemented. This could be the reason why we did not find any linear or quadratic association between pre-saccadic pupil size and SRTs in neurotypical participants, because the task was not challenging enough to influence arousal and attention in this group. For another task of our testing battery (passive auditory attention task) we had found that participants with ADHD showed signs of hypo-arousal (Bellato et al., 2021). Considering the similarity with the gap-overlap task (no ‘action’ was required to complete the task), we speculate that low vigilance/arousal, mind-wandering (commonly reported in ADHD; Frick et al., 2020) and dysregulated—therefore, more variable—autonomic arousal (reflected in smallest and largest pupil size) may have affected oculomotor performance in children with ADHD. In autistic participants, it seems that increased arousal facilitated attention orienting, considering that largest pupil size was associated with fastest SRTs, in line with the findings from previous studies (Jainta et al., 2011; Yamagishi & Furukawa, 2020). Future studies should aim at investigating the relationship between autonomic arousal and oculomotor attentional mechanisms in neurotypicals and clinical groups, in more details, e.g., by designing eye-tracking tasks that challenge arousal regulation at different levels.

We did not find any evidence in support of either the additive or interactive models of ADHD/autism co-occurrence, considering that no group differences were detected on any of the measures or effects investigated. It is challenging to interpret why in children with a dual diagnosis no association between pre-saccadic pupil size and SRTs was found, considering that significant effects were found in children with a single diagnosis. It could be that this group was too heterogeneous (it was indeed the group with most reported comorbidities, including anxiety and depression, conduct problems and tics) for any condition-specific pattern to emerge, while children with a single diagnosis of either ADHD or autism were more homogeneous in their behavioral phenotype and arousal profile. Previous studies have also postulated that there may be more than one type of double diagnosis (e.g., primarily ADHD with autism as secondary presentation, or primarily ASD with co-occurring ADHD), suggesting another form of heterogeneity within the comorbid group (van der Meer et al., 2012), which we advise to further investigate in future research on large samples of children with ADHD and/or autism.

In another set of tasks from the same study (i.e., EEG battery), we found that children with ADHD + autism showed the same arousal profile as children with a single diagnosis, i.e., hypo-arousal during resting-state and a passive auditory attention task (as children with ADHD-only), and hyper-arousal during the active response conflict task (as in children with a single diagnosis of autism) (Bellato et al., 2021). It could be that for some children, within the ADHD + autism group, the gap-overlap task did not challenge arousal regulation, while others may have experienced hypo- or hyper-arousal, making it difficult for a clear association between pupil size and SRTs to become evident. We suggest that future research will focus on clarifying what factors are associated with specific arousal profiles in children with a diagnosis of ADHD and autism, and if the same

associations can be found in children with a double diagnosis.

Our study has several limitations. First, as we have already reported, the findings we present emerged from a laboratory testing session and might not fully generalize to naturalistic environments, such as home or school. Secondly, children under treatment with non-stimulant medication could not take part in our study because nonstimulant medications cannot be temporarily withdrawn, meaning our findings are not generalizable to this sub-group of children with ADHD. Thirdly, this was a single-center study conducted for the completion of a doctoral degree, and with appropriate but minimal resources; hence, although effort was put in recruiting an appropriate number of participants, and these were very carefully clinically defined, our sample size may be considered small (99 children). In relation to our specific hypotheses (reported in Table 2) we had at least 92% power to detect large effects ( $f \geq .4$ ), at least 51% power to detect medium effects ( $f = .25-.39$ ), and at least 13% power to detect small effects ( $f = .1-.25$ ). We can therefore conclude that statistically non-significant effects are likely to be in the small-to-medium range and we were underpowered to detect these. Further research is needed to determine whether our findings can be replicated in larger samples and with greater power.

In conclusion, we found evidence for the presence of the ‘gap effect’, the ‘face effect’ and the ‘modality effect’ (and interactions between these) during a gap-overlap task, in a sample of children and adolescents. Although we did not find any evidence of alterations in basic oculomotor mechanisms in autistic children and with ADHD, and although they paid attention to the screen and completed the gap-overlap task, as neurotypicals did, we found that visual attention in children with ADHD and/or autism did not respond to the modality of presentation of stimuli in the same way as neurotypicals. We speculate that this could have been caused by overall reduced engagement with the task, particularly in children with ADHD, who may have not benefitted from the additional sensory information in the same way as neurotypicals. Lastly, we found that slower oculomotor performance characterized both hypo- and hyper-arousal transient states in children with ADHD, while in autistic participants slower SRTs were predicted by smaller pupil size only. These findings highlight the importance of further research considering the role of autonomic arousal in visual attention differences in ADHD and autism.

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