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ORIGINAL PAPER

Altered Relationship Between Microsaccades and Inattention in ADHD and the Effects of Stimulant Medication

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Abstract

Objectives Research into the role of the oculomotor system in attention deficit hyperactivity disorder (ADHD) has grown recently, but studies examining microsaccades are limited and contradictory, with tasks often confounded by high cognitive load. The current study aimed to explore the relationship between microsaccades and ADHD traits in adults with a diagnosis of ADHD, both those receiving medication (ADHD-M) and those who were unmedicated (ADHD-U), and a healthy control (HC) group.

Methods All participants (ADHD-M, $N=18$; ADHD-U, $N=22$; HC, $N=31$) completed the Adult ADHD Self Report Scale and a simple sustained attention task whilst binocular microsaccades were measured in a cross-sectional study.

Results A significant positive correlation was found between total ASRS score and microsaccade rate for the HC group, driven by a correlation between inattentive traits rather than hyperactive/impulsive traits. The same relationship between inattention and microsaccade rate was found for the ADHD-M group but not the ADHD-U group, who showed an opposite relationship.

Conclusions This research aligns with previous work finding that it is inattentive symptoms which are related to microsaccade features and demonstrates the need to consider medicated and unmedicated groups separately, which may have contributed to mixed findings previously. The findings here suggest that medicated individuals with ADHD show comparable microsaccade-inattention trait relationships to healthy controls which warrants further investigation to establish if medication normalises this relationship. Similarly, the distinct pattern of results for unmedicated individuals should be explored further in the context of conceptualisation of ADHD traits as a continuum.

Keywords ADHD · Oculomotor · Microsaccades · Inattention

Attention deficit hyperactivity disorder (ADHD) is the most common neurodevelopmental condition and is characterised by heightened inattention, impulsivity and hyperactivity (American Psychiatric Association, 2022). Although initially conceived as a childhood condition only, it is now accepted to impact individuals across the lifespan with around 3% of adults worldwide experiencing ADHD (Song et al., 2021). The impact of ADHD is considerable; those with the condition typically have poorer educational outcomes and employment status, both linked to 75% lower lifetime earnings than those without ADHD (Christiansen et al., 2021; Gordon & Fabiano, 2019; Pelham et al., 2020). Additionally, they have a greater likelihood of developing various co-occurring mental and physical conditions when compared to individuals without ADHD (Instanes et al., 2018; van der Plas et al., 2025) and a recent study indicated lower life expectancy by up to 9 years (O'Nions et al., 2025). Given the substantial impact of ADHD on an individual, it is imperative that the condition is fully understood. One area of growing interest within ADHD research is the oculomotor system, which has been found to show deficits at several levels including the basal ganglia, cerebellum and superior colliculus (SC) within ADHD (Aylward et al., 1996; Krain & Castellanos, 2006; Overton, 2008). It has been suggested that oculomotor functioning could be a biomarker of ADHD and therefore a diagnostic tool, as well as a research method

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for examining cognition and management approaches (Andreou & Argatzopoulou, 2025; Shiva Janmohammadi et al., 2020a, b).

Perhaps unsurprisingly, given the alterations in brain structures within the oculomotor system, research has shown alterations to the generation of saccades, with studies largely assessing pro-saccades, anti-saccades, visually-guided saccades (VGS) and memory-guided saccades (MGS) (Gooding & Basso, 2008; Sherigar et al., 2023). Results have consistently revealed deficits in anti-saccades, MGS and VGS which appear to improve with stimulant medication in those with ADHD (Castellanos et al., 2000; Feifel et al., 2004; Gould et al., 2001; Mostofsky et al., 2001; Munoz et al., 2003; O'Driscoll et al., 2005; Ross et al., 2000). Evidence for deficits in pro-saccades is mixed (Hanisch et al., 2006; Klein et al., 2003), but where they are found, methylphenidate appears to reduce the deficits (Bucci et al., 2017; Klein et al., 2002). Collectively, the results suggest that performance on these oculomotor paradigms reveals deficits in response inhibition in ADHD (Mahone et al., 2009), which is perhaps expected given the core symptoms of ADHD. These same saccadic measures have been proposed to offer a diagnostic or screening tool for ADHD (Yoo et al., 2024), and general visual-motor skill training (S. Janmohammadi et al., 2020a, b) and visual search training (Caldani et al., 2020) have been found to improve attention in a small sample of children with the condition (S. Janmohammadi et al., 2020a, b).

However, one type of saccade that has received comparatively less attention is the microsaccade. These eye movements are fixational movements which means that they occur when we attempt to fixate on a stimulus. They are very small, jerky movements, which can be triggered on demand, without special training and can be visual- or memory-guided, and are controlled by the SC (Willeke et al., 2019). They are considered an important window into cognitive functions (Hung et al., 2023) and emerging evidence suggests that they are relevant to ADHD, although the issue is far from clear. Studies using adults without a clinical diagnosis of ADHD have shown a significant positive relationship between microsaccade rate and scores on the Adult ADHD Self-Report Scale (ASRS), with those with higher scores having a higher rate of microsaccade production (Panagiotidi et al., 2017). However, other findings have been contradictory. Although two other studies both report a relationship between microsaccade rate and the inattention/memory subscale of the Conners' Adult ADHD Rating Scale (CAARS) (Motomura et al., 2023; Poynter et al., 2013), that relationship is in the negative direction, i.e. those with higher scores had lower microsaccade rates. Others found no relationship between microsaccade rate and ASRS scores (Perquin & Bompas, 2019).

Evidence continues to be contradictory in those studies where participants with a clinical diagnosis have been studied. Research conducted with a small group of adults with a diagnosis of ADHD, along with an age-matched control group, found that those with ADHD who were currently unmedicated had a significantly higher microsaccade rate than controls (Fried et al., 2014). In contrast, more recent work examining microsaccades in adults with ADHD and controls has found that unmedicated participants with ADHD have fewer microsaccades (Dankner et al., 2017) or when part of a mixed group with medicated participants they do not differ from controls (Roberts et al., 2018).

The main problem with research to date, with just one exception (Panagiotidi et al., 2017), is that microsaccades have been measured in the context of tasks with a range of levels of cognitive difficulty, from viewing faces (Motomura et al., 2023) to undertaking TOVA testing (Fried et al., 2014). Evidence suggests that microsaccades are sensitive to cognitive load (Dalmaso et al., 2017; Krejtz et al., 2020). Furthermore, stimulant substances (present in some studies) can decrease microsaccade rate (Hampsey et al., 2019). Consequently, in the current study, we chose to measure microsaccades in participants with a clinical diagnosis of ADHD (and controls) using a simple, cognitively undemanding paradigm and to explore the impact of medication on the production of microsaccades in the absence of cognitive load.

Methods

Participants

Ethical approval was granted by institutional ethics committees prior to data collection (Ref: LRM-23/24-27210; Ref 012498). All participants provided written consent in accordance with the Declaration of Helsinki.

Healthy Control Group (Non-ADHD) Participants were recruited from the University of Sheffield participant panel and received course credit. Inclusion criteria were (i) at least 18 years of age, (ii) currently residing in the UK and (iii) had normal or corrected-to-normal vision. Exclusion criteria for this group were (i) a current or previous diagnosis of ADHD and (ii) any major mental health or neurological condition.

ADHD Group Participants were recruited via the university research recruitment listing at King's College London, the 'Call for Participants' website, advertisements on the 'UK Adult ADHD Network' (UKAAN), and the laboratory social media. Those participating received a £10 voucher. Inclusion criteria were (i) be aged 18–35 years, (ii) currently residing in the UK, (iii) have normal or corrected-to-normal vision, (iv) declare a current diagnosis of ADHD from a healthcare

professional, either alone or with comorbid autism spectrum disorder (ASD), depression or anxiety, (v) score ≥ 14 on the ASRS Screener items, aligning with the clinical cut-off (Kessler et al., 2007). Additionally, for those receiving medication, they had to (i) have been on the same medication and dose for at least 4 weeks at the time of testing and (ii) have adherence between 70 and 100% to ensure that medication effects were reasonably consistent across participants. Exclusion criteria for this group included a diagnosis of any other neurological or psychiatric condition or learning difference. Note that the different age criteria for the two groups stemmed from this data being collected as part of a wider study where we wished to avoid age-related cognitive and oculomotor decline (Irving et al., 2006; Salthouse, 2009). We deliberately included these common co-occurring conditions found in ADHD to ensure an ecologically valid sample. This choice is supported by evidence that these conditions frequently co-occur with ADHD and are clinically relevant to symptom expression and functioning (Gnanavel et al., 2019).

Eligibility for this group was assessed using an online survey in which participants provided basic demographic details along with details of diagnoses and medication. They also completed the full 18-item ASRS (Kessler et al., 2007) and a two-item measure of adherence where they reported medication use (Safren et al., 2007).

Procedures

Eligible participants attended a laboratory visit. For those in the healthy control group, this is the point at which they completed the ASRS. For those in the ADHD group, completing the ASRS was part of the screening, so it was completed in advance of the visit. All other procedures were the same during the visit.

Eye tracking was performed using an EyeLink 1000 Plus (SR Research Osgood, ON, Canada) which recorded binocular eye movement at a sample rate of 500 Hz in pupil-only mode. The eye tracker was mounted above a head and chin rest located 50.1 cm in front of the screen on which task stimuli were displayed. The fixation task was created using the EyeLink-associated experiment builder software (SR Research Osgood, ON, Canada). Participants were seated on a padded chair in front of a 24-inch Dell OptiPlex Plus screen with a 60 Hz refresh rate (width 520 cm × height 300 cm, 1920 × 1080-pixel resolution) in a room with the lights turned off and window blinds shut to maintain a constant level of illumination. Participants were verbally asked to place their chin on the padded chin rest, firmly plant their forehead on the head rest and maintain a stable posture and head positioning for the duration of the experiment. A keyboard was made easily accessible in front of the participant.

A 9-point grid was used for calibration. During this stage, nine fixation points appeared in a random sequence, and participants were instructed to make saccades towards and fixate on each point until it disappeared, and a new one was presented. Calibration was passed if disparities were less than 1°. Once calibration was completed, a second validation step followed in which the same procedure was followed. Passing validation also required disparities to be less than 1°. If calibration or validation failed, these steps were repeated up to three times before participation would be discontinued. This step took approximately 3 min to complete. Calibration/validation was performed twice for each participant, at the beginning of each block of the task.

The task itself was a simple sustained attention task and is described in detail elsewhere (Panagiotidi et al., 2017), but briefly, participants were required to fixate on a black cross appearing on a white background for 20 s. The cross was displayed in the centre of the screen with an 86 Hz refresh rate. Two blocks, each consisting of 10 trials, were presented to each participant. There was a break between each trial with the experiment self-paced; the participant was asked to press the spacebar to begin each trial. Each trial was preceded by the instructions screen. The whole process took less than 1 h to complete.

Data Pre-processing

EyeLink EDF data were exported to sample-wise time series containing left/right horizontal and vertical gaze positions (pixels) per sample, with missing data in either eye being flagged as blink periods. All durations used in rate denominators excluded blink-masked samples, due to blinks being indistinguishable from noise in recordings. Trial ideal length was 20 s (10,000 samples at 500 Hz).

Trials with < 6500 valid samples were excluded (i.e. less than 65% of the possible samples). Participants contributing < 15 valid trials (i.e. 75% of trials) after trial-level screening were excluded. Additional removals were made for excessive noise, monocular-only recordings, incomplete sessions, or file corruption prior to analysis. This resulted in data being excluded from 7 healthy control participants ($N = 1$ missing data; $N = 6 < 15$ valid trials) and 13 participants in the ADHD group (medicated $N = 6$, unmedicated $N = 7$) for missing data ($N = 3$ computer failure, $N = 1$ monocular data collected only), completing an incorrect number of trials ($N = 3$) and finally, due to < 15 valid trials ($N = 6$).

Microsaccades were detected using the commonly adopted algorithm (Engbert & Kliegl, 2003) which has also been used previously with this specific task (Panagiotidi et al., 2017). This approach allows detection of binocular and monocular microsaccades but in line with previous

research only binocular microsaccades were considered (Hauperich et al., 2020). All the trials from all sessions were collapsed and included in the analysis. Firstly, the position data was transformed to velocities using a moving average of three data samples (6 ms) for each eye. Secondly, the velocity threshold was computed and multiplied by the relative velocity threshold (6.0). If the average velocity exceeded the velocity threshold in at least three consequent samples, the movement was defined as a monocular microsaccade. Amplitude and direction of binocular microsaccades were determined using the average horizontal and vertical displacement across the two eyes (Engbert, 2006; Engbert & Kliegl, 2003; Otero-Millan et al., 2008). Samples where no tracking data were detected were characterised as blinks and excluded from analysis. To reduce the likelihood of blink-related artifacts being detected as microsaccades and to standardise data processing between the two groups, samples 20 ms before and after blinks were also removed (Thaler et al., 2013). We used $\lambda = 6$ in all computations reported here. A minimal duration of three data samples (6 ms) was assumed to further reduce noise.

To suppress spurious macro-saccades and transient artefacts whilst aligning with the majority of studies, detected events were retained only if amplitude $\leq 1°$ (Hauperich et al., 2020). Classical fixed-fixation studies defined microsaccades as extremely small, typically ≤ 12 arcmin (0.2–0.5°) (Ditchburn & Foley-Fisher, 1967; Ditchburn & Ginsborg, 1952; St Cyr & Fender, 1969). Over time, definitions loosened, with thresholds up to 1–1.5° and even 2° used in some literature (Møller et al., 2002; Rucci & Poletti, 2015; Willett & Mayo, 2023). However, researchers have cautioned that movements exceeding 1° substantially shift the retinal locus of stimulation, moving the image from the foveola (the central 1° rod-free zone that mediates highest acuity) to the parafovea (Poletti & Rucci, 2016). Even though a 1° movement represents only a tiny fraction of the visual field (~ 200° horizontally), it completely changes which retinal cells are engaged, from the densely packed cones at the very centre to less specialised areas nearby. In functional terms, these larger displacements resemble small voluntary saccades more than stabilising fixational microsaccades. This physiological distinction, combined with the need for methodological clarity across studies, motivated our conservative 1° amplitude cap as a compromise between the strict classical definition and broader modern usage.

In addition to a maximum amplitude of 1°, we set a peak velocity $\leq 120°/s$ for microsaccades with events exceeding this discarded before summary statistics were computed. This threshold helps eliminate fast, larger saccadic intrusions. It reflects the upper limit commonly seen in microsaccades which typically fall well below this range whilst filtering out rare, high-velocity outliers that are likely not functional microsaccades. Literature consistently reports microsaccade velocities within this bound (Gu et al., 2024; Rucci & Poletti, 2015), and previous studies have also used our definition of microsaccades in their research (Engbert & Kliegl, 2003; Martinez-Conde et al., 2009). Importantly, regardless of threshold choice, most detected microsaccades naturally cluster at $< 0.5°$ amplitude, making our strict cut-offs conservative yet minimally impactful on core results.

Data Analyses

Statistical analysis to examine the relationship between microsaccade characteristics and ADHD traits in those with and without ADHD, with the latter group split into those currently receiving, and adhering to medication, and those unmedicated, was conducted in SPSS v.29 (IBM Statistics). The sample was initially characterised using descriptive statistics and group differences examined using one-way ANOVA for continuous variables and chi-square or Fisher's exact for categorical variables. The characteristics of the detected movements were manually checked and confirmed by plotting peak microsaccade velocity against amplitude, as well as by plotting amplitude distributions (Hafed et al., 2009; Zuber et al., 1964) for each of the three groups. Correlation analyses were then conducted to assess the relationship between ADHD traits and microsaccade rate, amplitude, duration and peak velocity. These correlations were calculated for the total ASRS score and the two subscales of inattention (IA) and hyperactivity/impulsivity (HI). Finally, group differences in microsaccades were assessed using an ANCOVA with age as a covariate due to significant group differences. Post hoc *t*-tests using Bonferroni–Holm correction (Gaetano, 2013) were applied where significant differences were found. This method of correction provides a better balance between controlling family-wise error rate (FWER) and maintaining statistical power. It is less conservative than the Bonferroni method, meaning it is more likely to detect true effects whilst still controlling for the risk of false positives (Giacalone et al., 2018). This correction method was also used in the sample characterisation where significant differences were found.

Results

Sample Characterisation

The final sample consisted of 31 healthy controls (HC: 3 males, 28 females) and 40 individuals with ADHD who could be further divided into 18 receiving medication (ADHD-M: 2 males, 16 females) and 22 not on medication (ADHD-U: 1 male, 21 females, 1 non-binary). Given the small number identifying as non-binary and the low expected cell counts, Fisher's exact test was used in place of

chi-square and revealed no significant association between group and gender (χ^2 (2) = 0.723, p = 0.755). Most participants in each group were right-handed with only 8 identifying as left-handed across all groups (HC = 3, ADHD-M = 2, ADHD-U = 2). There was no significant association between handedness and group (χ^2 (2) = 0.279, p = 1.000) according to Fisher's exact test. Age did differ significantly between the three groups (F (2, 71) = 16.672, p < 0.001) with post hoc tests using a Bonferroni–Holm correction showing the two ADHD groups significantly older than the control group (p < 0.003, 95% CI [−8.79, −2.19], ADHD-U p < 0.003, 95% CI [−9.78, −3.65]) (Table 1). As expected, there were also significant differences between groups for the ASRS measures: inattention (F (2, 71) = 41.458, p <0.001), hyperactivity/impulsivity (F (2, 71) = 28.933, p < 0.001) and the total score (F (2, 71) = 47.610, p < 0.001). In all cases, significant differences were between the two ADHD groups and the healthy control group (IA: ADHD-M p < 0.003, 95% CI [−12.31, −6.20], ADHD-U p < 0.003, 95% CI [−11.82, −6.15]; HI: ADHD-M p < 0.003, 95% CI [−16.00, −7.41], ADHD-U p < 0.003, 95% CI [−13.69, −5.72]; total IA: ADHD-M p < 0.003, 95% CI [−27.15, −14.77], ADHD-U p < 0.003, 95% CI [−24.44, 12.94]) only (Table 1).

Table 1 Characteristics of the three groups for continuous variables. In all cases, the significant differences were between the two ADHD groups and the healthy control group

	Healthy control ($M \pm$ SD)	ADHD-medicated ($M \pm$ SD)	ADHD-unmedicated ($M \pm$ SD)	Sig
Age	20.68 ± 3.24	26.17 ± 4.50	27.39 ± 5.90	< 0.001
ASRS				
Inattention (IA)	20.58 ± 4.74	29.83 ± 3.82	29.57 ± 3.67	< 0.001
	14.90 ± 6.52	26.61 ± 5.04	24.61 ± 5.63	< 0.001
Hyperactivity/impulsivity (HI)	35.48 ± 9.81	56.44 ± 7.13	54.17 ± 7.52	< 0.001
Total				

Of those with ADHD, 24 (58.5%) reported only a diagnosis of ADHD, whilst 7 (17.1%) reported additionally having ASD, 13 (31.7%) reported also having depression and 9 (22.0%) identified that they had anxiety. Note that the total percentage for ADHD alone and co-occurring conditions exceeds 100% (129.3%) because participants could report multiple co-occurring conditions. There was no significant association between the presence of a co-occurring condition and whether individuals were medicated or not (χ^2 (1) = 0.540, p = 0.462). There was also no significant association between the specific co-occurring condition and medication status (depression χ^2 (1) = 1.038, p = 0.308; anxiety χ^2 (1) = 0.892, p = 0.345; ASD χ^2 (1) = 0.135, p = 0.713). All those receiving medication reported taking stimulants and provided adherence information indicating high levels of adherence (M = 93.5%, SD = 7.6%).

Microsaccade Features

Microsaccades have often been shown to follow the main sequences (Hafed et al., 2009; Zuber et al., 1964), that is, an approximately linear relationship between the amplitude of the eye movement and the peak velocity. This is shown in Fig. 1 for each of the three groups.

Relationship Between Microsaccades and ADHD Traits

For the healthy control group, the total ASRS score significantly positively correlated with microsaccade rate

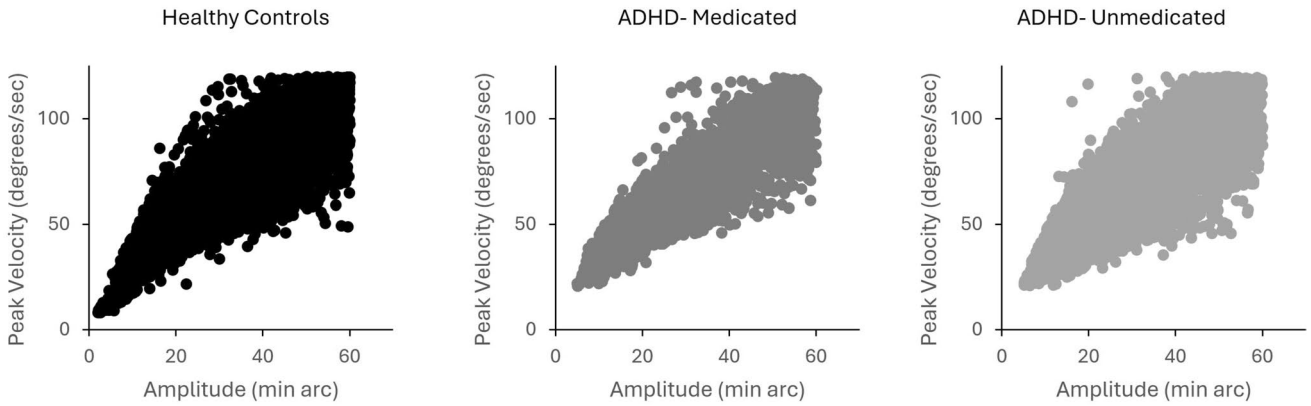

Fig. 1 The peak microsaccade velocities were plotted against their amplitudes for healthy controls (left column) (N = 9174), individuals with ADHD currently taking medication (middle column) (N = 3881), and those with ADHD not currently taking medication (right column) (N = 7784)

($r = 0.447$, $p = 0.012$, 95% CI [0.110, 0.692]) so that as ADHD symptom scores increased so did the rate of microsaccades. However, the subscales showed only a significant correlation between inattention (IA) scores and rate ($r = 0.508$, $p = 0.004$, 95% CI [0.187, 0.731]) and not the hyperactive impulsive scores (HI: $r = 0.304$, $p = 0.097$, 95% CI [−0.057, 0.594]). No other microsaccade feature (i.e. amplitude, peak velocity of duration) significantly correlated with any ASRS measure ($p \geq 0.114$). For those with ADHD taking medication (ADHD-M), there was a trend towards significance when looking at the relationships between the total ASRS score and microsaccade rate ($r = 0.465$, $p = 0.052$, 95% CI [−0.002, 0.766]), and again this was significant for IA scores ($r = 0.563$, $p = 0.015$, 95% CI [0.130, 0.815]) but not HI scores ($r = 0.231$, $p = 0.355$, 95% CI [−0.264, 0.630]). No other microsaccade feature significantly correlated with any ASRS measure ($p \geq 0.424$). Finally, for those with ADHD not currently on medication (ADHD-U), there was no significant correlation between total ASRS score and microsaccade rate ($r = -0.070$, $p = 0.757$, 95% CI [−0.477, 0.362]) but there was a significant correlation for IA traits albeit in the opposite direction to that seen with the other groups ($r = -0.441$, $p = 0.040$, 95% CI [−0.728, −0.024]), so as ADHD symptom scores increased so did the rate of microsaccades. There was no significant correlation for HI traits and rate ($r = 0.199$, $p = 0.374$, 95% CI [−0.243, 0.573]). For this group, the IA score also significantly positively correlated with peak velocity ($r = 0.453$, $p = 0.034$, 95% CI [0.039, 0.735]). There were no other significant correlations between microsaccade feature and ASRS score ($p \geq 0.166$). Figure 2 shows the correlations between rate and ASRS scores.

Group Differences in Microsaccades

ANCOVA with age as a covariate revealed no significant main effect of group on microsaccade rate (F (2, 67) = 0.617, $p = 0.542$, $\eta p^2 = 0.018$). There was a significant

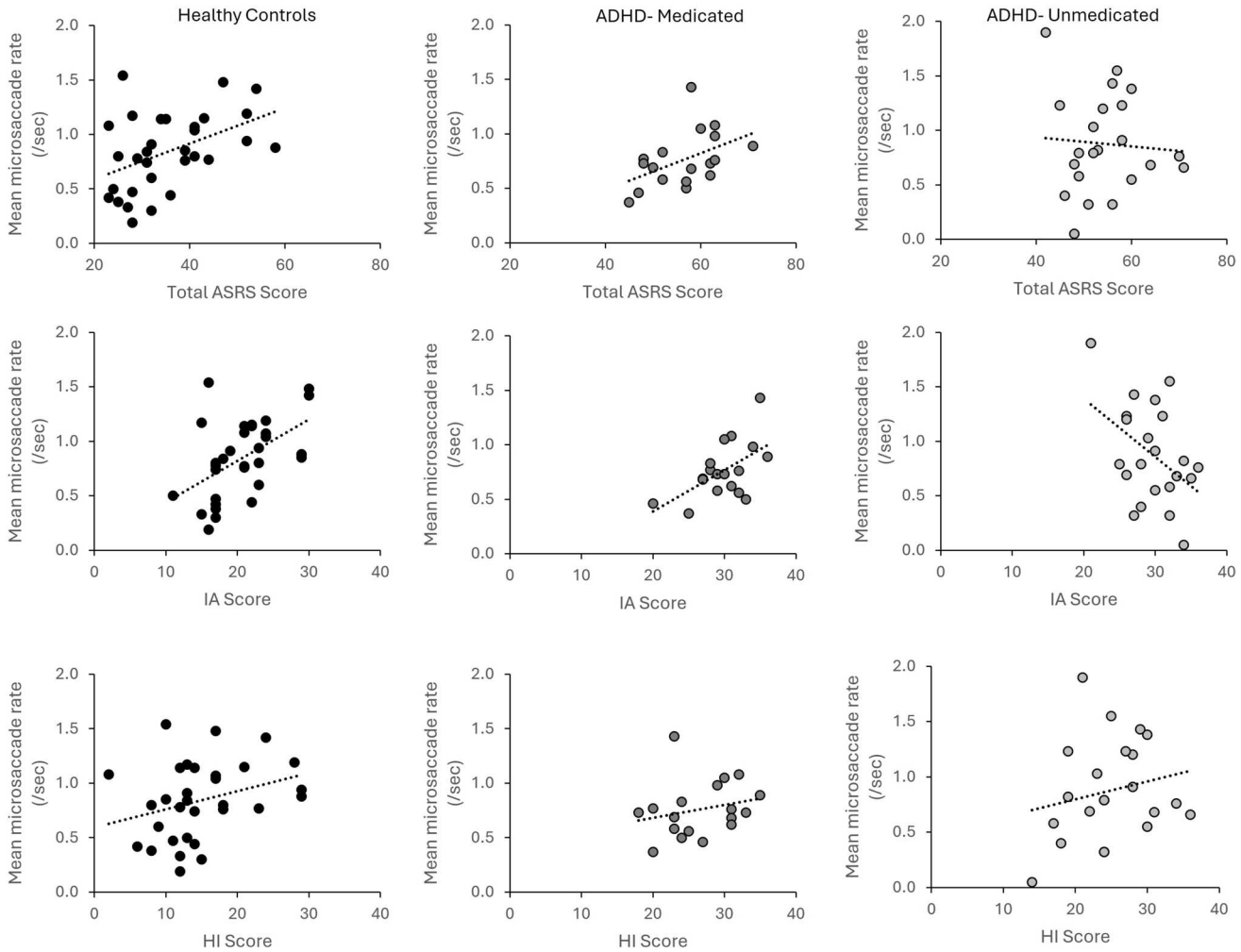

Fig. 2 The relationships between microsaccade rate and total ASRS scores (top row), IA scores (middle row) and HI scores (bottom row)

difference between groups for microsaccade amplitude (F (2, 67) = 5.780, p = 0.005, ηp^2 = 0.147). Post hoc t-tests using a Bonferroni–Holm correction revealed only one significant result indicating that healthy controls (0.55 ± 0.09°) had a significantly higher amplitude than the ADHD-M (0.45 ± 0.10°) (t (47) = 3.287, p = 0.003, 95% CI [0.042, 0.156]). There was also a significant difference between groups for microsaccade duration (F (2, 67) = 5.331, p = 0.007, ηp^2 = 0.137). Post hoc t-tests revealed significant differences between healthy controls (12.89 ± 1.48 ms) and ADHD-M (11.65 ± 1.46 ms) (t (47) = 2.835, p = 0.021, 95% CI [0.359, 0.2.111]) only, with healthy control participants having longer duration microsaccades. Finally, there were significant group differences in peak velocity (F (2, 67) = 3.514, p = 0.035, ηp^2 = 0.095); however, the post hoc test revealed that the difference between the healthy control group (69.93 ± 11.34°/s) and the ADHD-M group (61.49 ± 11.68°/s) (t (47) = 2.482, p = 0.051, 95% CI [1.560, 15.265]) and the two ADHD groups (ADHD-U = 71.10 ± 13.83°/s) (t (38) = −2.341, p = 0.051, 95% CI [−17.913, −1.297]) did not withstand the Bonferroni–Holm correction.

Discussion

The aim of the present study was to examine the relationship between microsaccade characteristics and ADHD traits in those with and without ADHD, with the latter group split into those currently receiving and adhering to medication and those unmedicated, during a simple task in the absence of cognitive load. We found that in those without ADHD there was a significant positive correlation between overall ADHD traits and microsaccade rate but that this was driven by a significant correlation with inattention traits rather than the hyperactivity impulsive traits which did not significantly correlate with rate. The positive relationship between overall ADHD traits and rate showed a trend towards significance in those with ADHD who were receiving medication. The strong positive correlation between inattention traits and microsaccade rate remained, whilst hyperactive impulsive traits showed no significant correlation. By way of contrast, there was no significant correlation between overall ADHD traits and microsaccade rate in those with ADHD not receiving medication. Like the other groups, there was no significant correlation between the hyperactive impulsive traits and the microsaccade rates but there was a significant negative correlation between inattentive traits and rate, indicating that for this group higher inattentive traits meant lower rate of microsaccades. The same group also had a significant positive correlation between inattentive traits and microsaccade duration, indicating they had longer duration saccades as inattentive symptoms increase, which aligns with reduced rate. ANCOVA also found group differences in microsaccades between the three groups for amplitude and duration. For amplitude, healthy controls had significantly higher amplitude microsaccades compared to those with ADHD receiving medication. Similarly, healthy controls had significantly longer duration microsaccades compared to those receiving medication with ADHD. Differences in peak velocity did not withstand correction for multiple comparisons.

As indicated above, previous research investigating the relationship between microsaccades and ADHD traits has reported mixed results. Work in those without a diagnosis has revealed both significant positive association between ADHD traits and microsaccades (Panagiotidi et al., 2017) and significant negative associations (Motomura et al., 2023; Poynter et al., 2013) or even no association (Perquin & Bompas, 2019). The current study removed the possibility of cognitive load demands with a simple task and found a positive association between rate and ADHD traits and, in particular, inattention. The finding that inattention was critical rather than hyperactivity/impulsivity aligns with previous work where relationships have also identified attentional symptoms as critical (Motomura et al., 2023; Poynter et al., 2013). None of these prior studies that have had participants without an ADHD diagnosis conducted a full diagnostic interview to determine whether participants could have undiagnosed ADHD, and our results suggest that this may contribute to the different findings. In particular, if the participant cohort contained several cases of undiagnosed (and therefore unmedicated) ADHD, equivalent to our ADHD-U group, this would potentially lead to a negative association between ADHD traits and microsaccades (as in Motomura et al., 2023; Poynter et al., 2013).

Our results fit well with studies where participants have a clinical diagnosis of ADHD. For example, it has been shown that unmedicated participants with ADHD have fewer microsaccades (Dankner et al., 2017). This partially aligns with our finding that unmedicated individuals with ADHD have longer duration microsaccades with worsening symptoms and that higher levels of inattention are associated with fewer microsaccades. Similarly, when medicated and unmedicated participants with ADHD are combined into a single group, they did not differ from controls in terms of their microsaccade rate (Roberts et al., 2018). This would be expected from the current results given the opposing direction of effects in medicated and unmedicated individuals, effectively cancelling out any overall differences. The negative relationship between microsaccade rate and inattention symptoms in unmedicated participants with a diagnosis of ADHD contrasting with the positive relationship in healthy controls warrants further investigation. Whilst current perspectives on ADHD assume that it is a continuum disorder which grades from the general population to the clinic

(Hudziak et al., 2007; Martin et al., 2014), these findings may indicate a qualitative difference in ADHD in the relationship between microsaccades and traits which could challenge this view. As such more research is needed to explore these relationships in larger samples, over time.

The group comparisons only showed differences between those on medication and healthy controls for amplitude and peak velocity; the latter of which did not withstand correction for multiple comparisons. This contrasts to previous work which revealed a higher rate of microsaccades in unmedicated individuals with ADHD but not medicated individuals (Fried et al., 2014), although the difference may result from task differences between the two studies in terms of cognitive load.

A key finding in the current study is that the presence of medication appeared to result in a relationship between rate and ADHD traits similar to that found in healthy controls when compared to the unmedicated group. We tentatively speculate that this could occur because the medication, in this case stimulants, restored the typical relationship between attention and microsaccade rate. The current cross-sectional design cannot confirm this possibility, and further research should explore this in within-subjects longitudinal designs with both on and off medication conditions. Although this study cannot determine normalisation, the possibility of this aligns with previous research that found that microsaccade rate was normalised in the presence of medication (Fried et al., 2014). Exactly what mechanism could underlie this is unclear. Previous research has suggested that medication increases inhibitory oculomotor control by elevating arousal or the general tonic level of the attention system (Fried et al., 2014). A possible neural correlate of this activity is the SC which has been implicated in ADHD with theory suggesting that heightened activity in the SC during ADHD may result in the over-production of microsaccades (Overton, 2008). Whilst the current study does indicate that for healthy controls and those on medication rate increased with trait score this was not also reported for unmedicated individuals suggesting that it may not simply be an increase in microsaccade rate with ADHD. An alternative explanation is that ADHD disrupts the normal relationship between attentional abilities and microsaccades and that medication re-establishes this relationship. This would align with previous work indicating that stimulant medications alter collicular activity in a manner that is thought to stabilize activity in the region and adjust the signal-to-noise ratio (Dommett et al., 2009; Turner et al., 2018).

Here we reported statistically significant correlations between microsaccade rate and ADHD traits in those with and without ADHD and differentiated between those with the condition on medication and not on medication. The results of the current study suggest that it is critical to consider medicated and unmedicated participants separately as different, contradictory relationship may be found in the two groups. Whilst this study is one of the largest studies to date investigating microsaccades and includes an ecologically valid sample of adults with ADHD and common co-occurring conditions as well as a healthy control group, there are limitations. Firstly, we did not conduct full clinical interviews to verify reported diagnosis or lack thereof. Instead, we asked participants to declare if they had received an ADHD diagnosis from a healthcare professional and used the ASRS to confirm the category (diagnoses/undiagnosed) they identified. It is possible, therefore, that this information was misreported by participants or that undiagnosed ADHD was found within the control group. Given comparison to prior studies with both clinical and non-clinical groups revealed mixed results, we cannot rule out this was due to inaccurate classification. Further studies should include full clinical interview screening. Secondly, whilst the inclusion of those with co-occurring conditions allows for ecological validity, it also means the effects of these conditions cannot be extracted. Whilst the presence of these conditions was associated with medication, severity of them was not assessed. Additionally, the medicated group all reported taking stimulant medication and so we cannot be sure if our results would generalise to non-stimulant medication. Furthermore, the exact dose and type of medication were not recorded and, although medicated participants were instructed to take medication as normal on the day of testing, we did not record when this was in relation to the testing session. Future research should examine all types of medication and consider factors such as specific formulation and peak effectiveness with regard to testing time, as well as control for co-occurring conditions which was not possible in the current study due to sample size. Fourthly, although the correlations, supported by confidence intervals, indicate different relationships between microsaccade rate and inattentive traits in the groups, a larger sample would be needed to statistically compare the slopes. Finally, it should be noted that our sample was predominantly female and there was a small but significant age difference between groups. The presence of a female-dominated sample, whilst addressing a gap in the literature where females are under-represented in ADHD research (Platania et al., 2025), does mean that findings may not generalise to males. Furthermore, although limited, there is evidence for oculomotor functioning, albeit not specifically microsaccades, varying by gender and age (Lee et al., 2022; Popowczak & Zwierko, 2025) meaning future research with more closely matched samples, ideally large enough to incorporate gender comparisons, is required.

Author Contribution Conceptualisation and methodology (all authors); Investigation (APM, MP); formal analysis (APM, MP, EJD); supervision (PGO, EJD), writing – original draft preparation (APM, EJD), writing – reviewing and editing (APM, PGO and EJD).

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Data Availability Data will be made available on reasonable request from the corresponding author.

Declarations

Ethics Approval The study was approved by the Research Ethics Committee of King's College London (LRM-23/24-27210) and Sheffield University (012498).

Consent to Participate All participants provided informed consent to participate in the research and for the results of the study to be published.

Conflict of Interest The authors declare no competing interests.

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