Postural Control Deficits in Autism Spectrum Disorder: The Role of Sensory Integration

Doumas, M., McKenna, R., & Murphy, B. (2016). Postural Control Deficits in Autism Spectrum Disorder: The Role of Sensory Integration. *Journal of Autism and Developmental Disorders*, *46*(3), 853-861. https://doi.org/10.1007/s10803-015-2621-4

Published in:
Journal of Autism and Developmental Disorders

Document Version:
Peer reviewed version

Queen's University Belfast - Research Portal:
Link to publication record in Queen's University Belfast Research Portal

Postural control deficits in Autism Spectrum Disorder: The role of sensory integration

Michail Doumas, Roisin McKenna and Blain Murphy

School of Psychology, Queen's University Belfast, Belfast, UK

Address correspondence to Michail Doumas, School of Psychology, Queen's University Belfast, 18-30 Malone Road, Belfast, BT9 5BN, UK.

Email: m.doumas@qub.ac.uk, Tel: +44 (0)28 9097 4605, Fax: +44 (0)28 9097 5486.

Abstract

We investigated the nature of sensory integration deficits in postural control of young adults with ASD. Postural control was assessed in a fixed environment, and in three environments in which sensory information about body sway from visual, proprioceptive or both channels was inaccurate. Furthermore, two levels of inaccurate information were used within each channel (gain 1 and 1.6). ASD participants showed greater postural sway when information from both channels was inaccurate. In addition, control participants' ellipse area at gain 1.6 was identical to ASD participants' at gain 1, reflecting hyper-reactivity in ASD. Our results provide evidence for hyper-reactivity in posture-related sensory information, which reflects a general, rather than channel-specific sensory integration impairment in ASD.

Keywords: Postural control, Balance, Autism Spectrum Disorder, Sensory Integration, Proprioception, Vision.

Postural control deficits in Autism Spectrum Disorder: The role of sensory integration

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder mainly characterized by "persistent deficits in social communication and social interaction across multiple contexts" (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, DSM-5). Sensory impairments were previously not part of the core definition of the disorder, but the DSM classification now includes the expression of "hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of the environment." A key sensorimotor control process affected by ASD is the control of upright standing, or postural control (Fournier, Amano, Radonovich, Bleser, & Hass, 2014; Fournier, Kimberg, et al., 2010; Graham et al., 2015; Greffou et al., 2012; Minshew, Sung, Jones, & Furman, 2004; Molloy, Dietrich, & Bhattacharya, 2003). This task is critical for daily life and independence in both children and adults, and is useful in assessing impairments, not only in general movement control, but also in some of its more specific aspects, including the quality of sensory input from individual channels (e.g. vision and proprioception) and the mechanisms of sensory integration.

Postural control relies on sensory information from visual, vestibular and proprioceptive channels, utilised by a feedback process to produce corrective muscle responses to resist gravity (Balasubramaniam & Wing, 2002; Maurer, Mergner, & Peterka, 2006). Control in this task does not rely equally on the three channels, rather, information from each channel is weighted depending on its relative reliability following a sensory integration, or reweighting process (Peterka, 2002; Peterka & Loughlin, 2004). For example, when we move from a well lit to a dark environment, visual information becomes less reliable and is down-weighted and as a result,

information from proprioceptive and vestibular channels is up-weighted. In stable environments, the sensory channel with the highest weight during this task is proprioception. However, when we step on a compliant surface like grass or sand, proprioceptive information becomes less reliable and is also down-weighted (Peterka, 2002). In these examples, fast and accurate sensory integration is critical for quick postural adjustments and fall prevention (Horak, 2005). Thus, reliability of sensory information from the three channels and the way this information is integrated, are likely to be two key contributing factors to the postural control deficits observed in ASD. Deficits in both of these aspects of sensory processing in vision and proprioception have been previously assessed in children and adults with ASD (for review see Gowen & Hamilton, 2013).

Visual information is differently affected by ASD depending on the level in which processing takes place, low or high (Bertone, Mottron, Jelenic, & Faubert, 2005; Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005; Pellicano & Gibson, 2008). For example, Pellicano and Gibson (2008) assessed integrity of dorsal stream visual processing in ASD and showed that children with ASD exhibited intact lower-level but impaired higher-level dorsal stream functioning. In a similar vein, proprioception was assessed in adolescents with ASD and Typically Developing (TD) controls using proprioceptive matching tasks (Fuentes, Mostofsky, & Bastian, 2011). In this study, although ASD participants were impaired in general sensory and motor performance, their proprioceptive abilities were not different from typically developing adolescents'. Furthermore, Glazebrook et al. (2009) showed that in a manual pointing task without vision, when proprioception was the dominant modality, no ASD-related impairments were shown. However, when both modalities were present and sensory integration

demands increased, ASD participants took considerably more time to perform the pointing movements. Together, this evidence suggests that processing of unimodal sensory information in ASD, including low-level visual and proprioceptive processing, is relatively intact. Thus, the observed deficits in postural control in participants with ASD are likely to arise at the level of multisensory integration. This idea is in line with ASD-related multisensory integration deficits in the temporal domain, shown in tasks assessing temporal integration of auditory and visual stimuli (Stevenson, Siemann, Schneider, et al., 2014; Stevenson, Siemann, Woynaroski, et al., 2014; Wallace & Stevenson, 2014).

Postural control deficits in ASD have been identified primarily using clinical and diagnostic tests (for meta analysis see Fournier, Hass, Naik, Lodha, & Cauraugh, 2010), with very few studies specifically examining sensory integration of vision and proprioception in this disorder. In one of these studies, Greffou et al. (2012) showed impaired integration of visual information in individuals with ASD when standing in a virtual tunnel that oscillated in different frequencies. TD adolescents', but not adults', sway increased with tunnel frequency, especially in the highest frequency, however, this was not the case in adolescents with ASD. This finding was attributed to a sensory integration impairment reflected in hypo-reactivity to visual sway-inducing information. In the proprioceptive domain, Molloy et al. (2003) asked TD and ASD children to stand on a fixed surface and on foam with and without vision. They showed that children with ASD exhibited greater sway areas, with this effect increasing with task difficulty. Furthermore, Minshew et al. (2004) reported that effects of ASD emerged only when proprioceptive information was manipulated by means of support-surface sway reference. However, none of the previous studies has systematically manipulated

sensory integration demands by means of changing the reliability of both visual and proprioceptive information in a continuous manner.

The aim of this study was to assess the nature of sensory integration deficits in postural control in young adults with ASD. Sensory integration demands were manipulated by means of inducing inaccurate visual and proprioceptive information about body sway using the well-established technique of sway reference (Black, Wall, & Nashner, 1983; L. Nashner, 1984; L. M. Nashner, Black, & Wall, 1982). Modelling and experimental work in typically developing individuals suggests that postural sway is less sensitive to inaccurate visual, compared with inaccurate proprioceptive information (Clark & Riley, 2007; Peterka, 2002). Thus, we predicted little or no group differences in postural sway in a fixed environment and when visual information was inaccurate, due to their low sensory integration demands. However, when sensory integration demands increased by means of introducing inaccurate proprioceptive information and especially inaccurate visual and proprioceptive information simultaneously, we expected postural sway in the ASD group to show a steeper increase compared with controls, reflecting impaired sensory integration in ASD. Finally, we expected this increase to be larger when sensory integration demands were further increased by means of greater sway-reference gains (gain level 1 vs. 1.6). The two levels of gain were selected on the basis of a previous study assessing effects of increasing levels of sway reference gain on postural sway (Clark & Riley, 2007) and on our own pilot testing. This gain manipulation, which was equivalent in vision and proprioception, allowed for a direct contrast of sensory integration deficits in the two channels.

Methods

Participants

Fifteen young adults with ASD and 15 controls participated in the study. Detailed group characteristics are presented in Table 1. All participants had full-scale IQ greater than 80, measured using the Wechsler Abbreviated Scale of Intelligence (WASI, Wechsler 1999). ASD participants were recruited through Autism Initiatives Northern Ireland and from the wider community. All participants with ASD met Diagnostic and Statistical Manual for Mental Disorders-Fourth Edition (DSM-IV, American Psychiatric Association, 2013) criteria for ASD. Diagnostic proof of ASD was obtained by a General Physician, Clinical Psychiatrist, or Psychologist. The Social Responsiveness Scale (SRS, Constantino & Gruber, 2005), completed by a parent or carer, was used to obtain an ASD severity score. In SRS, an overall score of 76 or above is considered within the severe range of ASD, a score in the range of 60-75 indicates the mild to moderate range or high functioning ASD and a score of 59 or less is considered indicative of typical development and is not compliant with an ASD diagnosis. SRS provides a valid assessment of autism severity as shown by correlation coefficients greater than 0.64 between SRS and the Autism Diagnostic Interview Revised (Hilton et al., 2007).

Insert Table 1 Here

Both groups completed a medical pre-screening questionnaire to ensure no comorbid diagnoses, such as Attention Deficit Hyperactivity Disorder (ADHD), as studies have indicated their influence on postural performance (Ghanizadeh, 2011;

Sergeant, Piek, & Oosterlaan, 2006). Pre-screening also ensured no history of major neurological disorders and no intake of medication affecting postural control, including sleeping medication and tricyclic antidepressants (Mamo et al., 2002). Basic sensory processing was assessed using the adolescent/adult Sensory Profile, a 60-item self-questionnaire probing sensory behaviors through questions about everyday experiences (Brown & Dunn, 2002). The Sensory Profile is a self-report questionnaire with questions such as "I trip or bump into things" which requires responses in a five point likert scale format e.g. (1 = Never and 5 = Always). It measures and profiles effects of sensory processing on functional performance by means of assessing participants' neurological thresholds (i.e. their sensitivity to touch or smell stimuli) and their response/self-regulation patterns (i.e. whether they change the environment to meet their sensory needs or they adapt their needs to the environment). Our ASD participants showed increased sensitivity in low registration and sensation avoiding aspects of the test compared with controls (Table 1). This is in line with previous studies using various versions of this test (Baker, Lane, Angley, & Young, 2008; Baranek, David, Poe, Stone, & Watson, 2006; Kern et al., 2007; Watling, Deitz, & White, 2001). Participants provided written informed consent and the protocols were approved by the School of Psychology, Queen's University Belfast Research Ethics Committee.

Insert Figure 1 here

Apparatus and Tasks

Postural control was assessed using the SMART Balance Master System (Neurocom inc.), comprising mechanically locked dual force plates and a three-sided visual surround. The system provided ground reaction forces in the Anterior-Posterior (AP) and Medio-Lateral (ML) directions in a sampling rate of 100Hz. Participants were asked to stand on the force plates and to be as stable as possible while looking at a fixation cross positioned in front of them at eye level. Foot placement was marked on the force plates in the beginning of the session and was identical in all trials. Stance width was adjusted to each participant's height in a standardised position, as advised by the system's manufacturer (Distance between lateral borders of the heels: 26cm apart for height=154.9-165cm and 30.5cm for height=166-190.5cm). A safety harness that ensured safety in the event of loss of stability but did not limit motion was worn throughout postural assessment.

The experiment comprised four posture conditions (Figure 1): one including no surround or surface perturbations (Fixed) and three sway-reference conditions during which the visual three-sided surround (Visual), the support surface on which participants were standing (Proprioceptive), or both surround and support (Both) were tilted in the sagittal plane (Anterior-Posterior direction) using a servo-controlled motor in proportion to participants' own body sway, or sway reference (Black et al., 1983; L. Nashner, 1984; L. M. Nashner et al., 1982). Sway reference is a well-established method of inducing inaccurate proprioceptive and visual information about body sway (Peterka & Loughlin, 2004). During visual sway reference, when the participant sways forward 1°, the surround is tilted 1° forward (Figure 1b), thereby inducing inaccurate visual information about body sway. Similarly, during proprioceptive sway reference (Figure 1c), when the participant sways forward 1°, the support surface is tilted 1° forward,

thereby keeping ankle-angle constant and inducing inaccurate proprioceptive information about body sway. It is important to emphasize that during sway reference, visual and proprioceptive information per-se were still accurate, but they were not providing veridical information about body sway. Surround and support movements were implemented in direct proportion to AP body sway as in the examples above (gain=1) or in proportion greater than 1 (gain=1.6), thereby increasing the amplitude of surround and support perturbations (Clark & Riley, 2007).

During testing there were conditions in which participants with ASD exhibited a large amount of sway and high instability. This was particularly true of the condition inducing the largest amount of sway (both visual and proprioceptive sway reference, gain=1.6). Loss of stability was observed in four trials in total, performed by three ASD participants. As soon as loss of stability was observed, the trial was interrupted and repeated. Interrupted trials were excluded from analysis. During all loss-of-stability incidents, a small step response was sufficient to maintain balance.

Procedure

The experiment comprised two sessions, on different days, no more than one week apart. The first session took place in the participant's home or in the laboratory and comprised the pre-screening measures including demographic and medical information, the intelligence test (WASI) and the Sensory Profile. The second session took place in the laboratory and lasted 45 minutes. Postural assessment started with a practice block comprising two trials in each of the seven posture conditions with increasing sensory integration demands: Fixed, then Visual, Proprioceptive and Both at

gain 1 followed by the last three conditions at gain 1.6. After practice, for the main experiment participants performed two blocks of trials: one block comprising fixed (3 trials), visual gain 1 (6 trials), proprioceptive gain 1 (6 trials) and both gain 1 (6 trials), and the other block included the last three conditions with gain 1.6. Trial duration was 20s. The order of blocks was counterbalanced across participants.

Data analysis

The Anterior-Posterior and Medio-Lateral COP trajectories exported from the balance system were low pass filtered (4th order Butterworth dual-pass filter, cut off frequency: 4 Hz). Then, an ellipse was fitted to the COP trajectory on the x-y plane. The two main axes of the ellipse, reflecting AP and ML sway were determined using Principal Component Analyses. The length of the ellipse's axes was equal to 2 SD along each axis, fitting approximately 88% of the COP trajectory within the ellipse, excluding any extreme excursions of the COP trajectory (for details on this methodology see Duarte & Zatsiorsky, 2002). Postural sway measures for each trial were the size of the ellipse, and the SD of sway in the AP and ML directions calculated as the two main axes of the ellipse. Single-trial measures were then averaged for statistical purposes.

Data analysis software was developed in MATLAB (2013a; The Mathworks, MA, USA). Results for the three posture measures were analyzed first using an independent samples t-test to contrast group performance in the fixed platform condition, and then by a mixed design ANOVA with gain (1 and 1.6) and posture condition (Visual, Proprioceptive and Both) as within-, and group (control and ASD) as between-subjects

factors. Statistical analyses were performed using SPSS 22 for MAC (Armonk, NY: IBM Corp.).

Results

Ellipse area

Ellipse area results are depicted in Figure 2. In the fixed platform condition, analysis showed no group differences in ellipse area (P>.05). In conditions containing sway reference manipulations (Visual, Proprioceptive and Both), results showed that overall, ellipse area was greater in participants with ASD compared with controls [*group,* $F(1,28)=12.09$, $P<.05$, $\eta^2=0.3$]. Ellipse area was also greater in conditions with gain 1.6 compared with gain 1 [*gain,* $F(1,28)=25.62$, $P<.01$, $\eta^2=0.48$] and increased with posture condition [*posture condition*, $F(1.6,46.3)=82.46$, $P<.01$, $\eta^2=0.75$]. Furthermore, the difference between ASD participants and controls increased with posture condition [*posture condition by group* $F(1.6,46.3)=10.4$, $P<.05$, $\eta^2=0.27$] and less so with gain, because the latter interaction only approached significance [*gain by group*, $F(1,28)=4.01$, $P=.053$, $\eta^2=0.13$]. Also, ellipse area differences between gain 1 and 1.6 increased with posture condition [*gain by posture condition*, $F(1.1,31.7)=25.83$, $P<.01$, $\eta^2=0.48$]. In line with these findings, a 3-way interaction $F(1.6,46.3)=4.68$, $P<.05$, $\eta^2=0.14$ suggested that the increase in ellipse area with posture condition was steeper in ASD compared with controls, and this group difference became even greater, especially in the 'both' condition when gain increased from 1 to 1.6.

Insert Figure 2 here

To interrogate this three-way interaction, separate mixed design ANOVAs were performed for the two gain levels, 1 and 1.6. Results showed no group by posture condition interaction in gain 1 (P>.05) but this interaction was present in gain 1.6 $F(2,56)=8.95$, $P<.01$, $\eta^2=0.24$, suggesting that group interactions were driven by conditions with high sensory integration demands. To identify which posture conditions were driving this interaction, we performed post-hoc independent samples t-tests with Bonferroni correction in all posture conditions and for both gains. Results showed that group differences were significant only in the 'both' condition at a gain of 1.6 $t(28) = 3.46$, $P=.002$. In all other group comparisons, the ASD group showed greater ellipse areas than controls, however, none of these differences reached significance (all P-values = .024 - .074). Interestingly, the two groups showed identical performance (P>.05) when controls were performing at gain 1.6 and ASD participants at gain 1 -a result reflecting the ASD group's hyper-reactivity to sensory information.

AP and ML sway SD

For AP SD (Figure 3a) in the fixed platform condition, participants with ASD showed greater SD than controls ($t(28) = 2.2$, $P<.05$). In sway-reference conditions, AP SD was greater in participants with ASD [*group*, $F(1,28)=4.64$, $P<.05$, $\eta^2=.14$] and increased with gain [*gain*, $F(1,28)=29.36$, $P<.01$, $\eta^2=.51$]. AP SD also increased with posture condition [*posture condition*, $F(1.9,52.7)=105.06$, $P<.01$, $\eta^2=.79$] and this increase was greater for gain 1.6 relative to 1 [*posture condition* by gain, $F(1.6,45.3)=23.82$, $P<.01$, $\eta^2=46$]. However, unlike ellipse area comparisons, no group interactions were shown.

Insert Figure 3 here

For ML SD (Figure 3b) in the fixed platform condition, no differences were observed between ASD and control groups (P>.05). A mixed design ANOVA showed that ML SD was greater for participants with ASD compared with controls [*group*, $F(1,28)=10.32$, $P<.05$, $\eta^2=.27$] and increased with gain [*gain*, $F(1,28)= 6.31$, $P<.05$, $\eta^2=.37$] and posture condition [*posture condition*, $F(2,55.8)=16.31$, $P<.01$, $\eta^2=.37$]. Similar to ellipse area, differences in ML SD between the two gain levels increased with posture condition [*gain by posture* condition, $F(1.4,38)=19.13$, $P<.01$, $\eta^2=.41$], and more importantly, the increase in ML SD with posture condition was greater in participants with ASD compared with controls [*group by posture condition*, $F(2,55.8)=8.97$, $P<.01$, $\eta^2=.24$]. Visual inspection of Figure 3b suggests that, similar to ellipse area, this interaction is due to the large increase in ML SD when gain increases in the 'both' condition. However, this interaction was not followed by a group by gain interaction and the three way interaction in this analysis only approached significance (P=.069).

Discussion

The aim of this study was to assess the nature of sensory integration deficits in postural control of young adults with ASD. Ellipse area results showed no ASD-related deficits when visual information was inaccurate, but these deficits emerged when both visual and proprioceptive information was inaccurate. Furthermore, when gain increased from 1 to 1.6, ASD participants' ellipse area increased to a much larger extent than controls'. These results suggest that the gradual increase in sensory integration

demands, induced by both posture condition and gain manipulations, resulted in a respective increase in postural sway differences between ASD and control groups. In addition, the ASD group at gain 1 showed the same ellipse area with the control group at gain 1.6 in all posture conditions. This result suggests that the amount of correction applied by individuals with ASD following a postural perturbation is much greater compared with control participants, reflecting hyper-reactivity in the ASD group. Finally, we assessed variability in the two directions of postural sway, AP and ML. The pattern of results largely replicated ellipse area results, with AP showing large effects of ASD, gain and posture conditions, and ML showing very clear group interactions with posture condition and gain.

Our results are in agreement with previous studies assessing postural control using visual and proprioceptive sway reference manipulations in control populations (Clark & Riley, 2007; Doumas, Smolders, & Krampe, 2008; McCollum, Shupert, & Nashner, 1996; L. M. Nashner, 1976; Peterka & Black, 1990). This pattern can be explained using linear models of sensory integration for postural control (Peterka, 2002; Peterka & Loughlin, 2004). When participants stand on a fixed environment, proprioceptive and vestibular information are the key sources of information, with vision having a smaller contribution (or weight) to overall stability (Peterka, 2002). Thus, when inaccurate visual information is introduced, sensory information from the two other channels is sufficient to produce the appropriate corrective movements and little or no increase in sway is observed. However, when inaccurate proprioception is introduced, accurate vestibular and visual information may not be sufficient to produce appropriate corrections, due to the large contribution of proprioception to postural control. This perturbation results in an increase in postural sway, which is even greater

when information from both vision and proprioception is inaccurate because in this case vestibular is the only accurate source of information. In the present study, even though proprioceptive manipulations alone did not show group differences, the 'both' condition exhibited not only the largest postural sway in the control group, but also the largest ASD-related differences, confirming our hypothesis for a sensory integration deficit in ASD.

Our results suggest that participants with ASD exhibit the same general pattern of postural control as control participants. However, the main group difference lies on the sensitivity of ASD individuals' postural control system to increases in sensory integration demands. When these demands are low, in the case of visual manipulations, no group differences were shown. Similarly, Greffou et al. (2012) showed that adolescents with ASD show hypo-reactivity to visual stimuli, whereas, TD adolescents show higher reactivity to visual stimuli. However, in agreement with our findings, this difference in reactivity was not present in young adults (Greffou et al. 2012). On the other hand, when these demands were high, in the case of high gain and manipulation of both channels, our results showed that this increase affected ASD participants more than controls (Minshew et al., 2004; Molloy et al., 2003). Together, this gradual increase in group differences with sensory integration demands reflects a general, rather than sensory channel-specific impairment in ASD. This is because a channel-specific decline would have been reflected in a greater impairment in only one of the channels (e.g. vision), together with a lack of increase in instability between this channel and the condition in which both channels were inaccurate. Similar channel-specific impairments have been shown in ASD in a recent study using a motor learning task (Marko et al., 2015).

An alternative explanation for the increase in postural sway in ASD could be that this impairment is due to vestibular dysfunction. Under this idea, when sensory integration demands increase in the condition involving inaccurate visual and proprioceptive information, postural control needs to rely solely on vestibular information as the only reliable source of sensory information. Thus, vestibular impairment in ASD may also explain our findings. Even though we cannot fully rule out this possibility, studies assessing vestibular function in ASD suggest intact vestibulo-ocular reflex function in studies assessing children (Goldberg, Landa, Lasker, Cooper, & Zee, 2000) and children and adults with ASD (Furman, Osorio, & Minshew, 2015). This evidence is in line with the intact nature of sensory information in ASD, including low-level visual information (Bertone et al., 2005; Pellicano et al., 2005; Pellicano & Gibson, 2008) and proprioceptive acuity (Fuentes et al., 2011).

We also assessed sway variability (SD) in the two directions of postural sway, AP and ML. AP variability was greater in the ASD group and increased with posture condition and gain manipulations, but unlike ellipse area results, there were no group interactions. In contrast, in ML, ASD participants showed greater variability and this difference increased with posture condition. This result suggests that sway in the ML direction reflects ellipse area results more accurately than results in the AP direction, and is unexpected because sway reference manipulations mainly targeted the AP direction. One explanation for this finding can be found in the trade-off, reciprocal links shown between AP and ML sway in a precision aiming task performed during quiet standing (Balasubramaniam, Riley, & Turvey, 2000). When aiming constraints require minimization of postural sway in one direction to enhance aiming accuracy, in tasks like shooting or archery, this minimization is followed by a reciprocal increase in sway in

the other direction. Similarly, in our study, sway minimization was required in the AP direction because this was the direction of our sway reference manipulations. Following this idea, our ASD participants are likely to have actively kept AP sway at bay when sensory integration demands increased, in order to minimize the possibility of a fall. As a result, there were no group differences in AP variability. However, this sway reduction in one direction resulted in a reciprocal increase in the other direction, but only in participants with ASD. Further research is required to interpret this asymmetry in the reciprocal increase of the two directions of postural sway in ASD.

The main focus of our study was on postural control in adults, rather than children or adolescents with ASD. Even though ASD has been primarily studied as a neurodevelopmental disorder affecting children and adolescents, many of the symptoms and characteristics of ASD persist in adulthood and are likely to be exacerbated in older age (Happe & Charlton, 2012). This approach to ASD research is important, especially in postural control, because performance in this task declines during adulthood as instability increases with age and this dysfunction leads to the large incidence of fall accidents commonly observed in older adults (Rubenstein, 2006). It is possible that the ASD-related balance impairments shown in the present study also increase with age and become critical after the age of 65, leading to an even greater likelihood of fall accidents in ASD than in healthy older adults. Recent studies have identified very effective ways of reducing fall accidents in healthy older adults through targeted physical activity comprising balance-training exercises (Sherrington, Tiedemann, Fairhall, Close, & Lord, 2011; Sherrington et al., 2008). Future research could emphasize the role of physical activity and the role of exercise in ASD. Little is known about ASD individuals' ability to improve their balance, yet, a recent study

(Cheldavi et al. 2014) showed that children with ASD improved their balance over a 18-week practice program, including postural control with and without vision and on a fixed or compliant surface (foam). However, this study did not contrast ASD and control groups, thus, it is not clear whether the capacity for balance improvement is the same in the two groups.

The neural underpinnings of sensory integration deficits in ASD are not well understood, however the cerebellum has been identified as a critical structure, both for ASD and for sensorimotor control. On the ASD side, studies have shown a reduction in purkinje cell numbers (Bailey et al., 1998; Ritvo et al., 1986) and a reduction in volume of the cerebellar vermis (Hashimoto et al., 1995; Murakami, Courchesne, Press, Yeung-Courchesne, & Hesselink, 1989; Scott, Schumann, Goodlin-Jones, & Amaral, 2009) and on the sensorimotor control side, it is well established that the cerebellum is critical for postural control, sensory integration and motor learning (for a review see Therrien & Bastian, 2015). While little is known about the role of the cerebellum in ASD individuals' postural control, a recent study assessed the role of the cerebellum in ASD and TD children's ability to learn a simple reaching task using visual and proprioceptive feedback (Marko et al., 2015). Children with ASD were faster than controls in proprioceptive-based learning but slower in visual-based learning. More importantly, this study showed that parts of the anterior cerebellum extending to lobule VI and part of lobule VII involved in sensorimotor control, were smaller in volume in ASD children, even though the overall size of the brain and the cerebellum did not differ between the two groups. Given the critical role of the cerebellum in postural control, both in terms of receiving sensory input and in terms of regulating motor output, these findings suggest

that it is possible that the hyper-reactivity in ASD participants' postural control shown in the present study is due to dysfunction of the sensorimotor regions of the cerebellum.

Our study had a number of limitations. We did not study the developmental trajectory of ASD-related changes in postural control, thus, our findings are applicable only to high functioning adults with ASD. Furthermore, balance control in our study was assessed in a highly controlled laboratory environment, which means that it may not generalize to real life dynamic balance tasks like standing on a moving bus, or in a crowded room. Further research using more ecologically valid tasks is needed to uncover ASD-related differences in real-life conditions.

References

Bailey, A., Luthert, P., Dean, A., Harding, B., Janota, I., Montgomery, M., . . . Lantos, P. (1998). A clinicopathological study of autism. *Brain, 121 (Pt 5)*, 889-905.

Baker, A. E., Lane, A., Angley, M. T., & Young, R. L. (2008). The relationship between sensory processing patterns and behavioural responsiveness in autistic disorder: a pilot study. *Journal of Autism and Developmental Disorders, 38*(5), 867-875. doi: 10.1007/s10803-007-0459-0

Balasubramaniam, R., Riley, M. A., & Turvey, M. T. (2000). Specificity of postural sway to the demands of a precision task. *Gait and Posture, 11*(1), 12-24.

Balasubramaniam, R., & Wing, A. M. (2002). The dynamics of standing balance. *Trends in Cognitive Sciences, 6*(12), 531-536. doi: 10.1016/S1364-6613(02)02021-1

Baranek, G. T., David, F. J., Poe, M. D., Stone, W. L., & Watson, L. R. (2006). Sensory Experiences Questionnaire: discriminating sensory features in young children with autism, developmental delays, and typical development. *Journal of Child Psychology and Psychiatry, 47*(6), 591-601. doi: 10.1111/j.1469-7610.2005.01546.x

Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2005). Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. *Brain, 128*(Pt 10), 2430-2441. doi: 10.1093/brain/awh561

Black, F. O., Wall, C., 3rd, & Nashner, L. M. (1983). Effects of visual and support surface orientation references upon postural control in vestibular deficient subjects. *Acta Otolaryngologica, 95*(3-4), 199-201. doi: 10.3109/00016488309130936

Brown, C. E., & Dunn, W. (2002). *Adolescent/adult sensory profile*. San Antonio, TX: Pearson.

Clark, S., & Riley, M. A. (2007). Multisensory information for postural control: sway-referencing gain shapes center of pressure variability and temporal dynamics. *Experimental Brain Research, 176*(2), 299-310. doi: 10.1007/s00221-006-0620-6

Constantino, J. N., & Gruber, C. P. (2005). *The social responsiveness scale*. Los Angeles, CA: Western Psychological Services.

Doumas, M., Smolders, C., & Krampe, R. T. (2008). Task prioritization in aging: effects of sensory information on concurrent posture and memory performance. *Experimental Brain Research, 187*(2), 275-281. doi: 10.1007/s00221-008-1302-3

Fournier, K. A., Amano, S., Radonovich, K. J., Bleser, T. M., & Hass, C. J. (2014). Decreased dynamical complexity during quiet stance in children with autism spectrum disorders. *Gait Posture, 39*(1), 420-423. doi: 10.1016/j.gaitpost.2013.08.016

Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: a synthesis and meta-analysis. *Journal of Autism and Developmental Disorders, 40*(10), 1227-1240. doi: 10.1007/s10803-010-0981-3

Fournier, K. A., Kimberg, C. I., Radonovich, K. J., Tillman, M. D., Chow, J. W., Lewis, M. H., . . . Hass, C. J. (2010). Decreased static and dynamic postural control in children with autism spectrum disorders. *Gait and Posture, 32*(1), 6-9. doi: 10.1016/j.gaitpost.2010.02.007

Fuentes, C. T., Mostofsky, S. H., & Bastian, A. J. (2011). No proprioceptive deficits in autism despite movement-related sensory and execution impairments. *Journal of Autism and Developmental Disorders, 41*(10), 1352-1361. doi: 10.1007/s10803-010-1161-1

Furman, J. M., Osorio, M. J., & Minshew, N. J. (2015). Visual and Vestibular Induced Eye Movements in Verbal Children and Adults with Autism. *Autism Research*. doi: 10.1002/aur.1481

Ghanizadeh, A. (2011). Sensory processing problems in children with ADHD, a systematic review. *Psychiatry Investigations, 8*(2), 89-94. doi: 10.4306/pi.2011.8.2.89

Glazebrook, C., Gonzalez, D., Hansen, S., & Elliott, D. (2009). The role of vision for online control of manual aiming movements in persons with autism spectrum disorders. *Autism, 13*(4), 411-433. doi: 10.1177/1362361309105659

Goldberg, M. C., Landa, R., Lasker, A., Cooper, L., & Zee, D. S. (2000). Evidence of normal cerebellar control of the vestibulo-ocular reflex (VOR) in children with high-functioning autism. *Journal of Autism and Developmental Disorders, 30*(6), 519-524. doi: 10.1023/A:1005631225367.

Gowen, E., & Hamilton, A. (2013). Motor abilities in autism: a review using a computational context. *Journal of Autism and Developmental Disorders, 43*(2), 323-344. doi: 10.1007/s10803-012-1574-0

Graham, S. A., Abbott, A. E., Nair, A., Lincoln, A. J., Muller, R. A., & Goble, D. J. (2015). The Influence of Task Difficulty and Participant Age on Balance Control in ASD. *Journal of Autism and Developmental Disorders, 45*(5), 1419-1427. doi: 10.1007/s10803-014-2303-7

Greffou, S., Bertone, A., Hahler, E. M., Hanssens, J. M., Mottron, L., & Faubert, J. (2012). Postural hypo-reactivity in autism is contingent on development and visual environment: a fully immersive virtual reality study. *Journal of Autism and Developmental Disorders, 42*(6), 961-970. doi: 10.1007/s10803-011-1326-6

Happe, F., & Charlton, R. A. (2012). Aging in autism spectrum disorders: a mini-review. *Gerontology, 58*(1), 70-78. doi: 10.1159/000329720

Hashimoto, T., Tayama, M., Murakawa, K., Yoshimoto, T., Miyazaki, M., Harada, M., & Kuroda, Y. (1995). Development of the brainstem and cerebellum in autistic patients. *Journal of Autism and Developmental Disorders, 25*(1), 1-18. doi: 10.1007/BF02178163

Hilton, C., Wente, L., LaVesser, P., Ito, M., Reed, C., & Herzberg, G. (2007). Relationship between motor skill impairment and severity in children with Asperger syndrome. *Research in Autism Spectrum Disorders, 1*(4), 339-349. doi: 10.1016/J.Rasd.2006.12.003

Kern, J. K., Trivedi, M. H., Grannemann, B. D., Garver, C. R., Johnson, D. G., Andrews, A. A., . . . Schroeder, J. L. (2007). Sensory correlations in autism. *Autism, 11*(2), 123-134. doi: 10.1177/1362361307075702

Mamo, D. C., Pollock, B. G., Mulsant, B., Houck, P. R., Bensasi, S., Miller, M. C., . . . Reynolds, I. C. (2002). Effects of nortriptyline and paroxetine on postural sway in depressed elderly patients. *American Journal of Geriatric Psychiatry, 10*(2), 199-205. doi: 10.1097/00019442-200203000-00011

Marko, M. K., Crocetti, D., Hulst, T., Donchin, O., Shadmehr, R., & Mostofsky, S. H. (2015). Behavioural and neural basis of anomalous motor learning in children with autism. *Brain*, 138(Pt 3), 784-797. doi: 10.1093/brain/awu394

Maurer, C., Mergner, T., & Peterka, R. J. (2006). Multisensory control of human upright stance. *Experimental Brain Research*, 171(2), 231-250. doi: 10.1007/s00221-005-0256-y

McCollum, G., Shupert, C. L., & Nashner, L. M. (1996). Organizing sensory information for postural control in altered sensory environments. *Journal of Theoretical Biology*, 180(3), 257-270. doi: 10.1006/jtbi.1996.0101

Minshew, N. J., Sung, K., Jones, B. L., & Furman, J. M. (2004). Underdevelopment of the postural control system in autism. *Neurology*, 63(11), 2056-2061. doi: 10.1212/01. WNL. 0000145771. 98657. 62

Molloy, C. A., Dietrich, K. N., & Bhattacharya, A. (2003). Postural stability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 33(6), 643-652. doi: 10.1023/B:Jadd.0000006001.00667.4c

Murakami, J. W., Courchesne, E., Press, G. A., Yeung-Courchesne, R., & Hesselink, J. R. (1989). Reduced cerebellar hemisphere size and its relationship to vermal hypoplasia in autism. Archives in Neurology, 46(6), 689-694. doi: 0.1001/archneur.1989.00520420111032

Nashner, L. (1984). Analysis of stance posture in humans Handbook of Behavioral Neurobiology (pp. 527–565). New York: Plenum.

Nashner, L. M. (1976). Adapting reflexes controlling the human posture. *Experimental Brain Research*, 26(1), 59-72. doi: 10.1007/BF00235249

Nashner, L. M., Black, F. O., & Wall, C., 3rd. (1982). Adaptation to altered support and visual conditions during stance: patients with vestibular deficits. *Journal of Neuroscience*, 2(5), 536-544. doi: 10.1007/BF00235249

Pellicano, E., Gibson, L., Maybery, M., Durkin, K., & Badcock, D. R. (2005). Abnormal global processing along the dorsal visual pathway in autism: a possible mechanism for weak visuospatial coherence? *Neuropsychologia*, 43(7), 1044-1053. doi: 10.1016/j.neuropsychologia.2004.10.003

Pellicano, E., & Gibson, L. Y. (2008). Investigating the functional integrity of the dorsal visual pathway in autism and dyslexia. *Neuropsychologia*, 46(10), 2593-2596. doi: 10.1016/j.neuropsychologia.2008.04.008

Peterka, R. J. (2002). Sensorimotor integration in human postural control. *Journal of Neurophysiology, 88*(3), 1097-1118. doi: 88: 1097–1118, 2002; 10.1152/jn.00605.2001.

Peterka, R. J., & Black, F. O. (1990). Age-related changes in human posture control: sensory organization tests. *Journal of Vestibular Research, 1*(1), 73-85.

Peterka, R. J., & Loughlin, P. J. (2004). Dynamic regulation of sensorimotor integration in human postural control. *Journal of Neurophysiology, 91*(1), 410-423. doi: 10.1152/jn.00516.2003

Ritvo, E. R., Freeman, B. J., Scheibel, A. B., Duong, T., Robinson, H., Guthrie, D., & Ritvo, A. (1986). Lower Purkinje cell counts in the cerebella of four autistic subjects: initial findings of the UCLA-NSAC Autopsy Research Report. *American Journal of Psychiatry, 143*(7), 862-866. doi: 10.1176/ajp.143.7.862

Rubenstein, L. Z. (2006). Falls in older people: epidemiology, risk factors and strategies for prevention. *Age and Ageing, 35 Suppl 2*, ii37-ii41. doi: 10.1093/ageing/afl084

Scott, J. A., Schumann, C. M., Goodlin-Jones, B. L., & Amaral, D. G. (2009). A comprehensive volumetric analysis of the cerebellum in children and adolescents with autism spectrum disorder. *Autism Research, 2*(5), 246-257. doi: 10.1002/aur.97

Sergeant, J. A., Piek, J. P., & Oosterlaan, J. (2006). ADHD and DCD: a relationship in need of research. *Human Movement Science, 25*(1), 76-89. doi: 10.1016/j.humov.2005.10.007

Sherrington, C., Tiedemann, A., Fairhall, N., Close, J. C., & Lord, S. R. (2011). Exercise to prevent falls in older adults: an updated meta-analysis and best practice recommendations. *New South Wales Public Health Bulletin, 22*(3-4), 78-83. doi: 10.1071/NB10056

Sherrington, C., Whitney, J. C., Lord, S. R., Herbert, R. D., Cumming, R. G., & Close, J. C. (2008). Effective exercise for the prevention of falls: a systematic review and meta-analysis. *Journal of the American Geriatrics Society, 56*(12), 2234-2243. doi: 10.1111/j.1532-5415.2008.02014.x

Therrien, A. S., & Bastian, A. J. (2015). Cerebellar damage impairs internal predictions for sensory and motor function. *Current Opinion in Neurobiology, 33*, 127-133. doi: 10.1016/j.conb.2015.03.013

Wallace, M. T., & Stevenson, R. A. (2014). The construct of the multisensory temporal binding window and its dysregulation in developmental disabilities. *Neuropsychologia*, 64C, 105-123. doi: 10.1016/j.neuropsychologia.2014.08.005

Watling, R. L., Deitz, J., & White, O. (2001). Comparison of sensory profile scores of young children with and without autism spectrum disorders. *American Journal of Occupational Therapy*, 55(4), 416-423. doi: 10.5014/ajot.55.4.416

Figure Caption Sheet

Figure 1. Posture conditions. The stick figures depict the four posture conditions: a) Fixed, b) Visual, c) Proprioceptive and d) Both. Straight arrows depict body sway.

Figure 2. Ellipse area measures for controls and participants with ASD in all conditions. Error bars represent ± 1 standard error of the mean.

Figure 3. a) AP SD and b) ML SD measures for controls and participants with ASD in all conditions. Note that the scale in 2a is ten times larger than in 2b. Error bars represent ± 1 standard error of the mean.

Figure 1

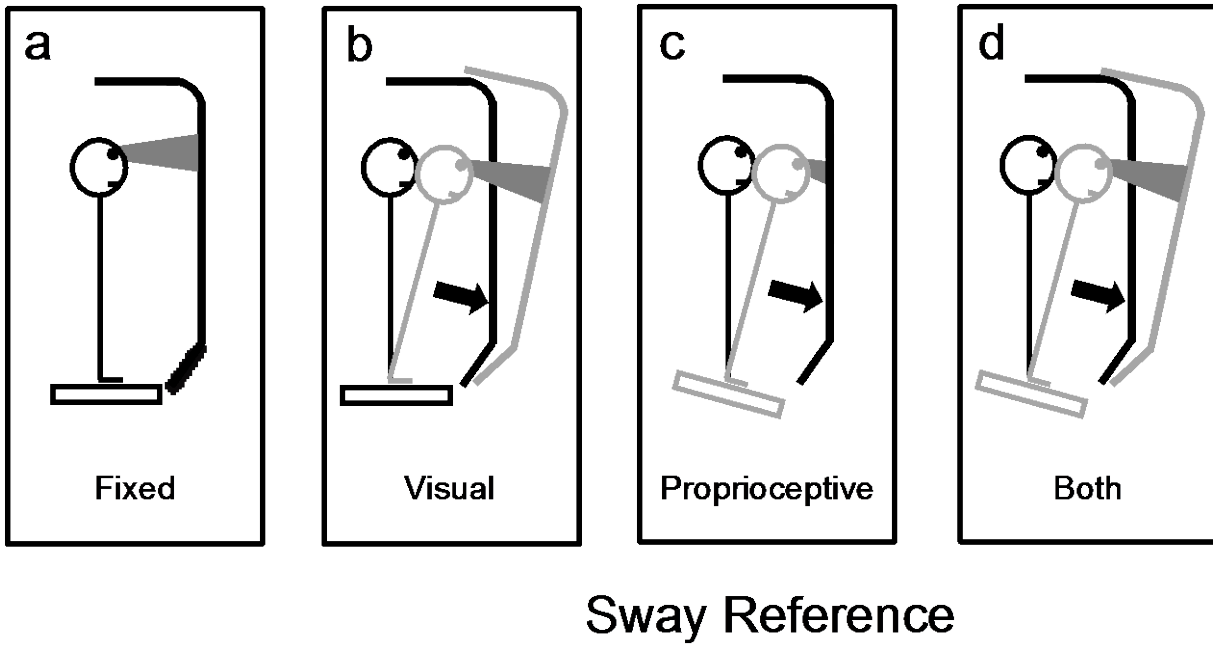

Figure 2

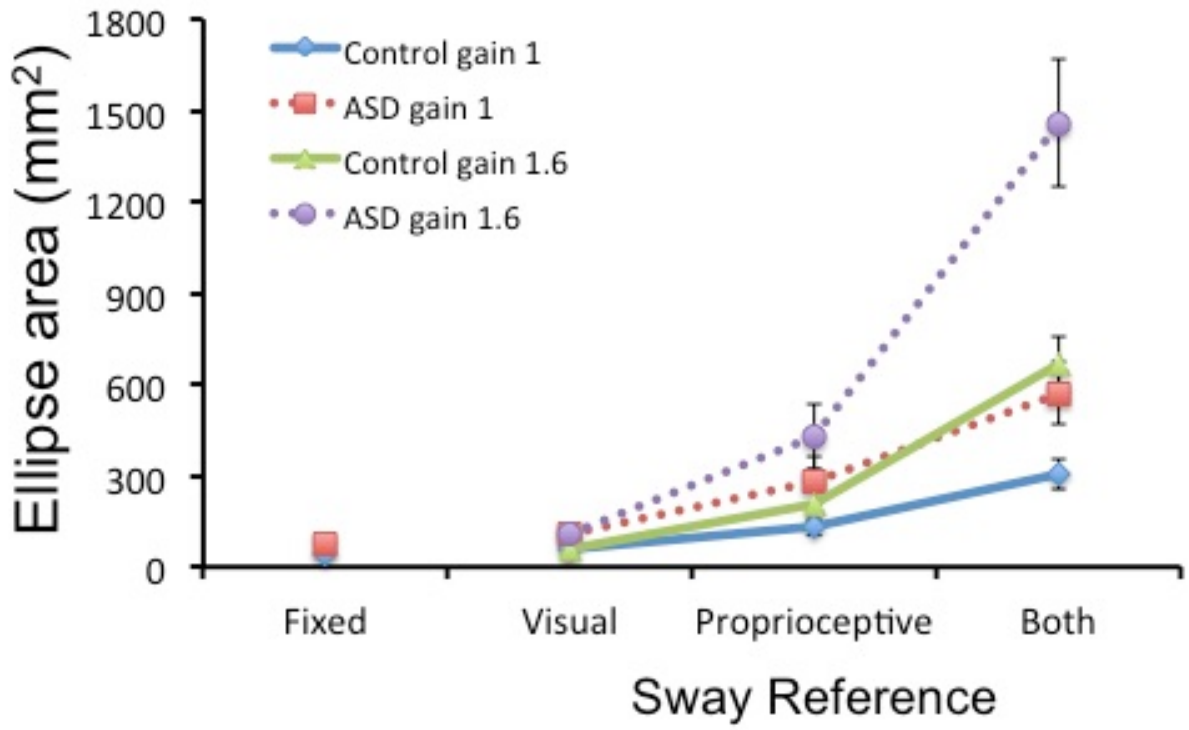
Ellipse area (mm²)
1800
1500
1200
900
600
300
0
Control gain 1
ASD gain 1
Control gain 1.6
ASD gain 1.6
Fixed
Visual
Proprioceptive
Both
Sway Reference

Figure 3

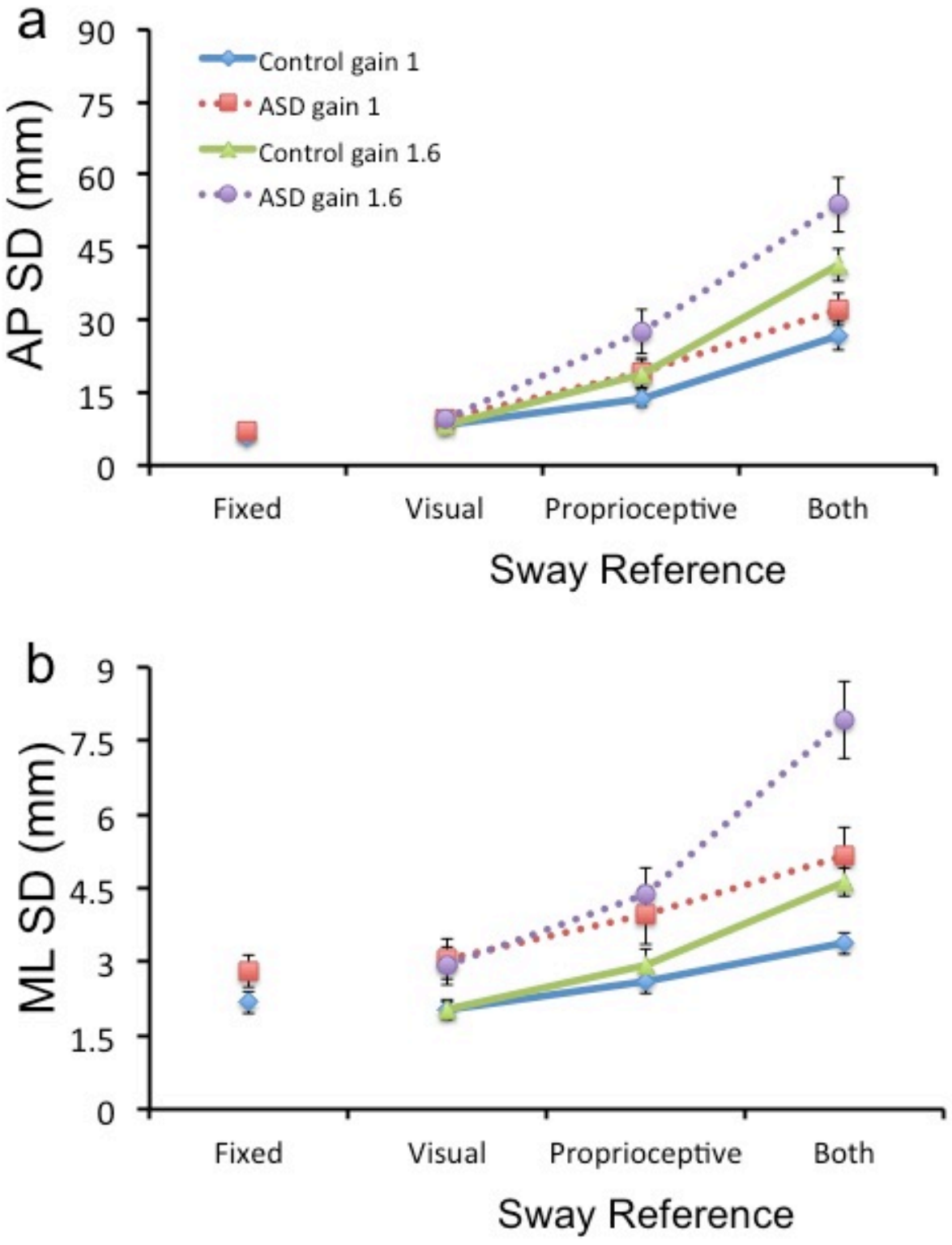
a
AP SD (mm)
90
75
60
45
30
15
0
Control gain 1
ASD gain 1
Control gain 1.6
ASD gain 1.6
Fixed
Visual
Proprioceptive
Both
Sway Reference
b
ML SD (mm)
9
7.5
6
4.5
3
1.5
0
Fixed
Visual
Proprioceptive
Both
Sway Reference

Table 1. Participant characteristics, group means and SD, and p-values from group comparisons using independent samples t-tests for all screening tests

Characteristic	ASD (n=15) Mean (SD)	Controls (n=15) Mean (SD)	p-value
Age	23.9 (5.7)	26.1 (6.9)	0.365
Sex (female/male)	2/13	2/13	N/A
Height (cm)	177.0 (11.3)	173.4 (12.9)	0.410
Full scale IQ (WASI)	105.5 (11.9)	113.4 (14.8)	0.117
SP Low Registration	2.5 (0.7)	2.0 (0.4)	0.016
SP Sensation Seeking	3.0 (0.4)	3.0 (0.3)	0.564
SP Sensory Sensitivity	2.6 (0.6)	2.4 (0.3)	0.279
SP Sensation Avoiding	2.9 (0.7)	2.2 (0.3)	0.002
Social Responsiveness Scale (SRS)	72(11.9)	N/A	N/A

SP: Sensory Profile

Author Note

Michail Doumas, Roisin McKenna and Blain Murphy, School of Psychology, Queen's University Belfast, 18-30 Malone Road, Belfast, BT9 5BN, UK.

The authors would like to thank Sarah Trimby and Lauren Logan for their help with data collection, Autism Initiatives, our participants and their families for their support.

Correspondence concerning this article should be addressed to Michail Doumas, School of Psychology, Queen's University Belfast, 18-30 Malone Road, Belfast, BT9 5BN, UK. E-mail: m.doumas@qub.ac.uk