Postural Control Processes During Static and Dynamic Activities in Autism Spectrum Disorder

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Abstract

Individuals with autism spectrum disorder (ASD) show multiple postural control deficits, including reduced postural stability during standing and reduced amplitude and frequency of anticipatory postural adjustments (APA) prior to planned movements. This study aims to identify mechanisms of postural control deficits in ASD during more challenging standing conditions including coordination of postural control processes used to support mediolateral (ML) and anteroposterior (AP) adjustments. We also examined APAs made during the initiation of walking to characterize predictive motor processes supporting postural control in ASD. Seventeen individuals with ASD were matched with 20 typically developing (TD) controls on age, gender ratio, nonverbal IQ, and body mass index (BMI). Participants completed three tests of postural control. During the first test, they stood with their feet shoulder width apart (neutral stance). During the second test, they stood with feet close together (Romberg one) in order to assess postural control during a more challenging standing condition in which the base of support is reduced. During the third test, participants stood with feet shoulder width apart and swayed their torso in a circle (circular sway). The standard deviation (SD) of their center of pressure (COP) in the ML and AP directions and the COP trajectory length were examined for each condition. We also assessed mutual information (MI), or the shared dependencies between COP in the ML and AP directions. Finally, individuals completed a step initiation task in which they took a step forward from one force platform to another. The APA amplitude and duration prior to stepping were measured, as were the maximum lateral sway during stepping, step distance, step velocity, and step duration. Individuals with ASD showed increased COP trajectory length relative to TD controls but no differences in COP SD during the standing tests. Compared to controls, participants with ASD showed greater levels of MI during static stance but reduced levels of MI during circular sway. During the step initiation task, groups did not differ on the amplitude or

duration of APAs. During stepping, individuals with ASD showed reduced lateral sway, shorter step durations, and increased step velocity. Our finding that individuals with ASD show increased MI during circular sway suggests that they have a reduced ability to effectively coordinate distinct joint movements during dynamic postural adjustments. Our finding that individuals with ASD show reduced lateral sway when stepping suggests that motor rigidity may interfere with balance and gait in patients implicating basal ganglia circuits involved in guiding rapid or ballistic movements.

Key Words: Autism Spectrum Disorder, Postural Control, Mutual Information, Step Initiation, Anticipatory Postural Adjustments

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Postural control processes during static and dynamic activities in autism spectrum disorder

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by social and communication deficits and restricted and repetitive behaviors (American Psychiatric Association, 2013). Multiple comorbid issues also are common in individuals with ASD including intellectual disability, anxiety, self-injurious behaviors, and impulsivity and hyperactivity as well as medical conditions including gastrointestinal issues, and epilepsy (Veenstra-VanderWeele & Blakely, 2012). In addition, motor problems, including poor upper and lower limb coordination during reaching and walking, reduced postural control, and reduced anticipatory control of motor behavior (Bhat, Landa, & Galloway, 2011) frequently are seen in individuals with ASD. Studies of infants with ASD have suggested that motor deficits are present early in development, may be among the earliest signs of the disorder (Baranek, 1999; Esposito, Venuti, Maestro, & Muratori, 2009; Flanagan, Landa, Bhat, & Bauman, 2012; Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998), and contribute to worse social, language, and cognitive outcomes (Travers et al., 2016). Further, the brain systems that support motor behavior in healthy individuals have been repeatedly implicated in post-mortem and MRI studies of ASD (Bailey et al., 1998; Stanfield et al., 2008; Whitney, Kemper, Bauman, Rosene, & Blatt, 2008) suggesting that defining the basic motor processes that are affected in individuals with ASD may provide insights into the neural bases of this disorder.

Postural control deficits consistently have been shown in individuals with ASD (Bhat et al., 2011; Kohen-Raz, Volkmar, & Cohen, 1992; Memari, Ghanouni, Shayestehfar, & Ghaheri, 2014; Minshew, Sung, Jones, & Furman, 2004; Molloy, Dietrich, & Bhattacharya, 2003) and they are considered associated features supporting a diagnosis (American Psychiatric Association, 2013). Further, delays in postural stability during sitting, standing, and walking

have been documented in the first years of life in ASD (Ozonoff et al., 2008; Teitelbaum et al., 1998) and appear to be predictive of worse outcomes (MacDonald, Lord, & Ulrich, 2014; Travers et al., 2016). Despite these findings, the motor control processes that disrupt postural stability in ASD are not well understood.

Postural control reflects the integration of both feedforward and feedback motor control mechanisms (Massion, 1994). Feedforward motor control processes play a more prominent role in initial or rapid movements made prior to sensory feedback being available to guide actions. For example, anticipatory postural shifts are made prior to initiating a step forward in order to maintain stability as weight is displaced. During ongoing movements, sensory feedback input from proprioceptive, somatosensory, vestibular, and visual systems are used to continuously adjust behavioral output (Horak, 2006; Massion, 1994). During postural control, individuals use sensory feedback information to correct sway caused by inherent physiological noise as well as internal (e.g., muscle fatigue) and external perturbations (e.g., carrying a heavy item). When sensory feedback processes are disrupted, postural adjustments are less coordinated, less smooth and less precise (Crapse & Sommer, 2008; Subramanian et al., 2017; van Beers, 2009). These distinct postural control processes involve separate brain systems suggesting that determining the extent to which feedforward and feedback motor control mechanisms are impaired in ASD may provide key insights into neural underpinnings of the disorder.

Research also has shown the importance of coordinating joint movements to maintain postural stability. When maintaining postural stability while standing still, adjustments along mediolateral (ML) and anteroposterior (AP) axes are synchronized to correct sway in all directions. This is especially important for more challenging postural conditions such as when one foot is placed in front of the other in tandem (Wang & Newell, 2014), and during the

"Romberg one" stance in which individuals place their feet side by side (Notermans, Van Dijk, Van der Graaf, Van Gijn, & Wokke, 1994). The coordination of joint movements is necessary to prevent falling during both self-initiated movements (e.g. reaching for an object or taking a step) and in response to external perturbations (e.g. getting pushed) (Horak, 2006). For example, younger adults rely on the coordination of hip and ankle joints when they encounter external perturbations, while older adults, whose joint movements are more restricted, use multiple discrete control processes to maintain balance which may result in reduced stability (Wang & Newell, 2014).

Anticipatory postural adjustments (APAs) are critical for planning movements or shifting bodyweight in order to maintain postural stability prior to the onset of a goal-directed movement (Aruin, 2002; Schmitz, Martineau, Barthélémy, & Assaiante, 2003). APAs reflect feedforward processes, or predictive commands, that aim to shift the center of pressure (COP) in anticipation of perturbations that are associated with a planned movement (Massion, 1992). APAs were first demonstrated using electromyography (EMG) recording of muscle activity during voluntary upper limb movements (Belen'kii, Gurfinkel', & Pal'tsev, 1967). The authors documented anticipatory muscle activation in the trunk and legs prior to voluntary arm lifting and found that this anticipatory muscle activation helped maintain balance. APAs also have been shown using COP recordings. Studying APAs during upper-limb movements, Riach and Hayes (1990) found that anticipatory COP adjustments are evident in children as young as age four years. The authors indicated that APAs in the AP direction were not made as consistently as anticipatory ML adjustments in young children suggesting that distinct APAs mature at different rates. Additional research measuring both COP displacement and muscle activation during self-initiated arm movements has suggested that by age seven years, typically developing (TD)

children are able to generate and utilize APAs at an adult level (Girolami, Shiratori, & Aruin, 2010).

APAs are especially important during the initiation of walking. Walking is a complex set of coordinated movements requiring multiple dynamic postural adjustments in order to maintain balance (Assaiante, Woollacott, & Amblard, 2000). It requires the movement of the center of mass of the body from a large base of support to a small base of support (Fournier et al., 2010). Gait initiation involves two distinct phases, including an anticipatory adjustment of the COP made before the voluntary step and a compensatory shift in COP made once the individual has started taking a step. In order to efficiently maintain postural control, anticipatory and compensatory postural adjustments must be dynamically applied prior to, during, and subsequent to a desired movement (Girolami et al., 2010). In studying anticipatory motor behaviors during step initiation in children, Ledebt, Bril, and Breniere (1998) found that, while APAs were seen in children as young as 2.5 years old, they were not seen consistently until later childhood. Further, posterior directed anticipatory movements were evident during early childhood prior to the development of lateral APA movements (Ledebt, Bril, & Breniere, 1998). These findings implicate multiple distinct APA processes involved in the initiation of walking that develop at different rates.

Postural Control During Static and Dynamic Stances in ASD

Studies have suggested that children with ASD are less stable than controls during standing tasks (Kohen-Raz et al., 1992; Memari et al., 2013; Memari et al., 2014; Molloy et al., 2003). In a series of postural conditions, including Romberg eyes open and eyes closed conditions, Kohen-Raz, Volkmar, and Cohen (1992) found that, compared with TD children and children with intellectual disabilities (ID), children with ASD (ages 6-20 years) exhibited

elevated levels of COP variability and lateral sway. Children with ASD also showed increased COP variability in the ML compared to AP direction, whereas TD children showed the opposite pattern (Memari et al., 2013). Given that increased lateral sway is seen in TD toddlers, these results suggest that the development of postural control in ASD is delayed or remains immature into early adulthood (Kohen-Raz et al., 1992). Additional studies have indicated that individuals with ASD show an increased reliance on visual and proprioceptive information to maintain stability as evidenced by more severe decreases in postural stability relative to controls during conditions where sensory feedback information is disrupted (Minshew et al., 2004; Molloy et al., 2003). Similarly, Wang et al. (2016) found that participants with ASD demonstrate increased COP variability during both static and dynamic stances in which they were instructed to lean in a particular direction as far as possible. Elevations in COP variability in ASD were more severe during dynamic standing conditions, suggesting that patients show greater levels of impairment during conditions in which demands on feedback motor control processes are increased.

Wang et al. (2016) also examined the extent to which postural control processes involving ankle joints that control AP sway operated independently or in concert with hip joint processes that control ML sway. During static stance, these distinct mechanisms show considerable cross-talk, or mutual information (MI), which helps coordinate postural control mechanisms to reduce sway in all directions. During intentional sway in a target direction, ankle and hip processes operate more independently to ensure that sway is directional. When engaging in intentional sway, individuals with ASD showed a reduced ability to decouple hip and ankle joint control processes as reflected by increased MI relative to controls (Wang et al., 2016). Therefore, patients' dynamic, intentional sway was less directional than that seen in control individuals. These findings indicate that individuals with ASD may show deficits in adaptively

modulating the degree of coordination among distinct joints across different naturalistic postural conditions. By determining the extent to which individuals with ASD are able to adapt their level of MI across different joint processes during naturalistic conditions, tests of dynamic postures may provide important insights into affected motor control mechanisms and the integrity of neural systems that support the coordination of distinct movements.

Anticipatory Postural Adjustments (APAs) in ASD

Individuals with ASD also appear to show deficits in feedforward motor control processes, evident as early as infancy (Brisson, Warreyn, Serres, Foussier, & Adrien-Louis, 2012; Landa, Haworth, & Nebel, 2016). For example, Schmitz, Martineau, Barthélémy, and Assaiante (2003) found that APAs were reduced in amplitude in children with ASD (ages 5-10 years) during a bimanual load-lifting task. During this task, participants sat with their left forearm horizontal and a load was either suspended below the arm or placed on a platform. During involuntary conditions, the experimenter caused the release of the load at an unpredictable time. During a voluntary condition, the load was placed on the platform and participants were instructed to lift it at their own pace (Schmitz, Martin, & Assaiante, 2002). The authors found that during the voluntary condition, TD children anticipated the muscle perturbation leading to a decrease in muscle activation, whereas children with ASD did not show this same anticipatory muscle response. Instead, children with ASD responded with a compensatory postural adjustment resulting from feedback inputs processed after the voluntary load lifting. These results suggest that children with ASD have deficits in feedforward control of motor behavior and may compensate for this deficit by relying on feedback motor adjustments to maintain postural control. Studying a similar task, Martineau, Schmitz, Assaiante, Blanc, and Barthélémy (2004) examined sensorimotor cortical activity involved in anticipatory control of

voluntary movements. The authors found that TD children used APAs that were associated with increased activity localized to the primary motor cortex. In contrast, individuals with ASD did not show anticipatory motor cortical activity suggesting that deficits in APAs reflect reduced preparatory activity within neocortical systems involved in motor planning (Martineau, Schmitz, Assaiante, Blanc, & Barthelemy, 2004). The extent to which deficits in feedforward control processes contribute to deficits in postural control has not yet been determined.

Brain Processes Supporting Postural Control

Brain systems involved in feedforward and feedback control of postural stability have been well delineated in non-human primate and human lesion studies as well as non-invasive neuroimaging studies. These studies have indicated that the anterior vermis of the cerebellum plays a key role in feedforward and feedback postural control and is innervated by extrastriate and striate cortices involved in processing visual feedback information (Apps & Garwicz, 2005; Mosconi, Wang, Schmitt, Tsai, & Sweeney, 2015). Additionally, increases in brain activation are seen in anterior and posterior vermis and inferior occipital and temporal cortices when transitioning from a supine to Romberg one position (Ouchi, Okada, Yoshikawa, Nobezawa, & Futatsubashi, 1999). The vermis and intermediate areas of the cerebellum rely on input from both motor and parietal cortices to process visual-spatial information, whereas input from the spinal cord provides somatosensory and proprioceptive feedback information (Apps & Garwicz, 2005; Horak, 2006).

Cortico-cerebellar systems involved in postural control have been repeatedly implicated in ASD (Bailey et al., 1998; Whitney et al., 2008). The cerebellum includes multiple distinct subregions that are involved in different types of sensorimotor behaviors, including postural control, eye movements, and upper limb movements (e.g. reaching). Each of these types of

actions has been found to be impaired in ASD (for review see Mosconi et al., 2015). A meta-analysis of structural MRI studies has suggested that cerebellar hemispheric volumes are increased in individuals with ASD relative to healthy controls, and that volumes of cerebellar vermal lobules VI-VII and VIII-X are decreased in patients (Stanfield et al., 2008). These findings are particularly notable in the context of the medial cerebellum's (i.e., vermis and intermediate zones) prominent role in postural control and walking (Apps & Garwicz, 2005). Additionally, it has been demonstrated that individuals with ASD also show atypical functional connectivity between cerebellum and motor cortex during gross motor behaviors (Mostofsky et al., 2009).

Cortico-basal ganglia circuits also are implicated in the maintenance of postural stability through the initiation and planning of movements such as walking (Baev et al., 2002; Patla, 1996). Studies of these circuits have shown that the striatum, which is comprised of the caudate nucleus and the putamen, is the primary input nucleus of the basal ganglia (Graybiel, 2000; Gunaydin & Kreitzer, 2016) and receives inputs from cortex and thalamus. Direct and indirect pathways from the striatum work together to facilitate and inhibit movements (Gunaydin & Kreitzer, 2016). The output structures of the basal ganglia include the internal globus pallidus which connects to the brainstem as well as cortex through the thalamus. These cortico-basal ganglia circuits are proposed to control the initiation and termination of goal-directed behaviors (Gunaydin & Kreitzer, 2016).

Post-mortem studies, neuroimaging studies, and studies of animal models have implicated cortico-basal ganglia circuits in ASD with impairments influencing a range of motor and core ASD symptoms (for a review see Subramanian et al., 2017). A post-mortem study of individuals with ASD showed that older individuals with ASD show increased volumes of

caudate nuclei and nucleus accumbens (Wegiel et al., 2014) associated with more severe repetitive behaviors (Hollander et al., 2005). Neuroimaging studies of individuals with ASD also showed putamen enlargement (Sato et al., 2014). Additionally, a study by Vilensky et al (1981) found that individuals with ASD show gait patterns similar to patients with striatal dysfunction and resembling parkinsonian gait as evidenced by decreased stride length (Vilensky, Damasio, & Maurer, 1981).

Based on findings that feedforward and feedback motor control processes and their neural underpinnings are compromised in ASD, we propose the following three primary aims. Aim 1 will identify mechanisms of postural control deficits in individuals with ASD during both static and dynamic standing conditions. During the static conditions, participants will stand with feet shoulder width apart (neutral stance) or feet close together (Romberg one stance). The Romberg one condition requires a reduced base of support and therefore increases the challenge placed upon feedback control of motor systems. Postural control tests using the Romberg one stance are frequently administered in neurology to assess cerebellar and proprioceptive deficits (Lanska & Goetz, 2000). During the dynamic condition, participants will initiate and maintain a circular sway while in a neutral stance. We predict that individuals with ASD will show increased COP variability and COP trajectory length across all static and dynamic stance conditions relative to TD controls. Further, we expect that the severity of postural control deficits in ASD will increase as greater demands are placed on feedback motor control processes. Specifically, we hypothesize more severe impairments in ASD relative to controls during Romberg one compared to neutral stance, and during circular sway compared to both Romberg one and neutral stances.

Aim 2 will examine the coordination of distinct postural control processes used to support ML and AP adjustments in ASD during both static and dynamic standing conditions. By

examining these distinct processes, this study will determine the extent to which individuals with ASD are able to effectively coordinate their joint movements during standing conditions in which increased MI is advantageous in contrast to our prior study examining sway restricted to a single direction when MI should be reduced. We hypothesize that TD controls will show a decrease in MI in the Romberg one stance relative to the neutral stance and that this decrease will be smaller in the ASD group compared to the TD group. Additionally, we hypothesize that there will be an increase in MI during the circular sway condition relative to both static conditions in TD controls and that this increase will be smaller in the ASD group.

Aim 3 will examine step initiation to understand mechanisms of postural control during walking in ASD. We also hypothesize that APAs used in individuals with ASD, will be smaller in amplitude and duration. During the compensatory phase, we hypothesize that individuals with ASD will show reduced lateral sway suggesting greater instability when stepping. Based on previous findings showing that deficits in postural control are associated with more severe ASD symptoms (Radonovich, Fournier, & Hass, 2013; Travers, Powell, Klinger, & Klinger, 2013), we also will investigate the extent to which our measures of feedforward and feedback control of posture are associated with social-communication abnormalities and repetitive behaviors in individuals with ASD.

Methods

Participants

Seventeen individuals with ASD (ages 6-19 years) and 20 age, sex, non-verbal IQ, and Body Mass Index (BMI) matched TD control individuals completed tests of postural control and step initiation (Table 1). IQ was assessed using the Wechsler Abbreviated Scales of Intelligence (Stano, 1999). One TD control subject was unable to complete IQ testing due to scheduling

difficulties. Individuals with ASD were recruited through community advertisements and local clinics. For all patients, a diagnosis of ASD was established using the Autism Diagnostic Inventory-Revised (ADI-R) (Lord, Rutter, & Le Couteur, 1994), the Autism Diagnostic Observation Schedule – Second Edition (ADOS-2) (Lord et al., 2012) and expert clinical opinion based on DSM-V criteria. Three ASD subjects' parents were unable to complete the ADI-R, but these participants met ASD classification on the ADOS-2 and DSM-V criteria for ASD. Potential participants were excluded if they had any known genetic condition associated with ASD.

TD participants were recruited from the community and were required to have a score of eight or lower on the Social Communication Questionnaire (SCQ) (Berument, Rutter, Lord, Pickles, & Bailey, 1999). TD participants were excluded for current or past psychiatric or neurological disorders, family history of ASD in first- or second-degree relatives, or a history of developmental or learning disorders, psychosis, or obsessive-compulsive disorder in first-degree relatives based on a screening interview.

No participants were taking medications known to affect motor performance at the time of testing, including antipsychotics, stimulants, or anticonvulsants (Reilly, Lencer, Bishop, Keedy, & Sweeney, 2008). No participant had a history of head injury, birth asphyxia or non-febrile seizure. Participants 18 years of age or older provided written consent and minors provided assent in addition to written consent from their parent or legal guardian. All study procedures were approved by the local Institutional Review Board.

Apparatus and Procedures

Tests of static and dynamic standing and stepping were administered either during the initial study visit after completion of the clinical assessment or during a second visit. Participants completed four postural control tasks using two adjacent AMTI (American Mechanical

Technology, Inc., Watertown, MA) force platforms (Model: AccuGait; size: 49.78 × 49.78 cm; sampling rate: 1000 Hz). The four experimental conditions totaled 30-40 minutes. Short breaks were given between each trial and testing condition and additional breaks were given when necessary. Three successful trials were completed for each of the four conditions. Prior to each condition, the experimenter modeled the task. Participants were given practice before the test to ensure they understood the instructions.

Static stances. Two static standing conditions were administered: neutral stance and Romberg one stance. During neutral stance, participants stood as still as possible with their feet shoulder-width apart and their arms resting at their side in a relaxed position. During Romberg one stance, individuals stood with their feet close together and arms at their sides. Participants completed three 30-second trials for each static stance condition. COP variability (in the AP and ML directions), MI, and COP trajectory length were measured for each static stance condition.

Dynamic stance. To examine postural control during a more dynamic stance, participants completed a circular sway condition. They were asked to stand with their feet hip width apart and make a circle with their body. Participants were told to complete the circular sway condition using a natural speed, and they were reminded to keep both feet flat on the platform throughout testing. Data collection began five seconds after the participant started their circular sway. Participants rested for 30 seconds between each trial and for one minute between conditions. COP variability (in the AP and ML directions), MI, and COP trajectory length were measured.

Nine postural and dynamic stance trials were completed (three conditions x three trials). When participants did not follow testing instructions, additional trials were completed to reach three successful trials. The order of administration of static and dynamic standing conditions was counterbalanced across participants.

Step initiation. Stepping and APAs made prior to stepping were examined during a step initiation test. Participants completed trials in which they initiated one step forward from one force platform to an adjacent force platform. During each trial, participants stood still in a neutral stance for three to five seconds, received an auditory cue of either "right" or "left" prompting them to step with their right or left foot, and then stepped toward the second force platform at a comfortable speed and distance. In order to finish the trial, participants needed to have both feet resting on the second plate and standing in their neutral stance. For participants with shorter legs, the standing location on the first platform was adjusted prior to data collection to ensure that they stepped on the second force platform without either exaggerated extension of their leading leg or landing their feet in between the two force platforms. The direction of the auditory cue was randomized across trials, and the timing was randomly presented within three to five seconds after the experimenter confirmed that the participant was neutrally standing on the first platform. Each trial was followed by 10-seconds of rest. Three successful trials were collected for each participant. COP data from each trial were used to calculate APA duration and amplitude as well as step length, duration, velocity, and maximum lateral sway.

Data processing and analysis. Force and moment time series collected from the two force platforms were processed and analyzed in Matlab 2016b (MathWorks, Inc., Natick, MA). All kinetic data were down sampled to 100 Hz and low pass filtered using a 4^{th}-order double pass Butterworth filter with a cutoff frequency of 6 Hz. The COP time series for each force platform were derived from force and moment data consistent with prior methods (Prieto, Myklebust, Hoffmann, Lovett, & Myklebust, 1996). The COP_{NET} includes the COP time series in both AP ($COP_{AP\text{-}NET}$) and ML ($COP_{ML\text{-}NET}$) directions. The COP_{NET} time series were derived from COPs as well as the vertical ground reaction force of both plates (Wang, Jordan, & Newell, 2012;

Winter, 1995). For the static and dynamic standing conditions, the first five seconds of data and the last five seconds of data were removed in order to eliminate additional movements that were made while participants started and ended the task.

To assess participants' postural stability during the static and dynamic standing trials, we measured the SD of the COP time series in both the AP and ML directions as well as the COP trajectory length, or the sum of distances between points on the COP path (Prieto et al., 1996). To examine postural coordination during dynamic standing conditions, we measured the amount of MI shared between the COP in ML and AP directions. MI is a measure of shared dependency between two time series (Wang & Newell, 2014; Winter, 1995). In this study, MI is used to quantify the amount of shared information between the ankle and hip joints during different postural control conditions. Higher MI suggests more shared information across the COP_{AP} and COP_{ML} time series while lower MI suggests independent movements in the AP and ML directions (Wang et al., 2016).

The step initiation condition was separated into anticipatory and compensatory phases. To examine the anticipatory phase, APA amplitude and duration were examined. The onset of the anticipatory phase is defined as the first point where the COP in the ML direction is greater than two SDs from the baseline stance and remains for at least 50 milliseconds. In cases where there was no identifiable baseline stance recorded, the APA could not be calculated. The APA offset was defined as the point where the COP returned back to baseline before beginning the step. The APA contains a ML shift typically towards the stepping leg before the shift towards the standing leg and the onset of the step. APA amplitude is the maximum range of motion in the ML direction throughout the APA phase. The APA duration is measured as the time series between the onset and offset of the APA. During the compensatory phase of step initiation, we

measured participants' maximum lateral sway, step distance, step length, and step velocity. The maximum lateral sway is measured as the maximum $COP_{ML\text{-}NET}$ range of motion during the trial. The step distance is the amplitude of the forward step, which is defined as the $COP_{AP\text{-}NET}$ range of motion between the leading heel contacting the second force platform and the back toe lifting off the first platform. The step duration is calculated as the length of time between the leading heel contact on the second force platform and the back toe lifting off the first platform. Finally, the mean step velocity is calculated as the step distance over the step duration.

Clinical ratings of ASD severity. The ADI-R and ADOS-2 were used to examine ASD symptoms for each ASD participant and determine the extent to which postural control, APA, and step kinematics were associated with core symptoms of the disorder. The ADI-R (Lord et al., 1994) is a semi-structured parent/caregiver interview assessing current and past social interaction and communication behaviors characteristic of ASD, as well as the presence of restricted and repetitive behaviors. Higher scores reflect more severe abnormalities. The social, communication, and RRB algorithms were examined.

The ADOS-2 (Lord et al. 2012) is a semi-structured play-based assessment that uses developmentally appropriate social and play based interactions to elicit behaviors commonly impaired in ASD, including language and communication, reciprocal social interaction, play, stereotyped behaviors and restricted interests. The ADOS-2 has five different module levels (i.e., Toddler Module and Modules 1-4). Modules are differentiated based on age and individuals' language ability. Participants in the present study completed either Module 3 (children or adolescents) or 4 (adults). Empirically derived algorithm scores ranging from 1-10 (1 being low severity and 10 being the highest severity) are calculated from raw ADOS totals separated by Module and age (Gotham, Pickles, & Lord, 2009).

Statistical Analyses

Data for each dependent variable was averaged across trials for each participant. To examine between group differences in COP variability we ran a 3 (condition: neutral stance vs. Romberg one vs. circular sway) x 2 (COP direction: AP vs. ML) x 2 (group: ASD vs. TD) repeated measures ANOVA. In this model, condition and COP direction were the within subject factors and group was the between-subject factor. In order to assess MI across static and dynamic stances, we ran a 3 (condition: neutral stance vs. Romberg one vs. circular sway) x 2 (group: ASD vs. TD) repeated measures ANOVA. The three conditions were the within-subject factor and group was the between subject factor. In cases where Mauchly's test of sphericity was significant, results were interpreted using the Greenhouse-Geisser correction. In order to examine anticipatory and compensatory control processes during step initiation, we compared step variables between diagnostic groups using separate one-way ANOVAs. In the case of significant interactions ($p<0.05$), we ran post-hoc analyses using Bonferroni pairwise comparisons to correct for multiple comparisons. Cohen's d effect sizes also were calculated for group comparisons for all dependent variables (Cohen, 1988). Based on prior studies showing that both height and weight may be associated with postural control (Chiari, Rocchi, & Cappello, 2002; Hue et al., 2007) , we examined the relationship between height and weight and each postural control variable. We also examined outliers for height and weight and participants that were greater than three deviations from the mean were removed from the sample. Given the significant association between height and many of our primary dependent variables, it was included as a covariate.

To assess the relationships between postural control measures and symptom severity, we used Spearman correlations with ADOS-2 and ADI algorithm scores. Given that prior research

has shown that postural control improves over childhood and into adolescence, we also examined the relationships between postural control measures and age using Pearson correlations. Due to the high number of correlations for the clinical and demographic comparisons, only correlations with $|r| > 0.5$ were interpreted as significant.

Results

Postural Control

The ASD and TD groups did not differ in COP variability overall ($F(1,34)=1.137$, $p=0.294$) or as a function of stance (group x stance interaction: $F(1.038,34)=0.023$, $p=0.888$) or direction (group x direction interaction: $F(1,34)=1.032$, $p=0.317$). There also were no significant effects of direction ($F(1,34)=0.014$, $p=0.874$) or stance conditions ($F(1.038,34)=0.363$, $p=0.559$) (Table 2; Figure 1).

Individuals with ASD showed significantly greater COP trajectory length relative to TD controls ($F(1,34)=4.166$, $p=0.049$), however, COP trajectory length did not vary between groups as a function of stance (stance x group interaction: $F(1.010, 34.327)=2.704$, $p=0.109$). For COP trajectory length, there was a significant effect of stance condition ($F(1.010, 34.327)=4.592$, $p=0.039$). Participants showed greater COP trajectory length during the circular sway condition compared to the Romberg one condition ($F(1,34)=4.456$, $p=0.042$). (Table 2, Figure 2).

Participants with ASD showed greater MI during the neutral stance and Romberg one conditions and reduced MI during the circular sway condition compared to TD controls (group x stance interaction: $F(2,68)=5.03$, $p=0.009$) (Table 2, Figure 3). There was no effect of stance condition ($F(2,68)=1.473$, $p=0.236$).

Step Initiation

During the anticipatory phase of the step initiation task, individuals with ASD and controls did not differ on the maximum amplitude ($F(1,32)=0.002$, $p=0.966$) or the duration of their APAs ($F(1,32)=0.707$, $p=0.407$). During the compensatory phase, compared to controls, individuals with ASD showed greater mean step velocity ($F(1,34)=5.966$, $p=0.020$), as well as reduced step duration ($F(1,34)=10.486$, $p=0.003$). They also showed reduced ML range of motion compared to controls ($F(1,32)=3.704$, $p=0.063$), though this effect was marginal. Individuals with ASD did not differ from controls on step distance ($F(1,34)=0.884$, $p=0.354$).

Demographic and Clinical Correlations

For individuals with ASD, greater APA amplitude was associated with higher FSIQ ($r=0.587$, $p=0.013$) (Table 5, Figure 5). For TD individuals, none of the postural or step initiation measurements were associated with IQ (Table 4). For both the ASD and TD control groups, increased age was associated with decreased neutral stance COP_{ML} variability, neutral stance COP trajectory length, and Romberg one COP trajectory length, as well as increased Romberg one MI, circular sway MI, and step duration. For both groups, increased height was associated with increased Romberg one MI, circular sway MI, and step duration. In the TD group, increased height also was associated with increased lateral sway during step initiation. In the ASD group, increased height also was associated with decreased neutral stance COP_{ML} and Romberg one COP_{AP} variability as well as decreased COP trajectory length during both static conditions. Increased height also was associated with decreased step mean velocity. All correlation coefficients are provided in Tables 4 (TD) and 5 (ASD).

For individuals with ASD, greater neutral stance COP_{ML} variability ($r=0.546$, $p=0.023$) and decreased lateral sway during step initiation ($r=-0.548$, $p=0.023$) were associated with more severe ADOS-2 ratings of restricted repetitive behaviors (Figure 6). Greater APA amplitudes

during step initiation were associated with more severe clinically rated repetitive behaviors on the ADI-R, though this effect was marginal (r=0.517, p=0.058). Additionally, increased step duration (r=-0.534, p=0.049) and increased APA duration (r=0.507, p=0.064) were associated with more severe clinical ratings of communication abnormalities based on the ADI-R, though this effect was marginal. Decreased mean step velocity (r=-0.503, p=0.067) also was marginally associated with more severe ADI-R ratings of communication abnormalities (Table 6).

For TD individuals, COP_{AP} and COP_{ML} variability were positively correlated with each other and with COP trajectory length across the neutral and Romberg one stance conditions (Table 7). For the circular sway condition, increased MI was associated with decreased neutral stance variability in both directions as well as COP trajectory length. Increased circular sway MI was associated with decreased Romberg one COP_{ML} variability, COP trajectory length and Romberg one MI. Circular sway COP trajectory length also was associated with circular sway COP_{ML} variability. For the step initiation variables, increased APA duration was associated with increased circular sway COP_{ML} variability. Increased step duration was associated with decreased COP_{ML} variability for both neutral and Romberg one stances. Increased step velocity was associated with decreased step duration and increased Romberg one COP_{ML} variability. Increased maximum lateral sway during the step initiation was associated with increased circular sway MI and increased maximum APA.

For individuals with ASD, COP_{AP} and COP_{ML} variability were positively correlated with each other and with COP trajectory length across the neutral and Romberg one stance conditions (Table 8). For the neutral stance, increased MI was associated with increased circular sway MI and decreased circular sway COP trajectory length. For the step initiation variables, increased APA was associated with increased circular sway COP_{AP} variability. Increased step duration was

associated with decreased Romberg one COP_{ML} and COP trajectory length. Increased step velocity was associated with increased COP_{ML} variability and trajectory length for the neutral and Romberg one stances, increased COP_{AP} variability for the Romberg one stance, and decreased Romberg one MI. Increased step velocity also was associated with decreased step duration.

Discussion

This study examined postural control during both static and dynamic standing conditions in order to examine feedforward and feedback motor control processes in ASD. Five key findings are reported. First, individuals with ASD showed reduced MI during circular sway relative to controls suggesting a reduced ability to effectively coordinate distinct postural control mechanisms in order to maintain stability. Second, individuals with ASD showed increased COP trajectory length across stance conditions compared to controls suggesting that individuals with ASD are less stable during standing. Third, there were no significant differences between individuals with ASD and controls in the amplitude or duration of APAs suggesting that feedforward mechanisms involved in maintaining stability during walking are relatively unaffected in this sample of individuals with ASD. Fourth, during the step initiation task, children with ASD showed reduced lateral sway as well as shorter step durations and higher step velocities than controls suggesting that they had greater instability when stepping. Last, greater neutral stance COP_{ML} variability and decreased lateral sway when stepping were associated with more severe restricted and repetitive behaviors in ASD suggesting that deficits of postural control may contribute to or reflect mechanisms overlapping with core ASD symptoms. Taken together, these results suggest that individuals with ASD show impairments involving multiple

motor control mechanisms supporting postural stability, and that these impairments may contribute to motor issues seen in everyday activities such as walking and reaching.

Coordination of Distinct Motor Processes During Postural Control

We found that individuals with ASD showed reduced MI during circular sway suggesting an inability to coordinate distinct motor control processes during a task where fluid coordination is required. Typically, during more challenging stance conditions, healthy individuals show increased coordination of joint movements in order to maintain stability (Wang & Newell, 2014). During static stance conditions, a moderate level of shared dependency is seen in healthy individuals (Wang et al., 2016), with relative increases in shared dependency between joints seen in patients with neurodegenerative disorders (Rosenblum, Firsov, Kuuz, & Pompe, 1998). Additionally, In the current study, individuals with ASD showed increased MI during static stances indicating a reduced ability to modulate distinct processes and suggesting that they show an overreliance on the coordination between hip and ankle joints similar to patients with neurodegenerative disorders. These results also are consistent with previous findings that individuals with ASD show elevated MI during intentional sway along a single axis (Wang et al., 2016). During intentional sway along a single axis, optimal performance involves decoupling distinct control processes supporting postural adjustments in either the ML or AP directions. In contrast, the circular sway condition studied here elicits greater MI in order to support greater fluidity of non-linear movements. Individuals with ASD failed to adapt to these specific task demands and showed lower MI relative controls indicating an overall reduced ability to flexibly coordinate or decompose distinct control processes according to different postural tasks. The ability to flexibly modulate the amount of shared coordination between hip and ankle joints is necessary in order to maintain stability across changing environmental and postural demands.

When standing still or during a single direction sway, the increased MI seen in individuals with ASD may suggest a compensatory process in which they increase their body sway in multiple directions in order to decrease the likelihood of losing balance (Wang et al., 2016). Alternatively, the decreased MI during the dynamic circular sway condition may reflect reduced adjustments used during the more complex dynamic movements which could be due to greater rigidity or reduced central coordination of distinct movement processes.

Postural Stability Across Stance Conditions

Our finding of increased COP trajectory length in individuals with ASD is consistent with prior work showing greater trajectory length and COP variability in patients (Fournier et al., 2010; Wang et al., 2016). Greater COP trajectory suggests that individuals with ASD show more movement when attempting to remain still. In contrast to our hypothesis and prior work, we did not find any difference in COP variability between individuals with ASD and controls. Given the significant difference between groups for COP trajectory length and the medium effect size difference between groups for COP variability during static stances, the null findings for COP variability reported here may simply reflect a lack of power. Another possible explanation for this finding is that only individuals with ASD and average or above average IQs were included in the present study. Previous studies have shown that individuals with ASD and lower cognitive abilities show reduced postural stability relative to those with higher IQs (Minshew et al., 2004; Travers, Mason, Gruben, Dean, & McLaughlin, 2018). We also found that increased age was associated with reduced COP variability in ASD. Given that our sample was matched on age, this may suggest that the ability to control postural variability in individuals with ASD reaches the same levels as controls during later childhood/adolescence.

Methodological factors also may have contributed to differences between our results on COP variability and prior work. In the present study, we removed the first five and last five seconds of each COP recording for each stance, whereas prior work has focused on the most stable segment of the force trace (Wang et al., 2016). The current method of data analysis is more objective and reliable and may have better captured individual variability; however, more individualized methods such as identifying the most stable segment for each trial may help determine optimal performance for each individual and also may be more sensitive to performance deficits in patients whose "best" performance is more limited.

Feedforward Mechanisms Supporting Step Initiation

In contrast to our hypothesis, individuals with ASD did not show evidence of APA abnormalities consistent with feedforward control deficits during walking. Though deficits of anticipatory control have been documented during a bimanual load lifting task, this was the first known study to examine APAs during step initiation in individuals with ASD. Our results suggest that abnormalities during walking may not reflect the same deficits of anticipatory control seen in other planned motor movements. Another possible explanation is that not all individuals with ASD experience deficits in feedforward mechanisms. We found that increased FSIQ was associated with greater maximum APA amplitude. Given that we did not find impairments in APAs in this sample of individuals with ASD, this suggests that feedforward deficits may be specific to individuals with ASD with comorbid IDD. In contrast, we found that increased APA amplitude and duration were associated with more severe ASD symptoms including communication abnormalities and restricted and repetitive behaviors. This suggests that individuals with more severe ASD symptoms without IDD may demonstrate atypical APAs. It also is important to note that we examined APAs using COP measurements, which is an

indirect measure of APAs; the most sensitive measurement of APAs is EMG recording of the muscle (Belen'kii et al., 1967). Our findings that APAs are not affected in individuals with ASD may reflect reduced sensitivity of our measure.

In contrast to findings on feedforward postural control processes, we found evidence that feedback control of stepping is disrupted in individuals with ASD. When initiating a step and moving from stationary to walking, momentum in the ML direction is required to maintain stability. Specifically, when shifting from stationary with double leg support to initiate a step with a single leg of support, the body is required to make a lateral shift towards the stance leg (Fournier et al., 2010). In our study, individuals with ASD showed decreased lateral sway, consistent with a prior study of individuals with ASD (Fournier et al., 2010) suggesting that patients show a reduced lateral swing towards their stance leg resulting in the need for more active postural control in order to maintain stability. This may be due to greater rigidity when stepping and decreased balance similar to the pattern seen in aging individuals and individuals with Parkinson's disease (Hass et al., 2004; Hass, Waddell, Fleming, Juncos, & Gregor, 2005). This decreased lateral sway during step initiation seen in both individuals with ASD and individuals with Parkinson's disease suggests possible overlapping neural circuitry including dysfunction of basal ganglia circuits, consistent with prior structural and functional MRI studies of ASD (for review see Subramanian et al., 2017).

Associations Between Postural Control Deficits and ASD Severity

We found associations between more severe postural control deficits and restricted and repetitive behaviors in ASD. Specifically, decreased ML sway during step initiation and increased COP_{ML} variability during neutral stance were associated with more severe restricted and repetitive behaviors. This is consistent with previous studies showing that reduced postural

symmetry (Travers et al., 2013) and increased postural sway (Radonovich et al., 2013) in ASD are associated with more severe restricted and repetitive behaviors. In individuals with IDD, more severe motor control deficits are a predictor of more severe repetitive behaviors (Bodfish, Parker, Lewis, Sprague, & Newell, 2001). This finding suggests that shared neural mechanisms may be responsible for the development of both motor control impairments and restricted and repetitive behaviors in ASD, or that one of these deficits may cause the other. The basal ganglia has been implicated in repetitive behaviors in both mouse models of ASD (Lewis, Tanimura, Lee, & Bodfish, 2007) and in patients (Langen, Durston, Kas, van Engeland, & Staal, 2011). The basal ganglia also plays a key role in learning and completing complex motor movements (Graybiel, 2008; Yin & Knowlton, 2006) suggesting that alterations of basal ganglia development and its cortical targets may impact both basic motor control and more complex behavioral flexibility abilities in ASD. Further studies should examine possible mechanisms underlying the relationship between basic motor deficits and repetitive behavior issues in ASD.

We also found that increased step duration and decreased step velocity as well as increased APA duration were associated with more severe communication abnormalities. This is in contrast to our finding that individuals with ASD show increased step velocity and decreased step duration. However, these findings should be interpreted in the context of prior studies showing that patients with Parkinson's disease demonstrate reduced gait velocity. This reduced gait velocity is associated with more severe degeneration of basal ganglia nuclei (Hausdorff, Cudkowicz, Firtion, Wei, & Goldberger, 1998). In the context of ASD, communication abnormalities include behaviors such as repetitive speech and language, which is associated with disrupted corticostriatal circuit function (Langen et al., 2011). Our results indicate that individuals with ASD and more severe communication symptoms demonstrate similar gait

instability patterns to individuals with Parkinson's disease and may suggest shared neurophysiological alterations involving basal ganglia networks.

Brain Processes of Postural Control

Motor control is supported at least partially by basal ganglia and cerebellar circuits (Subramanian et al., 2017). The basal ganglia and cerebellum are involved in feedback loops necessary for sensorimotor accuracy and motor learning (Hikosaka, Nakamura, Sakai, & Nakahara, 2002) and for goal directed motor behaviors (Subramanian et al., 2017). Additionally, cerebellar circuits involving vermis and intermediate zones are utilized in the control of balance and walking (Apps & Garwicz, 2005). Spinocerebellar inputs provide proprioceptive feedback information that is integrated with other sensory information to maintain balance (Mosconi et al., 2015). For example, patients with cerebellar lesions show postural control deficits similar to those seen in individuals with ASD including increased AP sway (Diener, Ackermann, Dichgans, & Guschlbauer, 1985). Additionally, the cerebellum plays a key role in the coordination of movements including the ability to make immediate corrections to ongoing motor behavior (Baev et al., 2002). The lack of coordination between hip and ankle joints during the circular sway condition suggests possible cerebellar dysfunction similar to the lack of multiple joint coordination during gait seen in patients with cerebellar lesions (Palliyath, Hallett, Thomas, & Lebiedowska, 1998). The role of the basal ganglia in motor control also is demonstrated by neuromotor diseases that selectively affect basal ganglia circuits, including Parkinson's and Huntington's diseases. In these diseases, gait variability, another measure of gait instability, is associated with disease severity implicating the basal ganglia in the control of gait (Hausdorff et al., 1998). Additionally, stride height and length are associated with putamen and

nucleus accumbens volumes suggesting that the basal ganglia may play a direct role in gait control (McGough et al., 2018).

The overlap between postural control deficits and ASD symptom severity, specifically increased restricted and repetitive behaviors, suggests that overlapping neural systems may be responsible for both motor control deficits and ASD symptoms (Radonovich et al., 2013). Regions of the basal ganglia have been implicated in studies of animal models as well as human structural and functional MRI studies of disorders involving repetitive behaviors, including ASD, Obsessive Compulsive Disorder (OCD), and Parkinson's Disease (Langen et al., 2011). MRI studies of individuals with ASD have shown an association between repetitive behaviors and striatal volumes (Hollander et al., 2005; Langen et al., 2011; Rojas et al., 2006; Sears et al., 1999)). Patients with Parkinson's Disease, a neurogenerative condition characterized by basal ganglia defects, also show motor disturbances involving both repetitive and reduced movements similar to individuals with ASD. Studies of Parkinson's disease suggest that increased perseverative behavior is related to disrupted interaction between striatum and the frontal cortex (Langen et al., 2011). In the context of these studies, our findings on patients with ASD suggest that neurodevelopmental disruptions involving basal ganglia-cortical communication may contribute to both repetitive behaviors and less controlled postural movements.

Limitations and Future Directions

This study presents new evidence for multiple distinct forms of postural control deficits in ASD. When interpreting these findings, multiple study limitations should be considered. First, this was a small sample study conducted across a relatively wide age range (6-19 years). Larger developmental studies are warranted to more clearly determine growth rates and patterns of postural deficit in ASD across the lifespan. Second, we excluded participants with IDD, though

data from our study and others suggests that postural control may be more severely impacted in individuals with comorbid IDD. Finally, comparisons of APAs and motor variability should be examined across distinct behaviors to determine the specificity of the pattern of deficit documented here to postural control systems.

Conclusions

Overall, our findings identify deficits of joint coordination and sensory feedback processes during postural control in ASD. Given the amount of impairment that motor deficits can cause, these findings highlights potential targets for intervention that can be examined using a precise and easily quantified measure. The relationships between these postural control deficits and core symptoms of ASD suggests that their study may provide important insights into neurobiological mechanisms contributing to both motor and core clinical issues in patients.

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Appendix

Table 1

Participant characteristics

	ASD (n=17)	**TD (n=20)**
Age (years)	13.67 (3.13)	12.48 (4.17)
Height (cm)	161.80 (15.88)*	149.46 (17.61)*
Weight (kg)	58.82 (15.41)*	45.81 (18.41)*
Leg Length (cm)	85.31 (9.13)	77.39 (15.34)
BMI	22.18 (4.04)	19.71 (4.13)
% Male	88%	80%
FSIQ	97.76 (17.27)	108.47 (14.53)
PIQ	99.76 (16.41)	104.32 (11.57)
VIQ	96.18 (17.46)*	110.58 (15.34)*

Note. Data are reported as mean and standard deviation in parentheses. FSIQ = full-scale IQ; PIQ = performance IQ; VIQ = verbal IQ

*$p < 0.05$

Table 2

Estimated means and effect sizes for postural control measurements

	Estimated Mean (SE)		
	ASD (n=17)	**TD (n=20)**	Effect size
Neutral Stance			
COP_{ML}	0.436 (0.058)	0.261 (0.054)	0.73
COP_{AP}	0.606 (0.065)	0.474 (0.059)	0.50
MI	0.611 (0.044)	0.553 (0.04)	0.32
COP Length	34.645 (3.912)	24.11 (3.588)	0.65
Romberg 1			
COP_{ML}	0.767 (0.061)	0.621 (0.056)	0.58
COP_{AP}	0.826 (0.096)	0.582 (0.088)	0.62
MI	0.66 (0.024)	0.617 (0.022)	0.44
COP Length	44.36 (4.009)	36.234 (3.676)	0.49
Circular Sway			
COP_{ML}	7.355 (0.480)	6.889 (0.440)	0.24
COP_{AP}	3.804 (0.235)	4.034 (0.215)	0.24
MI	0.572 (0.035)	0.691 (0.032)	0.83
COP Length	454.836 (40.946)	351.813 (37.55)	0.61

Note. COP_{ML} = COP variability in the ML direction; COP_{AP} = COP variability in the AP direction; MI = mutual information; COP Length = COP trajectory length. Covariates appearing in the model are evaluated at the following value: Height = 155.130 cm. Effect size was calculated using Cohen's d. Data are reported as estimated mean and standard error in parentheses.

Table 3

Estimated means and effect sizes for step initiation measurements

	Estimated Mean (SE)		Effect size
	ASD (n=17)	**TD (n=20)**	
Step maximum APA	4.567 (0.492)	4.537 (0.478)	0.01
Step duration APA	0.491 (0.047)	0.434 (0.046)	0.29
Step duration	0.177 (0.014)	0.24 (0.013)	1.11
Step distance	32.108 (1.084)	30.68 (0.994)	0.32
Step mean velocity	194.728 (14.648)	144.618 (13.433)	0.83
Step maximum ML	10.824 (0.796)	13.019 (0.772)	0.67

Note. Covariates appearing in the model are evaluated at the following value: Height = 155.130 cm. Effect size was calculated using Cohen's d. Data are reported as estimated mean and standard error in parentheses.

Table 4

Correlations coefficients between dependent and demographic variables for TD Controls

	Age	**FSIQ**	**Height**	**Weight**
Neutral COP_{ML}	-0.619**	0.127	-.454	-0.438
Neutral COP_{AP}	-0.435	0.019	-0.298	-0.244
Neutral MI	-0.089	-0.059	-0.076	0.06
Neutral Length	-0.588**	-0.004	-0.470	-0.438
ROM1 COP_{ML}	-.506*	0.125	-0.418	-0.42
ROM1 COP_{AP}	-0.345	0.289	-0.183	-0.156
ROM1 MI	0.633**	0.198	0.519*	0.389
ROM1 Length	-0.565**	0.153	-0.404	-0.383
Circle COP_{ML}	0.43	-0.094	0.453	.456
Circle COP_{AP}	0.464	0.046	0.359	0.168
Circle MI	0.515*	-0.015	0.517*	0.386
Circle Length	0.058	-0.003	-0.014	-0.013
Step APA	0.106	-0.010	0.205	-0.01
Step Duration APA	0.267	0.138	0.45	0.412
Step Duration	0.729**	0.218	0.752**	0.820**
Step Distance	0.359	0.152	0.426	0.413
Step Mean Velocity	-0.419	-0.067	-0.433	-0.431
Step ML	0.436	0.321	0.683**	0.478

Note. Neutral = Neutral stance condition; ROM1 = Romberg one condition; Circle = Circular sway condition; COP_{ML} = COP SD in the ML direction; COP_{AP} = COP SD in the AP direction; MI = mutual information; Step APA = Step initiation APA amplitude; Step ML = Step initiation maximum lateral sway.

*$p < 0.05$ level;**$p < 0.01$ level

Table 5

Correlations coefficients between dependent and demographic variables for ASD

ASD	**Age**	**FSIQ**	**Height**	**Weight**
Neutral COP_{ML}	-0.602*	-0.118	-0.565*	-0.314
Neutral COP_{AP}	-0.324	-0.233	-0.332	-0.012
Neutral MI	0.397	-0.140	0.411	0.492
Neutral Length	-0.710**	-0.167	-0.675**	-0.436
ROM1 COP_{ML}	-0.497	0.332	-0.484	-0.281
ROM1 COP_{AP}	-0.629**	-0.101	-0.623**	-0.291
ROM1 MI	0.502*	-0.148	0.584*	0.478
ROM1 Length	-0.688**	0.157	-0.690**	-0.387
Circle COP_{ML}	0.097	0.115	0.234	0.077
Circle COP_{AP}	0.431	0.407	0.444	0.078
Circle MI	0.584*	-0.066	0.599*	0.571*
Circle Length	-0.428	-0.043	-0.353	-0.378
Step APA	0.270	0.587*	0.277	-0.103
Step Duration APA	0.006	-0.099	-0.047	0.351
Step Duration	0.586*	-0.160	0.645**	0.639**
Step Distance	0.475	0.060	0.462	0.551*
Step Mean Velocity	-0.605*	0.093	-0.670**	-0.505*
Step ML	0.056	0.115	0.057	-0.107

Note. Neutral = Neutral stance condition; ROM1 = Romberg one condition; Circle = Circular sway condition; COP_{ML} = COP SD in the ML direction; COP_{AP} = COP SD in the AP direction; MI = mutual information; Step APA = Step initiation APA amplitude; Step ML = Step initiation maximum lateral sway.

*$p < 0.05$ level;**$p < 0.01$ level

Tables 6

Correlation coefficients between dependent variables and ASD clinical ratings

	ADI Social Communication	ADI Communication	ADI Repetitive Behavior	ADOS Repetitive Behavior	ADOS Severity Score
Neutral COP_{ML}	-0.029	-0.024	-0.009	0.546*	0.211
Neutral COP_{AP}	0.142	0.211	-0.058	0.165	0.138
Neutral MI	-0.182	0.324	-0.165	0.041	-0.047
ROM1 COP_{ML}	-0.480	-0.040	-0.051	0.040	0.004
ROM1 COP_{AP}	-0.178	0.093	-0.116	0.213	0.047
ROM1 MI	0.042	0.297	-0.083	-0.240	0.071
Circle COP_{ML}	0.469	0.171	-0.056	0.283	0.097
Circle COP_{AP}	0.196	-0.308	0.156	-0.251	0.037
Circle MI	0.411	0.490	-0.025	0.093	0.105
Step APA	-0.084	-0.180	0.517	-0.441	0.067
Step Duration APA	0.239	0.507	-0.290	0.349	0.383
Step Duration	0.393	0.534*	-0.090	-0.041	0.301
Step Distance	0.264	0.390	0.069	0.125	-0.021
Step Mean Velocity	-0.393	-0.503	-0.100	0.177	-0.381
Step ML	-0.156	-0.069	0.111	-0.548*	-0.473

Note. Neutral = Neutral stance condition; ROM1 = Romberg one condition; Circle = Circular sway condition; COP_{ML} = COP SD in the ML direction; COP_{AP} = COP SD in the AP direction; MI = mutual information; Step APA = Step initiation APA amplitude; Step ML = Step initiation maximum lateral sway.

*$p < 0.05$

Table 7

Correlation matrix of postural control and step initiation variables for TD controls

	Neut COP_{ML}	Neut COP_{AP}	Neut MI	Neut Len	ROM1 COP_{ML}	ROM1 AP	ROM1 MI	ROM1 Len	Circ COP_{ML}	Circ COP_{AP}	Circ MI	Circ Len	Step APA	Dur APA	Step Dur	Step Vel	Step ML
Neut COP_{ML}	1.00																
Neut COP_{AP}	.891**	1.00															
Neut MI	.404	.287	1.00														
Neut Len	.857**	.916**	.017	1.00													
ROM1 COP_{ML}	.822**	.656**	.622**	.598**	1.00												
ROM1 COP_{AP}	.839**	.734**	.600**	.556*	.750**	1.00											
ROM1 MI	-.498	-.487	.324	-.656**	-.140	-.220	1.00										
ROM1 Len	.973**	.878**	.384	.824**	.788**	.896**	-.509*	1.00									
Circ COP_{ML}	-.154	.169	-.261	.066	-.327	-.074	-.046	-.103	1.00								
Circ COP_{AP}	-.412	-.291	-.304	-.310	-.372	-.215	.350	-.331	.394	1.00							
Circ MI	-.640**	-.541*	-.157	-.605**	-.636**	-.404	.519*	-.576**	.136	.495	1.00						
Circ Len	.121	.268	-.295	.282	.059	.099	-.358	.154	.515*	.289	-.459	1.00					
Step APA	-.224	-.228	-.351	-.196	-.248	-.050	-.131	-.109	-.007	.176	.401	-.126	1.00				
Step APA Dur	.164	.475	-.281	.298	-.115	.187	-.252	.211	.634**	-.056	-.110	.414	.011	1.00			
Step Dur	-.523*	-.344	-.261	-.444	-.661**	-.371	.307	-.470	.464	.152	.496	-.116	.001	.447	1.00		
Step Vel	.448	.276	.476	.316	.798**	.365	-.019	.388	-.300	-.175	-.428	.076	-.149	-.431	-.744**	1.00	
Step ML	-.494	-.400	-.230	-.477*	-.439	-.212	.302	-.426	.329	.206	.599**	-.132	.601**	.170	.425	-.235	1.00

Note. Neut = Neutral stance condition; ROM1 = Romberg one condition; Circ = Circular sway condition; COP_{ML} = COP SD in the ML direction; COP_{AP} = COP SD in the AP direction; MI = mutual information; Len = COP trajectory length; Step APA = Step initiation APA amplitude; APA Dur = Step initiation APA duration; Step Dur = Step Initiation duration; Step Vel = Step initiation mean velocity; Step ML = Step initiation maximum lateral sway; *p<0.05 level; **p<0.01.

Table 8

Correlation matrix of postural control and step initiation variables for ASD individuals

	Neut COP_{ML}	Neut COP_{AP}	Neut MI	Neut Len	ROM1 COP_{ML}	ROM1 COP_{AP}	ROM1 MI	ROM1 Len	Circ COP_{ML}	Circ COP_{AP}	Circ MI	Circ Len	Step APA	Dur APA	Step Dur	Step Vel	Step ML
Neut COP_{ML}	1.00																
Neut COP_{AP}	.725**	1.00															
Neut MI	-.233	-.308	1.00														
Neut Len	.891**	.816**	-.480	1.00													
ROM1 COP_{ML}	.514*	.292	.011	.417	1.00												
ROM1 COP_{AP}	.798**	.570*	-.031	.763**	.712**	1.00											
ROM1 MI	-.494	-.116	.294	-.380	-.213	-.178	1.00										
ROM1 Len	.796**	.470	-.099	.678**	.880**	.862**	-.498	1.00									
Circ COP_{ML}	-.094	.005	-.323	.094	-.220	-.280	.004	-.233	1.00								
Circ COP_{AP}	-.429	-.280	-.329	-.263	-.287	-.422	.339	-.492	.441	1.00							
Circ MI	-.427	-.320	.541*	-.418	-.302	-.268	.394	-.445	.365	.354	1.00						
Circ Len	.153	.177	-.655**	.316	.010	-.130	-.250	.072	.401	-.044	-.574*	1.00					
Step APA	-.309	-.369	-.197	-.358	-.015	-.341	.136	-.178	.069	.606**	-.161	.115	1.00				
Step APA Dur	.359	.341	.080	.174	.362	.139	-.242	.387	-.013	-.449	-.238	.359	-.139	1.00			
Step Dur	-.320	-.108	.441	-.414	-.520*	-.339	.476	-.506*	.092	-.042	.483	-.315	-.110	-.068	1.00		
Step Vel	.526*	.233	-.229	.561*	.650**	.571*	-.587*	.724**	.064	-.158	-.257	.204	-.079	.196	-.801**	1.00	
Step ML	-.083	.021	.098	.017	.008	.104	.175	-.034	.018	.358	.095	-.295	.334	-.440	-.258	.219	1.00

Note. Neut = Neutral stance condition; ROM1 = Romberg one condition; Circ = Circular sway condition; COP_{ML} = COP SD in the ML direction; COP_{AP} = COP SD in the AP direction; MI = mutual information; Len = COP trajectory length; Step APA = Step initiation APA amplitude; APA Dur = Step initiation APA duration; Step Dur = Step Initiation duration; Step Vel = Step initiation mean velocity; Step ML=Step initiation maximum lateral sway; *p<0.05 level; **p<0.01

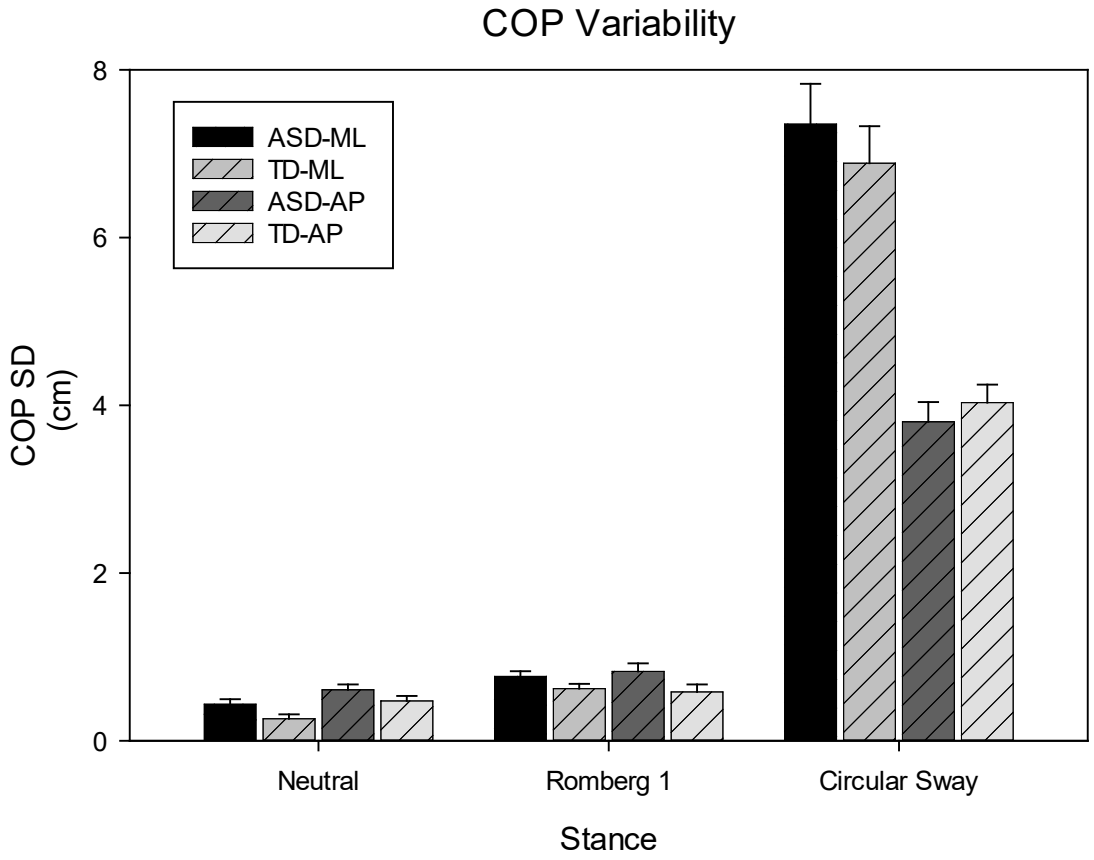


Figure 1. Covariates appearing in the model are evaluated at the following value: Height = 155.130 cm. Individuals with ASD did not differ from TD controls in COP variability. However, medium effect size differences were seen between groups across AP and ML directions for Neutral and Romberg one stance conditions.

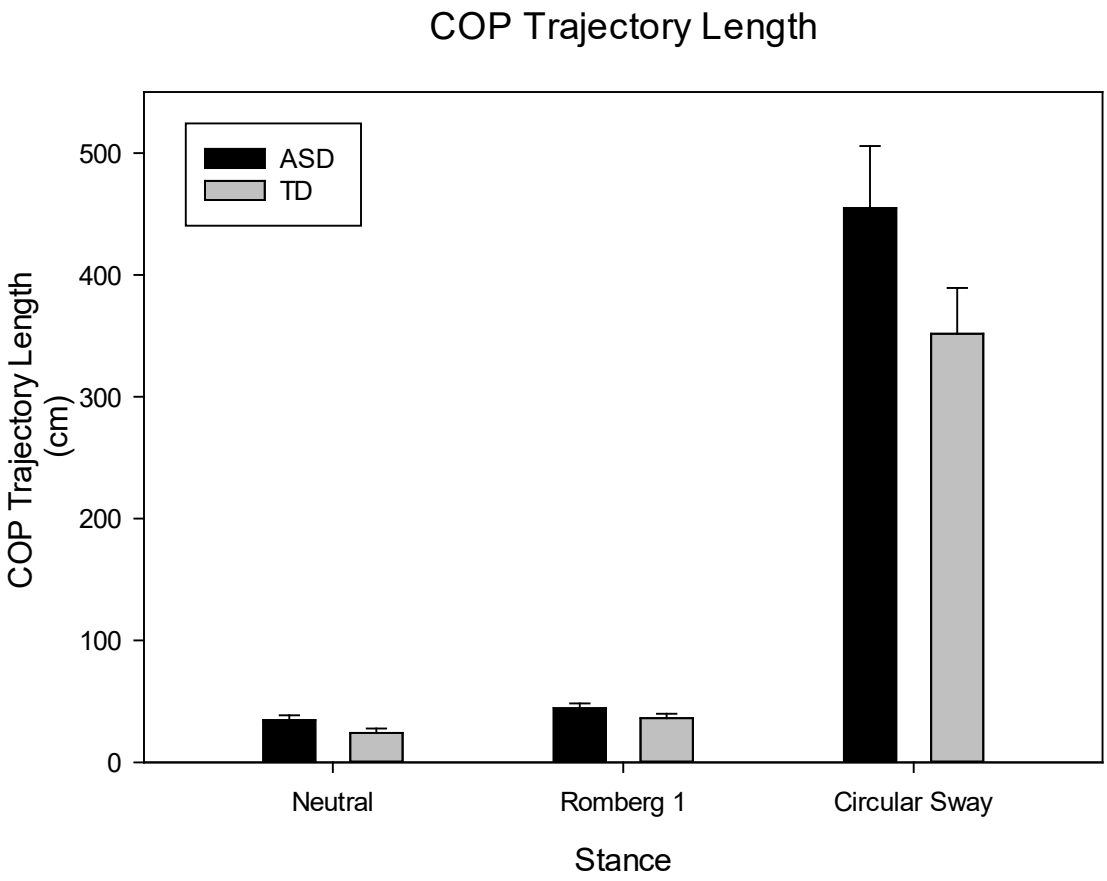


Figure 2. Covariates appearing in the model are evaluated at the following value: Height = 155.130 cm. Individuals with ASD showed significantly greater COP trajectory length than TD controls, ($p < 0.05$).

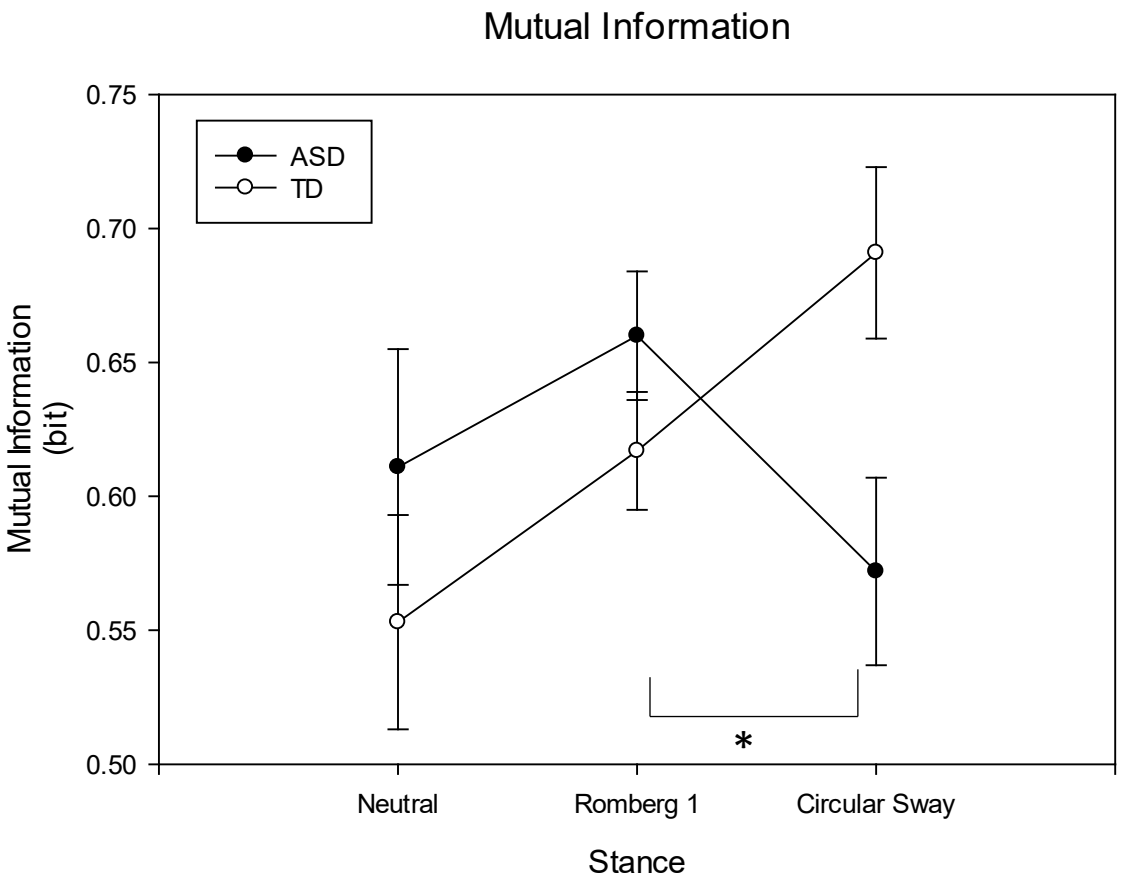


Figure 3. Covariates appearing in the model are evaluated at the following value: Height = 155.130 cm. There was a significant Group X Stance interaction with individuals with ASD showing decreased MI during the circular sway condition relative to TD controls.
*$p < 0.05$.

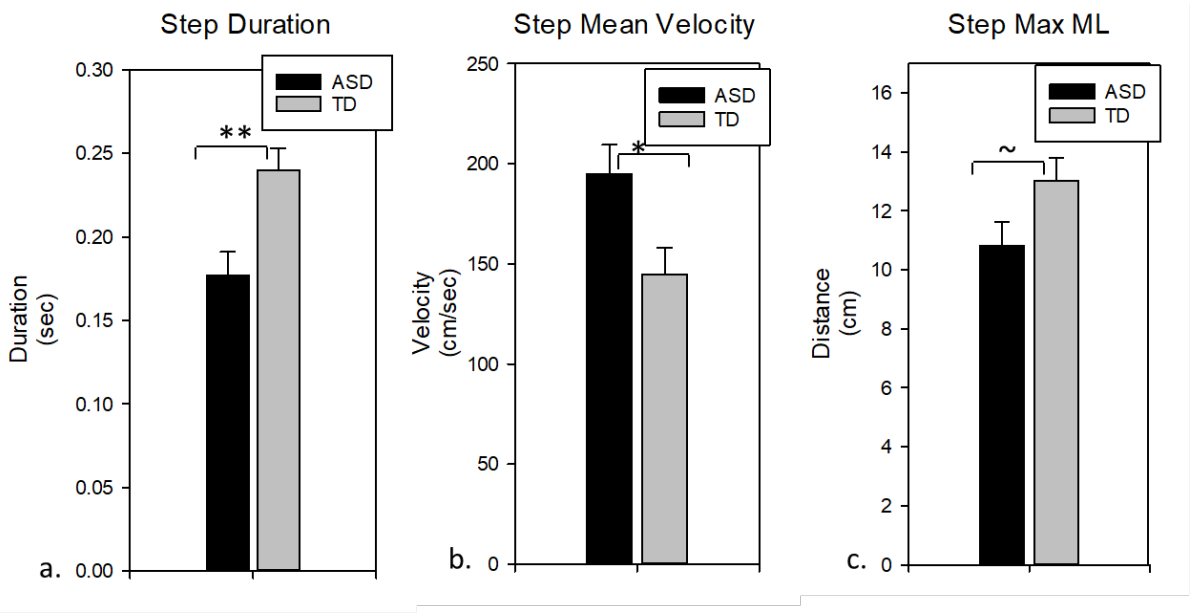


Figure 4. a. TD individuals showed increased step duration compared to individuals with ASD. b. Individuals with ASD showed increased mean step velocity compared to TD individuals. c. Individuals with ASD showed decreased maximum lateral sway during step initiation compared to TD individuals.
*$p < 0.05$; **$p < 0.01$; ~ $p=0.063$

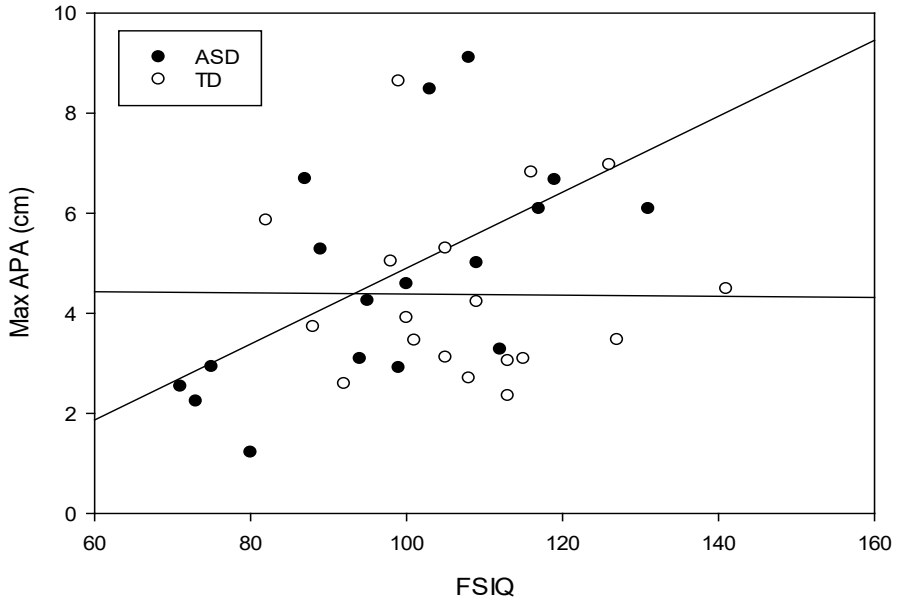
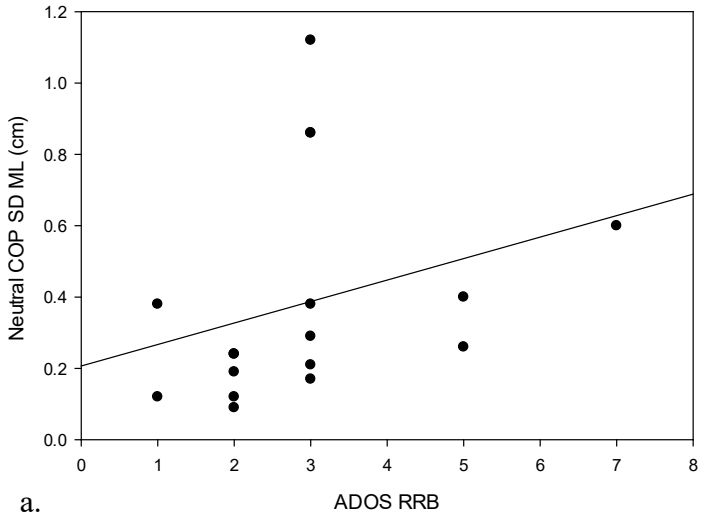
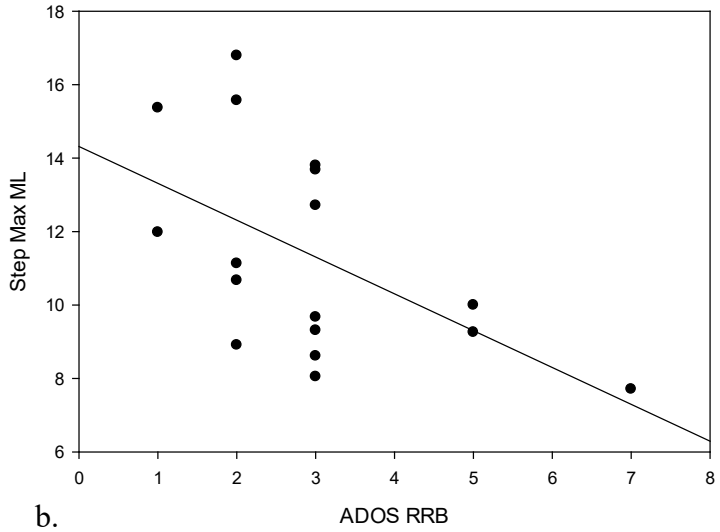


Figure 5. FSIQ = Full Scale IQ. Increased FSIQ it associated with decreased maximum APA amplitude in individuals with ASD (r = 0.587) but not TD controls (r = -0.010).



a.



b.

Figure 6. RRB = Restricted and repetitive behaviors. a. Increased neutral stance COP_{ML} variability was associated with more severe repetitive behaviors in ASD (r = 0.546). b. Decreased step initiation lateral sway was associated with more severe repetitive behaviors in ASD (r = -0.548).