



Coupling between visual information and body sway in adults with Down syndrome



Matheus Machado Gomes^{a,*}, Renato Moraes^a, José Angelo Barela^{b,c}

^a School of Physical Education and Sport of Ribeirão Preto, University of São Paulo, EEFERP-USP, São Paulo, Brazil

^b Institute of Physical Activity and Sport Sciences and Graduate Program in Human Movement Sciences, Cruzeiro do Sul University, São Paulo, Brazil

^c Institute of Bioscience, São Paulo State University, Rio Claro, Brazil

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ABSTRACT

Background: Prior studies suggest that infants with Down syndrome (DS) need more experience to acquire a similar relationship between visual information and body sway than infants without DS. However, it is unclear how adults with DS deal with visual information to control posture.

Aim: To examine the coupling between visual information and body sway in adults with DS.

Methods: Twenty adults with DS (25.8 ± 4.0 years) and twenty age- and sex-matched controls (25.6 ± 4.0 years) stood upright inside a “moving room” in two experimental conditions: continuous (room oscillated continuously at 0.1, 0.2, and 0.5 Hz) and discrete (room moved forward or backward for a brief moment). Tridimensional body sway and moving room displacement data were registered.

Results: Individuals with DS coupled their body sway to the imposed visual stimulus, but showed higher position variability at frequencies other than the frequency of room movement (0.48 cm) and lower coherence (0.80) than controls (0.40 cm and 0.90, respectively).

Conclusions: Adults with DS were able to couple to the visual cue, but with differences in terms of the scaling of postural responses to spatial parameters of the visual stimulus.

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What this paper adds?

This study clearly showed that individuals with DS were able to obtain visual cues from the movement of the room, and to produce correspondent body sway. However, they seem to experience problems in obtaining visual cues that would provide accurate information about body position and velocity in conditions in which the visual cues are not highlighted. When visual cues were manipulated and visual information is enhanced, for example when the room moved, individuals with Down syndrome properly coupled their body sway to this information. These findings not only contribute to understanding about the functioning of the postural control system of individuals with Down syndrome, but also draw attention to future strategies that could be implemented to enhance visual cues, providing more reliable information about body sway in this population.

* Corresponding author at: EEFERP – USP, Avenida Bandeirantes, 3900, Ribeirão Preto, SP, CEP: 14049-907, Brazil.
E-mail addresses: mmgomes@usp.br, prof.matheus.gomes@gmail.com (M.M. Gomes).

1. Introduction

Individuals with Down syndrome (DS) show several differences in their motor performance as compared to controls (Cabeza-Ruiz et al., 2011; Cimolin et al., 2011; Galli et al., 2008; Rigoldi, Galli, Mainardi, Crivellini, & Albertini, 2011; Spanò et al., 1999; Viergege, Schulze-Rava, & Wessel, 1996; Webber, Virji-Babul, Edwards, & Lesperance, 2004). In general, movements of individuals with DS are slow (Almeida et al., 2000; Davis & Kelso, 1982; Shumway-Cook & Woollacott, 1985), with excessive muscle co-activation (Almeida, Corcos, & Latash, 1994; Almeida et al., 2000; Carvalho & Almeida, 2009; Latash, Kang, & Patterson, 2002), characterized by muscle weakness (Angelopoulou, Tsimaras, Christoulas, Kokaridas, & Mandroukas, 1999; Croce, Pitetti, Horvat, & Miller, 1996), and coordination problems (Ringebach, Chua, Maraj, Kao, & Weeks, 2002; Spanò et al., 1999). Sensory deficits (Chen & Fang, 2005; Cole, Abbs, & Turner, 1988; Hodges, Cunningham, Lyons, Kerr, & Elliott, 1995; Virji-Babul & Brown, 2004), muscle hypotonia (Kokubun et al., 1997; Rigoldi et al., 2011) and atypical cerebellar volume (Aylward et al., 1997) has been suggested as the key causes for these motor differences.

Despite these well-known differences, some studies have shown that in specific conditions the motor performance of individuals with DS is similar to controls (Almeida et al., 2000; Gomes & Barela, 2007; Latash, 2000; Villarroja et al., 2012; Vuillerme, Marin, & Debú, 2001). Latash, Almeida, and Corcos (1993) Latash, (2000) have suggested that instead of problems in the motor control system, motor performance differences of individuals with DS are due to impairment in the decision making process. Likewise, our earlier results (Gomes & Barela, 2007) did not indicate any functional changes in the postural control system of adults with DS, as they were able to use the sensory information similarly to controls during maintenance of upright stance.

The performance of any motor task depends on the capacity of the motor control system to integrate and “weight” accurately sensory cues coming from multiple sources (Oie, Kiemel, & Jeka, 2002) and to use the information furnished by these cues to produce appropriate muscle activity. Moreover, acquisition and refinement of motor skills have also been suggested to occur as this intricate relationship between sensory information and motor activity is mastered (Barela, 1999; Barela, Jeka, & Clark, 1999; Barela, Jeka, & Clark, 2003). Unfortunately, very little is known regarding how individuals with DS use sensory information to modulate motor activity, and most of the available knowledge relates to infants (Butterworth & Cicchetti, 1978; Polastri & Barela, 2005) and children (Wade, Emmerick & Kernozek, 2000), and many important issues still remain unclear.

A clever strategy to examine how sensory information is related to motor activity is to manipulate cues from a sensory channel, while maintaining the cues from the other channels unaltered, and to observe changes in motor activity due to the sensory manipulation (Schöner, Dijkstra, & Jeka, 1998). A few studies have employed this strategy, using a moving room to examine how visual information is integrated into motor action in individuals with DS (Butterworth & Cicchetti, 1978; Polastri & Barela, 2005; Wade et al., 2000). Polastri and Barela (2005), for instance, observed that infants with DS need more time and experience in order to acquire a similar relationship between visual information and body sway as infants without DS. Moreover, the delay to achieve the developmental milestones exhibited by infants with DS could also be due to their need for more experience and exposition to sensory and motor experiences. However, it is unclear if adults with DS still differ in the use of visual cues to control postural sway when compared to neurologically normal adults. Therefore, the goal of this study was to examine the use of visual information to control postural sway and to examine the coupling between visual information and body sway in adults with DS.

2. Methods

2.1. Participants

Twenty adults with Down syndrome (13 males and 7 females, mean age: 25.8 ± 4.0 years) and twenty age- and sex-matched controls (mean age: 25.6 ± 4.0 years) participated in this study. Participants with DS were recruited from special schools, whereas participants in the control group were university employees and undergraduate and graduate students. Participants in the control group and parents of the participants with DS reported no known musculoskeletal injuries or neurological disorders (besides DS) that might have affected their ability to maintain the upright stance. All participants had normal or corrected-to-normal vision. Prior to the study, participants or parents, in the case of participants with DS, gave informed consent for participating in the study. The study was conducted in accordance with the Declaration of Helsinki and approved Institutional Ethical Review Board.

2.2. Procedures

Each participant came to the Movement Studies Laboratory at São Paulo State University where the experimental procedure was outlined to participants and body mass and height measures were obtained. The experimental task consisted of maintaining upright position inside a “moving room”. The moving room consisted of three walls and a ceiling (2.1 m long \times 2.1 m wide \times 2.1 m height) mounted on four wheels that slid on rails, allowing back and forth movement of the room, independent of the floor. The walls were painted in white and black, creating a pattern of 22 cm wide vertical white and black stripes in order to increase visual contrast. A 800 lumens compact fluorescent light bulb was used to maintain consistent light conditions throughout the data collection process. The room movement was produced by a servo-mechanism system con-

sisting of a controller (Compumotor – Mod. APEX6151), a controlled stepper motor (Compumotor – Mod. N0992GR0NMSN), and an electrical cylinder (Compumotor – Mod. EC3-X3xxn-1004A-MS1.MT1 M). A custom software (Compumotor – Motion Architect for Windows) initiated and controlled room movement.

The movement of the room was controlled in order to produce two experimental conditions: continuous and discrete movements. In the continuous condition, the room oscillated continuously at frequencies of 0.1, 0.2, and 0.5 Hz, amplitudes of 1.0, 0.5, and 0.2 cm, respectively, and peak-to-peak velocity of 0.6 cm/s, during 60 s. In the discrete condition, the room remained stationary, in the first 4 s, then moved forward or backward (away or approaching the participant, respectively), with an amplitude of 2.6 cm and mean velocity of 1.3 cm/s, during 2 s, and remained stationary again for the last 14 s, totaling 20 s.

Tridimensional body sway and moving room displacement data were obtained by an OPTOTRAK system (Digital Northern, Inc.), using infra-red emitting diodes (IREDs) placed on participants' back between their scapulae, and on the frontal wall of the room. Data were sampled with a frequency of 100 Hz.

In both conditions, participants stood inside the moving room at 1 m from the front wall. They were asked to assume a comfortable upright stance, barefooted, with their feet parallel at shoulder width, and to maintain this position, keeping as stable as possible, while looking at a target (white circle with 5 cm diameter) fixed on the front wall of the room at eye level.

Each participant first performed a trial, lasting 60 s, in which the room remained stationary and this was used as the baseline. Next, participants performed 9 trials in the continuous condition, organized in 3 blocks in which room frequency (0.1, 0.2, and 0.5 Hz) was randomly defined. Finally, participants performed 6 trials in the discrete condition, also organized in 3 blocks in which room direction (forward or backward) was randomly defined.

Participants had a brief rest interval between trials. In order to check if participants with DS kept looking at the target throughout the trial, they were videotaped (Panasonic camera – Model WV – CL350) from a frontal view. If they did not look to the target, the trial was repeated. This was the case for about 5% of the trials. In all trials, one experimenter was positioned close to the participants to assist them and to prevent any loss of balance. However, none of the participants needed any assistance.

2.3. Data analysis

Body sway and moving room displacement data were filtered using a second order Butterworth digital filter with a cut off frequency of 5 Hz. For the no room movement trial, mean sway amplitude was calculated. Initially, a first order polynomial was fitted and subtracted from all the trial values, along with the mean value, in order to eliminate any postural orientation adjustment not related to body sway throughout the trial. Then, the standard deviation was computed indicating the variance of body sway during the trial. Such procedures were employed for body sway in the anterior-posterior (AP) and medial-lateral (ML) directions.

Mean sway amplitude was also computed in those trials in which the room moved continuously, following the same procedures employed for the no room movement trial. However, because the room movement occurred in the AP direction and induced postural sway in this direction, mean sway amplitude was only computed for the AP direction. In addition, predominant frequency of body sway was obtained as the frequency with the highest spectral power among all the frequency of the spectrum.

The relationship between body sway and moving room, in the AP direction, was examined using the following variables: gain, phase, variability and coherence. Gain was calculated as the ratio between body sway amplitude and moving room amplitude spectrum at the driving frequency of the room (0.1, 0.2, and 0.5 Hz). Gain indicated the influence of the room movement on body sway, with values close to 1 indicating body sway with the same spectral amplitude of the moving room, and values below/above 1 indicating body sway with smaller/larger amplitude, respectively, than the moving room. Phase was calculated based on the temporal difference between the extremes of the position of the moving room and body sway cycles (Dijkstra, Schöner, & Gielen, 1994). Phase was used to examine the temporal relationship between room movement and body sway. Phase values close to zero indicate that the participant swayed together with the room, and positive/negative phase values indicate that the participant swayed ahead of/behind the room. Position variability represents the body sway amplitude at frequencies other than that of the driving stimulus of the room. To calculate the variability, the component of body sway due to room movement was removed by subtracting the sinusoid corresponding to the Fourier transform of the trajectory at the stimulus frequency (Barela et al., 2003). The variability was then defined as the root mean square of the residual postural sway trajectory. Low variability means that most of the sway amplitude is due to the driving stimulus frequency; conversely, high variability indicates that body sway also occurred at frequencies other than that of the driving stimulus. Coherence measured the strength of the relationship between body sway and room movement, that is, how strong body sway was coupled to the visual stimulus. Coherence was calculated as:

$$Coherence = \frac{|P_{yx}(\omega)|^2}{P_{xx}(\omega) P_{yy}(\omega)}$$

where $P_{xy}(\omega)$ is the cross-spectrum of two signals $x(t)$ and $y(t)$, moving room and body position, and $P_{xx}(\omega)$ and $P_{yy}(\omega)$ are the auto-spectra of $x(t)$ and $y(t)$, respectively, calculated at a specific frequency (ω). Coherence values close to 1 indicate

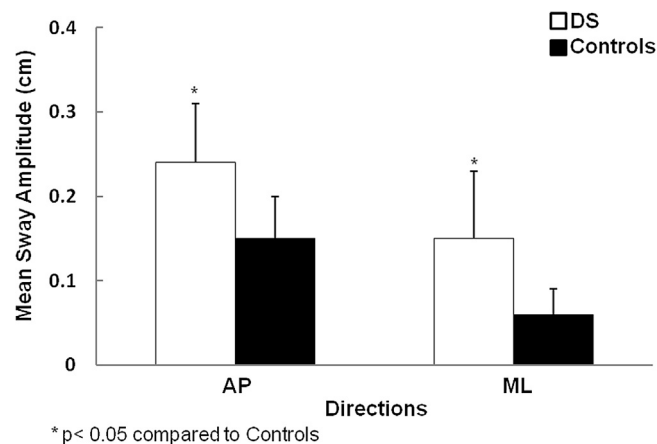


Fig. 1. Mean and standard deviation of the mean sway amplitude in the anterior-posterior (AP) and medial-lateral (ML) directions for participants from both groups: Down syndrome (DS) and Controls.

strong dependence between the signals and values close to zero indicate no dependence between the signals (Prioli, Cardozo, Freitas Junior, & Barela, 2006).

For trials in which the room moved discretely, the following dependent variables were obtained: body displacement; time to body sway reversal; and mean sway amplitude before and after the room movement. Body displacement was obtained as the absolute difference between the body position at the moment that the moving room began to move and the body position at the moment that the participant stopped and/or reversed the induced body sway due to the visual manipulation. Time to body sway reversal corresponded to the time elapsed between the beginning of the room movement and the body sway reversal. Finally, mean sway amplitude was obtained for the periods before and after the room movement as previously described for the baseline trial. In this case, mean sway amplitude before the room movement indicated postural control performance without any visual manipulation, whereas mean sway amplitude after the room movement indicated postural control performance to reestablish postural equilibrium after the visual disruption.

2.4. Statistical analysis

Analyses were performed after the respective statistical assumptions were fulfilled. In the trial with no room movement, postural control performance was compared between groups using a multivariate analysis of variance (MANOVA) with mean sway amplitude for both AP and ML directions as dependent variables.

In the continuous condition, two MANOVA and two ANOVA were carried out, with group (DS and Controls) and frequency of the room (0.1, 0.2, and 0.5 Hz) as factors, this last one treated as repeated measures. For the MANOVA, the dependent variables were: 1) mean sway amplitude and predominant frequency and, 2) gain and phase. The dependent variables for each ANOVA were variability and coherence.

In the discrete condition, two MANOVA were performed, with group (DS and Controls) and movement (forward and backward directions) as factors, this last one treated as repeated measures. The dependent variables for the MANOVA were: 1) body displacement and time to body sway reversal and, 2) mean sway amplitude before and after the room movement.

When necessary, follow up univariate analyses and Tukey (HSD) post hoc tests were employed. In all comparisons the alpha level was set at 0.05 and analyses were performed using SPSS software.

3. Results

3.1. Postural control performance with no visual manipulation

MANOVA revealed a significant effect for group, Wilks' Lambda = 0.54, $F(2,37) = 15.52$, $p = 0.001$. Univariate analyses exhibited an effect for mean sway amplitude in both AP, $F(1,38) = 24.59$, $p = 0.0001$, and ML directions, $F(1,38) = 20.60$, $p = 0.0001$. Participants with DS displayed larger body sway amplitude than the controls in both AP (0.24 ± 0.07 cm; 0.15 ± 0.05 cm, respectively) and ML (0.15 ± 0.08 cm; 0.06 ± 0.03 cm, respectively) directions (Fig. 1).

3.2. Continuous visual manipulation

Continuous visual manipulation from the moving room induced body sway in both participants with DS and controls in all three visual manipulation frequencies. Fig. 2 depicts exemplar time series of the moving room oscillation and body

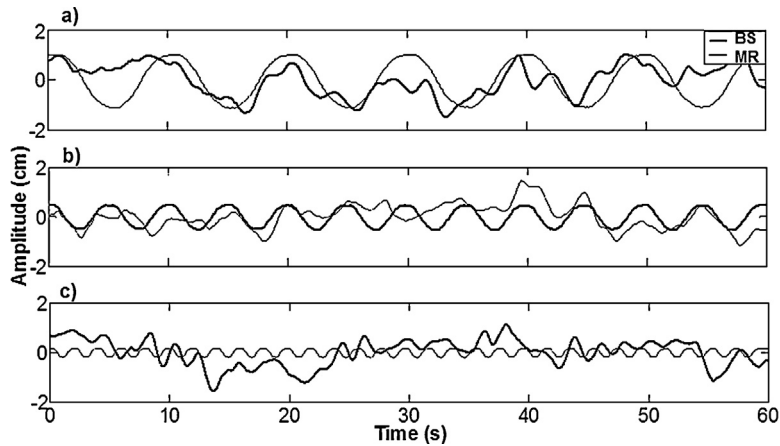


Fig. 2. Moving room (MR) displacement and body sway (BS) exemplar time series for a participant with Down syndrome at 0.1 (a), 0.2 (b), and 0.5 Hz (c) frequencies of the room movement.

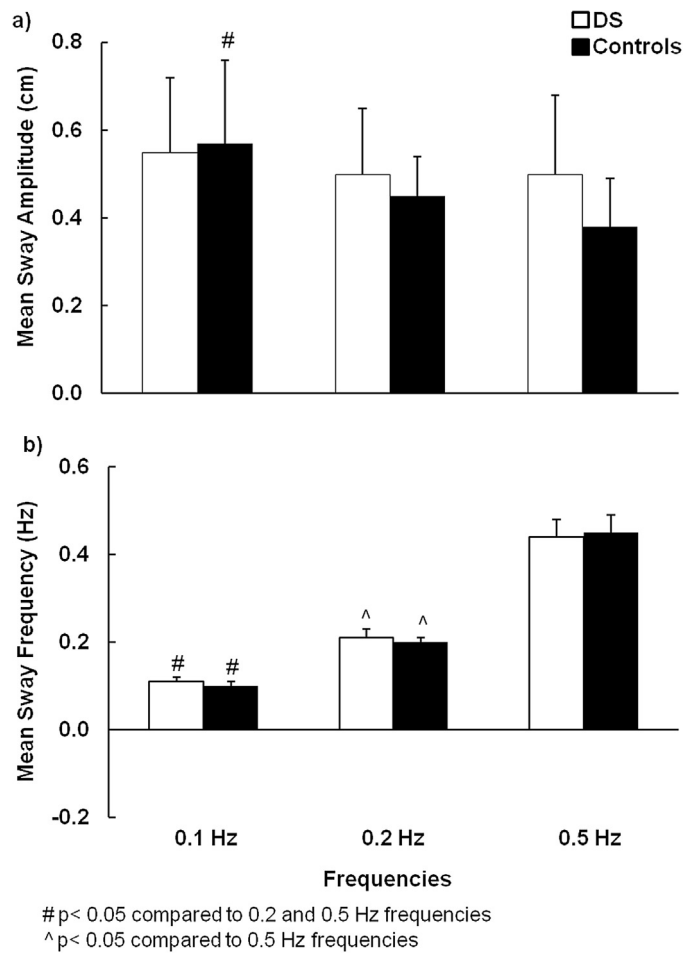


Fig. 3. Mean and standard deviation of the mean sway amplitude (a) and the mean sway frequency (b) for participants with Down syndrome (DS) and Controls at 0.1, 0.2 and 0.5 Hz frequencies of the room movement.

sway for a participant with DS, in each of the stimulus frequencies. It is possible to see that body sway characteristics differ according to the visual stimulus frequency.

Fig. 3 depicts mean sway amplitude and frequency for both participants with DS and controls in all three moving room frequencies. MANOVA revealed no group effect, Wilks' Lambda = 0.96, $F(2, 37) = 0.67$, $p = 0.516$, but it identified a significant effect

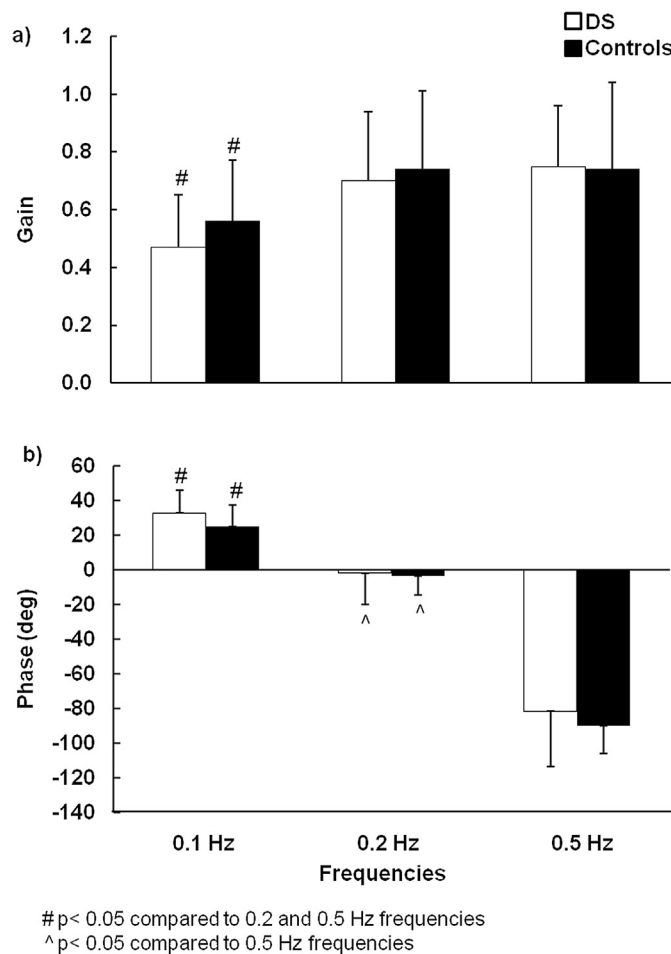


Fig. 4. Mean and standard deviation of the gain (a) and phase (b) for participants with Down syndrome (DS) and Controls at 0.1, 0.2 and 0.5 Hz frequencies of the room movement.

of frequency, Wilks' Lambda = 0.01, $F(4,35) = 1324.68$, $p = 0.0001$, and group by frequency interaction, Wilks' Lambda = 0.73, $F(4,35) = 3.23$, $p = 0.023$. For mean sway amplitude (Fig. 3a), univariate analysis revealed a significant frequency effect, $F(2,76) = 24.42$, $p = 0.0001$, and group by frequency interaction, $F(2,76) = 7.58$, $p = 0.001$. Post hoc tests indicated that while participants with DS swayed with the same amplitude at all three frequencies (0.1 Hz: 0.55 ± 0.17 cm; 0.2 Hz: 0.50 ± 0.15 cm; 0.5 Hz: 0.50 ± 0.18 cm), controls swayed with larger amplitude at 0.1 Hz (0.57 ± 0.19 cm) than at 0.2 Hz (0.45 ± 0.09 cm) and 0.5 Hz (0.38 ± 0.11 cm). For mean sway frequency (Fig. 3b), univariate analysis revealed a significant effect only for frequency, $F(2,76) = 1886.85$, $p = 0.0001$. Post hoc tests indicated that both participants with DS (0.1 Hz: 0.11 ± 0.01 Hz; 0.2 Hz: 0.21 ± 0.02 Hz; 0.5 Hz: 0.44 ± 0.04 Hz) and controls (0.1 Hz: 0.10 ± 0.01 Hz; 0.2 Hz: 0.20 ± 0.01 Hz; 0.5 Hz: 0.45 ± 0.04 Hz) increased sway frequency as room frequency increased.

Fig. 4 depicts gain and phase for both participants with DS and controls in all three moving room frequencies. MANOVA revealed no effect of group, Wilks' Lambda = 0.929, $F(2,37) = 1.41$, $p = 0.257$, but it revealed a significant frequency effect, Wilks' Lambda = 0.037, $F(4,35) = 229.35$, $p = 0.0001$. There was no group by frequency interaction, Wilks' Lambda = 0.89, $F(4,35) = 1.03$, $p = 0.407$. Univariate analysis revealed a significant frequency effect for both gain, $F(2,76) = 30.61$, $p = 0.0001$, and phase, $F(2,76) = 444.02$, $p = 0.0001$. Post hoc tests indicated that gain values were lower when the room oscillated at 0.1 Hz (0.52 ± 0.20) than at 0.2 Hz (0.72 ± 0.26) and 0.5 Hz (0.75 ± 0.26) (Fig. 4a). Participants swayed ahead of the room at 0.1 Hz (28.82 ± 12.81), with the room at 0.2 Hz (-2.60 ± 14.67), and lagged behind the room at 0.5 Hz (-85.70 ± 23.71) (Fig. 4b).

Fig. 5 depicts coherence for both participants with DS and controls in all three moving room frequencies. ANOVA revealed a significant group effect, $F(1,38) = 13.18$, $p = 0.0001$, but no frequency effect, $F(2,76) = 2.78$, $p = 0.069$, and no group by frequency interaction, $F(2,76) = 0.80$, $p = 0.451$. Participants with DS presented lower coherence (0.80 ± 0.12) than controls (0.90 ± 0.09).

Fig. 6 depicts position variability for both participants with DS and controls in all three moving room frequencies. ANOVA revealed a significant group, $F(1,38) = 5.01$, $p = 0.031$, and frequency effects, $F(2,76) = 8.84$, $p = 0.0001$, but no group by frequency interaction, $F(2,76) = 2.61$, $p = 0.080$. Participants with DS exhibited higher position variability (0.48 ± 0.16 cm)

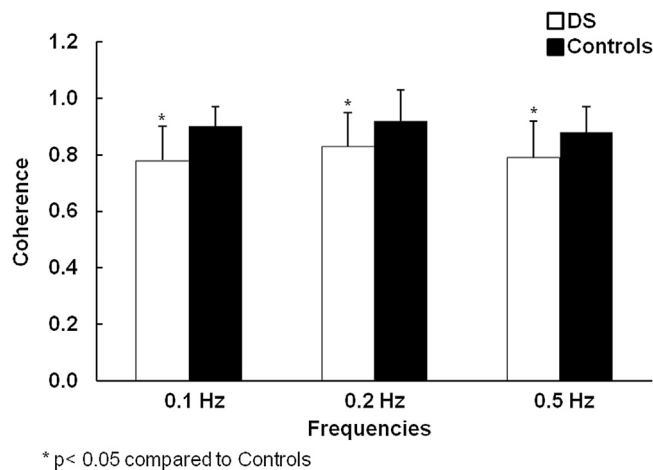


Fig. 5. Mean and standard deviation of the coherence for participants with Down syndrome (DS) and Controls at 0.1, 0.2 and 0.5 Hz frequencies of the room movement.

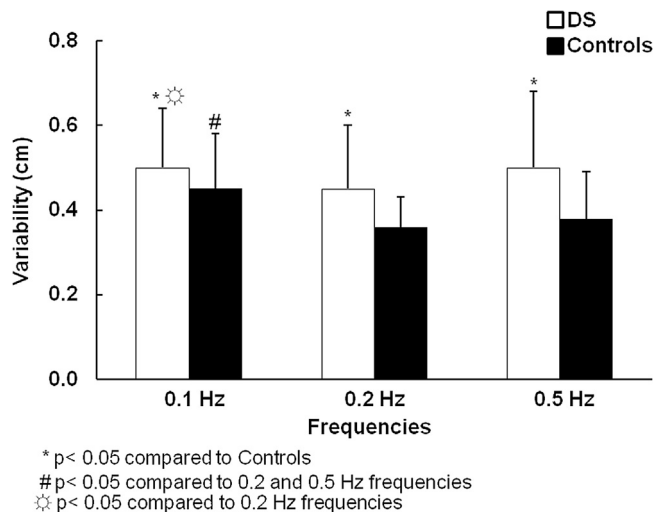


Fig. 6. Mean and standard deviation of the variability for participants with Down syndrome (DS) and Controls at 0.1, 0.2 and 0.5 Hz frequencies of the room movement.

than controls (0.40 ± 0.10 cm). Post hoc tests indicated that participants presented higher position variability at 0.1 Hz (0.47 ± 0.14 cm) than at 0.2 Hz (0.41 ± 0.12 cm).

3.3. Discrete visual manipulation

Discrete visual manipulation from the moving room induced correspondent body sway in both participants with DS and controls in both a forward and backward direction.

Fig. 7 depicts sway displacement and duration of time displacement for both participants with DS and controls at forward and backward discrete movement of the room. MANOVA revealed no group effect, Wilks' Lambda = 0.87, $F(2,37) = 2.74$, $p = 0.078$, but a significant effect of direction, Wilks' Lambda = 0.84, $F(2,37) = 3.58$, $p = 0.038$, and no group by direction interaction, Wilks' Lambda = 0.97, $F(2,37) = 0.64$, $p = 0.533$. Univariate analyses revealed a significant direction effect only for body displacement, $F(1,38) = 7.32$, $p = 0.010$, with participants showing larger body displacement when the room was moved forward (1.01 ± 0.46 cm) than backward (0.82 ± 0.40 cm) (Fig. 7a). Time of displacement was unaffected by either group or direction (Fig. 7b).

Fig. 8 depicts mean sway amplitude for both participants with DS and controls before and after discrete movement of the room. MANOVA revealed a significant group effect, Wilks' Lambda = 0.43, $F(2,37) = 24.02$, $p = 0.0001$, but no direction effect, Wilks' Lambda = 0.95, $F(2,37) = 1.03$, $p = 0.368$, and no group by direction interaction, Wilks' Lambda = 0.89, $F(2,37) = 2.26$, $p = 0.119$. Univariate analyses revealed group differences before, $F(1,38) = 49.03$, $p = 0.0001$, and after, $F(1,38) = 29.22$, $p = 0.0001$, the discrete movement of the room. The participants with DS showed larger sway amplitude than the con-

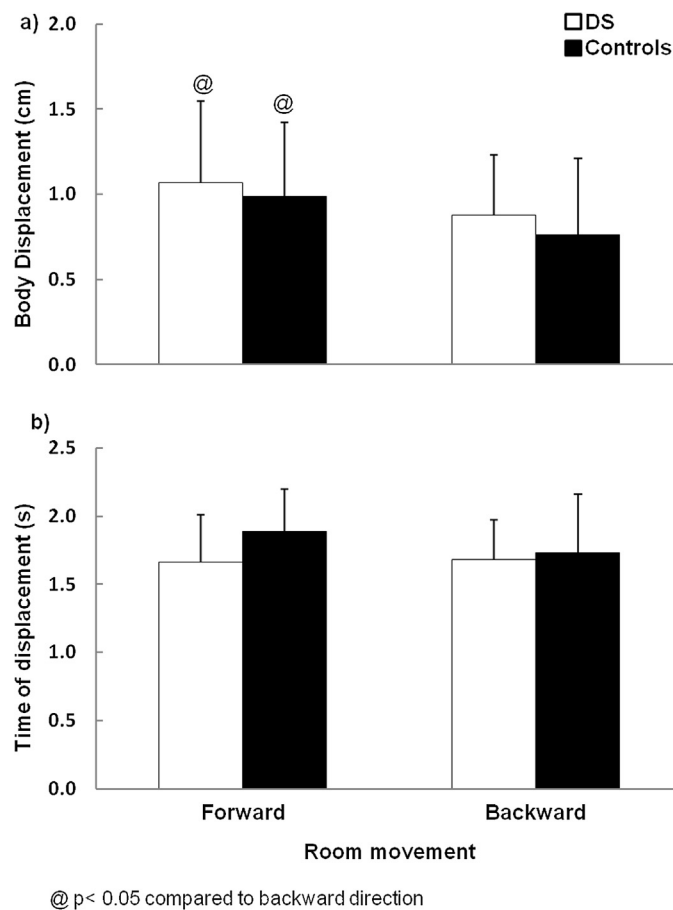


Fig. 7. Mean and standard deviation of the body displacement (a) and time of displacement (b) for participants with Down syndrome (DS) and Controls in the forward and backward movement of the room.

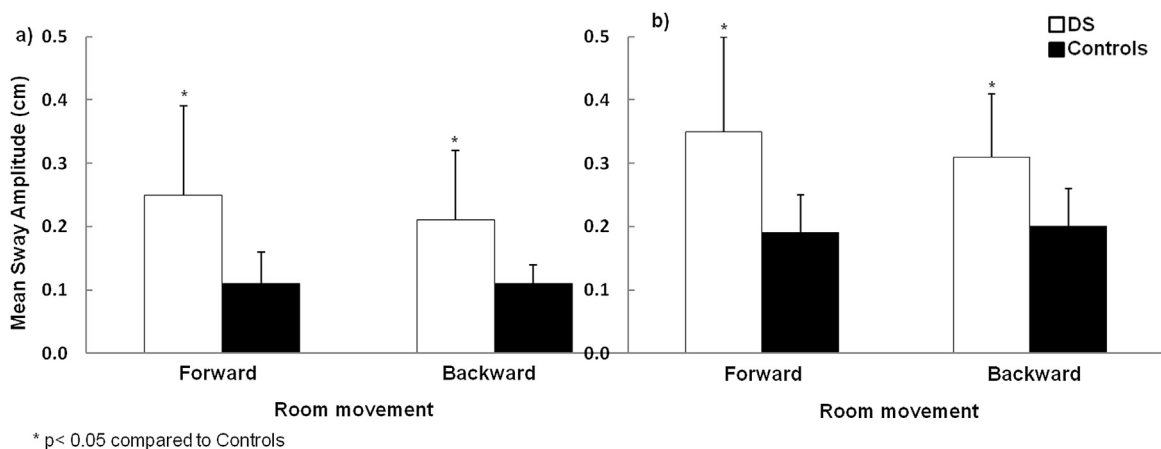


Fig. 8. Mean and standard deviation of the mean sway amplitude before (a) and after (b) room movement for participants with Down syndrome (DS) and Controls in the forward and backward movement of the room.

controls both before (DS: 0.25 ± 0.14 cm; controls: 0.11 ± 0.05 cm) and after (DS: 0.35 ± 0.15 cm; controls: 0.19 ± 0.06 cm) the forward movement of the room. For the backward movement of the room, the sway amplitude was also larger for the participants with DS before (DS: 0.21 ± 0.11 cm; controls: 0.11 ± 0.03 cm) and after (DS: 0.31 ± 0.10 cm; controls: 0.20 ± 0.06 cm) room movement.

4. Discussion

The present study investigated the use of visual information to control postural sway and the coupling between visual information and body sway in adults with DS. Adults with DS swayed with larger amplitude compared to controls in the quiet standing task. However, participants with DS were able to modulate their postural sway in response to the visual stimulus in a moving room, but with some differences in terms of the scaling of postural responses to spatial parameters of the visual cue, showing higher position variability and lower coherence than the control participants. In the discrete visual manipulation condition, adults with DS presented similar postural responses to control participants.

The finding that adults with DS swayed with a larger amplitude than their peers without DS have been observed in several other studies (Cabeza-Ruiz et al., 2011; Cimolin et al., 2011; Galli et al., 2008; Gomes & Barela, 2007; Vieregge et al., 1996; Webber et al., 2004), suggesting differences in postural control functioning. However, the underpinning changes that lead to poor postural control stability in adults with DS remain unclear. In order to achieve body stability, the postural control system needs to obtain and integrate the relevant sensory cues to identify body sway dynamics (i.e., position and velocity) and activate appropriate muscles to maintain or balance the forces acting on the body (Barela et al., 2003; Metcalfe et al., 2005). In this perspective, the fact that individuals with DS sway with a larger amplitude than controls might be due to several factors such as: difficulties to obtain the appropriate sensory cues (Biec et al., 2014; Vuillerme et al., 2001; Webber et al., 2004) or impairment in the attentional focus (Dulaney & Tomporowski, 2000); deficits to activate the muscles in the correct sequence and magnitude (Aruin & Almeida, 1997; Carvalho & Almeida, 2009; Shumway-Cook & Woollacott, 1985; Vuillerme et al., 2001; Webber et al., 2004); and difficulties to properly couple sensory information and motor action (Polastri & Barela, 2005).

Individuals with DS seem to experience problems in obtaining sensory cues that would provide accurate information about body position and velocity in conditions in which the sensory cues are reduced or deteriorated (Biec et al., 2014; Vuillerme et al., 2001; Webber et al., 2004). On the other hand, when sensory cues are manipulated and sensory information is enhanced, for example when lightly touching a rigid surface, individuals with DS are able to properly detect and use this additional information to improve postural control performance, reducing body sway amplitude and reaching the performance level of controls (Gomes & Barela, 2007). Our present results clearly showed that individuals with DS were able to detect the visual stimulus coming from the movement of the room, and to produce a correspondent body sway response, but their responses were not identical to the controls. The participants with DS showed a weaker relationship between body sway and the visual stimulus than controls, as evidenced by the coherence measure. Moreover, the mean sway amplitude in the continuous condition suggests qualitative differences between participants with DS and controls. Individuals with DS were not able to scale their body sway amplitude according to the visual stimuli, swaying with the same amplitude across all three moving room amplitudes/frequencies (1.0 cm at 0.1 Hz; 0.5 cm at 0.2 Hz and 0.2 cm at 0.5 Hz) while controls swayed with a larger amplitude at 0.1 Hz compared to the other frequencies (0.2 and 0.5 Hz). Therefore, individuals with DS are able to detect dynamic visual information and use it to organize the spatial-temporal dynamics of body sway, but, at the same time, they show difficulties in extracting all of the characteristics embedded in the visual stimuli in order to modulate body sway responses. These results are in accordance with a previous study that used the same moving room paradigm with similar parameters (i.e. amplitude of 1 cm at 0.1 Hz frequency), but with a different clinical population (i.e. individuals with developmental coordination disorder) (Chung & Stoffregen, 2011).

Another possible reason for the postural sway differences between adults with DS and controls may be differences in the focus of attention. According to Chiviakowsky, Wulf, and Wally (2010) and McNevin and Wulf (2002), an external focus of attention improved balance control. Thus, it is possible that the controls adopted an external focus of attention (i.e., looking at the target fixed on the front wall of the room), while participants with DS preferred an internal focus of attention (i.e., maintaining the body position as quiet as possible). This could explain the higher body sway amplitude in the baseline condition and the weaker coupling between body sway and visual stimulus in the room movement condition for the participants with DS.

Results from several studies have suggested that individuals with DS show different muscle activation (longer latencies) and muscle coordination (Aruin & Almeida, 1997; Carvalho & Almeida, 2009; Shumway-Cook and Woollacott, 1985; Vuillerme et al., 2001; Webber et al., 2004) that can be related to the increased body sway amplitude observed in individuals with DS. However, individuals with DS showed a similar latency to respond to the discrete movement of the room compared to controls. Based upon these results, we can suggest that despite all the observed differences regarding specific muscle activation, such differences do not seem to affect the performance of individuals with DS during upright stance even under visual perturbation. The discrepancy of our results compared to those previously observed (Aruin & Almeida, 1997; Carvalho & Almeida, 2009) might be due to methodological differences, in which the discrete moving room perturbation is less demanding than the ones employed in other studies.

Despite the similarity in using visual cues to produce body sway, in both continuous and discrete conditions, individuals with DS exhibited a larger sway amplitude when visual cues were not manipulated and higher position variability and lower coherence during continuous visual manipulation than controls. These results may be due to an inherently increased noise level in the postural control system (Barela et al., 2003; Van der Kooij & Peterka, 2011), which may be related to noise in the commands sent to the peripheral musculature. Considering that several studies have observed important differences inherent to muscle properties and control in individuals with DS (Almeida et al., 2000; Aruin & Almeida, 1997; Carvalho & Almeida, 2009; Latash et al., 2002), these differences might contribute to errors between the actual movement and the one

specified by the central nervous system. Another potential source of noise in individuals with DS could relate to estimation of body orientation (i.e., position and velocity). Individuals with DS show differences regarding the functioning of the sensory system such as increased somatosensory latency, reduced visual acuity and contrast sensitivity (Chen & Fang, 2005; Cole et al., 1988; John, Bromham, Woodhouse, & Candy, 2004), and an less reliable sensory system might account for the difficulties in estimating body sway properly.

It is worthy noticing that when sensory cues are manipulated, the contribution of sensory information is augmented, as observed in the postural control performance with enhanced haptic cues in individuals with DS (Gomes & Barela, 2007). Enhancement of sensory cues would overcome the noisier system, by providing more reliable information about body sway. This seems to be the case, according to our results, since individuals with DS swayed with the same amplitude as controls when visual information was manipulated, but with higher position variability and lower coherence (in the continuous room movement condition), that could relate to the less functional motor system of individuals with DS.

Our results indicate that postural control functioning of adults with DS is similar to adults without DS, as suggested previously (Gomes & Barela, 2007; Vuillerme et al., 2001). Performance differences are due to poor functioning of the motor control system and to less precise inputs coming from the visual system. Vuillerme et al. (2001) suggested that rather than functional differences, quantitative differences were most likely to be related to the poor performance of the postural control system. Based on the findings of the present study we can add to this suggestion that these quantitative differences are related to the motor control system and to the quality of the visual inputs coming from the visual system. Moreover, in conditions and situations in which visual cues are manipulated, visual information is enhanced and postural control performance is improved in individuals with DS.

In summary, we showed that adults with DS were able to couple their body sway to the visual stimulus, but with higher position variability and lower coherence than controls. Furthermore, individuals with DS showed poor performance in postural control because of an inherently high noise level in the postural control system, that might be due to differences in motor control commands sent to the musculature, differences in attentional focus, and less accurate visual cues coming from the visual system. These differences can be minimized by providing more informative visual cues, leading to enhanced visual information related to body sway.

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