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Unstable coupling of body sway with imposed motion precedes visually induced motion sickness

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ABSTRACT

Motion sickness is preceded by differences in the quantitative kinematics of body sway between individuals who (later) become sick and those who do not. In existing research, this effect has been demonstrated only in measures of body sway, relative to the earth. However, body sway can become coupled with imposed oscillatory motion of the illuminated environment, and the nature of this coupling may differ between individuals who become sick and those who do not. We asked whether body sway would become coupled to complex oscillations of the illuminated environment, and whether individual differences in such coupling might be precursors of motion sickness. Standing participants (women) were exposed to complex oscillation of the illuminated environment. We examined the strength of coupling as a function of time during exposure. Following exposure, some participants reported motion sickness. The nature and temporal evolution of coupling differed between participants who later reported motion sickness and those who did not. Our results show that people can couple the complex dynamics of body sway with complex imposed motion, and that differences in the nature of this coupling are related to the risk of motion sickness.

1. Introduction

One of the least controversial observations about motion sickness is that the malady is associated with imposed motion. Perhaps the paradigmatic example is physical displacement of the support surface, as occurs on ships at sea, and in terrestrial vehicles (e.g., Reason, 1978; Stoffregen & Riccio, 1991). Motion sickness also is associated with displacement relative to the illuminated environment, as occurs in video games (e.g., Stoffregen, Faugloire, Yoshida, Flanagan, & Merhi, 2008), virtual environments (e.g., Villard, Flanagan, Albanese, & Stoffregen, 2008) and moving rooms (e.g., Lee & Lishman, 1975; Stoffregen, 1985; Stoffregen & Smart, 1998). Motion sickness is common when participants are exposed to multi-frequency oscillations in the range 0.1–0.4 Hz. This is true for physical displacement (e.g., Guignard & McCauley, 1982; Lawther & Griffin, 1988), and for imposed oscillations of the illuminated environment (e.g., Bonnet, Faugloire, Riley, Bardy, & Stoffregen, 2006; Koslucher, Haaland, Malsch, Webeler, & Stoffregen, 2015; Stoffregen & Smart, 1998; Stoffregen, Yoshida, Villard, Scibora, & Bardy, 2010).

In a given situation, motion sickness typically occurs in some participants, but not in others. For example, on a ship at sea some people may develop seasickness, while others do not (e.g., Lawther & Griffin, 1988; Stoffregen, Chen, Varlet, Alcantara, & Bardy, 2013). In the moving room paradigm, in which the illuminated environment oscillates along the line of sight of an inertially

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stationary observer, imposed oscillations in the 0.1–0.4 Hz range typically lead to motion sickness in approximately 40% of participants (e.g., Bonnet et al., 2006; Stoffregen & Smart, 1998; Stoffregen et al., 2010). Effects of this kind are understood to mean that susceptibility is related to individual differences in responses to imposed motion. An important question, then, is to identify the individual differences that account for susceptibility to motion sickness. It is commonly assumed that the relevant individual differences exist in inferential mental models, or expectations about sensory stimulation (e.g., Oman, 1982; Reason, 1978). An alternative view is that the relevant individual differences exist in the ability to adapt perceptual-motor activity to changing constraints on bodily control (e.g., Stoffregen, 2011). We asked whether motion sickness might be related to coupling or synchronization of body sway with imposed motion of the illuminated environment. Our study was not designed to distinguish between different theories of motion sickness etiology. Rather, in this exploratory study, our primary aim was to evaluate a new measure of body sway as a possible precursor of visually induced motion sickness.

1.1. Postural precursors of motion sickness

In the postural instability theory of motion sickness, Riccio and Stoffregen (1991) proposed that instability in the control of posture (of the entire body, or of its segments) was a necessary and sufficient condition for motion sickness. An important test of this theory is to measure postural activity (e.g., body sway) during exposure to potentially nauseogenic stimuli, and to compare postural activity between persons who (later) report motion sickness and those who do not. This test has been conducted numerous times, in diverse circumstances, and the prediction has been confirmed. Distinctive patterns of postural kinematics have preceded subjective symptoms of motion sickness in military flight simulators (Stoffregen, Hettinger, Haas, Roe, & Smart, 2000), virtual environments (Villard et al., 2008), moving rooms (e.g., Koslucher, Haaland, & Stoffregen, 2014; Koslucher, Munafo, & Stoffregen, 2016; Smart, Stoffregen, & Bardy, 2002; Stoffregen & Smart, 1998; Stoffregen et al., 2010), and head-mounted displays (Merhi, Faugloire, Flanagan, & Stoffregen, 2007). Postural precursors of motion sickness have been identified in qualitatively different parameters of body sway, such as the spatial magnitude of movement (e.g., Bonnet et al., 2006; Stoffregen et al., 2010), and the temporal dynamics of movement, including movement multifractality (e.g., Koslucher et al., 2016; Munafo, Diedrick, & Stoffregen, 2017).

The diversity of the settings that have been tested, and of the dependent variables that have been evaluated, provide powerful confirmation of the prediction that postural activity should differ between persons who experience motion sickness and persons who do not, and that such differences should exist before the onset of any subjective symptoms of motion sickness.

Research on motion sickness often is focused upon the time course of subjective symptoms during exposure to imposed stimulus motion (e.g., Keshavarz & Hecht, 2011). Subjective symptoms change over time during exposure. However, in most cases the induction of symptoms is not instantaneous (Stanney et al., 1998). This fact is central to our method. Our motion stimuli are subtle, such that many participants are not aware of any imposed motion (e.g., Stoffregen & Smart, 1998). In part for this reason, subjective symptoms of motion sickness often occur only after 10–20 min of exposure (e.g., Bonnet et al., 2006; Stoffregen & Smart, 1998; Stoffregen et al., 2010; Villard et al., 2008). Explicitly and repeatedly, we instruct participants to discontinue their participation immediately if they experience any symptoms of motion sickness, however mild. Taken together, these aspects of our design mean that body sway during exposure occurs before the onset of subjective symptoms. It is in this sense that differences in body sway between participants who (later) report motion sickness and those who do not can be considered to be precursors of motion sickness.

Motion sickness susceptibility differs between the sexes. For example, seasickness is more common among women than men (e.g., Lawther & Griffin, 1988). Similar effects have been reported for visually induced motion sickness. Koslucher et al. (2015) evaluated visually induced motion sickness in a moving room. They found a large, statistically significant sex difference in the incidence of motion sickness, with women (38%) far more likely to become sick than men (9%). Given this result, in the present study we elected to use only female participants, so as to more nearly approach equal sample sizes in our Well and Sick groups.

1.2. Coupling and motion sickness

In most existing research, body sway has been measured relative to the earth, that is, relative to motion sensing devices that were, themselves, stationary relative to the earth. When motion sickness is induced by imposed motion, there exists the logical possibility that postural precursors of motion sickness might exist, not in movement of the body relative to the earth, but in movement of the body relative to the imposed motion. This possibility is plausible in relation to the general literature documenting the tendency of standing participants to couple body sway with imposed motion relative to the illuminated environment, in the moving room paradigm. Several studies have documented such coupling when a moving room oscillated at 0.2 Hz (i.e., a simple sinusoid) with small amplitude (e.g., 2 cm); (e.g., Dijstra, Schoner, & Gielen, 1994; Lee & Lishman, 1975; Stoffregen, 1985). Motion sickness is most commonly associated with imposed oscillations that are not simple sinusoids but, rather, are complex, comprising sum-of-sines functions (e.g., Bonnet et al., 2006; Koslucher et al., 2015; Stoffregen et al., 2010; Stoffregen & Smart, 1998). Outside the motion sickness literature, some studies have identified individual differences in the strength of coupling between postural control and imposed motion stimuli (e.g., Faugloire, Bardy, & Stoffregen, 2006; Toledo & Barela, 2014). The existence of individual differences in the strength of coupling between imposed oscillations and postural activity is consistent with the logical possibility that motion sickness susceptibility could be related to individual differences in the strength of such coupling. Hence, we predicted that the coupling of body sway with complex oscillations would differ between participants who reported motion sickness and those who did not, and that these differences would exist before the onset of subjective symptoms of motion sickness.

In the present study, our primary interest was in the coupling of body sway with imposed motion (that is, body sway relative to a moving room). Several measures of coupling between two time series are robust to the complexity of animate movement, including

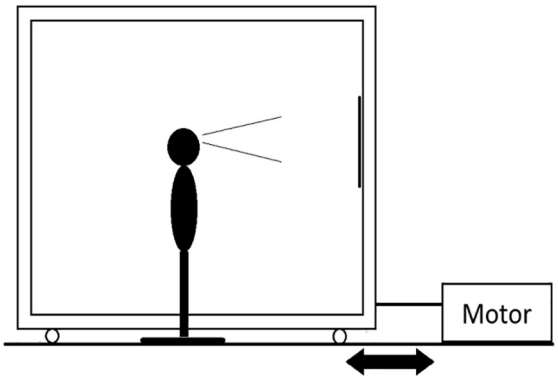

Fig. 1. The moving room.

cross-spectral coherence (e.g., Toledo & Barela, 2014; Varlet, Bardy, Chen, Alcantara, & Stoffregen, 2015), the cross-wavelet transform (Issartel, Marin, Bardaienne, Gaillor, & Cadopi, 2006; Varlet, Marin, Legarde, & Bardy, 2011), and average mutual information (Balasubramaniam, Riley, & Turvey, 2000; Boker, Schreiber, Pompe, & Bertenthal, 1998; Stoffregen, Villard, Kim, Ito, & Bardy, 2009). We elected to use average mutual information, or AMI, which assesses the uncertainty in the prediction of a given measurement in one time series according to measurements in another time series.

We also evaluated two other orthogonal measures of body sway relative to the earth; the spatial magnitude of sway, and its degree of multifractality. These measures allowed us to determine whether results from the current study were compatible with effects reported in the literature.

2. Method

2.1. Participants

The participants were 30 women, with mean age 21.13 years (SD = 2.28 years), mean height 1.66 m (SD = 1.4 m), and mean weight 64.5 kg (SD = 9.19 kg). Each participant had normal or corrected-to-normal vision, and reported that they had no history of disease or malfunction of the vestibular apparatus, recurrent dizziness, or falls. The experimental protocol was approved, in advance, by the University of Minnesota IRB.

2.2. Apparatus

Visual stimulus motion was created using a moving room, which consisted of a cubical frame, 2.44 m on a side, mounted on wheels and moved along one axis on rails (Fig. 1). The interior surfaces of the walls and ceiling were covered with blue and white marble-patterned adhesive paper. At the center of the front wall was a map of the continental United States (53 × 80 cm; 19° × 28°). Illumination was provided by floodlights mounted inside the room and oriented so that shadows were minimized. Movement of the room (oscillation along an axis parallel to the line of sight) was powered by an electric motor under computer control. Participants stood on the floor of the laboratory, such that there was no imposed inertial motion.

We monitored body sway using a magnetic tracking system (FasTRAK, Polhemus, Colchester VT). To measure motion, we used three sensors, each sampled at 40 Hz. One sensor was attached to a safety helmet worn on the head, and one between the shoulder blades (at the level of the C7 vertebra), using cloth medical tape. A third sensor was attached to the moving room. We retained data for the anterior-posterior (AP) and mediolateral (ML) axes.

2.3. Procedure

As part of the informed consent process, participants were instructed to discontinue the experiment immediately upon experiencing any motion sickness symptoms, however, mild. After the informed consent process, we separately (i.e., independently) assessed participants' initial motion sickness status and symptom levels. Participants completed the Simulator Sickness Questionnaire, or *SSQ* (Kennedy, Lane, Berbaum, & Lilienthal, 1993), which allowed us to assess the initial level of symptoms (SSQ-1). To assess motion sickness status, participants responded to a forced-choice, yes/no question, *Are you motion sick?* Next, we measured height and weight.

Participants stood upon marked lines on the floor, such that the midlines of the heels were 17 cm apart, and the angle between the feet was 10°. Participants were instructed that, during trials, they should not move their feet, and should keep their hands at their sides.

We used a within-participants experimental design. We began with four brief, 60 s trials, in which we measured body sway in the absence of nauseogenic motion stimuli. Data from these trials will be reported elsewhere (cf. Koslucher et al., 2016). Participants

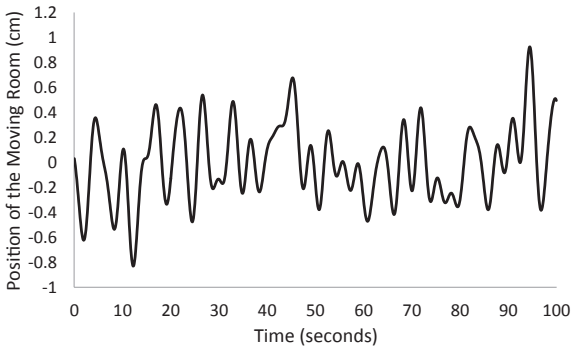

Fig. 2. A portion of the sum-of-sines motion function of the moving room. The actual motion was a non-repeating function lasting up to 600 s.

were then exposed to a sum of ten sines in the range 0.1–0.4 Hz, with maximum displacement amplitude of 2.5 cm (Fig. 2). The motion function was identical to that used in previous studies of visually induced motion sickness (e.g., Bonnet et al., 2006; Koslucher et al., 2014; Stoffregen et al., 2010). Each trial was 10 min long, and participants were exposed to a maximum of four trials. During these trials, participants were instructed to maintain their gaze on the map on the front wall of the moving room. Before each trial, participants were reminded to discontinue participation immediately if they experienced any symptoms of motion sickness, however mild.

Between trials, the moving room was stationary and the participant was required to sit for 1 min. Upon discontinuation or after the completion of the experimental protocol (whichever came first), participants completed SSQ-2, and the forced-choice question, *Are you motion sick?* Participants who stated that they were not sick after the exposure to room motion (i.e., at SSQ-2) were given a printed copy of the SSQ (SSQ-3), which included the forced-choice, *yes/no* question, *Are you motion sick?*, and asked to complete it immediately if they became sick at any time in the following 24 h or, if they did not become sick, after 24 h.

2.4. Data analysis and inferential statistics

We assigned participants to the Well and Sick groups based solely upon their responses to the forced-choice, yes/no question, *Are you motion sick?* Participants who responded in the affirmative after exposure were assigned to the Sick group. All others were assigned to the Well group. For SSQ data, we evaluated the Total Severity Score, which we computed in the recommended manner (Kennedy et al., 1993). As SSQ scores are not normally distributed, in analyzing these data we used non-parametric statistics.

We evaluated the evolution of sway over the course of exposure to the sum-of-sines stimulus. To evaluate the evolution of sway over time, we selected three 2-min time windows from the data, using the method of Bonnet et al. (2006), Koslucher et al. (2014), and Stoffregen et al. (2010). For the Sick group, we choose the first, the middle, and the final two minutes for each participant, with the restriction that no window included a boundary between two trials (that is, each window included only continuous data from within a single trial). For the Well group, the selection of time windows was based upon the mean exposure duration for the Sick group, which ensured that the sway data for both groups corresponded to the same mean duration of exposure to the sum-of-sines stimulus.

Using data from the three time windows, we conducted three orthogonal analyses. For each dependent variable, we separately analyzed body sway in the AP and ML axes. Our first two analyses addressed body sway relative to the earth. We evaluated the spatial magnitude of body sway in terms of positional variability, which we defined operationally as the standard deviation of head and torso positions (e.g., Koslucher et al., 2016). We evaluated the temporal dynamics of sway using multifractal detrended fluctuation analysis, or MF-DFA (e.g., Kelty-Stephen, Palatinus, Saltzman, & Dixon, 2013). MF-DFA is an extension of more traditional detrended fluctuation analysis (Lin, Seol, Nussbaum, & Madigan, 2008). Several studies have documented the existence of multifractality in standing body sway (e.g., Kantelhardt et al., 2002; Munafo, Curry, Wade, & Stoffregen, 2016). Other studies have shown that postural multifractality can be a precursor of visually induced motion sickness (Koslucher et al., 2016; Munafo et al., 2017). The multifracality of movement is evaluated in terms of the *singularity spectrum*. The width of the singularity spectrum indicates the degree of multifractality: A wider singularity spectrum indicates more multifractal movement (Kelty-Stephen et al., 2013). Using open-source software (Ihlen, 2012), we computed the width of the singularity spectrum for each time window. We selected a minimum scaling range of 16 data points with 19 evenly spaced increasing segment sizes to a maximum of the length of the time series. This range was the same for each time series. The computation of spectrum width is susceptible to outliers (Kelty-Stephen et al., 2013). For this reason, before conducting inferential statistics on spectrum width we removed data from participants for which spectrum width was greater than three standard deviations from the overall mean (i.e., the mean across the three windows and all participants).

For our third analysis, we computed average mutual information (AMI) as a measure of the extent to which body sway was coupled with imposed motion of the illuminated environment. AMI is a form of cross-correlation analysis. Traditional cross-correlation techniques can reveal linear relations between time series. Recent research has revealed that movement in upright stance can contain significant nonlinearities (e.g., Balasubramaniam et al., 2000; Duarte & Zatsiorsky, 2000). The complexity of postural activity led us to consider nonlinear dependencies in coupling between the room motion and postural activity. AMI consists of assessing the uncertainty in the prediction of a given measurement according to a preceding measurement (for equations, see Stoffregen et al.,

2009). In previous research, AMI has proved useful in identifying nonlinear coupling in postural activity relating to motion sickness (e.g., Dong, Yoshida, & Stoffregen, 2011). In computing AMI, we used the method of Boker et al. (1998), as implemented by Stoffregen et al. (2009). Following Boker et al. (1998) and Stoffregen et al. (2009) we analyzed 201 time-lags around 0, from −100 to +100, and from these we selected the maximum of AMI. This analysis yielded the mean maximum AMI for each condition, where the unit of mutual information is bits.

In analyzing data on postural activity, the factors were Body Axis (AP vs. ML), Body Segment (Head vs. Torso), Time Windows (1 vs. 2 vs. 3) and Sickness Groups (Well vs. Sick). Where kinematic data satisfied the sphericity and normality assumptions, we used repeated-measures analysis of variance (ANOVA), which permitted us to report partial η^2 as a measure of effect size. According to Cohen (1988), values of partial $\eta^2 > 0.14$ indicate a large effect, and values of partial $\eta^2 > 0.06$ indicate a medium effect. Where kinematic data violated the sphericity assumption, we used mixed effects modeling (e.g., Kreft & de Leeuw, 1998; Yu et al., 2010). Mixed effects models are constructed from fixed effects, random effects, and covariance structures, and analyzed using restricted maximum likelihood, which estimates the variance components of the random effects without influence from the fixed effects or outlying values. Independent variables are usually selected as fixed effects, as in traditional ANOVA. Factors that introduce added variability, such as multiple observations taken on subjects, are selected as random effects. Mixed effects models do not compute degrees of freedom in the traditional manner (i.e., $n - p$, where p refers to the number of parameters in the model). Instead, degrees of freedom are estimated from null distributions of F-statistics. In our mixed effects models, we classified participants as random variables and simplified the random effects model using backward elimination and refitting at each step. The optimal model was found when the Akaike Information Criterion was lower than in previous models. For our mixed effects models, we did not evaluate effect sizes because methods for calculating effect sizes in linear mixed models have not been widely established. For each dependent variable we conducted separate 2 (body axes; AP vs. ML) × body segment (Head vs. Torso) × Time Windows (Window 1 vs. Window 2 vs. Window 3) × Sickness Groups (Well vs. Sick) analyses. The task, condition, body axes, body segments, and time windows factors were within-participants, while the Sickness Groups factor was between-participants. To account for multiple comparisons, we set $\alpha = 0.01$.

3. Results

3.1. Motion sickness incidence and discontinuation

Nine of 30 participants (30.00%) stated that they were motion sick, and so were included in the Sick group. Each of these nine participants stated that they were motion sick at SSQ-2. All other participants were included in the Well group.

Among participants assigned to the Well group, none discontinued. Among participants assigned to the Sick group, all nine discontinued, with average exposure duration of 20 m 39 s. That is, the mean time of discontinuation was shortly after the beginning of the third 10-minute trial.

3.2. Symptom severity

Data on symptom severity are summarized in Fig. 3. Before exposure to room motion (i.e., at SSQ-1), the difference between Total Severity Scores for the Well and Sick groups was not significant, $U = 93.5$, $p = .98$. After exposure to room motion, symptoms in the Sick group were greater than in the Well group, $U = 8.0$, $p < .001$. Following exposure to the moving room, SSQ scores were higher than before exposure for the Sick group, $z = 2.67$, $p < .05$, but also for the Well group, $z = 3.92$, $p < .001$.

3.3. Body sway

As noted above, we used the mean exposure duration of the Sick group (20 m 39 s) to define the Time Windows for the Well group. Accordingly, for the Well group Time Window 1 corresponded to the first 2 min of the first sum-of-sines trial (0:00–2:00), Time

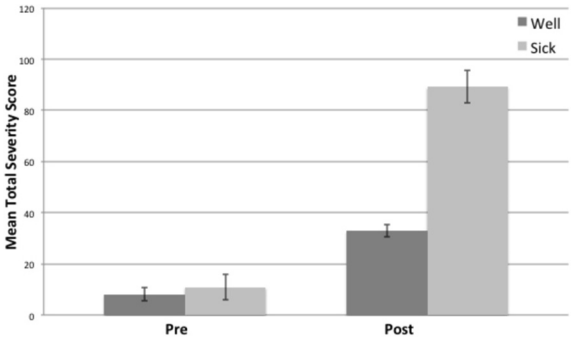

Fig. 3. Subjective symptoms (mean SSQ total severity score) before and after exposure to sum-of-sines room motion, for the Well and Sick groups. The error bars represent the standard error of the mean.

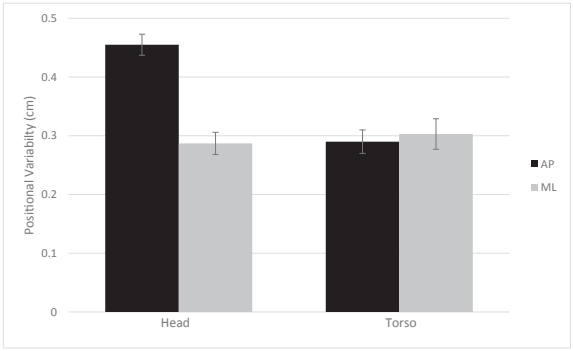

Fig. 4. Mean positional variability, illustrating the statistically significant interaction between Body Segment (head vs. torso) and Body Axis (AP vs. ML). The error bars represent the standard error of the mean.

Window 2 corresponded to the first 2 min of the second sum-of-sines trial (10:00–12:00), and Time Window 3 corresponded to the final 2 min of the second sum-of-sines trial (18:00–20:00).

For positional variability, mixed effects modeling revealed that the main effect of Axis was significant, $F(2,179.17) = 32.27$, $p < .001$. Positional variability was greater in the AP axis (mean = 0.372 cm, SE = 0.02 cm) than in the ML axis (mean = 0.295 cm, SE = 0.02 cm). The main effect of Time Windows was significant, $F(2,98.1) = 21.14$, $p < .001$. Positional variability increased over time (Window 1 mean = 0.249 cm, SE = 0.01 cm; Window 2 mean = 0.331 cm, SE = 0.02 cm; Window 3 mean = 0.421 cm, SE = 0.03 cm). In addition, the Body Segment × Axis interaction was significant, $F(2,146.49) = 32.97$, $p < .001$. As can be seen in Fig. 4, positional variability was greatest for the head in its AP axis. The primacy of motion in AP is consistent with the fact that this was the axis of room motion. Beyond this, the interaction suggests that participants responded to room motion primarily with the head, rather than the entire body. Finally, the Time Windows × Sickness Groups interaction was significant, $F(2,56) = 8.44$, $p = .008$, partial $\eta^2 = 0.23$ (Fig. 5A). Post-hoc tests did not reveal any significant difference among the groups (Well group Window 1 mean = 0.269 cm, 99% CI = 0.221 cm < mean < 0.317 cm, Window 2 mean = 0.337 cm, 99% CI = 0.237 cm < mean <

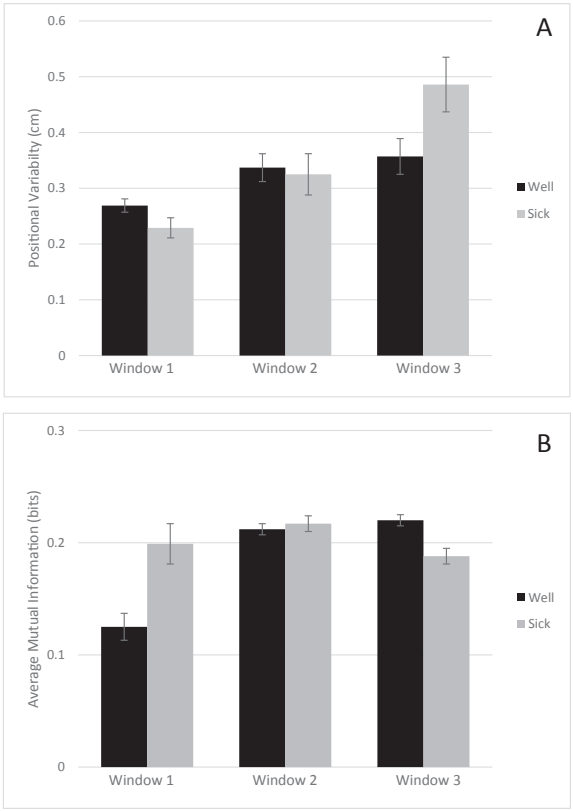

Fig. 5. Data on postural activity, illustrating statistically significant Time Windows × Sickness Groups interactions. A. Positional variability. B. Average Mutual Information. The error bars represent the standard error of the mean.

0.437 cm, Window 3 mean = 0.357 cm, 99% CI = 0.229 cm < mean < 0.485 cm; Sick group Window 1 mean = 0.229 cm, 99% CI = 0.157 cm < mean < 0.301 cm, Window 2 mean = 0.325 cm, 99% CI = 0.177 cm < mean < 0.473 cm, Window 3 mean = 0.486 cm, 99% CI = 0.290 cm < mean < 0.682 cm).

For MF-DFA, two participants (1 Well, 1 Sick) exhibited values of spectrum width that were more than three standard deviations from the group mean, for at least one trial and, accordingly, were removed as outliers. ANOVA revealed that the main effect of Axis was significant, $F(1,26) = 42.77$, $p < .001$, partial $\eta^2 = 0.62$. The multifractal spectrum was wider for movement in the AP axis (mean = 0.539, 99% CI = 0.467 < mean < 0.611) than for movement in the ML axis (mean = 0.415, 99% CI = 0.355 < mean < 0.475). The main effect of Segment was significant, $F(1,26) = 29.32$, $p < .001$, partial $\eta^2 = 0.53$. The multifractal spectrum was wider for movement of the head (mean = 0.530, 99% CI 0.466 < mean < 0.594) than for movement of the torso (mean = 0.423, 99% CI 0.347 < mean < 0.499). The main effect of Time Windows was significant, $F(2,52) = 6.31$, $p = .004$, partial $\eta^2 = 0.20$. The width of the multifractal spectrum increased over time; however, there was not a significant difference between any two Time Windows (Window 1 mean = 0.448, 99% CI = 0.364 < mean < 0.532; Window 2 mean = 0.460, 99% CI = 0.408 < mean < 0.512; Window 3 mean = 0.522, 99% CI = 0.434 < mean < 0.610).

For AMI, mixed effects modeling revealed that the effect of Axis was significant, $F(1,28.20) = 72.20$, $p < .001$. AMI in the AP axis (mean = 0.21 bits, 99% CI = 0.187 bits < mean < 0.227 bits) was greater than in the ML axis (mean = 0.18 bits, 99% CI = 0.161 bits < mean < 0.201 bits). The effect of Time Windows was significant, $F(2,29.21) = 13.13$, $p < .001$. Changes in AMI across time windows were not linear; however, there was not a significant difference between any two Time Windows (Window 1 mean = 0.16 bits, 99% CI = 0.118 bits < mean < 0.206 bits, Window 2 mean = 0.22 bits, 99% CI = 0.199 bits < mean < 0.231 bits, Window 3 mean = 0.20 bits, 99% CI = 0.184 bits < mean < 0.224 bits). The Time Windows × Sickness Groups interaction was significant, $F(2,28) = 11.80$, $p < .001$ (Fig. 5B). For the Well group, coupling was low at Window 1, increased significantly by Window 2, and remained stable at Window 3 (Well Group: Window 1 mean = 0.125 bits, 99% CI = 0.077 bits < mean < 0.173 bits, Window 2 mean = 0.212 bits, 99% CI = 0.192 bits < mean < 0.232 bits, Window 3 mean = 0.220 bits, 99% CI = 0.200 bits < mean < 0.240 bits). By contrast, for the Sick group, coupling did not change across Time Windows (Window 1 mean = 0.199 bits, 99% CI = 0.127 bits < mean < 0.271 bits, Window 2 mean = 0.217 bits, 99% CI = 0.189 bits < mean < 0.245 bits, Window 3 mean = 0.188 bits, 99% CI = 0.160 bits < mean < 0.216 bits).

4. Discussion

We measured body sway in standing women during prolonged, complex oscillation of the illuminated environment. Following exposure to oscillation of the illuminated environment, 30% of participants reported motion sickness. Ratings of symptom severity were greater among participants who reported motion sickness than among participants who did not. We documented nonlinear coupling of body sway with complex, sum-of-sines motion of the illuminated environment. In addition, we identified postural precursors of motion sickness during exposure to complex oscillation of the illuminated environment. Postural precursors of motion sickness were found in the spatial magnitude of body sway, replicating previous studies. In a novel effect the Well and Sick groups differed in the degree to which body sway was temporally coupled with the stimulus motion.

4.1. Motion sickness incidence and symptom severity

The incidence of motion sickness, the severity of reported symptoms, and the duration of exposure were representative of women in a moving room (Koslucher et al., 2015). Exposure to sum-of-sines motion increased symptom severity in both Well and Sick groups. Yet, at post-exposure, symptom severity was greater for the Sick group than for the Well group. The finding that imposed motion can increase symptoms among both Well and Sick groups is compatible with previous studies (e.g., Stanney et al., 1998). This effect underscores the value of using independent measures of motion sickness incidence and severity, and also underscores the idea that measures of symptom severity, such as the SSQ, are not reliable indicators of motion sickness incidence (Stoffregen, 2011).

4.2. Body sway independent of motion sickness

We observed within-participants effects of our manipulations that were independent of motion sickness status, that is, effects of room motion that were general across our sample. Perhaps most conspicuous is the fact that the main effect of Time Windows was significant for each of our dependent variables. Prolonged exposure to sum-of-sines room motion yielded changes in the spatial magnitude (positional variability) and fractality of body sway, and to the strength of coupling between room motion and the body. Similarly, the main effect of body axis was significant for each dependent variable. In the AP axis, body movement was greater, exhibited greater fractality, and was more strongly coupled with room motion than in the ML axis. These effects are straightforward results of the fact that room motion was confined to the AP axis. In addition, the multifractality of movement was greater for the head than for the torso. This effect appears to be novel, both for women and in general, and indicates that control of movement multifractality can differ between body segments.

During exposure to sum-of-sines room motion, statistically significant within-participants effects on AMI revealed that body sway can become coupled with imposed oscillations that are complex, and that coupling can evolve over time during exposure. These effects underscore the existence of robust coupling between the complex dynamics of human movement and the sum-of-sine dynamics of imposed motion of the illuminated environment, extending effects reported when imposed motion was inertial (Varlet et al., 2015). Taken together, such effects underscore the need for models (both mathematical and theoretical) that are robust to sum-

of-sines movement (cf. Haken, Kelso, & Bunz, 1985).

4.3. Body sway in relation to motion sickness

For positional variability, we found a statistically significant Sickness Groups × Time Windows interaction (Fig. 5A), revealing that positional variability increased over time for the Sick group, but not for the Well group. This interaction replicated several previous studies that have included both women and men (e.g., Bonnet et al., 2006; Koslucher et al., 2014; Stoffregen et al., 2010).

An important novelty of the present study was our analysis of coupling between postural activity and oscillatory motion of the illuminated environment, in relation to motion sickness. During exposure to sum-of-sines room motion, the temporal evolution of coupling differed between the Well and Sick groups (Fig. 5B).

It is useful to compare Fig. 5A and B, which show that the time course of group-specific responses differed between absolute movement (positional variability) and coupling (AMI). For positional variability, the Well and Sick groups diverged over time during exposure. By contrast, coupling of body sway with room motion differed between the Well and Sick groups at the beginning of room motion (i.e., in Window 1) as well as at the end (Window 3). The differences across dependent measures underscore the qualitative difference between measures of body sway relative to the earth (positional variability) versus body sway relative to imposed motion (coupling). The results suggest that our female participants could modulate the strength of coupling independent of the positional variability and multifractality of sway. If we interpret the behavior of the Well group as reflecting stable control, then the results also suggest that the immediate strong coupling between body sway and imposed motion observed in the Sick group constituted a form of postural instability. A full interpretation of this effect would require measures of coupling in the context of imposed inertial motion: It may be that susceptible individuals inappropriately couple their sway to any imposed motion (i.e., inertial or optical), or it could be the susceptible individuals preferentially couple their sway only relative to the illuminated environment.

The postural instability theory of motion sickness etiology (Riccio & Stoffregen, 1991) predicts that prior to the onset of subjective symptoms of motion sickness postural activity should differ between persons who are susceptible to motion sickness and those who are not susceptible. We confirmed the existence of postural precursors of motion sickness in the spatial magnitude of body sway. The principal contribution of the present study is our demonstration that postural precursors of motion sickness occurred between body sway and the temporal dynamics of oscillatory motion of the illuminated environment, and that these precursors differed between participants who (later) reported motion sickness and those who did not. The present results constitute a qualitatively novel confirmation of the postural instability theory of motion sickness.

5. Conclusion

We measured women's body sway during exposure to imposed oscillatory motion of the illuminated environment. Exposure to sum-of-sines motion of the illuminated environment induced motion sickness in some participants. We identified postural precursors of motion sickness during exposure to sum-of-sines motion. Precursors were identified in the spatial magnitude of body sway, but also in the coupling of body sway with the sum-of-sines stimulus. This latter result provides the first confirmation of a prediction of Riccio and Stoffregen (1991) that postural precursors of motion sickness should include variations in coupling between body sway and the temporal dynamics of imposed environmental motion. This effect offers a qualitatively new type of confirmation of the general prediction that postural instability should precede the subjective symptoms of motion sickness. Our results motivate the evaluation of coupling as a postural precursor of motion sickness in terrestrial vehicles, such as automobiles, and on ships at sea (cf. Varlet et al., 2015).

Our sample comprised only women. In previous studies, it has been found that the postural precursors of motion sickness differ between women and men (e.g., Koslucher et al., 2014, 2016; Munafo et al., 2017). Thus, we should not assume that the results of the present study would apply to men. Our results directly motivate future research to determine whether motion sickness in men is preceded by distinctive patterns of coupling between stimulus motion and postural activity.

Analytic techniques, such as AMI, make it possible to quantify complex coupling of body sway with the temporal dynamics of complex imposed visual motion. In the present study, we demonstrated the existence of such coupling, and found that it differed between participants who (later) reported motion sickness, and those who did not. These findings suggest that it might be possible to evaluate coupling of body movement with the complex dynamics of nauseogenic motion in non-laboratory situations. Potential examples include the unpredictable dynamics of many interactive technologies (such as head-mounted displays, which update displayed information based upon user-generated head movements), and the aperiodic, 6-degree-of-freedom oscillations that characterize motion of ships at sea (cf. Varlet et al., 2015). Motion sickness naturally occurs outside the laboratory, such that the assessment of coupling between bodily movement and complex natural motions simultaneously can advance our understanding of motion sickness in both theoretical and applied contexts.

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Appendix A. Supplementary data

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