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# Frequency Characteristics of Visually Induced Motion Sickness

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**Objective:** The aim of this study was to explore the frequency response of visually induced motion sickness (VIMS) for oscillating linear motion in the fore-and-aft axis.

**Background:** Simulators, virtual environments, and commercially available video games that create an illusion of self-motion are often reported to induce the symptoms seen in response to true motion. Often this human response can be the limiting factor in the acceptability and usability of such systems. Whereas motion sickness in physically moving environments is known to peak at an oscillation frequency around 0.2 Hz, it has recently been suggested that VIMS peaks at around 0.06 Hz following the proposal that the summed response of the visual and vestibular self-motion systems is maximized at this frequency.

**Methods:** We exposed 24 participants to random dot optical flow patterns simulating oscillating fore-and-aft motion within the frequency range of 0.025 to 1.6 Hz. Before and after each 20-min exposure, VIMS was assessed with the Simulator Sickness Questionnaire. Also, a standard motion sickness scale was used to rate symptoms at 1-min intervals during each trial.

**Results:** VIMS peaked between 0.2 and 0.4 Hz with a reducing effect at lower and higher frequencies.

**Conclusion:** The numerical prediction of the "crossover frequency" hypothesis, and the design guidance curve previously proposed, cannot be accepted when the symptoms are purely visually induced.

**Application:** In conditions in which stationary observers are exposed to optical flow that simulates oscillating fore-and-aft motion, frequencies around 0.2 to 0.4 Hz should be avoided.

**Keywords:** simulator sickness, frequency, fore-and-aft motion, stimulus parameters

## INTRODUCTION

Simulation and virtual reality technologies are increasingly used for research, training, design, and entertainment (Stanney, 2002). The ability to immerse users in interactive synthetic environments offers some distinct advantages in that it provides a controlled and safe environment in which individuals can repeatedly be exposed to scenarios that in real life are too costly, dangerous, or simply nonexistent. The ultimate acceptability and usability of these technologies is, however, seriously limited by the fact that they are often reported to induce visually induced motion sickness (VIMS), which is characterized by signs and symptoms such as nausea, headache, fatigue, and drowsiness (Bos, 2011b; Kennedy, Hettlinger, & Lilienthal, 1990; Lawson, Graeber, Mead, & Muth, 2002; Wilson, 1996). VIMS significantly interferes with the intended goals for which these technologies are used. In the context of training, it may hinder the learning process, prevent individuals from participating in the training, limit the length of time for which training can occur, and lead to negative transfer of training (see also Kennedy et al., 1990). In the wider context of entertainment, VIMS has been reported not only when head-mounted displays have been used but also when computer games have been played with the use of stand-alone monitors, along with the widespread occurrence during some TV programs and cinema films (see Howarth, 2008). Thus, there is a strong practical motivation to gain a better understanding of the underlying causes of VIMS.

VIMS is a form of motion sickness that may occur when stationary observers are exposed to moving visual images. Provided certain conditions are met (see Dichgans & Brandt, 1978), moving visual images can induce an illusory sensation of self-motion, known as *vection* (Tschermak, 1931). When visual motion is unaccompanied by physical

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self-motion, the discrepancy between the self-motion cues provided by the visual system (i.e., vection) and the lack of consistent signals from the vestibular and somatosensory systems is thought to underlie the generation of VIMS (Bles, Bos, de Graaf, Groen, & Wertheim, 1998; Hettinger, 2002; Hu & Stern, 1998; Oman, 1991; Reason & Brand, 1975).

Motion environments, including simulators, virtual environments, and commercially available video games that create an illusion of self-motion, are frequently reported to induce VIMS (Lawson et al., 2002) and may result in participant dropout rates as high as 50% (Reed, Diels, & Parkes, 2007). To be able to predict the incidence and severity of VIMS, one first needs to identify contributing factors. More specifically, given that VIMS is visually induced, a logical first step would be the identification of the visual stimulus characteristics that are most conducive to VIMS. This approach has previously been shown to be fruitful with regard to seasickness. The “motion sickness dose value” for predicting seasickness on the basis of the vertical motion of vessels (British Standards Institution, 1987) has been shown to be in accordance with conditions that cause sickness at sea and is therefore of practical value in minimizing motion sickness (Griffin, 1990). Ultimately, the development of a “cyber sickness dose value” (So, Ho, & Lo, 2001) may also prove to be instrumental in minimizing the occurrence of VIMS in synthetic environments.

For true motion sickness, the important physical characteristic of the provocative motion is predominantly the frequency and, to a lesser extent, the acceleration or amplitude of the motion (Griffin, 1990; Guignard & McCauley, 1990). In laboratory studies involving both linear and angular oscillation, motion sickness peaks at a frequency of approximately 0.2 Hz, whereas motion at other frequencies produces little or no sickness (Bos & Bles, 1998; Donohew & Griffin, 2004; Golding, Finch, & Stott, 1997; Golding & Markey, 1996; Golding, Phil, Mueller, & Gresty, 2001; Griffin, 1990; Guignard & McCauley, 1990; O’Hanlon & McCauley, 1974). This finding is consistent with what is known about the provocative motion profiles of transport systems associated

with motion sickness, including ships, trains, aircraft, cars, and camels (e.g., Förstberg, Andersson, & Ledin, 1998; Golding, Bles, Bos, Haynes, & Gresty, 2003; Guignard & McCauley, 1990; Lawther & Griffin, 1988).

The dominant frequency of oscillation of the visual scene or image may also play an important role in the generation of VIMS (Kennedy, Berbaum, Dunlap, & Hettinger, 1996), and, like true motion sickness, imposed visual motion at a frequency around 0.2 Hz has been suggested to be most provocative (Hettinger, Berbaum, Kennedy, Dunlap, & Nolan, 1990). However, until recently (Diels & Howarth, 2006; Golding et al., 2009), there has been no published data to substantiate this specific frequency dependence of VIMS. Golding et al. (2009) showed that visual off-vertical-axis rotation was significantly more provocative at 0.2 Hz than at lower or higher frequencies, as also observed with real motion. Parker and colleagues (Duh, Parker, Phillips, & Furness, 2004; Parker, Duh, Phillips, & Furness, 2001), on the other hand, hypothesized VIMS to peak at a much lower frequency. Support for their hypothesis was provided in a study employing concurrent visual and vestibular stimulation (Duh et al., 2004) in which they evaluated the frequency response of the visual component by evaluating postural balance while a visual scene was oscillating. They concluded that “simulator sickness may be most readily evoked by visual-vestibular conflicts at the ‘cross-over frequency’—the frequency at which the summed response from the visual and vestibular self-motion systems is maximum” (p. XX[AQ: 1]), which they stated to be around 0.06 Hz. However, there exist no published data to substantiate their hypothesis for the situation that is found far more often, in which stationary observers are exposed to moving images, such as are encountered in fixed-base simulators as well as in the consumer context of cinema and television. It is these circumstances that are relevant if one wishes to provide a “design guidance curve that indicates the frequency range of simulated motion that is likely to evoke simulator or virtual reality sickness” (Duh et al., 2004, p. XX[AQ: 2]).

In this article, we report two experiments designed to explore the frequency dependence

of VIMS. Studies into VIMS tend to expose observers to visual rotation about a vertical axis (e.g., Bubka & Bonato, 2003; Duh et al., 2004; Golding et al., 2009), but rotation has, however, only a limited role in the normal locomotion of the human observer. The principal motion components that occur during normal (simulated) locomotion of a person are generally translations and, more specifically, are usually translation along the line of sight in the forward direction. Accordingly, in this study, stationary observers were exposed to random dot radial optical flow patterns simulating oscillating linear motion in the fore-and-aft axis. The starting point in the first experiment was Duh et al.'s (2004) hypothesis, and we investigated VIMS in the lower frequency range—0.025, 0.05, 0.1, and 0.2 Hz—around the hypothesized maximum. Following the failure to obtain results consistent with this hypothesis, a second experiment was then conducted to extend the frequency range to 1.6 Hz. For brevity, the methods and results for Experiments 1 and 2 are presented together.

## METHOD

### Participants

Following approval by the Loughborough University Ethical Advisory Committee, 24 participants gave their informed consent to participate in the study. The first experiment included 12 participants (7 male and 5 females) with a mean ( $\pm$  *SD*) age of 29.8 ( $\pm$  5.8) years. In the second experiment, another 12 individuals (5 female and 7 male) with a mean ( $\pm$  *SD*) age of 24.6 ( $\pm$  2.8) years participated. All participants had intact vestibular function, were not receiving any medication, and had normal or corrected-to-normal vision. The mean Motion Sickness Susceptibility Questionnaire (MSSQ) percentile score for the participants in both experiments was 44%, indicating the sample to be slightly less susceptible to motion sickness than the normal population (Golding, 1998).

### Apparatus and Stimuli

The experiments took place in a dark room, and each participant had his or her head stabilized by means of a head-and-chin rest (Figure 1). The images were viewed binocularly from a fixed

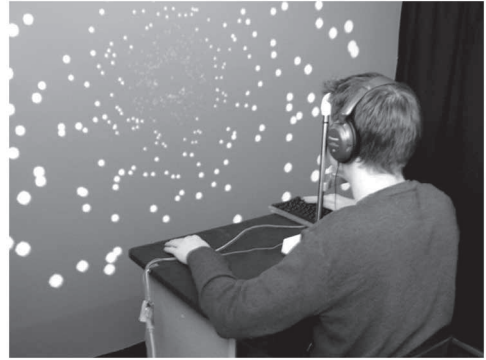


Figure 1. Experimental setup.

viewpoint at a distance of 90 cm from the screen. To occlude the edges of the screen and other peripheral features, we had participants wear goggles, which limited the visual field to  $65^\circ$  (horizontal)  $\times$   $59^\circ$  (vertical) of angle. Acoustic localization cues were masked by pink noise (75 dB) transmitted to earphones. In addition, auditory alerting bleeps (500, 750, and 1000 Hz at 100 dB) were played at random intervals throughout the exposure duration. Communication with the participants during exposure was via a microphone. To control for eye movements, we instructed participants to fixate a red dot ( $0.57^\circ$  of visual angle) projected at eye height in the center of the screen. By means of an infrared camera aimed at the participants' face, instruction compliance was monitored in real time by the experimenter.

We generated the stimuli in real time with a frame rate of 60 Hz using Matlab (Version 6.5) running on a Dell GX computer fitted with a Matrox Millennium P750 graphics card (64 Mb). The images were back-projected onto a tangent screen ( $190 \times 145$  cm) with a Hitachi CP-X958W/E projector ( $1,024 \times 768$  pixels). The display consisted of 500 white dots with a luminance of  $10.82 \text{ cd/m}^2$  randomly positioned on a black background of  $0.35 \text{ cd/m}^2$ . Dot velocity and size varied exponentially as a function of their simulated location in depth (Andersen & Braunstein, 1985). Dot size at the eye ranged from  $0.22^\circ$  at the middle to  $2.97^\circ$  at the periphery. For technical reasons, there were no dots at the very center of the visual scene, and as a consequence, there was a black disc subtending

8.75° of visual angle. All participants were exposed to random dot optical flow patterns simulating oscillating linear motion in the fore-and-aft axis.

In Experiment 1, participants were exposed to oscillating linear motion at the frequencies of 0.025, 0.05, 0.1, and 0.2 Hz. In Experiment 2, the frequencies employed were 0.2, 0.4, 0.8, and 1.6 Hz. At each frequency, the stimuli oscillated with a peak angular velocity of 34°/s, which pertains to a perceived peak velocity of 0.97 m/s. Since peak optical velocity was held constant in this study, displacement and acceleration covaried with frequency. The appearance to the participant was similar to the opening sequence of the TV program *Star Trek*, or the early MS Windows “starfield” screensaver, but with back-and-forth motion rather than forward motion alone.

### Experimental Design and Procedure

Participants were exposed to each of the conditions for 20 min, and we separated trials by at least 24 hr to limit any habituation to the stimulus (Hill & Howarth, 2000). To avoid possible circadian rhythm effects, we held each trial at the same time of day. A repeated-measures design was used, and to minimize order effects, we balanced the sequence in which the conditions were presented using a Latin square design. Prior to the first session, participants received written and verbal instructions. When they indicated that they fully understood the task, the experiment commenced. They were instructed to focus on the central fixation dot for the duration of the experiment.

### Metrics

Motion sickness symptoms were assessed with the Simulator Sickness Questionnaire (SSQ; Kennedy, Lane, Berbaum, & Lilienthal, 1993). Measures of interest were the change (postexposure minus preexposure score) in the SSQ total scores and the change in SSQ subscores (Kennedy et al.’s [1993] N, O, and D scales). In addition, participants rated the severity of their motion sickness every minute on the standard sickness scale produced by Bagshaw and Stott (1985; 1 = *no symptoms*, 2 = *mild*

*symptoms but no nausea*, 3 = *mild nausea*, 4 = *moderate nausea*). The experiment was stopped when a malaise rating of 4 was reached or after 20 min, whichever was sooner. Participants who reached a malaise rating of 4, and stopped, before 20 min were assigned continuation values of 4. All the participants were initially symptom free, and the measures of interest were (a) the time for participants to first report a sickness rating of 2 (S2), (b) the time to first report a rating of 3 (S3), (c) the maximum sickness rating, and (d) the sum of the sickness ratings across the 20-min exposure duration (accumulated sickness rating). If no symptoms were reported, an accumulated sickness rating and symptom onset time of 21 were recorded.

We assessed the occurrence of vection post-exposure by asking participants the following questions: “Whilst watching the moving images, did you get the feeling of motion? Did you experience a compelling sensation of self-motion as though you were actually moving?” Vection was defined as a compelling feeling of self-motion, such as “the feeling you get when a train moves next to you and you mistake it for your own motion.” To ensure that participants differentiated between object and self-motion, prior to the first session, they were exposed to oscillating roll motion (0.125 Hz; peak-to-peak amplitude of 120°) until a compelling sensation of self-motion was reported. This sensation typically occurred after about 15 s.

### Data Analysis

Data analysis was performed with the software package SPSS (Version 13). The data were analyzed twice. In the first analysis, we considered the effects of session order, and because none was identified, the analyses were repeated with the assumption that no session order effect existed. Since the motion sickness scales were not at an interval level of measurement, the data collected by using these scales were analyzed with the use of a nonparametric approach. The symptom onset time and accumulated sickness rating distributions were heavily negatively skewed because of the large number of participants who reached the 20-min maximum exposure without reporting any symptoms. To minimize the number of ties, a



**TABLE 1:** Number of Participants Reaching Each Sickness Rating Stage Before the 20 Min Cutoff for Each Frequency in Experiments 1 and 2

Sickness Rating	Frequency (Hz)							
	Experiment 1				Experiment 2			
	0.025	0.05	0.1	0.2	0.2	0.4	0.8	1.6
1. No symptoms	12/12	12/12	12/12	12/12	12/12	12/12	12/12	12/12
2. Mild symptoms but no nausea	5/12	5/12	7/12	8/12	10/12	9/12	8/12	6/12
3. Mild nausea	0/12	0/12	2/12	3/12	2/12	4/12	2/12	1/12
4. Moderate nausea	0/12	0/12	0/12	2/12	2/12	1/12	0/12	0/12

similar approach to that previously performed by Golding et al. (2003) was adopted. This approach involved the fact that different SSQ total severity scores were observed between the four conditions in some participants, indicating certain conditions to be more provocative to them than others. SSQ total severity scores for such participants were then employed to break ties. If SSQ total severity scores at 20 min were the same for different conditions, the results were accepted as tied. Because of the abnormal distribution of the data, we tested differences between conditions for significance using non-parametric Wilcoxon signed ranks tests.

**RESULTS**

**Vection**

In Experiment 1, 11 out of 12 participants experienced vection in the direction opposite that of the display motion in all four conditions. The remaining participant did not experience any vection during 0.025 Hz oscillation but did so during oscillation at the other frequencies. In the second experiment, 3 participants did not report any vection during 0.8 Hz oscillation, whereas 1 participant did not report vection during 1.6 Hz oscillation.

**Sickness Ratings**

Table 1 shows the number of participants reaching each sickness rating stage before the 20-min cutoff. It can be seen that in Experiment 1, an increase in frequency produced greater motion sickness. None of the participants

reported nausea (sickness rating of 3) during 0.025 Hz and 0.05 Hz oscillation. During 0.2 Hz oscillation, however, 2 participants asked to terminate the experiment before the maximum 20-min time cutoff (at Minute 17 and 18), having reached a sickness rating of 4. The results of Experiment 2 show the reverse in that an increase in frequency beyond 0.2 Hz resulted in reduced motion sickness. In the second experiment, 2 participants had to terminate the experiment during 0.2 Hz oscillation after 6 and 8 min; one of these participants also requested to stop the experiment during 0.4 Hz oscillation after 6 min.

**Accumulated Sickness Rating**

The mean accumulated sickness ratings for each frequency are shown in Figure 2a. In Experiment 1, an increase in accumulated sickness rating was observed with increasing frequency. The accumulated rating during 0.2 Hz oscillation was significantly higher than during 0.05 Hz oscillation ( $Z = 2.524, p = .012$ ) and 0.025 Hz oscillation ( $Z = 2.240, p = .025$ ). The rating during 0.1 Hz oscillation was significantly higher than that of the 0.025 Hz oscillation ( $Z = 2.384, p = .017$ ). The other differences seen were not statistically significant. Beyond 0.2 Hz, as evaluated in Experiment 2, however, participants reported lower sickness ratings with increasing frequency. Post hoc comparisons revealed that the accumulated sickness rating during 0.2 Hz oscillation was significantly higher than during 1.6 Hz oscillation ( $Z = -2.158, p = .031$ ).

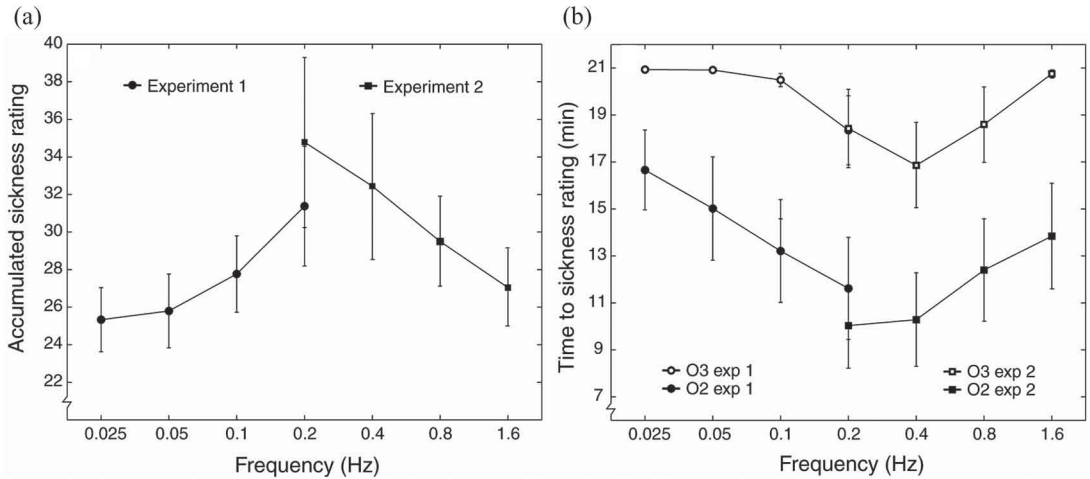


Figure 2. (a) Mean ( $\pm$  SEM) accumulated sickness rating and (b) mean ( $\pm$  SEM) time to sickness ratings of 2 (O2) and 3 (O3) as a function of frequency for Experiments 1 and 2.

### Symptom Onset Time

Figure 2b shows the mean times to achieve sickness ratings of 2 (*mild symptoms but no nausea*) and 3 (*mild nausea*). Since both measures failed to pass the tests for normality, nonparametric statistics were used. In Experiment 1, the time to achieve sickness ratings of 2 and 3 both became shorter with higher frequencies. Post hoc analysis showed that time to a sickness rating of 2 during 0.2 Hz oscillation was significantly shorter than during either 0.05 Hz oscillation ( $Z = -2.449, p = .014$ ) or 0.025 Hz oscillation ( $Z = -2.668, p = .008$ ). Time to a sickness rating of 2 was significantly shorter during 0.1 Hz oscillation compared with oscillation at 0.025 Hz ( $Z = -2.670, p = .008$ ). Time to a sickness rating of 3 during 0.1 Hz oscillation was significantly shorter than during 0.025 Hz oscillation ( $Z = -2.124, p = .034$ ). No other differences were found to be significant. As for the accumulated sickness ratings, in Experiment 2, the same effect was observed whereby time to achieve a sickness rating of 2 was shortest during 0.2 Hz oscillation and became consistently longer with increasing frequencies above this frequency. Time to achieve a sickness rating of 3 was shortest during 0.4 Hz oscillation and became longer with frequencies both below and above 0.4 Hz. Because of the abnormal distribution of time to sickness

ratings of both 2 and 3, nonparametric tests were employed. Post hoc comparison showed that time to a sickness rating of 2 during 1.6 Hz oscillation was significantly longer than during 0.4 Hz oscillation ( $Z = 2.123, p = .031$ ). No other differences were found to be significant.

### SSQ

Table 2 shows the mean (SEM) SSQ total scores and the SSQ N, O, D subscores for each frequency for Experiment 1 and 2. In line with the other metrics in Experiment 1, SSQ total scores and subscores consistently increased with increasing frequency, with the highest SSQ scores observed during 0.2 Hz oscillation. Post hoc analysis showed that the SSQ total score and N subscore were significantly higher during 0.1 Hz than during 0.025 Hz oscillation ( $Z = 2.173, p = .030$ ;  $Z = 2.692, p = .007$ , respectively). No other differences were found to have reached statistical significance. In Experiment 2, the SSQ total scores showed a steady decrease with increasing frequency. However, no clear trend was observed in the SSQ subscores. Post hoc comparisons revealed no differences to have reached statistical significance.

### DISCUSSION

This study was conducted to explore the frequency dependence of VIMS for linear oscillatory



**TABLE 2:** Mean (SEM) SSQ Total Scores and N, O, D Subscores for Each Frequency

Sickness Rating	Frequency (Hz)								
	Experiment 1				Experiment 2				
	0.025	0.05	0.1	0.2	0.2	0.4	0.8	1.6	
Total	19.0 (5.0)	25.6 (7.9)	35.5 (10.9)	36.8 (12.8)	17.3 (5.4)	15.0 (3.1)	14.9 (3.7)	14.6 (3.1)	
N	12.7 (3.9)	19.9 (8.3)	31.0 (8.9)	33.4 (13.5)	13.9 (6.9)	14.7 (5.2)	11.3 (3.8)	6.1 (1.9)	
O	19.0 (5.3)	25.3 (7.1)	28.4 (9.2)	27.8 (9.2)	17.2 (4.7)	13.8 (1.7)	16.5 (4.1)	19.3 (3.4)	
D	17.4 (5.5)	19.7 (6.3)	34.8 (13.7)	37.1 (13.7)	12.7 (5.5)	8.9 (4.3)	8.9 (3.9)	10.1 (5.3)	

Note. SSQ = Simulator Sickness Questionnaire (Kennedy, Lane, Berbaum, & Lillenthal, 1993).

motion in the fore-and-aft axis, and within the limits of our testing, 0.025 to 1.6 Hz, the level of motion sickness was maximal within the frequency range of 0.2 to 0.4 Hz. Although the SSQ total scores, accumulated sickness rating, and time to a sickness rating of 2 all indicated motion sickness to peak at 0.2 Hz, time to a sickness rating of 3 indicated 0.4 Hz oscillation to be most provocative (see Figure 2). The largest number of participants reaching a sickness rating of 2 was at a frequency of 0.2 Hz, but the largest number of participants reaching a sickness rating of 3 was at a frequency of 0.4 Hz.

The frequency of maximum nauseogenicity would appear, from our data, to lie between 0.2 Hz and 0.4 Hz, and it is clear that the results do not lend support to the hypothesis proposed by Duh et al. (2004), according to which VIMS is expected to peak at a frequency of around 0.06 Hz. This frequency is the value at which the visual and vestibular tuning functions described by Duh et al. cross and that they expect to have the maximum nauseogenicity. However, the “crossover frequency” will change if these functions are not weighted equally, and our results would suggest that they should not be.

The striking similarity in frequency dependence between true motion sickness and VIMS observed in the present study lends support for Hettinger et al.’s (1990) proposition that both true and visual motion at a frequency around 0.2 Hz most readily evokes motion sickness. In this context, it is worth examining how theories of motion sickness deal with its frequency dependence.

Benson (1988) proposed that during low-frequency oscillation, motion sickness occurs because of a phase error in motion signals from the otoliths and somatosensory receptors (a suggestion first put forward by Mach, 1875). Von Gierke and Parker (1994) further elaborated on this proposal by suggesting a potential conflict not only between the otoliths and somatosensory receptors but also between the otoliths and the visceral graviceptors. Stott (1986), on the other hand, suggested an intraotolith conflict at low-frequency oscillations. The central nervous system expects the otoliths’ overall output to average 1G across periods longer than approximately 0.5 s. Unlike walking or running, which occur at higher frequencies (>1 Hz), this expectation is violated during sustained low-frequency oscillations. However, as there is no direct involvement of the vestibular system, other than its being silent, neither of these hypotheses would appear to be able to explain the frequency response of VIMS on the basis of the vestibular signals, apart from the fact that the *expected* signals are absent.

An alternative explanation for the frequency tuning of motion sickness as well as its etiology is provided by the postural instability theory (Riccio & Stoffregen, 1991), according to which motion sickness occurs only in conditions of prolonged postural instability. The frequency dependence of motion sickness is explained by the overlap between imposed stimulus motion and postural sway, resulting in waveform interference, which would be greatest in the area of maximum overlap at around

0.2 Hz (Stoffregen & Smart, 1998). However, whereas several studies provide support for this theory, there are numerous findings that appear difficult to reconcile with this theory. These were recently reviewed by Bos (2011a) and include observations of negative correlations between postural instability and motion sickness, decreased instability over time accompanied by increases as opposed to decreases in sickness, the fact that Ménière patients suffer from motion sickness at night while lying still in bed, and that individuals without functioning organs of balance do not get sick from motion despite the fact that they generally show more postural instability than do healthy individuals.

Irrespective of how exactly instability is defined (see Riccio & Stoffregen, 1991), these examples illustrate that there are clearly conditions in which motion sickness occurs in the absence of any postural instability, which argues against the theory's basic premise that postural instability is a necessary and sufficient condition for motion sickness to occur. As pointed out by Bos (2011a), postural stability and motion sickness may be related via a common mechanism, but this relation does not imply causality.

Currently, the most promising theoretical framework to explain the frequency dependence of motion sickness appears to be the subjective vertical conflict model (Bles et al., 1998; Bos, Bles, & Groen, 2008). Within the subjective vertical conflict model, relevant visual and vestibular sensory signals pass through a low pass filter with a time constant of 5 s (= 0.2 Hz). At the same time, the equivalent "efference copy" signals (so-called internal model) pass through a filter with the same frequency characteristics, before matching with the processed sensory signals in a comparator. Because of filter characteristics, a significant mismatch is detected by the comparator at 0.2 Hz, and an output is given that initiates motion sickness (Bos et al., 2008). At frequencies both below and above 0.2 Hz, the degree of mismatch reduces, as ultimately reflected in lower motion sickness levels.

One limitation of the current experiments was that velocity was held constant across frequencies,

and thus, acceleration and displacement covaried with frequency. Although an effect of displacement and acceleration on motion sickness cannot be ruled out, the consistent frequency effect found with both constant (Duh et al., 2004) and varying (Lin, Razzaque, & Parker, 2005) peak velocity during rotational motion suggests the frequency dependence of VIMS to be largely independent of displacement and acceleration. Furthermore, if motion sickness were dependent solely on the peak velocity of the stimulus, the graph relating motion sickness to frequency would have a gradient of zero. Alternatively, if motion sickness were governed simply by acceleration, motion sickness and frequency would have shown a monotonic relationship. This result was clearly not the case, and it appears that, as for true motion sickness, the principal physical characteristics of provocative motion include the frequency (or spectrum in the case of complex motions) and, to a lesser extent, the intensity (i.e., acceleration, amplitude) of the motion.

Nevertheless, it is acknowledged that future research will benefit from the independent manipulation of both frequency and intensity to further enhance understanding of visual stimulus characteristics and VIMS. Considerations should also be given to the use of optical flow patterns that allow for distance perception containing familiar objects as opposed to abstract dots. However, whereas the stimuli used in the present study may be less powerful than more realistic stimuli, there is no reason to believe that the tuning effect observed would be different.

In summary, it has been previously argued that designers need to know the frequency response of the visual stimulus provided to viewers of displays that have the potential to cause VIMS. In our experiment, which involved participants viewing a starlike pattern of stars, the maximum level of VIMS was found in the region of 0.2 to 0.4 Hz, with higher and lower frequencies proving less powerful in generating symptoms. Thus the numerical prediction of the "crossover frequency" hypothesis, and the design guidance curve previously proposed, cannot be accepted when the symptoms are purely visually induced.

## KEY POINTS

- Visually induced motion sickness peaks between 0.2 and 0.4 Hz, with a reducing effect at lower and higher frequencies.
- The numerical prediction of the “crossover frequency” hypothesis cannot be accepted when the symptoms are purely visually induced.
- In conditions in which stationary observers are exposed to dynamic visual displays, optical flow that simulates oscillating fore-and-aft motion in the frequency range of 0.2 to 0.4 Hz should be avoided.

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