Frequency characteristics of visually induced motion sickness

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Abstract

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:** 24 participants were exposed to random dot optical flow patterns simulating oscillating fore-and-aft motion within the frequency range of 0.025 – 1.6 Hz. Before and after each 20 min exposure VIMS was assessed using the SSQ. Also, a standard motion sickness scale was used to rate symptoms at one minute 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:** Under conditions in which stationary observers are exposed to optical flow which simulates oscillating fore-and-aft motion at frequencies around 0.2-0.4 Hz should be avoided.

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**Short title version:** Frequency Characteristics of VIMS

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

**Précis:** Effect of oscillating optokinetic fore-and-aft motion on visually induced motion sickness
INTRODUCTION

Simulation and Virtual Reality (VR) 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 non-existent. 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 characterised by signs and symptoms such as nausea, headache, fatigue, and drowsiness (Bos, 2011a; Kennedy et al., 1990; Lawson et al., 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 may 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 using stand-alone monitors, along with the widespread occurrence during some TV programmes 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 and 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 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 (Reason and Brand, 1975; Oman, 1982; Bles et al., 1998).

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 drop-out rates as high as 50% (Reed et al., 2007). In order to be able to predict the incidence and severity of VIMS, one first needs to identify contributing factors. More specifically, considering VIMS to be 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 based on the vertical motion of vessels (BSI, 1987) has been shown to be in accordance with conditions that cause sickness at sea and is therefore of practical value in minimising motion sickness (Griffin, 1990). Ultimately, the development of a ‘Cyber Sickness Dose Value’ (So et al., 2001) may also prove to be instrumental in minimising 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 and McCauley, 1990). In laboratory studies using 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 and Bles, 1998; Donohew and Griffin, 2004; Golding and Markey, 1996; Golding et al., 1997, 2001; Griffin, 1990; Guignard and McCauley, 1990; O’Hanlon and McCauley, 1974). This 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. Guignard and McCauley, 1990; Lawther and 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 et al., 1996), and, like true motion sickness, imposed visual motion at a frequency around 0.2 Hz has been suggested to be most provocative (Hettinger et al., 1990). However, until recently (Diels and 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 (OVAR) was significantly more provocative at 0.2 Hz than at lower or higher frequencies, as also observed with real motion. Parker and co-workers (Duh et al., 2004; Parker et al., 2001), on the other hand, hypothesised 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 whilst 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”, which they stated to be around 0.06 Hz. However, there exists no published data to substantiate their hypothesis for the situation which 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 which 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).
We report here 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 and 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 hypothesis, and we investigated VIMS in the lower frequency range: 0.025, 0.05, 0.1, and 0.2 Hz, around their hypothesised 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 experiment 1 and 2 are presented together.

METHODS

Participants

Following its 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 (± SD) age of 29.8 (± 5.8) years. In the second experiment, a further 12 individuals (5 female and 7 male) with a mean (± SD) age of 24.6 (± 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 their head stabilised by means of a head/chin rest (figure 1). The images were viewed binocularly from a fixed viewpoint at a distance of 90 cm from the screen. To occlude the edges of the screen and other peripheral features, participants wore goggles, which limited the visual field to 65º (h) x 59º (v) of angle. Acoustic localisation 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, participants were instructed to fixate a red dot (0.57º of visual angle) projected at eye height in the centre of the screen. By means of an infrared camera aimed at the participants’ face, instruction compliance was monitored in real-time by the experimenter.

![Figure 1: Experimental setup.](image)

The stimuli were generated 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 Millenium P750 graphics card.
The images were backprojected onto a tangent screen (190 cm x 145 cm) with a Hitachi CP-X958W/E projector (1024 x 768 pixels). The display consisted of 500 white dots with a luminance of 10.82 cd/m² randomly positioned on a black background of 0.35 cd/m². Dot velocity and size varied exponentially as a function of their simulated location in depth (Andersen and Braunstein, 1985). Dot size at the eye ranged from 0.22° at the middle to 2.97° at the periphery. For technical reasons, there were no dots at the very centre 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 programme “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 mins, and trials were separated by at least 24 hrs to limit any habituation to the stimulus (Hill and Howarth, 2000). To avoid possible circadian rhythm effects, each trial took place at the same time of day. A repeated measures design was used, and to minimise order effects the sequence in which the conditions were presented was balanced 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 using the Simulator Sickness Questionnaire (SSQ) (Kennedy et al., 1993). Measures of interest were the change (post – pre exposure score) in the SSQ total scores and the change in SSQ subscores (N, O, D). 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 once malaise rating 4 was reached or after 20 mins, whichever was the sooner. Participants who reached a malaise rating of 4, and stopped, before 20 mins were assigned continuation values of 4. All the participants were initially symptom-free and the measures of interest were (i) the time for participants to first report a sickness rating of 2 (S2), (ii) the time to first report a rating of 3 (S3), (iii) the maximum sickness rating, (iv) the sum of the sickness ratings over 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.

The occurrence of vection was assessed post exposure by asking participants the following question: “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 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 typically occurred after about 15 seconds.

**Data analysis**

Data analysis was performed using the software package SPSS (version 13). The data were analysed twice. The first analysis considered the effects of session order, and because none were identified, the analyses were repeated assuming 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 analysed using a non-parametric approach. The symptom onset time and accumulated sickness rating distributions were heavily negatively skewed due to the large number of participants reached the 20 min maximum exposure without reporting any symptoms. To minimise the number of ties, a similar approach to that previously performed by Golding (2003) was adopted. This used 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, differences between conditions were tested 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. One participant did not experience any vection during 0.025 Hz oscillation but did so during oscillation at the other frequencies. In the
second experiment, three participants did not report any vection during 0.8 Hz oscillation whereas one 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 cut-off. It can be seen that in experiment 1 an increase in frequency produced greater motion sickness. None of the participants reported nausea (sickness rating 3) during 0.025 and 0.05 Hz oscillation. During 0.2 Hz oscillation, however, two participants asked to terminate the experiment before the maximum 20 min time cut off (at minute 17 and 18), having reached sickness rating 4. The results of experiment 2 show the reverse in that an increase in frequency beyond 0.2Hz resulted in reduced motion sickness. Two 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.

Table 1. Number of participants reaching each sickness rating stage before the 20 min cut-off for each frequency in Experiments 1 and 2

<table>
<thead>
<tr>
<th>Frequency (Hz)</th>
<th>Experiment 1</th>
<th>Experiment 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sickness rating</td>
<td>0.025</td>
<td>0.05</td>
</tr>
<tr>
<td>1. No symptoms</td>
<td>12/12</td>
<td>12/12</td>
</tr>
<tr>
<td>2. Mild symptoms but no nausea</td>
<td>5/12</td>
<td>5/12</td>
</tr>
<tr>
<td>3. Mild nausea</td>
<td>0/12</td>
<td>0/12</td>
</tr>
<tr>
<td>4. Moderate nausea</td>
<td>0/12</td>
<td>0/12</td>
</tr>
</tbody>
</table>

**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 0.2Hz oscillation was significantly higher than...
during 0.05 Hz oscillation ($Z = 2.524, p = 0.012$) and 0.025 Hz oscillation ($Z = 2.240, p = 0.025$). The rating during 0.1 Hz oscillation was significantly higher than that of the 0.025 Hz oscillation ($Z = 2.384, p = 0.017$). 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 ($Z = -2.158, p = 0.031$).

![Figure 2](image)

Figure 2. (a) Mean (± SEM) accumulated sickness rating and (b) mean (± SEM) time to sickness rating 2 (O2) and 3 (O3) as a function of frequency for experiment 1 and 2.

**Symptom onset time**

Figure 2b shows the mean times to achieve sickness ratings 2 (mild symptoms, but no nausea) and 3 (mild nausea). Since both measures failed to pass the tests for normality, non-parametric statistics were used. In experiment 1, the time to achieve sickness ratings 2 and 3 both became shorter with higher frequencies. Post-hoc analysis showed that time to sickness rating 2 during 0.2 Hz oscillation was significantly shorter than during either 0.05 Hz oscillation ($Z = -2.449, p = 0.014$) or 0.025 Hz oscillation ($Z = -2.668, p = 0.008$). Time to sickness rating 2 was significantly shorter during 0.1 Hz oscillation compared with oscillation...
at 0.025 Hz (Z = -2.670, p = 0.008). Time to sickness rating 3 during 0.1 Hz oscillation was significantly shorter than during 0.025 Hz oscillation (Z = -2.124, p = 0.034). 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 sickness rating 2 was shortest during 0.2 Hz oscillation and became consistently longer with increasing frequencies above this frequency. Time to achieve sickness rating 3 was shortest during 0.4 Hz oscillation and became longer with frequencies both below and above 0.4 Hz. Due to the abnormal distribution of both time to sickness rating 2 and 3, non-parametric tests were employed. Post-hoc comparison showed that time to sickness rating 2 during 1.6 Hz oscillation was significantly longer than during 0.4 Hz oscillation (Z = 2.123, p = 0.031). No other differences were found to be significant.

**Simulator Sickness Questionnaire (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 = 0.030; Z = 2.692, p = 0.007, 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.
<table>
<thead>
<tr>
<th></th>
<th>Experiment 1</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Experiment 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sickness rating</td>
<td>0.025</td>
<td>0.05</td>
<td>0.1</td>
<td>0.2</td>
<td>0.2</td>
<td>0.4</td>
<td>0.8</td>
<td>1.6</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>19.0(5.0)</td>
<td>25.6(7.9)</td>
<td>35.5(10.9)</td>
<td>36.8(12.8)</td>
<td>17.3(5.4)</td>
<td>15.0(3.1)</td>
<td>14.9(3.7)</td>
<td>14.6(3.1)</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>12.7(3.9)</td>
<td>19.9(8.3)</td>
<td>31.0(8.9)</td>
<td>33.4(13.5)</td>
<td>13.9(6.9)</td>
<td>14.7(5.2)</td>
<td>11.3(3.8)</td>
<td>6.1(1.9)</td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>19.0(5.3)</td>
<td>25.3(7.1)</td>
<td>28.4(9.2)</td>
<td>27.8(9.2)</td>
<td>17.2(4.7)</td>
<td>13.8(1.7)</td>
<td>16.5(4.1)</td>
<td>19.3(3.4)</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>17.4(5.5)</td>
<td>19.7(6.3)</td>
<td>34.8(13.7)</td>
<td>37.1(13.7)</td>
<td>12.7(5.5)</td>
<td>8.9(4.3)</td>
<td>8.9(3.9)</td>
<td>10.1(5.3)</td>
<td></td>
</tr>
</tbody>
</table>

**DISCUSSION**

This study was conducted to explore the frequency dependence of VIMS for linear oscillatory 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 - 0.4 Hz. Although the SSQ total scores, accumulated sickness rating and time to sickness rating 2 all indicated motion sickness to peak at 0.2 Hz, time to sickness rating 3 indicated 0.4 Hz oscillation to be most provocative (see figure 2). The highest number of participants reaching sickness rating 2 was at a frequency of 0.2 Hz, but the highest number of participants reaching sickness rating 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 is the value at which their visual and vestibular tuning functions cross, and which 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 due to a phase error in motion signals from the otoliths and somatosensory receptors. Von Gierke and Parker (1994) further elaborated on this by suggesting a potential conflict not only between the otoliths and somatosensory receptors but also 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 over periods of time greater than around 0.5 seconds. 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 it 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 aetiology is provided by the postural instability theory (Riccio and Stoffregen, 1991) according to which motion sickness only occurs under 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 0.2Hz (Stoffregen and Smart, 1998). However, whereas several studies provide support for this theory, there are numerous findings which appear difficult to reconcile with this theory. These were recently reviewed by
Bos (2011b) 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 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 healthy individuals. Irrespective of how exactly instability is defined (see Riccio and 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 (2011b), postural stability and motion sickness may be related via a common mechanism, but this 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, 2008). Within the subjective vertical conflict model, relevant visual and vestibular sensory signals pass through a low pass filter with a Time Constant of 5s (=0.2Hz). 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 0.2Hz and an output is given which initiates motion sickness (Bos et al., 2008). At frequencies both below and above 0.2Hz, 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 et al., 2005)
peak velocity during rotational motion, suggests the frequency dependence of VIMS to be
largely independent of displacement and acceleration. Furthermore, if motion sickness was
dependent solely upon 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 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 our
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 which have the potential to
cause VIMS. In our experiment, which involved participants viewing a star-like pattern of
stars, the maximum level of VIMS was found in the 0.2 – 0.4 Hz. region, 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
- Under conditions in which stationary observers are exposed to dynamic visual displays, optical flow which simulates oscillating fore-and-aft motion in the frequency range of 0.2-0.4 Hz should be avoided
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Biographies

Cyriel Diels

Cyriel Diels is a senior lecturer in human factors in the Department of Industrial Design at the School of Arts and Design, Coventry University. Following his degree in Psychonomics at Utrecht University, he studied the effects of stimulus characteristics on visually induced motion sickness and received his PhD from Loughborough University in 2007. Since then he has worked in the Human Factors and Simulation group at the Transport Research Laboratory (TRL) with a focus on driving behaviour and simulation technology. Before taking up his current position, he worked in the Jaguar Land Rover research department on the development and evaluation of new HMI technologies in the automotive environment.

Peter A. Howarth

Peter A. Howarth started his working life as an Optometrist (an Ophthalmic Optician) before returning to academia. His Masters in Ergonomics, from Loughborough University, was followed by a spell on the West Coast of the USA when he worked in the Lawrence Berkeley Laboratory at the University of California at Berkeley. During this period he investigated the Human Factors issue of how the human pupil responds to flicker. A year after he was awarded his PhD from University of California at Berkeley in Physiological Optics, he returned to England and took up his present position in what is now the Environmental Ergonomics Research Centre, Loughborough Design School, Loughborough University.