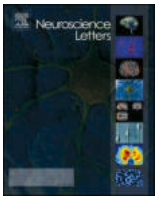
Neuroscience Letters 830 (2024) 137767

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet



Commentary

Concussion can increase the risk of visually induced motion sickness

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ARTICLE INFO

Keywords:
Motion sickness
Concussion
Visual dependence
Field dependence
Motion sensitivity
Dizziness

ABSTRACT

Concussion can lead to various symptoms such as balance problems, memory impairments, dizziness, and/or headaches. It has been previously suggested that during self-motion relevant tasks, individuals with concussion may rely heavily on visual information to compensate for potentially less reliable vestibular inputs and/or problems with multisensory integration. As such, concussed individuals may also be more sensitive to other visually-driven sensations such as visually induced motion sickness (VIMS). To investigate whether concussed individuals are at elevated risk of experiencing VIMS, we exposed participants with concussion (n = 16) and healthy controls (n = 15) to a virtual scene depicting visual self-motion down a grocery store aisle at different speeds. Participants with concussion were further separated into symptomatic and asymptomatic groups. VIMS was measured with the SSQ before and after stimulus exposure, and visual dependence, self-reported dizziness, and somatization were recorded at baseline. Results showed that concussed participants who were symptomatic demonstrated significantly higher SSQ scores after stimulus presentation compared to healthy controls and those who were asymptomatic. Visual dependence was positively correlated with the level of VIMS in healthy controls and participants with concussion. Our results suggest that the presence of concussion symptoms at time of testing significantly increased the risk and severity of VIMS. This finding is of relevance with regards to the use of visual display devices such as Virtual Reality applications in the assessment and rehabilitation of individuals with concussion.

1. Introduction

Concussion is a common type of mild traumatic brain injury resulting in various symptoms such as temporary impairment of attention and memory, mood changes, balance problems, and/or headaches [1,2]. Among these symptoms, visual vertigo and visually induced dizziness (VID) are sensations that often persist for a prolonged period after the actual concussion-causing event [3,4]. Although these symptoms may be caused by injuries to the inner ear or vestibular trauma (e.g., benign paroxysmal positional vertigo) as a direct result of the concussion event, they can also be observed in concussed individuals without any measurable vestibular end organ deficits or pathologies [5]. One potential explanation for the presence of these symptoms could be a heightened sensitivity to visual motion in concussed individuals [6–10]. In healthy individuals, under typical everyday circumstances, congruent and redundant information provided by the visual, vestibular, auditory, and somatosensory systems are integrated in an optimal fashion in order to precisely estimate one's position in and movement through space [11,12]. That is, the brain integrates and weights these sensory inputs based on their individual reliability, with more reliable cues weighted higher than less reliable cues [13–16]. It has been suggested that individuals with concussion may show non-optimal visual-vestibular integration and may over-weight dynamic visual information compared to vestibular information [17]. This over-weighting of visual information, or *visual dependence* [18], and the non-optimal integration of sensory information may increase both visual motion sensitivity and VID-related symptoms in some individuals post-concussion.

Visual dependence can be considered a certain type of *field dependence,* a cognitive style describing an individual's tendency to generally rely more on internal (e.g., bodily) versus external (e.g., environmental)

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https://doi.org/10.1016/j.neulet.2024.137767
Received 1 March 2024; Accepted 7 April 2024
Available online 8 April 2024

cues [19]. The concept of field dependence represents a continuum ranging from highly field dependent (e.g., relying strongly on external cues such as visual information) to highly field independent (e.g., relying strongly on internal cues such as vestibular information). Field dependence may also play a role when estimating one's body position and self-motion in space [20,21], as this process requires the integration of external (e.g., visual) and internal (e.g., vestibular) cues. In participants without concussion, increased visual dependence has been associated with dizziness [22] and balance disorders [23].

Stronger dependence on visual motion inputs may also increase one's susceptibility to other visually induced phenomena such as vection (i.e., illusion of self-motion in the absence of physical movement, [24,25]) or visually induced motion sickness (VIMS). VIMS is a sensation similar to traditional motion sickness, with the difference being that actual, physical movement is typically missing during VIMS in, for instance, large-scale movie theaters, Virtual Reality (VR) applications, or simulation-based amusement park rides [26,27]. Common symptoms of VIMS include, but are not limited to, nausea, cold sweating, pallor, fatigue, eyestrain, dizziness, or headache. Note that VIMS is different from VID [28], since dizziness is not considered one of the cardinal symptoms of VIMS, which is predominantly driven by gastrointestinal, arousal, and oculomotor issues. Although the perceptual sensation of dizziness might not be distinguishable between both cases, VID typically starts immediately after visual stimulation begins, whereas dizziness as a symptom of VIMS typically builds up over time and requires at least a few minutes to manifest itself. Importantly, while VID is more often observed in clinical populations, VIMS can be experienced by every individual, except for those with a complete bilateral vestibular deficit [29]. It is often assumed that a sensory conflict between the information from the visual, vestibular, and somatosensory senses is the root cause of VIMS [30,31]. For instance, stationary VR users may experience VIMS when the visual system indicates self-motion through optical flow, whereas the vestibular and somatosensory systems indicate stasis. If this conflict is novel to the individual and habituation or adaptation has not been successfully established, VIMS may occur [32]. Interestingly, higher field dependence in healthy individuals has been discussed as a potential contributor to greater motion sickness susceptibility [21,33] and VIMS [34], although the evidence for this relationship remains sparse.

Compared to those without concussion, concussed individuals might, in general, be at elevated risk of experiencing a visual-vestibular conflict resulting in VIMS given their potentially increased sensitivity to visual motion. Interestingly, to the best of our knowledge, empirical evidence to evaluate this postulation is missing and no research study has yet investigated whether concussion affects the severity of VIMS. Although previous studies have found that a slower concussion recovery is linked to general motion sickness susceptibility [35,36], the basic question of whether or not a concussed individual experiences more motion sickness or VIMS relative to a non-concussed individual has not been examined. The goal of the present study was to investigate the effect of concussion on VIMS, while also considering the relationships among VIMS, visual dependence, and chronic dizziness (i.e., participants' general tendency to experience dizziness during everyday life). To achieve this, we exposed individuals with a history of concussion and healthy controls to an immersive VR scenario. As concussed individuals often report symptoms when they are exposed to situations that contain visually complex information or contain moving visual environments [37,38], such as shopping in a supermarket, a VR scene of a grocery store aisle was chosen. Participants were virtually moved down the grocery store aisle and reported their level of VIMS before and after exposure to the visual experimental stimulus. In addition, a Rod-and-Disk test measured participants' level of visual dependence and questionnaires recorded participants' self-reported level of everyday dizziness and general somatic sensations at baseline.

2. Material and methods

2.1. Participants

Thirty-one adults (M_{age} = 36.3 years, age range 20–54 years) participated in this study, including 15 healthy controls (9 female, 6 male) and 16 participants with a medically diagnosed history of concussion. Concussions were defined based on clinical criteria current at the time of recruitment [39]. For individuals who had recently experienced a concussion, additional information including the date it occurred, whether it was diagnosed by a medical professional, signs and symptoms, as well as treatments were recorded.[1] Participants with concussion were categorized as either symptomatic ($n = 7$; 6 female) or asymptomatic (n = 9; 5 female). Symptomatic participants reported chronic post-concussion symptoms (i.e., three or more months past their date of injury) at the time of participation, whereas asymptomatic participants were no longer experiencing symptoms based on self-report. The Rivermead Post-Concussion Symptom Inventory [40] was used to quantify the number and intensity of symptoms experienced by participants with a history of concussion. Participants were recruited from the community and local concussion clinics. Participants reported no recent history of stroke, psychiatric, vestibular, or musculoskeletal disorders or other major health conditions (e.g., diabetes) and had normal or corrected-to-normal visual acuity as assessed via the Early Treatment for Diabetic Retinopathy Study [41] eyechart. The Dynamic Visual Acuity test [42] was also used to assess vestibular-ocular function. Two participants with concussion (one asymptomatic and one symptomatic) reported migraines post-concussion. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and was approved by the Research Ethics Board of the University Health Network.

2.2. Baseline measures

Several baseline measures were acquired to measure sensory and perceptual characteristics of the participants in each group. Note that some of these baseline measures, including the Vestibular Disorders of Activities of Daily Living Scale [43] and the Rivermead Post-Concussion Symptom Inventory [40], are not further discussed in the present paper (see Gabriel et al., submitted).

2.2.1. Visual dependence

The computerized Rod-and-Disk task [8] was used to measure visual dependence. Participants were seated in front of a computer monitor looking through a 20 cm long tube with a cloth covering their head to occlude any environmental visual cues. The stimulus consisted of a white line ("rod") presented on a black background filled with 220 randomly distributed green dots. Participants were instructed to align the rod vertically with respect to gravity, providing a measure of their subjective visual vertical. The background dots were either stationary or rotated clockwise/counterclockwise at 10° per second (12 trials each, total 36 trials). The static condition was presented first and served as the baseline for the rotating conditions, which were presented in randomized order.

For data analysis, outliers (i.e., trials with extreme scores substantially deviating from the median) were removed (9 trials in total across 3 participants), and the difference in degrees between the true vertical (0°) and participant's placement angle of the rod was calculated for all static and rotating trials. The difference between the mean angle recorded for the static condition and the mean angle recorded for each of

[1] Of the symptomatic concussed participants, only 2 did not report being diagnosed by a health professional, but all received treatment. For the asymptomatic participants, 3 did not report being diagnosed by a health professional, and only 4 received treatment.

the counterclockwise and clockwise conditions was calculated and converted to absolute values before they were averaged (RD score) for further analysis.

2.2.2. Dizziness & somatic symptoms

The extent to which dizziness impacted the participant's everyday life was assessed using the Dizziness Handicap Inventory (DHI) [44], a questionnaire with 25 items where participants have to self-report the perceived impact of dizziness on functional, emotional, and physical levels during their everyday lives. The Patient Health Questionnaire-15 (PHQ-15) [45], including 15 somatic symptoms such as stomach pain, back pain, and headaches, was administered to measure any somatic symptoms participants were experiencing.

2.3. Dependent measure

VIMS severity was measured using the Simulator Sickness Questionnaire (SSQ) [46], containing 16 items (e.g., nausea, fatigue, headache) rated on a 4-point scale ranging from 0 (*not at all*) to 3 (*severe*). Three subscales – nausea, oculomotor, disorientation – as well as a total score were calculated following a weighting procedure suggested by the authors of the SSQ. The SSQ was administered once before and once after stimulus presentation (see below for details).[2] The pre-stimulus SSQ captured participants' level of well-being prior to the study at baseline and was therefore not associated with VIMS per se. Instead, it can be interpreted as a general measure of different symptoms unrelated to stimulus exposure. The difference between the SSQ scores reported at baseline and after stimulus exposure (ΔSSQ) was used to estimate the increase in symptom severity elicited by the stimulus.

2.4. Stimuli and apparatus

The study was conducted at the Challenging Environment Assessment Lab (CEAL) located at the KITE Research Institute. Stimuli were presented in a dome-shaped VR laboratory (*StreetLab*; Fig. 1) on a curved projection screen with a large field-of-view (240° horizontally, 105° vertically). No sounds were presented. The stimulus consisted of a virtual grocery scene designed in Unity (2019.2.2f1, Unity Technologies Inc.) and was presented using MATLAB (2015b, The MathWorks Inc.). The virtual scene depicted a straight grocery store aisle (approx. 145 m long and 4.4 m wide) stocked with dry-goods on the shelves. Other areas of the grocery store, including other aisles and a produce section, were visible when participants were positioned at the start of the aisle (see Fig. 1). During stimulus presentation, the visual scene created optic flow, virtually simulating participants' moving forward down the aisle. Navigation speed varied across trials (0.07 – 4.0 m/s), and trials included either constant velocity or intermittent visual perturbations (i.e., visual motion presented in 1-second bursts with 15 bursts total per trial). Constant velocity trials lasted 30 s and visual perturbation trials lasted 60 s, including a 0.5 s acceleration and deceleration phase for each. Thirty trials were presented (15 constant velocity, 15 intermittent), resulting in a total stimulus exposure duration of 22.5 min. Note that the velocity manipulations are not relevant for the present paper and will not be further discussed.

2.5. Procedure

Participants provided written informed consent before completing the baseline assessments including the Rod-and-Disk task, the pre-stimulus SSQ, and the clinical assessments (e.g., DHI). After entering StreetLab, participants were asked to comfortably stand at the centre of the lab on a force plate with their feet approximately hip-width apart and were outfitted with a loose harness to prevent falls. At the beginning of each trial, a fixation cross was displayed in the centre of the screen to direct the participant's attention to the visual scene; the fixation cross disappeared as soon as the visual scene began to move. After all trials in the experiment were completed, participants filled out the SSQ a second time. Participants were then debriefed and received their compensation.

2.6. Data analysis

All statistical analyses were conducted using R (v. 3.6.2, R Foundation for Statistical Computing, 2016). Non-parametric Kruskall Wallis tests, followed-up with Wilcoxon post hoc tests (Benjamini-Hochberg corrected), were calculated to compare the three experimental groups (healthy controls, symptomatic concussion, asymptomatic concussion). For a comparison of combined concussion groups and healthy controls see the Supplementary Materials. Spearman correlations were calculated to analyze the relationship between the baseline measures (RD, DHI) and the SSQ scores; however, given the small sample size in the two concussion groups, correlations were run for both concussion groups (symptomatic, asymptomatic) combined.

3. Results

3.1. VIMS and concussion

Averaged SSQ scores for all three groups are shown in Fig. 2. At baseline, a significant effect of group (control, symptomatic, asymptomatic) was observed for all SSQ subscales, including nausea, $H(2) = 9.26$, $p = .009$, $\eta^2 = .26$, oculomotor, $H(2) = 12.26$, $p = .002$, $\eta^2 = .37$, disorientation, $H(2), = 13.26$, $p = .001$, $\eta^2 = .40$, and the total score, $H(2) = 12.00$, $p = .002$, $\eta^2 = .36$. For all SSQ subscales and the total score, post hoc tests revealed that the symptomatic concussion group showed significantly higher SSQ scores compared to healthy controls (all p's $< .014$) and the asymptomatic concussion group (p's $< .018$; except for the SSQ subscale nausea, $p = .095$), suggesting that they experienced an elevated level of symptoms before stimulus exposure. No difference between the asymptomatic concussion group and healthy controls showed at baseline.

After stimulus presentation, a significant effect of group showed for all SSQ subscales, including nausea, $H(2) = 7.36$, $p = .025$, $\eta^2 = .19$, oculomotor, $H(2) = 11.113$, $p = .004$, $\eta^2 = .33$, disorientation, $H(2), = 13.36$, $p = .001$, $\eta^2 = .41$, and the total score, $H(2) = 12.23$, $p = .002$, $\eta^2 = .37$. Again, post hoc tests indicated that the symptomatic concussion group showed significantly higher SSQ scores compared to the other two groups (all p's $< .05$), but no difference between the asymptomatic concussion group and healthy controls were found.

Comparing the ΔSSQ scores, a significant effect of group showed for the SSQ subscales oculomotor, $H(2) = 7.97$, $p = .019$, $\eta^2 = .21$, disorientation, $H(2), = 9.65$, $p = .008$, $\eta^2 = .27$, and the total score, $H(2) = 8.73$, $p = .013$, $\eta^2 = .241$, but not for the subscale nausea, $H(2) = 2.13$, $p = .35$, $\eta^2 = .01$. Post hoc tests suggested that for the significant ΔSSQ subscales the symptomatic concussion group showed significantly higher SSQ scores compared to the other two groups (all p's $< .05$), but no difference between the asymptomatic concussion group and healthy controls was found when comparing the difference pre- vs. post-stimulus exposure.

3.2. Visual dependence, dizziness, somatic symptoms, and VIMS

Fig. 3 shows the results of the Rod-and-Disk task (Panel A), DHI (Panel B), and PHQ-15 (Panel C) separated by the experimental groups. For the Rod-and-Disk task, no statistically significant difference between groups was found in a Kruskall-Wallis test, $H(2) = 0.457$, $p = .80$, $\eta^2 = -.02$, although the raw scores may suggest larger misalignments of the

[2] In addition to VIMS measures, vection and postural sway measures were recorded. However, the present paper will solely focus on the VIMS data; the relationship between vection, postural sway, and concussion is discussed in detail in Gabriel et al. (submitted).

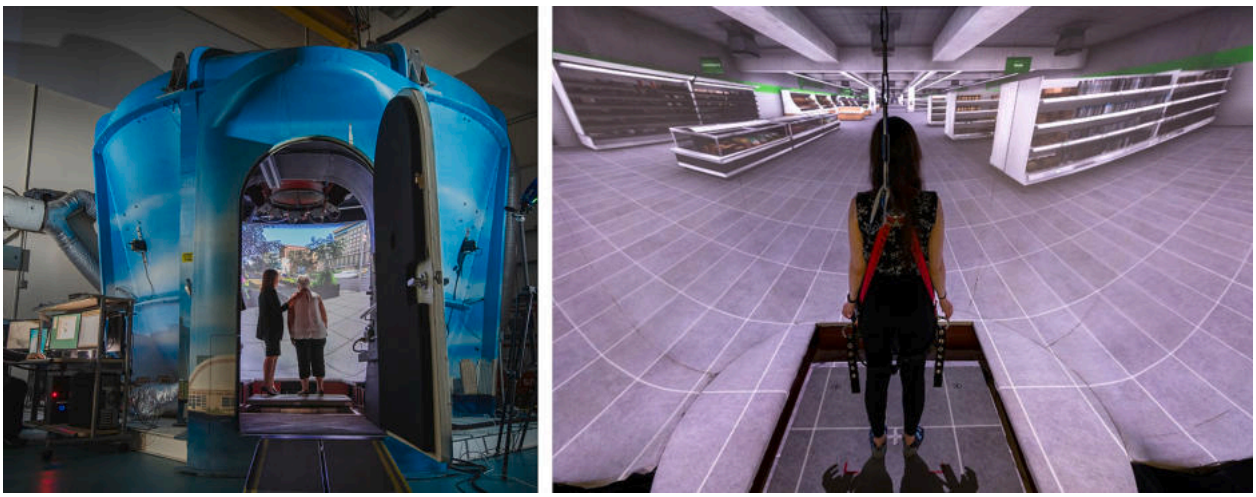

Fig. 1. External view of StreetLab (left) and an interior view of StreetLab and the virtual scene (right) showing the grocery store aisle.

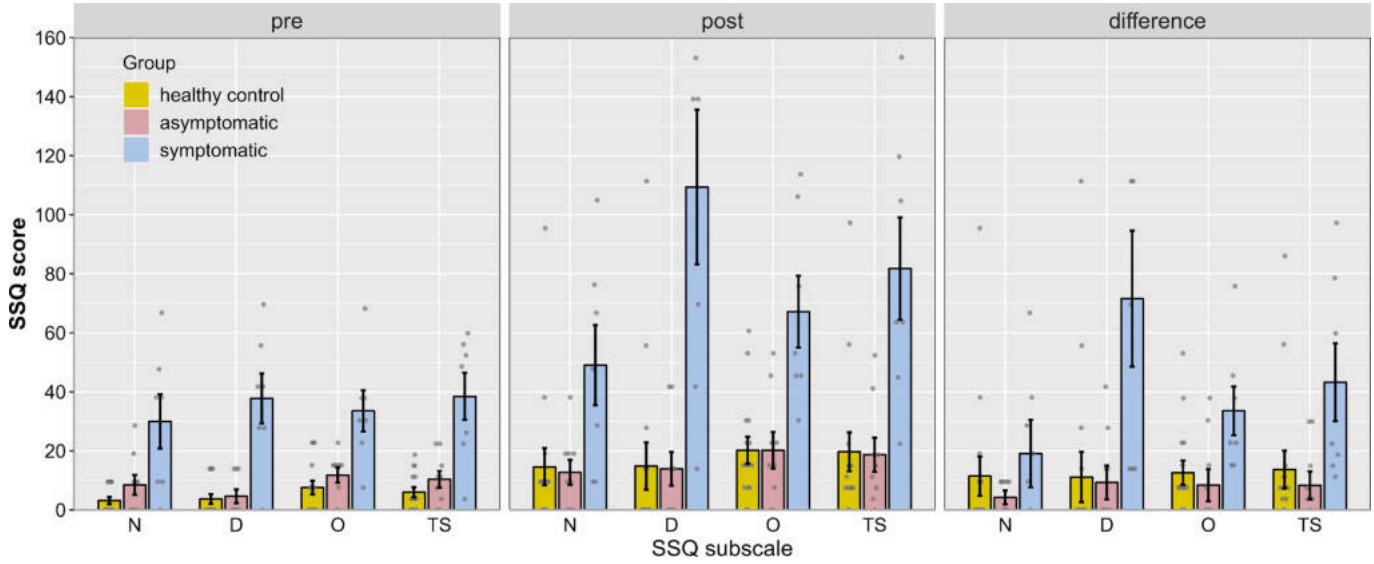

Fig. 2. Averaged SSQ scores for each subscale pre (left) and post (middle) stimulus presentation and the difference between pre- and post-stimulus (ΔSSQ, right) separated by group. *Note:* Error bars represent +/- SEM; dots represent individual scores. N = nausea, D = disorientation, O = oculomotor, TS = total score.

rod (i.e., greater visual dependence) in the symptomatic concussion group. For the DHI, Kruskall-Wallis tests showed a significant main effect of group, $H(2) = 13.84$, $p < .001$, $\eta^2 = .42$, with post hoc tests indicating significantly higher DHI scores in the symptomatic group compared to the healthy control group ($p = .002$) and the asymptomatic group ($p = .012$), whereas healthy controls did not differ from the asymptomatic group ($p = .388$). Similarly, a main effect of group was found for the PHQ15, $H(2) = 10.37$, $p = .005$, $\eta^2 = .30$. Post hoc tests showed that PHQ-15 scores were significantly higher in the symptomatic group compared to healthy controls ($p = .006$) and the asymptomatic group ($p = .029$), whereas healthy controls did not differ from those who were asymptomatic ($p = .434$).

Spearman correlations were calculated separately for healthy controls and the combined concussion groups (Table 1). For healthy controls, moderate-to-strong positive correlations were found between the RD score and the ΔSSQ scores nausea, disorientation, and the total score (Fig. 4), suggesting that participants who were more visually dependent (i.e., larger misalignment of the rod) experienced a larger increase in VIMS after stimulus exposure. For participants with concussion, a strong positive correlation was found between the ΔSSQ score nausea and visual dependence, but not with the other ΔSSQ scores. With regards to the DHI and PHQ-15 scores, strong, positive correlations were found with some the ΔSSQ scores in the concussed groups but not in the control group.

4. Discussion

The goal of the present study was to investigate how concussion affects the severity of VIMS. Additionally, we explored the relationship between VIMS and visual dependence, dizziness, and somatic symptoms as influencing factors. Overall, individuals who reported concussion symptoms at the time of testing also reported significantly more severe VIMS and significantly higher levels of general dizziness and somatic symptoms than individuals with asymptomatic concussion and healthy controls. Positive, moderate-strong correlations between visual dependence and VIMS were found both in healthy controls and participants with concussion.

4.1. VIMS severity following concussion

To the best of our knowledge, no study has yet investigated how VIMS severity is affected by concussion. In this study, we found evidence

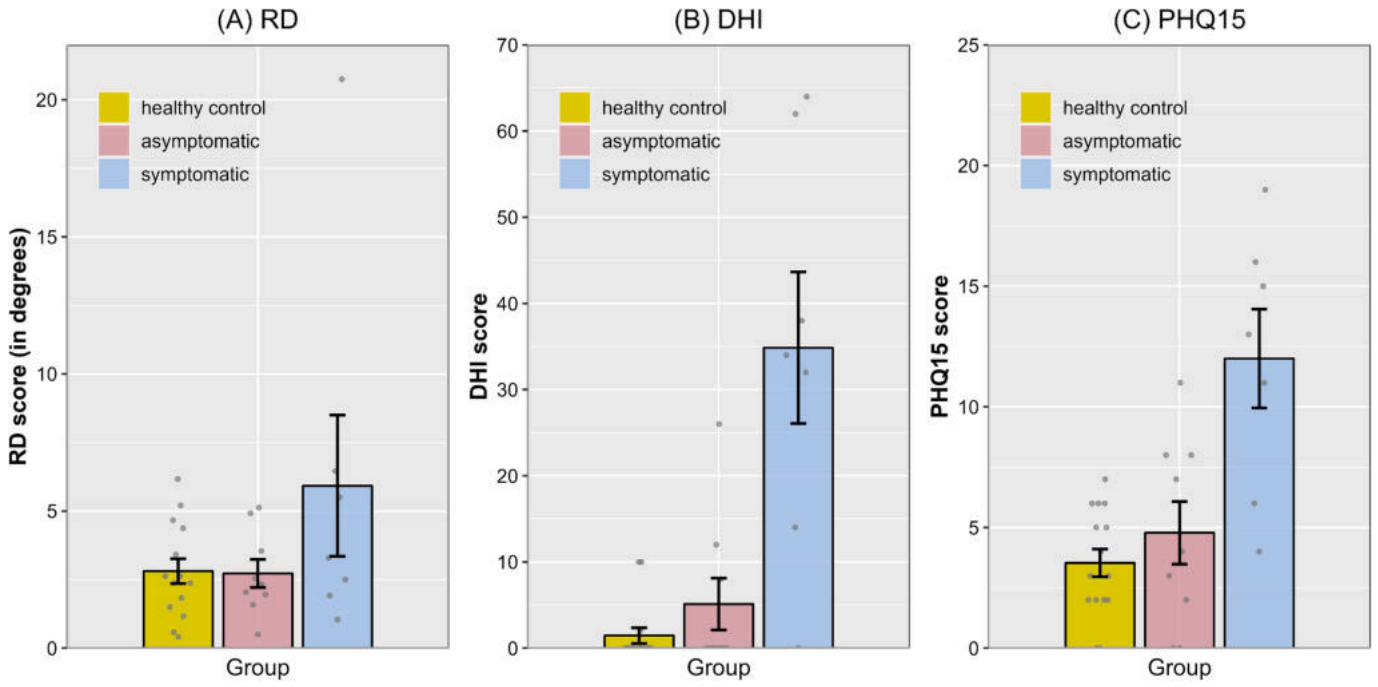

Fig. 3. Averaged RD score (A), DHI score (B), and PHQ-15 score (C) separated by group. *Note:* Error bars represent the standard error of the mean, and the grey dots represent individual participant values.

Table 1
Pearson correlations between the different ΔSSQ scores and RD, DHI, and PHQ-15.

Group		SSQ subscale			
	Variable	Nausea	Disorientation	Oculomotor	Total score
Healthy control	PHQ-15	−.007	−.073	−.036	−.040
$n = 15$	RD	.524	.636*	.299	.550*
	DHI	−.199	−.324	−.396	−.324
Concussion (combined)	PHQ-15	.442	.720**	.573*	.660**
$n = 16$	RD	.670**	.334	.262	.432
	DHI	.122	.671**	.379	.479

Note: *p < .05, **p < .01.

that concussion can significantly increase VIMS severity in individuals who currently experience concussion symptoms (e.g., dizziness). This group reported significantly higher difference scores on the SSQ subscales disorientation and oculomotor, as well as the total SSQ score compared to healthy participants and to asymptomatic individuals with concussion. Interestingly, no difference between healthy participants and asymptomatic individuals with concussion was found. This increase in VIMS severity in symptomatic individuals with concussion seems plausible, given that VIMS is likely rooted in a sensory conflict between the visual, vestibular, and proprioceptive systems. As symptomatic concussed individuals may show particularly pronounced non-optimal integration of visual and vestibular information, this may result in an over-weighting of visual cues and larger visual-vestibular conflict, thereby bolstering the severity of VIMS [17,18]. One component that could explain differences in VIMS severity between symptomatic and asymptomatic individuals with concussion is habituation. It is well known that VIMS decreases over time due to repeated exposure [47,48], but the time it takes to show effective habituation may be inter-individually different. It is possible that asymptomatic people with concussion may have already successfully habituated to a potential re-weighting of sensory information post-concussion, whereas symptomatic individuals have not. Given the observed relationship between VIMS and (symptomatic) concussion, our findings suggest that VR-based concussion rehabilitation approaches should be used with caution, as VR users with a history of concussion could be at elevated risk of experiencing adverse side effects associated with VIMS. However, the effects observed here reflect acute changes in VIMS during and immediately after stimulus exposure, whereas prolonged effects of dynamic visual stimulus exposure (negative or positive) remain unknown.

4.2. *The role of visual dependence, dizziness, and somatization*

We found positive, moderate-to-strong correlations between visual dependence and VIMS for healthy and concussed participants, suggesting that a stronger reliance on visual cues was linked to higher VIMS severity. Specifically, visual dependence was associated most strongly with disorientation in healthy controls and most strongly with nausea in concussed participants. From a theoretical perspective, this positive relationship between visual dependence and VIMS makes sense given that more visually dependent individuals rely more on external (e.g., visual) than internal (vestibular) cues with regards to their position in space. However, relying on visual cues also increases the risk of a visual-vestibular mismatch during dynamic visual stimulation in stationary observers. Interestingly, we did not see any significant differences in visual dependence between the experimental groups. Although symptomatic concussed individuals showed higher visual dependency scores as measured by the Rod-and-Disk test compared to the other two groups, these differences were not statistically significant. It is important to interpret these correlations with care given the small sample size in each experimental group. That is, for healthy individuals, the strong positive

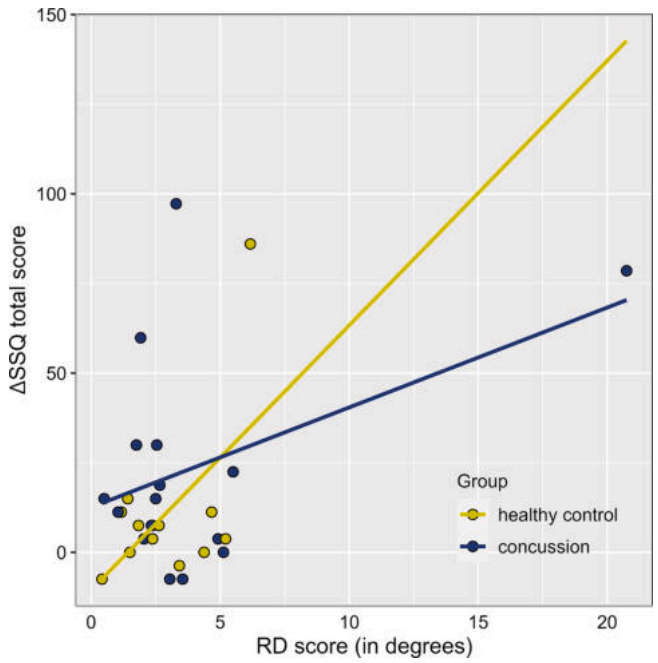

Fig. 4. Scatterplot between the RD score and the ΔSSQ total score for all groups.

correlations seem to be primarily driven by one individual data point (high VIMS and high visual dependency), and removing this data point from the analysis may alter the correlation substantially. Thus, more data is indeed needed to be able to better address the relationship between visual dependence and VIMS in future research studies.

With regards to dizziness and somatization, not surprisingly, we found that symptomatic participants with concussion reported significantly higher scores in general compared to the asymptomatic and healthy participants. However, when looking at the relationship between these factors and VIMS, it stands out that dizziness and somatization are only strongly correlated with changes in VIMS in concussed participants but not in healthy individuals. The strongest correlation with dizziness was found for the SSQ subscale disorientation, which again seems logical. Somatization was positively correlated with changes in all SSQ subscales, suggesting that more pronounced pre-existing somatic symptoms may lead to more severe VIMS after dynamic visual stimulation. Similar results were found for healthy participants in our previous work, where general discomfort prior to a VIMS-inducing stimulus was found to be a good predictor of VIMS severity after stimulus exposure [49].

4.3. Limitations and future direction

One of the main limitations of the present study is the small sample size, specifically when splitting concussed individuals into symptomatic and asymptomatic groups. Small sample sizes are not uncommon for studies involving participants with concussion given the limited access to this group [45,50] and the significant differences that were observed between groups are quite pronounced as demonstrated by large effect sizes. However, we need to interpret non-significant findings (e.g., differences in visual dependency, differences in ΔSSQ between healthy and asymptomatic concussed participants) with care. Similarly, the small sample size did not allow us to investigate potential sex-related differences with regards to VIMS.

Another limitation is that we did not collect information on motion sickness susceptibility, particularly susceptibility pre-concussion in the concussion group. In retrospect, it would have been interesting to know whether one's general susceptibility to motion sickness and VIMS may have affected the severity of symptoms following dynamic visual stimulation. The motion sickness susceptibility questionnaire [51] or the visually induced motion sickness susceptibility questionnaire [52,53] are validated tools that allow an estimation of one's susceptibility to these phenomena and would be a valuable addition for similar studies in the future.

Lastly, it is important to note that the visual stimulus that we used was not very powerful for inducing VIMS in all participants. However, this was done by design as we deliberately chose a visual scene that is commonly known to be naturally disturbing for many individuals with concussion during their everyday interactions, rather than choosing a stimulus that would confidently induce VIMS. Thus, it is not surprising that healthy participants reported little VIMS overall. We can speculate that a more intense stimulus may further increase VIMS levels in all groups, including those who reported to be asymptomatic and controls and may indeed make any other group-related differences more pronounced (e.g., between healthy controls and asymptomatic). Future research could apply a more intense stimulus to further investigate these questions.

5. Conclusions

Our results are the first to demonstrate an increase in VIMS susceptibility following concussion. We found convincing evidence that VIMS severity is significantly higher after experiencing concussion, but only for those with lingering self-reported post-concussion symptoms. Additionally, positive correlations between visual dependency and VIMS severity suggested that relying more heavily on visual cues over other non-visual sensory inputs may increase the risk of VIMS.

6. Significance statement

The results of this study suggest that the presence of concussion symptoms significantly increases the risk and severity of visually induced motion sickness (VIMS) and that the level of visual dependence was positively correlated with VIMS.

CRediT authorship contribution statement

Behrang Keshavarz: Formal analysis, Supervision, Visualization, Writing – original draft. **Meaghan S. Adams:** Conceptualization, Investigation, Methodology, Project administration, Writing – review & editing. **Grace Gabriel:** Data curation, Writing – review & editing. **Lauren E. Sergio:** Conceptualization, Funding acquisition, Methodology, Writing – review & editing. **Jennifer L. Campos:** Conceptualization, Funding acquisition, Investigation, Methodology, Resources, Supervision, Writing – review & editing.

Data availability

Data will be made available on request.

Acknowledgements

Funding: This work was supported by a Canadian Institutes for Health Research project grant awarded to Sergio and Campos (Grant #451503).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neulet.2024.137767.

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