Effect of virtual reality and whole-body heating on motion sickness severity: A combined and individual stressors approach

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ABSTRACT

Background: Virtual reality (VR) use is limited by the potential side effects of prolonged exposure to vection, leading to motion sickness. Air temperature (T_a) may exacerbate the severity of such side effects through a synergistic interaction. This study assessed the individual and combined impact of a hot T_a and VR on motion sickness severity. ***Method:*** Thirteen healthy volunteers were exposed to a 20 min visual stimulus, across four experimental conditions: N_CS: 22℃ T_a with computer screen; N_VR: 22℃ T_a with VR; H_CS: 35℃ T_a with computer screen; H_VR: 35℃ T_a with VR. Motion sickness was assessed via fast motion sickness scale (FMS) and simulator sickness questionnaire (SSQ). Physiological indices of motion sickness including, sweat rate, rectal temperature, cutaneous vascular conductance (CVC), skin temperature, blood pressure and heart rate were also examined. ***Results:*** FMS and SSQ ratings indicate a significant main effect for VR, increasing sickness severity ($p<0.001$). A significant main effect of T_a was observed for SSQ, but not FMS ratings (FMS, $p=0.07$; SSQ, $p<0.04$). Despite trends towards synergism, no interaction (T_a x VR) was observed for FMS ($p=0.2$) or SSQ scores ($p=0.07$), indicating an additive response. Synergistic trends were also observed for sweat rate and CVC. ***Conclusion:*** Synergism between VR and heat on motion sickness remains unclear, possibly as a result of considerable inter-individual variation in the reported subjective responses. Understanding of the questions raised by this study inform safe working guidelines for the use of VR in commercial and occupational settings.

KEY WORDS

Nausea, Thermoregulation, Heat stress, Toxic hypothesis, VR

1.0 INTRODUCTION

Advances in Virtual Reality technology (VR) has attracted interest across a range of occupational fields, including medical, military and educational industries. For example, users are now able to artificially experience complex occupational scenarios with reduced risk of injury, damage or cost. Yet, despite the efforts of many manufacturers, there are concerns regarding the associated side effects of prolonged VR use, including malaise, dizziness, headache and eyestrain [1,2]. In many cases VR use has shown to increase the risk of visually-induced motion sickness (VIMS), also known as cybersickness, with ongoing debate concerning to the direct influence of perceived self-motion (vection) [3–9]. For example, an observational study comparing ratings of VIMS in 497 healthy adults, reported a 55% vs. 14% prevalence of sickness, having viewed a 3D and 2D movie respectively [10]. Symptom intensity was also observed to be significantly higher following 3D compared to 2D viewing. Comparable to 'classical' motion sickness, common symptoms of VIMS may present as dizziness, vertigo, sweating, stomach awareness and nausea, which may further progress to vomiting with sufficient stimulus duration or intensity [11–13]. Given the scope and utility of VR technology, it is important to better understand the underlying mechanisms, mediating factors and interactions which may link VR with VIMS, thus maximising its future potential.

Due to the complex nature of motion sickness there is limited agreement on a holistic and theoretical understanding of the mechanisms that cause the syndrome [14]. Indeed, it has even been reported that motion sickness can develop prior to exposure to a provocative stimulus, perhaps due to expectancy or anxiety effects [14]. At present, several hypotheses exist to explain the cause of motion sickness; (i) Sensory Conflict and Rearrangement Theory [15], in which sensory information from visual, vestibular and somatosensory systems, either become mismatched, or if these senses fail to match those stored in the central nervous system from

past experiences; (ii) Postural Instability Theory [16], in which an organism attempts to maintain postural stability in relation to its environment throughout daily activities, with sickness occurring when a stable state can no longer be obtained, along with a perceived lack of control; (iii) Poison Theory or the Toxic Hypothesis [17], an evolutionary response in which emesis acts as a defence mechanism to intoxication of the body due to toxin induced stimulation of the vestibular senses. The net result of the latter is a mismatch between perceived moving vestibular and static visual signals, consequently leading to emesis, vertigo, dizziness and postural instability.

There is also limited research on the secondary and mediating factors that subsequently impact upon VIMS susceptibility during VR use. One such factor is air temperature (T_a), including both hot and cold stimuli. Interestingly, previous studies have highlighted interactions between motion sickness and thermoregulation, showing an increased risk of deep-body cooling in motion sick individuals, when exposed to cold environments [18,19]. In view of the toxic hypothesis, its appears conceivable that this reduction in deep-body temperature may act in conjunction with motion sickness, to further protect the body against perceived intoxication via a slowing of metabolic rate [20]. We further postulate that an added thermoregulatory load induced via heat stress, in addition to a provocative VR stimulus, may artificially strengthen the body's belief that it is under threat from intoxication, subsequently triggering a heightened nauseogenic response [20]. As such, it is possible that a mechanistic interaction of a synergistic nature may exist between heat exposure (i.e. prolonged exposures to $T_a > 30°C$) and VR use, on VIMS susceptibility [21]. While the toxic hypothesis provides a potential explanation for a synergistic interaction between heat and VR, if an interaction is not observed (i.e. additive effects), this may better support the role of other theories such as sensory conflict, in the aetiology of motion sickness.

To understand the role of a hot air temperature in modulating nausea in virtual reality, this investigation examined the combined and differential impacts of heat and VR on motion sickness severity. Three hypotheses were constructed; *1.* individuals would report significantly greater perceptions of VIMS whilst viewing VR, when compared to a computer screen control, *2.* individuals would not report any difference in VIMS under hot conditions, compared to a thermoneutral control, *3.* combined VR and hot conditions would synergistically interact to significantly increase VIMS ratings when compared to either factor independently. An understanding of such main effects and potential interactions is likely to better inform safe working guidelines for the use of VR in commercial and occupational settings, as well as elucidating some of the underlying mechanisms impacting VIMS.

2.0 METHODS

2.1 Participants

Thirteen healthy volunteers, five male and eight females (age, 25 ± 3 yrs), were recruited from the Loughborough, UK between June and November 2017. Inclusion criteria detailed: non-smoking, otherwise healthy individuals, reporting no significant (>6 hrs per week) gaming or VR experience. All participants provided written informed consent. Ethical approval was granted by the Loughborough University Ethics Committee and the research was conducted in accordance with the Declaration of Helsinki, 2008.

2.2 Study Design

The study utilised a repeated measures randomised design and was conducted in the Environmental Ergonomics Research Centre at Loughborough University. Participants were exposed to a standardised visual stimulus, presented across four independent experimental

conditions in a random order: N_CS: neutral air temperature with computer screen (T_a, 22.2 ± 1.0 ℃; RH, 40 ± 3 %); N_VR: neutral air temperature with VR headset (T_a, 22.3 ± 1.0 ℃; RH, 40 ± 4 %); H_CS: hot air temperature with computer screen (T_a, 35.0 ± 0.9 ℃; RH, 38 ± 3 %); H_VR: hot air temperature maintained with VR headset (T_a, 35.1 ± 0.9℃; RH, 37 ± 4 %). One week prior to the first trial, participants were fully briefed in relation to the study aim, design and test requirements. Twenty-four hours prior to each laboratory visit, participants were asked to refrain from alcohol, caffeine and gaming use, and avoid heat exposure. Participants commenced all tests at the same time each day to reduce the effect of circadian rhythm. A minimum 24 hr wash-out period was observed between trials to eliminate any carry-over of effects. Air temperature and relative humidity were monitored throughout the investigation via Testo probe and data logger (Probe- 0635 1535, Logger- 435, Testo Ltd, Germany), and maintained within 1.0℃ T_a / 5% RH of the desired set-point for each trial.

2.3 Visual Stimulus.

The visual stimulus consisted of a 20 min pre-recorded series of computer-generated driving clips (5 merged clips at 3 to 5 mins in duration each), presented through software Project Cars™ (Slightlymad Studious Ltd.). In the control conditions, participants observed the clip sequence on a computer screen (HP LA2306x, 23”, 1920 x 1080) positioned 1 m away at head height. The same driving clip was presented to participants using a VR headset (Oculus Rift Developmental Kit 2), offering a 100° horizontal field of view, with 960x1080 resolution in each eye.

Pre-recorded videos were used to potentiate motion sickness, due to a reduction in sense of control that the participant experiences relative to their environment [22]. Furthermore, the sequencing of the clips was strategically ordered based on the number of turns in the track

(least to most), with the assumption that more turns would equate to an increased risk of motion sickness [23]. Each video clip was presented from the perspective of the driver. Whilst computer screen conditions were fixed in 2D, VR conditions allowed participants to manipulate their visual array in accordance with the pitch, roll and yaw of their own head. Fixation was not controlled in the current study to better align with real world scenarios. Sound levels were standardised across all conditions using a commercial 2.1 multimedia speaker system (Phillips Ltd., Netherlands).

2.4 Experimental Procedures

On arrival to the laboratory, participants first completed a simulator sickness questionnaire (SSQ) to ensure no carry-over of motion sickness symptoms from previous trials [24]. Perceptual ratings for 16 motion sickness symptoms were provided on a 4-point scale (0-none, 1-slight, 2-moderate, 3-severe), and added together to provide a total score. In a separate preparation room, participants donned the appropriate experimental apparatus; heart rate assessed by 3-lead electrocardiogram (Tango M2, SunTech Medical Inc., USA), skin temperature at the calf, thigh, pectoral and tricep by surface thermistor (iButton^{TM}, Maxim, USA), deep-body temperature by rectal thermistor (Grant Instruments Ltd., UK), local sweat rate at the palm, mid-upper-back and shin by ventilated sweat capsules (Q-Sweat, TestWorks, WR Medical Co., USA), skin blood flow at the inner forearm by laser doppler (Moor Instruments Ltd, Devon) and blood pressure by automated sphygmomanometer (Tango M2, SunTech Medical Inc., USA). Once ready, participants entered the temperature controlled experimental room, were asked to sit in front of the computer system on a car seat and find a comfortable position which could be maintained with minimal need for movement.

Participants undertook a 5-min acclimatisation period, remaining quiet and still. At this point, all equipment was calibrated and zeroed ready for data collection. Participants were also briefed on the subsequent use of the Fast Motion Sickness Scale (FMS), used to instantaneously assess perceived severity of motion sickness. The FMS was presented via a visual-analogue scale ranging 0 (no sickness) to 20 (incapacitating sickness), in which a score of 10 should represent moderate levels of motion sickness. The use of this simplistic scale allowed for easy memorisation and recall during VR conditions in which the headset was used. Participants were asked to focus on nausea, general discomfort and stomach awareness, and to ignore perceptions such as boredom, fatigue and nervousness [25]. At minute five, a member of the research team initiated the 20-min visual stimulus, either via computer screen or VR headset. After each minute, participants were asked to provide a verbal FMS rating. Heart rate, skin temperature, rectal temperature, local sweat rate and skin blood flow were all continuously assessed throughout the 20-min period, sampled at 1 Hz. Blood pressure, was assessed every 5 mins. On completion of the 20-min visual stimulus, the SSQ were immediately completed once again, after which the trial finished. To complete the experiment, participants were handed a last copy of the SSQ when leaving the laboratory in order to record simulator sickness two hours post-visual stimulus.

2.5 Data Analysis

Statistical significance was set at $P \leq 0.05$. Two-way, repeated measures analysis of variance (ANOVA) was used to evaluate the main effects of air temperature and visual stimulus, addressing hypothesis 1 and 2, in addition to the interaction between the two factors (T_a x VR), addressing hypothesis 3. Inferential statistical analysis was conducted using the software package IBM SPSS Statistics for Windows (version 23, IBM Corp., USA). Physiological variables were interpreted as a mean and peak value across each 20 min trial. Data are

presented as mean and [95% Confidence Intervals (CI)] unless stated otherwise in figures and tables.

3.0 RESULTS

All participants successfully completed the experiment, undertaking all trials, despite varying reports of motion sickness.

3.1 Motion Sickness

Taken independently (hypothesis 1), VR significantly increased motion sickness severity compared to computer screen viewing when expressed as mean FMS rating (*CS trials*, 0.6 [0.2 - 1.1] vs. *VR trials*, 4.3 [2.6 – 6.0]; $p<0.001$). Air temperature, independently of VR (hypothesis 2), did not significantly influence mean FMS ratings (*N trials*, 2.1 [1.3 – 2.9] vs. *H trials*, 2.8 [1.5 – 4.2]; $p=0.07$). No interaction (hypothesis 3) was observed between VR and air temperature ($p=0.2$), indicating an additive effect when these factors are combined. Peak FMS ratings reflect the results seen for mean FMS ratings, with a significant main effect observed for VR (*CS trials*, 1.6 [0.8 – 2.4] vs. *VR trials*, 8.7 [5.5 – 11.8]; $p<0.001$), however no effect for air temperature (*N trials*, 4.8 [3.2 – 6.4] vs. *H trials*, 5.5 [3.1 – 7.9]; $p=0.3$), and no interaction seen between VR and air temperature ($p=0.9$). Whilst statistical analysis of the data reveals no significant evidence for synergism (T_a x VR), a difference in the magnitude of mean change between H_CS and H_VR (Δ FMS, 4.0), compared to N_CS and N_VR (Δ FMS, 3.2), indicates a trend towards a synergistic interaction (*Fig 1.*). Furthermore, inspection of individual data shows a large inter-individual difference in the magnitude of response across subjects, providing partial evidence for distinct groups of responders or non-responders. Approximately six individuals showed a clear visible difference in mean FMS

224 rating between control and VR conditions, four individuals showing no visible difference, and
225 three interspersed between (*Fig 2.*).
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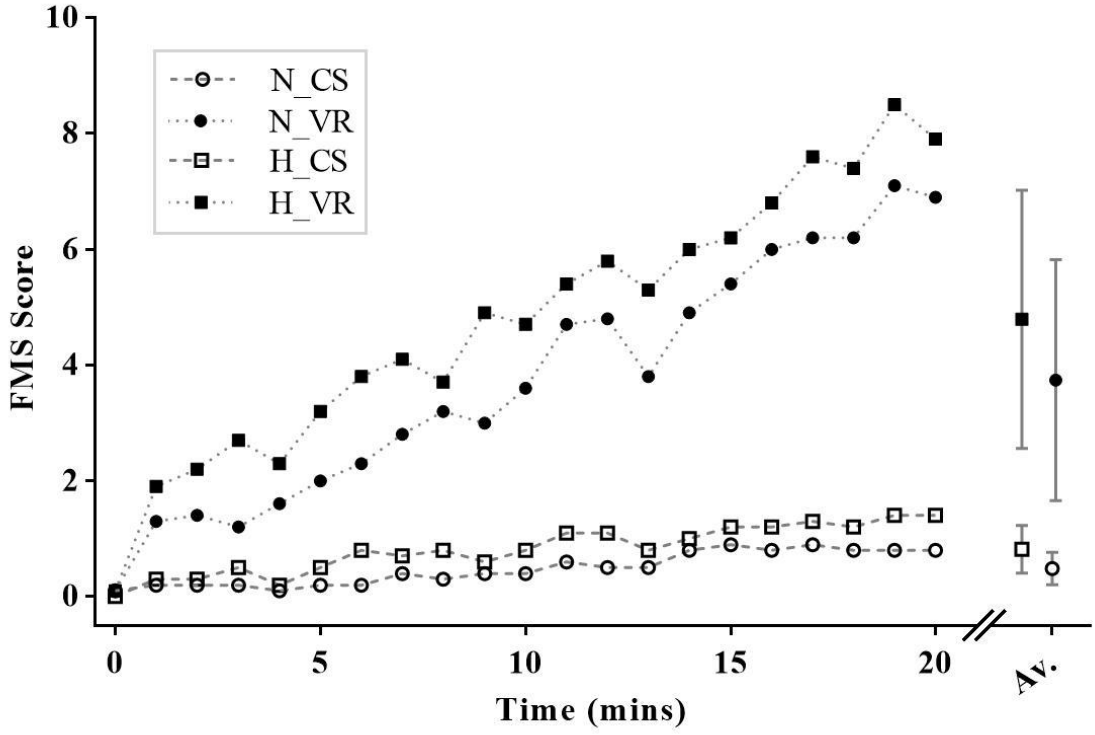

Figure 1: Impact of virtual reality use and air temperature on perceptions of visually induced motion sickness assessed via a Fast Motion Sickness scale.

Note: Data are mean ± SD; n=13. Experimental conditions; N_CS, 22°C Ta with computer screen; N_VR, 22°C Ta with virtual reality headset; H_CS, 35°C Ta with computer screen; H_VR, 35°C Ta with virtual reality headset.

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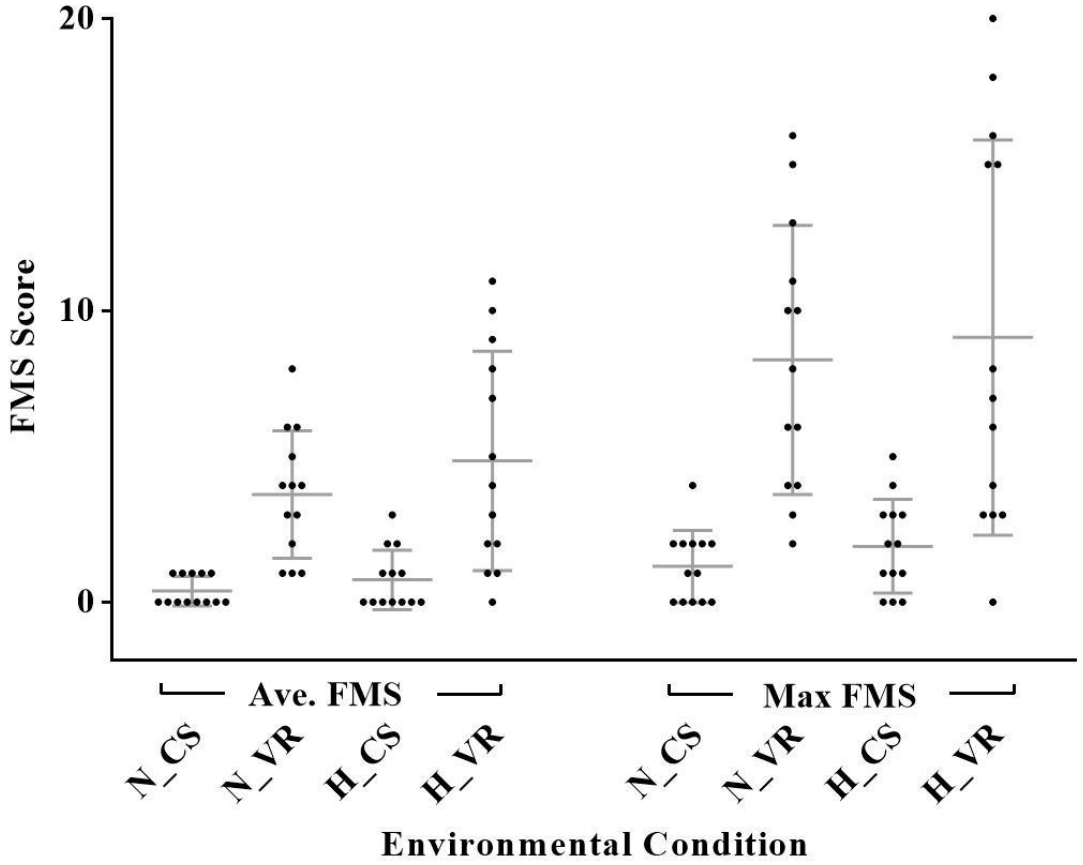

Figure 2: Impact of virtual reality use and air temperature on perceptions of visually induced motion sickness assessed via a Fast Motion Sickness scale.

Note: Data are mean ± SD, with individual data points presented; n=13. Experimental conditions; N_CS, 22°C Ta with computer screen; N_VR, 22°C Ta with virtual reality headset; H_CS, 35°C Ta with computer screen; H_VR, 35°C Ta with virtual reality headset.

On arrival to the laboratory, all participants reported zero ratings of motion sickness via SSQ across all trials. The SSQ ratings immediately following each trial paralleled FMS observations. The results demonstrate significant independent main effects (hypothesis 1 and 2) for VR (*CS trials*, 4.1 [1.8 – 6.5] vs. *VR trials*, 12.8 [6.7 – 19.0]; p=0.001) and air temperature (*N trials*, 7.1 [3.6 – 10.6] vs. *H trials*, 9.9 [4.8 – 15.0]; p=0.04) on reported

motion sickness severity. Again, no significant interaction (hypothesis 3) was observed between VR and air temperature (p=0.07), although a clear trend for synergism was observed. Perceptions of motion sickness diminished across all conditions at 2 hrs post trial (*Fig 3.*), with no main effects observed (p>0.1).

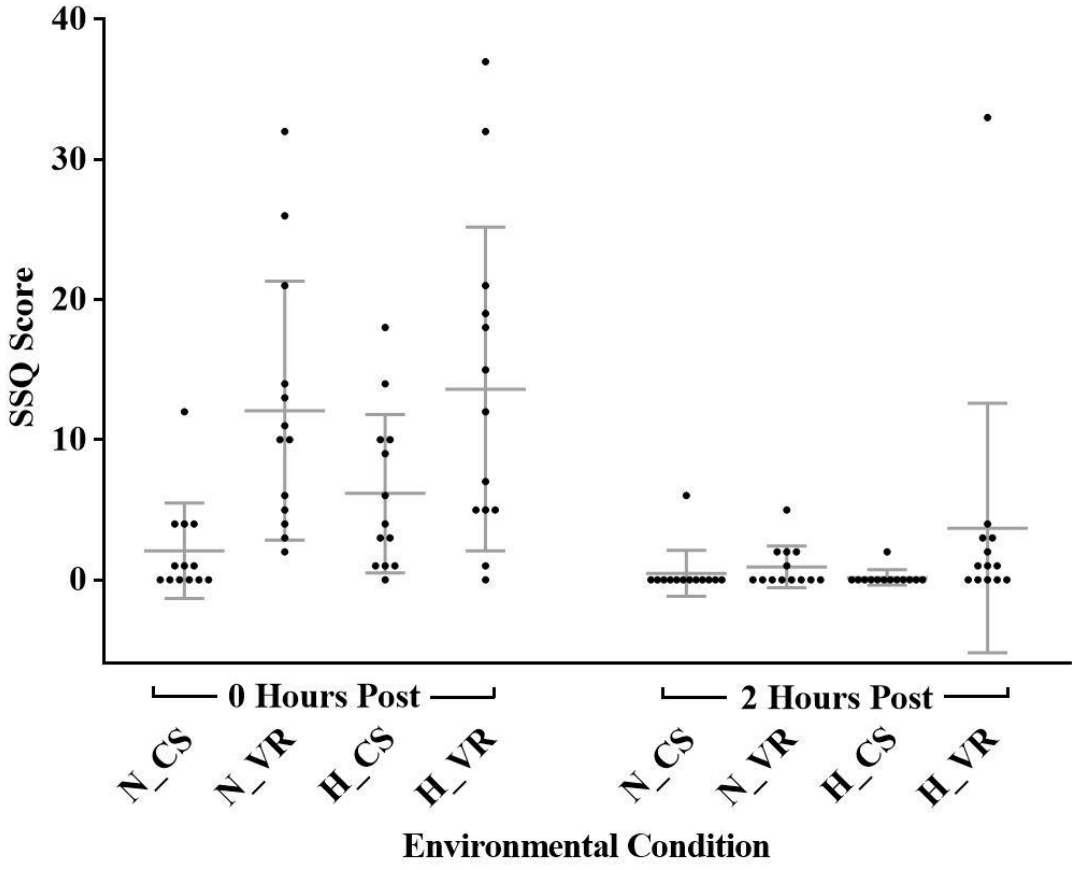

Figure 3: Impact of virtual reality use and air temperature on perceptions of visually induced motion sickness assessed via a Simulator Sickness Questionnaire.

Note: Data are mean ± SD, with individual data points presented; n=13. Experimental conditions; N_CS, 22°C Ta with computer screen; N_VR, 22°C Ta with virtual reality headset; H_CS, 35°C Ta with computer screen; H_VR, 35°C Ta with virtual reality

3.2 Physiological Parameters

No differences ($p > 0.2$) were observed in physiological variables at baseline across experimental conditions upon immediate entry to the room. Independently, VR significantly increased local sweat rate (*CS trials*, 45.6 [36.2 – 54.9] g/m^2/hr vs. *VR trials*, 59.9 [46.6 – 73.2] g/m^2/hr; $p=0.001$) and reduced cutaneous vascular conductance (*CS trials*, 0.34 [0.25 – 0.44] au vs. *VR trials*, 0.26 [0.18 – 0.34] au; $p=0.02$). No independent main effect of VR was seen on any other physiological variables ($p>0.6$). Air temperature significantly increased mean skin temperature (*N trials*, 32.1 [31.6 – 32.7]℃ vs. *H trials*, 34.7 [34.4 – 35.1]℃; $p<0.001$), heart rate (71.3 [68.0 – 74.6] bpm vs. 75.5 [72.3 – 78.6] bpm; $p<0.05$), cutaneous vascular conductance (0.25 [0.17 – 0.33] au vs. 0.36 [0.25 – 0.46] au; $p<0.05$) and local sweat rate (37.4 [31.2 – 43.7] g/m^2/hr vs. 68.0 [50.8 – 85.2] g/m^2/hr; $p<0.001$). No independent main effects of air temperature were observed in mean arterial pressure or rectal temperature ($p>0.1$). Finally, no interactions were observed between air temperature and VR across any of the measured variables ($p>0.1$) (*Table 1*). However, as seen in *Table 1*, a noticeable difference in the magnitude of change between H_CS and H_VR, compared to N_CS and N_VR was observed for sweat rate data (H_VR - H_CS Δ, 18.2 vs. N_VR - N_CS Δ, 10.5). The was also observed for CVC data (H_VR - H_CS Δ, 11.6 vs. N_VR - N_CS Δ, 2.5). Peak values for all physiological variables reflect the patterns seen in mean values. Taken together, the data supports a trend for synergism when combining heat and VR on skin blood flow and local sweating responses.

Table 1: Impact of virtual reality use and air temperature on assessed physiological parameters.

	N _CS	N_VR	H_CS	H_VR	Effects
Heart Rate (bpm)	72 ± 7	71 ± 8	74 ± 7	77 ± 6	T_a
Systolic Blood Pressure (mm Hg)	121 ± 12	121 ± 13	120 ± 12	120 ± 10	
Diastolic Blood Pressure (mm Hg)	75 ± 6	73 ± 7	73 ± 6	74 ± 7	
Skin Temperature (°C)	31.9 ± 1.0	32.3 ± 1.1	34.7 ± 0.7	34.8 ± 0.5	T_a
Rectal Temperature (°C)	37.1 ± 0.2	37.2 ± 0.2	37.2 ± 0.3	37.0 ± 0.4	
Local Sweat Rate (g/m^2/hr)	32.2 ± 9.8	42.7 ± 14.8	58.9 ± 24.0	77.1 ± 30.6	T_a, *VR*
Cutaneous Vascular Conductance	23.5 ± 13.8	21.0 ± 12.9	37.1 ± 21.4	25.5 ± 11.9	T_a, *VR*

Note: Data are mean ± SD. Main effects determined via paired sampled T-test for air temperature (T_a) and Visual Stimulus (*VR*), and repeated measures ANOVA for interaction (T_a**VR*); *n*=13. Experimental conditions; N_CS, 22°C T_a with computer screen; N_VR, 22°C T_a with virtual reality headset; H_CS, 35°C T_a with computer screen; H_VR, 35°C T_a with virtual reality headset.

4.0 DISCUSSION

This study aimed to assess the combined and differential impact of a hot air temperature and virtual reality on motion sickness severity and the associated thermoeffectors (sweating, vasomotor activity). In accordance with the aim, three hypotheses are discussed:

4.1 Hypothesis 1 – Independently of T_a, VR use would elicit motion sickness

In agreement with hypothesis 1, the results of the present study highlight a main effect of VR, indicating a significant increase in perceptions of VIMS with VR use relative to a 2D computer screen control. This finding supports previous research, including Akiduki et al. [3] and Ohyama et al. [4], who also demonstrated evidence for VIMS with VR; a consideration for both providers and users of VR. It appears that symptoms of VIMS may present themselves with as little as 10-20 mins of provocative VR exposure, aligning with previous data [26]. Importantly, this finding shows that the VR stimulus utilised within the current study was sufficient to elicit VIMS in the present participant cohort.

Findings across physiological parameters provide additional support for hypothesis 1. An increase in sweat rate was seen with VR use, highlighting the presence of 'cold-sweating', a well-documented symptom of motions sickness. Indeed, a correlation of sweat rate with nausea and VIMS has previously been determined at $r = 0.67$, $p < 0.01$ (Nalivaiko, Rudd and So, 2014). Numerous other studies have documented increased sweating response during motion sickness [27–30]. Interestingly, the current investigation reported a significant reduction in CVC with VR use compared to a CS control, perhaps resulting from increased sympathetic nervous drive during sickness. Previous literature addressing the link between motion sickness and CVC remains inconclusive [20], with some studies highlighting a reduction in CVC during exposure to a provocative stimulus [31] and others highlighting an increase in CVC [19,32]. No changes were observed in rectal temperature, likely due to the short nature of each experimental trial.

4.2 Hypothesis 2 – Independently of VR, hot conditions would not elicit motion sickness

Physiological parameters responded in a manner expected relative to increases in air temperature [33]. Results showed significant increases in the sweat rate, skin blood flow, skin temperature and heart rate in hot conditions relative to the neutral control. No change was seen in rectal temperature across trials due the short exposure time used in the present study.

Alternatively, mixed findings were observed in accordance with hypothesis 2; in agreement, no main effect was observed for air temperature on FMS ratings, however, a significant main effect for air temperature was seen in SSQ scores. Though there appears to be no clear physiological rationale for which hot air conditions alone may directly elicit motion sickness, one must consider whether general feelings of discomfort that individuals experienced as a result of heat exposure inadvertently translated across to subjective motion sickness symptom

reporting. Indeed, as part of the 16-point SSQ to assess VIMS, individuals were asked to rate their perceptions of 'general discomfort', 'sweating' and 'fatigue'; symptoms which are elevated under hot conditions. While all participants were asked to only report on motion sickness related symptoms, it is not always possible for participants to delineate the effects of thermoregulatory sweating and discomfort, against that of VIMS induced symptoms. Finally, attention paid to psychological constructs may help further to explain increased SSQ ratings with heat alone in the current study. Conceivably, anxiety would increase with the discomfort experienced during hot trials. Indeed, considering the symptoms of anxiety, which also include increased stomach upset, dizziness and heart rate, one could consider anxiety as another rationale as to why SSQ reporting increased in hot conditions alongside the spill over of thermoregulatory responses [34]. Whilst the links between trait anxiety and motion sickness have been previous explored [35,36], little research has investigated whether state anxiety in response to heat stress may also add to the onset of motion sickness.

4.3 Hypothesis 3 - Combined effect of heat and VR on motion sickness

The findings of the present study do not support hypothesis 3, providing no clear evidence for a synergistic interaction between increased air temperature and VR. Notwithstanding, a noticeable difference in the magnitude of change between H_CS and H_VR trials, compared to N_CS and N_VR trials for FMS and SSQ, indicates potential signs of synergism in some, but not all participants. Indeed, a closer look at individual FMS and SSQ data shows partial evidence for responders and non-responders (Fig 2 & 3). This is supported by the findings of many large scale studies which report disparity in the motion sickness susceptibility across individuals exposed to an identical provocative stimulus, either artificial or true motion [10,37]. Indeed, the presence of non-responders in the study cohort limits the power available for the interaction statistic, thereby potentially masking any synergistic effects of heat on VR

induced motion sickness. Note, due the limited sample size within the current study, it was not possible to partition the data into groups to explore this further. A repeat of the current investigation, utilizing only individuals who are known to 'respond' to motion sickness would provide an intriguing area of investigation; however, this may also reduce the generalisability of the results to a wider population of users.

It is also plausible that modifications made to the magnitude and type of visual VR stimulus may in turn increase the magnitude and consistency of any potential interaction between VR induced vection and heat. Though it was conjectured that a fast-moving motor sport video sequence via VR would elicit vection and subsequently motion sickness, in addition to the positive findings discussed for hypothesis 1, future investigations may wish to trial a video sequence with a wider array of planes of motion; e.g. motion experienced on a rollercoaster or simulated human movement through a range of obstacles. In support, Bonato and colleagues [23,38] found that some VR stimuli are effective in evoking VIMS, while others are less so. Note, the extent of vection directly experienced by participants was not assessed in the current investigation, thus should be included in future research. Investigation into the combined stress of heat and true motion, in place of VR, for direct comparison to the current study, also provides an intriguing area for future exploration.

Physiological parameters do not provide unequivocal evidence for synergism between T_a and VR. Yet, in parallel with the subjective motion sickness findings, analysis of sweat rate and cutaneous vascular conductance show potential trends towards a synergistic interaction between heat exposure and VR use. As seen in Table 1, a difference in the magnitude of change between H_CS vs. H_VR trials, compared with N_CS vs. N_VR trials, shows evidence for increased sudomotor activity when VR and heat are combined. Interestingly,

vasomotor activity showed conflict between the vasoconstriction induced by VR and vasodilatation induced by heat. In view of the toxic hypothesis, such findings are intriguing and worth further investigation; yet due to the large inter-individual variation across the current data set, an unequivocal conclusion is not possible.

4.4 Conclusion

This investigation assessed the individual and combined impact of VR use and a hot air temperature on motion sickness severity. Independently, VR evoked a significant increase in self-reported motion sickness. Nonetheless, the data herein does not provide unequivocal evidence of a clear synergistic interaction between VR and T_a. While definitive evidence for a synergistic interaction was not obtained, potential trends were identified that warrant further investigation. Considerable variation was seen in the inter-individual resistance to motion sickness, conceivably limiting the statistical power available for a significant interaction between stressors.

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