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Depersonalisation/derealisation in virtual reality is associated with vestibular sensitivity

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A Part of Sage

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Abstract

Despite significant advancements in Virtual Reality (VR) technology, exposure to VR can induce side effects on the user. One such effect is Depersonalisation and Derealisation (DPDR): feelings of detachment from the self and surroundings. The exact mechanism behind the development of DPDR symptoms following VR use is unknown. Stable representations of the bodily self depend on the integration of visual and vestibular signals. However, VR disrupts this integration. The brain is thought to resolve visuo-vestibular conflict by downweighting vestibular signals. However, whether this reweighting accounts for VR-induced DPDR has not been tested. We investigated whether changes in vestibular sensitivity are associated with DPDR. Thirty-two participants were exposed to 10 min of either roll optic flow, inducing visuo-vestibular conflict, or random dot motion, inducing no conflict. Vestibular sensitivity was assessed before and after VR using a psychophysical detection task. A moderation analysis revealed that in the roll condition, participants whose sensitivity to GVS increased following VR reported the greatest increases in DPDR, whereas no such relationship was seen in the random condition. Thus, these findings suggest that individuals who are unable to resolve the sensory conflict by vestibular-downweighting are most susceptible to DPDR.

Keywords

depersonalisation/derealisation (DPDR), virtual reality, visuo-vestibular conflict, vestibular sensitivity, galvanic vestibular stimulation, multisensory reweighting

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Introduction

Virtual Reality (VR) has become more integrated into our everyday life, with applications spanning entertainment, education, clinical rehabilitation, and professional training.¹ Yet despite rapid improvements in display resolution, frame rates, and body tracking, users still report unwanted side effects that continue after the headset is removed.^{2–4} These can vary from visual disturbances and altered depth perception to disruptions in body ownership and a need to verify the reality of one's surroundings through touch.⁵ VR aftereffects also spread to cognitive and sensorimotor domains: cognitive performance and reaction times can be impaired following exposure, even after cybersickness symptoms have resolved.⁶ Among the more disconcerting of these aftereffects is the experience of Depersonalisation and Derealisation (DPDR): a subjective sense of detachment from the body, self, and surroundings that can happen upon returning to the real world from a virtual one.^{7,8}

Depersonalisation refers to feelings of unreality and disconnection from the self, while Derealisation describes the experience of the external world appearing strange, distant, or dreamlike. In clinical settings, transient DPDR is extremely common; it is estimated that up to 74% of the general population will experience at least one episode in their lifetime, although only 1 to 2% develop symptoms that require clinical intervention.⁹ VR appears to be a potent trigger for these experiences. A retrospective survey of VR users found that almost 84% reported DPDR symptoms after its use,¹⁰ and symptoms are most common among newer and female users.¹¹ Importantly, compared to traditional screen-based gaming, DPDR symptoms are

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significantly increased after VR exposure, suggesting that something inherent VR's immersive qualities is responsible.⁸

What drives DPDR after VR is still less understood. One explanation is that DPDR results from the subjective sense of "being there" in the virtual world, or the illusion of presence.¹² The reasoning is simple: the more immersed you feel in the VR, the more jarring it may feel coming back to reality, which can trigger feelings of detachment and unreality. Presence itself may be made of at least two subcomponents.¹³ The Place Illusion (PI) is the sense of being spatially located inside the virtual world. It relies on sensory contingencies similar to those we expect in the real world, for instance a stable visual scene during head movements and a wide field of view. The Plausibility Illusion (Psi) is the sense that events in the virtual world are really happening. This is driven by realistic illumination, physical plausibility, and interactive responsiveness.^{12,14} This is supported by psychophysiological studies: high levels of Presence are connected with increased bodily responsiveness to virtual stimuli, including elevated heart rate and skin conductance when encountering anxiety-evoking scenarios.^{15,16} However, while a link between Presence and DPDR is plausible, it has not been determined which specific component of Presence, if any, drives symptoms.

Moreover, it may be possible that DPDR arises not from Presence itself, but from more fundamental sensory processes that bind sensory information for self-motion. During physical movement, vision and the vestibular system work closely together with proprioceptive and tactile inputs to construct a coherent representation of self-motion in space.^{17–22} However, VR exposure breaks this visuo-vestibular integration. In VR, vision can indicate that the user is moving while the vestibular system indicates that the user is stationary. For example, while driving through a simulated environment, the user will receive optic flow information that they are accelerating and turning, however, their vestibular senses will indicate that they are not moving.²³ This difference creates a visuo-vestibular conflict, widely regarded as a primary driver of cybersickness,²⁴ and can also reduce Presence.²⁵ The severity of cybersickness symptoms increases with the magnitude of this conflict,²⁶ and symptoms typically develop within the first minutes of VR exposure.^{27,28} Improvements in VR display quality have not reduced the problem; in fact, more realistic VR environments can produce even greater symptoms, which may be due to richer optic flow generating a stronger mismatch with the absent vestibular input.^{29–31}

The brain can resolve sensory conflicts by reweighting signals depending on their reliability, placing greater weight on the more reliable modality.^{32–34} This concept extends to visuo-vestibular integration for self-motion, where humans shift reliance between visual and vestibular input depending

on signal coherence.^{18,35–37} In VR the vestibular signals are uninformative because the user is stationary, while visual signals are highly informative and coherent. The expected outcome is a "downweighting" of vestibular input and a lower reliance on vestibular information to resolve the conflict.^{23,38}

There is previous evidence that this vestibular-downweighting occurs both during and after VR exposure. Exposure tovection-inducing optic flow in VR has been shown to change Vestibular-Evoked Myogenic Potential (VEMP) asymmetry ratios, with increased amplitude on the left sternocleidomastoid muscle after just one minute of exposure.³⁹ This suggests that visuo-vestibular conflict rapidly affects otolith-mediated vestibular reflexes. The ability to detect vestibular stimulation has also been shown to be reduced after exposure to optic flow in VR, but only when the visual and vestibular signals are presented on the same plane of motion, consistent with the predictions of optimal multisensory integration.⁴⁰ These effects appear to extend beyond unimodal vestibular processing as well, as visuo-vestibular conflict presented within the roll plane has also been shown to reduce sensitivity to multisensory verticality percepts.⁴¹ Finally, previous studies have reported reduced vestibulo-ocular reflex gain⁴² and impaired eye-head-hand coordination³ following VR exposure, further strengthening the notion of vestibular-downweighting as a significant aftereffect.

If VR triggers vestibular-downweighting, could this also account for VR-induced DPDR? Evidence from clinical populations suggests it might. Patients with peripheral vestibular disease report DPDR symptoms at significantly higher rates than healthy controls,^{43,44} and those who have more severe symptoms are impaired at updating their spatial orientation independent of their dizziness.⁴⁵ Elyoseph et al. (2023) proposed that this occurs through a cascade in which vestibular disruption leads to spatial disorientation and anxiety, which in turn gives rise to DPDR.⁴⁶ More broadly, the vestibular system contributes to bodily self-consciousness, including body ownership and self-location,^{20,47} and a recent systematic review has confirmed the association between vestibular dysfunction and DPDR across both patient and healthy populations.⁴⁸ These findings raise the possibility that vestibular-downweighting induced by visuo-vestibular conflict in VR drives DPDR through similar mechanisms.

However, a direct test of this hypothesis has not been conducted. Previous studies examining VR aftereffects have typically used immersive virtual environments with rich visual scenes, interactive elements, and spatial cues, all of which contribute to the illusion of Presence.¹² As a result, it has not been possible to determine whether DPDR is caused by visuo-vestibular conflict and the associated vestibular-downweighting, from the Presence illusion itself, or from some interaction between the two. Here, we investigated whether visuo-vestibular conflict in VR

modulates vestibular sensitivity, and whether such modulation is associated with DPDR symptoms. We used VR stimuli that only induced visuo-vestibular conflict through optic flow but contained no cues for Place Illusion or Plausibility Illusion. This allowed us to isolate the contribution of sensory conflict from Presence. Vestibular sensitivity was measured before and after VR exposure using a psychophysical detection task based on Galvanic Vestibular Stimulation (GVS). We predicted that exposure to visuo-vestibular conflict would reduce vestibular sensitivity, and that this reduction would be associated with increased DPDR.

Methods

Ethics

The experimental protocol was approved by local ethics committee. The study was conducted in line with the Declaration of Helsinki. Written informed consent was obtained from participants prior to commencing the study.

Participants

Power analysis with G*Power suggested a minimum sample size of 24 participants for a within-between ANOVA interaction effect, a medium-large effect size ($f = 0.3$, based on⁴⁰) with 80% power and $\alpha = .05$. To account for dropouts and exclusions, total of 42 participants (32 female, 8 male, 1 genderfluid, 1 non-binary, mean age = 20.67, $SD = 5.34$) were recruited from the School of Psychology Research Participation Scheme (RPS). Eight participants (6 female, 2 male, mean age = 19.25, $SD = 0.88$) whose d' score in the pre-VR condition was less than zero (indicating poor understanding of task instructions and/or poor sensitivity to GVS) were replaced prior to final analysis, leaving a final sample of 34 participants. Two further participants (both female, aged 18 and 20) were excluded at the analysis phase due to extreme baseline (first participant baseline score = 740) and pre-post changes (pre = 1, post = 821) in Cambridge Depersonalisation Scale (CDS) scores,⁴⁹ which significantly influenced the statistical model according to Cook's Distance.

The final sample comprised 32 participants (24 female, 6 male, 1 genderfluid, 1 non-binary, M age = 21.13, $SD = 6.05$), randomly allocated to either the Roll ($n = 17$) or Random ($n = 15$) condition. Exclusion criteria were any history of neurological, psychiatric, hearing, or vestibular disorders, epilepsy or family history of epilepsy, or any contraindications to GVS. No specific vestibular assessments were undertaken prior to participation, but no participants reported any vestibular/balance conditions during screening.

Twenty-nine participants were right-handed according to their Edinburgh Handedness Inventory (EHI⁵⁰) scores,

two were left-handed, and one had no preference. Motion sickness susceptibility was assessed using the Motion Sickness Susceptibility Scale Short Form (MSSQ-Short⁵¹), which estimates susceptibility to motion sickness from self-reported history of sickness across various forms of transport and motion. MSSQ-Short scores were similar across conditions, with the Roll group ($N = 17$) scoring $M = 3.28$ ($SD = 1.16$) and the Random group ($N = 15$) scoring $M = 3.58$ ($SD = 1.11$), both indicating low-to-moderate susceptibility to motion sickness. Prior gaming and VR experience were assessed using a brief Gaming Experience Questionnaire (GEQ) which asked participants about their frequency of computer use, prior VR use, and gaming habits. Most participants reported prior computer experience ($n = 27$, 84%) and approximately two-thirds had previous VR experience ($n = 21$, 66%; 12 in the random condition, 10 in the roll condition). Of those who had previously used VR, this was reported as rarely (less once per week). Gaming frequency was generally low, with most participants playing less than once per week ($n = 13$, 41%).

Design

A 2 x 2 mixed design was used, with Time (Pre-VR vs. Post-VR) as a within-subjects factor and Condition (Roll vs. Random) as a between-subjects factor.

Measures

Galvanic Vestibular Stimulation and detection task. Two commercial DC stimulators (NeuroConn GmbH, Ilmenau, Germany; The Magstim Company Ltd., Whitland, Wales, UK) were used to deliver GVS and sham stimulation. Carbon rubber electrodes measuring approximately 4 cm² were coated in NaCl electrode gel and placed on the left mastoid (anode) and right mastoid (cathode) in a binaural-bipolar configuration for GVS delivery, and on the base of the neck for sham delivery (Figure 1(c)). Both GVS and Sham were administered at the same intensities using sinusoidal waveform of 5 s duration with an amplitude of 1.4 mA at 2.00 Hz. A sham stimulation condition was used to control for non-specific sensations associated with electrode application. Electrodes placed on the base of the neck deliver cutaneous sensations comparable to those produced by GVS, without activating the vestibular nerve and therefore without inducing any sensation of head movement or rotation.

Vestibular sensitivity to GVS was assessed using two-alternative forced-choice detection task. The task consisted of 60 trials (30 GVS, 30 sham), presented in a randomised order. Each trial began with a high-pitched short tone indicating stimulus onset and concluded with a low-pitched longer tone indicating stimulus offset. Participants pressed "L" on a keyboard if they perceived a sensation of head movement or "S" if they perceived no head movement,

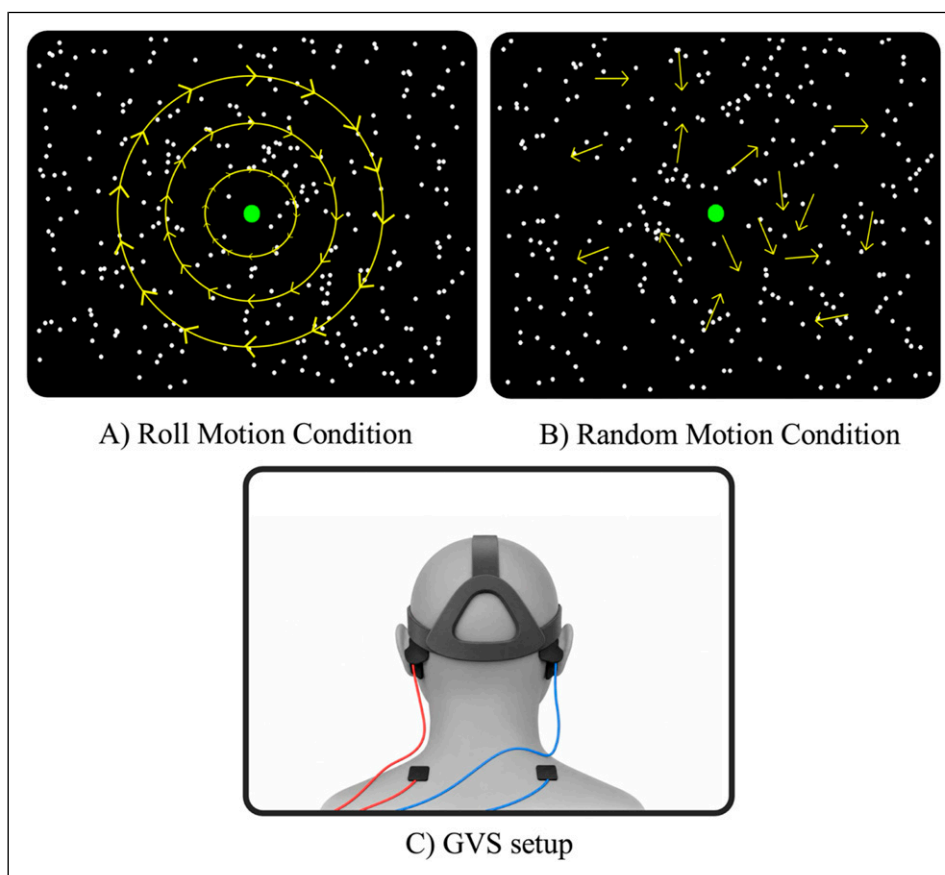


Figure 1. Experimental setup and visual stimuli. (a) Roll/Conflict condition: white dots rotated around the central fixation point, producing optic flow consistent with roll rotation and inducing a visuo-vestibular conflict. (b) Random/No Conflict condition: white dots moved randomly on a black background with no coherent self-motion signal. Arrows indicate the direction of dot motion. A stationary green fixation dot was displayed at the centre in both conditions. Neither stimulus contained cues for Place Illusion or Plausibility Illusion (c) Participants wore a Meta Quest 3 head-mounted display (HMD). Carbon rubber electrodes were placed on the left mastoid (anode) and right mastoid (cathode) in a binaural-bipolar configuration for GVS delivery, and on the base of the neck (approximately between C7 and T1) for sham delivery.

responding only after the second tone. The task lasted approximately 15 min per administration and was completed both before and after VR exposure.

Visual stimuli. Visual stimuli were presented using a Meta Quest 3 head-mounted display (HMD). While other VR displays such as Oculus, CAVEs or surround screens may also induce visuo-vestibular conflicts,^{39,52} the present study focused specifically on stimuli delivered via an HMD. Participants viewed a full-field pattern of moving white dots on a black background (Figure 1) with a stationary green fixation dot at the centre. These stimuli were created in MATLAB R2024b with Psychtoolbox (version 3.0.19) and exported as videos (1920 × 1080 resolution, 30 FPS). In the Roll/Conflict condition, 1000 white dots on a black background rotated continuously in an anticlockwise direction around the central fixation point at approximately 90°/s, producing optic flow consistent with roll rotation (Figure 1(a)) and thereby inducing a visuo-vestibular conflict, since vision signalled rotation

while the vestibular system registered no corresponding head movement. In the Random/No Conflict condition (Figure 1(b)), each dot moved on a random trajectory, providing no coherent signal for self-motion and thus producing no visuo-vestibular conflict. These stimuli were chosen on the basis of previous research indicating that roll optokinetic stimulation can modulate sensitivity to vestibular signals⁴⁰ and verticality.^{41,53,54} However, it remains an interesting avenue for future research to investigate whether other planes of motion and/or more complex dynamic visual stimuli would produce similar effects. Critically, neither stimulus contained cues sufficient to generate Place Illusion or Plausibility Illusion, minimising the contribution of Presence to any observed effects on DPDR. Participants were instructed to fixate on the central green dot throughout the VR exposure.

Simulator sickness. Simulator sickness was assessed using the Simulator Sickness Questionnaire (SSQ;⁵⁵). The SSQ comprises 16 items rated on a 4-point scale (0 = none, 3 =

severe), measuring symptoms across three subscales: Nausea, Oculomotor, and Disorientation. A Total SSQ score was computed using the standard weightings, with higher scores indicating greater simulator sickness. The SSQ was administered before VR exposure (pre-VR) and following the post-VR detection task (post-VR).

Depersonalisation/derealisation. Depersonalisation/derealisation (DPDR) was measured using the State-Version of the Cambridge Depersonalisation Scale (CDS;⁴⁹). The state CDS comprises 22 items assessing the present intensity of depersonalisation and derealisation experiences, such as feelings of unreality, emotional numbing, and altered body perception (Table 1). Each item is rated on a visual analogue scale (VAS) ranging from 0% (“I’m not having it at all”) to 100% (“It’s as bad as it can get”). A total CDS score was computed by summing responses across all 22 items, yielding a possible range of 0 to 2200, with higher scores indicating greater DPDR symptomatology. The CDS was administered immediately prior to detection task (pre-VR) and immediately following the post-VR detection task (post-VR).

Presence. Presence was assessed using the Slater-Usold-Steed (SUS) questionnaire,⁵⁶ administered immediately after VR exposure. The SUS comprises six items measuring the subjective sense of “being there” in the virtual environment. Each item is rated on a 7-point scale, with higher scores indicating a greater sense of presence. A mean score was computed across all six items for each participant,

yielding a single Presence score on a 1 to 7 scale. This mean Presence score was used as the Presence predictor in subsequent analyses.

Procedure

Participants were informed of the study procedures and any potential discomfort associated with GVS. Informed consent was obtained, after which participants completed the EHI, MSSQ-Short, GEQ, SSQ (pre-VR), and CDS (pre-VR).

GVS and sham electrodes were then placed on the participants. Participants were told that they might feel sensations of skin tingling and faint head movements, but no information was provided regarding the difference between GVS and Sham stimulation. Participants then completed the pre-VR detection task as described previously. Following the detection task, participants donned the Meta Quest 3 HMD and viewed their assigned visual condition (Roll or Random) for 10 minutes. Immediately after VR exposure, participants closed their eyes and were blindfolded by the experimenter before completing the post-VR detection task using the same parameters. Following this, the blindfold was removed and participants completed the SSQ (post-VR), CDS (post-VR), and SUS Presence questionnaire before being fully debriefed.

Data analysis

The responses from the detection task were classified according to signal detection theory (SDT) as hits (“Head

Table 1. CDS-S items.⁴⁹

1. I'm feeling strange, as if I were not real or as if I were cut off from the world.
2. Things around me are now looking “flat” or “lifeless,” as if I were looking at a picture.
3. I am feeling as if parts of my body don't belong to me.
4. I'm now having the feeling of being a “detached observer” of myself.
5. My body is feeling very light now, as if I were floating on air.
6. I am not feeling any emotions at all.
7. If I read this sentence aloud, my voice sounds remote and unreal.
8. I am having a feeling of complete emptiness in my head so that I am not having any thoughts at all.
9. I'm having the feeling that my hands or my feet have become larger or smaller.
10. My surroundings are feeling detached or unreal, as if there was a veil or a fog between me and the outside world.
11. It seems now as if things that I have recently done took place a long time ago. For example, anything which I have done this morning feels as if it were done weeks ago.
12. If I try to remember important events in my life (e.g. graduation, wedding etc.), I feel so detached from the memories that it seems as if I had not been involved in them.
13. I don't seem to be feeling any affection towards my family or close friends.
14. Objects around me are looking smaller or further away.
15. I cannot feel properly the pencil that I have in my hand, as if it were not me who were holding it.
16. If I now try to imagine the face of a relative or friend whom I frequently see (but who is not with me at the present), I do not seem able to picture it in my mind.
17. If I pinch myself in my own arm now, I feel so detached from the pain that it feels as if it were “somebody else's pain.”
18. I am now having the feeling of being outside of my body.
19. I am feeling as if I were not in charge of my movements, so that I feel “automatic” and mechanical, as if I were a “robot.”
20. I am feeling so detached from my thoughts that they seem to have a “life” on their own.
21. I feel like touching myself to make sure that I have a body or a real existence.
22. I am still having the same strange feelings as when I stated to answer this questionnaire.

moved” response + GVS stimulus), false alarms (“Head moved” response + Sham stimulus), misses (“No head movement” response + GVS stimulus), or correct rejections (“No head movement” response + Sham stimulus).

Hit rate was calculated as the proportion of GVS trials correctly identified as GVS:

$$p_{Hit} = \frac{hits}{n_{GVS}}$$

where n_{GVS} is the total number of GVS trials. Where the number of hits was equal to zero, the hit rate was adjusted to $\frac{0.5}{n_{GVS}}$, and where the number of hits was equal to n_{GVS} , the hit rate was adjusted to $\frac{hits-0.5}{n_{GVS}}$. False alarm rate was calculated as the proportion of sham trials incorrectly identified as GVS:

$$p_{FA} = \frac{FA}{n_{Sham}}$$

where n_{Sham} is the total number of sham trials. Where the number of false alarms was equal to zero, the false alarm rate was adjusted to $\frac{0.5}{n_{Sham}}$ and where the number of false alarms was equal to n_{Sham} , the false alarm rate was adjusted to $\frac{FA-0.5}{n_{Sham}}$. These adjustments were applied to avoid proportions of 0 or 1, which would produce undefined z-scores.⁵⁷

Vestibular sensitivity was indexed by d' , calculated as:

$$d' = z(p_{Hit}) - z(p_{FA})$$

where z denotes the inverse of the cumulative standard normal distribution function.⁵⁸ A higher d' reflects greater ability to discriminate GVS from sham stimulation, and thus greater vestibular sensitivity. Response bias was indexed by criterion (c), calculated as:

$$c = -\frac{[z(p_{Hit}) + z(p_{FA})]}{2}$$

Positive values of c indicate a conservative bias (tendency to respond “sham”), and negative values indicate a liberal bias (tendency to respond “GVS”).

Participants whose pre-VR d' was less than zero were excluded from analysis, as they were unable to distinguish GVS from sham stimulation at baseline. These participants were replaced prior to final analysis to ensure an adequate sample size. In addition, two further participants were excluded from analysis due to extreme changes in DPDR

scores. A moderated linear regression analysis was used to explore the relationship between Presence, Vestibular Sensitivity and DPDR.

Results

Descriptive statistics for vestibular sensitivity (d'), response bias (criterion), and DPDR pre-post and mean presence in each condition can be seen in Table 2:

Vestibular sensitivity

A 2×2 Mixed ANOVA with factors Time (Pre vs Post, within-subjects) and Condition (Random vs Roll, between-subjects) was conducted to investigate changes in vestibular sensitivity. No significant main effect of Time ($F(1,30) = 2.52, p = .12, \eta^2 = .012$), Condition ($F(1,30) = 0.62, p = .44, \eta^2 = .017$), or interaction ($F(1,30) = 0.41, p = .52, \eta^2 = .002$) were found.

Response bias

A 2×2 Mixed ANOVA with factors Time (Pre vs Post, within-subjects) and Condition (Random vs Roll, between-subjects) was conducted to investigate changes in response bias. No significant main effect of Time ($F(1,30) = 0.11, p = .74, \eta^2 = .001$), Condition ($F(1,30) = 0.09, p = .76, \eta^2 = .002$), or interaction ($F(1,30) = .002, p = .96, \eta^2 = .00002$) were found.

Presence

An independent t-test was used to investigate differences in mean Presence scores between Roll and Random conditions. A significant difference was found ($t^{30} = 2.38, p = .02$, Cohen's $d = .84$). Mean presence was higher in the Roll (3.36) vs Random (2.61) condition. However, as expected, Presence scores were generally low, reflecting the sparse nature of the visual stimulus.

Depersonalisation/derealisation

The change in DPDR scores by condition and individual CDS-S Item can be seen in Figure 2. The majority of individual CDS-S items showed at least ~5 point increases on average following VR exposure. Specific items which showed the largest increases in both conditions included

Table 2. Mean (SD) vestibular sensitivity, response bias, DPDR and Presence by condition.

Condition	d' pre	d' post	Criterion pre	Criterion post	DPDR pre	DPDR post	Presence
Random	2.52 (1.09)	2.36 (1.21)	0.30 (0.33)	0.27 (0.41)	95.40 (122.5)	202.00 (208.43)	2.61 (0.93)
Roll	2.95 (1.32)	2.57 (1.31)	0.26 (0.45)	0.23 (0.53)	134.59 (193.72)	288.49 (299.33)	3.36 (0.86)

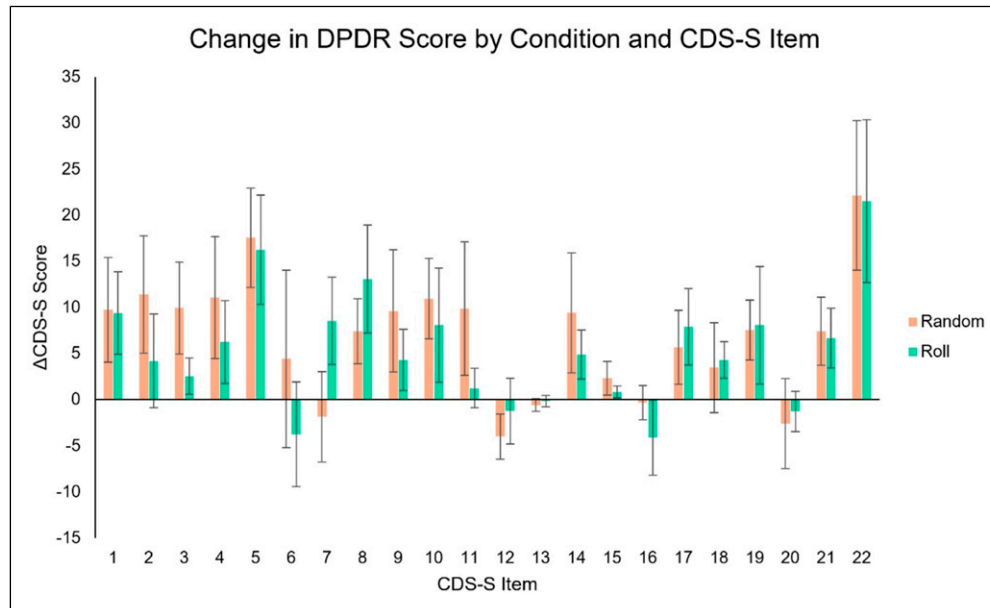


Figure 2. Changes in DPDR score by Condition and CDS-S Item. Δ CDS-S scores were calculated by subtracting pre- from post CDS-Scores, and averages calculated for each condition. Average differences are shown by item and condition. Error bars represent standard error of the mean.

Item 5 (“My body feels very light now, as if I were floating on air”) and Item 1 (“I’m feeling strange, as if I were not real or as if I were cut off from the world.”), indicating that VR could induce general feelings of DPDR and alterations to body-related sensations regardless of condition. Items which did not show much change post vs pre-VR exposure tended to be those relating to emotion and memory. Item 22 (“I am still having the same strange feelings as when I stated to answer this questionnaire.”) showed the largest increase across both conditions, indicating that sensations of DPDR may be longer-lasting than pre-VR. Future research is needed to investigate these specific sensations in more detail. Below, we focus on overall changes in total DPDR scores for analysis.

A 2×2 Mixed ANOVA with factors Time (Pre vs Post, within-subjects) and Condition (Random vs Roll, between-subjects) was conducted to investigate changes in DPDR. A significant main effect of Time ($F(1, 30) = 17.09, p < .001, \eta^2 = .09$) was found, with higher levels of DPDR post (248.19) vs pre (115.75) VR exposure. No main effect of Condition ($F(1, 30) = 0.82, p = .37, \eta^2 = .02$), or interaction ($F(1, 30) = 0.53, p = .47, \eta^2 = .003$) was found.

Relationship between presence, vestibular sensitivity and depersonalisation/derealisation

A moderation analysis was conducted to investigate the relationship between Presence, d' and DPDR scores. The difference in DPDR post-pre-exposure (Δ DPDR) was the outcome variable, while the difference in d' post-pre-

exposure ($\Delta d'$) and mean presence scores were used as predictor variables. The interaction between Condition (Random vs Roll) was used as a moderator variable, thus encompassing two interaction terms (Condition* $\Delta d'$, Condition*Presence). The Random condition was used as the reference group. The Presence and $\Delta d'$ predictors were mean-centred prior to analysis. The model was constructed using the `lm` function in R and RStudio (version RStudio 2026.01.0) specified as:

$$\Delta DPDR = Condition * (\Delta d' + Presence)$$

Estimates for each predictor and interaction can be seen in Table 2. The overall model significantly predicted Δ DPDR ($F^{5,26} = 2.59, p = .049$), explaining approximately 20% of the variance ($R^2_{Adj} = .20$). No significant main effects of Condition (Roll) or Presence were found (Table 3). However, the interaction between Condition and $\Delta d'$ was significant ($B = 160.56, SE = 68.92, t = 2.33, p = .028$). Simple slopes revealed a positive relationship between Δ DPDR and $\Delta d'$ in the Roll condition, such that

Table 3. Model estimates.

	B (SE)	t-value (p)
Intercept	133.66 (47.85)	2.79 (.009)**
Condition (roll)	46.86 (65.42)	0.72 (.48)
$\Delta d'$	-13.66 (50.01)	-.27 (.78)
Presence	61.26 (51.88)	1.18 (.25)
Condition* $\Delta d'$	160.56 (68.92)	2.33 (.028)*
Condition*Presence	-92.35 (74.80)	-1.24 (.23)

participants whose d' increased after exposure to roll rotation had higher Δ DPDR (Figure 3). No relationship between $\Delta d'$ and Δ DPDR was seen in the random condition.

Discussion

Visuo-vestibular conflict is widely considered to be a primary consequence of VR exposure, arising when visual signals indicate self-motion while the vestibular system signals that the individual is stationary.^{23,24} The brain is thought to resolve this conflict through a downweighting of vestibular input, and previous evidence suggests that this process alters vestibular reflexes and perception both during and after VR use.^{39,40,42} Whether vestibular-downweighting also accounts for the DPDR symptoms frequently reported after VR has not previously been tested. The present study addressed this question using VR stimuli that induced visuo-vestibular conflict through optic flow but contained no cues for Place Illusion or Plausibility Illusion, with vestibular sensitivity measured before and after exposure via a GVS detection task. A complex relationship was found between DPDR and vestibular sensitivity, such that participants who became more sensitive to vestibular cues following VR exposure had worse levels of DPDR, but only following visuo-vestibular conflict. These findings suggest that Presence alone is not a sufficient explanation for development of DPDR following VR exposure.

The study revealed several findings. First, a moderation analysis indicated that there was a significant interaction between Condition and $\Delta d'$, such that in the Roll condition, participants whose vestibular sensitivity increased after VR exposure reported the greatest increases in DPDR, with no such relationship observed in the Random condition. Second, there was no significant change in vestibular sensitivity (d') or response bias (criterion) following VR exposure in either condition. Third, DPDR increased

significantly from pre- to post-VR exposure, and this increase did not differ between conditions. Fourth, Presence was significantly higher in the Roll than the Random condition. Each of these findings is discussed in turn.

The most informative finding was from the moderation analysis. The overall model significantly predicted Δ DPDR, explaining approximately 20% of the variance, and a significant interaction between Condition and $\Delta d'$. In the Roll condition, participants who showed increase vestibular sensitivity after VR exposure reported the greatest increases in DPDR. Initially this direction of effect seems counter-intuitive: the vestibular-downweighting hypothesis predicts that reduced vestibular sensitivity should be associated with greater DPDR. If the effect is not explained by simple downweighting, what is causing it? One explanation is the process of vestibular recalibration upon return to the real world. During VR exposure, the brain downweights vestibular signals to resolve the visuo-vestibular conflict.²³ This reweighting is not an immediate but a dynamic, ongoing process of sensory adjustment. When the headset is removed, the sensory world changes abruptly: vestibular signals are once again consistent with the environment, but the system has been recalibrated to ignore them. The brain must therefore upweight vestibular cues to restore accurate self-motion perception. An increase in d' following VR exposure may reflect this compensatory upweighting, as the system shifts towards greater reliance on vestibular input. Increased VEMP amplitude after VR vection has also been interpreted before in a similar way, as representing an upweighting of vestibular cues as they become reliable again.³⁹ Previous research investigating the time-course of changes to verticality sensitivity provide insight into this dynamic reweighting.⁵³ Participants were asked to indicate whether upright or slightly tilted lines were vertical or not during exposure to roll optic flow or random motion over approximately 9 minutes of exposure. Rapid decreases in verticality sensitivity were observed within the first few minutes of exposure to roll optic flow, while no such decrease was seen during random motion. A follow-up study which continued to measure verticality sensitivity once the conflicting roll stimulus was removed indicated a rapid recovery of sensitivity, suggesting swift recalibration of sensory cues.⁵³ However, whether a similar process is present for sensitivity to vestibular stimulation as in the present study requires further research. The finding that greater vestibular sensitivity was associated with greater DPDR in the Roll condition suggests that it is the recalibration process itself, rather than the loss of vestibular sensitivity, that drives dissociative experiences.

Existing clinical evidence suggests clear associations between vestibular (dys)function and DPDR symptoms. Patients with peripheral vestibular disease experience DPDR more than healthy controls,^{43,44} and a recent systematic review has confirmed these findings in both patient and healthy populations.⁴⁸ This work suggests that

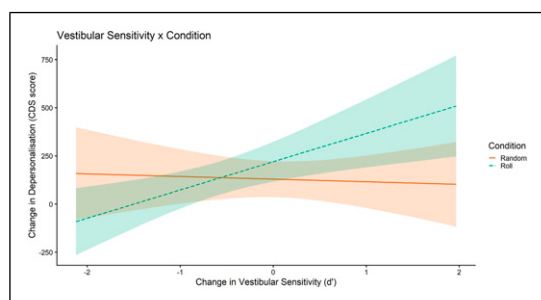


Figure 3. Simple slopes from the moderation analysis showing the relationship between change in vestibular sensitivity ($\Delta d'$, post minus pre) and change in DPDR (Δ CDS score, post minus pre) by condition. In the Roll condition, participants whose vestibular sensitivity increased after VR exposure showed greater increases in DPDR (significant positive slope). No such relationship was observed in the Random condition. Shaded regions indicate 95% confidence intervals.

vestibular disruption triggers DPDR through a sequential process which involves spatial disorientation and anxiety rather than dizziness alone.^{45,46} However, this clinical picture reflects a state of chronic vestibular loss closer to persistent downweighting than to the dynamic upweighting we propose here. Whether vestibular signals are chronically lost (clinical populations) or are actively recalibrating after recent conflict (the present study), the result is the same: the vestibular contribution to multisensory self-representation is destabilised. These findings thus strengthen the concept that the vestibular system plays a central role in bodily self-consciousness, including the sense of body ownership and self-location.^{20,47}

Importantly, neural representations of vestibular signals have significant overlap with regions implicated in bodily self-consciousness, supporting the above interpretation. The parieto-insular vestibular cortex (PIVC) integrates vestibular input with visual and somatosensory signals, while the neighbouring temporo-parietal junction (TPJ) helps locate the self within the body and in space.^{20,59} Disrupting these regions can alter bodily self-consciousness, including out-of-body experiences and feelings of detachment from the self.^{60,61} The posterior insula links vestibular processing to interoception and the sense of one's own bodily state.⁴⁷ Since the regions that process vestibular signals are also those that anchor the self in the body, a vestibular system still recalibrating after VR exposure might, for example, feed unstable input into them. This could explain why vestibular readjustment tracked with DPDR in the present study, and future neuroimaging could test it directly.

Although the moderation analysis revealed clear individual differences in vestibular change, at the group level there was no significant change in vestibular sensitivity (d') or response bias (criterion) after VR exposure in either condition. This contradicts with previous work showing that visuo-vestibular conflict alters VEMP asymmetry,³⁹ reduces GVS detection during congruent optic flow,⁴⁰ and lowers sensitivity to multisensory verticality percepts.⁴¹ However, the timing of vestibular assessment may have played a role. In the previous studies, vestibular function was tested during or immediately after short-duration exposure to optic flow. Here, it was measured afterwards, once the headset was removed and vestibular signals were again congruent with the stationary body. This introduces a temporal gap during which vestibular recalibration may have begun. Vestibular reweighting is believed to occur quickly, with cybersickness symptoms developing within the first minutes of VR exposure,^{27,28} suggesting that the recalibration process upon returning to the real world may similarly begin promptly. Di Girolamo and Pic (2001) found that reduced VOR gain after 20 min of VR had recovered within 30 min.⁴² Similarly, Perry et al. (2026) reported extremely fast recovery of verticality sensitivity, within the first 90 s following removal of conflicting optic

flow.⁵³ With a 10-min exposure followed by a 15-min detection task, our vestibular measurements were taken nearly 17 to 20 min post-exposure, a window in which any transient reduction in sensitivity may already have already recovered. Future research should therefore investigate the time-course of recovery of vestibular function following visuo-vestibular conflict exposure.

Unlike the vestibular measures, DPDR increased significantly from pre- to post-VR exposure in both conditions, consistent with reports that VR induces dissociative symptoms.^{7,8,10} This increase did not differ between the Roll and Random conditions. If visuo-vestibular conflict were the primary driver of DPDR, we would expect greater symptom increases in the Roll condition. What, then, is driving the effect? One reason is that the HMD itself introduces sensory alterations that contribute to DPDR. Wearing an HMD constrains the field of view, alters visual resolution, and disrupts proprioceptive feedback from the head and face, and the abrupt return from a virtual display to the real world may itself produce a perceptual discontinuity that contributes to DPDR regardless of visual content. This interpretation aligns with evidence that DPDR is elevated specifically after VR compared to conventional screen-based experiences,⁸ implicating the immersive characteristics of the HMD as a contributing factor. The experimental procedure may also have played a role, since electrode placement, blindfolding, and a prolonged detection task could promote a general sense of detachment in both groups. A non-VR control group completing the same procedure without VR exposure would establish whether these DPDR increases are specific to VR or arise from the setup itself.

Presence was significantly higher in the Roll than the Random condition, despite neither stimulus being designed to generate Place Illusion or Plausibility Illusion. The stimuli contained no spatial cues, interactive elements, or recognisable environmental features, and lacked the cues that Slater et al. (2022) identified as driving both PI and Psi.¹² Nevertheless, the coherent roll rotation generated a stronger sense of Presence than random dot motion. One possibility is that the rotating display induced vection, the visual sense of self-motion, which may support Presence by suggesting that the observer is situated within a space and moving through it. This would fit with the idea that self-motion alone can support Presence. We did not measure vection, so this remains a possibility for future work to test directly. Presence was also measured only by self-report, which can be biased by how people read items about the sense of "being there." Future studies should add other measures, such as physiological or behavioural indices, to test it more robustly.

These findings have wider relevance for how VR is used and for whom. As VR moves further into clinical settings such as exposure therapy, pain management, and rehabilitation, users are exposed to it for longer and more often and

the aftereffects of that exposure matter. If dissociative symptoms arise not from sensory conflict alone but from the vestibular system readjusting once the headset comes off, then the period immediately after VR use deserves more attention than it currently gets. It also raises the possibility that people differ in how readily their vestibular system recalibrates, which could help explain why few people are more prone to dissociative aftereffects than the others. Testing vestibular processing will require measuring it at multiple points after exposure rather than once, as a single post-VR assessment cannot capture a process unfolding over minutes.⁴² Complementary physiological measures like VEMPs can also help track recalibration as it occurs. Furthermore, screening for individual differences in vestibular function could eventually identify users most at risk in clinical settings.

While the present study provides initial insights into the role of vestibular functioning in DPDR following VR exposure, several limitations are apparent. The overall model explored here explains only 20% of the variance in DPDR. Thus, there remain many possible avenues for future research to identify predictors of the remaining variability. Such predictors may include personality and demographic factors. For example, previous research suggests that younger, female, inexperienced VR users, and those who experience high levels of embodiment may have higher levels of DPDR¹¹ – although embodiment is an unlikely contributor in the present study given the lack of a VR avatar and the sparse optic flow used here. As such, a more complete evaluation of these factors in future participants can give more insights into how these factors interact with vestibular sensitivity and DPDR. In addition, other factors such as visual sensitivity and vestibular physiological functioning were not measured here, but could also plausibly play a role in symptoms, given their association with non-VR DPDR.⁴⁶ Accordingly, measures such as VEMPs and visual acuity (including stereoacuity which may be relevant for VR exposure given vergence-accommodation conflicts driven by head-mounted displays,⁶²) could provide more extensive insights into physiological factors contributing to VR-induced DPDR. Finally, the present study deliberately used a sparse visual stimulus, with few cues for Presence to deliberately and closely explore the relationship between DPDR and vestibular sensitivity. As such, it is possible that more complex environments, such as those used during real VR exposure, may reveal additional interactions with Presence, vestibular sensitivity, and embodiment which drive DPDR, providing a more complete explanation of relevance to real-world VR use. Accordingly, future research is vital for further exploring the association between these complex factors.

Conclusion

The present study found a complex relationship between DPDR and vestibular processing in VR. While VR exposure increased DPDR irrespective of whether

the visual stimulus contained coherent self-motion signals, in the Roll condition, increases in vestibular sensitivity following VR exposure were associated with greater DPDR, suggesting that the dynamic recalibration of vestibular signals upon returning to the real world may contribute to dissociative experiences. These results indicate that the relationship between vestibular processing and DPDR extends beyond simple downweighting, and that the process of sensory readjustment following VR may be as consequential as the conflict itself. Future research should explore this recalibration process using time-resolved assessments to capture how vestibular readjustment unfolds following VR exposure.

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Ethical considerations

The study methodology was approved by the University of Kent School of Psychology ethics committee (Ethics ID: 2025176157061110138).

Consent to participate

Participants provided written informed consent prior to participation in the study, and were debriefed following participation.

Author contributions

SD: Methodology, Data collection and curation, Analysis, Writing – original draft, project administration. MG: Conceptualisation, Methodology, Research design, Analysis, Writing – review and editing, Supervision, Funding acquisition

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Declaration of conflicting interests

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Data Availability Statement

The raw data is available from the Open Science Framework: <https://osf.io/v8a7d>

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