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ORIGINAL RESEARCH PAPER

The neverending trip: Associations between Hallucinogen Persisting Perception Disorder (HPPD) and non-visual perceptual disturbances

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ABSTRACT

Objective: Hallucinogen Persistent Perception Disorder (HPPD) is a condition where the effects of hallucinogenic drugs reoccur long after the acute effects have stopped. No established risk factors or mechanisms for HPPD have been identified. However, reports have suggested a risk phenotype for HPPD due to associations with other perceptual disturbances. With recent increases in therapeutic psychedelic drug use, it is essential to consider the existence of HPPD risk factors. Therefore, exploring potential links between HPPD and other perceptual disturbances, such as tinnitus and migraine with aura, is a necessary first step. This study aimed to investigate the association between HPPD and other perceptual disorders. *Methods:* One hundred thirty-eight individuals with HPPD and 116 controls participated in a survey assessing the prevalence of various perceptual disturbances: photosensitivity, phonosensitivity, tinnitus, migraine with aura, vertigo, paraesthesia, and synaesthesia. *Results:* The survey results showed a significant association between HPPD and photosensitivity (OR = 10.65), phonosensitivity (OR = 8.00), and the number of perceptual disturbances (OR = 1.59) in the HPPD group compared to the control group. The study also observed trends of dual prevalence between HPPD and tinnitus, migraine with aura, vertigo, paraesthesia, and synaesthesia. Participants with both HPPD and other perceptual disturbances were likelier to experience additional perceptual disturbances after the onset of HPPD. *Conclusions:* These findings suggest a common vulnerability or pathophysiological mechanism among these perceptual disturbances. Given the increasing therapeutic use of hallucinogens, the results of this study provide essential considerations for HPPD risk profiles. Moreover, they may guide future investigations into HPPD's pathophysiology and management options.

KEYWORDS

Hallucinogen Persistent Perception Disorder, HPPD, flashbacks, LSD, psilocybin, synaesthesia, paraesthesia, tinnitus, visual snow syndrome, VSS

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INTRODUCTION

Hallucinogen Persistent Perception Disorder (HPPD) is a condition in which the effects of hallucinogens persist long after drug cessation (Ford et al., 2022), resulting in nonpsychotic hallucinations primarily affecting visual perception. The prevalence of HPPD is challenging to estimate accurately due to the lack of population studies and HPPD presentation

variability (Ford et al., 2022). While the DSM-V only recognises one type of HPPD, there are two widely accepted forms: Type 1, characterised by brief "flashbacks," and Type 2, characterised by chronic symptoms that can last for months or a lifetime (Halpern, Lerner, & Passie, 2018). In all cases, the symptoms involve visual disturbances, which the patient recognises as such, rather than delusional hallucinatory images (Abraham, 1983). HPPD is triggered by a previous history of drug use, with Lysergic Acid Diethylamine (LSD) being the most frequently documented drug associated with HPPD. However, cases linked to other drugs, including those outside the hallucinogenic class (Baggott, Coyle, Erowid, Erowid, & Robertson, 2011), have also been reported. The onset of HPPD is often associated with a triggering event.

There is no widely accepted understanding of the risk factors or mechanisms for Hallucinogen Persisting Perception Disorder (HPPD). However, links have been observed between HPPD and other perceptual comorbidities such as Visual Snow Syndrome (VSS), migraine with aura, and tinnitus. VSS is a neurological condition characterised by a continuous visual disturbance in the form of uncountable dots in the visual field, similar to a poorly-tuned analogue television (Renze, 2017). Despite being clinically like HPPD, VSS occurs in individuals without a history of hallucinogenic drug use (Schankin et al., 2014a, 2014b). Some researchers have hypothesised that HPPD and VSS may share a common pathophysiology (Puledda, Schankin, & Goadsby, 2020) or exist within the same spectrum of conditions with different triggers (Puledda & Goadsby, 2018). Schankin et al. have discussed the strong association between visual snow, tinnitus, and migraine aura and proposed that these could be distinct syndromes with a shared pathophysiological mechanism (Schankin, Maniyar, Sprenger, & et al, 2014). Several VSS patients have reported a connection between migraines and the appearance or worsening of visual snow symptoms (Klein & Schankin, 2021).

Apart from migraine, VSS has also been correlated with tinnitus (Puledda et al., 2020), which is the perception of sound without an external source (Han, Lee, Kim, Lim, & Shin, 2009). Puledda et al. have proposed that visual snow and tinnitus may be two manifestations of a similar disorder, with an increased perception of subthreshold or absent sensory stimuli (Puledda et al., 2020). Tinnitus has been used to predict VSS severity, suggesting that tinnitus and VSS might share a common pathophysiological mechanism (Puledda et al., 2020). Thalamocortical dysrhythmia (Hepschke et al., 2022) and cortical dysexcitability have been hypothesised to explain both pathologies, with links also observed with migraines (Puledda et al., 2020).

Given the known associations between HPPD and VSS, tinnitus, and migraine aura, it is reasonable to hypothesise that HPPD may also be associated with non-visual perceptual comorbidities. In a systematic review of 64 different HPPD symptoms reported by 97 patients across 66 publications, Vis et al. reported concurrent non-visual disturbances such as vertigo (3.1%), hypersensitivity and hyposensitivity (5.2%), and synaesthesia (2.1%) (Vis, Goudriaan, Ter Meulen, & Blom, 2021). The review also revealed a history of migraines in 4.2% of patients with documented medical histories (Vis et al., 2021). Litjens et al. also found associations between HPPD and non-visual symptoms, including tinnitus, oversensitivity to sound, and tingling in the extremities (Litjens, Brunt, Alderliefste, & Westerink, 2014).

The aetiology and pathogenesis of HPPD involve poorly understood mechanisms. One neurobiological hypothesis proposes that LSD use leads to central nervous system dysfunction, characterised by the disinhibition of visual processes (Martinotti et al., 2018). This may occur due to the destruction of GABAergic cortical serotonergic inhibitory interneurons, which can cause a breakdown in the mechanisms that filter unnecessary stimuli (Martinotti et al., 2018). Additionally, a theory of reverse tolerance or sensitisation has been proposed to account for flashbacks even after the stimulus (LSD) has been removed (Stahl, 2021).

The Lateral Geniculate Nucleus (LGN) is also thought to be implicated in HPPD (Abraham & Aldridge, 1993). As an area of the thalamus involved in visual perception pathways, the theory is supported by the discovery of LSD-sensitive neurons in this region (Abraham & Aldridge, 1993). Abraham proposed that flashbacks, common in Type 1 HPPD, could originate from the LGN as a type of visual seizure (Abraham, 1983). Other theories suggest that the subtle overactivation of neural-visual pathways causes symptoms after intoxication in predisposed patients (Halpern et al., 2018). Alternatively, flashback experiences may be triggered by environmental stimuli that resemble the original experience (Holland & Passie, 2001) or anxiety against a background of vulnerability in perception processing (Halpern et al., 2018).

The existing literature on HPPD contains some high-evidence systematic reviews, e.g., (Halpern & Pope, 2003; Martinotti et al., 2018; Vis et al., 2021). However, most studies have small sample sizes, and the definition of the disorder varies widely across the literature, leading some to call for revising the current diagnostic criteria (Halpern et al., 2018). Some authors discuss HPPD but group patients under other conditions, such as "Visual Snow" (Schankin, Maniyar, Digre, & et al, 2014) and "acquired synaesthesia" (Yanakieva, Luke, Jansari, & Terhune, 2019). Investigating a population of HPPD patients can be challenging due to stigma, misdiagnosis (Anderson, Lake, & Walterfang, 2018), and the condition's rarity.

A significant gap in the literature is the absence of studies focusing on the dual incidence of HPPD and other perceptual comorbidities. Such a relationship may raise questions about whether HPPD's pathology involves more than just the visual system. Perceptual disturbances may be a risk factor for HPPD, predisposing patients to the disorder. While the possibility of pre-drug use tinnitus indicating vulnerability to HPPD has been discussed (Halpern et al., 2018), it has not been thoroughly scrutinised. Alternatively, HPPD may cause non-visual perceptual disturbances in patients who did not previously have them. Knowledge of such an association could have major implications, particularly for assessing the suitability of hallucinogens for

therapeutic purposes. A study using hallucinogens in healthy controls found that 9.6% developed ongoing flashback experiences, though none met the clinical criteria for HPPD (Muller et al., 2022). As therapeutic hallucinogens represent a growing area of psychiatric research (De Gregorio et al., 2021), it is crucial to know whether risk factors predispose some people to HPPD. Such factors could impact the risk-benefit ratio of such treatments.

This study aims to determine whether there is an increased prevalence of perceptual disturbances, including tinnitus, migraine with aura, vertigo, paraesthesia, synaesthesia, photosensitivity, phonosensitivity, and visual snow, in a population with HPPD compared to a control group without HPPD. Because the implications of these disturbances preceding HPPD development or occurring after the fact differ, this study will also investigate and discuss their relative time course.

METHODS

This study received approval from the Macquarie University Human Research Ethics Committee HREC EXEC Medical Sciences Committee on 10/06/2021, with ethics approval number 52021945728525. Informed, written consent was acquired via an online consent form attached to the survey. It was an observational case-control study that utilised a digital survey created using Limesurvey and hosted on the Macquarie University website. Participants in the HPPD group were recruited from HPPD online interest groups, such as the Neurosensory Research Foundation and the Perceptual Restoration Foundation, where the study information and link were posted. A comparative control group was recruited from the Macquarie University Psychology cohort and remunerated with course credits for participation.

Questionnaire

The survey obtained information about participants' demographics, HPPD phenomenology, illicit drug use, and their perceptual, psychiatric, pain, and neurological comorbidities. The survey consisted of a combination of multiple-choice and free-text questions. This report focused on perceptual comorbidities. Participants were asked about the name of the comorbidity in question and its short description to avoid misunderstanding medical terminology (see Appendix 1). Participants were also asked about the time course of these comorbidities relative to the onset of their HPPD symptoms. In contrast, controls were only asked about perceptual symptoms and were not given questions about HPPD.

Inclusion/exclusion criteria

To be included in the HPPD cohort of this study, participants had to self-identify as having HPPD, with perceptual changes occurring after consuming a hallucinogenic drug. Of the 233 participants who submitted responses, 82 were excluded because they were incomplete, and five were excluded because they indicated they did not believe they had HPPD. Additionally, participants who had visual epilepsy, tumours in the brain or visual system, or neuroinflammatory disorders, or who had diagnosed schizophrenia/other psychotic disorders were also excluded following DSM-V criteria for HPPD. In total, 138 HPPD responses were available for review and analysis. The control group comprised 143 responses, of which 27 were excluded due to being incomplete, leaving 116 for analysis.

Data analysis

The data were presented descriptively using absolute and relative frequencies and means and standard deviations. A series of multivariate logistic regression models were used to assess the association between comorbidities and the presence of HPPD. To control for potential confounding factors, available demographic variables (age, gender, country of residence, ethnicity, and education) were included in the model. The outcome of each model was the presence or absence of specific comorbidities (tinnitus, migraine with aura, vertigo, paraesthesia, synaesthesia, photosensitivity, and phonosensitivity). Additionally, a linear regression model was used to analyse the number of comorbidities present in each participant. The primary exposure considered was the group (HPPD patients vs. control). As a large number of models were created, the level of significance (α) was adjusted using the Sidak procedure to maintain an overall type I error rate of 5%: $\alpha = 1 - (1 - 0.05)^{(1/k)}$, where k is the number of models created (i.e., 8).

RESULTS

Demographics

After applying the exclusion criteria, 254 responses were included in the analysis, with 138 in the HPPD group and 116 in the control group.

The HPPD group ($n = 138$) had a mean age of 30.3 (SD = 11.55) and consisted of 71% ($n = 98$) males, 28.3% ($n = 39$) females, and 0.7% ($n = 1$) non-binary participants. The majority of the HPPD group identified as white (87%, $n = 120$), with 29.7% ($n = 41$) residing in Australia and 70.3% ($n = 97$) residing elsewhere. Most participants in the HPPD group had completed or commenced tertiary education (55.8%, $n = 77$).

The control group ($n = 116$) had a different demographic profile than the HPPD group. They had a mean age of 20.8 (SD 7.77) and a near-inverse gender distribution, with 81.9% ($n = 95$) females and 18.1% ($n = 21$) males. Most of the control group identified as white (60.3%, $n = 70$) or Asian (26.7%, $n = 31$), and all participants (100%, $n = 116$) resided in Australia. All control participants were undergraduate university students, with some having completed other degrees (7.8%, $n = 9$) or non-university tertiary education (6.9%, $n = 8$), while others reported their highest level of education as high school (33.6%, $n = 39$).

Demographically, the two groups in our study exhibit important differences, particularly in age and gender

Table 1. Demographic summary of the HPPD group and the control group

Demographic		HPPD (N, %)	Control (N, %)
Age (mean, SD)		30.3 (11.55)	20.8 (7.77)
Gender	Female	39 (28.3)	95 (81.9)
	Male	98 (71.0)	21 (18.1)
	Non-binary	1 (0.7)	0 (0)
Ethnicity	White	120 (87.0)	70 (60.3)
	Asian	1 (0.7)	31 (26.7)
	Hispanic	7 (5.1)	3 (2.6)
	Middle-Eastern	3 (2.2)	7 (6.0)
	Indigenous Australian	1 (0.7)	5 (4.3)
	Black	3 (2.2)	0 (0)
	Other/Unspecified	3 (2.2)	0 (0)
Country of residence	Australia	41 (29.7)	116 (100)
	Other	97 (70.3)	0 (0)
Education level[$]	Did not complete high school	12 (8.7)	0 (0)
	High school	24 (17.4)	39 (33.6)
	Non-university tertiary qualification	25 (18.1)	8 (6.9)
	Undergraduate	35 (25.4)	60 (51.7)
	Postgraduate	42 (30.4)	9 (7.8)

This table depicts the demographics of age, gender, ethnicity, country of residence and education level and compares the HPPD group with the controls.
[$]The control group were all Macquarie University School of Psychology undergraduate students.

distribution (see Table 1). To account for these differences and differences in ethnicity, country of residence, and education level, we controlled for them in our statistical model (Table 2).

We investigated the quantity of perceptual comorbidities by tallying and averaging each individual's total "yes" responses. Our results show that the HPPD group had significantly more perceptual comorbidities than the control group, with 1.59 more comorbidities on average ($p < 0.001$).

We also compared the frequencies of each comorbidity between the two groups (see Fig. 1). Overall, there was a trend of a higher prevalence of comorbidities in the HPPD group. Specifically, odds ratios above 1.5 were found for migraine with aura and vertigo, above 2 for synaesthesia, and above 3 for paraesthesia and tinnitus. However, after accounting for demographic differences, these results yielded $p > 0.05$ (see Fig. 3).

Regarding photosensitivity and phonosensitivity, the odds ratios were significant at 10.65 and 8.00, respectively ($p \leq 0.001$). Participants with HPPD were 10.65 times more likely to report photosensitivity than controls (95% CI 2.67 – 48.45), and 8.00 times more likely to report phonosensitivity (95% CI 1.85 – 38.78). These results suggest an overall trend towards increased perceptual comorbidity in HPPD participants, with significantly more comorbidities overall, and a statistically higher occurrence of photo- and phono-sensitivity (Fig. 2).

To investigate the time course of comorbidity symptom onset, participants in the HPPD group who responded "yes" were asked whether the symptoms had commenced "before," "after," or if they were "not sure." Across all seven surveyed domains, we found a trend towards comorbidities occurring after the onset of HPPD. However, it's important to note that Fig. 3 represents small sample sizes, as it is restricted to those who indicated having comorbidity and HPPD, so interpretation should be made with caution.

These findings suggest that HPPD may have a causative impact on these comorbidities or that they may share a common trigger.

DISCUSSION

This study aimed to investigate the prevalence and time course of predominantly non-visual perceptual comorbidities in a group with HPPD and compare it to the prevalence in a control group after adjusting for demographic differences.

Our findings revealed a significantly higher prevalence of perceptual disturbances in the HPPD group compared to the control group (OR 1.59, $p < 0.001$). The HPPD group was also significantly more likely to have photosensitivity (OR 10.66, $p < 0.0001$) and phonosensitivity (OR 1.81, $p = 0.001$) than the control group. However, after correcting for demographic differences, tinnitus, migraine with aura, vertigo,

Table 2. Relative frequencies of perceptual comorbidities

Perceptive comorbidity	Control ($N = 116$)	HPPD ($N = 138$)	Relative frequency HPPD	Relative frequency Control	OR	p-value
Tinnitus	40	103	74.638	34.483	3.27	0.147
Migraine with Aura	14	46	33.333	12.069	1.50	0.994
Vertigo	34	56	40.580	29.310	1.52	0.981
Paraesthesia	36	61	44.203	31.034	3.19	0.142
Synaesthesia	4	15	10.870	3.448	2.45	0.964
Photosensitivity	20	94	68.116	17.241	10.65	<0.001
Phonosensitivity	15	53	38.406	12.931	8.00	0.001
Number of Comorbidities[$]	34	18	13.043	29.310	1.59	<0.001

[$]Effect of group on number of comorbidities is represented in natural scale.
This table depicts the perceptive comorbidities' relative frequencies and odds ratios, comparing the HPPD group with the control group.

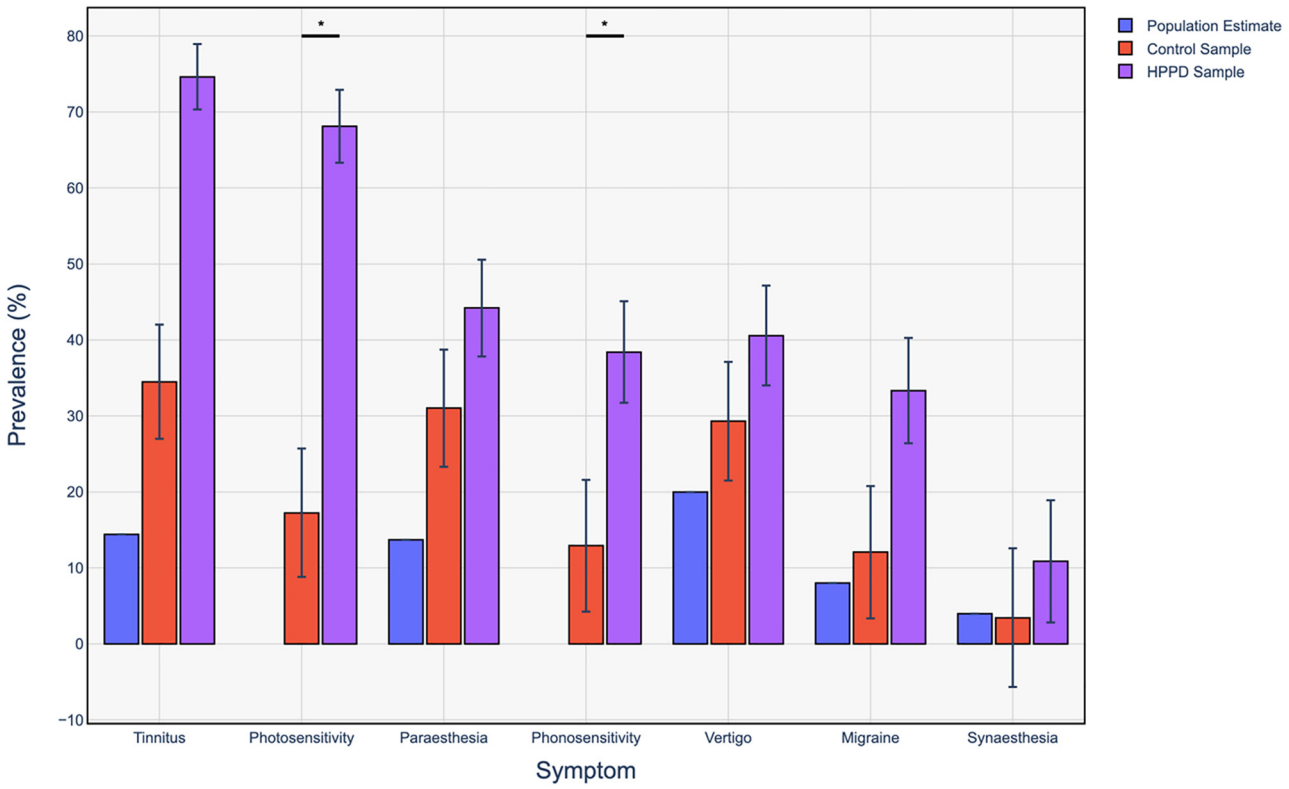

Fig. 1. Comparison of relative frequencies of perceptual comorbidities in HPPD and control groups. The purple bars represent the HPPD group, while the red bars represent the control group. The frequencies are adjusted for demographic differences between the two groups. Blue bars represent population estimates extracted from the literature where available. The details of the population estimated are available in Appendix 2. $^{*}p < 0.05$ after correction

paraesthesia, and synaesthesia did not have significant associations with HPPD. Nonetheless, Fig. 1 shows a clear trend of higher proportions of these comorbidities in the HPPD group. It is worth noting that photosensitivity, as defined in Appendix 1, is a visual phenomenon, and its occurrence in HPPD has been documented before (Ford et al., 2022; Puledda et al., 2020).

Although HPPD is typically regarded as a disorder predominantly of the visual system (American Psychiatric Association. and American Psychiatric Association. DSM-5 Task Force., 2013), the significant relationship between phonosensitivity and HPPD, as well as the overall significantly higher quantity of perceptual disorders in this group, suggests an association between having HPPD and non-visual symptomology.

It is important to note that this study design does not allow investigating a causative relationship. However, we observed an overall trend towards the onset of all perceptual comorbidities after a participant's HPPD, as seen in Fig. 3.

Theories explaining results

Several possible explanations could account for the relationships observed in this study. One possibility is that HPPD is not limited to the visual system and instead affects multiple sensory modalities. HPPD may cause auditory (tinnitus, phonosensitivity), tactile (paraesthesia), and vestibular (vertigo) disturbances, as well as synaesthesia and migraine aura. This may be explained by the thalamocortical dysrhythmia and cortical dysexcitability (Puledda et al., 2020) theory, which posits that HPPD lowers the threshold for stimuli across multiple senses and leads to heightened perceptions of subthreshold or absent stimuli, such as in hallucinations, tinnitus, para- and photosensitivity, and phonosensitivity. Additionally, there may be dysregulation in response to such stimuli, as seen in synaesthesia or migraine aura (Puledda et al., 2020).

Two theories suggest a mechanistic link between migraines with aura and HPPD. Firstly, psilocybin, a drug associated with HPPD, can cause migraine-like headaches in a dose-dependent manner (Johnson, Sewell, & Griffiths, 2012). Secondly, some hallucinogens, such as tryptamines/LSD, are structurally similar to migraine medications, such as triptans/ergotamines (Litjens et al., 2014; Wilkinson, 2004).

Another explanation is that perceptual disturbances act as risk factors for developing HPPD. These disorders and HPPD may share a pathophysiological vulnerability related to a reduced ability to filter unnecessary stimuli. Disorders such as tinnitus may predict a higher vulnerability to HPPD, with drug use as a trigger (Halpern et al., 2018). This study supports the idea that HPPD is more than an isolated visual experience. Although it is difficult to pinpoint the exact cortical centre for this vulnerability, this

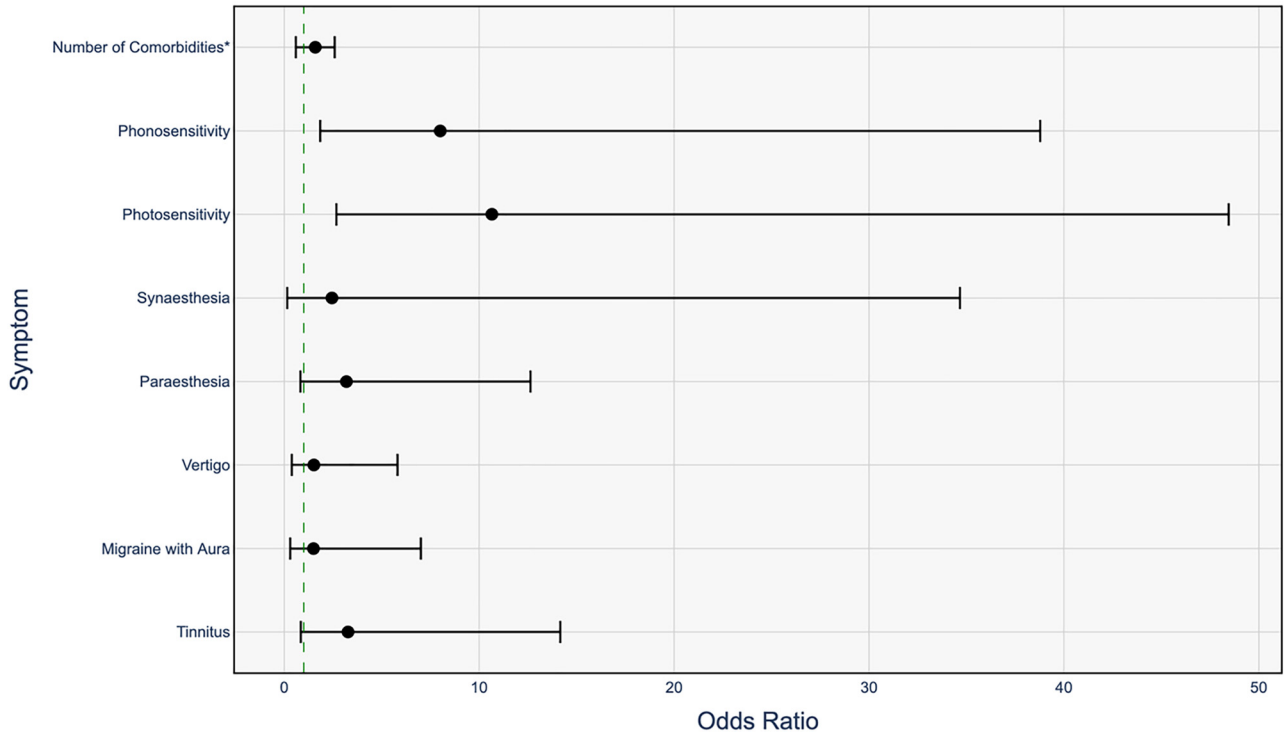

Fig. 2. Comparison of odds ratios of perceptual comorbidities in HPPD and control groups, adjusted for demographic differences. The figure also includes the odds ratios of the number of comorbidities.
[$]Effect of group on number of comorbidities is represented in natural scale

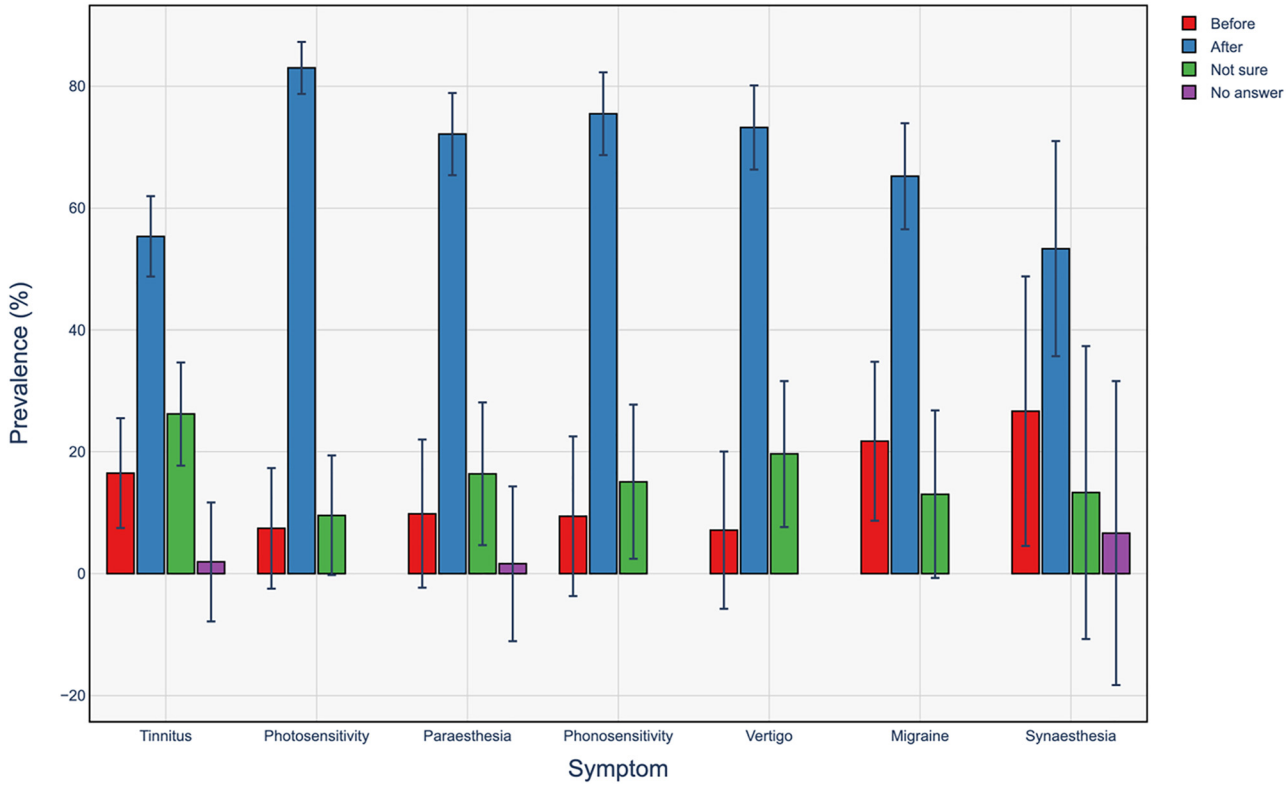

Fig. 3. Time course of comorbidities relative to the onset of HPPD. Participants who responded "yes" within the HPPD group were asked about the timing of their comorbidity symptom onset in relation to their HPPD: "before", "after", or "not sure". The figure shows a trend towards comorbidities occurring after the onset of HPPD across all seven surveyed domains. However, it should be noted that Fig. 3 represents a small sample size, limited to those who reported having both HPPD and the comorbidity, so caution is advised when interpreting the results. These findings suggest that HPPD may have a causative impact on these comorbidities or may share a common trigger

study suggests that it is higher than the level of the visual system. This vulnerability may arise from a genetic predisposition, an anatomical difference in the brain, or exposure to a causative pathogen or event. Future studies could use neuroimaging techniques such as fMRI or magnetoencephalography and genetic studies to investigate this vulnerability further.

Limitations

The study design has certain limitations that affect the generalisability of the results. Since it was a survey, it was not possible to confirm whether participants who reported having a disorder, including HPPD or any of the assessed comorbidities, met the clinical criteria for that disorder. Although efforts were made to align questions for the HPPD group with the DSM-V criteria and exclude certain patients, such as those with visual epilepsy or schizophrenia, the results for other comorbidities were based solely on subjective self-reporting of an experienced symptom or disorder. The absence of individual assessments for each participant makes it challenging to confirm the clinical accuracy of these subjective experiences. The definitive design would involve assessing participants in a clinic environment to confirm diagnoses, although this may be difficult to do on the same scale. We have also not used a design that facilitates the assigning of any causation in our conclusions and it would be ideal to consider this in any future follow up studies.

Another major limitation of this study design is the control group, which comprised Macquarie University psychology students. As shown in Table 1, they differed demographically from the HPPD group, and these differences could have implications, such as the gender disparity affecting the likelihood of perceptual disorders like migraine (Eisenstein, 2020). The younger age of controls could also affect perceptual disturbances like tinnitus (Al-Swiahb & Park, 2016). Although demographic differences in age, gender, ethnicity, country of residence, and education were accounted for statistically, there may have been other hidden confounding differences that were not adjusted for.

Appendix 2 (Table A1) examines the population estimates for some perceptual comorbidities and compares them to the HPPD and control groups. Significant differences exist between the psychology student group and population estimates, particularly in tinnitus, vertigo, and paraesthesia. A larger sample size may balance this difference, but it could also be coincidental. Some literature suggests that psychology students have higher rates of some mental illnesses than students in other fields, and the same could be true for subjective perceptual experiences (Mojs et al., 2015).

A future study could address some of these limitations using a better-matched control group, particularly regarding age and gender. An ideal control group may consist of individuals with similar drug use but without HPPD symptoms, although it may be challenging to recruit such a group.

CONCLUSION

The data presented in this study supports the theory that HPPD is not only a visual disorder but also involves significant photosensitivity, phonosensitivity, and a range of perceptual disorders compared to controls. Furthermore, the study identified trends between HPPD and other conditions such as tinnitus, migraine with aura, vertigo, paraesthesia, and synaesthesia. Participants with both HPPD and perceptual disturbances were more likely to experience these after the onset of their HPPD, indicating a potential common vulnerability or pathophysiological mechanism between these disorders. Therefore, the idea of HPPD as solely a visual disorder may need revision.

This finding has increasing relevance, particularly in light of the recent upswing in the use of hallucinogens in clinical trials for mental illness treatment. Future studies in this field may employ neuroimaging or genetic investigations to further elucidate these disorders' underlying mechanisms.

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Appendix 1: Survey questions

Do you experience any of the following:

Tinnitus: Tinnitus is when you experience ringing, buzzing, or other noises in your ears. This isn't caused by an external sound, and other people usually can't hear it. It can also sound like roaring, clicking, hissing, or humming, and may be present all the time, or may come and go.

Migraine with aura: This is when you have recurring headaches that come at the same time, or just after a sensory disturbance. This disturbance might be in the form of tingling in your hands or face, or visual changes such as flashes of life, floaters in vision, or blind spots.

Vertigo: This is a feeling of being off-balance. It may be as though you are moving or spinning or the world is moving or spinning around you. This feeling may be present all the time, or may come and go.

Numbness or tingling: This may occur in your arms, legs, or somewhere else on your body. It can feel numb, like 'pins and needles', or as though your skin is crawling or itching with no reason. This feeling may be present all the time, or may come and go.

Synaesthesia: Synaesthesia is a condition where information that would normally stimulate one sense instead stimulates another or several senses. For example, you might hear sounds, but also see them in the form of colourful swirls. Alternatively, you may 'hear' things that most people would only see visually, or 'taste' something most people would only hear or see. This can be with any of the senses.

Sensitivity to light: High sensitivity to light, or 'photophobia', is when the sun or bright lights can cause you to feel uncomfortable, or may even cause pain. This can be linked with headaches, or may occur on its own. This feeling may be present all the time, or may come and go.

Sensitivity to sound: High sensitivity to sound, or 'phonophobia', is when loud or sudden sounds make you feel very uncomfortable, or may even cause pain. This can be linked with headaches, or may occur on its own, and is not usually associated with hearing problems or hearing loss. This feeling may be present all the time, or may come and go.

Did your [symptom] start before or after you first experienced HPPD?

These questions are part of a larger survey conducted and analysed at Macquarie University.

Appendix 2: Comparison of data with population prevalence

Table A1. Percentage of HPPD and control groups surveyed with perceptual comorbidities, compared to estimated population prevalence

	HPPD	Control	Population estimates
Tinnitus	74.6	34.5	14.4 (Jarach et al., 2022)
Migraine with aura	33.3	12.1	8 (Russell, Rasmussen, Thorvaldsen, & Olesen, 1995; Viana, Tronvik, Do, Zecca, & Hougaard, 2019)
Vertigo	40.6	29.3	15–20 (Neuhauser, 2016)
Paraesthesia	44.2	31.0	13.7 (Inoue et al., 2013)
Synaesthesia	10.9	3.4	2–4 (Simner et al., 2006)
Photosensitivity	68.1	17.2	
Phonosensitivity	38.4	12.9	

This table compares the HPPD and control group data from this survey with population estimates. Photosensitivity and phonosensitivity population prevalence were not included due to their subjective nature.