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Complete List of Authors:	Trinidade, Aaron; Southend University Hospital; Anglia Ruskin University Medical School Cabreira, Verónica; Centro Hospitalar Universitário de São João; University of Edinburgh, Center for Clinical Brain Sciences/University of Edinburgh Goebel, Joel; Washington University in St Louis School of Medicine Staab, Jeffrey; Mayo Clinic, Department of Psychiatry and Psychology Kaski, Diego; University College London, Department of Neuro-otology Stone, Jon; University of Edinburgh, Centre for Clinical Brain Sciences
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Predictors of persistent postural-perceptual dizziness (PPPD) and similar forms of chronic dizziness precipitated by peripheral vestibular disorders: a systematic review.

Aaron Trinidade, FRCS (ORL-HNS)[1,2]

Verónica Cabreira, MD[3,4]

Joel A Goebel, MD FACS FRCS[5]

Jeffrey P Staab, MD, MS[6]

Diego Kaski, PhD MRCP[7]

Jon Stone, PhD MRCP[4]

[1]Southend University Hospital, Westcliff-on-Sea, UK.

[2]Anglia Ruskin University Medical School, Chelmsford, UK.

[3]Centro Hospitalar Universitário de São João, Porto, Portugal.

[4]Centre for Clinical Brain Sciences, University of Edinburgh, Edinburgh, UK.

[5]Washington University in St. Louis School of Medicine, St. Louis, Missouri, USA.

[6]Mayo Clinic, Rochester, Minnesota, USA.

[7]University College London Hospital, London, UK.

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Correspondence to:

Aaron Trinidade, FRCS (ORL-HNS)

Department of Otolaryngology, Southend University Hospital NHS Foundation Trust, Prittlewell Chase, Westcliff-on-Sea, UK. Email: aaron.trinidade@nhs.net. Tel: +44 01702 385145

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Abstract

BACKGROUND: The literature on predictors of persistent postural-perceptual dizziness (PPPD) following peripheral vestibular insults has not been systematically reviewed.

METHODS: We systematically reviewed studies on predictors of PPPD and its four predecessors (phobic postural vertigo, space-motion discomfort, chronic subjective dizziness, and visual vertigo). Investigations focused on new onset chronic dizziness following peripheral vestibular insults, with a minimum follow-up of 3 months. Precipitating events, promoting factors, initial symptoms, physical and psychological comorbidities, and results of vestibular testing and neuroimaging were extracted following the PRISMA guidelines.

RESULTS: We identified 13 studies examining predictors of PPPD or PPPD-like chronic dizziness. Pre-existing anxiety, early maladaptive symptoms following vestibular injury, dependent personality types, increased body vigilance following precipitating events, and visual dependence, but not the severity of initial or subsequent structural vestibular deficits or compensation status, were the most important predictors of chronic dizziness. Disease-related abnormalities of the otolithic organs and semi-circular canals and age-related brain changes seem to be important only in a minority of patients.

CONCLUSIONS: Early symptoms, predisposing physiological and psychological factors and behavioural responses are the most likely predictors of PPPD following peripheral vestibular illnesses. Future studies are needed to validate these results, identify the most sensitive and specific early indicators of PPPD, and factors amenable to preventative and rehabilitation interventions.

Key words: persistent postural-perceptual dizziness; functional dizziness; phobic postural vertigo; space-motion discomfort; visual vertigo; chronic subjective dizziness; chronic dizziness; predictors; prognosis.

Introduction

Persistent postural-perceptual dizziness (PPPD) is a chronic functional vestibular disorder characterised by dizziness, unsteadiness, or non-spinning vertigo that are present on most days for three months or more. These are often exacerbated by upright posture, active or passive movement, and moving or complex visual stimulus exposure. PPPD may be precipitated by conditions that disrupt balance or cause vertigo, unsteadiness, or dizziness, including peripheral or central vestibular disorders, other medical illnesses, and psychological distress. It presents alone or co-exists with other conditions (1).

PPPD's diagnostic criteria were introduced into the beta draft of the WHO's International Classification of Diseases (11th edition) in 2015 and adopted with the final version in 2022 (2) following a consensus document from the Behavioural Subcommittee of the Bárány Society's Classification Committee for Vestibular Disorders (1). Although the term PPPD is relatively new, the potential of acute vertigo to develop into distinct chronic dizziness without clear structural correlates has been recognised for centuries. PPPD brings together varying aetiological and mechanistic models, and evolves from previously described visual vertigo, space-motion discomfort, phobic postural vertigo and chronic subjective dizziness, the latter which removed the need for specific psychological factors to be present. Its pathophysiology is thought to include locomotion and spatial orientation alterations (e.g., abnormal postural control stiffening (3) and visual dependence (4)), appropriate adaptations during mobility threats (e.g., acute vestibular illnesses), but maladaptive when maintained after acute illness remission. Current pathophysiologic models (5) hypothesize that these brain functioning shifts are linked to predisposing factors that may be identifiable early in the illness, perhaps before PPPD's onset, making them possible PPPD predictors.

PPPD is a significant public health concern, being the principal diagnosis for 20% of all dizzy patients in general neurology clinics (6), typically causing moderate-to-severe handicap and poor quality of life (7). We review the literature on predictors of PPPD or PPPD-like chronic dizziness following acute vestibular illnesses to 1) identify early risk factors for developing PPPD following peripheral vestibular insults that might be clinically useful; and 2) improve knowledge about the pathophysiology involved in PPPD's development.

Methods

We searched PubMed, Embase and PsycINFO databases. The search strategy included: (vertigo OR dizziness OR "vestibular neuritis" OR "benign paroxysmal positional vertigo" OR "Meniere* disease" OR "vestibular migraine*" OR "acoustic neuroma" OR "vestibular schwannoma") AND ("persistent postural perceptual dizziness" OR “PPPD" OR "visual vertigo" OR "space motion discomfort" OR "space motion phobia" OR "chronic dizziness" OR "functional dizziness" OR "peripheral dizziness" OR "somatoform dizziness" OR "psychogenic dizziness" OR "chronic vertigo") AND (“predict*” OR “likelihood” OR "risk factor*" OR “prognosis” OR “pathophysiologic” OR "follow-up*" OR “followup*” OR “persist*” OR "on-going" OR “ongoing”). English-language studies from database inception through 25 January 2022 were included. We also searched bibliographies of included studies, and added relevant articles proposed by JAG, JPS, DK and JS. The review followed PRISMA guidelines (Figure 1).

We extracted information on study design, sample size, age and gender of participants, follow-up duration and proportion followed up, previous vestibular events, chronic dizziness nomenclature, baseline assessments, physical and psychometric measurements, and findings about significant chronic dizziness predictors following vestibular events. Retrospective or prospective studies with peripheral vestibulopathy as the symptom trigger and a minimum 3-month follow-up were included. Studies investigating treatments were excluded. Table 1 lists inclusion and exclusion criteria.

Results

Included study characteristics

Thirteen studies were included, comprising 780 participants (Figure 1, Table 2) (8–20). Female proportion ranged from 36-76.7% (average: 53.6%); mean age was 50.2 years. Faralli, *et al.* (16) focused on young patients (mean age: 26 years). Imate, *et al.* (20) did not specify a mean age, but 10 patients (23.3%) were <30 years, of which three were ≤10 years.

Six studies were prospective, utilizing within-subjects designs or subgroup comparisons (9,11,13–15,17); four were retrospective, utilizing within-subjects designs or subgroup comparisons (8,10,12,16);

two were previously-evaluated patient reassessments (19,20); one was a prospective case-control investigation (18).

Regarding precipitating events (Table 2), seven studies included patients with vestibular neuritis only (9,11,13,17,19,20). The largest was by Godemann, *et al.* (17) who enrolled 103 patients within 24 hours of vestibular neuritis onset and retained 75 over one year to investigate persisting vertigo risk factors. Sixty-seven were from a previous case-control study (18) comparing patients with persistent vertigo 6 months after vestibular neuritis to 89 randomly selected healthy controls. Two studies included patients with benign paroxysmal positional vertigo (BPPV) only: one focused on persistent symptom correlation with vestibular-evoked myogenic potentials (VEMPs) (10), the other with findings from brain structural MRI (12). Three studies included patients with a mixture of diagnoses (8,14,15). Kabaya, *et al.* (8) studied 155 patients with peripheral vestibular disorders (vestibular neuritis, BPPV, Ménière's disease, and unspecified peripheral vestibular dysfunction (n=89)) and non-peripheral conditions (vestibular migraine, psychiatric dizziness, other/unknown illness (n=66)). This was the only study that included patients with illnesses other than peripheral vestibular disorders. It was retained because most patients had a peripheral disorder, it was the largest study, and it was the only one that specifically investigated PPPD. Tschan, *et al*. (14) studied 52 patients with BPPV, vestibular neuritis, Ménière's disease or vestibular migraine. Heinrichs, *et al.* (15) studied 43 patients with vestibular neuritis or BPPV. Faralli, *et al*. (16) did not specify a diagnosis in their 20 patients but reported that they had experienced sudden, spontaneous vertigo lasting more than 24 hours accompanied by nausea and vomiting, suggesting acute unilateral vestibulopathy.

Selected studies included a variety of physiological, psychological, and radiological tests. Most used a combination of physiological testing and questionnaires (8,9,11,15,17,18), except for one using physiological tests only (14).

Chronic dizziness frequency

Chronic dizziness rates following precipitating vestibular insults were reported in 11 studies (Figure 2). They were generally higher for studies focusing on vestibular neuritis only or non-specified acute unilateral vestibulopathy (Faralli, *et al.*) (16), and lowest following BPPV. The highest rate was 53% in

the relatively small vestibular neuritis study by Berginius, *et al.* (n=19) (19). The lowest rates were 7.5% in a larger study of 102 BPPV patients by Cha, *et al.* (12) and 10.3% in the study of 155 patients with various conditions, mainly BPPV and Ménière's disease, by Kabaya, *et al.* (8).

Risk factors

Demographics

Only one study found gender as a chronic dizziness predictor (18) despite a female preponderance in seven (8,10,12,13,15,16,18). Older age was associated with chronic dizziness in three studies. Two examined age-related brain changes after BPPV (12) and vestibular neuritis (13) using MRI. The third analysed peripheral vestibular function after vestibular neuritis with various measures (19) (Figures 2 & 3).

Acute vestibular symptoms

Heinrichs, *et al.* (2007) (15) created a composite measure of vestibular symptom severity by summing intensity, frequency, and duration of symptoms on a patient-reported visual analogue scale. Acute dizziness severity after vestibular neuritis or BPPV was *not* predictive of chronic dizziness, but the interaction between acute dizziness severity and acute anxiety about bodily symptoms (discussed later) was associated with chronic dizziness severity after three months. Kabaya, *et al.* (8) found that 18 months after acute vestibular symptom onset, Dizziness Handicap Inventory (DHI) total and subscale scores measured at 33 days (mean) did not differ between patients with (n=8) or without (n=147) PPPD. In contrast, early Niigata PPPD Questionnaire (NPQ) total scores and scores on upright/walking and self-motion, but not visual stimulation, subscales were significantly higher among those later developing PPPD versus those who did not. Early positive NPQ scores (total >27) had high sensitivity (0.88), but low specificity (0.52) for identifying patients who develop PPPD. Thus, standard measures of vestibular symptom severity do not generally predict chronicity, but a measure focused on the types of dizziness exacerbators experienced in PPPD, perhaps unsurprisingly, do.

Anxiety and body vigilance at onset

Godemann, *et al.* (18) reported that 85% of 67 patients who developed chronic dizziness experienced acute anxiety following vestibular neuritis. In a later study of the same cohort , using the Agoraphobic

Cognitions Questionnaire (ACQ) and Bodily Symptoms Questionnaire (BSQ), they found that patients who experienced acute vertigo as an alarming event over which they had no control or focused intensively on their vestibular symptoms as harbingers of negative outcomes were more likely to experience ongoing dizziness than those who did not develop catastrophic thinking or hypervigilance about bodily sensations (17). In a 3-month prospective investigation following patients after acute vestibular neuritis or BPPV, Heinrichs, *et al.* (15) found that interaction between acute vestibular symptom severity and heightened body vigilance measured by BSQ predicted chronic dizziness in patients with vestibular neuritis but not BPPV. Cousins, *et al.* (11) also identified the importance of acute psychological symptoms in 40 patients with acute vestibular neuritis. Dizziness handicap severity (DHI scores) at 10 weeks was linked to body vigilance (BSQ scores) and anxiety-related autonomic arousal (Vertigo Symptom Scale – autonomic/anxiety subscale), all measured within 1-5 days of illness onset.

Visual dependence

Visual dependence is normally distributed but increases in some patients following acute peripheral or central vestibular insults (21). It is also thought to underlie PPPD's visually-induced dizziness component (1). Cousins, *et al*. (11) used the Rod and Disk Test to show that visual dependence – the overweighting of visual versus vestibular and somatosensory cues in determining vertical orientation perception with respect to gravity – was associated with chronic dizziness. Acute visual dependence provides a mechanistic link with acute body vigilance and anxiety-related autonomic arousal in promoting chronic dizziness.

Pre-existing psychological factors

Two studies (14,15) found that patients with premorbid psychiatric disorders or dependent personality traits were more at risk of developing chronic dizziness . Conversely, two other studies (11,18) did not. Tschan, *et al.* (14) found that patients with higher resilience, sense of coherence, and general satisfaction with life were *less* likely to develop secondary functional dizziness one year after a vestibular disorder. .

Vestibular clinical signs and testing

Imate, *et al.* (20) found an increased frequency of positional nystagmus among 43 patients with chronic dizziness following acute vestibular neuritis. They hypothesized that this was caused by increased residual resting activity in the ipsilateral vestibular nucleus, which could persist for >10 years. Berginius, *et al.* (19) found that a small caloric asymmetry was associated with increased residual unsteadiness or vertigo risk following acute vestibular neuritis in 19 individuals. Even though caloric testing measured only horizontal semi-circular canal dysfunction, they used this result to postulate vestibular neuritis-induced damage to the vertical canals or otoliths. More recently, Lee, *et al.* (9) found that lower vestibulo-ocular reflex (VOR) gain, higher occurrence and peak velocity of overt corrective saccades, and lower covert corrective saccade occurrence on video head impulse testing (vHIT) were associated with poorer symptomatic recovery post-vestibular neuritis. These variables, together with suppression HIT results, predicted residual symptoms. Faralli, *et al.* (16) reported that VEMPs were absent in all 20 patients with persistent dizziness following unilateral acute vestibulopathy and suggested the potential role of inferior vestibular nerve dysfunction in long-term symptoms and postural impairment. Oh, *et al.* (10) found an association between an increased cervical VEMP-modified inter-aural difference ratio favouring the affected side among 65 BPPV patients with residual dizziness.

In contrast, Godemann, *et al.* (18), Heinrichs, *et al.* (15), and Cousins, *et al*. (11) observed *no* association between vestibular tests and chronic dizziness. Using factor analyses, Cousins, *et al*. (11) found that caloric asymmetry loaded with rotary chair motion detection threshold on a factor explained only 12% of outcome variance, whereas a separate factor that contained visual dependence, body vigilance, anxiety-related autonomic arousal, and DHI scores explained 59% of outcome variance. Furthermore, Adamec, *et al.* (13) found that VEMPs were non-predictive of outcomes in patients with vestibular neuritis and Kabaya, *et al*. (8) found no significant differences in pure-tone audiometry, positional nystagmus testing, electronystagmography, caloric testing, cervical VEMP, or vHIT between patients who developed PPPD or not.

Neuroimaging

Four studies used brain MRI (8,12,13,15). Cha, *et al.* (12) reviewed brain MRIs of 120 BPPV patients and concluded that grey matter atrophy and white matter hyperintensities, a feature of cerebral small

vessel disease (cSVD), were independently associated with long-lasting dizziness. Adamec, *et al.* (13) reached similar conclusions in their study of patients post-vestibular neuritis.

Discussion

This review investigated chronic dizziness predictors in patients experiencing acute vestibular events. To our knowledge this is the first study summarizing risk factors for the development of PPPD and its predecessors, taking into consideration demographic variables, vestibular tests, imaging and psychological factors. It shows that overall long-term prognosis appears to be much less dependent on the magnitude of peripheral vestibular damage compared to a combination of psychophysical factors, personality traits, anxiety, altered vestibular perception and increased visual dependence.

Structural vestibular deficits are poor PPPD predictors

Although the otolithic organ has been proposed as the seat of PPPD (22), evidence to support a predictive role for peripheral vestibular injuries to the severity or persistence of chronic vestibular symptoms is inconsistent (11,23–27). Strikingly, in this review only a minority of patients with PPPD-like chronic dizziness in prospective studies (e.g., 10% in Godeman's work) have evidence of a chronically uncompensated peripheral deficit (17,18). While a small cross-sectional study (19) found small differences on caloric irrigation in patients with chronic dizziness, other studies failed to replicate this. Similarly, two retrospective (10,16) and one prospective (9) study associated patterns of absent VEMPs and vHIT changes with chronic dizziness, but the trend was inconsistent. Besides their small sample sizes, these studies did not measure relevant psychological or physiological variables (e.g., visual dependence), factors that outperformed structural variables as chronic dizziness predictors in larger investigations. Furthermore, it would be unexpected for any factor to be present or absent 100% of the time as was reported for absent VEMPs by Faralli, *et al*. (16). Such absolute results would more likely arise from technical factors, selection bias, or other confounders. Further supporting our results, Kim, *et al.* (28) assessed otolith dysfunction in 51 patients six weeks post-vestibular neuritis and found that otolith-related tests such as VEMPs recovered rapidly in almost every patient and were not residual symptom predictors. While canal-related tests such as HIT initially imply vestibular nerve injury severity (28), their usefulness seems to wane, as shown by Patel, *et al.* (29) who tested patients cross-sectionally three months post-vestibular neuritis and found that chronic dizziness was not associated

with HIT gain abnormalities of single or combined ipsilesional semi-circular canals. Recently, Herdman, *et al.* (30) evaluated 185 patients attending a neuro-otology clinic for persistent dizziness and vertigo following unilateral peripheral vestibulopathy, BPPV or vestibular migraine. They found that vestibular deficits were not associated with symptom severity, except for the presence of an abnormal caloric test result. In contrast, general and dizziness-specific physiological factors were associated with dizziness handicap and severity more strongly than objective vestibular deficits or the diagnosis itself. Therefore, *ongoing* peripheral vestibular damage is contributory to PPPD in only a small fraction of patients and is not likely the main determinant of symptom burden or handicap (11,15,16,29–36) (Figure 4). This discrepancy between objective findings and symptom burden is consistent with other areas of neurological practice. For example, there is little relationship between degenerative lumbar disease and pain, either cross-sectionally or as a predictive factor (37).

Central brain structural pathology does not appear to be a strong PPPD predictor

In two studies, chronic dizziness was associated with brain MRI abnormalities, particularly mediated by older age, indicating that age-related brain atrophy and white matter changes may be associated with subsequent impaired recovery (12,13). These abnormalities were not specifically located in vestibular cortical regions, suggesting that they may have disrupted one or more wide-ranging networks that support locomotion and spatial orientation. Most large-scale cerebral small vessel disease (cSVD) studies have focused on gait disturbance rather than dizziness. However, Ahmad, *et al.* (38) identified that >80% of patients with high cSVD grades suffered from otherwise unexplained dizziness, implying that cSVD may be an independent factor in imbalance and dizziness in the elderly, especially among those with subtle neuro-otologic or neuro-ophthalmologic signs (e.g., smooth pursuit abnormalities or saccades on videonystagmography) (39–41). Thus, a reasonable caveat to the general finding that long-term outcomes were not predicted by structural abnormality severity is that incomplete compensation for peripheral structural deficits or the existence of sufficient central lesions may have contributed, in part, to chronic dizziness in some, especially older, patients (Figure 4).

Demographic and psychological variables are better PPPD predictors

Most studies reported a female preponderance among patients developing chronic symptoms (8,10,12,13,15,16,18) , but an association was found in only one study (18). This is consistent with

recent large cross-sectional investigations reporting a 2:1 ratio of women:men and an average age in the late 50s among PPPD patients in tertiary neurology practices (6). Older age was also associated with chronic dizziness in two studies of patients following acute vestibular neuritis or BPPV, linked to age-related changes in grey and white brain matter (12,16). Neither gender nor age (PPPD has been reported from middle childhood to late adulthood (6)) are likely to be useful isolated outcome predictors but may be in combination with other factors.

Specific early dizziness symptoms as chronic dizziness predictors are promising. The largest study reviewed (8), and only one assessing PPPD specifically, found that high susceptibility to three provoking factors for PPPD (upright posture/walking, self-motion, visual stimulation) measured by NPQ had high sensitivity (0.88) for identifying patients later developing PPPD. However, this retrospective study only included 8 PPPD patients and had low specificity (0.52). Nevertheless, the NPQ was more promising than acute severity of dizziness alone (15) or dizziness handicap (8), neither of which predicted chronic dizziness. Furthermore, the provoking factors captured by NPQ subscales had high sensitivities and specificities for distinguishing PPPD from BPPV, Ménière's disease, and vestibular migraine, reinforcing its clinical value (24).

In 1958, Moore defined psychogenic dizziness as "anxiety preceding and causing the symptom of vertigo instead of being its result." (16). Our data indicates that it is not just pre-existing anxiety and anxiety-related personality traits, in particular neuroticism, that are relevant but also the hypervigilant state of increased introspective self-monitoring that arises *following* acute vestibular disease and the fear of further vertigo attacks. Two specific anxiety-related variables received significant support as potential PPPD predictors: high body vigilance (i.e., conscious attention to dizziness) and catastrophic worries about the causes and consequences of vestibular symptoms in response to acute vestibular events (11,15,17,18) (Figure 4). These symptoms can be quantified with validated measures of body sensation and anxiety cognition. Heinrichs, *et al*. (15) found that acute vestibular symptoms interacted strongly with acute body vigilance to predict persistent dizziness in vestibular neuritis patients suggesting an added value of incorporating these psychological factors into a short questionnaire covering acute vestibular symptoms and factors provoking PPPD. This is further supported by several studies finding higher psychological comorbidity frequency, including anxiety and panic attacks, in

patients with ongoing dizziness following vestibular vertigo (18,31,42), although this association cannot be proven retrospectively.

Data on pre-existing psychiatric disorders as outcome predictors were mixed (14,15) and like gender and age, need to be considered in light of high base rates of these illnesses in the general population (up to 30% lifetime prevalence for anxiety disorders, 10-20% lifetime prevalence for mood disorders). A study by Dyukova, *et al.* (43), not included in the review because follow-up was only at one month, looked at patients with ongoing dizziness complaints versus those without persistent dizziness after successful BPPV treatment and found that patients with ongoing dizziness were more likely to have a history of premorbid panic attacks (80% vs 29.3%), implying that an anxiety-related predisposition might influence the long-term course of symptom burden. Regarding pre-existing personality traits, cross-sectional investigations found a higher frequency of neuroticism between PPPD patients and patients with other vestibular disorders or the general public (18). In contrast, positive traits may be protective, including resilience, sense of coherence and life satisfaction (14). Best, *et al*. (31) also evaluated patients with acute vestibular syndromes over one year and concluded that patients with premorbid psychiatric disorders were more likely to suffer from emotional distress after an acute vestibular event, especially perhaps those with vestibular migraine (Figure 4). Finally, Staab, *et al.* (44) noted that primary anxiety disorders can cause dizziness and be exacerbated by physical neuro-otologic conditions just as the latter may cause psychopathology (Figure 4).

Visual dependence is an independent physiological predictor of PPPD

Cousins, *et al*. (11) found that visual dependence was an independent predictor of dizziness handicap and strongly interacted with psychological variables of body vigilance and anxiety-related autonomic arousal to generate chronic dizziness. They measured visual dependence with a portable laptop computer system making it an interesting early physiologic measure which could also be a relevant PPPD treatment target, given further validation is achieved (Figure 4).

Relevance to treatment

Understanding PPPD predictors could help tailor effective treatments. Published studies of all three existing treatment modalities for PPPD and its predecessors, namely vestibular rehabilitation (45),

medication (46), and psychotherapy (47), hint that early therapeutic intervention outcomes are more effective. One study examining positive predictors of CBT effectiveness in the long-term found that the presence or absence of comorbid anxiety disorders was the most significant positive predictive factor for improvement of DHI at 6-month follow-up (48). Another study analysing visual rehabilitation in 27 PPPD patients found that after 6 months, 41% achieved remission and 74% achieved remission and/or treatment response (49).

Strengths and limitations

Most studies were completed before PPPD was defined and thus investigated one of PPPD's four predecessors (14) or non-specific persistent dizziness following acute vestibular illnesses (9–13,15–20). The available evidence mainly focused on chronic dizziness post-vestibular neuritis and post-BPPV, although some studies also included other conditions. Review of chronic dizziness descriptions in those studies indicated that many patients would have met current PPPD criteria, either as their only active diagnosis following resolution of their acute illnesses or co-existing with partially uncompensated peripheral vestibular deficits. Thus, similarities across studies and concordance between older (9–20) and newer (8) investigations is reassuring. Some study sizes were small (n<30), but the total participant number in selected studies (n=780) was acceptable for this report's aims. As with all reviews, its conclusion reflects the available data and further replication in larger and more representative populations is required.

Conclusion

Overall, the evidence points to the brain, cognitions and emotions as the key drivers of residual dizziness. After acute vestibular events, the brain undergoes central adaptation and in PPPD patients it is unable to de-adapt once these have resolved. Individuals who develop chronic dizziness following a vestibular insult seem to be inherently prone to experiencing such events more acutely and fearfully and become more sensitive to their resulting bodily changes. Psychological factors and a tendency to visual dependence appear important in predicting poor outcomes; otoneurotologic factors such as disease-related abnormalities of the otolithic organ and semi-circular canals and age-related brain changes, appear to have a smaller role. Vestibular testing remains essential in PPPD assessment to

look for common comorbidities but an approach that identifies and attempts to modify predictors appears to be more important.

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Table 1. Criteria for study inclusion and exclusion.

Inclusion criteria	Exclusion criteria
Articles in English	Chronic dizziness secondary to any non-vestibular condition
Longitudinal studies analysing risk factors for chronic dizziness following a vestibular insult, retrospective or prospective in design, including either within group of between group comparisons to relevant controls.	Assessment of links between a psychiatric illness and chronic dizziness in the absence of vestibular disease
Mean follow-up period of at least 3 months	Examination of treatment outcomes of vestibular conditions
Clinical outcomes at baseline and follow-up assessed with standardized measures of symptoms, associated variables, and test results.	Only abstract available
Studies focused primarily on adults	Studies focused exclusively on children

Table 2. Chronological summary of characteristics and outcomes of studies specifically attempting to determine predictors of chronic dizziness following an acute peripheral vestibular insult.

Author (year)	Vestibular insult (n)	Chronic dizziness nomenclature used	Chronic dizziness rate (%)	Mean baseline assessment (weeks)	F/U duration (months)	F/U rate (%)	Mean age±SD (years)	F (%)	Study design	Study components[$]	Findings and cited predictors
Kabaya (2022) (8)	BPPV (51) MD (23) Psychiatric dizziness (12) Peripheral vestibular dysfunction (9) VM (8) VN (6) Other (18) Unknown (28)	PPPD	10.3	4 (32.7 days)	>3	100	55.4±16.1	60	Retrospective analyses between recovered patients and patients with PPPD.	PTA, positional/positioning nystagmus testing ENG, caloric testing, cVEMP, vHIT CT scan and MRI *Q:* NPQ, DHI	(1) The total NPQ score of the PPPD group was significantly higher than that of the non-PPPD group; among the three exacerbating factors, the upright posture or walking and the movement scores of the PPPD group were significantly higher than those of the non-PPPD group, while there were no significant differences in the visual stimulation score; (2) there were no significant differences in the results of the vestibular function test between the two groups; (3) the total scores of the DHI, and physical, emotional, and functional subscales of the DHI were not significantly different between the PPPD group and the non-PPPD group.
Lee (2020) (9)	VN (27)	Residual symptoms of VN	29.6	<1	6	100	56.4±12.7	37	Prospective comparison of recovered and chronically symptomatic patients	Video-nystagmography, vHIT (HIMP and SHIMP), cVEMP, calorics *Q:* DHI, locally standardised symptom VAS	(1) Recovered group HIMP showed higher VOR gain, higher occurrence of covert corrective saccades, lower occurrence and peak velocity of overt corrective saccades than group with residual symptoms; (2) Recovered group SHIMP showed higher VOR gain, higher occurrence and peak velocity of anti-corrective saccades than group with residual symptoms; (3) HIMP and SHIMP at 1 month are important factors to predict the residual symptoms in chronic phase of VN.
Oh (2019) (10)	BPPV (65)	Residual dizziness	24.6	<1	4	100	53.1	73.8	Retrospective analyses within patients	cVEMP Caloric testing	BPPV patients with increased cVEMP-modified IAD ratio* (≥25%) at the affected side were more likely to have residual dizziness after recovery of BPPV (OR: 6.62).
Cousins (2017) (11)	VN (40)	Chronic dizziness	-	<1	2.5 (recovery) 10 (long-term recovery)	80 65	50	36	Prospective comparison of recovered and chronically symptomatic patients	Calorics, vestibular perceptual tasks, rod-and-disk task *Q:* DHI, HADS, BSQ, VSS_A	(1) DHI score at recovery stage (10 weeks) was significantly correlated with acute autonomic arousal and acute visual dependency; (2) acute autonomic arousal (VSS_A), visual dependency and fear of bodily sensations also predicted long-term outcome (10 months); (3) worse recovery was not associated with age, baseline depression or vestibular variables; (4) recovery at 10 weeks correlated with anxiety and depression (HADS), autonomic arousal and fear of bodily sensations (BSQ) measured at 10 weeks.
Cha (2017) (12)	BPPV (120)	Chronic dizziness	7.5	<1	16±3.7	100	51.5±9.41	76.7	Retrospective analyses within patients	MRI	Brain atrophy was independently associated with long-lasting dizziness after BPPV (OR: 4.39).
Adamec (2014) (13)	VN (26)	Chronic vestibular insufficiency	42	<1	12	100	53	53.8	Prospective comparison of patients with healthy controls (for VEMP only)	VEMP, MRI Neurological examination including assessment of nystagmus and Fukuda test.	(1) Older age and chronic white matter lesions on brain MRI were positive predictors for the development of chronic vestibular insufficiency after vestibular neuritis; (2) VEMPs, nystagmus assessment and the Fukuda test were not useful to predict the development of chronic vestibular insufficiency.
Tschan (2010) (14)	BPPV (15) VN (15) MD (8) VM (24)	Somatoform dizziness and vertigo	43	1-24	12	100	52±13	41	Prospective analyses within patients	*Q:* VSS, and a psychometric test battery measuring resilience, sense of coherence, and satisfaction with life	(1) Patients with higher scores of resilience, sense of coherence, and satisfaction with life at baseline were less likely to acquire secondary somatoform dizziness at 12 months; (2) baseline mental comorbidity, lifetime mental disorders and stressful live events play a major predictive role for the long-term prognosis of dizziness and vertigo.

Heinrichs (2007) (15)	VN (24), BPPV (19)	Persisting psychogenic (phobic) dizziness	30	<1	12	100	56.2 ± 13.5	58	Prospective analyses within patients; secondary analyses within groups with VN and BPPV	DIPS, head MRI, AEP *Q:* locally standardised symptom VAS, ACQ, BAI, BDI, BSQ, MI, SCL-90-R	(1) General psychological distress, depressive and anxiety symptoms and avoidance were not associated with persisting dizziness; (2) Interpretation of bodily sensations (BSQ) was a significant predictor of persisting dizziness in patients with VN, but not in patients with BPPV, while ACQ did not significantly predict the persistence of dizzy feelings; (3) the interaction between initial subjective severity of dizziness and the BSQ was a significant predictor for the persistence of dizziness; (4) patients classified as having "phobic dizziness" with normal medical tests had a higher percentage of mental disorders in the past with all of them having experienced a depressive disorder.
Faralli (2006) (16)	Acute unilateral vestibulopathy (20)	Persistent dizziness	45	≥24	-	100	26	60	Retrospective case review	VEMPs Stabilometry	(1) VEMPs (P1/N1 and P2/N2 waveforms) were absent in all patients with persistent vertigo; three (33.3%) showed the recovery of previously absent canal function. The absence of VEMPs raises a role for otolithic dysfunction for the development of dizziness; (2) Stabilometry showed significantly higher pathological S values in all the 9 patients with persistent dizziness.
Godemann (2005)§ (17)	VN (103)	Persisting vertigo	29	6	12	78	50 ± 4.5	50	Prospective analyses within patients	DIPS, calorics, dynamic posturography *Q:* ACQ, BSQ, SCL-90-R, STAI, VSS,	(1) Patients who experience vertigo 1 year after VN experience more dysfunctional cognitions (ACQ) and fearful body sensations (BSQ); (2) patients with persisting vertigo also experience phobic avoidance, anxiety and a tendency towards somatization; (3) posturographic results did not differentiate between the two patient groups; (4) depression sub-scale did not contribute to the variation of vertigo; (5) ACQ and the BSQ together can predict the severity of the vertigo up to 30%.
Godemann (2004)§ (18)	VN (67)	Chronic vertigo	19.4	6	6	100	52 ± 14.3	56.7	Prospective case-control (patients compared to healthy volunteers)	DIPS, calorics *Q:* ACQ, BSQ, FKV, PSSI, STAI, VSS	(1) Female gender and dysfunctional coping were predictors of chronic vertigo; (2) patients who developed chronic vertigo after acute vestibular disorders were significantly more anxious, had a stronger impression of losing control over their bodies and experienced the acute dysfunction as a serious crisis; (3) "loyal-dependent", "compulsive" and "schizo-typical" personality types were significantly more prevalent among patients with chronic vertigo; (4) a tendency to evaluate body sensations fearfully (ACQ) and increased interpretation of bodily sensations (BSQ) were significantly associated with vertigo after 6 months.
Berginius (1999) (19)	VN (19)	Residual vestibular symptoms	53	<1	84-96	100	47	42.1	Reassessment of previously evaluated patients – analyses within patients	Calorics, ENG, Bekesy audiometry, stapedial reflexes *Q:* locally standardised questionnaire	(1) A small caloric side difference and a higher age at disease onset increased the risk of residual symptoms such as positioning vertigo or unsteadiness; (2) there were no differences in the mean caloric side difference at the 7- or 8-year follow-up in the groups with and without symptoms; (3) pathologically elevated stapedius reflex thresholds were not associated with larger mean caloric side difference compared with the group of patients with normal threshold.
Imate (1993) (20)	VN (43) Divided into 4 groups: **I, II, III, IV**		Exact % not provided: **I** > **II** > **IV** > **III**	**I:** 6 (n=6) **II:** 6-36 (n=7) **III:** 36-120 (n=24) **IV:** >120 (n=6)	**I:** 6 (n=6) **II:** 6-36 (n=7) **III:** 36-120 (n=24) **IV:** >120 (n=6)	58	Exact age not provided: *At onset* **2** > **4** > **1** > **3** *At re-exam* **4** > **2** > **3** > **1**	-	Reassessment of previously evaluated patients – analyses within patients	Calorics, ENG	Persistent positional nystagmus increases in patients with persistent dizziness after 3 years post-VN, which may reflect residual increase of resting activity in the ipsilateral vestibular nucleus to the affected side.

[$]In addition to structured or semi-structured interview and bedside examination
[§]Overlapping studies
*Modified IAD ratio was defined as follows: (affected side ear amplitude - unaffected side ear amplitude) ÷ (affected side ear amplitude + unaffected side ear amplitude).
ACQ – Anxiety Cognition Questionnaire; **AgCQ** – Agoraphobic Cognitions Questionnaire; **ASI** – Anxiety Sensitivity Index; **AEP** – acoustic evoked potentials; **BAI** – Beck Anxiety Inventory; **BDI** – Beck Depression Inventory; **BSQ** – Body Sensation Questionnaire; **BPPV** – benign paroxysmal positional vertigo; **cVEMP** – cervical vestibular-evoked myogenic potential; **DDI** – Depersonalisation Derealisation Inventory; **DHI** – Dizziness Handicap Index; **DIPS** – *Diagnostisches interview bei psychischen störungen* (Diagnostic interview for psychiatric disorders); **ENG** – electronystagmogram; **FKV** – *Freiburger Fragebogen zur Krankheitsverarbeitung* (Freiburg Coping Illness Questionnaire); **F/U** – follow-up; **Gp** – group; **GAD-7** – Generalised Anxiety Disorder Assessment-7; **HADS** – Hospital Anxiety and Depression Scale; **HIMP** – head impulse test; **HSN** – head shaking nystagmus; **IAD** – interaural amplitude difference; **MD** – Ménière's disease; **MI** – Mobility Inventory; **MRI** – magnetic resonance imaging; **NPQ** – Niigata PPPD Questionnaire; **OT** – ocular torsion; **oVEMP** – ocular vestibular-evoked myogenic potential; **PHQ-9** – Patient Health Questionnaire-9; **PHQ-15** – Patient Health Questionnaire-15; **PPPD** – persistent postural-perceptual dizziness; **PPV** – phobic postural vertigo; **PSSI** – *Persönlichkeitsstil- und -störungsinvetar* (Personality type and disorder inventory); **PTA** – Pure Tone Audiogram; **Q** – questionnaire; **RS** – resilience; **SCL-90-R** – Symptom Checklist-90-R; **SHIMP** – suppression head impulse test; **SOC** – sense of coherence; **STAI** – State-Trait Anxiety Inventory; **SVV** – subjective visual vertical; **SWLS** – satisfaction with life; **VAS** – visual analogue scale; **VEMP** – vestibular-evoked myogenic potential; **vHIT** – video head impulse test; **VHQ** – Vertigo Handicap Questionnaire; **VM** – vestibular migraine; **VN** – vestibular neuritis; **VOR** – vestibulo-ocular reflex; **VSS** – Vertigo Symptom Scale; **VSS_A** – Vertigo Symptom Score_arousal; **VSS-SF** – Vertigo Symptom Scale-Short Form

Table 3. Summarised interpretation of current evidence based on predictors and acute vestibular events.

Predictor	Positive studies	Negative studies	Did not assess it	Interpretation
Demographics				
Female gender	Godemann (18) (VN) (n=67)	Kabaya (8) (mixed population) (n=155) Oh (10) (BPPV) (n=65) Cha (12) (BPPV) (n=120) Adamec (13) (VN) (n=26) Heinrichs (15) (VN and BPPV) (n=43) Faralli (16) (acute vestibulopathy) (n=20)	Lee (9) Cousins (11) Tschan (14) Godemann (17) Berginius (19) Imate (20)	Female gender was found to be a predictor of chronic dizziness after VN in a single study.
Older Age	Cha (12) (BPPV) (n=120) Adamec (13) (VN) (n=26) Berginius (19) (VN) (n=19)	Lee (9) (VN) (n=27) Oh (10) (BPPV) (n=65) Cousins (11) (VN) (n=40)	Kabaya (8) Tschan (14) Heinrichs (15) Faralli (16) Godemann (17) Godemann (18) Imate (20)	Advanced age might be independently associated with a higher rate of dizziness symptoms after VN. Indirectly, brain atrophy (commonly observed in older people) was found to be associated with chronic symptoms after BPPV in a single study.
Early vestibular symptoms				
Severity of dizziness (assessed by DHI or VAS)	Heinrichs (15) (VN and BPPV) (n=43)	Kabaya (8) (mixed population) (n=155) Lee (9) (VN) (n=27)	Oh (10) Cousins (11) Cha (12) Adamec (13) Tschan (14) Faralli (16)	Severity of dizziness interacts with acute body vigilance and anxiety to predict chronic dizziness. This was found true post-VN but not after BPPV. Other studies did not find significant differences for the severity of disease between the PPPD group and the non-PPPD groups.

			Godemann (17) Godemann (18) Berginius (19) Imate (20)	
Dizziness exacerbators	Kabaya (8) (mixed population) (n=155)	-	All the other studies.	Early mean NPQ total score (a measure focused on the types of dizziness exacerbators) as well as the upright/walking and self-motion subscales, but not the visual stimulation subscale, might be useful to discriminate those who will end up developing chronic symptoms with high sensitivity but low specificity.
Visual dependence				
Visual dependence	Cousins (11) (VN) (n=40)	-	All the other studies.	An increase of visual dependence (sensitivity to visual motion and complex visual stimuli) is observed in patients with chronic dizziness, who have a higher reliance on visual input for spatial orientation and balance. Visual dependence associated with severity of vertigo, increased acute body vigilance and anxiety was found to predict persistence of dizziness. This might be a treatment target in future rehabilitation programs.
Psychological factors				
Premorbid psychiatric disorder	Tschan (14) (BPPV, VN, MD and VM) (n=62) Heinrichs (15) (VN and BPPV) (n=43)	Cousins (11) (VN) (n=40) Godemann (17) (VN) (n=103)	Lee (9) Oh (10) Cousins (11) Cha (12) Adamec (13) Faralli (16) Godemann (18) Berginius (19) Imate (20)	Having a premorbid psychiatric disorder might pose patients at a higher risk of chronic dizziness. Depression was not a predictor of chronic dizziness in two studies.
Higher resilience and general satisfaction with life	Tschan (14) (BPPV, VN, MD and VM) (n=62)	-	All the other studies.	Measures of subjective well-being seem to be protective against the development of secondary chronic dizziness.
Fear of bodily sensations, anxiety, greater autonomic arousal and heightened awareness of bodily sensations	Cousins (11) (VN) (n=40) Heinrichs (15) (VN and BPPV) (n=43) Godemann (17) (VN) (n=103) Godemann (18) (VN) (n=67)	-	Kabaya (8) Lee (9) Oh (10) Cha (12) Adamec (13) Tschan (14) Faralli (16) Berginius (19) Imate (20)	Persistent dizziness is often determined by a combination of psychological factors including a heightened awareness of bodily sensations, fear of bodily sensations and autonomic arousal. Subjects who experience vertigo as a particularly alarming event tend to focus more intensely than other patients on the negative symptoms. These factors are related to the initial severity of dizziness and were consistently reported in patients with VN, but not in BPPV.
Dependent personality traits and dysfunctional coping	Godemann (18) (VN) (n=67)	-	All the other studies.	Neuroticism and loyal-dependent", "compulsive" or "schizo-typical" personality traits might drive the shift of vertigo to a somatoform process.

Vestibular tests				
Caloric testing	Berginius (19) (VN) (n=19)	Kabaya (8) (mixed population) (n=155) Lee (9) (VN) (n=27) Oh (10) (BPPV) (n=65) Cousins (11) (VN) (n=40) Faralli (16) (acute unilateral vestibulopathy) (n=20) Godemann (17) (n=103) Godemann (18) (n=67) Imate (20) (VN) (n=43)	Cha (12) Adamec (13) Tschan (14) Heinrichs (15)	Small caloric side differences were identified as a risk factor for residual unsteadiness or vertigo after VN in a single study with a small sample size. Subsequent studies did not find caloric differences suggesting ongoing canal paresis in patients with persisting vertigo.
Positional/positioning nystagmus (including ENG and VNG)	Imate (20) (VN) (n=43)	Kabaya (8) (mixed population) (n=155) Lee (9) (VN) (n=27) Adamec (13) (VN) (n=26) Berginius (19) (VN) (n=19)	Oh (10) Cousins (11) Cha (12) Tschan (14) Heinrichs (15) Faralli (16) Godemann (17) Godemann (18)	The increase in the incidence of positional nystagmus post-VN may be due to a residual increase of resting activity in the ipsilateral vestibular nucleus to the affected side, and such abnormalities can persist for over 10 years from onset, possibly driving chronic vertigo. Studies using ENG and VNG failed to demonstrate a role for nystagmus assessment in the acute phase as a mean to predict PPPD.
HIT (VOR gain and corrector saccade)	Lee (9) (VN) (n=27)	Kabaya (8) (mixed population) (n=155)	Oh (10) Cousins (11) Cha (12) Adamec (13) Tschan (14) Heinrichs(15) Faralli (16) Godemann (17) Godemann (18) Berginius (19) Imate (20)	On the suppression HIT, some recovered patients show higher VOR gain, higher occurrence and peak velocity of anti-corrective saccades in comparison with the group with residual symptoms. These findings were not replicated by others, particularly because the usefulness of HIT seems to wane over time.
VEMP	Faralli (16) (acute unilateral vestibulopathy) (n=20) Oh (10) (BPPV) (n=65)	Adamec (13) (VN) (n=26) Kabaya (8) (mixed population) (n=155) Lee (9) (VN) (n=27)	Cousins (11) Cha (12) Tschan (14) Heinrichs (15) Godemann (17) Godemann (18) Berginius (19) Imate (20)	Increased cVEMP inter-aural difference ratios in the affected side and absent VEMPs waveforms were associated with residual dizziness, suggesting a potential role of inferior vestibular nerve function for symptom persistence and long-term postural impairment. This was not replicated in three other studies.
Stabilometry	Faralli (16) (acute unilateral vestibulopathy) (n=20)	-	All the other studies.	A single study described higher pathological values on stabilometry in patients with persistent dizziness after acute unilateral vestibulopathy.
Posturography	-	Godemann (17) (VN) (n=103)	All the other studies.	Current evidence does not support the use posturography to predict PPPD.

		Faralli (16) (acute unilateral vestibulopathy) (n=20)		
Audiometry	-	Faralli (16) (acute unilateral vestibulopathy) (n=20) Berginius (19) (VN) (n=19) Kabaya (8) (mixed population) (n=155)	All the other studies.	Current evidence does not support the use of audiometry to predict PPPD.
Brain imaging				
Age-related brain atrophy	Cha (12) (BPPV) (n=120) Adamec (13) (VN) (n=26)	Kabaya (8) (mixed population) (n=155) Heinrichs (15) (VN and BPPV) (n=43)	Lee (9) Oh (10) Cousins (11) Tschan (14) Faralli (16) Godemann (17) Godemann (18) Berginius (19) Imate (20)	Otoneurotologic disease-related changes in brain structure might accompany brain atrophy development and long-term dizziness, but further investigation is needed.
White matter hyperintensities	Cha (12) (BPPV) (n=120) Adamec (13) (VN) (n=26)	Kabaya (8) (mixed population) (n=155) Heinrichs (15) (VN and BPPV) (n=43)	Cousins (11) Tschan (14) Godemann (17) Godemann (18) Imate (20) Berginius (19) Lee (9) Faralli (16) Oh (10)	White matter hyperintensities (a feature of chronic small vessel disease) were independently associated with residual dizziness and may reflect underlying physiological and psychological changes in patients with PPPD. Further investigation is needed to confirm a potential role of imaging biomarkers in predicting PPPD.

BPPV – benign paroxysmal positional vertigo; **ENG –** electronystagmography; **HIT –** head impulse test; **MD –** Ménière's disease; **NPQ** – Niigata PPPD Questionnaire; **PPPD –** persistent postural-perceptual dizziness; **VEMP –** vestibular-evoked myogenic potential; **VM –** vestibular migraine; **VN –** vestibular neuritis; **VNG** – videonystagmography; **VOR –** vestibulo-ocular reflex

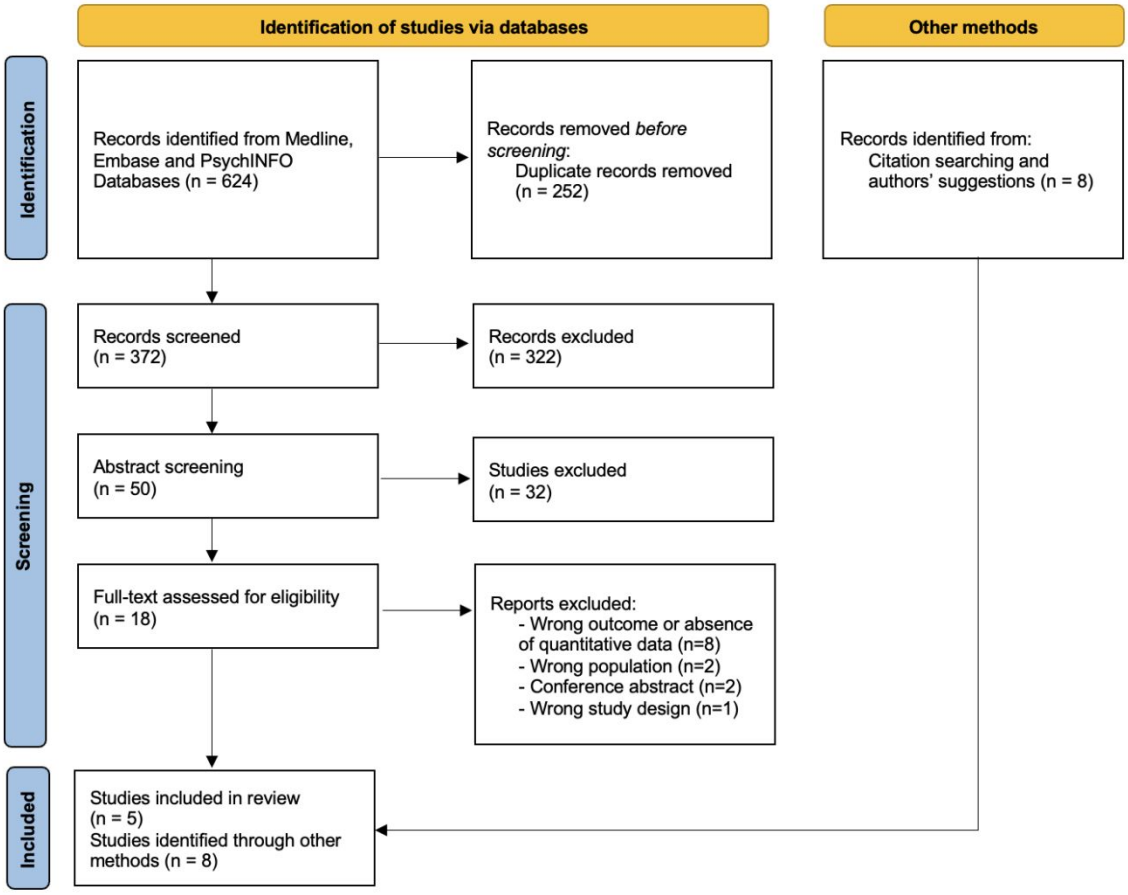
Legend

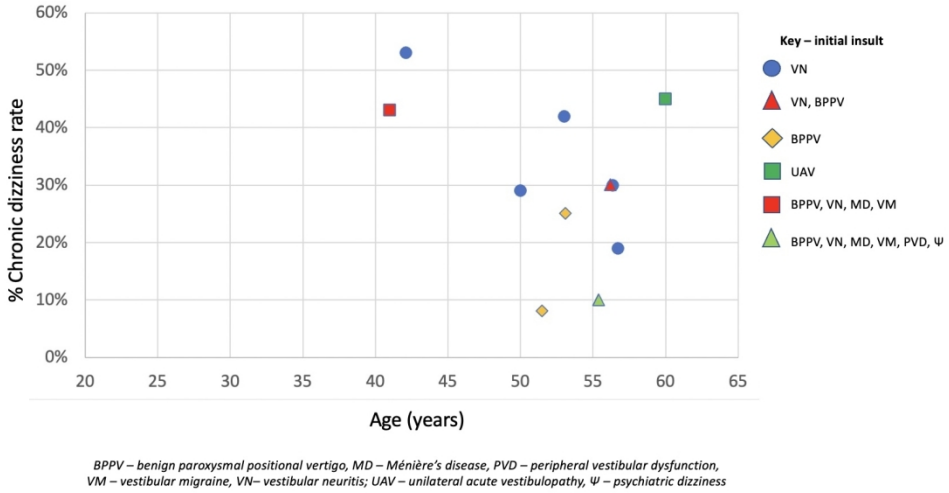
Figure 1. PRISMA flowchart for study selection.

Figure 2. Scatter plot showing relationship of three study characteristics: median age, initial insult pathology and chronic dizziness rate. Studies that did not record an exact chronic dizziness rate (10,19) are not shown.

Figure 3. Scatter plot showing relationship of three study characteristics: female gender, initial insult pathology and chronic dizziness rate. Studies that did not record an exact chronic dizziness rate or female distribution (11,20) are not shown.

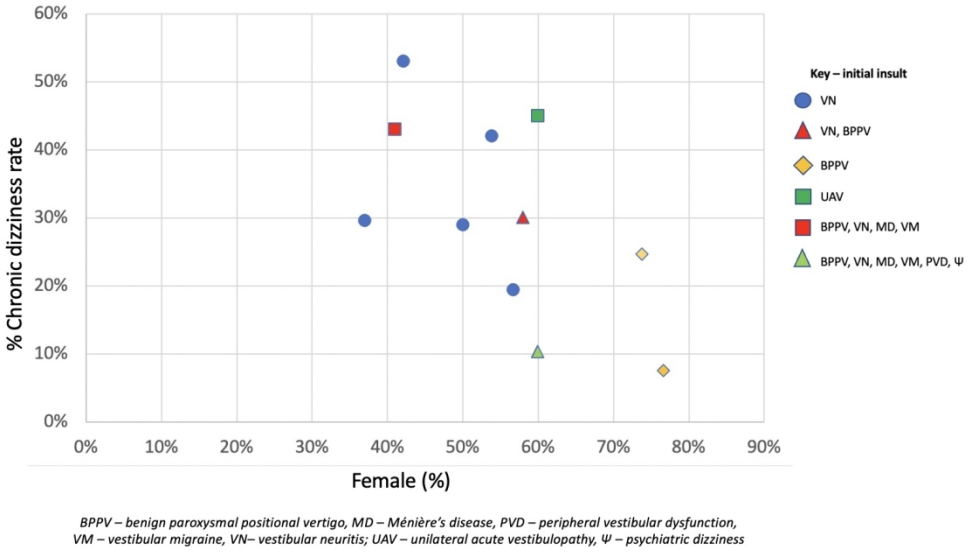
Figure 4. Schematic showing pathways linking pre-existing conditions, acute events, and chronic outcomes in patients with persistent dizziness. Blue text and arrows show links associated with PPPD. Red text and arrows show links associated with comorbid psychiatric disorders. Green text and arrows depict links related to structural illnesses. The thin green arrows compared to the thick blue and red arrows denote the smaller contribution of structural illnesses to chronic symptoms compared to PPPD and comorbid psychiatric disorders. (-) indicates that resilient personality traits decrease risk of downstream effects in contrast to all other connections that increase risk.





BPPV – benign paroxysmal positional vertigo, MD – Ménière's disease, PVD – peripheral vestibular dysfunction, VM – vestibular migraine, VN– vestibular neuritis; UAV – unilateral acute vestibulopathy, Ψ – psychiatric dizziness

310x162mm (144 x 144 DPI)



BPPV – benign paroxysmal positional vertigo, MD – Ménière's disease, PVD – peripheral vestibular dysfunction, VM – vestibular migraine, VN– vestibular neuritis; UAV – unilateral acute vestibulopathy, Ψ – psychiatric dizziness

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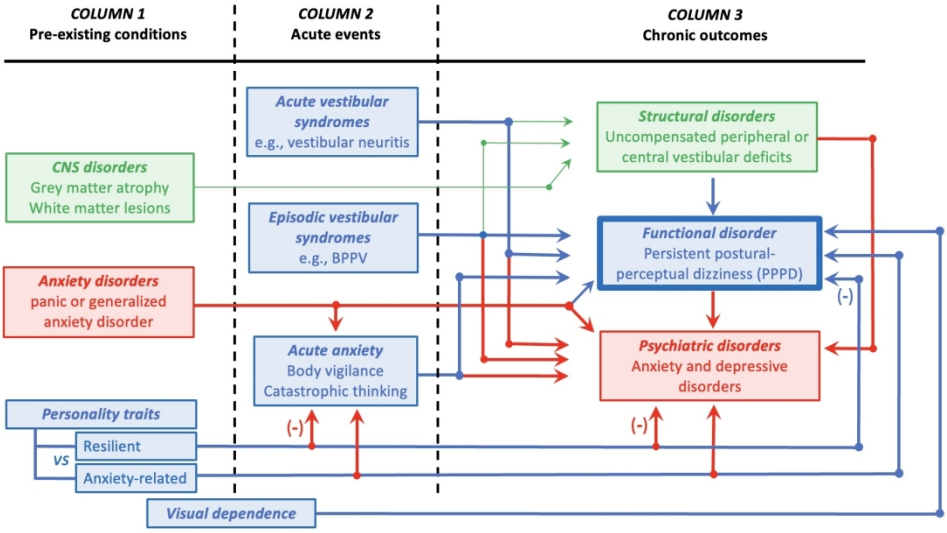

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