Exploring Factors Associated with Persistent Postural-Perceptual Dizziness (PPPD): A Mixed-Methods Approach

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Thesis submitted for the degree of

Doctor of Philosophy

DECLARATION

This work has not been submitted in substance for any other degree or award at this or any other university or place of learning, nor is being submitted concurrently in candidature for any degree or other award.

Signed: Ryan Gamble (candidate). Date: 24/12/2021. Corrected Submission Date:18/05/2022.

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This Thesis is being submitted in partial fulfilment of the requirements for the degree of PhD.

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This Thesis is the result of my own independent work/investigation, except where otherwise stated. Other sources are acknowledged by explicit references. The views expressed are my own.

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Thesis Summary

Persistent Postural-Perceptual Dizziness (PPPD) is a functional (non-organic), debilitating neuro-vestibular condition characterised by chronic episodes of visually induced dizziness (Bronstein, 1995; Staab et al., 2017). Symptoms are triggered by situations of visuo-vestibular conflict, intense visual-environments and active and passive motion (Bronstein, 2004; Pavlou, 2010). PPPD is thought to develop due to an over reliance on vision for postural control mechanisms (Bronstein, 1995). The aim of this Thesis was to explore factors associated with PPPD. The aim of Chapter 2 was to explore how individuals with PPPD make sense of their symptoms and condition and to better understand the lived experiences of PPPD, including the psycho-social impacts of the condition. Findings suggest identity loss, dismissal and non-belief, poor psychological well-being, out of body experiences and processes of sense-making are factors associated with PPPD. Shared themes included poor metal well-being, sensory overload, sleep impairments and PPPD not affecting television/movie watching. The aim of Chapter 3 was to test if short-term exposure to optokinetic stimulation can produce changes in markers of visual dependence and to establish the promise of multi-media technology for producing recalibration effects. Findings suggest that passively viewing movies are the most effective optokinetic stimulus for recalibration effects. The aim of Chapter 4 was to explore the relationships between self-reported PPPD symptoms and performance on traditionally used laboratory measures of visual dependence in the non-clinical student and sub-clinical student populations. Findings suggest that PPPD symptoms do not correlate with levels of visual dependence. Furthermore, traditional measures of visual dependence also failed to correlate. Taken together, this Thesis provides the scientific community with a deeper understanding of factors associated with PPPD in clinical and non-clinical communities.

Contents

Acknowledgements

"Our lives are not our own. From womb to tomb, we are bound to others. Past and present. And by each crime and every kindness, we birth our future." – Somni-451 (David Mitchell's Cloud Atlas).

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Chapter 1: General Introduction

1.1. Overview

Persistent Postural-Perceptual Dizziness (PPPD) is a neuro-vestibular condition characterised by chronic episodes of visually induced dizziness, disequilibrium, non-spinning vertigo and postural instability (Bronstein, 1995; Deitrich & Staab, 2017; Seemungal & Passamonti, 2018; Staab et al., 2017; Wurthman et al., 2017). Symptoms are triggered by situations of visuo-vestibular conflict, intense visual-environments, and active or passive motion (Bronstein, 2004; Pavlou, 2010). Commonly reported triggers include cluttered supermarket aisles, repeated patterns, and busy moving traffic (McCabe,1975; Soheston, Bittar & Staab, 2016; Staab et al., 2017). The aim of this Thesis was to explore factors associated with PPPD.

Chapter 1 aims to provide the reader with a comprehensive understanding of the neuro-vestibular condition PPPP and the factors associated with the condition. Chapter 1 is split into four main parts for clarity. Each section aims to disseminate key information and literature regarding the condition PPPD. The first section of the General Introduction aims to introduce the PPPD condition, which includes an explanation of the leading theory of how it emerges and the laboratory measures that can be used to indicate symptoms. The second section of the Chapter aims to disseminate how PPPD is rehabilitated. This reflects background literature for the experiments presented in Chapter 3. The third section of Chapter 1 explores specific conditions and factors associated with PPPD. This offers information relevant to the investigations presented in of Chapter 2 and 4. The fourth section of Chapter 1 documents the lived experiences of PPPD. This will prepare the reader for the empirical studies reported in Chapter 2. The General Introduction can therefore be thought of as four complementing sections that will allow the reader to have a comprehensive introduction to PPPD and factors associated with the condition.

1.2. Section 1: Introducing Persistent Postural-Perceptual Dizziness

The first section of Chapter 1 aims to introduce PPPD, explore the history of the disorder and present the reader with the leading theory of how it is theorised to emerge. This section also documents the laboratory measures that can be used to indicate the presence of symptoms.

1.2.1. What is Persistent Postural-Perceptual Dizziness?

PPPD is a functional debilitating neuro-vestibular disorder characterised by chronic episodes of dizziness, disequilibrium, postural instability and non-spinning vertigo (Bronstein, 1995; Deitrich & Staab, 2017; Seemungal & Passamonti, 2018; Staab et al., 2017; Wurthman et al., 2017). Symptoms are triggered by situations of visuo-vestibular conflict, intense visual-environments and active and passive motion (Bronstein, 1995; Bronstein, 2004; Pavlou, 2010; Staab et al., 2017). Commonly reported triggers include cluttered supermarket aisles and busy moving traffic (McCabe,1975; Söheston, Bittar & Staab, 2016; Staab et al., 2017). PPPD is the second most common condition reported in dizziness clinics and is most prevalent in females and the middle aged (Dietrerich, Staab & Brandt, 2016; Strupp et al., 2003). Typically, people develop this disorder upon recovery from an acute vestibular compromising illness such as vestibular neuritis or labyrinthitis (Cousins et al., 2014; Trinidade & Goebel, 2018). PPPD is correlated with anxiety and it is common for PPPD patients to develop this secondary psychiatric illness (Guerraz et al., 2001; Staab, Eckhardt-Henn, Horri, Jacob & Stupp, 2017; Zur et al., 2015). More recently it has, however, been suggested that anxiety may be a pre-existing risk factor for developing the disorder, introducing a 'chicken and egg problem' between anxiety and PPPD (Honaker, 2018). Regardless, the PPPD condition results in a debilitated patient with complex co-occurring illnesses that implicate quality of life (Zur et al., 2015).

1.2.2. Labels for Visually Induced Dizziness

PPPD is a relatively new diagnostic label (Staab et al., 2017); however, the disorder has a rich documented history, albeit under a range of different names. Thus, although the diagnostic label of PPPD is new, the recognition of its symptoms and the categorisation of the disorder is not.

Visually induced dizziness was first documented in the late 1800's where symptoms of chronic subjective dizziness were associated with either the marketplace, anxiety or both (Benedikt, 1870; Cordes, 1872; Wetphal, 1871). Benedikt (1870) documented patient reports of visually induced dizziness as Platzschwindel, which translates to '*vertigo* in the plaza or square'. Benedikt hypothesised the condition to be of an ophthalmologic (eye) or neurological (nerves, nervous system) nature. Westphal (1871) documented similar patient experiences, labelling the condition Die Agoraphobie, which translates to a '*fear* of the marketplace'. This was the first-time experiences of visually induced dizziness were associated with anxiety, including anxiousness of the sensations of vertigo, and unsteadiness, but also of any actions, behaviours and scenarios that may trigger symptoms, such as going to the marketplace. Cordes (1872) then wrote of patients reporting the same visual syndrome and, like Westphal, wrote of the anxiety these people report. Cordes labelled this condition Platzangst, which translates to '*fear* of the plaza or the square' again placing emphasis on

anxiety. Later, Lannois and Turner (1898) documented similar experiences concluding that this was a condition that arises post-otologic (ear) disease, especially in the anxious. Thus, even when considered to be of an otologic nature, the idea that anxiety was a central part of the condition was always present. Thus, early documentation on visually induced dizziness phenomena has always associated the condition with either the visually complex marketplace, anxiety or both.

The idea of cluttered and visually complex marketplaces resurfaced in the form of 'Supermarket Syndrome' (McCabe, 1975). With this condition people reported severe dizziness, disequilibrium and non-spinning vertigo when walking along supermarket aisles. Like the preceding disorders, Platzschwindel, Platzangst and Die Agoraphobie, people with Supermarket Syndrome also reported anxiety. This reinforces the idea that the condition is inextricably linked to the visually cluttered and complex marketplace, and in more contemporary times the supermarket, and anxiety.

Similar dizziness conditions have also been documented in other cluttered visual environments. Marks and Bebbington (1976) wrote on 'Space Phobia' where people avoided (and then learned to fear) complex visual environments. Marks and Bebbington posited that Space Phobia maniftested in those with an intolerance to visuo-spatial motion cues (Marks, 1981). Hoffman and Brooker (1978) also documented patients with similar experiences; however, the authors stated that these experiences were due to a predominately visual problem, and these experiences should therefore be called 'visually induced motion symptoms'. This term effectively captured the visual nature of the condition. Visually induced dizziness symptoms are therefore not solely present in marketplaces and supermarkets, but also occur in other visually cluttered and complex environments.

Research has also suggested that personality may be related to experiences of visually induced dizziness. Brandt and Dietricht (1986) explored symptoms in a relatively large vestibular patient group and formed the first contemporary diagnostic label for the condition. A label that was not context specific and could capture a diverse range of triggers of dizziness and all associated symptoms such as; disequilibrium; disorientation; postural instability and *anxiety*. This label was Phobischer Attacken-Schwankschwindel or rather 'Phobic Postural Vertigo'. The researchers documented that anxiety may manifest because exogenous or endogenous motion cues and/or because of the anticipation of these experiences. Anticipating motion and vestibular symptoms has been shown to produced hypervigilant state where the individual forms an unhealthy conscious awareness of self, and world, motion which may promote vestibular symptoms (see Wuerhr et al., 2013). Brandt and Dietricht (1986) posited that personality factors could affect, and predict, the existence of symptoms. Notably, they found that those with a neurotic personality were more likely to develop dizziness symptoms and to display depressive tendencies. This suggests that visually induced dizziness may not

be a solely visual problem (Hoffman & Brooker, 1978); the condition must have some psychological basis due to its association with psychological factors such as anxiety and the neurotic personality type.

Jacob, Moller, Turner and Wall (1986) explored the prevalence of vestibular symptoms in clinically anxious patient groups. This was done in order to form a theoretical basis of visually induced dizziness. Jacob and colleagues established a condition that they referred to as 'Space and Motion Discomfort' (Jacob et al., 1989; Jacob et al., 1993). This condition captured an increased awareness of self and world motion and the general discomfort that can be involved in spatial orientation and navigation in these people (Jacob et al., 1993). Jacob, Redfern and Furman (2009) later suggested that Space and Motion Discomfort occurs when exposed to complex and/or moving visual stimuli and symptoms can occur when the observer is stationary or moving. It was suggested that these experiences occur due to an over-reliance on somatosensory (sensation) cues for postural control mechanisms, resulting in an over sensitivity to actual or presumed changes in somatosensory experiences. This was the first theoretical model of visually induced dizziness to suggest that the condition manifests due to a sub-optimal sensory organisation of the multi-sensory cues that facilitate postural control and self-motion perception.

Bronstein (1995), in his seminal paper, used the term visual vertigo to describe dizziness, disequilibrium, non-spinning vertigo and nausea from exposure to complex visual patterns when stationary or during ambulation (Bronstein, 2004; Dieterich & Staab, 2017; Staab, 2006). Bronstein documented reported that approximately 1/3 of his acute vestibular patients presented with visually induced dizziness (or vertigo) after recovery from their initial vestibular insult (such as vestibular neuritis: Cousins et al., 2014). This specific sub-set of vestibular patients no longer had an active vestibular compromising illness but seemed to have developed a secondary visually induced dizziness condition that persisted after recovery of their first illness. Visual vertigo has consistently been found to correlate with anxiety; in line with findings using different diagnostic labels (Balaban & Jacob, 2001; Furman, Balaban & Jacob, 2001; Guerraz et al., 2001; Lannois & Tournier, 1898; Zur et al., 2015). The adoption of the term visual vertigo placed the emphasis of the disorder onto somatosensory cues – specifically the visual cue. Bronstein suggests that this residual dizziness in the by-product of the central nervous systems ability to dynamically re-weight cues when the reliability of the vestibular signal is compromised (or unreliable) which becomes maladaptive once the acute vestibular illness surpasses. This results in an over-dependence on vision for postural control. This is the theory of visual dependence and is discussed in detail later in this section of Chapter 1.

More recently, the diagnostic label PPPD was preceded by Chronic Subjective Dizziness (Staab & Ruckenstin, 2007). Chronic Subjective Dizziness reflected earlier disorders such as Phobic Postural Vertigo but focused primarily on the sensory and physical nature of the condition rather than any associated psychological factors. (Staab et al., 2017). To satisfy a diagnosis of Chronic Subjective Dizziness the patient had to present with the typical persistent non-spinning vertigo and disequilibrium symptoms and these symptoms had to have been triggered by self and world motion. Staab and Ruckenstein were the first to suggest that this visually induced dizziness is the by-product of the individual developing hypersensitivity to motion cues. The diagnostic label of Chronic Subjective Dizziness therefore overlaps with the theory of visual dependence and visual vertigo by also suggesting some underlying sensitivity to visual motion cues or some maladaptive, sub-optimal, sensory organisation.

1.2.3. The Newly Established Persistent Postural-Perceptual Dizziness

A new diagnostic label was recently developed that unifies previous nomenclature. This diagnostic label is PPPD. The term PPPD reflects the nature of the visually induced dizziness condition. A condition that is *persistent* and triggered or exacerbated by the upright *posture* and one that produces *perceptions* of *dizziness*, disequilibrium and vertigo. The condition may also impair *postural* control mechanisms and produce inaccurate *perceptions* of motion based on sensory input (predominantly from the visual channel). This produces a sensitivity to visual motion. Visual motion cues that arise from self-motion [egocentric cues] and/or world-motion [eccentric cues] may produce symptoms and destabilise postural control mechanisms (Popkirov, Staab & Stone, 2018; Popkirov, Stone & Holle-Lee, 2018; Yan et al., 2017).

Staab et al. (2017) state that there are 5 diagnostic criteria that must be satisfied for the diagnosis of the disorder:

1. One or more symptoms of dizziness, unsteadiness, or non-spinning vertigo are present on most days for 3 months or more. This captures the persistence element of the condition.
2. Persistent symptoms occur without specific provocation but are exacerbated by three specific factors: a) the upright posture, b) active or passive motion (regardless of direction or position, and c) exposure to moving or complex visual patterns.
3. The disorder is precipitated by conditions that cause vertigo, unsteadiness, dizziness or problems with balance including acute, episodic or chronic vestibular syndromes, other neurological or medical problems, or psychological distress.
    a. When the precipitant is an acute or episodic condition, symptoms settle into the pattern of criteria A as the precipitant resolves, but they may occur intermittently at first, then consolidate into persistent course.

b. When the precipitant is a chronic syndrome, symptoms may develop slowly at first and worsen gradually.

4. Symptoms cause significant distress or functional impairment.
5. Symptoms are not better accounted for by another disorder.

Accurate detection and diagnosis of PPPD relies on subjective clinical judgement. These judgments are made clinical scientists and/or vestibular therapists or other allied medical scientists (otologists, neurologists, ophthalmologists etc.). Potential PPPD patients are typically asked to give contextual background information relating to their symptoms, triggers, functional impairments and any associated distress. It is the role of the experienced clinician to establish how well the diagnosis criteria explains the individual's experiences and presentation. The clinician would likely explore the structural, functional and psychological history of the individual. For example, the clinician may assess if the patient presents with a history of vestibular compromise, stress or psychological illness. Clinicians may also judge whether the patient presents with signs of anxiety or a neuroticism. The putative mechanisms of PPPD are presented in Figure 1.1 showing the domains the clinician may probe as part of the clinical assessment.

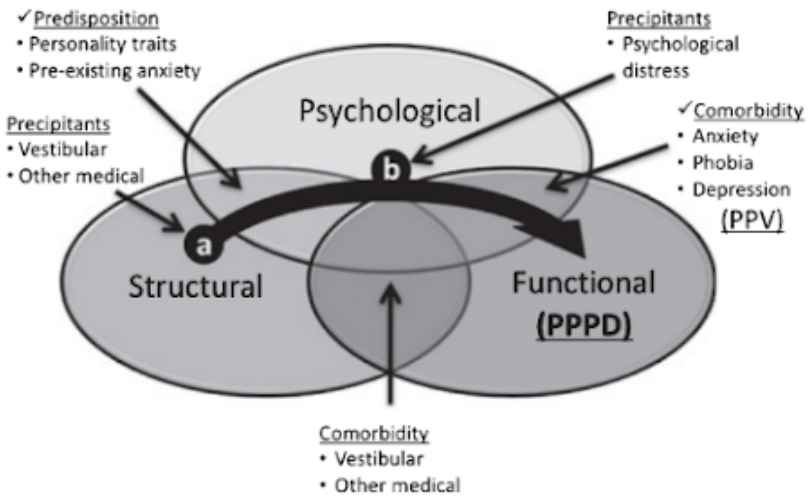

Figure 1.1. The putative mechanisms of PPPD (adapted from Staab et al., 2017, p.204).

1.2.4. Visual Dependence: The leading theory of Persistent Postural-Perceptual Dizziness

PPPD is thought to be the by-product of overly depending on vision for postural control mechanisms (Bronstein, 1995; Bronstein, 2004; Pavlou, 2010). Humans are visually dominant beings, meaning they rely on the visual cue for perception, action and postural control (Dovidio & Ellyson, 1985; Ray, Horvat Croce, Mason & Wolf, 2008; Rushton, Harris, Lloyd & Wann, 1998; Stins et al., 2009),

though the degree to which people rely on this cue differs greatly between people (Sherman, 1967; Willey & Jackson, 2014; Witkin & Goodenough, 1977; Witkin, Moore, Goodenough & Cox, 1977; Witkin, 1967). This is a normal function; however, the importance of vision can be *weighted* to adapt to the demands of life. Evidence from psychophysical and neurophysiological studies strongly suggest that weighting is based on reliability of the sensory signal (Fetsch, Pouget, DeAngelis & Angelaki, 2011; Mejier & Noppeney, 2020). The senses that facilitate balance and postural control rely on functioning in a state of equilibrium for optimum performance and perception. PPPD is therefore theorised to develop as the central nervous system has coded the visual cue as more reliable and it is therefore given more weight when sensory integration occurs. For a graphical overview see Figure 1.2.

Typically, the PPPD patient presents as having had a vestibular compromising illness, such as vestibular neuritis in recent months (Cousins et al., 2014). Once the vestibular system has recovered, and full compensation returns, a residual chronic dizziness persists. Vestibular signals become unreliable and thus are down-weighted by the central nervous system in favour of the alternative cues for self-motion perception and postural control; namely visual signals and the proprioceptive systems. As humans are visually dominant beings, the emphasis is mainly placed on the visual cue for postural control (Dovidio & Ellyson, 1985; Ray, Horvat Croce, Mason & Wolf, 2008). This mechanism is adaptive as it is responding to the unreliability and error of erroneous vestibular signals that result in inaccurate and unreliable estimates of self-motion. This results in perceptions of self-motion whilst physically stationary. This cue is down weighted in an attempt to avoid incorrect estimates and perceptions to occur (Fetsch, Pouget, DeAngelis & Angelaki, 2011; Fetsch, DeAngelis & Angelaki, 2010; Medendorp, Alberts, Verhagen, Koppen & Selen 2018.) It is therefore theorised that PPPD manifests due to a maladaptive dependence on vision after recovery of the vestibular system. The process that was once adaptive now becomes maladaptive and has results in an over-dependence on vision for postural control mechanisms and self-motion perception. The visual signal is therefore up-weighted and is coded as the most reliable estimate for self-motion; a process theorised to be conducted by a central re-weighting system (for an introduction to reliability-based weighting see; Ernst & Banks, 2002; Aller & Noppeney, 2019; Rohe & Noppeny, 2015) though the location and neural network facilitating this function is still debated. Thus, what was once an adaptive process, during the initial vestibular insult, ultimately persists after it is functionally required, becoming maladaptive. The dependence on vision is no longer necessary but persists meaning that sensory organisations between the senses that facilitate postural control and self-motion perception become sub-optimal and this is thought to facilitate PPPD.

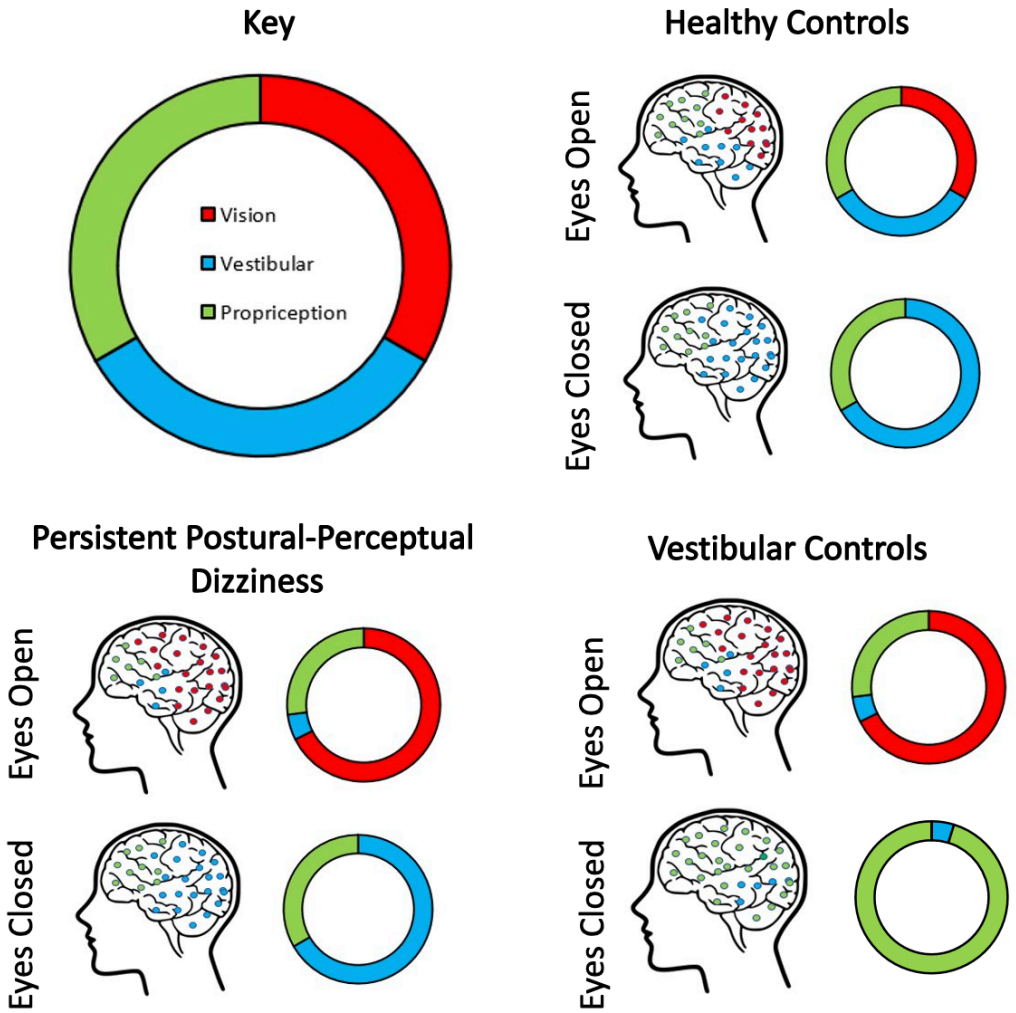

Figure 1.2. Graphical depiction of the sensory contributions for postural control mechanisms in healthy controls, the PPPD and vestibular control groups.

1.2.5. Evidence in Support of the Theory of Visual Dependence

Evidence has supported the theory of visual dependence for the manifestation of PPPD in patients (Guerraz et al., 2001). PPPD patients (then called visual vertigo patients), 'labyrinthine defective controls' (a vestibular control group free form visually induced dizziness) and healthy people free from vestibular compromise and/or PPPD were recruited by the researchers. All participants were required to complete two common laboratory measures of visual dependence; the Rod and Frame Test of the Subjective Visual Vertical (Asche & Wilkin, 1948a; Asche & Wilkin, 1948b) and Postural Kinematics - measures of physical sway in response to visual information (Barnes & Crutchfeild, 1990; Ledin et al., 1991; Koozekanani, Stockwell, McGhee & Firoozmand, 1980; Guerraz et al., 2001; Redfern, Yardley & Bronstein, 2001; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). These tests are explained in detail in the section 3.2.1.2, Chapter 3. Postural and perceptual impairments were apparent in both vestibular groups; however, only the labyrinthine defective control

patients showed an increased ratio between their sway path with eyes closed and eyes open (Romberg Quotient) representing a larger stabilizing effect of vision (Bronstein, Hood, Gresty & Panagi, 1990; Colledge et al., 1994; Vartianen, Holm, Kosinen & Hokkanen, 2017). This suggests that when eyes are closed, the labyrinthine defective controls patients were unable to re-weight and rely on the vestibular cue for self-motion, as this cue is fully compromised. This effect is not present in the healthy controls or PPPD group, who can rely on their vestibular cue when forced to do so. This suggests that PPPD patients have a down-weighted vestibular cue when visual information is available (see Figure 1.2. Note, this is an oversimplification and other information sources such as efferent copies and muscle are neglected for theoretical parsimony, in line with the literatures approach).

Relatedly, Guerraz et al. (2001) documented specific postural responses to visual motion in the PPPD patient group. The ratio between sway when visual (rotational) motion is present and sway when static visual information is presented. This ratio is referred to as a Vection Quotient (see section 3.2.1.2.2, Chapter 3). PPPD patients were shown to have increased Vection Quotients, but this was not evident in healthy controls or vestibular compromised controls. As this quotient represents the degree to which an individual is destabilised by visual motion, this suggests that PPPD patients found it more difficult than either control group to supress inaccurate visual cues for self-motion. This suggests that PPPD patients struggle to resolve visuo-vestibular conflict. This seminal finding suggests a maladaptive dependence on vision in those who report PPPD symptoms and this is evidenced by the lack of change in the increased destabilisation caused by a moving visual stimulus. This may suggest that the key to rehabilitating PPPD patients may lie in desensitising them to visual cues of self-motion.

1.2.6. Constraints and Limitations of the Theory of Visual Dependence

Visual dependence may not explain the existence of symptoms in all cases of PPPD, as not all PPPD patients present with previous vestibular compromising illness (Bronstein, 1995). For example, some present with high levels of stress or psychological illness (Dieterich & Staab, 2016; Popkirov, Staab & Stone, 2018; Trindade & Goebel, 2018). This may suggest that psychological or functional disorders may also affect the calibration of the senses that facilitate postural control mechanisms. Alternatively, this may suggest that the theory of visual dependence may best account for PPPD symptoms in people with no previous history of vestibular compromises.

Research is yet to establish where in the brain this central re-weighting process occurs and how it functions beyond the idea of reliability-based weighting. The theory of visual dependence may therefore fall criticism to the black-box problem – the idea that researchers cannot truly establish if

the theories of cognitive processing best represent the true underlying bio-mechanical processes (for a primer on the black-box theory consult: Adadi & Berrada, 2018; Blanco, 1995). Visual dependence can therefore be conceptualised as the best scientific model of PPPD manifestation that is possible, that is yet to be falsified.

PPPD symptoms have also been identified in the general healthy population (using the Visual Vertigo Analogue Scale (VVAS): Dannenbaum, Chilingaryan & Fung, 2011). The VVAS and its psychometric properties are documented in section 2.6.1.2.1 of Chapter 2. See Appendix 1.1 for the scale. Recent published research has shown that although most generally healthy people report no PPPD symptoms, approximately 11% of the adult population report PPPD symptoms that are greater than or equal than the lowest scoring PPPD patient – a score of 25 on the VVAS (Powell, Derry-Sumner, Rajenderkumar, Rushton & Sumner, 2020). To date, it is not known if these people have an over-dependence on vison. The aim of Chapter 4 was to better explore this by correlating scores on two commonly used laboratory measures of visual dependence with PPPD scores in the generally healthy student population. If the theory of visual dependence could explain the existence of symptoms in these people then scores should correlate positively, with a higher visual dependence co-occurring with high levels of PPPD symptoms.

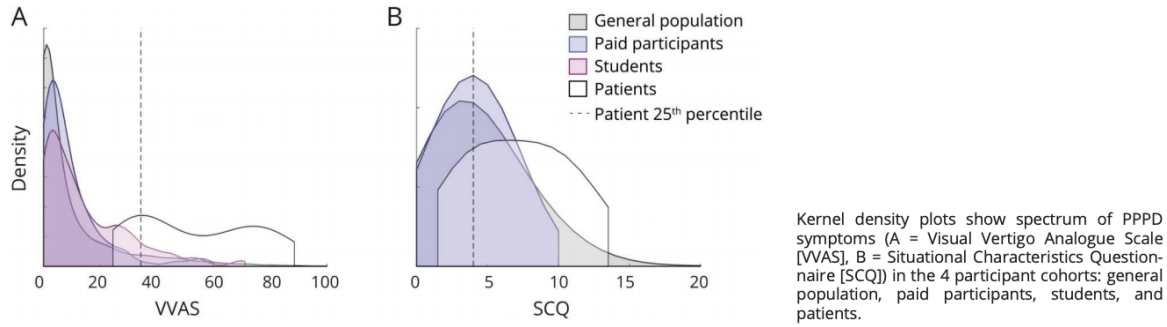

Figure 1.3. Data from Powell et al. (2020, p.1933) showing that VVAS scores are on a spectrum in the general healthy population and that approximately 11% of respondents report PPPD levels equal to or greater than the lowest scoring PPPD patients.

1.2.7. Measuring Persistent-Postural Perceptual Dizziness: The use of Laboratory Measures

A number of laboratory measures can be used can be used to indicate the presence of symptoms related to PPPD. Such measures include the questionnaire-based tools like the Visual Vertigo Analogue Scale (VVAS; Dannenbaum, Chilingaryan & Fung, 2011) and the Niigata PPPD Questionnaire (NPQ; Yagi et al., 2019).

Measures of visual dependence can also be used to infer the presence of PPPD (see Bronstein, 1995; Guerraz et al., 2001). There are two classic laboratory measures of visual dependence. The first is the Rod and Frame test of the Subjective Visual Vertical (Asch & Wilkin, 1948a; Asch & Wilkin, 1948b). The second is the use of Postural Kinematics (body sway) and the formation of a Vection Quotient; a ratio which represents the destabilising effect of visual motion (Barnes & Crutchfeild, 1990; Ledin et al., 1991; Koozekanani, Stockwell, McGhee & Firoozmand, 1980; Guerraz et al., 2001; Redfern, Yardley & Bronstein, 2001; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). These measures are thought to capture the underlying dependence on vision for postural control and balance mechanisms. These measures do not detect actual PPPD symptoms but can be used to infer the presence of PPPD for they quantify visual dependence. These methods are explained in detail in Chapter 3, section 3.2.1.2.

Note, in wider vestibular research another quotient can be formed to estimate visual dependence that also assesses vestibular function – the Romberg Quotient. The Romberg Quotient is a postural sway measure that indicates the degree to which an observer relies on visual information for postural control. It is a commonly used measure in vestibular research and includes postural sway data when no visual information is available (Cornilleau-Pérès, 2005; Lanska, 2002; Romberg, 1843; Vartiainen, Holm, Koskinen & Hokkanen, 2018). It is important to note that people with PPPD usually present with typically healthy Romberg Quotients (Guerraz et al., 2001) as mentioned above.

This first section of the General Introduction Chapter has introduced the condition PPPD, offered the leading theory of how it is thought to develop and considered the laboratory measures/tools that can infer its presence. This provides the reader with key information relevant to Chapter 3 where the researcher aimed to explore the short-term effects of optokinetic stimulation on markers of visual dependence. This section also provides information relevant to Chapter 4 where the researcher aimed to correlate scores PPPD symptoms (as measured by the VVAS: Dannenbaum, Chilingaryan & Fung, 2011) and performance on the two commonly used laboratory measures of visual dependence.

1.3. Section 2: Rehabilitating Persistent Postural-Perceptual Dizziness

The second section of Chapter 1 aims to discuss how PPPD can be rehabilitated and provides background literature relevant to Chapter 3. This section covers an introduction to Vestibular and Balance Rehabilitation Therapy, Visual Desensitisation approaches for rehabilitating PPPD and explores the different visual stimuli that have been used as rehabilitation tools for desensitising the PPPD patient to visual triggers of symptoms. This section will highlight that rehabilitation tools are often simplistic and unengaging. Thus, this section will also provide the motivation for Chapter 3; a

Chapter designed to test the potential for engaging multi-media stimuli to be used as rehabilitation tools.

Several rehabilitation paradigms are available to the PPPD patient (see Popkirov, Stone & Holle, 2018; Popkirov, Staab & Stone, 2018). They fall into three categories; psychological therapies (Holmberg, Karlberg, Harlacher & Mgnusson, 2007; Mahoney, Edelman & Cremer, 2013), psychopharmacological interventions (Esin, Khairullin, Mukhametova & Esin, 2017; Staab, Ruckstein & Amsterdam, 2004; Sluck, Elliot, Dvorak, Ding & Farris, 2017), and Vestibular and Balance Rehabilitation Therapy (VRBT: Hannsson, 2007; Meldrum & McConn-Walsh, 2018; Pavlou, 2010; Pavlou, Davies & Bronstein, 2006; Pavlou et al., 2012; Whitney et al., 2006; Wrigley & Pavlou, 2005; Shumway-Cook & Horak, 1990) with VRBT being the favoured approach (Umphred & Lazaro, 2012).

1.3.1. Vestibular and Balance Rehabilitation Therapy: The 'Gold Standard'

The most effective chronic rehabilitation tool for PPPD is Vestibular and Balance Rehabilitation Therapy (VBRT; Cooksey, 1946; Cawthorne, 1946; Hannsson, 2007; Meldrum & McConn-Walsh, 2018; Umphred & Lazaro, 2012). VBRT is a specialised form of physical therapy that works to rehabilitate primary and secondary conditions caused by vestibular disorders. The regime aims to alleviate vertigo and dizziness, gaze instability and promote postural control. This is done by actively encouraging the patient to produce vestibular compensation and efficient sensory-conflict resolution by placing the person into a state of sensory conflict where their central nervous system must resolve conflict in order to recalibrate the weighting of the senses (Pavlou, 2010; Pavlou et al., 2011; Pavlou, Davies & Brinstein, 2006; Shepard, Telion, Smith-Wheelock & Raj, 1993; Shumway-Cook & Horak, 1990; Umphred & Lazaro, 2012; Whitney et al., 2006). For the PPPD patient, this involves placing the patient into a state of visual-vestibular conflict in order to improve their conflict resolution capabilities whilst working to desensitise the patient to visual triggers of symptoms (Pavlou et al., 2011; Pavlou, Davies & Bronstein, 2006). VBRT therefore works to return the senses to a state of equilibrium by promoting compensation and habituation.

Rehabilitation programs should always be tailored to the specific patient with graded exposure to triggers. Given this, clinical rehabilitation programs may differ greatly among patients with the same condition (Whitney, Wrisley, Marchetti Furman, 2002). Typical clinical regimes last approximately 8 weeks; however, they may be extended to up to 16 weeks and this formal program should be complimented with at home exercises that may continue after completion of the clinical program (see Umphred & Lazaro, 2012). As PPPD is triggered by visual motion, rehabilitation often requires exposure to visual motion patterns (optokinetic stimulation: Pavlou, 2010). To date, it is unknown if

desensitisation techniques work by producing an immediate short-term reduction in visual dependence. As rehabilitation uses graded exposure each session may produce some significant effect on the observers underlying visual dependence or smaller changes to sensory organisation may amalgamate over time. Van Ombergen et al. (2016) tested if optokinetic stimulation can produce an immediate effect on commonly used laboratory measures of visual dependence (Van Ombergen et al., 2016). Findings suggests that exposure to short-term optokinetic stimulation can change the postural but not perceptual marker of visual dependence. The researchers report that in clinical participants postural sway is increased immediately after exposure to optokinetic stimulation. This may be evidence of a transient after after-effect from exposure but does not help understand if the postural marker of visual dependence may decrease after such a transient effect dissipates. In order to better understand this in Chapter 3 reports research conducted to explore the short-term effects of optokinetic stimulation on markers of visual dependence. This was done to establish if visual desensitisation techniques can produce short-term changes in visual dependence and over what timeframes.

1.3.2. Visual Desensitisation Therapy: Using Optokinetic Stimulation to Rehabilitate Persistent Postural-Perceptual Dizziness (PPPD)

Exposing the PPPD patient to optokinetic stimuli is an effective way to desensitise the patient to visual triggers of symptoms (Bronstein, Lemptert & Seemungal, 2010; Bronstein, 2004; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). Pavlou, Lingeswaran, Davies, Gresty and Bronstein (2004) compared VBRT in PPPD patients with and without optokinetic stimulation. Forty chronic peripheral vestibular patients were randomly allocated to one of the two groups and given VBRT twice a week for eight weeks – supported by at home exercises. Postural sway and subjective symptoms were measured at four weeks and eight weeks. Upon completion of the programme, posturography scores significantly improved for both groups; however, the largest improvement was evident in the visual desensitisation condition. Note, no subjective visual vertical measures were employed. Visual vertigo symptoms (as measured by the older Visual Vertigo Symptoms Scale; Yardley, Masson, Verschuur, Haacke & Luxon, 1992) were shown to reduce in the desensitisation group only.

Visual desensitisation techniques are effective management and rehabilitation tools for PPPD (Whitney et al., 2006; Pavlou et al., 2011; Popkirov, Stone & Holle-Lee, 2018; Thompson, Goetting, Staab & Shepard, 2015). Pavlou et al. (2012) recruited 16 PPPD patients and randomly allocated participants to one of three visual exposure groups: a static visual world, a dynamic visual world or a combination of static and visual environments. Participants were placed in a large immersive theatre where visual stimulation was administered. In the two conditions that contained visual motion cues

(incongruent sensory signals) abnormal posturography scores were shown to reduce. The largest reduction in physical sway was visible in the dynamic stimulation group. Pavlou Bronstein and Davies (2013) suggest effects are evident when optokinetic stimulation is administered at home using a passive DVD video. Sixty chronic peripheral vestibular patients were recruited and randomly allocated to one of three treatment groups: typical optokinetic stimulation using a full visual field rotator, or exposure to an at home visual motion DVD, either supervised or unsupervised. No significant difference between group outcomes was visible at eight weeks post intervention, however all groups displayed within-group reductions in vestibular and PPPD symptoms. Posturography and functional gait scores were shown to improve for both supervised stimulation groups but findings were not present in the unsupervised group. Attrition was 40% higher when treatment was unsupervised. Finally, patients who undertook supervised visual desensitisation therapy (in the form of a DVD) also showed decreased levels of anxiety. This suggests that at home novel methods of visual desensitisation therapy coupled with supervision and support may be key for holistic rehabilitation of PPPD. Visual desensitisation using optokinetic stimulation, administered at home or in the clinic, are therefore effective tools to support in the rehabilitation the PPPD patient.

1.3.3. The Nature of the Optokinetic Stimulation

The quality and content of optokinetic stimuli used to introduce visuo-vestibular conflict in PPPD patients differ greatly across clinics and laboratories. Original paradigms have included spinning striped umbrellas (Thompson, Goetting, Staab & Shepard, 2015), rolling striped visual drums (Bos & Bles, 2004) and optic-flow videos with high spatial frequency features (Pavlou et al., 2011). Such froms of optokinetic stimulation may, however, become monotonous and may result in patient attrition or non-compliance. Thus, there is need for more engaging content. For example, Van Kerckhovan and Mert (2014) employed visual illusions as a form of optokinetic stimulation to produce visuo-vestibular conflict. In this pilot work, the researchers present a case study of an elderly patient with bilateral vestibular compromise, whom upon after receiving optokinetic based rehabilitation was shown to reduce in self-reported Dizziness Handicap Scores by 50% (16/100 reduced to 8/100) post programme. Thus, a diverse range of stimuli have been used to introduce visuo-vestibular conflict, but approaches are now favouring virtual environments and simulations.

Immersive virtual environments have been used to instigate visuo-vestibular conflict (Meldrum et al., 2012; Meldrum et al., 2015; Meldrum & Jahn, 2019; Popkriov, Stone & Holle-Lee, 2018). Commonly employed paradigms require the patient to be placed in front of a large screen and exposed to low-level visual features known to trigger symptoms (see Pavlou et al., 2011). This design has been adapted by enhancing the visual stimuli and exposing the patient to simulations of triggers such as the supermarket aisle and walking in crowded environments (Aharoni, Lubetzy, Wang, Goldman &

Krasvosky, 2019; Aharoni, Lubetzy, Arie & Krasovsky, 2021; Whitney et al., 2006). Other techniques have simulated situations such as navigating busy subways (Meldrum et al., 2012). These simulations aim to recalibrate balance control mechanisms whilst improving confidence at comfort with triggering scenarios.

Low quality virtual environments or basic computerised stripes are effective rehabilitation tools for PPPD. However, such forms of optokinetic stimulation may be criticised as boring or unengaging. Thus, there is a need to establish more immersive simulations to enhance the user experience and encourage patient adherence. Furthermore, more advanced simulations of motion may produce greater effects visual dependence if the low-level features are more pronounced. Potential avenues for visual desensitisation therapy may be virtual video games or engaging movies. Playing a virtual video game or viewing an engaging movie, high in visual motion, may allow for a more immersive and enjoyable form of rehabilitation. There is research to suggest that engaging in virtual video game play can enhance sensorimotor skills, perception and psychological state (Green & Baelier, 2012; Lee & Peng, 2006; Li, Chen & Chen, 2016); Rosser et al., 2007). Furthermore, experimental data in the Multiple Sclerosis patient group has shown promising results on balance and postural control when interactive games are employed (Ortiz-Gutiérrez et al., 2013). This suggests there is potential for virtual video game-based optokinetic stimulation to enhance postural control mechanisms and balance and reduce visual dependence. Virtual video games and/or engaging movies may therefore be promising rehabilitation tools with PPPD patients – a hypothesis currently untested.

To date, no research has explored the potential for virtual video games or engaging movies to be used as rehabilitation tools PPPD. A plausible explanation for this gap in the literature may be due to concerns that actively playing a virtual video game would require the user to actively control and interact with the optokinetic properties of the stimulus. To date, it is not known if this real-time interactivity affects the efficacy of a given visual motion stimulus.

The aim of Chapter 3 was to address this gap in the literature by exploring the potential from multi-media stimuli to be used as rehabilitation tools. The researcher experimentally tested a range of engaging multi-media tools that could be used as optokinetic stimulation that were active and passive in nature. This was done in order to establish which form of visual motion may be the most promising rehabilitation tool (and the time frames effects occur over). Studies were conducted with a non-clinical sample to test different rehabilitation approaches and stimuli without wasting patient time. Originally, this was intended to be foundational research before testing the most efficacious tool with the PPPD community. Due to the constraints of Covid-19 this could not be done.

Section 2 of Chapter 1 aimed to discuss how PPPD can be rehabilitated and has provided background literature relevant for the experiments presented in Chapter 3. This section has introduced Vestibular and Balance Rehabilitation Therapy, Visual Desensitisation approaches for the rehabilitation of PPPD and considered current optokinetic stimuli used as rehabilitation tools. This section highlighted that rehabilitation tools are often simplistic and unengaging. This section provides the reader with key information relevant to Chapter 3 where the researcher aimed to explore the short-term effects of novel multi-media based optokinetic stimulation on markers of visual dependence.

1.4. Section 3: Conditions and Factors Associated with Persistent Postural-Perceptual Dizziness

Section 3 of Chapter 1 aims to consider conditions and factors associated with PPPD. It will act as background information relevant to empirical Chapter 2, and 4 and supplementary research presented in Appendix S2.1. This section of the Chapter therefore explores how PPPD is related to age, anxiety, women, migraine, stress and Cyber Sickness.

1.4.1. Age and Persistent Postural-Perceptual Dizziness

PPPD is most commonly reported in the middle aged (Dietrerich, Staab & Brandt, 2016; Neuhauser, 2016; Strupp et al., 2003). Presumably, this is because as humans age the senses (in particular the vestibular system) degrade and become more unreliable forcing them to become more naturally dependent on vision for postural control (Schwartz, Rohe, Eggers & Shephard, 2014). As such, visual dependence increases with age (Agathos et al., 2015). Age-related vestibular degradation explains why sensations of dizziness are more likely to occur as humans age and thus are more apparent in the middle aged and are even more likely in elderly populations (Iwasaki & Yamasoba, 2015; Jahn, Kressig, Bridenaugh, Brandt & Schniepp, 2015; Katsarkas, 1994; Strupp et al., 2013; Yardley, Owen, Nazareth and Luxon, 1998). However, this explains vestibular symptoms but not necessarily PPPD symptoms. As PPPD is more commonly reported in the middle aged, if vestibular degradation fully explained PPPD then visually induced dizziness symptoms should be more prevalent in aging communities which is not the case.

Counter-intuitively, PPPD symptoms correlate negatively with age (Powell, Derry-Sumner, Rajenderkumar, Rushton & Sumner, 2020). This was found when using the generally healthy adult population as a sample, with PPPD symptoms assessed using the Situational Characteristics Questionnaire (SCQ: Jacob et al., 1993; Jacob, Lilienfeld, Furman, Durrant & Turner, 1989) and the

shorter Visual Vertigo Analogue Scale (VVAS: Dannenbaum, Chilingaryan & Fung, 2011). This finding is in contrast with what may expected from the literature and suggests that the relationship between PPPD and age is not fully understood. Thus, although age may increase visual dependence it does not appear to correlate with PPPD symptoms.

1.4.2. Anxiety and Persistent Postural-Perceptual Dizziness

Anxiety has long been established as a risk factor and/or consequence of developing PPPD. Potentially due to the interrelated threat assessment mechanisms that facilitate dizziness and anxiety (Trindade & Goebel, 2018; Van Ombergen et al., 2016). Both conditions have also been associated with hypervigilance and a heightened awareness of the self in physical space (Morisod, Mermod & Maire, 2018; Bronstein, 1995). Furthermore, this underlying anxious predisposition feeds the PPPD disorder by heightening the fear of triggers, symptoms and consequential falling and triggering envrionments (such as the supermarket; McCabe, 1975). This encourages and validates agoraphobic tendencies and produces a vicious cycle between anxiety and PPPD (Holmberg, Karlberg, Haralacher & Magusson, 2005; Yu, Xue, Zhang & Zhou, 2018).

Fifteen percent of PPPD patients' pre-existing illness is psychogenic in nature and the result of anxiety or panic attacks (Staab et al., 2017). This may suggest that PPPD is not a solely sensory condition and must encapsulate some psychogenic elements. This idea is supported by the condition's association with anxiety (Cordes, 1872; Westphal, 1871; Powell et al., 2020). Evidence in support of this claim is presented in Chapter 5.

1.4.3. Women and Persistent Postural-Perceptual Dizziness

PPPD predominantly affects females; for every one male diagnosed with PPPD two females are diagnosed (Dietrerich, Staab & Brandt, 2016; Strupp et al., 2003). This may suggest gender-based health inequalities when it comes to the PPPD condition. However, it is currently not known if this is due to the increased likelihood of anxiety in the female gender (Bander & Betz, 1981; Hong & Karstensson, 2002; Lewinsohn, Gotlib, Lewinsohn, Seely & Allen, 1998), increased reporting of symptoms in females (Nam et al., 2010) or due to psychological gender differences (Dias, Cruz & Fonseca, 2010) or biological sex differences (Bangasser & Curarenta, 2021; Bekker & van Mens-Verhulst, 2007; Jalnaurkar, Allen & Pigott, 2018). This raises interesting questions around historically marginalised communities and PPPD, such as the prevalence of PPPD symptoms in the Trans and Non-Binary communities where traditional ideas of gender and sex do not reflect authentic identities. Conceptually, psychologists differentiate gender as the social construct an individual, does or does not, identify with and sex and the chromosomal make up of an individual (Lips, 2020; Johnson &

Repta, 2012; Moore, 2002). A better understanding of the relationships between PPPD, the female gender and the female sex would help the field understand if the disorder is associated with the female sex or the female gender. This may have important bio-psycho-social implications for health and health disparity and better account for the prevalence of PPPD in females. This information is given as context but was beyond the scope of the Thesis.

1.4.4. Migraine and Persistent Postural-Perceptual Dizziness

PPPD patients often report migraines (Staab & Ruckenstein, 2007; Popkirov, Staab & Stone, 2018). Twenty-six percent of PPPD patients report migraines (Bittar and Lins, 2015). The same data set also stated that PPPD is often co-morbid with a range of other diseases and conditions such as hypercholesterolemia (31%), migraine (26%), carbohydrate metabolic disorder (22%), cervical syndrome (21%), benign paroxysmal positional vertigo (15%) dysautonomia (7%), disorders of the middle ear (7%), hypertension (5%), cardiac arrhythmia (5%), diabetes (5%), hypothyroidism (5%), and Menière's syndrome (5%). Interestingly, migraine (post puberty) also affects women more than men at a ratio of 3:1 (Raña-Martinez, 2008). An initial precipitating vestibular migraine accounts for 15% of PPPD patients and often co-occurs with PPPD and this finding also translates to the sister syndrome Mal de Debarquement syndrome (Bisdorff, von Brevern, Lempert & Newman-Toker, 2009; Cha, 2009; Staab et al., 2017). Little is known about the origin and manifestation of vestibular and non-vestibular migraines. Less is known about the intersection of patients who report migraine and PPPD. Regardless, their co-occurring relationship is well document (see Staab & Ruckenstein) and data presented in Chapter 5 also support this association.

1.4.5. Stress and Persistent Postural-Perceptual Dizziness

Stressful life circumstances have also been known to precipitate both organic and non-organic vestibular disorders, including PPPD (Dieterich & Staab, 2016). Few empirical investigations have been employed to directly explore the relationship between PPPD and stress as studies have favoured anxiety. Data from Radjziej, Schmid, Dinkel, Zwergal and Lahmann (2015) suggest that adverse life experiences childhood and/or adulthood are equally prevalent in structural and functional dizziness. This suggests that people with adverse life experiences are more at risk from experiencing both structural and functional dizziness, of which PPPD is the latter. There may, therefore, be health inequalities in the prevalence of dizziness. For example, a recent qualitative report has documented dizzy symptoms in case studies of the LGBTQ+ community (Mustamam, Rajathurai, Gusdian & Khan, 2020). Generally, this community are more likely to experience adverse life experiences due to their sexual orientation, including bullying, social isolation and marginalisation, physical violence, health inequalities and mental health disorders (Stonewall, 2017; Stonewall, 2018). The authors

concluded that stress from adverse life experiences may affect dizziness and as these may and thus those more at risk of such stress may facilitate the manifestation of dizzy like symptoms (Mustamam et al.*)*. It is important to note that it is not clear if this dizziness is general in nature or visually induced. Regardless, stressful life circumstances are associated with higher levels of dizziness, including PPPD, and there may therefore be health inequalities for high stressed individuals and minority groups at greater risk of stress from society and social interactions with the majority.

Note, it is unknown how visual dependence and stress are related but what is clear is that stress may be a risk factor for developing dizziness, and in particular PPPD with some patients reporting no pre-existing vestibular insult but extremely high levels of self-reported stress (Staab et al., 2017). However, currently it is not known how experiences of PPPD and psycho-social factors interact with one and other. These relationships were explored relationships between PPPD and psycho-social factors in Chapter 2 in the student population of a Russell Group University – a naturally stressed group due to their academic commitments.

1.4.6. Cyber Sickness & Persistent Postural-Perceptual Dizziness

Characteristic symptoms of PPPD over-lap with those of Cyber Sickness. Cyber Sickness is defined as a unique form of motion sickness that results from the stationary observer being exposed to virtual visual motion patterns that produces a sensation of self-motion and/or vection (LaViola, 2000). From this definition, Cyber Sickness is conceptualised as a similar condition to PPPD, where triggers are simulated motion. As such, some researchers use the term 'Simulator Sickness' and measure the construct using the Simulator Sickness Questionnaire (Kennedy, Lane, Berbaum & Lilenthal, 1993; see Appendix 1.2); however, the appropriateness of this is heavily debated within the literature (see Stanney & Kennedy, 1997). Given the similarities between the conditions, it has been posited that Cyber Sickness is not a unique form of visually induced dizziness, rather a general visually induced dizziness from motion patterns that are virtual in their nature (Mazloumi, Walker, Hodgson & Nalivaiko, 2018). This may suggest that the condition is the same as PPPD, simply bound to the virtual world. Cyber Sickness does, however, involve factors such as eyestrain due to convergence, accommodation cues, fixed focal demands, and, where relevant, the 'fit' of head mounted displays. These do not appear to have obvious equivalents in PPPD. However, this does not mean that the two conditions are unrelated and there may be some shared factors that explain both conditions. To date, no research has looked to explore the relationship between PPPD and Cyber Sickness and if the two conditions have shared factors that can explain symptom manifestation. This was explored as supplementary research (see Appendix 2.1) in order to better support the investigations of Chapter 2.

The third section of Chapter 1 aimed to discuss conditions and factors associated with PPPD. This included a consideration of age, anxiety, women, migraine, stress and Cyber Sickness. This section has provided the reader with background literature related to Chapter 2 where the lived experiences of PPPD were explored. It also provides information relevant to the supplementary research presented in Appendix 2.1 where the researcher aimed to explore the relationship between PPPD symptoms and Cyber Sickness symptoms.

1.5. Section 4: The Lived Experiences of Persistent Postural-Perceptual Dizziness

The fourth section of Chapter 1 aims to present the reader with literature of the lived experiences of PPPD. It will act as background information relevant for the mixed-methods research presented in Chapter 2.

1.5.1. Qualitative Methods are Neglected in Persistent Postural-Perceptual Dizziness Research

The lived experiences of those with PPPD are not well understood and presents a research area that has been neglected to date. This may be due to the fact that lived experiences are often probed by qualitative research methods; research often neglected in PPPD research (for an introduction to qualitative research methods see Braun & Clarke, 2013). PPPD can be assessed using qualitative research methods and clinical assessments of patients are fundamentally qualitative. Qualitative research paradigms have been successfully employed to assess the lived experiences of PPPD (Bigelow, Semenov, du Lac, Hoffman & Agarwal, 2016; Teleaven, Peterson, Ludigsson, Kammerlind & Peolsson, 2016). These studies have used a range of qualitative techniques including thematic analysis, content analysis and grounded theory (Mendel, Lutzen, Bergenius and Bjorvell, 1997; Tinetti, Williams & Gill, 2000; Herdman, Evetovits, Everton & Murdin, 2020); however, these investigations are rare and not all possible qualitative methods have been employed with the PPPD group.

1.5.2. Qualitative Findings in Dizzy Populations

Qualitative enquires have established that chronic dizziness can produce a confused state in the sufferer where they are unable to make sense of their symptoms or illness (Bigelow, Semenov, du Lac, Hoffman & Agarwal, 2016; Teleaven, Peterson, Ludigsson, Kammerlind & Peolsson, 2016). For people with the specific diagnosis of PPPD, qualitative data suggests they may show vulnerable

reactions to physical and psychological situations, require affirmation and constantly seek new ways to cope and continue with their lives – usually via healthcare services (Mendel, Lutzen, Bergenius & Bjorvell, 1997). The cohort also tend to avoid disclosing their health compromised status but are often forced to do so when symptoms ultimately persist, and they realise they need bio-psycho-social support or intervention (Yardley & Beech, 1998). Qualitative research has also found that PPPD symptoms result in people questioning their own sanity, especially when medical professionals misdiagnose their symptomatic presentation or do not make the patient feel that their experiences are valid or 'real' (Sezier, 2016; Sezier, Saywell, Terry, Taylor & Kayes, 2019). These accounts suggest that the persistence of symptoms not only results in a loss of the self but fears that they cannot navigate life with PPPD. Unfortunately, these few studies are the only documented accounts of the lived experiences of PPPD in the published literature, to date. This shows a general neglect of qualitative research methods within PPPD research and highlights the lack of knowledge and understanding of the lived experiences of the disorder and how PPPD affects patients across all domains of life.

1.5.3. Interpretative Phenomenological Analysis and Persistent Postural-Perceptual Dizziness

No studies have adopted the hermeneutic [qualitative] approach of Interpretative Phenomenological Analysis (IPA). IPA is an experiential qualitative research method that generates rich contextual data based on lived experiences. Questions are formed around a narrative of the patient and encourages disclosure of experiences through candid story telling supported by a compassionate qualitative researcher (Smith, Flowers & Larkin, 2009). IPA has been successful in capturing the lived experiences of numerous health compromising illnesses that are hard to articulate such as Anorexia, Multiple Sclerosis, Fibromyalgia and HIV (Ashe, Furness, Taylor, Haywood-Small & Lawson, 2011; Flowers, Davis, Larkin, Church & Marriott, 2011; Fox & Diab, 2015; Strickland, Worth & Kennedy, 2015). IPA may therefore be a promising tool for exploring the lived experiences of PPPD. Furthermore, as the approach promotes a commitment to ideography applying this tool to the PPPD patient groups allows us to generate deeply rich personalised accounts of lived experiences of PPPD and help them articulate their experiences in a supportive manner. The tool may also help validate patient experiences by actively listening to their accounts in a non-judgemental way free from time constraints.

In Chapter 2, the researcher aimed to better explore the lived experiences of PPPD. In particular, the research was designed to probe the psycho-social impacts of the condition on all domains of life. As the qualitative research method, IPA, had yet to be applied to this patient group, this method was adopted to encourage candid disclosure of hard to articulate experiences.

The fourth and final section of the General Introduction aimed present the reader with an introduction on literature on the lived experiences of PPPD. Although studies exploring the lived experiences are sparse this section explored the issue of the neglect of these research methods, qualitative findings in dizzy populations. This section also provided the reader with background information relevant to the empirical investigations of Chapter 2.

1.6. Summary and Thesis Research Questions

PPPD is a functional (non-organic), debilitating, neuro-vestibular disorder characterised by chronic episodes of dizziness, disequilibrium, non-spinning vertigo and postural instability (Bronstein, 1995; Deitrich & Staab, 2017; Seemungal & Passamonti, 2018; Staab et al., 2017; Wurthman et al., 2017). Symptoms are triggered by situations of visuo-vestibular conflict, intense visual-environments and active and passive motion (Powell, Derry-Sumner, Rajenderkumar, Rushton & Sumner, 2020; Trinidade & Goebel, 2018). Commonly reported triggers include cluttered supermarket aisles and busy moving traffic (McCabe,1975; Söheston, Bittar & Staab, 2016; Staab et al., 2017). The aim of this Thesis was to explore factors associated with PPPD.

This General Introduction aimed to introduce the Persistent Postural-Perceptual Dizziness (PPPD) condition, disseminate the leading theory of PPPD manifestation and present commonly used laboratory measures for indicating the presence of symptoms. The introduction then presented three unique strands of literature which related to the specific empirical Chapters presented in the wider Thesis. First, the Chapter considered how PPPD can be rehabilitated. This reflects background literature for the experiments presented in Chapter 2. Second, conditions associated with PPPD were considered. This should prepare the reader for the investigations presented in Chapter 3 and 4. Third and final, the lived experiences of PPPD were documented. This will prepare the reader for the empirical studies that are included in Chapter 5. Having introduced these key areas in relation to PPPD research, the main aims and research questions for each empirical Chapter will now be stated.

1.6.1. Chapter 2: Aims and Research Questions

PPPD patients often present with secondary, or potentially precipitating, psychiatric illnesses which result in a debilitated patient with complex co-occurring illnesses that diminishes quality of life (Zur et al., 2015) – though the lived experiences of people with PPPD are seldom probed (Guerraz et al., 2001; Staab, Eckhardt-Henn, Horri, Jacob & Stupp, 2014; Zur et al., 2015). The aim of Chapter 2 was to explore how individuals with PPPD make sense of their symptoms and condition and to better

understand the lived experiences of PPPD, including the psycho-social impacts of the condition. A supplementary aim of the research was to deepen the scientific community's conceptual understanding of the condition, improve sensitivity to context (Yardley, 2008), and stimulate further qualitative research. Thus, the objective of Chapter 2 was to directly access PPPD patients' voices in order to probe the lived experiences of PPPD using the IPA method. The second objective was to explore both ideographic and across-participant accounts of the condition. The third objective was to quantitively explore the relationships between any emergent themes and PPPD symptoms in a large non-clinical sample.

1.6.2. Chapter 3: Aims and Research Questions

VBRT is the preferred rehabilitation route for those with PPPD, though for best results rehabilitation should involve optokinetic visual desensitisation techniques, which to date are somewhat mundane and monotonous (Bronstein, 2004; Bronstein, Lemptert & Seemungal, 2010; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). Optokinetic stimulation techniques allow the patient to habituate to retinal cues for self-motion, which, over time, allows the individual to desensitise to the [visual] triggers of their symptoms and research in clinical populations has shown that brief exposure to an optokinetic stimulus can produce measurable changes in the postural, but not perceptual, marker of visual dependence (Van Ombergen et al., 2016). The aim of Chapter 3 was test if short-term exposure to optokinetic stimulation can produce changes in markers of visual dependence. The research also aimed to explore the short-term timeframes effects occur on. Finally, as current optokinetic stimuli used in the rehabilitation of PPPD may be considered monotonous and unengaging, (see stimuli presented in: Meldrum et al., 2012; Whitney et al., 2006) experiments were designed in a manner that would establish if multi-media technology could be used to produce recalibration effects and establish the most effective stimulus for doing so. Thus, a supplementary aim was to establish if engaging multi-media technology can be used to facilitate optokinetic stimulation. The objectives of this Chapter were to establish if short-term exposure, in the form of different engaging multi-media stimuli, can produce a short-term reduction in traditional laboratory measures of visual dependence.

1.6.3. Chapter 4: Aims and Research Questions

Visual dependence is the leading theory of PPPD manifestation (Bronstein, 1995; Bronstein, 2004; Pavlou et al., 2010; Staab et al., 2017). Thus, one may hypothesise that performance on the two commonly used measures of visual dependence (Rod and Frame test and postural sway – Vection Quotient) would correlate with PPPD symptoms. Furthermore, as these measures are assumed to measure the same entity one would expect performance on these measures to also correlate - though

research has suggested this is not always the case (see Guerraz et al., 2001). The aim of Chapter 4 was to explore the relationships between self-reported PPPD scores (as measured by the Visual Vertigo Analogue Scale: Dannenbaum, Chillingryan & Fung, 2011) with scores on the two commonly used laboratory measures of visual dependence. The objectives of this Chapter were to explore these relationships in the heathy student and sub-clinical student groups.

Chapter 2: Using Interpretative Phenomenological Analysis to Probe the Lived Experiences of Persistent Postural-Perceptual Dizziness

2.1. Introduction

PPPD can be a disabling experience resulting in functional impairments such as difficulties in walking, navigating three-dimensional space, maintaining postural control, or controlling active and passive motion (Dierterich & Staab, 2017; Olsson Möller, 2014). These functional impairments are often so physically restricting that they satisfy the diagnostic criteria for a medically recognised disability (Mueller, Schuster, Strobl & Grill, 2012). These individuals do however fight to gain control of the condition and the disabling status it produces (Olsson Möller, 2014). PPPD can then result in behavioural adaptations in order to avoid triggering symptoms, such as developing an abnormal [stiffened] gait or posture and becoming hypervigilant to self-motion (Jahn, Kressig, Bridenbaugh, Brandt & Schniepp, 2015; Mueller, Schuster, Strobl & Grill, 2012).

Limited previous research has explored the lived experiences of PPPD. Qualitative data suggest chronic dizziness can produce a confused state where individuals are unable to make sense of their symptoms or illness (Bigelow, Semenov, du Lac, Hoffman & Agarwal, 2016; Teleaven, Peterson, Ludigsson, Kammerlind & Peolsson, 2016). Patients also show misconceptions surround the aetiology of their dizziness and have a poor understanding of potential treatment avenues but develop their own 'work arounds' to avoid a loss of mobility and independence (Kruschinski, Theile, Drier & Hummers-Pradier, 2010). In the case of PPPD, the diagnostic label itself may have negative psycho-social implications as the name and acronym of the condition may be considered conceptually confusing, without medical merit, or a label given to psychosomatic experience (Hedman, Evetovits, Everton & Murdin, 2020; Seizier, Saywell, Terry, Taylor & Kayes, 2019). In contrast, being able to make sense of symptoms through diagnostic labels can allow for patients to better articulate health compromising illnesses which, in turn, can result in more positive health outcomes (Beach, Keruly & Moore, 2006; Stewart et al., 2000).

Despite being one of the most common causes of chronic dizziness, very little is known about the lived experience of people with PPPD. It is important to understand the diverse range of experiences of this condition and how it affects individuals across the different domains of their lives. This is useful for researchers as it could potentially spark new avenues for investigation, and useful for

clinicians who often have to identify PPPD within complex sets of symptom presentation. Previous research has found patients typically become less confident in themselves and in conducting daily activities, and this ultimately results in a subjective loss of self where they struggle to navigate living with PPPD (see Seizer et al., 2019). Living with chronic dizziness has also been shown to encourage feelings of insecurity, exhaustion and a loss of dignity which can be reinforced if interactions with healthcare professionals are perceived as dismissive (Mendel, Lützén, Bergenius & Björvell, 1997).

Previous qualitative research in this area have used traditional qualitative research methods such as thematic analysis, content analysis and grounded theory (Mendel, Lutzen, Bergenius and Bjorvell, 1997; Tinetti, Williams & Gill, 2000; Herdman, Evetovits, Everton & Murdin, 2020). However, no research probing the lived experiences of PPPD have adopted the hermeneutic approach of IPA to probe lived experiences of PPPD. IPA is an experiential qualitative research method that generates rich contextual data based on lived experiences (see Smith, Flowers & Larkin, 2009). The method has been successful in helping patients articulate difficult health compromising illnesses (Ashe, Furness, Taylor, Haywood-Small & Lawson, 2011; Flowers, Davis, Larkin, Church & Marriott, 2011; Fox & Diab, 2015; Strickland, Worth & Kennedy, 2015) and may therefore be a promising tool for probing the lived experiences of PPPD.

2.1.1. Aims and Objectives of Chapter 2

The aim of Chapter 2 was to explore how individuals with PPPD make sense of their symptoms and condition and to better understand the lived experiences of PPPD, including the psycho-social impacts of the condition. A supplementary aim of the research was to deepen the scientific community's conceptual understanding of the condition, improve sensitivity to context (Yardley, 2008), and stimulate further qualitative research. Thus, the objective of Chapter 2 was to directly access PPPD patients' voices in order to probe the lived experiences of PPPD using the IPA method. The second objective was to explore both ideographic and across-participant accounts of the condition. The third objective was to quantitively explore the relationships between any emergent themes and PPPD symptoms in a large non-clinical sample.

2.2. Study 1: Using Interpretative Phenomenological Analysis to Explore the Psycho-Social Impacts of Persistent Postural-Perceptual Dizziness: An Idiographic Approach

The aim of this study was to explore how individuals with PPPD make sense of their symptoms and condition and to better understand the lived experiences of PPPD, including the psycho-social impacts of the condition.

2.2.1. Study 1: Method

2.2.1.1. Participants

Six participants (four females and two males), with an active diagnosis of PPPD, between the ages of 29 and 54, were recruited. Five participants were recruited through clinics (i.e. following an assessment with a clinical scientist who diagnosed PPPD). The patients were given a web link to access if they would like to be contacted to take part in research in this area. The remaining participant contacted me stating they had recently been given a diagnosis of PPPD and would like to be involved in patient-focused research projects. All participants currently live in the United Kingdom. One participant also had an active diagnosis of Fibromyalgia and one with Endometriosis. All participants are given a pseudo-name for anonymity.

This study was approved by the Cardiff University School of Psychology Ethics committee and NHS ethics panel.

2.2.1.2. Qualitative Research Training in Interpretative Phenomenological Analysis

The researcher contacted an expert in the IPA method to undertake training and knowledge exchange in relation to the IPA method, its epistemological stance, questionnaire design and effective phenomenological interactions. Furthermore, this training also included guidance on generating, analysis and interpreting the data produced by the IPA method. As part of this training, the researcher was required to critically appraise key papers in the field and conduct practice interviews and data analysis with the trainer. Furthermore, as part of the training one PPPD interview was conducted with the instructor in order to gain confidence in the method and witness an expert utilise the method in practice.

2.2.1.3. Procedure

Data were collected using semi-structured interviews. Interviews lasted between 1 and 2 hours. Two interviews were conducted online (with video) whilst the remaining four were conducted using the telephone, depending on the participant's preference. The semi-structured interview schedule can be found in Appendix 2.2 Participants were asked questions when the topics naturally arose. All questions related to lived experiences of PPPD. All interviews were audio-recorded and transcribed verbatim.

2.2.1.4. Developing the Semi-Structured Interview Guide

2.2.1.4.1. Supplementary Research to Inform Interview Questions and Topics

Supplementary research exploring experiences and symptoms associated with PPPD were explored as supplementary research to better inform question design. This supplementary work (Chapter) can be found Appendix 2.1. Supplementary findings suggested that two clusters of symptoms – those that manifest in the head and represent confusion and those that manifest in the body representing 'sickly' vertiginous symptoms. This understanding helped the researcher probe the different types of experiences that PPPD may produce.

2.2.1.4.2. Semi-Structure Interview Guide Development

The interview guide was developed by the researcher in conjunction with an expert in IPA methodology and practice (Professor Kevin Wilson-Smith: see Wilson-Smith & Bates, 2014; Wesig & Wilson-Smith, 2021). Each item was designed to elicit a narrative-based response that focused on experience. Draft versions of the interview guide were passed to the expert and feedback was integrated based on his expertise.

The interview guide was iteratively developed during data collection. After each participant was interviewed, the guide was updated to reflect areas of focus or to adjust or remove items. Any unforeseen areas for exploration were added to the guide if they arose from a participants narrative account of PPPD.

2.2.1.5. Piloting Interpretative Phenomenological Analysis

The research paradigm and semi-structured interview guide was piloted with a PPPD patient. This participant contacted the researcher raising their interest in taking part in PPPD research. The pilot session was conducted with an experienced IPA researcher and the participant was informed that this

was the researchers first IPA interview and that the session was being ran with an experienced researcher so that adequate training and skill acquisition could be ascertained. This pilot session was led by the experienced IPA researcher with the support of the researcher so that style, tone and probing could be observed and then practiced. The data created during this session was used as part of the analysis (see participant 1 Lara).

2.2.1.6. Analytic Strategy

All six transcripts were analysed using Interpretative Phenomenological Analysis (IPA: Smith, 1996). IPA is a qualitative research method which offers a systematic approach to analysing qualitative data by exploring an individual's personal journey (Riggs & Coyle, 2002; Smith, Flowers & Osborn,1997, Smith, Jarman & Osborn, 1999). The method aims to establish how the participant makes sense of their world and its events and obtain information regarding their cognitions, emotions and how they understand events in their internal and external worlds (Riggs & Coyle, 2002). IPA is epistemologically congruent with the intent of exploring the lived experience of those living with complicated health conditions and is used often in the field of health psychology (Ashe, Furness, Taylor, Haywood-Small & Lawson, 2011; Flowers, Davis, Larkin, Church & Marriott, 2011; Fox & Diab, 2015; Strickland, Worth & Kennedy, 2015). In addition, its idiographic nature was deemed ideal for use with patients who report being dismissed or not listened to, as the method fully invests in their unique story in a supportive non-judgemental manner (Seizier, Saywell, Terry, Taylor & Kayes, 2019).

The analysis followed Smith, Flower and Larkins (2009) guidelines and involved iterative reading of transcripts and listening to the audio files. Transcripts were annotated to highlight thoughts, ideas and key phases, and these annotations were built into notes for each transcript, which were then refined and condensed to represent superordinate and subordinate themes (Riggs & Coyles, 2002). Each quote presented in the analysis is referenced by indicating the pseudo-name of participant and the line number of the quote in the transcript.

2.2.1.7. Ensuring Rigour in the Qualitative Data Analysis

All data generated were iteratively coded by the researcher. The researcher immersed himself in the data by iteratively reading transcripts of the interview and repeated listening to the audio recordings of the interview. Each line of each transcript was annotated with notes and these notes were condensed to codes representing initial themes in the data and any thoughts the research had in relation to these. The transcripts, notes and codes were then used as the core data for the IPA data analysis process.

A second researcher (an expert in the field), simultaneously, analysed the data. Both researchers then met virtually upon completion of the initial analysis of each participant's account. The researchers discussed emerging themes from the explained their reasoning for interpreting each theme. Themes had to be identified by both researchers for inclusion in the analysis. Researchers did not disagree on any emerging themes.

The transcripts, notes and themes were read by a clinical scientist familiar with co-occurring conditions, to examine whether experiences and symptoms ascribed to PPPD were not more clearly associated with one of the co-occurring conditions.

2.2.1.8. Post Interview Debriefing and Self-Reflection

Upon completion of each interview the researcher met with the IPA expert to reflect on the conversation and discuss any successes, challenges or barriers to candour. These reflection sessions were used to reflect on practice in order to avoid or develop strategies to encourage or avoid specific scenarios or interactions in the future, Furthermore, as some accounts of the lived experiences of PPPD are personal, moving and emotional these sessions were also used to debrief and articulate any emotions the researcher may be experiencing and how to manage navigating empirically charged sessions professionally and empathetically. Debrief sessions typically lasted 1 hour. One session last 2 hours due to the moving nature of the account (see Participant 6 – Sian). This is considered good qualitative research practice (McMahon & Winch, 2018).

2.3. Study 1: Results - Case by Case Interpretative Phenomenological Analysis

2.3.1. Participant 1: Lara

Lara is a 29-year-old woman who has completed an undergraduate degree, a master's degree and three years of PhD study and now works in agriculture. Lara has been experiencing PPPD for 2 years. Prior to this she would describe herself as a 'Jolly. Energetic. And not in a conceited way but like Superwoman' (**Lara: 597– 598).**

2.3.1.1. Superordinate Theme 1.1: Identity Crisis

Lara stated that living with PPPD has impaired her life so much so that she no longer recognises who she is:

> ‘I felt like I lost my identity. Completely. I was known as the person that who did stuff all the time, I was so active, I was really jolly all the time and like really energetic and I lost all of those things. And I had a massive identity crisis. I was like “I don’t even know who I am if I can’t do all this stuff and if I’m just really sad all the time and I don’t adventure”’. (**Lara: 585-588**) ‘I was so fun! I could do all these things and now I’m not. And I’m sad. And I’m crap. And I can’t do my job. And my self-confidence and ability, as like, to do my job and be a friend and cool person like plummeted’ **(Lara: 666-668)**

The condition made her unable to take part in many activities that she considered crucial to her identity and ultimately left her feeling like her identity was stolen from her by the condition and replaced with vestibular and [negative] psychological symptoms.

2.3.1.2. Superordinate Theme 1.2: Sexism: Men’s Microaggressions in Cinical Assessments

Lara believes that negative, unempowering and ‘dismissive’ clinical interactions surround PPPD are, in part, due to her perceived gender and sex; that she is prejudged based on outdated and offensive associations of the female. She alludes to this stigma and how it resonates with negative connotations of anxiousness, neuroticism, attention-seeking and medical malingering which she believes affects her clinical treatment.

> ‘Like the doctors were [emphasised] so rude like some of them it was so bad it was because I’d been in a couple of times about something before and I think it was just like ‘oh she’s, it’s just this anxious woman just keeps coming in’. (**Lara: 246-249**)

She also states that this is not a ‘one off’ event but one that has prevailed, insidiously, throughout her journey to a diagnosis:

> ‘So, it was a doctor and even before I’d gone in (and I was upset) he’d already decided there was nothing wrong with me. You could tell. Like, you could tell. Middle-aged, middle-class man. Crying lady. I’ve seen it, because it happened at the doctors before’ (**Lara: 379- 382**)

Lara interprets the behaviour she has witnessed and experienced, from male medical professionals as ‘so [emphasis] dismissive and rude’ **(Lara: 401**) and that these interactions are microaggressions evident of covert sexist attitudes that affect clinical interactions.

2.3.2. Participant 2: Zaynab

Zaynab is a 43-year-old female Optometrist. Zaynab was diagnosed with endometriosis 14 years prior to the study and has undergone numerous operations in relation to the health compromising illness - 'about 10 operations in the last 10 years' (**Zaynab: 19-20**). PPPD symptoms developed approximately three-four years ago and after recovery from acute Benign Paroxysmal Positional Vertigo (BPPV). In addition, she is also a migraineur and is vitamin B12 deficient.

2.3.2.1. Superordinate Theme 2.1: Fear & Anxiety

This superordinate theme captures Zaynab's fear. Subordinate themes are the composites of fear she faces: (a) the fear of when *symptom* will present (b) the fear of the potential *consequences* of symptoms and (c) the fear that, because of PPPD, she will *fail to function* adequately.

2.3.2.1.1. Subordinate Theme 2.1a: Unknown Nature of Persistent Postural-Perceptual Dizziness

Zaynab discloses that she fears the unknown nature PPPD and when the next episode will present.

> 'I'd say dizziness made me anxious (…) The anxiety of is it going to happen? When is it going to happen? I've got no way of determining whether it's going to happen or not. It just randomly comes out of nowhere (…) If I knew the trigger factors at the time, then maybe I'd be a little bit more confident but because I didn't know why it was happening, I had no control over it' (**Zaynab: 444-446**)

Zaynab articulates her anxiety that surrounds not knowing where or when the episodes would strike. Interestingly, her account does not indicate that she fears the symptoms themselves but rather the unexpected nature of them. Her use of the past-tense also suggests that once she was informed by healthcare professionals how and why her symptoms persist that control is established, and fear diminishes.

2.3.2.1.2. Subordinate Theme 2.1b: Potential Consequences of an Episode

Symptoms may trigger at any place and/or time and this may cause catastrophic events. This is the cause of great anxiety and fear for Zaynab resulting in behavioural adaptations to avoid potentially calamitous scenarios.

> ‘I came home and said to my husband I don’t feel safe driving. I don’t want to have a dizzy spell and, you know, have an accident or something (…) I'm worried that I might be causing danger to myself or to anyone’ (**Zaynab: 277-278, 464**)

Here, Zaynab reflects on her experiences of PPPD and integrates a conscious awareness of how symptoms may, in potentially dangerous scenarios such as driving, put herself and others in danger. This fear of causing harm results in behavioural adaptations that avoid behaviours that may be dangerous if an unexpected bout of dizziness or disequilibrium was triggered.

2.3.2.1.3. Subordinate Theme 2.1c: Failure to Function Adequately

The final manifestation of fear Zaynab shows is the fear that she will be unable to function adequately as both a person and optometrist.

> ‘If I feel like I’m not 100% (…) I would say to my husband; I don't know if I, you know, want to work full time anymore. So, I started to like work half a day’ (**Zaynab: 457, 216**)

It is however important to note that some of her symptoms may be due to BPPV if this has not been fully resolved.

2.3.2.2. Superordinate Theme 2.2: Social Withdrawal and Isolation

Living with PPPD and dealing with chronic symptoms results in social withdrawal and isolation for Zaynab.

> ‘What I’d do is just, you know, isolate myself (…) I’ll just go off, you know, make an excuse and just go alone. Just to be able to concentrate on it and deal with rather than having all the other things around me’ (**Zaynab: 244, 258-259**)

The experiences of dizziness are shown to be over-powering and to cloud mental focus to the detriment of social interactions. Note, this account resonates with other themes such as sensory sensitivity and visual-overload described in study 2.

2.3.2.3. Superordinate Theme 2.3: Time

Adequate clinical time with a patient is crucial for Zaynab to feel like effective assessment and rehabilitation of health problems has occurred - both as a practitioner of optometry and as a PPPD patient.

> ‘Time is very important. Even in our job, you know, if we’re given more time with our patients, I think we can do more for them and patients feel that as well (…) to be honest the most informative experience and the most helpful experience was seeing Anna [the clinical scientist]. Because she erm took probably an hour to go through all my case history, do everything, try to (she did!) loads of tests, trying to figure out what was going on’ (**Zaynab: 336-337, 304-306**)

In contrast to the quote above, Zaynab discloses that interactions with general practitioners and specialists who are pressed for time have led to dismissive interactions and misdiagnosis– an experience that is, unfortunately, shared among the participants of the study.

> ‘When I saw the specialist, I literally saw him for like 5 minutes. He was quite dismissive to be honest he was like “oh are you dizzy now?” and I said “no” because I wasn't at the time and I hadn't had an episode for a while and he just said, “oh it's just a migraine” and said “it's probably just a vestibular migraine” and that's it and “I'm discharging you”. And I was like OK…’ (**Zaynab: 85-86,100**)

> ‘if you feel like somebody's running late or somebody's actually very late you don't want to take up much of their time’ (**Zaynab: 338-339**).

2.3.3. Participant 3: Owen

Owen is a 33-year-old male computer games designer who is an avid gamer and enjoys spending time playing online virtual video games (including head-mounted virtual reality games) and building computers. Owen was also diagnosed with ADHD in childhood. When asked how long he has experienced PPPD symptoms he stated ‘supposedly, I’ve had it all my life’ (**Owen: 88**); however, he has only recently been given the diagnosis of PPPD by a clinical scientist. He has also been diagnosed with non-epileptic seizures.

2.3.3.1. Superordinate Theme 3.1: Making Sense of Symptoms through Meaningful Metaphor

Articulating experiences of PPPD is reported to be qualitatively difficult with Owen stating:

> ‘it’s just unless you’ve experienced it yourself, it’s hard to put into words’ (**Owen: 662**)

In order to make sense of the condition and its physiological and psychological effects and their impact on him, Owen uses a meaningful metaphor (the computer metaphor) to make sense of his affliction and to articulate his lived experiences:

> 'the best way I can describe it is like a driver conflict where you got a driver for your mouse but then suddenly your mouse stops working but it's interfering with the keyboard and you start trying to type out erm trying to use the keyboard to get to the restart option, but things keep getting inverted or relocated and they're constantly shifting. They're not making sense even on the keyboard even though the letters are printed their actual functions have changed and the more, when you try fighting it, the more you complicate the actual issue on the PC itself' (**Owen: 950-956**)

The computer metaphor is used to articulate complicated vestibular and sensory experiences. The idea that he uses this metaphor to make sense of his condition is further reinforced by his use of language that supports the motif throughout the narrative such as:

> 'rebooting (…) programmed myself to (…) process (…) reset (…) restart' (**Owen: 936, 497, 940, 782, 937**).

2.3.3.2. Superordinate Theme 3.2: Out of Body Experiences

This superordinate theme captures Owen's experiences that are analogous to out of body experiences. The subordinate themes reflect the two types of experience he documents, (a) hallucinations and (b) dissociative experiences.

2.3.3.2.1. Subordinate Theme 3.2a: Hallucinations

Owen openly discusses that his experiences of PPPD sometimes include hallucinogenic experiences. Note, it is important to acknowledge that the experiences he is referring to may originate from PPPD, seizures, ADHD or a combination of the comorbidities and thus although the symptoms are present and valid may, or may not, be the direct consequence of PPPD alone.

> 'About two years ago I was helping the neighbour carry basic white chairs up the stairs. I was talking to her and I just, to me was like someone grabbed the back of my head and just pulled me back but I ended up swimming in the ocean. So, I was in the middle of the ocean it was night and I was confused like wait, wasn't I just on the stairs? And umm ended up having to

> swim for a bit. To me it felt like 20 minutes. Got to that beach. As soon as I got to that beach I was waking up and I was like what the hell?' (**Owen: 490-496**)

2.3.3.2.2. Subordinate Theme 3.2b: Disassociation

He also recalls episodes that result in a dissociative state, where he isn't aware of his embodied state.

> 'Like this one time in a dizziness episode my Dad challenged me said "okay if you could do 50 laps of the pool@ - (which was an Olympic size swimming pool) – " I'll give you £30. And I ended up going into this absolute trance I can't remember doing it… until my mum slapped me out of it!' (**Owen: 704-708**)

These experiences are interesting as, to date, no formal documentation of PPPD patients reporting such symptoms is published. The manifestation of 'out of body experiences' have been suggested in the past to be due to the vestibular system's role in body and self-perception in space. However, as this type of experience is socially stigmatised this may discourage disclosure. It should, however, be noted that the trance-like state Owen reports could be associated with his seizures or other co-morbidities. However, interestingly Owen is not the only PPPD participant to disclose such experiences (see Sian - participant 6, section 6.).

2.3.4. Participant 4: Regina

Regina is a 40-year-old woman with active diagnoses of PPPD and Fibromyalgia. Regina was a health care worker before ultimately having to give up work due to the demands of her health problems.

2.3.4.1. Superordinate Theme 4.1: Embodied Understanding of Health

This superordinate theme captures Regina's sense making techniques through her understanding of embodiment. Subordinate themes reflect her (a) sense making of illness through the physical familiarity of symptoms, (b) acceptance of the limits of the human body and (c) understanding of health compromising illness as analogous to war and conflict.

2.3.4.1.1. Subordinate Theme 4.1a: Understanding through Physical Familiarity

Regina explains that she simultaneously experiences multiple health compromising illnesses and that she understands conditions with more predictable symptoms best.

> ‘I think fibro I probably understand best. And then migraines, because I know the warning signs. And then vertigo because its unpredictable. Erm, yeah it’s a little more difficult to understand’ (**Regina: 330-332**)

2.3.4.1.2. Subordinate Theme 4.1b: Accepting the Limits of the Human Body

Regina’s daily interactions with health compromising illnesses have forced her to accept the limits of her body.

> ‘Yeah, just learning to cope and make the most of any good days erm yeah just learning to listen to what your body says and rest when you need to (…) I know there’s a certain way I’m feeling before a flare up and in that case, I try and take it easy… I don’t ask why me I just think well it’s a part of me and just wait for it to eventually go away’ (**Regina: 185-187, 224-225, 371-372**)

The persistence and the demands of her health compromising illnesses are shown to have given Regina an embodied awareness of her state and how she has learned to listen to and accept the limits of her body. She also expresses gratitude for symptom free days. This understanding of herself within her physical body may also be the reason as to why she does not centralise PPPD and its experiences as fibromyalgia plays a more central role in her life.

2.3.4.1.3. Subordinate Theme 4.1c: Health is Analogous to War and Conflict

Linguistic imagery devices that denote conflict and war are present throughout Regina’s narrative when discussing her health issues.

> ‘It’s just a matter of battling my way home (…) it can be a bit of a mind battle (…) just keep going (…) manage what’s happening and move forward (**Regina: 163-164, 372, 534-535**)

These indicators that Regina interprets living with her conditions as conflict are interesting given that she also explains that she has come to accept the limits of chronic illness. This implies there is acceptance of the tension between herself and her chronically ill identity, rather than full acceptance.

2.3.5. Participant 5: James

James is a 40-year-old engineer. James describes himself as ‘a normal guy, quite like sports and being active (…) just a regular guy’ (**James: 5-7**). During his training to become an engineer he developed labyrinthitis. After recovery of the infection, he then went on to develop PPPD. His mother also has a

history of labyrinthitis. James states that he is 'pretty much over it now' (**James: 225**) and feels 'pretty normal again' (**James: 226**) but slight symptoms do persist.

2.3.5.1. Superordinate Theme 5.1: An Anxious Predisposition

James is the only participant within the study to disclose that he believes that he was, to some degree, an anxious person before vestibular insult and the manifestation of PPPD.

> 'I am a bit of a worrier- I think. I never used to think I was, but I've realised I am a bit in comparison to others' (**James: 369-370**).

2.3.5.2. Superordinate Theme 5.2: The Disabling Nature of Persistent Postural-Perceptual Dizziness

This superordinate theme captures James', thoughts, feelings and perceptions of PPPD as a disability. Three subordinate themes reflect the physical, psychological and social dimensions of this theme: (a) the physical disability, (b) feeling drained and sub-optimal functioning and (c) cognitive dissonance between living with a disability and accepting the label of disabled.

2.3.5.2.1. Subordinate Theme 5.2a: Persistent Postural-Perceptual Dizziness (PPPD) is Physically Disabling

This subordinate theme captures the physically disabling nature of the condition that incapacitated James.

> 'Initially I was incapacitated for months (…) I was like handicapped weren't I, so I just felt, everything just felt more difficult (…) well when I was peak ill, I wasn't anything! I was just lying on my left side doing nothing. I was yeah, I was just erm; miserable, frustrated erm crippled' (**James: 300-303, 391-393**)

The idea that James could not, or would not, view himself as anything could reflect the effect of this incapacitation on his identity, which we know can be affected by life with PPPD (see participant 1, theme 1).

2.3.5.2.2. Subordinate Theme 5.2b: Feeling Drained: Sup-Optimal Functioning

James' narrative emphasises the secondary fatigue that arises from living with PPPD and having to manage the demands of the condition through general daily life.

> ‘It was so draining. It was just, like I said I felt like I was functioning at 70-80 percent (…) everything was more difficult. More draining (…) Gradually started getting better but I just felt horrendous…. But for months (like months!) after, I felt like I was functioning at sort of 80 percent (…) You just feel drained. Everything was draining. It was mentally draining. Cause things were harder than they should be… it was tough’ (**James: 301-303, 235, 268, 329-331**)

Note that James highlights both physical and mental fatigue, which may reflect the physical and psychological demands of the condition.

2.3.5.2.3. Subordinate Theme 5.2c: The Cognitive Dissonance between a Disability and the Disabled Identity

The use of language that denotes disability is prevalent throughout James’ narrative which builds and reinforces the motif of PPPD as a disability.

> ‘debilitating (…) crippled (…) handicapped’ (**James: 562, 393, 302**)

Despite the linguistic technique building a theme of disability, James shows that he would be uncomfortable identifying as disabled and labelling PPPD as a disability:

> ‘If it was a permanent thing then its 100 percent a disability. But I mean the fact that it passed; disability is a bit strong - but at the time it was disabling, yeah… but I dunno if I’d want to be classed as disabled. But yeah, it is disabling so it is, yeah’ (**James: 313-315**).

This extract raises two interesting points; the first, that James believes PPPD is a disability whilst it persists and would be classifiable as a disability if it were permanent. Since some patients do not ever fully recover from PPPD, this raises the question of whether they would consider themselves disabled. And if they should be recognised as such. Secondly, the extract alludes to the stigma of being associated or classified as disabled; functionally he described himself as disabled, but he would not feel comfortable being labelled so.

2.3.6. Participant 6: Sian

Sian is a 49-year-old wife and mother and describes herself as a family oriented ‘home-bird’ (**Sian: 10**). She has two adult children with her husband and identifies strongly with the role of caregiver,

mother and wife. Sian has developed severe anxiety and agoraphobia due to her PPPD and feels this has 'in some ways completely ruined my life' (**Sian: 735**).

2.3.6.1. Superordinate Theme 6.1. The Identity Loss of the Matriarch

Sian heavily identifies with the role of the matriarch and explains that living with PPPD has challenged her ability to function as a matriarch within her family unit.

The identity of the mother is central to Sian's understanding of herself and her identity. The experiences of PPPD have inhibited her ability to function as the mother she always has been.

> 'Then you know I say when It's all passed and I'm feeling and I've got a grip on, this is my mind playing tricks on me. I'll say right, to my daughter, tomorrow when you know in between my work calls or whatever let's go to the park, stick our trainers on and just walk around the park and enjoy the flowers then come home. And she's like "yeah we'll do that" - by the following day I'm finding an excuse not to do it because I'm convinced something bad will happen when I get there' (**Sian: 385-390**).

Sian's account shows us that anxiety is inhibiting her from conducting typical behaviours and actions of the mother. Interestingly, Sian's general narrative shows that she interprets her anxiety as the by-product of PPPD. This is explored further in Sian's subordinate theme 6.3. Sian discloses that she maintains the motivations to undertake motherly tasks but her experiences and symptoms, in particular the fear and anxiety around symptoms, stop her from executing these behaviours. This places Sian in a state of additional psychological distress as she then must deal with the psychological guilt her identity loss as a mother brings (see subordinate theme 6.1c).

As part of her identity as the matriarch, Sian identifies with the role of wife. Living with PPPD, she explains, has resulted in her avoiding typical activities that a partner may do with, or for, their significant other.

> 'Now my husband will say "do you want to go for a walk on the beach?" and I'll be like, "you can drive to the beach and I'll sit in the car "and and I try to… I try to dress something else up as nice like I'll… I'll make us a picnic and flask and we'll have a car picnic. Almost like I'll go but I... But I have to stay in my safe place. And I try to make it attractive to stay in the car rather than going out. And I've also done things like said okay then no problem knowing that its perhaps really windy then purposefully not taken a coat or a jumper. And when we've got

there, I've said "Oh no. I haven't got a coat or a jumper- you go for a quick walk I'll be alright" [laughs]' (**Sian: 374-381**)

Sian also shows how deep rooted this this issue is and how she can no longer execute general behaviours that, to her, are crucial to successfully functioning, and thus identifying, as the archetype of the wife.

> 'My husband has said I really fancy a chicken salad for tea, you know, when I get home and he's got home, and I've said "you'll have to take me- you'll have to go with me to the supermarket" cause we haven't got the chicken and I have not been able to leave the house to go and get the chicken. You know to, to.. that's not right. That's not right.' (**Sian: 462-466**)

These extracts show that living with PPPD has rendered Sian unable to conduct everyday behaviours and tasks that she believes are crucial to her identity as a wife. They also show the cumulative effects of PPPD on previously taken-for-granted activities, and the potential consequences of these insidious impacts on self-esteem and relationships.

Sian goes on to express guilt associated with her limitations:

> 'I feel extreme guilt as a mother and as a wife. Because I know that they would perhaps like to go to the park and you know, don't get me wrong they're old enough to go and do it themselves but as a family to go to the park (…) go the cinema, go for a picnic (…), go have an hour on the beach, go to the cinema, whatever. You know when you can, and all that sort of stuff and and I'll do anything to get out of it. I'll say 'oh go on your own you're 23 I don't want to watch those sorts of films" or "go with your father" or you know, and I feel [pause] guilty for not sharing in those things with them or not helping them experience those things. But I don't feel sad that I'm missing out, cause I don't feel as though I'm missing out because the fear is so extreme. I don't want to feel that fear– I'm happier not feeling the fear and being safe in my own home. (..) But I also feel that it's a shame.' (**Sian: 401 – 408**)

Sian also shows the inner struggle that she feels where she must deal with both the guilt of her avoidant behaviours and the relief that she does not have to engage in activities that may trigger her symptoms. This results in a cognitive dissonance between the two opposing states and a complicated cycle between guilt and relief and guilt for feeling relief.

2.3.6.2. Superordinate Theme 6.2: Persistent Postural-Perceptual Dizziness as the Cause of Anxiety and Panic

Throughout Sian's narrative she makes it clear that she believes that her anxiety and panic conditions are the by-product of living with PPPD.

> 'So, so this [PPPD] has created an awful situation with anxiety and panic and agoraphobia (…) which then has created an agoraphobia situation (…) So I know I'm in this, I know I'm on a hamster wheel of visual things can trigger this off balance feeling and that then triggers the anxiety and panic which just exacerbates the whole thing. So, I feel like I'm in a bit of a mess with it actually [laughs] (…) There's a vestibular problem, you know, there's a mental health problem now really, I think has been created as a result of it and has compounded as a result of it' (**Sian: 150, 172, 193-196, 740-742**)

Sian's secondary psychological conditions are understood in relation to PPPD, and she shows that she believes that the PPPD has caused the psychological conditions. She also notes that she is stuck in a cycle between the vestibular and psychological symptoms but again documents that it is the symptoms associated with PPPD that triggers her anxiety and panic.

2.3.6.3. Superordinate Theme 6.3: Ownership Through Language

Sian uses language that captures her lived experiences to form psychological ownership over her symptoms. She articulates and re-labels her symptoms in ways she finds accessible for herself.

> 'I say to my husband oh I'm really *boaty* at the minute (…) I get that a lot with just people passing me or I might be moving that way and somebody else is moving that way and that creates the *boaty*. That creates the *boaty* feeling. So the vision causes the kind of *whoozy* in the head and mine and other peoples motion causes the *boaty*.' (**Sian: 122, 294-297**)

By labelling her symptoms in relation to how she understands and experiences them this allows her to gain psychological ownership over them and make the symptoms her own and more predictable.

2.3.6.4. Superordinate Theme 6.4: Disassociation and Depersonalisation

Experiences of disassociation and depersonalisation are disclosed by Sian. She states that during episodes she commonly feels experiences of dissociation or disconnected from the body she inhabits.

> ‘I feel sometimes like I… I… I can’t really put into words… the, you know, the disassociation that they refer to? That I’m not... there. I’m not there! I’m sort of there but I’m not there (…) it was very bright and I hadn’t taken sunglasses and I just (...) You know I wasn’t blind, I could hear noises, but it was like I can remember standing there talking to my parents, by my car (by my safe place!) so I can get in anytime I want to and just chatting away and it was very bright and I hadn’t taken my sunglasses and I just.. I could hear them talking but it was like… I don’t know what you’re on about it was like blah bah blah blah blah blah… it was like... as if I was zoned out, I was there but I wasn’t there. I could see. You know I wasn’t blind. I could hear noises, but it was like as if I was just not really there. And I get that a lot and I then straight away and the panic hits in and I think straightway what’s going on?! What’s going on?! Is there something wrong with my brain cause I’m not really here? What’s going on? What’s going on? (…) It’s almost as if I’m having a night terror but awake’ (**Sain: 480-495**)

Sian captures the confusion these dissociative experiences give her by recounting the numerous rhetorical questions she would ask herself during an episode. Furthermore, by comparing the experience to a night-terror she captures the pure essence of fear that the experience brings. Finally, she states that:

> ‘I’ve never actually said: “you know what, I feel totally dissociated at the moment and that’s part of my panic”. So, I let it, let it… I’ve never actually said that….’ (**Sian: 504-506**)

This captures the fear that some symptoms may carry negative social connotations or worry loved ones. Furthermore, as Sian openly admits she has never disclosed this symptom before the reason why this symptom may not be associated with PPPD may be due to the social inhibitions of disclosing such symptoms.

2.4. Study 2: Using Interpretative Phenomenological Analysis to Establish the Shared Psycho-Social Impacts of Living with Persistent Postural-Perceptual Dizziness

The aim of study 2 was to explore cross-participant themes from the qualitative data generated in study 1. A sample of six is considered adequate for a cross-participant analysis using IPA as it allows the researcher to maintain the focus on idiographic expressions whilst giving enough narratives to form meaningful comparisons across participants (Smith, Flowers and Larkin, 2009). This is the most efficient way of using the deeply rich accounts produced in study 1 and may reveal themes that are important to PPPD but were not central to each participants world view and narrative.

2.4.1. Study 2: Method

2.4.1.1. Participants/ Procedure

Qualitative data from the same six participants from Study 1 were used in this Study 2. Thus, the procedure remained the same as in Study 1.

2.4.1.2. Analytic Strategy

The analytic strategy remained the same as in study 1; however, here I widened the scope of the investigation and analysed data across the participants qualitative accounts. I therefore analysed each idiographic case in tandem with one and established emerging superordinate and subordinate themes that were present across all the PPPD patient participants. These represent shared lived experiences of PPPD.

2.4.2. Study 2: Results - Cross Participant Interpretative Phenomenological Analysis

Of the six participants, four self-identified as migraineurs. Participants generally reported a qualitative loss of self, self-esteem or confidence in physical abilities – these are best represented in study 1. Five of the six participants reported that primary healthcare workers initially misdiagnosed PPPD for anxiety. Only James did not report experiencing this. Two of the six participants also reported out of body and dissociative symptoms - these are considered in in detail in study 1.

2.4.2.1. Superordinate Theme 1: Poor Mental Well-being

The narratives shared highlight the impact of PPPD on general mental well-being and mood. This is best described as the superordinate theme of poor mental well-being with two subordinate themes; (a) gender differences in disclosure of impaired mental well-being and (b) "look, am I going mad?" **(Sian: 779**).

> 'obviously it affected my mood, my overall persona and everything, because it was so draining (…) you feel low, don't you? (…) I mean it was tough' (**James: 300-301; 329**)

However, Regina states:

> "It's no use letting it upset you too much" (**Regina: 355)**

Regina's' use of the words 'too much' suggests that although she is accepting of her health compromising illnesses these experiences still do upset her and that this is something she must simply deal with. This invokes the idea of acceptance of one's limits.

2.4.2.2. Subordinate Theme 1.1a: Gender Differences in the Disclosure of Impaired Mental Well-Being

In regard to the general impact of PPPD on mental-wellbeing, female participants were more open to disclose their negative psychological experiences and feelings.

> 'I, I quite often... when I get upset now about the sort of situation I find myself in, the words I use are I just want to be how I was before all of this' (**Sian: 345-346)**

> 'I couldn't go a day without crying (…) There were days where I didn't want to get out of bed. Cause you knew it would be there' (**Lara: 332-334)**

Sian even goes as far to say:

> 'I feel in some ways it has completely ruined my life' (**Sian: 735**)

Accounts suggest that the physical symptoms are only one part of the disorder and that the daily psychological struggles overwhelm their minds and lives.

2.4.2.3. Subordinate Theme 1.1b: Look, am I going mad?

A failure to ascertain an adequate diagnosis and poor understanding of the disorder by primary and secondary healthcare professionals leaves the patient to begin to question their own sanity. This is further reinforced by the misguided, and often dismissive, diagnosis of anxiety:

> 'At the start I was like, 'you're actually mental" like "there is nothing wrong with you" - especially when you go to the doctors and one was just like - you've just got anxiety' (**Lara 246-247**)

Participants explain that after no adequate diagnosis or treatment for their symptoms, they ultimately grow and worsen to the point where they are forced to ask themselves, and healthcare professionals if they are going mad:

> 'It actually took me going to the doctors and crying saying "look, am I going mad? Is there something wrong with my mental health? Am I actually going mad?" Because I come back about it and it got to that point before I was offered you know the mental health side stuff' (**Sian: 779-782**)

The repetition of this question - 'am I going mad? - shows the helplessness and fear that comes from genuinely having to ask this question. This interaction is shown to be highly frustrating and almost terrifying where Sian does not know if she is mentally well or not. Ultimately, this leads her to question her own sanity: a narrative that is shared with other female participants.

2.4.2.4. Superordinate Theme 2: Sensory Overload

A key superordinate theme that emerges from the data is the idea of sensory overload; that there is just too much visual information for them to process.

> 'It's just an extra sense that I don't need to deal with. So, you know I'd just rather not. You know sort of try and look at anything. Its cause, it's almost like a sensory overload. So, if you cut off one of the senses it sort of helps you deal with it a bit better' (**Regina: 288-290)**

Simply put:

> "It just like became overwhelming really" (**James: 129**)

Participant's also state that this is obviously worse when more visual information is available, like when undertaking tasks outside the home:

> 'It's worse when I'm out the house cause so much is going on so much is moving – traffic, people, crowds erm too much things to look at in a supermarket you know when you've got rows and rows of things it's just too much (…) It's too much, it's too much erm too much visual information almost' (**Sian: 134-136; 141**)

This overload of visual information has also resulted in some patients having to adopt behavioural strategies to mitigate or ward from intense visual stimuli, showing that behavioural adaptations are necessary to complete everyday functions such as shopping:

> 'I used to wear sunglasses all the time because I really needed to tone down the world. It was like it was too like, too contrast, and visual! And it would like throw me.' (**Lara: 567-568)**

The use of the phrase 'tone-down the world' (**Lara: 567**) gives the impression that these experiences are heightened and too intense and need to be turned down in order to conduct simply daily tasks.

2.4.2.5. Superordinate Theme 3: Sleep Impairments

Surprisingly, sleep was reported as impaired as a function due to the experiences of PPPD. Interestingly, most participants causally alluded to this as if it is an obvious implication due to the disorder:

> 'It can happen quite randomly where I wake up in the night and you know my eyes are just going backwards and forwards and erm you know quite disorientated' (**Regina: 77-78**)

> 'I think it was waking me up. Like I said, it felt like I was on a ship and my head was being thrown from side to side and that was waking me up, so I was trying to force myself back to sleep' (**James: 100-102**)

To date, no research has documented an association between sleep and/or sleep quality and PPPD.

2.5. Supplementary Question 1: Do Persistent Postural-Perceptual Dizziness Symptoms Affect Patients' Ability to Engage with Recreational Visual Motion?

Supplementary research conducted to inform the interview guide suggested that PPPD symptoms may not be triggered by television and movie viewing. Thus, as part of the current qualitative studies a question relating patients' symptoms when engaging with media based visual motion activities was included.

Contrary to notions within in the literature, five of the six participants involved in the study stated that viewing television/movies did not trigger or exacerbate their symptoms. Only one participant reported being a video gamer; this participant stated that gaming did not trigger or exacerbate PPPD symptoms.

> 'Like, the only time I could get away from it was watching TV – for some reason watching TV was almost fine (…) normally I could watch TV and that was fine. So, so I spent a lot of time at the computer' (**Lara: 252 – 256**)

> 'I was like just like watching a Disney film as much as I could just in and out of everything' funny enough I could play a video games and I actually got a VR headset' (**Owen: 120, 311-315**)

> 'Watching tele was alright. Even when, Like I said it was at the peak cause when I was off originally it was when World cup was on so like if I was lay on my left-hand side, I could like I said it was like nothing was wrong I could watch the tele fine' (**James: 437 – 440, 452**)

What this may suggest is that, for these five PPPD patients, the current understanding of the disorder is not accurate – they can engage with passive visual motion without symptom provocation or exacerbation. Note, accounts do suggest that this is when seated, stationary provided and in a safe space. It is however important to highlight that the data presented in Chapter 4 suggested that PPPD experiences could not predict TV/movie behaviours in a student sample. The Thesis therefore presents converging mixed-methods evidence that PPPD experiences are not related to recreational visual motion use and therefore may affect recreational motion activities.

2.6. Study 3: Exploring the Relationships Between Persistent Postural-Perceptual Dizziness and the New Psycho-Social Constructs of Interest

The qualitative research presented in Chapter 2 has suggested that, in these six participants identity loss, sleep, identity and general mental well-being (including anxiousness) were negatively impacted due to living with PPPD. Furthermore, the participants also reported a general sensitivity across their wider senses. Previous research has shown that PPPD is associated with a general sensory sensitivity across sensory modalities (Powell et al., 2020); however, this could also be due to a cognitive construct known as somatic amplification - the tendency to perceive somatic sensations as more intense than the sensory signal suggests (Barsky, Goodson, Lane & Cleary, 1988).

The aim of study 3 was to explore the relationships between PPPD symptoms and the psycho-social variables identified in the qualitative enquires. To ascertain a large enough sample, relationships were explored in a large student sample.

2.6.1. Study 3: Methods

2.6.1.1. Study 3: Participants

Three hundred and ninety-seven undergraduate psychology student participants (350 females; 42 males; 2 non-binary people) self-selected to participate in an online study exploring PPPD and psycho-social factors. No wider demographic information was collected. No exclusion criteria were applied. All participants received one course credit for participation. Participants were in an imposed lockdown during data collection.

2.6.1.2. Study 3: Materials

2.6.1.2.1. Visual Vertigo Analogue Scale (VVAS)

The VVAS itself is a 9-item questionnaire using an analogue scale of representation, where responses can range between 0 and 10. Participants are asked report their dizziness levels in a range of intense visual environments, where 0 = no dizziness and 10 = extreme dizziness. Raw scores are transformed by calculating the mean score of items. This score is then multiplied by ten, meaning the minimum score on the VVAS is 0, and the maximum 100. Lower scores represent little to no PPPD symptoms, higher scores suggest PPPD symptoms. See Appendix 1.1 for the scale.

2.6.1.2.2. The Niigata PPPD Questionnaire (NPQ)

A new tool of measuring PPPD symptoms was recently published offering a diagnostic supporting measure of PPPD (Yagi et al., 2019). The Niigata PPPD Questionnaire (NPQ). The NPQ reliably assesses the three unique exacerbating factors of PPPD; upright posture/walking (Cronbach's alpha = 0.88), movement (Cronbach's alpha = 0.75) and visual stimulation (Cronbach's alpha = 0.83) which can be assessed individually or within the total scale (Cronbach's alpha = 0.91). The NPQ is a 12-item scale (four items per sub scale) which asks the respondent to indicate the difficulties in daily life activities due to dizziness on a scale of 0 (None) to 6 (Unbearable). Items for the upright posture/walking factor include 'walking at a natural pace' and 'sitting upright in a seat without back and arm support'. Items for the movement factor include 'quick movements such as standing up or turning your head' and 'riding a car, bus or train'. Items for the visual stimulation factor include 'looking at large store displays' and 'watching TV or movies with intense movement'. Scores on each item are summed to from a factor score and a general total score. The minimum score for each factor is 0 and the maximum 24. For the full scale the lowest possible score is 0 and highest is 71.

2.6.1.2.3. The Pittsburgh Sleep Quality Index (PSQI)

The Pittsburgh Sleep Quality Index (PSQI; Buysee, Reynolds, Monk, Berman & Kupfer, 1989) is a reliable 19-item scale that assess the general quality of sleep of the past month (Cronbach's alpha = 0.83). The scale captures seven unique composites of sleep quality which are sleep duration, habitual sleep efficacy, sleep disturbance, the use of sleep medications and daytime dysfunction. Cronbach's alphas for each of the component scores range between 0.76 (habitual sleep efficacy and subjective sleep quality) and 0.35 (sleep disturbances). The mean component correlation co-efficient = 0.58. Scores on all sub scales are summed to produce a total PSQI score that can range from 0 – 21 with higher scores representing poorer sleep quality. The global PSQI score is the result of summing all seven component scores where the low scores represent good sleep quality, and high scores represent poor sleep quality. The tool is able to detect sleep quality in clinical and non-clinical samples (Buysse et al., 1989). Note, in this study only used the global PSQI score. For the full psychometric properties see Appendix 3.3.

2.6.1.2.4. Warwick-Edinburgh Mental Well-Being Scale (WEMWBS)

The Warwick-Edinburgh Mental Well-Being Scale (WEMWBS; Tennant et al., 2007) is a reliable 14-item scale that measures general mental well-being in the general population (Cronbach's alpha = 0.89) and patient populations (Cronbach's alpha = 0.91). Participants are asked to respond to items such as, 'I've been feeling optimistic about the future' and 'I've been feeling good about myself' on a 5-point Likert scale where 'none of the time' = 1, 'rarely' = 2, 'some of the time' = 3, 'often' = 4 and

'all of the time' = 5. Items cover both hedonic and eudemonic aspects of mental health including positive affect, satisfying interpersonal relationships and positive functioning – though they do not represent their own sub-scales. Items are summed to create a total WEMWBS score. Total scores can range from 14 – 70 with a higher score on the WEMWBS indicating a higher level of mental well-being.

2.6.1.2.5. The Hospital Anxiety and Depression Scale (HADS)

The Hospital Anxiety and Depression Scale (HADS: Zigmond & Snaith, 1983) is a 14-item questionnaire that assesses depressive and anxious symptomatology. Seven items assess anxiety such as ''I feel tense or wound up' and seven assess depression such as, 'I still enjoy things as much as I used to'. Possible responses lie on a 4-point Likert scale. Note, the worded responses for each item vary across items. In some items such as 'I can laugh and see the funny side of things', participants may respond with, 'as much as I always could' (0), 'not quite so much' (1), 'definitely not so much now' (2), or 'not at all' (3). Whereas in items such as 'I feel restless as I have to be on the move' participants must respond with 'very much indeed' (3), 'quite a lot' (1), 'not very much' (2) or 'not at all' (0). Responses are therefore context and question specific. A total anxiety score is formed by summing scores from the seven anxiety items. The total depression score is formed by the same process, here summing the seven depression items. This scale has been used with PPPD patients in the past and in research exploring PPPD symptoms in the general healthy population (Pavlou, Davies & Bronstein, 2006; Powell et al., 2020).

2.6.1.2.6. The Rosenberg Self-Esteem Scale (RSE)

The Rosenberg Self-Esteem Scale (RSE; Rosenberg, 1965) is a 10-item scale that measures global self-worth by probing positive and negative feelings towards the self. The scale can therefore be used to capture sense of identity and self. Items include, 'on the whole I am satisfied with myself' and 'at times I think I am no good at all'. Responses to all 10 items are on a 4-point Likert scale where participants may respond with 'strongly agree', 'agree', 'disagree' or 'strongly agree'. On the five non-reversed items these responses translate to 3, 2, 1 and 0. For the five reversed items the same answers are coded as 1, 2, 3 and 4. Scores are summed to give a total RSE score. Scores may range between 0-30 with lower scores indicating lower self-esteem. Typically, respondents fall between 15 – 25, however below 15 can be considered as evidence of low self-esteem (Rosenberg, 1965).

2.6.1.2.7. The Somatic Amplification Scale (SA)

The Somatic Amplification Scale (SA; Barsky, Goodson, Lane & Cleary, 1988) is a 10-item scale which asks the respondent to state how 'characteristic of you in general', 10 items such as 'when someone else coughs it makes me cough too', 'I can't stand smoke, smog or pollutants in the air' and 'I hate to be too hot or too cold' are. Responses are on an ordinal scale of 1–5. Scores are summed to represent the general somatic amplifications of an individual. Scores may range between 10 and 50. A higher score on the Somatic Amplification Scale represents greater cognitive amplification of experience. Typical scores for healthy participants range between 24 - 29 (Nakao & Barksy, 2007).

2.6.1.3. Procedure

Participants self-selected to take part in an online study for course credit. Participants were free to complete the study using a smartphone, tablet, laptop or computer. The study took approximately 15 minutes to complete. Participants were free to withdraw at any time and omit any questions without explanation. Study information was presented online with consent taken digitally. Participants were debriefed upon completion of the survey.

2.6.1.4. Planned Statistical Analyses

Exploration of the relationships between n PPPD symptoms and psycho-social variables were planned to be explored using correlation and multiple regression analyses. For the multiple regression models were the psycho-social constructs would be entered into the model as predictor variables, with PPPD symptoms entered as the outcome variable. Thus, two multiple regression models where planned where anxiety, depression, self-esteem, general mental well-being, sleep and somatic amplification were entered into the model as predictor variables. In the first model scores on the VVAS would be entered into the model as the outcome variable. Model two would use scores on the NPQ.

The Median Absolute Deviation (MAD) was selected an outlier detection tool, where $k = 3.5$ would be used to identify outliers on each of the measured variables. This results in any score +/- 3.5*MAD to be considered an outlier and removed from data analysis. All analyses were pre-planned to be conducted using JASP.

2.6.2. Study 3: Results

2.6.2.1. Descriptive Statistics

The mean and standard deviation for each measure capturing PPPD symptoms were as follows: VVAS = 12.83 (12.13), NPQ = 10.52 (8.83), NPQ sub-scale 1 (upright posture) = 1.55 (2.04), NPQ sub-scale 2 (movement) = 5.32 (3.96) and NPQ sub-scale 3 (visual stimulation) = 2.87 (3.10).

The mean and standard deviation for each psycho-social construct were as follows: PSQI = 6.96 (3.26), HADS Anxiety = 9.28 (4.44), HADS Depression = 5.93 (3.78), RSE = 15.89 (205), WEMWS = 42.86 (12.18) and SA = 29.78 (6.17).

2.6.2.2. Inferential Statistics

2.6.2.2.1. Correlating Measures of Persistent Postural-Perceptual Dizziness (PPPD)

Data are presented in Table 2.1. The data suggest that the two measures of PPPD (the VVAS and NPQ) were moderately correlated, though not colinear, ($r = 0.68, p = <.001$). The VVAS was also found to correlate with the three subcomponent measures of the NPQ: upright right posture/walking ($r = 0.49, p = <.001$), movement ($r = 0.59, p = <.001$) and visual stimulation ($r = 0.61, p = <.001$).

2.6.2.2.2. Correlating Psycho-Social Constructs with Persistent Postural-Perceptual Dizziness Symptoms

Correlational data are presented in the correlation matrix in Table 5.1. Correlational analyses were performed between the all measures of PPPD symptoms (VVAS, NPQ and NPQ sub measures) and the psycho-social variables of interest. Data suggest that poor sleep quality, poorer general mental well-being, somatic amplification, higher levels anxiety and depression, and lower levels of self-esteem are all correlated with PPPD symptoms. Though the magnitude of the linear relationship varies between the scales and constructs used. Correlations exist regardless of which PPPD measure is used unless the construct is self-esteem, where when using the VVAS, no correlation was detected due to the fact that p values must be adjusted for multiple comparisons. See Table 5.1 for Rho correlation coefficients and associated p values.

Table 2.1

Correlation matrix showing the correlations between PPPD measures and psycho-social constructs.

Variable		VVAS	Niigata PPPD Questionnaire (NPQ)	NPQ 1: Upright posture	NPQ 2: Movement	NPQ 3: Visual Stimulation	Pittsburgh Sleep Quality Index	HADS Anxiety	Romberg Self Esteem	Warwick Edinburgh Mental Well Being Scale	Somatic Amplification	HADS Depression
1. VVAS	n	—										
	Spearman's rho	—										
	p-value	—										
2. Niigata PPPD Questionnaire (NPQ)	n	388	—									
	Spearman's rho	0.682 ***	—									
	p-value	< .001	—									
3. NPQ 1: Upright posture	n	354	354	—								
	Spearman's rho	0.492 ***	0.726 ***	—								
	p-value	< .001	< .001	—								
4. NPQ 2: Movement	n	393	389	354	—							
	Spearman's rho	0.587 ***	0.896 ***	0.483 ***	—							
	p-value	< .001	< .001	< .001	—							
5. NPQ 3: Visual Stimulation	n	380	380	349	381	—						
	Spearman's rho	0.607 ***	0.842 ***	0.526 ***	0.621 ***	—						
	p-value	< .001	< .001	< .001	< .001	—						
6. Pittsburgh Sleep Quality Index	n	355	350	317	356	343	—					
	Spearman's rho	0.219 ***	0.267 ***	0.184 ***	0.275 ***	0.196 ***	—					
	p-value	< .001	< .001	< .001	< .001	< .001	—					
7. HADS Anxiety	n	387	382	347	388	374	357	—				
	Spearman's rho	0.358 ***	0.409 ***	0.314 ***	0.367 ***	0.306 ***	0.329 ***	—				
	p-value	< .001	< .001	< .001	< .001	< .001	< .001	—				
8. Romberg Self Esteem	n	379	374	339	380	366	355	381	—			
	Spearman's rho	-0.115 *	-0.153 **	-0.107 *	-0.139 **	-0.109 *	-0.161 **	-0.340 ***	—			
	p-value	0.025	0.003	0.049	0.007	0.037	0.002	< .001	—			
9. Warwick Edinburgh Mental Well Being Scale	n	387	382	347	388	374	357	389	381	—		
	Spearman's rho	-0.202 ***	-0.259 ***	-0.236 ***	-0.210 ***	-0.164 **	-0.369 ***	-0.539 ***	0.452 ***	—		
	p-value	< .001	< .001	< .001	< .001	0.001	< .001	< .001	< .001	—		
10. Somatic Amplification	n	378	373	338	379	365	354	380	380	380	—	
	Spearman's rho	0.186 ***	0.257 ***	0.207 ***	0.279 ***	0.221 ***	0.158 **	0.241 ***	-0.009	-0.095	—	
	p-value	< .001	< .001	< .001	< .001	< .001	0.003	< .001	0.861	0.065	—	
11. HADS Depression	n	387	382	347	388	374	357	389	381	389	380	—
	Spearman's rho	0.209 ***	0.331 ***	0.271 ***	0.298 ***	0.244 ***	0.417 ***	0.600 ***	-0.366 ***	-0.617 ***	0.079	—
	p-value	< .001	< .001	< .001	< .001	< .001	< .001	< .001	< .001	< .001	0.124	—

* p < .05, ** p < .01, *** p < .001

2.6.2.2.3. Predicting Persistent Postural-Perceptual Dizziness When Symptoms are Quantified by the Visual Vertigo Analogue Scale

A stepwise multiple linear regression was fitted to the data to estimate the ability of anxiety, depression, self-esteem, general mental well-being, sleep and somatic amplification in predicting PPPD symptoms, as measured by the VVAS. The model suggests that anxiety and somatic amplification could significantly predict participants scores on the VVAS. The regression model could explain 14.8% of the variance and that this model was a significant predictor of PPPD symptoms: $F(2, 35) = 30.32$, $p = <.001$. The model suggests that anxiety ($\beta = 0.95$, $p = <.001$) and somatic amplification ($\beta = 0.28$, $p = 0.01$) were the only predictors that uniquely contributed to the model. The final predictive model was as follows: VVAS = -3.59 + (0.95*Anxiety) + (0.28*Somatic Amplification).

2.6.2.2.4. Predicting Persistent Postural-Perceptual Dizziness When Symptoms are Quantified by the Niigata PPPD Questionnaire (NPQ)

A second stepwise multiple linear regression was fitted to the data to estimate the ability of anxiety, depression, self-esteem, general mental well-being, sleep and somatic amplification in predicting PPPD symptoms, as measured by the NPQ. The model suggests that anxiety, somatic amplification and depression could significantly predict participants scores on the NPQ. The regression model could explain 20.3% of the variance and that this model was a significant predictor of PPPD symptoms: $F(3, 346) = 29.09$, $p = <.001$. The model suggests that anxiety ($\beta = 0.53$, $p = <.001$), somatic amplification ($\beta = 0.27$, $p = <.001$) and depression ($\beta = 0.41$, $p = 0.01$) were the only predictors that significantly contributed to the model. The final predictive model was as follows: NPQ = -4.32 + (0.53*Anxiety) + (0.27*Somatic Amplification) + (0.41*Depression). Supplementary multiple regression analyses were performed using each of the sub scales of the NPQ. These are provided in Appendix 2.4.

2.6.3. Study 3: Interpretations

Findings suggest that the psycho-social constructs are correlated PPPD symptoms; however only anxiety, somatic amplification are significant predictors of PPPD symptoms when using the VVAS. When using the NPQ the significant predictors are anxiety, somatic amplification and depression. Note, that although the models explain a relatively small amount of variance in the data this is meaning and interesting. This data suggests that PPPD is not a solely visual problem as between psycho-social factors can explain approximately 14.8% – 20.3% of the variance in symptoms. This highlights the notion that psycho-social factors are associated with the PPPD condition. Thus, PPPD is not a solely visual problem.

2.7. Chapter 2: Discussion

Living with PPPD can result in a debilitated patient with complex co-occurring illnesses that diminishes quality of life - though the lived experiences of people with PPPD are seldom probed in research (Guerraz et al., 2001; Mendel, Lutzen, Bergenius and Bjorvell, 1997; Sezier, Saywell, Terry, Taylor & Kayes, 2019). The aim of Chapter 2 was to explore the lived experiences of people with an active diagnosis of PPPD to better understand the lived experiences of PPPD and the psycho-social consequences of the condition. The analyses revealed a multitude of deeply personal superordinate and subordinate themes that were personalised to each idiosyncratic participant. The themes identified can generally be considered in terms of the following 5 motifs; identity loss, dismissal and non-belief, poor psychological well-being, out of body experiences and processes of sense-making. Shared themes included poor metal well-being, sensory overload, sleep impairments and PPPD not affecting television/movie watching. Quantitative explorations of the relationship between these constructs suggest that poor sleep quality, poorer general mental well-being, somatic amplification, higher levels anxiety and depression, and lower levels of self-esteem are all correlated with PPPD symptoms - though the strength of the relationships vary. However, only anxiety, somatic amplification were found to be significant predictors of PPPD when the PPPD is measured by the Visual Vertigo Analogue Scale (VVAS: Dannenbaum, Chilingaryan & Fung, 201. When PPPD is measured by the newer Niigata PPPD Questionnaire (NPQ; Yagi et al., 2019) anxiety, somatic amplification and depression were the only variables to significant predict PPPD symptoms. This leaves future researchers with promising avenues for future research into novel psycho-social factors associated with PPPD.

2.7.1. Identity Loss

The qualitative findings suggest that, in this sample, living with PPPD can result in a loss of the psychological self. This supports previous research has suggested that living with PPPD results in a loss of the sense of self (Seizier, Saywell, Terry, Taylor & Kayes, 2019). The analysis of lived experiences suggests that, for some participants, PPPD results in a disabled state. However, participants do not feel comfortable identifying with the label of disability. This may be due to the complex invisible nature of the condition and the fact that recovery is possible. Participants do however agree that PPPD is both physically and psychologically disabling resulting in a reduced quality of life. This data supports the works by Mueller, Schuster, Strobl and Grill (2012) who found that PPPD is a disabling condition. Future research should look to establish PPPD as a disability during active symptoms in hopes of giving validity to the condition and allowing patients to access a

range of support to manage their disabled life. Qualitative research should also consider empowering the patient group to accept the status of disabled should they wish to.

2.7.2. Out of Body Experiences

Two of the participants reported an out of body experience (explained as a hallucinogenic or dissociative experience). Research has associated such experiences with vestibular compromise due to the systems role in spatial mapping and the [perception of the self-within space (Ferre, Lopez & Haggard, 2014; Kaliuzhna, Vibert, Grivaz & Blanke, 2015; Lopez & Elziere, 2018). It is believed that this is due to these symptoms occur in the vestibular compromised as distorted vestibular signals 'mismatch' with alternative sources of sensory input, which creates an incoherent spatial reference frame resulting in experiences of detachment (Sang, Jauregi-Renaud, Green, Bronstein & Gresty, 2006). The research collected in Chapter 2 is, however, the first to present data suggest that out of body and dissociative experiences may be present in PPPD. To date, no research has currently explored out of body experiences, hallucinations or dissociative symptoms in PPPD, even though the theoretical assumptions of the origin of symptoms and condition visuo-vestibular mismatch and conflict resolution are key to the condition are almost identical. This data is the first to suggest that these symptoms can arise in PPPD. Future research should look to explore this promising area of future research by exploring the relationships between acute vestibular compromise, PPPD and out of body and/or dissociative symptoms to better understand how vestibular sensations are related to the sense of disconnection from the physical world.

2.7.3. Psychological Well-Being

The participants show that living with PPPD negatively impacts their general psychological well-being. This is evident through a number of superordinate and subordinate themes such as; social withdrawal and isolation, the manifestation of anxiety, dissociation and fear of symptoms and consequences. Each of these themes reflect some form of negative impact on the participant's general psychological well-being. This work is, however, the first to explore and analyse accounts in a case by case manner, allowing for the generation of rich themes that capture the composites of psychological well-being. However, the findings are congruent with those of previous literature (Seizier, Saywell, Terry, Taylor & Kayes, 2019; Yardley & Beech, 1998). Future research should look to establish if these idiosyncratic themes are emergent across a larger cohort of patients. This will complement the correlational research between PPPD symptoms and markers of psychological well-being included in this chapter.

2.7.4. Dismissal and Non-Belief

The majority of participants in this study disclosed that they did not feel listened to or validated when trying to seek out a diagnosis for their PPPD symptoms. It is commonly documented by the PPPD patient group that they do not feel listened to, taken seriously or supported by healthcare professionals in ascertaining the correct diagnosis and rehabilitation programmes (Seizier, Saywell, Terry, Taylor & Kayes, 2019). This actually led some participants to question their own sanity. Relatedly, PPPD symptoms have been known to trigger 'gas-lighting' effects, where patients believe they have 'lost their minds', especially when medical professionals misdiagnose their symptomatic presentation or do not make the patient feel that their experiences are valid or 'real' (Seizier, Saywell, Terry, Taylor & Kayes, 2019). The data presented in Chapter 2 strongly supports these previous findings.

Female participants interviewed believed that sexist attitudes towards women and PPPD symptoms negatively impacted their clinical interactions, journey to a diagnosis, and sense of self-worth and validation. Women often report that healthcare professionals do not take them seriously or take them, or their symptoms seriously and make them feel valid (Denny, 2004; Hoffman & Tarzian, 2001; Jones & Sutton, 2002; Tardos, 2019; Werner, Isaksen & Malterud, 2004). This suggests that PPPD may be another condition that is impacted by sexism in healthcare along with conditions such as chronic pain and endometriosis (Denny, 2004; Werner & Malterud, 2003).

PPPD patients, including some patients described here, often report that they feel that they are not listened to, that they have had negative experiences with healthcare professionals, and/or that their condition has been misdiagnosed or ignored (Seizier, Saywell, Terry, Taylor & Kayes, 2019). Thus, a key strength of the research collected in Chapter 2 is the commitment to the ideographic approach. This allowed the researcher to fully invest in the participant's story and support them through the process of disclosure, reflection and sense-making. Thus, these PPPD patients have been given a platform to share their lived experiences with a trained qualitative research psychologist in an informal semi-structure interview. This may be an empowering and validating experience for patients. However, by adopting an ideographic approach a limitation of the research may be the small sample sizes used. Future research should aim to collect data on the lived experiences from a large PPPD cohort to confirm findings outside of the patients sampled in Chapter 2. One plausible method of conducting such research may be to data mine lived experiences of PPPD from readily available online data from PPPD support and/or social media groups (see Chapter 5, 5.10.6).

2.7.5. Sleep

Sleep can facilitate neuro-plastic changes during vestibular rehabilitation (Whitney, Sparto & Furman, 2020); however, impaired sleep quality is usually considered to be due to acute vestibular illness, not PPPD. This therefore opens up a new avenue for future research providing that participants are referring to PPPD experiences and not any pre-existing condition affecting the vestibular system. This presents an interesting potential avenue for future research into the conditions and factors associated with PPPD. These findings suggest that in healthy participants the correlation may be mediated by anxiety and/or somatic amplification. Future research should look to explore relationships in the PPPD patient group to establish if this relationship translates to the patient community.

2.7.6. Quantifying Psychological Variables of Interest and Persistent Postural-Perceptual Dizziness

Many of the themes in Chapter 2 have been captured and/or alluded to in previous qualitative research paradigms (such as anxiety and the loss of self). However, adopting the IPA method has facilitated the candid disclosure of novel psychological factors related to PPPD. Such factors include poor sleep quality, guilt, depression, self-esteem and somatic amplification. Furthermore, the use of phenomenological methods has both established new variables whilst promoting a sensitivity to context (Yardley, 1993) within the research field offering an in-depth understand the lived experiences of the disorder. Thus, a quantitative approach was taken to seek quantitative data to support the relationships between PPPD symptoms and the newly established psychological variables. Data suggested anxiety and somatic amplification (and depending on the scale used, depression) uniquely predict PPPD scores, in the healthy student population. There are, however, significant indirect effects of sleep, general mental well-being, self-esteem and depression when mediated by anxiety. Taken together the mixed-methods data presented in Chapter 2 offers future research several promising avenues for PPPD investigations into previously unknown related psycho-social variables. Future research should look to explore the relationships between these psycho-social variables and PPPD symptoms in the PPPD patient group.

2.7.7. Using Interpretative Phenomenological Analysis with the Persistent Postural-Perceptual Dizziness Patient Group

A strength of the research collected in this Chapter is the application of the IPA method to the PPPD patient group. IPA has been a highly effective tool for encouraging discourse and the disclosure of highly sensitive health related information (Fox & Diab, 2015; Strickland, Worth & Kennedy, 2015). Adopting the tool allowed for deeply personal qualitative accounts of to be documented; accounts

shaped around experiences and individual life stories. As patients have often noted feeling dismissed or not listened to fully adopting a person-centred approach can be considered an ethical manner for interacting with patients (Seizier, Saywell, Terry, Taylor & Kayes, 2019); one that validates their experience. The success of these studies suggest that this tool is an effective knowledge elicitation tool when used with PPPD patient group. This may have clinical implications as the diagnosis of PPPD relies on the patient's presentation and previous history (Staab et al., 2017); a fundamentally qualitative interaction. Clinicians may be able to shape health related questions in an experiential story-based manner in order to enhance the quality of information provided by PPPD patients. Future research should look to explore the impact of the hermeneutic process on disclosure of symptoms in clinical settings.

2.8. Chapter 2: Summary

Living with PPPD can result in a debilitated patient with complex co-occurring illnesses that diminishes quality of life - though the lived experiences of people with PPPD are seldom probed in research (Guerraz et al., 2001; Mendel, Lutzen, Bergenius and Bjorvell, 1997; Sezier, Saywell, Terry, Taylor & Kayes, 2019). The aim of Chapter 2 was to explore the lived experiences of PPPD. In particular, it empathetically probed how individuals with a diagnosis of PPPD make sense of their symptoms and condition and the psycho-social impacts of the condition. The themes identified can generally be considered in terms of the following 5 motifs; identity loss, dismissal and non-belief, poor psychological well-being, out of body experiences and processes of sense-making. Shared themes included poor metal well-being, sensory overload, sleep impairments and PPPD not affecting television/movie watching. Poor sleep quality, poorer general mental well-being, somatic amplification, higher levels anxiety and depression, and lower levels of self-esteem were all shown to correlate with PPPD symptoms - though the magnitude of the linear relationship varies between the scales and constructs used. However, only anxiety and somatic amplification were significant predictors of PPPD when the PPPD is measured by the Visual Vertigo Analogue Scale (VVAS: Dannenbaum, Chilingaryan & Fung, 201. When PPPD is measured by the newer Niigata PPPD Questionnaire (NPQ; Yagi et al., 2019) anxiety, somatic amplification and depression were the only variables shown to significant predict PPPD symptoms. This leaves future researchers with promising avenues for future research.

Chapter 3: The Short-Term Effects of Optokinetic Stimulation on Markers of Visual Dependence

3.1. Introduction

Optokinetic stimulation can produce measurable changes in parameters associated with PPPD. Exposure to optokinetic patterns has been shown to reduce self-reported vestibular and psychological symptoms in the PPPD patient group (Pavlou, 2010). Optokinetic stimulation has also been shown to produce changes in markers of visual dependence in this group (Bronstein, 1995; Pavlou et al., 2012; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). Optokinetic stimulation techniques have recalibration effects that can affect the weighted contributions of the senses that facilitate postural control mechanisms (see Section 1, Chapter 1). This explains why optokinetic stimulation may alter levels of visual dependence. Recalibration effects typically occur over time; for example, over the typical 8-week rehabilitation programme (see Umphred & Lazaro, 2012). However, the optokinetic stimulation used with patients is often monotonous, unengaging and of low-quality meaning that not all patients are motivated to adhere to the rehabilitation programme (Meldrum et al., 2012; Whitney et al., 2006).

Little research has explored if optokinetic stimulation can produce short-term recalibration effects. Recent research has found that short-term exposure can affect visual dependence when captured using postural sway but not the Rod and Frame test (Van Ombergen et al., 2016). In PPPD patients, the researchers detected an increase in postural sway post optokinetic exposure. This may be early evidence of optokinetic stimulation having short-term recalibration effects. However, as this data shows an increase in sway, effects may be the result of a transient after-effect: the result visual motion destabilising postural control mechanisms. As, if the stimulus were to be an effective rehabilitation tool then it should reduce the postural marker of visual dependence . Thus, to date, it is not known if short term exposure to optokinetic stimulation can *reduce* visual dependence. Consequently, it is also not known if optokinetic stimulation can produce short-term positive visuo-vestibular recalibration effects and, if so, over what timeframes effects occur over.

Research from the sensory reweighting literature would suggest that humans can dynamically reweight sensory signals. For example, it is known that when driving in low-contrast situations such as fog or darkness, visibility is impaired (Snowden, Stimpson & Ruddle, 1998; Warren, Kay, Zosh, Duchon & Suhac, 2001). This reduces the reliability of the visual cue meaning that the driver must upweight vestibular and proprioceptive signals for more accurate velocity estimates

(Ramkhalawansingh, Keshavarz, Haycock, Shahab & Campos, 2016; Markkula et al., 2019) though confirmatory evidence of this is difficult to ascertain. Astronauts have also been shown to down weight proprioceptive signals during space flight (Carriot, Jamali & Cullen, 2015; Ganapathy, Da Rosa & Russomano, 2019). In relation to PPPD, patients report employing behaviours which facilitate the immediate down weighting of visual cues such as wearing sunglasses (see Chapter 2). Based on the interpretation of the pattern of results in this area, one may hypothesise optokinetic stimulation would produce dynamic short-term changes to sensory organisation. Thus, evidence from the sensory reweighting literature and lived experiences of PPPD may suggest that short-term exposure to an optokinetic stimulation may produce short-term effects on sensory organisation, importantly visual dependence.

3.1.1. Aims and Objectives of Chapter 3

The aim of Chapter 3 was test if short-term exposure to optokinetic stimulation can produce changes in markers of visual dependence – evidence of recalibration effects. These recalibration effects would be measured by the two commonly used laboratory measures of visual dependence where a reduction in each variable would indicate successful recalibration. As little is known about the timeframes optokinetic stimulation operate on, the researcher also aimed to explore the short-term timeframes effects occur on. Finally, as current optokinetic stimuli used in the rehabilitation of PPPD may be considered monotonous and unengaging (see stimuli presented in: Meldrum et al., 2012; Whitney et al., 2006), experiments were designed in a manner that would establish if multi-media technology could be used to produce recalibration effects and establish the most effective stimulus for doing so. Thus, a supplementary aim of Chapter 3 was to establish if engaging multi-media technology can be used to facilitate optokinetic stimulation. The objectives of this Chapter were to establish if short-term exposure, in the form of different engaging multi-media stimuli, can produce a short-term reduction in traditional laboratory measures of visual dependence. This was conducted in the student population to avoid wasting patients' time and to establish which stimuli may be the most promising rehabilitation tool – evident by the most pronounced reduction in levels of visual dependence.

3.2. Experiment 1: Using Novel Visual Media to Produce Short-Term Reductions in Markers of Visual Dependence

The first study of this Chapter had three aims. First, it sought to replicate previous findings that suggest that exposure to short-term optokinetic stimulation can produce changes in the postural, but not perceptual marker of visual dependence (Guerraz et al., 2001; Van Ombergen et al., 2016). Second, it aimed to establish if novel 'fun' optokinetic stimulation can be used to produce recalibration effects (i.e. reduce the dependence on vision). Third, it aimed to establish which t*ype* of optokinetic stimulation was the most effective at producing recalibration effects. As such, the researcher designed an experiment that would allow for the comparison of different types of engaging optokinetic stimuli such as virtual video games and movies.

Based on previous research, it was hypothesised that short-term exposure to optokinetic stimulation would *decrease* the postural, but not perceptual, marker of visual dependence (Guerraz et al., 2001; Van Ombergen et al., 2016). It was also hypothesised that the novel multi-media optokinetic stimuli would be effective in producing recalibration effects, in line with similar stimuli used in the past (Bronstein, 1995; Pavlou et al., 2012; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). There may, however, be differences in the efficacy of each stimulus.

3.2.1. Experiment 1: Methodology

3.2.1.1. Participants

Ninety-one participants were recruited through an online university participant panel for undergraduate psychology students (63 females; 28 males) with a mean age of 20.07 (SD: 4.22). Participants were asked to refrain from drinking alcohol for 24 hours before the study and were not eligible to take part in the study if they were currently taking anti-anxiety, anti-depression or vestibular suppressant medications. One participant was removed due to reporting PPPD (N = 90). All participants were randomly allocated to one of four visual stimuli groups. For group specific stimuli properties see section 3.2.1.2.3. Randomisation occurred using a digital randomisation tool where participant numbers were randomly allocated to one of the four groups. One participant was placed into the control condition for fears of visual motion producing PPPD like symptoms. This may have implicated the randomisation process. Twenty-three participants were allocated to the control condition (22 randomly, 1 due to concerns of PPPD symptoms). Twenty-two participants were randomly allocated to the movie condition, 22 to the active virtual video game condition and 24 to the passive virtual video game condition.

3.2.1.2. Apparatus and Stimuli

All stimuli were presented on a 55-inch LG high-definition organic light emitting diode television. Stimuli were viewed binocularly at a distance of 100cm. Manual responses, where necessary, were made by a Logitech smart accessible ball mouse held in both hands.

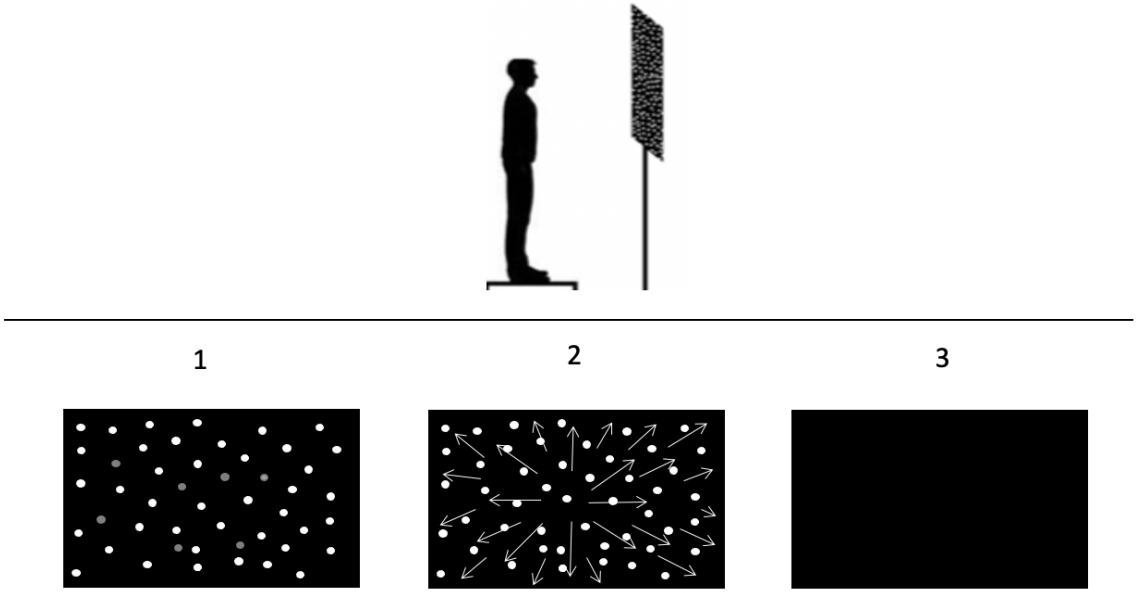

Figure 3.1. Graphical depiction of the postural sway equipment and the three visual conditions presented to participants whilst on the Wii Board.

3.2.1.2.1. Standardised Instructions

All participants were given standardised instructions in both written and verbal format. The researcher read aloud the instructions to ensure all participants received the same information. Participants were first asked to complete the postural sway test followed by the Rod and Frame test. Participants were then asked to view or interact with their randomly allocated visual stimuli. Participants were required to stand for all tasks. Details of each task and stimuli are presented below.

3.2.1.2.2. Postural Marker of Visual Dependence - Postural Sway

The Vection Quotient represents the degree to which the viewer is destabilised by external world motion (Guerraz et al., 2001). A Vection Quotient was formed by recording postural data in response to different visual information conditions. These visual conditions provided static information (typical vision), visuo-vestibular conflict (visual motion without vestibular motion cues) or no visual information. Data in response to these conditions was collected by placing the participant on a Nintendo Wii Board, using software to record postural data. The Vection Quotient was formed by capturing sway during visuo-vestibular conflict (condition 2), divided by sway present when visual

information is not manipulated (condition 1). This gave an estimate of how the user resolves visuo-vestibular conflict and can effectively suppress the inaccurate visual cue.

Three visual conditions were custom built using python code. In each condition a white limited lifetime dot cloud (maximum of 1240 dots) was presented against a black background (dot size = 1.14 degrees). Dots spawned at a random x, y, z coordinate in three-dimensional cubic space. In the congruent condition, dots spawned and respawned at a random x, y, z coordinate every 100ms. For the incongruent condition, dots spawned and then oscillated on the z dimension (anterior-posterior) as a function of a sinewave (peak velocity of 0.3 hz; amplitude of 100% of the viewing distance). Dots oscillated forwards and backwards. Visual conditions are presented in Figure 2.1.

A Nintendo Wii Fit Balance Board was Bluetooth connected to a Windows laptop running the opensource BrainBlox Neuromechanics Software Quotient (Cooper, Siegfried, Ahmed & 2014). Data was collected for each of the three visual conditions. This software records raw sway path lengths by detecting changes in centre of foot pressure when a user is stood on the board. Changes in foot pressure were calculated by measuring the distance between the first detected point (a) and the second detected point (b). This was calculated between all point-to-point changes. A mean sway path length was then calculated each of the three visual conditions (congruent/static vision, incongruent/visual motion and none). Participants were required to view each visual stimulus for 20 seconds whilst postural sway data was recorded.

The Vection Quotient was then computed by dividing Y axis only conflict (visual motion) condition sway by the congruent (static visual information) Y axis sway path length. This is done for each individual participant. Note, healthy controls typically display Vection Quotients of approximately 2.36, in vestibular patients this is approximately 2.74 and in PPPD patients approximately 3.93 (Guerraz et al., 2001).

3.2.1.2.3. Perceptual Marker of Visual Dependence - The Rod and Frame Test of the Subjective Visual Vertical

The Rod and Frame Test is a classic psychophysical test that assesses the subjective visual vertical (SVV) or rather, the perceived upright vertical in relation to the true earth-gravitational upright (Asch & Wilkin, 1948a; Asch & Wilkin, 1948b). The test itself is used to establish the contribution of visual and gravitational (vestibular) cues to set the perception of the earth-right vertical.

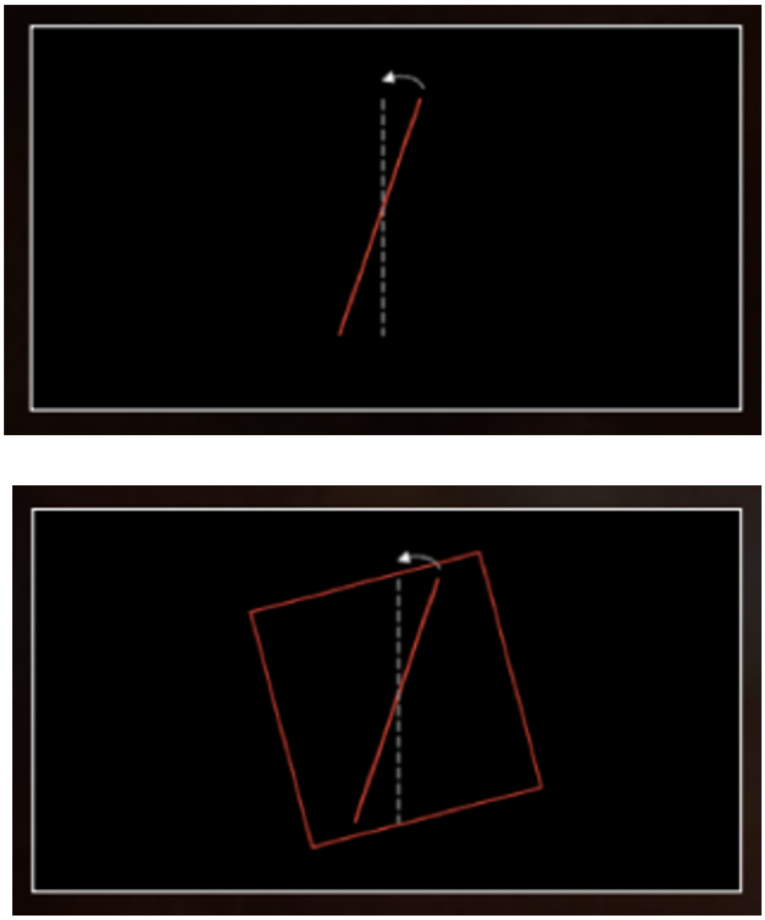

Figure 3.2. The Rod and Frame test of the subjective visual vertical (not to scale). Top: The rod alone condition. This shows the orientation that the rod is presented at and the true earth-right vertical which it should be returned to. Bottom: This illustrates a rod and frame condition (here the frame is tilted left) where the rod must again be orientated to the true earth vertical.

In this test the participant is placed in a dark environment free from any light sources and asked to orient a rod to their perceived gravitational upright. In contemporary research this line is usually presented on a large screen that does not emit any other light sources. A red rod is presented at the centre of the screen randomly orientated and it is the user's job to orient it to their perceived vertical. In some trials, participants view a rod in isolation which they must orient it to their perceived vertical. However, in other trials the rod is presented inside a much larger red frame that is titled to the left or right. Here, the participant must try and orient the rod to their perceived vertical and supress the unreliable information from the visual reference frame.

The users' Rod and Frame effect quantifies their level of visual dependence. A general Rod and Frame effect was formed by taking the left and right tilted frame conditions and (having established they do not significantly differ) collapsing the absolute (or unsigned) orientations of the rod and subtracting the mean orientation of the rod when no visual reference frame is present. For most people

this usually results in a rod oriented to the direction of the tilted reference frame, which in healthy controls this is approximately 2 degrees (Böhmer & Mast, 1999). Vestibular groups, including the PPPD patient group show absolute (unsigned) deviations larger than controls, around 5+ degrees (Hafström, Fransson, Karlberg & Magnusson, 2004a; Hafström, Fransson, Karlberg & Magnusson, 2004b). Note, unsigned deviations are used to show the general effect rather than a specific bias to the left or right.

In this experiment, a solid red rod (0.30 cd/m^2) was presented to the participant in the centre of the screen with a random orientation offset between -25 and 25 degrees. On 24 trials the rod was housed in a red frame (width = 24 degrees, height = 24 degrees). On 12 trials the orientation of the frame was manipulated 15 degrees clockwise and on 12, 15 degrees anti-clockwise. The psychophysical method of adjustment was used. A general Rod and Frame effect was then computed for all observers. This variable was formed by collapsing the absolute left and right frame effects and subtracting the mean rod only estimates from the collapsed frame effect. This gives an estimate of how much the observer relies on the visual reference frame for setting the gravitational vertical.

3.2.1.2.4. Visual Stimuli

Control Stimuli: A database of images with subjective ratings of visual discomfort was accessed (Pennachio & Wilkins, 2015). Images consisted of art, buildings or shapes. Ninety images were randomly selected from the lowest scoring 10% of images indicating these images were the least visually uncomfortable. Images were presented for 10 seconds each.

Movie: Situations known to trigger vestibular symptoms were recorded using a head-mounted camera whilst walking. Situations include; riding an escalator and navigating busy shop aisles. Scenes were complemented with clips of translating virtual corridor environments with high spatial frequencies. Each clip lasted 30 seconds.
Active Virtual Video Game: The racing simulator Mario Kart 8 run using the Nintendo Wii U console. Players completed the Electrodrome track followed by the Rainbow Road track without virtual opponents. Players navigated the vehicle using the consoles controller.

Passive Virtual Video Game: Recorded footage of Mario Kart 8 was passively shown to the observer. Participants were matched to a participant in the active video game condition and shown their gameplay footage. If no new footage was available, the participant was placed in the active condition and their gameplay recorded (n = 2).

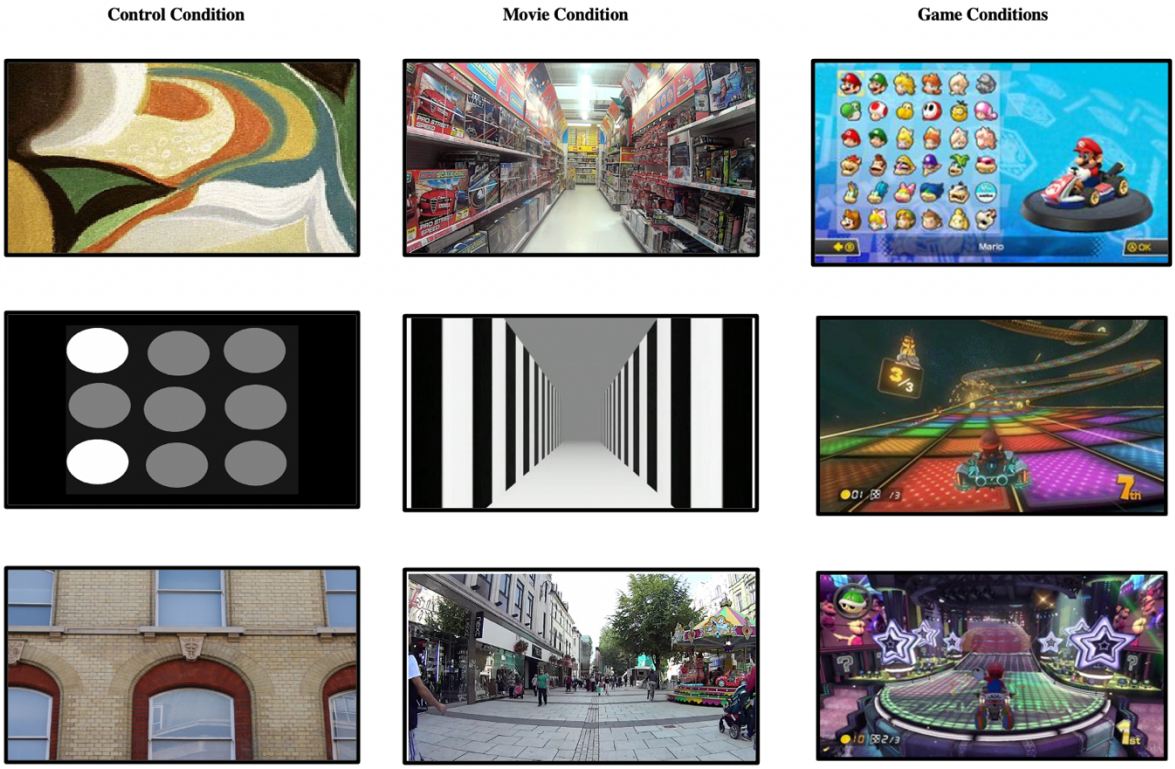

Figure 3.3. Experimental stimuli for experiment 1. Left column: typical stimuli in the control condition, where the first image is a picture of art, the second a shape-based image and the third an image of a building. Column 2: typical stimuli used in the movie condition, where the first image is of Toys R Us, the second the virtual striped corridors and the third walking in a crowded street. Column 3: virtual video game stimuli where the first image is the avatar selection page and the second and third images are of the racetracks used in the active and passive virtual video game conditions.

3.2.1.2.5. The Visual Vertigo Analogue Scale (VVAS)

The Visual Vertigo Analogue Scale (VVAS; Dannenbaum, Chilingaryan & Fung, 2011) identifies whether an individual experiences the characteristic symptoms of PPPD (formerly visual vertigo: see General Introduction Chapter). The full psychometric properties of the VVAS can be found in section 2.6.1.2.1 of Chapter 2.

3.2.1.3. Study 1: Procedure

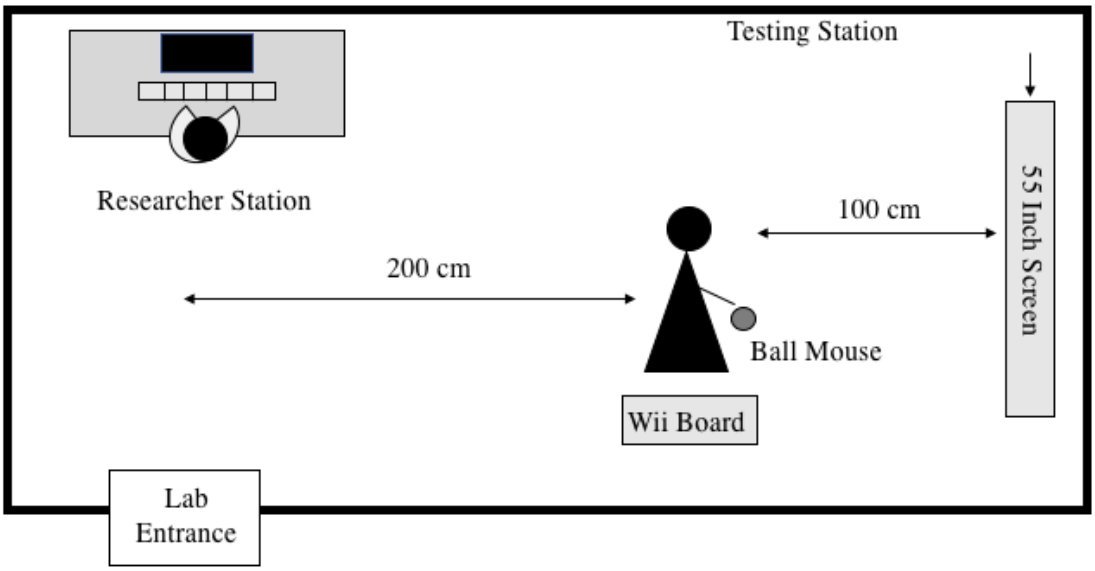

Figure 3.4. Graphical depiction of the laboratory set up.

Participants were invited into a psychophysical laboratory. The laboratory set up is presented in Figure 3.4. Participants were greeted and given verbal and written study information. If participants were comfortable proceeding they were asked to sign an informed consent form. This document also asked participants to provide key demographic details such as age and gender. The researcher remained in the room (at a distance of 200cm away from the participant) to control stimuli and troubleshoot any technology issues.

Before participants began the experiment, they were asked if they would like to take part in a short online study related to the study (Chapter 4). If participants agreed to complete the questionnaire they were awarded an additional course credit. This questionnaire was the Visual Vertigo Analogue Scale (VVAS: Dannenbaum, Chillingaryan & Fung, 2011) and was completed digitally at the researcher station.

Participants then progressed to the main experimental testing phase. The researcher read aloud standardised instructions to participants to explain how the tasks work and how they should respond. These instructions stated that the participant was required to keep their attention to the centre of the screen at all times during testing, to stand with feet apart and arms down by sides during the Wii board task, and to use the roll ball mouse with both hands. All tasks were undertaken with participants in a standing position. Participants stood barefooted on a Wii Balance Board and passively viewed, static, translating and then no visual information, for 20 seconds each. Participants then stepped off

the Wii board to orient a rod, with and without a frame, using a ball mouse held in both hands. Participants then viewed (or played) the visual stimuli allocated to them for 15 minutes. Afterwards, participants completed the Wii postural task, Rod and Frame task and a final Wii posture task in that order. It took approximately 1-2 minutes from participants ending on task to starting another. If participants required a longer break duration, they were welcome to do so. See Figure 3.5 for a diagram depicting the experimental procedure. All participants were thanked, debrief and awarded course credit.

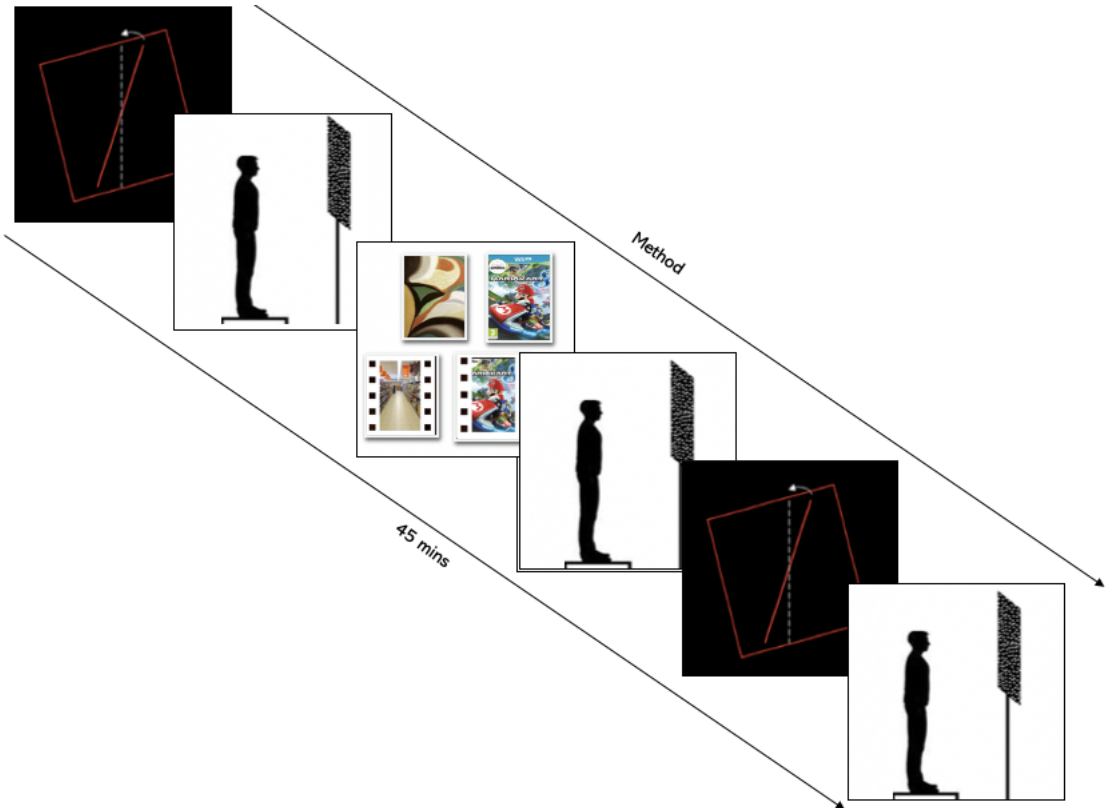

Figure 3.5. Graphical depiction of the experimental paradigm for Experiment 1.

Table 3.1

Experiment 1: Descriptive statistics.

				Visual Dependance Measures							
		n	Time	Rod and Frame Test				Vection Quotient			
				Valid Data	Missing Data/ Outliers	M	SD	Valid Data	Missing Data/ Outliers	M	SD
Condition	Control	23	Pre Exposure	22	1	1.88	1.14	20	3	1.63	0.36
			Post Exposure	22	1	1.79	1.14	22	1	1.61	0.51
			Post Exposure 2	–	–	–	–	19	4	1.56	0.37
	Movie	22	Pre Exposure	21	1	1.95	1.48	20	2	1.73	0.53
			Post Exposure	21	1	1.73	1.07	18	4	1.55	0.46
			Post Exposure 2	–	–	–	–	18	4	1.33	0.42
	Active Game	22	Pre Exposure	20	2	1.52	1.02	19	3	1.73	0.44
			Post Exposure	20	2	1.58	0.93	20	2	1.65	0.54
			Post Exposure 2	–	–	–	–	20	2	1.88	0.7
	Passive Game	24	Pre Exposure	22	2	1.45	1.34	24	0	1.69	0.45
			Post Exposure	22	2	1.36	1.13	24	0	1.58	0.45
			Post Exposure 2	–	–	–	–	23	1	1.56	0.46

3.2.1.4. Planned Statistical Analyses

ANCOVA models were pre-planned for the analysis of data, where pre-scores on the outcome measures would be entered into the models as covariates. The Median Absolute Deviation (MAD) or Modified Z (Iglewicz & Hoaglin, 1993) was selected to detect outliers on the outcome measures of visual dependence. Any score +/- 3.5*MAD were considered outliers and removed from the analysis.

3.2.2. Experiment 1: Results

The measures of visual dependence were the Rod and Frame effect and Postural Sway – Vection Quotient. A more pronounced effect on the Rod and Frame test indicates a larger reliance on the visual reference frame for the setting the gravitational vertical. A larger Vection Quotient suggests that the observer is more destabilised by motion on the retina. A reduction in either the postural or perceptual marker would be indicative of a short-term recalibration effect of optokinetic stimulation techniques.

3.2.2.1. Descriptive Statistics

The mean and standard deviations of each visual dependence measure at each timepoint are presented in Table 3.1. This table also documents sample sizes and valid data and missing data due to technology failure and outlier detection. The data set consists of 63 females and 28 males with a mean age of 20.07 (SD: 4.22).

Inspection of the data suggests that all experimental groups show levels of visual dependence that are to be expected in a generally healthy cohort. Healthy controls typically display a Rod and Frame effect of approximately 2 degrees (Böhmer & Mast, 1999). Vestibular patients and PPPD patients typically group show deviations of 5+ degrees (Hafström, Fransson, Karlberg & Magnusson, 2004a; Hafström, Fransson, Karlberg & Magnusson, 2004b). Further inspection of the data shows relatively normal levels of visual dependence across all experimental groups when measured by the Rod and Frame test. Similar findings are evident in participants' Vection Quotients. Visual motion has a destabilising effect on balance control mechanisms in humans capable of visual perception. As such, Vection Quotients are always greater than 1. Healthy controls typically display Vection Quotients of approximately 2.36, in vestibular patients this is approximately 2.74 and in PPPD patients approximately 3.93 (Guerraz et al., 2001). The data also suggest that the Vection Quotients detected across all experimental groups are typical of healthy individuals free from vestibular compromise and/or PPPD. Note, Vection Quotients in this sample do not surpass 1.73 prior to exposure to an optokinetic stimulus. This is lower than Guerrez et al. (2001) would suggest for health controls;

however, given the mean age of participants in their study was 42 and 20.07 in the current study, this difference in Vection Quotient is likely explained by age.

3.2.2.2. Inferential Statistics

Data are depicted as line graphs with 95% Confidence Intervals.

3.2.2.2.1. Experiment 1: Rod and Frame Analyses

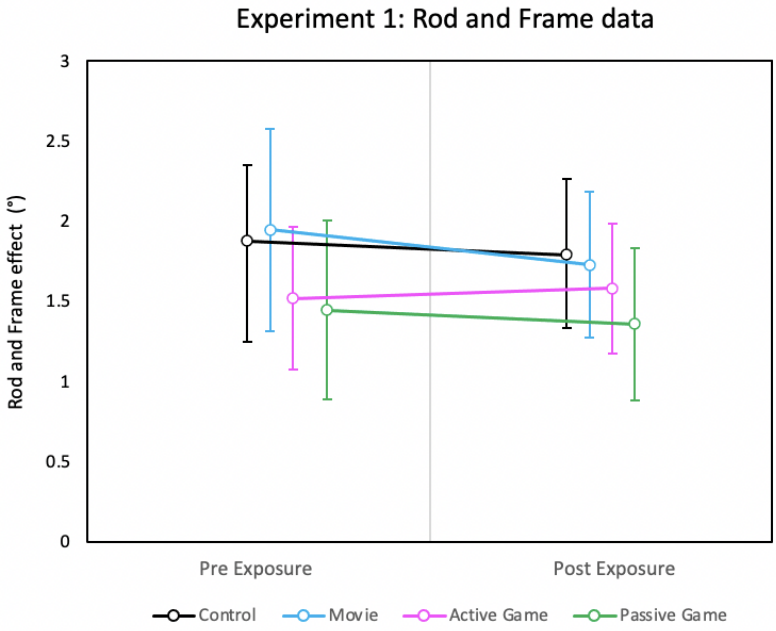

Figure 3.6. Rod and Frame effect results from Experiment 1. Mean Rod and Frame effect is plotted at time 1 (pre-exposure) and time 2 (post-exposure). Error bars represent 95% confidence. Each condition is presented individually; top left is the control condition (coloured black), top right is the movie condition (coloured blue), bottom left is the active virtual video game condition (coloured pink) and bottom right is the passive virtual video game condition (coloured green).

Results for the Rod and Frame test at time points one (pre-exposure) and two (post-exposure) can be found in Figure 2.5. Absolute left and right frame effects were compared using a paired samples t-test. Baseline (time 1: $t(88) = 0.85$. $p = 0.40$, $d = 0.09$) and post-exposure (time 2: $t(87) = -0.27$, $p = 0.79$, $d = -0.03$) and showed no significant difference. Frame effects were therefore collapsed to represent a general Rod and Frame effect. Groups did not significantly differ on the pre Rod and Frame effects, $F(3, 82) = 0.51$, $p = 0.68$, $\eta_p^2 = 0.02$). Therefore, an Analysis of Covariance was conducted on post-exposure Rod and Frame effects, whilst controlling for baseline (pre-exposure) Rod and Frame effects. The covariate, pre-exposure Rod and Frame effect, was significantly related to the post exposure effect; $F(1, 79) = 96.57$, $p = <.001$, $r = 0.75$); however, no significant effect of [visual stimuli] group was detected on post exposure Rod and Frame effects when controlling for baseline

pre-exposure effects; $F(3, 79) = 0.32, p = 0.81, \eta_p^2 = 0.01$). The data indicated no significant effect of visual motion on the reliance of a visual reference frame for the setting of the gravitational vertical. This means that the data fail to suggest that the perceptual marker of visual dependence can be reduced after short-term exposure to optokinetic stimuli. This is in line with the hypothesis and previous research (Van Ombergen et al., 2016).

3.2.2.2.2. Experiment 1: Postural Sway: Vection Quotient Analyses

Results for the postural sway (Vection Quotient) at time points one (pre-exposure), two (post-exposure 1) and three (post-exposure 2) are plotted in Figure 2.6. Two Analysis of Covariance's were conducted on the data. For each analysis the time 1 (pre-exposure) Vection Quotient was entered into the model as the covariate. Groups did not significantly differ on the covariate; $F(3, 79) = 0.22, p = 0.88, \eta_p^2 = 0.01$. The first analysis compared the Vection Quotient between the four visual groups, at time 2 (post-exposure 1), whilst controlling for baseline (pre-exposure) Vection Quotients. The covariate, time 1 (pre-exposure) Vection Quotient, was significantly related to the Vection Quotient at time 2 (post-exposure 1); $F(1, 76) = 6.68, p = 0.01, r = 0.28$; however, no significant difference between the four visual groups was detected; $F(3, 76) = 0.33, p = 0.80, \eta_p^2 = 0.01$.

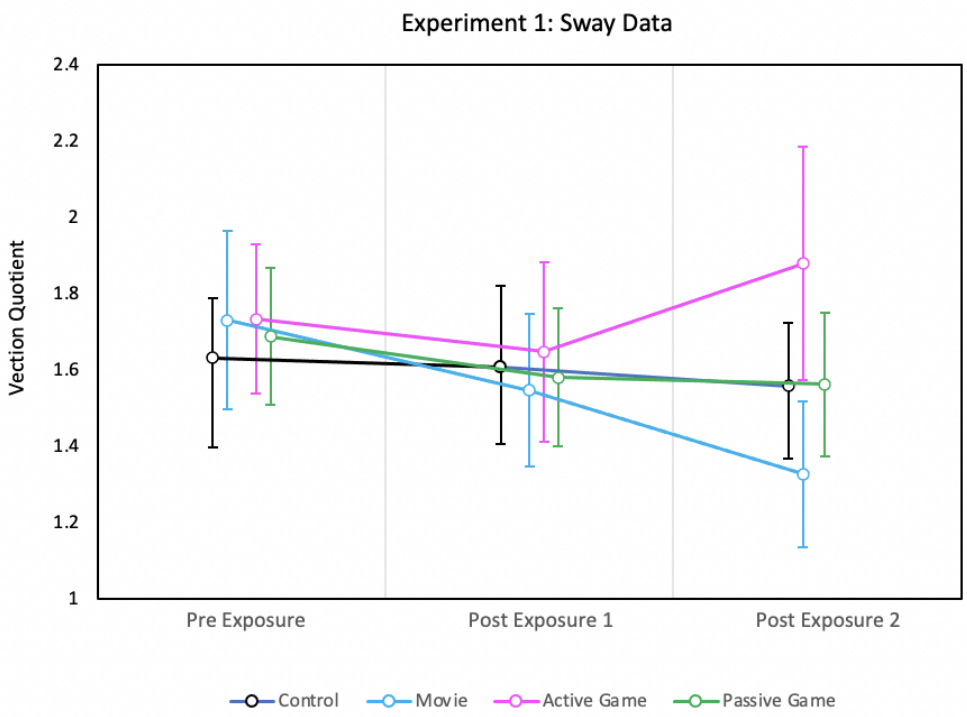

Figure 3.7. Postural sway Vection Quotient results from Experiment 1. Mean Vection Quotients are plotted for pre-exposure, post exposure 1 and post exposure 2. The quotient itself is formed by dividing the Y dimension sway path length detected in the visual motion (incongruent) condition by the no vision condition. The mean therefore represents a proportion that indicates the degree to which the viewer is destabilised by visual motion. Error bars represent 95% confidence. Conditions are coloured and a key is presented; the control condition is coloured black, the movie condition coloured blue, the active virtual video game condition is coloured pink and the passive virtual video game condition is coloured green.

The second analysis compared the Vection Quotient at time 3 (post-exposure 2) between the four groups, whilst controlling for baseline (pre-exposure) Vection Quotient. The covariate (pre-exposure) was not significantly related to the Vection Quotient present at time 3 (post exposure 2); $F(1, 70) = 1.42, p = 0.24, r = 0.13$, however a marginally significant effect was detected; $F(3, 70) = 2.51, p = 0.07, \eta_{p^2} = 0.10$. Post hoc comparisons, with Tukey corrections applied, revealed a significant difference between the movie and active virtual video game conditions only: $t(37) = 2.72, p = 0.04, d = 0.85$ (Bonferroni corrected comparison; $p = 0.05$). The results are in line with previous research that suggest visual motion can have a short-term effect on the postural marker of visual dependence. What the data may suggest is that the immediate change at time 2 may be thought of as a transient postural after- effect. However, what appears to be the true effect occurs 15 minutes after this (at time point 3), where a sustained reduction in the postural marker of visual dependence is visible when viewers were, passively, exposed to an optokinetic movie.

3.2.3. Experiment 1: Interpretations and Conclusions

The findings presented above suggest that the postural, but not perceptual, marker of visual dependence can be reduced by short-term exposure to an optokinetic visual stimulation technique. The data also show that passively viewing a movie is the most effective method of administering optokinetic stimulation, evidenced by the largest reduction in the Vection Quotient. These finding are congruent with previous literature that have suggested visual motion movies can reduce the postural marker of visual dependence (Van Ombergen et al., 2016; Pavlou, 2010). The data may suggest is that the immediate change at time 2 may be thought of as a transient postural after- effect. However, what appears to be the true effect occurs 15 minutes after this (at time point 3), where a sustained reduction in the postural marker of visual dependence is visible when viewers were, passively, exposed to an optokinetic movie. The data also suggest virtual video games are less efficacious tools for reducing the postural marker of visual dependence in this cohort.

3.3. Experiment 2: Increasing the Duration of Optokinetic Stimulation

The aim of Experiment 2 was to replicate the effect found in Experiment 1 using the most efficacious visual stimuli. This experiment was designed to confirm the preliminary findings that short-term exposure to an optokinetic stimulus (in the form of a movie) may reduce the postural, but not perceptual, marker of visual dependence. A finding in-line with previous research (Van Ombergen et al., 2016). However, as the effects detected in Experiment 1 were small this second experiment was designed with an increased duration of optokinetic stimulation. This was done in order to promote the manifestation of effects. Finally, as using a quotient as a main variable for analysis can introduce unsystematic variance (noise) to data, the amount of postural data collected was also increased.

It was hypothesised that increasing the amount of time the observer views the optokinetic stimulus may produce a larger effect on the postural marker of visual dependence. Furthermore, it was hypothesised that short-term exposure to an optokinetic stimulus, in the form of a movie, would reduce the postural but not perceptual marker of visual dependence.

3.3.1. Experiment 2: Methods

3.3.1.1. Participants

Fifty participants were recruited through the same psychology Undergraduate participation panel (40 identified as female; 10 as male). The mean age was 21.02 (SD: 4.57). The same inclusion/exclusion criteria applied in Experiment 1 was used. Twenty-five participants were randomly allocated to the control group and 25 to the movie condition. The same randomisation process as stated in Experiment 1 occurred. Two participants withdrew from the experiment due to sensations of disequilibrium and vertigo, therefore N = 48 control = 24; movie = 24). Both participants were female and in the control condition with no retinal motion. All participants were awarded course credit for participation.

3.3.1.2. Stimuli and Procedure

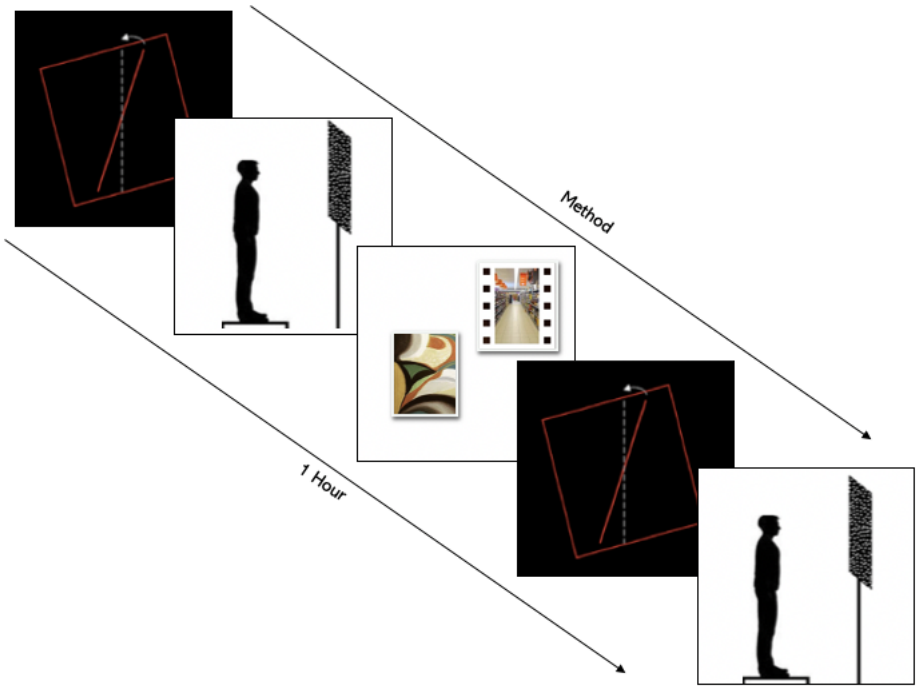

Figure 3.8. Graphical depiction of the experimental paradigm for Experiment 2.

Participants were invited into a psychophysical laboratory. The laboratory set up remained the same as in experiment one (see Figure 3.4). In line with the previous experiment, participants were greeted and given verbal and written study information. If participants were comfortable proceeding they were asked to sign an informed consent form and provide demographic information (age and gender). The researcher remained in the lab (200cm away from the participant) during testing.

As in Experiment 1, participants were asked if they would like to complete a short digital questionnaire probing their experiences with dizziness (VVAS: Dannenbaum, Chillingaryan & Fung, 2011) as part of another study (see Chapter 4) and was completed digitally at the researcher station.

Participants then progressed to the main experimental testing phase. Participants were randomly allocated to either the control or movie condition and informed which stimulus they would view. The researcher read aloud standardised instructions to participants to explain how the tasks work and how they should respond. The instructions were the same as those presented to participants in experiment 1; however, in these instructions, participants were asked to view their stimulus for 30 minutes. These instructions stated that the participant was required to keep their attention to the centre of the screen at all times during testing, to stand with feet apart and arms down by sides during the Wii board task, and to use the roll ball mouse with both hands.

All tasks were undertaken stood up. Participants stood barefooted on a Wii Balance Board and passively viewed, static, translating and then no visual information, for 20 seconds each. This was repeated 3 times, consecutively with no break. This resulted in 60 seconds worth of physical sway data for each of the three visual conditions Participants then stepped off the Wii board to orient a rod, with and without a frame, using a ball mouse held in both hands. Participants then viewed their randomly allocated visual stimuli for 30 minutes. In each condition, the original 15-minute stimulus from Experiment 1 was shown to the observer twice. Note, attention and gaze were not monitored. Afterwards, participants completed the Wii postural task and Rod and Frame task, in that order. It took approximately 1-2 minutes from participants ending on task to starting another. If participants required a longer break duration, they were welcome to do so. See Figure 3.7 for a diagram depicting the experimental procedure. All participants were thanked, debrief and awarded course credit for their participation. See Figure 3.8 for a graphical depiction of the procedure for Experiment 2.

3.3.1.3. Planned Statistical Analyses

ANCOVA models were pre-planned for the analysis of data, where pre-scores on the outcome measures would be entered into the models as covariates. The Median Absolute Deviation (MAD) or Modified Z (Iglewicz & Hoaglin, 1993) was selected to detect outliers on the outcome measures of visual dependence. Any score +/- 3.5*MAD were considered outliers and removed from the analysis. Data are depicted as line graphs with 95% Confidence Intervals.

3.3.2. Experiment 2: Results

3.3.2.1. Descriptive Statistics

The mean and standard deviations of each visual dependence measure at each timepoint are presented in Table 3.2. This table also documents sample sizes and valid data and missing data due to technology failure and outlier detection. The levels of visual dependence detected are typical for healthy controls free from vestibular comprise and/or PPPD (Böhmer & Mast, 1999; Hafström, Fransson, Karlberg & Magnusson, 2004a; Hafström, Fransson, Karlberg & Magnusson, 2004b; Guerraz et al., 2001). Values may be interpreted as lower than those reported in the literature; this is likely due to the age of the sample.

Table 3.2
Experiment 2: Descriptive statistics.

				Visual Dependance Measures							
		n	Time	Rod and Frame Test				Vection Quotient			
				Valid Data	Missing Data/ Outliers	M	SD	Valid Data	Missing Data/ Outliers	M	SD
Condition	Control	25	Pre Exposure	24	1	1.66	1.15	23	3	1.89	0.52
			Post Exposure	21	4	1.32	0.99	20	5	1.80	0.38
	Movie	25	Pre Exposure	23	2	1.32	1.04	23	2	1.90	0.53
			Post Exposure	24	1	1.18	0.97	22	3	1.84	0.55

3.3.2.2. Inferential Statistics

The mean Rod and Frame effect both pre and post exposure were used as the main dependant variable for the Rod and sway analysis. For both measures the pre-score was used as a covariate. The mean time 1 (pre-exposure) and time 2 (post-exposure) Vection Quotient from the three estimates was used as the main dependant variable for sway analyses weighted for the number of Vection Quotient estimates. Data are depicted as line graphs with 95% Confidence Intervals.

3.3.2.2.1. Experiment 2: Rod and Frame Analyses

Results for the Rod and Frame effects from Experiment 2 can be found in Figure 2.8.

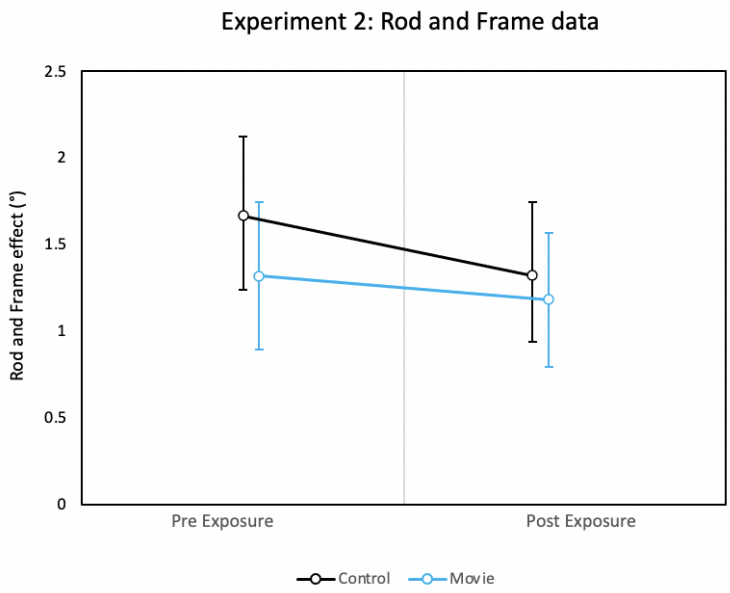

Figure 3.9. Rod and Frame effect results from Experiment 2. The mean Rod and Frame effect is plotted at time 1 (pre-exposure) and time 2 (post-exposure). The mean represents the average Rof and Frame effect. Error bars represent 95% confidence. Each condition is presented individually. The control condition is coloured black and the movie condition blue.

Rod and Frame effects were collapsed as no significant difference between absolute left and right frame effects were visible at time 1 (pre-exposure), $t(49) = -1.54$, $p = 0.13$, $d = -0.22$, or time 2, $t(45) = 0.02$, $p = 0.99$, $d = <.001$. The time 1 (pre-exposure) Rod and Frame effect was entered into an ANCOVA model as covariate, with the model comparing the two groups' time 2 (post exposure) Rod and Frame effects when controlling for baseline pre-exposure effects. Groups did not significantly differ on the covariate; $U = 32.00$, $p = 0.32$, Rank-Biserial Correlation = -0.17. Note, the Mann Whitney U test is used as data were not normally distributed. Baseline (pre-exposure) Rod and Frame effect was significantly related to the post-exposure effect, $F(1, 40) = 34.38$, $p = <.001$, $r = 0.68$; however, no significant difference between groups was detected when adjusting for the covariate; $F(1, 40) = 0.62$, $p = 0.44$, $\eta_p^2 = 0.02$. This affirmed the initial hypothesis and was in line with previous research, including that of Experiment 1.

3.3.2.2.2. Experiment 2: Postural Sway – Vection Quotient Analyses

Results for the Postural Sway Vection Quotients from Experiment 2 can be seen in Figure 2.9.

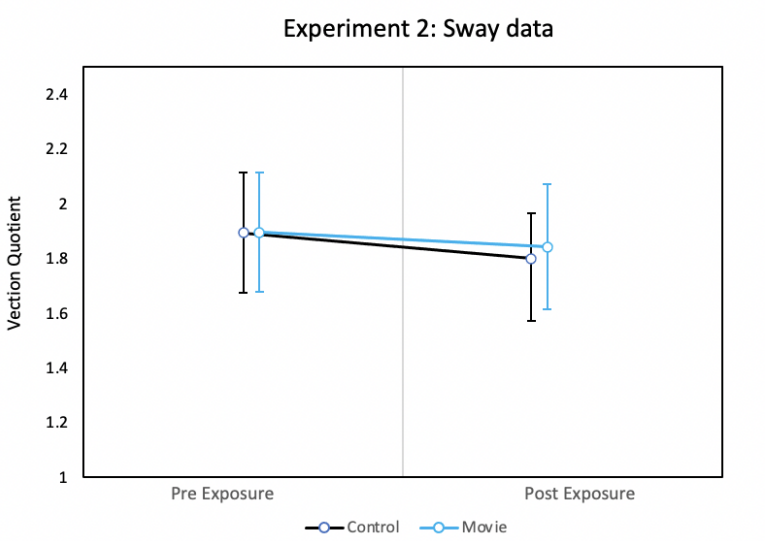

Figure 3.10. Postural sway Vection Quotient results from Experiment 2. Mean Vection Quotients are plotted at time 1 (pre-exposure) and time 2 (post-exposure). Error bars represent 95% confidence. The Vection Quotient plotted is the average Vection Quotient from up to 3 estimates. The control condition is coloured black and the movie condition coloured blue.

The average Vection Quotient at time 1 (pre-exposure) was entered into an ANCOVA analysis model as the covariate. The two groups did not significantly differ on the covariate: $U = 268.00, p = 0.74$, Rank-Biserial Correlation = -0.06. Note, the Mann Whitney *U* test was used as, again, data were not normally distributed. The average Vection Quotient at time 1 (pre-exposure) was significantly related to the average Vection Quotient at time 2 (post-exposure): $F(1, 38) = 38.34, p = <.001, \eta_p^2 = 0.50, r = 0.70$, however no significant difference was detected between the two groups when controlling for baseline (pre-exposure) Vection Quotients: $F(1, 38) = 0.36, p = 0.56, \eta_p^2 = 0.01$, when weighted for the number of observations. This finding did not align with previous findings and/or previous research (Van Ombergen et al., 2016). This may be due to using the same visual motion stimulus twice.

3.3.3. Experiment 2: Interpretations

Data suggest that when exposed to 30 minute of optokinetic stimulation, the stimulus doe not produce a recalibration effect. Thus, these data fail to replicate the initial finding of Experiment 1. This may be due to a limitation of the study – the use of the same optokinetic stimulus presented to the participant twice. The brain may be able to anticipate the motion patterns on the retina and thus impede the efficacy of the stimuli – as participants knew to expect certain motion patterns at certain points.

Alternatively, the participants may have simply become bored. The task requires them to stand for 30 minutes, which is actually an unusually long time to be stood in the same location. Furthermore, if participants did not find the stimulus to be engaging they may not have attended to it. These limitations informed the design of a third experiment using novel motion patterns (that are user centred and highly engaging) to explore such optokinetic stimulation could reproduce the findings of experiment 1. Furthermore, participants qualitative comments during testing indicated that the visual motion movie was not very intense and was monotonous. Thus, the researcher aimed to find a visual stimulus that would be both visually intense, with unpredictable motion patterns, and enjoyable. It was hypothesised exposing participants to unpredictable optic flow patterns would result in a reduction in the postural marker of visual dependence, once more.

3.4. Experiment 3.1: Pilot Study

Informal interactions with participants, colleagues and peers revealed that they experienced sensations of vection, disequilibrium and/or dizziness (usually in the cinema). Anecdotes for participants in experiment 1 and 2 had indicated a number of films that had resulted in dizziness; however, three motion picture films were consistently reported. These films were the live action movies *Avatar* and *Gravity* and the animated movie, *Spider-Man: Into the Spider-Verse*. In order to establish which motion picture was the most visually intense online pilot study (N = 292) was conducted. In thus study undergraduate participants viewed a 30 second clip of each of aforementioned movies (for course credit). All participants identified as either female (n = 255) or male (n = 37) but were free to identify however they saw fit. Wider demographic data was not collected.

Participants were asked to rate each motion picture on how much it made them feel subjectively a) dizzy b) sick c) uncomfortable and d) nauseous on an analogue scale between 0 (not at all) and 100 (very much so). All missing data were scored 0 in line with typical dizziness measures (see Dannenbaum, Chillingaryan & Fung, 2011; Kennedy, Lane, Berbaum & Lilienthal, 1993). The results of Experiment 3.1 can be found in Table 3.3. Gravity was chosen as the stimulus for Experiment 3 due to having the highest subjective ratings on all four visuo-vestibular factors. No inferential statistics were conducted on data as statistical significance was not the goal of this study.

Table 3.3.

Means and standard deviations for subjective vestibular experiences during the passive viewing of three motion picture films

Motion Picture	Subjective Vestibular Experience							
	Dizziness		Sickness		Nauseous		Uncomfortable	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Avatar	11.74	15.26	5.39	9.93	8.47	13.64	6.12	12.21
Gravity	20.14	20.56	8.63	14.59	27.21	26.75	9.01	15.23
Spider-Man	11.39	16.05	5.37	11.22	8.37	13.82	5.31	11.16

3.5. Experiment 3: Using a Visually Intense Motion Picture can Immediately Reduce the Postural Marker of Visual Dependence

The aim of Experiment 3 was to replicate the procedure of Experiment 2 using a more visually complex and engaging optokinetic stimulus to promote recalibration effects and attention to stimulus. It was hypothesised that the use of a narrative driven, high quality, and visually intense movie would produce a short-term reduction in the postural marker of visual dependence. Note, the Rod and Frame test was not completed in Experiment 3. This task was removed from Experiment 3 as data from Experiments 1 and 2 and previous literature suggest that short-term optokinetic stimulation does not produce a short-term change in the perceptual marker of visual dependence (Van Ombergen et al., 2016).

3.5.1. Experiment 3: Methods

3.5.1.1. Participants

Eighteen participants were recruited through the same Undergraduate Psychology participation panel as Experiments 1 and 2.. The same inclusion/exclusion criteria applied in experiments 1 and 2 were employed. Due to constraints with the Coronavirus 2019 pandemic data collection was stopped at N = 18. The mean age of the sample was 20.01 (SD: 1.89). Of the 18 participants 14 identified as female and the remaining 4 identified as male. Participants were randomly allocated to either the control (n = 11) or movie condition (n = 7) using the same randomisation process as stated in Experiment 1.

3.5.1.2. Stimuli and Procedure

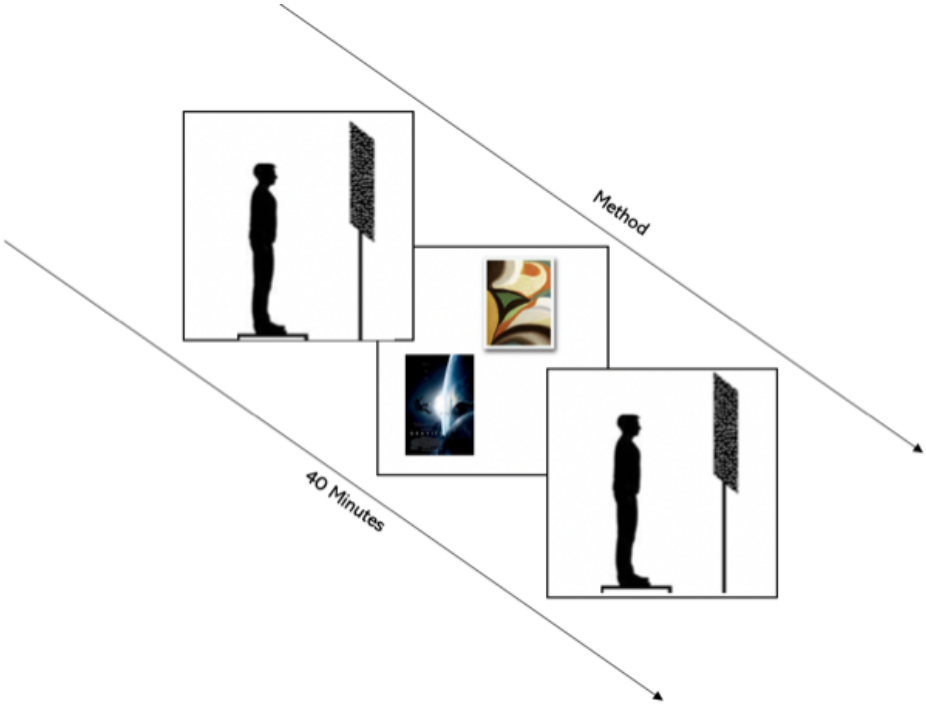

Figure 3.11. Graphical depiction of the experimental paradigm of Experiment 3.

Participants were invited into a psychophysical laboratory. The laboratory set up remained the same as in Experiment 1 (see Figure 3.4). In line with the previous experiment, participants were greeted and given verbal and written study information. If participants were comfortable proceeding they were asked to sign an informed consent form and provide demographic information (age and gender). The researcher remained in the lab (200cm away from the participant) during testing.

Participants were asked if they would like to complete a short digital questionnaire probing their experiences with dizziness (VVAS: Dannenbaum, Chillingaryan & Fung, 2011) as part of another study (see Chapter 4) and was completed digitally at the researcher station.

Participants then progressed to the main experimental testing phase. Participants were randomly allocated to either the control (no visual motion) condition or the Gravity movie condition and informed which stimulus they would view. The researcher read aloud standardised instructions to participants to explain how the sway task works and how they should respond. The instructions were the same as those presented to participants in Experiment 2; however, in these instructions participants were not asked to complete the Rod and Frame test at any point. These instructions stated that the participant was required to keep their attention to the centre of the screen at all times during testing and to stand with feet apart and arms down by sides during the Wii board task.

The participants were standing during all tasks. Participants stood barefooted on a Wii Balance Board and passively viewed, static, translating and then no visual information, for 20 seconds each. This was repeated 3 times, consecutively with no break. This resulted in 60 seconds worth of physical sway data for each of the three visual conditions Participants then viewed their randomly allocated visual stimuli for 30 minutes. Participants either viewed the same control stimulus from Experiment 2 or a 30-minute edited version of Gravity. Clips were edited so that motion intensity would be sinusoidal in nature. This new movie stimulus contained the main optokinetic scenes but also maintained the picture's narrative. See Figure 3.10 for a graphical depiction of the experimental procedure for experiment 3. Upon completion, participants were required to complete the Wii postural task once more. It took approximately 1-2 minutes from participants ending on task to starting another. If participants required a longer break duration, they were welcome to do so. See Figure 3.10 for a diagram depicting the experimental procedure. All participants were thanked, debrief and awarded course credit for their participation.

3.5.1.3. Planned Statistical Analyses

ANCOVA models were pre-planned for the analysis of data, where pre-scores on the outcome measures would be entered into the models as covariates. The Median Absolute Deviation (MAD) or Modified Z (Iglewicz & Hoaglin, 1993) was selected to detect outliers on the outcome measures of visual dependence. Any score +/- 3.5*MAD were considered outliers and removed from the analysis.

3.5.2. Experiment 3: Results

Due to the reduced sample size, no outlier detection and/or removal was employed. Five participants did not have a post exposure due to technological failures with the balance board. As in Experiment 2, the analysis was weighted for the number of the sway dependant variable parameter estimates.

3.5.2.1. Descriptive Statistics

The mean and standard deviations of Vection Quotients at each timepoint, for each group, are presented in Table 3.4. This table also documents sample sizes and valid data and missing data due to technology failure and outlier detection. The Vection Quotients appear to be somewhat lower than those reported in healthy controls (see Guerraz et al., 2001). As noted in Experiments 1 and 2, this difference is likely to reflect the fact that the participants recruited in these studies were young relative to the controls from Guerraz et al. (2001).

Table 3.4

Experiment 3: Descriptive statistics.

				Visual Dependance Measure			
		n	Time	Vection Quotient			
				Valid Data	Missing Data/ Outliers	M	SD
Condition	Control	11	Pre Exposure	11	0	1.85	0.44
			Post Exposure	5	2	1.96	0.82
	Movie	7	Pre Exposure	7	0	1.93	0.24
			Post Exposure	5	2	1.60	0.22

3.5.2.2. Inferential Statistics

Data are depicted as line graphs with 95% Confidence Intervals.

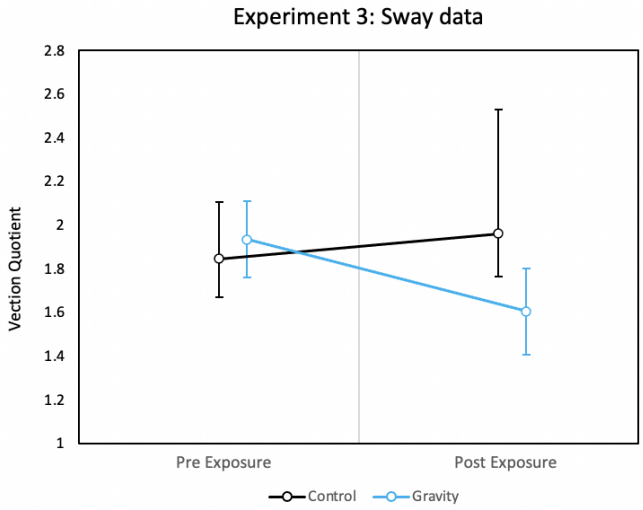

Figure 3.11. Postural sway Vection Quotient results from Experiment 3. Mean Vection Quotients are plotted at time 1 (pre-exposure) and time 2 (post-exposure). The Vection Quotient plotted is the average Vection Quotient from up to 3 estimates. The control condition is coloured black and the visual motion movie, Gravity, condition is coloured blue.

Results for the Postural Sway Vection Quotients from Experiment 3 can be seen in Figure 3.11. An ANCOVA model was used as the two groups did not significantly differ on the covariate ($t(16) = 0.49, p = 0.63, d = 0.24$. The average pre-exposure Vection Quotient was significantly related to the average post-exposure quotient: $F(1, 10) = 23.74, p = <.001, r = 0.77$. A significant difference between the two groups post-exposure (time 2) Vection Quotients was detected when controlling for baseline Vection Quotients: $F(1, 10) = 4.97, p = 0.05, \eta_{p}^{2} = 0.332$. These results are in line with the

hypothesis that unpredictable and intense short-term optokinetic stimulation (in the form of a movie) can reduce the postural marker of visual dependence. When taken together with the findings of Experiments 1 and 2, Chapter 3 presents evidence of a short-term effect of optokinetic stimulation on postural visual dependence; however, for this effect to be apparent the motion patterns must be novel, intense, unpredictable and engaging for measurable changes in sensory organisation and reweighting. More work must be undertaken to confirm such ideas, especially given the very different natures of stimuli used throughout Chapter 2.

3.6. Chapter 3: Discussion

VBRT is the preferred rehabilitation route for those with PPPD, though for best results rehabilitation should involve optokinetic visual desensitisation techniques, which to date are somewhat mundane and monotonous (Bronstein, 2004; Bronstein, Lemptert & Seemungal, 2010; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). It is theorised that optokinetic stimulation techniques allow the patient to habituate to retinal cues for self-motion, which, over time, allows the individual to desensitise to the visual triggers of their symptoms. Indeed, research in clinical populations has shown that brief exposure to an optokinetic stimulus can produce measurable changes in the postural, but not perceptual, marker of visual dependence (Van Ombergen et al., 2016).

Chapter 3 investigated whether short-term exposure to optokinetic stimulation techniques could be used to produce visuo-vestibular recalibration effects, that can in turn reduce markers of visual dependence. Findings suggest that short-term exposure to multi-media optokinetic stimulation can reduce the postural marker of visual dependence. This was in line with previous research (Van Ombergen et al., 2016). Data suggested passively viewing movies is the most effective stimulus to instigate change. Movies were shown to be more effective than passively viewing or actively playing a virtual videos game, in the student population. This is in-line with previous research (see Pavlou, 2010). It must however be noted that no claims can be made regarding the efficacy of such stimuli for the PPPD patient group with abnormally large visual dependencies. The sample sizes used within the studies are however small, which raises issues concerning power and reliability of findings.

The results reported in Chapter 3 suggest that immersive media can be used to administer optokinetic stimulation. Previous research has suggested that virtual worlds and videos can be used as optokinetic stimulation for use in the rehabilitation of PPPD (Pavlou, 2010; Pavlou et al., 2011; Whitney et al., 2006). A key aim of Chapter 3 was to understand what novel media based visual stimuli, comprising of optokinetic stimulation, could be used to produce visuo-vestibular recalibration effects. A

secondary aim was to establish which possible contemporary media would be the most efficacious stimuli to facilitate such changes. In line with the previous research (Pavlou, 2010; Van Ombergen et al., 2016), the data suggest that multimedia platforms can be used to administer optokinetic stimulation. Taken together the studies also suggest that passively viewing movies is the most effective tool to reduce the postural marker of visual dependence.

3.6.1. The Potential for Contemporary Media Platforms for Rehabilitation

The research reported in Chapter 3 is the first to explore viability of virtual video games and commercial movies as rehabilitation tools. The data suggests that virtual video games are not as effective at producing visuo-vestibular recalibration effects as passively viewing visual motion movies, albeit in a non-clinical participant cohort. This may be due to the observer's role in actively interacting with the motion patterns, which enables to user to estimate and anticipate where and when motion will appear. This could introduce more psychophysical variables such as haptic and proprioceptive signals and offer a reliable reference frame of the 'straight ahead' and vertical in relation to the earth-right upright. Future research should look to verify the preliminary findings that suggest virtual video games are less effective tools for reducing a reliance on vision in the healthy student population. Future research should also monitor gaze and attention in order to ensure participants are engaging with stimuli as protocols prescribe.

It is important to make it clear that no statements can be made on whether or not the same data pattern will be evident in the PPPD patient community and therefore gamified rehabilitation may still be a plausible avenue for future research and a promising rehabilitation tool. Future research should look to explore tis by replicating these studies with the PPPD patient group.

3.6.2. The Mixed Nature of Results

Throughout the studies of Chapter 3 what becomes evident is that the results appear to be inconclusive. Some effects and findings do not replicate, presumably because effect sizes are small, and this is may have been exacerbated by using a non-clinical sample who are not likely to have an over-reliance on the visual cue for postural control mechanisms. The studies presented in Chapter 3 should therefore be reproduced with the PPPD community in order to establish the effectiveness of tools in the right community with an (assumed) over-reliance on vision. Such studies should also be conducted with a larger sample to ensure statistical power for analyses and that if effects are visible, they are indeed detected.

3.6.3. Psychology Undergraduate Sample

A key limitation of the studies presented in this Chapter is that the participants were drawn from a relatively narrow population. I recruited undergraduate psychology students to complete the experiments however this demographic is known to a) have an over representation of females, b) binge-drink alcohol), c) be chronically stressed and anxious (Hupert & Brandt, 2018; Kurre, Straumann, van Gool, Gloor-Juzi & Bastianen, 2012; Piasecki et al., 2011; Trindade & Goebel, 2018) and d) have poor sleep hygiene (see Chapter 5). All factors known to interact with, and exacerbate, dizziness. These factors may be an issue as all can disrupt the sensory mechanics that facilitate postural control mechanisms. Furthermore, given the fact that the studies were psychophysical in nature and conducted in a dark room any of the above factors may encourage drowsiness, boredom or vestibular episodes which means the data collected may not be a reliable estimate of the human's perceptual system and sensory organisation. It should be noted I did attempt to avoid some of these limitations by stating in the recruitment information and participant information that participants must refrain from drinking for 24 hours prior to the study and have a good night sleep. Due to conducting research with undergraduate psychology students this was unavoidable but represents a key limitation of the research.

3.6.4. No Measure of Actual Persistent Postural-Perceptual Dizziness (PPPD) or Visually Induced Dizziness

A key limitation of the work collected in Chapter 3 is the lack of measurement of actual self-reported dizziness or disequilibrium symptoms. Rather, I focussed on psychophysical measures to capture the underlying visual dependence. Future research should consider the effect feature films may have on subjective visual vertigo symptoms (using the short Visual Vertigo Analogue Scale; Dannenbaum, Chillingaryan & Fung, 2011 or the Simulator Sickness Questionnaire; Kennedy, Lane, Berbaum & Lilienthal, 1993) and psychological symptoms. Future research should replicate the studies using the self-report tools to capture actual experiences of dizziness and disequilibrium rather than the psychophysical markers of visual dependence.

3.7. Chapter 3: Summary

Optokinetic stimulation has also been shown to produce changes in markers of visual dependence (Bronstein, 1995; Pavlou et al., 2012; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). Chapter 3 collects empirical investigations exploring the viability of virtual video games and commercial movies as optokinetic stimulation tools effective tools. A key aim of Chapter 2 was to explore if short-term exposure to multi-media optokinetic stimulation can reduce the postural marker of visual dependence. Chapter 3 presents a 'mixed-bag' of findings but may suggest exposure to optokinetic stimulation, in the form of movies, may produce short-term recalibration effects. Data from the first experiment suggest that multi-media tools can be used to administer optokinetic stimulation and effects are most pronounced when users passively view videos/movies. However, this finding could not be replicated in Experiment 2. In Experiment 3 a more visually intense commercial movie was used as stimulation and the effect found in Experiment 1 was replicated; however, the sample size in this study was too small to be conclusive. What this may suggest is that short-term exposure to passive visual movies can reduce the postural, but not perceptual, marker of visual dependence; however, when the nature of the optic flow must be intense and unpredictable. Taken together, what the data might suggest is that passively viewing commercial or homemade videos/movies may be the most effective tools for producing visuo-vestibular recalibration effects; however, no conclusions can be drawn about these tools in relation to the PPPD patient group.

Chapter 4: Correlating Symptoms of Persistent Postural Perceptual Dizziness with Scores on the Two Commonly used Laboratory Measures of Visual Dependence in the Student Population

4.1. Introduction

PPPD manifests due to an over-dependence on the vision for postural control mechanisms (Bronstein, 1995; Bronstein, 2016). Visual dependence refers to the degree to which an individual relies on cues from vision for postural control and balance mechanisms (Bronstein, 1995; Bronstein, 2004; Pavlou, 2010). In many situations, humans are visually dominant beings, meaning the visual cue is weighted more strongly than other senses for perception, action and postural control (Dovidio & Ellyson, 1985; Ray, Horvat Croce, Mason & Wolf, 2008; Rushton, Harris, Lloyd & Wann, 1998; Stins et al., 2009). The degree to which individuals rely on vision differs greatly between people (Witkin, 1967). Visual dependence is a normal function, however the role of vision or specifically its *weighting* can be changed and evidence strongly suggests that weighting is based on the reliability, or the precision, of the signal (Fetsch, Pouget, DeAngelis & Angelaki, 2011; Mejer & Noppeney, 2010). In the case of those with PPPD, these individuals become are overly-dependent on vision for postural control mechanisms, resulting in visually induced dizziness in highly intense or cluttered visual environments (Staab et al., 2017).

Approximately 1/3 of people who present with an acute vestibular insult (for example from an inner ear infection such as neuritis: Cousins et al., 2014) will go on to develop a chronic visually induced vertigo (Bronstein, 1995; Bronstein, 2004; Staab, 2020; Yardley, 1993). If visual dependence explains PPPD symptoms then one may assume that a higher degree of visual dependence would correlate positively with PPPD symptoms. Indeed, there is data available which suggests that visual dependence can explain the existence of PPPD symptoms in some patients (Bronstein, 1995; Pavlou, 2010). However, little is known about individuals who are free from the disorder but score highly on questionnaire-based measures of PPPD.

Recently published research has shown that although most generally healthy people report no PPPD symptoms, approximately 11% of the adult population report PPPD symptoms that are greater than or equal than the lowest scoring PPPD patient – a score of 25 on the VVAS (Powell, Derry-Sumner, Rajenderkumar, Rushton & Sumner, 2020). It is not known if these people have an over-dependence on vison. To date, no research has looked to correlate scores on the commonly used laboratory

measures of visual dependence (Rod and Frame test and the postural sway Vection Quotient) and self-report PPPD symptoms in non-clinical populations.

In addition, the literature indicates that visual dependence is assumed to be one entity. As such, both measures are used to detect the same underlying factor: visual dependence. Given this, one may hypothesise that scores on the two measures would correlate positively with one and other. However, research has not always found a significant correlation between the two measures of visual dependence (Bronstein, 1995; Guerraz et al., 2001; Pavlou, Davies & Bronstein, 2006). Furthermore, research is yet to correlate PPPD symptoms with the two measures of visual dependence; despite the need for such research (Powell, Derry-Sumner, Rajenderkumar, Rushton & Sumner, 2020).

4.1.1. Aims and Objectives

The aim of Chapter 4 was to explore the relationships between self-reported PPPD symptoms and performance on traditionally used laboratory measures of visual dependence. Thus, the objectives of this Chapter were to establish if any correlations existed between the postural and perceptual markers of visual dependence and PPPD symptoms. Studies were designed to explore the relationships in the non-clinical student and sub-clinical student populations.

4.2. Study 1: Correlating Symptoms of Persistent Postural-Perceptual Dizziness with Scores on the Two Commonly Used Measures of Visual Dependence

The aim of this study was to correlate self-reported PPPD scores with scores on the two commonly used laboratory measures of visual dependence (Rod and Frame test and the postural sway Vection Quotient) in the healthy student population.

4.2.1. Study 1: Methods

4.2.1.1. Participants

As already noted, the participants in Experiment 1-3 were asked if they would be interested in participating in a follow-on study using where their same postural and perceptual data would be used. The follow-on study would only require participants to complete the Visual Vertigo Analogue Scale

(VVAS: Dannenbaum, Chillingaryan & Fung, 2011). All participants asked to take part agreed and gave written and verbal consent. Participants were awarded 1 additional course credit for doing so.

One-hundred and fifty-nine participants were approached and asked to take part in the study Of the 159 participants, 18 participants did not have a baseline score on the Rod and Frame test, postural sway test or did not complete the required VVAS scale. The remaining sample was therefore N = 139. In this sample there were 106 females and 33 males. The mean age of the sample was 22.74 (SD 4.76).

4.2.1.2. Apparatus and Stimuli

4.2.1.2.1. Measures of Visual Dependence

The baseline (pre-exposure) Vection Quotient and Rod and Frame measure from Chapter 3 experiments was supplemented with a VVAS measure.

4.2.1.2.2. The Visual Vertigo Analogue Scale (VVAS)

The Visual Vertigo Analogue Scale (VVAS; Dannenbaum, Chilingaryan & Fung, 2011) identifies whether an individual experiences the characteristic symptoms of PPPD (formerly visual vertigo: see General Introduction Chapter). The full psychometric properties of the VVAS can be found in section 2.6.1.2.1 of Chapter 3.

4.2.1.3. Procedure

All participants were already taking part in one of the three experiments presented in Chapter 3. For each participant, the baseline (pre-experimental manipulation) raw data on the postural sway and Rod and Frame tests were collected. Sway and Rod and Frame data were treated in accordance to the same paradigm presented in Chapter 3 to form a Vection Quotient and Rod and Frame effect (see Experiment 1, Chapter 3).

4.2.1.4. Planned Statistical Analyses

Bayesian statistical analyses were chosen as analysis tools for the data. Bayesian statistics were chosen as these analyses are able to offer support both the alterative hypothesis and the conceptual null hypothesis. Thus, no hypothesis was made in regard to the relationship between variables and instead Bayesian statistics were utilised to allow the data to show support for either potential hypothesis. The Median Absolute Deviation (MAD) or Modified Z (Iglewicz & Hoaglin, 1993) was

selected to detect outliers on the outcome measures of visual dependence. Any score +/- 3.5*MAD were to be removed from the analysis.

4.2.2. Study 1: Results

The measures of visual dependence were the Rod and Frame effect and Postural Sway – Vection Quotient. A more pronounced effect on the Rod and Frame test indicates a larger reliance on the visual reference frame. A larger Vection Quotient suggests that the observer is more destabilised by motion on the retina. The measure of PPPD symptoms was the VVAS score – the higher the score on the VVAS, the more evidence of characteristic symptoms of PPPD.

4.2.2.1. Treatment of Data

Pooling the Data from Across the Three Experiments of Chapter 3

The data from across the three experiments of Chapter 3 were pooled. This was done in order to allow for a greater sample size and therefore enhance statistical power. In order to establish if it were appropriate to pool the data from across the three experiments, all baseline, pre-exposure data, for the postural and perceptual markers of visual dependence (from each experiment) were assessed. Raw data are presented in Figure 4.1. The patterns of data appeared to be homogeneous across experiments. A one-way ANOVA was conducted on each visual dependence variable, comparing the data from each experiment. No significant difference was detected for the Rod and Frame effects, $F(2, 133) = 0.866$, $p = 0.423$, $\eta_p^2 = 0.013$, or the Vection Quotient, $F(2, 133) = 1.642$, $p = 0.197$, $\eta_p^2 = 0.024$. Thus, visual dependence data from the three experiments of Chapter 3 were pooled and used for the analyses in Chapter 4.

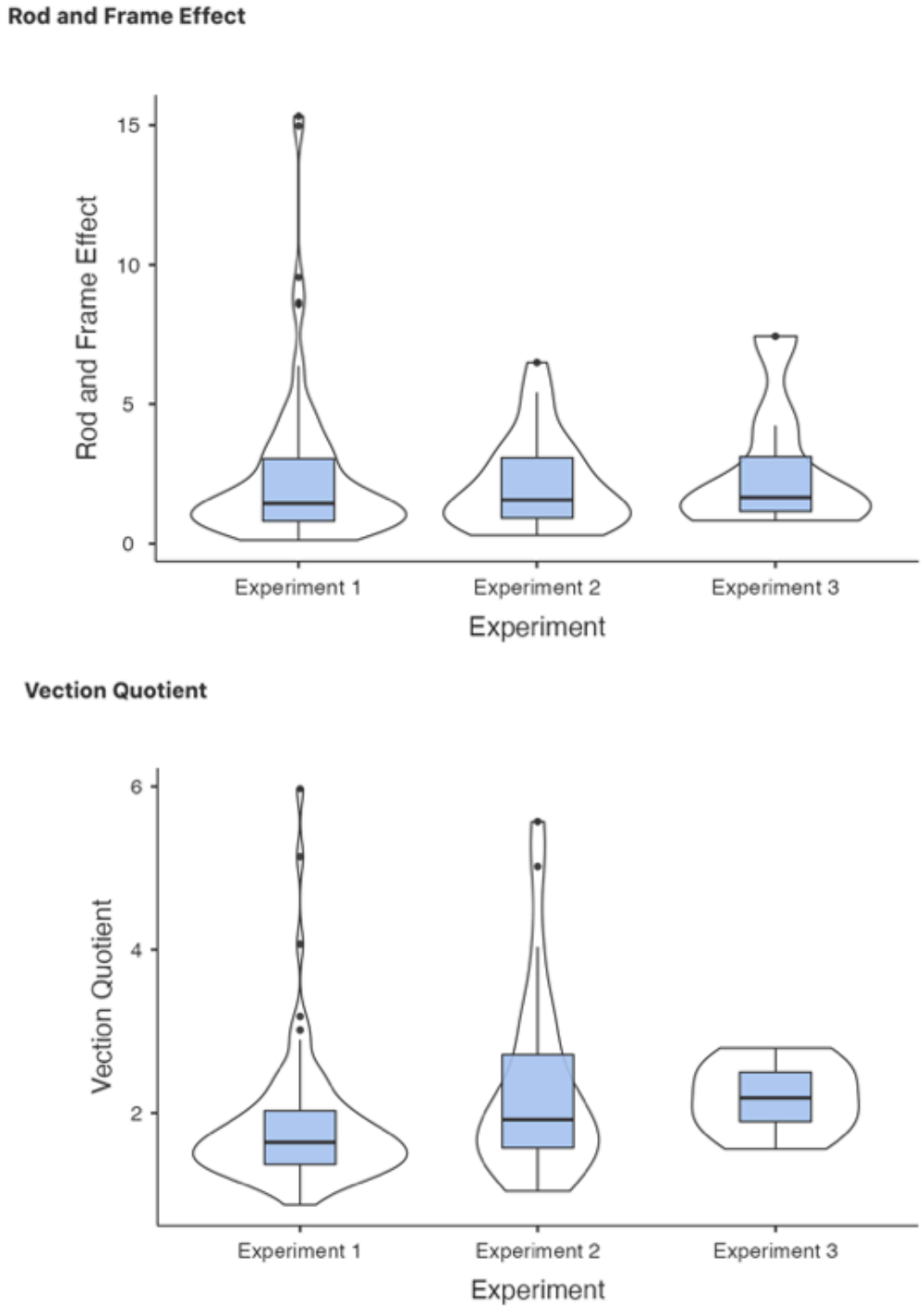

Figure 4.1. Data on the postural and perceptual markers from across the three experiments in Chapter 3. Top: the mean Rod and Frame effect for each participant is plotted on the y axis. Bottom: the mean Vection Quotient is plotted on the Y axis. On both graphs the x axis represents the experiment from Chapter 3. Each experiment's raw data is presented as a Boxplot presented within a violin frame to show where data clusters on the dependent variables.

4.2.2.2. Descriptive Statistics

The mean VVAS score in the dataset was 15.33 (SD: 12.92). Rod and Frame effects had a mean score of 1.61 (1.16) and Vection Quotients had a mean score of 1.76 (0.50). These scores are typical of a healthy sample free from PPPD and/or vestibular compromise (Böhmer & Mast, 1999; Hafström, Fransson, Karlberg & Magnusson, 2004a; Hafström, Fransson, Karlberg & Magnusson, 2004b; Guerraz et al., 2001). As noted in Chapter 3, scores on measures of visual dependence may be considered lower than those reported in the literature (see Gurraz et al., 2001). This is likely due to the use of a young student sample.

4.2.2.3. Inferential Statistics

4.2.2.3.1. Bayesian Correlation Analyses

Bayesian correlation analyses were used to establish if the three measures of interest correlated with one and other. For the Bayes analyses I report BF_{01} – the Bayes factor in favour of the null hypothesis. A stretched beta-width of 1 is set for all analyses meaning all correlations between -1 and +1 are given an equal prior probability (for a primer see Quintana & Williams, 2018). For all Bayesian analyses graphical depictions of the relationships between data are presented as scatterplots. Data were modelled and analysed using the Jasp software (Love et al., 2019) version 0.9.2.

4.2.2.3.2. Correlating Visual Vertigo Analogue Scale (VVAS) Scores and Measures of Visual Dependence

The results from the VVAS, Vection Quotient and Rod and Frame effects are presented in Figure 4.2. No correlation between Visual Vertigo scores and Vection Quotients was found ($r = 0.02$). The accompanying Bayes Factor was $BF_{01} = 8.9$ indicating that it is 8.9 times more likely that they are not related than related. No significant correlation with respect to VVAS scores and the Rod and Frame effects ($r = -0.01$) was found. The accompanying Bayes Factor was identical $BF_{01} = 8.9$ (moderate-strong evidence in support of the null hypothesis). No correlation between Rod and Frame effect and the Vection Quotient was detected ($r = 0.20$). The accompanying Bayes Factor was $BF_{01} = 0.9$, representing no evidence of a relationship (see Jeffereys, 1961; Lee & Wagenmakers, 2013).

4.2.3. Study 1: Interpretations

Taken together the data therefore supports the conceptual null hypothesis and the preliminary evidence presented by Pavlou, Davies and Bronstein (2006) and Guerraz et al. (2001) that measures of

visual dependence do not correlate. Furthermore, in the heathy student population PPPD symptoms fail to correlate with performance on traditional measures of visual dependence.

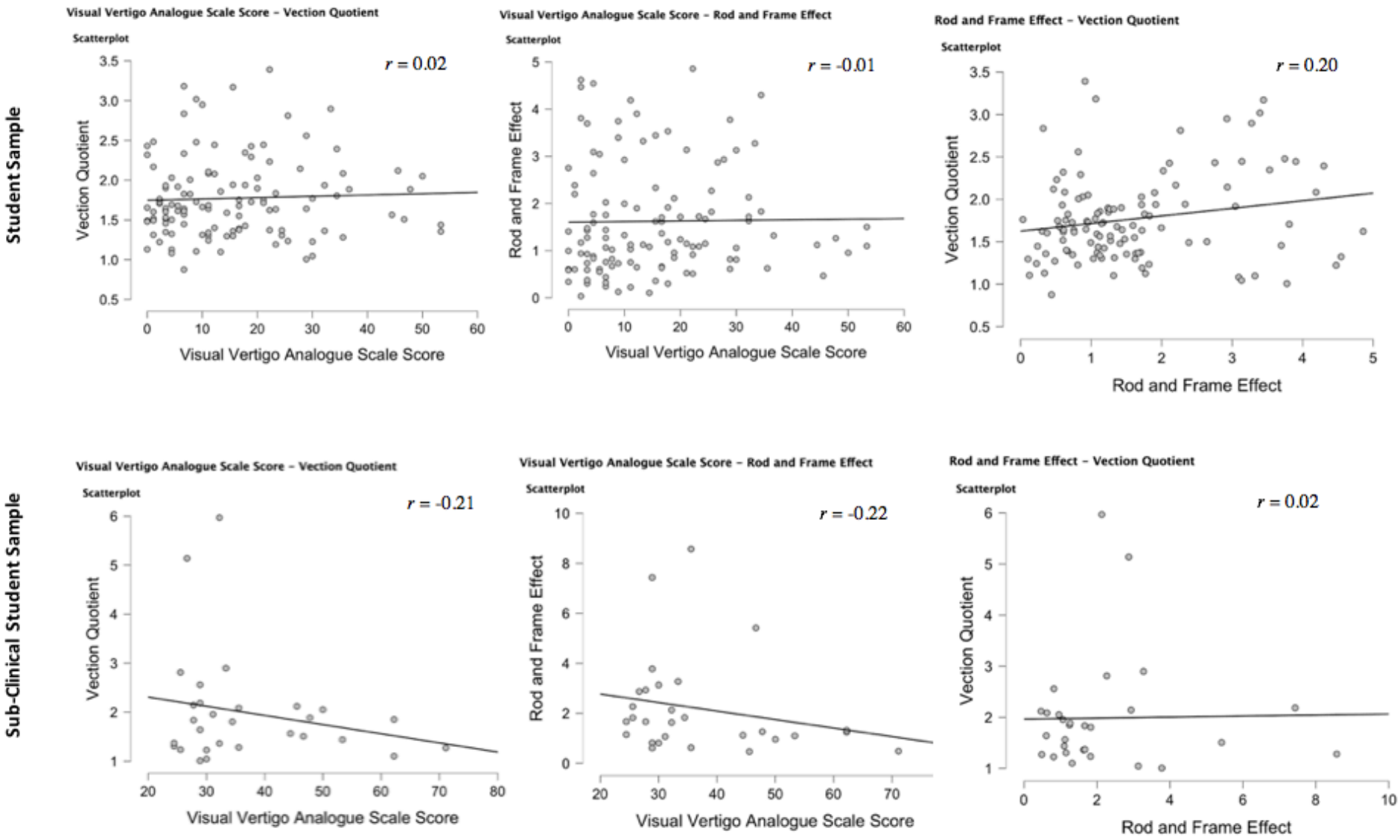

Figure 4.2. Bayesian inferential correlational models for the relationships between Visual Vertigo Analogue Scale (VVAS) Scores, Vection Quotients and Rod and Frame effects. Top: relationships in the healthy student population. Bottom: relationships in the sub-clinical student cohort.

4.3. Study 2: Correlating Symptoms of Persistent Postural-Perceptual Dizziness with Scores on Measures of Visual Dependence in Sub-Clinical Persistent Postural-Perceptual Dizziness Participants

The aim of the second study of Chapter 4 was to explore the relationships between PPPD symptoms and performance on measures of dependence in sub-clinical student participants.

4.3.1. Study 2: Methods

4.3.1.1. Participants & Procedure

Of the 139 participants whose data was used in Study 1 of Chapter 4, 29 participants self-reported PPPD symptoms that are equal to or greater than scores that actual PPPD patients report (minimum PPPD patient score on the VVAS = 25; Powell, Derry-Sumner, Rajenderkumar, Rushton & Sumner, 2020). These participants were used as the sample for Study 2.

4.3.1.2. Planned Analyses

The original Bayesian analyses were replicated using these sub-clinical participants to explore the relationships between VVAS scores, Vection Quotients and Rod and Frame effects in those who self-report clinical levels of visually induced dizziness.

4.3.2. Study 2: Results

4.3.2.1. Descriptive Statistics

The mean VVAS score in the sub-clinical dataset was 37.09 (SD: 12.72). The mean Rod and Frame effect was 2.19 (1.97). The mean Vection Quotient was 1.99 (1.11). These scores are typical of a healthy sample free from PPPD and/or vestibular compromise and do not suggest abnormal levels of visual dependence (Böhmer & Mast, 1999; Hafström, Fransson, Karlberg & Magnusson, 2004a; Hafström, Fransson, Karlberg & Magnusson, 2004b; Guerraz et al., 2001).

4.3.2.2. Inferential Statistics

Data are depicted in Figure 4.2. No significant correlation between VVAS scores and Vection Quotients was detected (r = -0.21). The accompanying Bayes Factor was BF_{01} = 2.4. No significant

correlation between VVAS scores and Rod and frame effect was detected (r = -0.22). The accompanying Bayes Factor was BF_{01} = 2.3, representing weak or anecdotal evidence in favour of the null hypothesis. Finally, no significant correlation between Rod and Frame effects and Vection Quotients were detected (r = 0.02). The accompanying Bayes Factor was BF_{01} = 4.3, offering moderate support for the conceptual null hypothesis.

4.3.3. Study 2: Interpretations

PPPD symptoms and laboratory measures of visual dependence fail to correlate in both the healthy student and sub-clinical student population. This may suggest that visual dependence may not be able to explain the existence of symptoms in this sub-clinical group. No claims about the actual PPPD group based on this data. This may however suggest that there is something uniquely different between people who experience high levels of PPPD like symptoms and actual PPPD patients. Perhaps this is where the key difference in visual dependence manifests. The data also suggest that the two measures of visual dependence again fail to correlate and thus data from across Chapter 4 suggest that traditional laboratory measures of visual dependence do not correlate. This may suggest that one or both of the measures do not actually capture visual dependence or perhaps more controversially may suggest that visual dependence is not one uniform construct.

4.4. Chapter 4: Discussion

The PPPD condition develops due to a maladaptive over-reliance on the visual cue for postural control and balance mechanisms (Bronstein, 1995). Some people in the student population report clinical levels of dizziness (Powell, Derry-Sumner, Rajenderkumar, Rushton and Sumner, 2020) and it is currently unknown if the theory of visual dependence can explain the existence of such symptoms in those without a diagnosis of PPPD. In addition, research has suggested that the commonly used measures of visual dependence fail to correlate (Bronstein, 1995; Guerrez et al., 2001; Pavlou, Davies & Bronstein, 2006). This Chapter aimed to explore the relationships between PPPD symptoms and the two commonly used measures of visual dependence in the healthy student and sub-clinical student groups.

The data presented in this Chapter suggest that, in the student population, PPPD symptoms do not correlate with performance on commonly used laboratory measures of visual dependence. When explorations focussed on sub-clinical PPPD students, PPPD symptoms and levels of visual dependence also failed to establish a significant correlation. Furthermore, in both groups, the two

commonly used laboratory measures of visual dependence also failed to correlate. These findings are in line with previous research that has not always found a significant correlation between the two measures of visual dependence (Bronstein, 1995; Guerraz et al., 2001; Pavlou, Davies & Bronstein, 2006). The findings are, however, the first to suggest that PPPD symptoms, in the typically healthy and sub-clinical student populations, do not correlate with commonly used laboratory measures of visual dependence. The findings in Chapter 4 may suggest that visual dependence may not be able to explain PPPD symptoms in these populations– though experimental data would be required to confirm this theory. As measures of visual dependence did not correlate with one and other, it would suggest one of three things. First, that the measures may not actually measure visual dependence. Second, that if they do measure visual dependence, then visual dependence may not be one uniform entity. Or third, that one or both of the measures are compromised.

4.4.1. The Rod & Disk Measure of Visual Dependence

The Rod and Disk test is a measure of visual dependence that works as a symbiosis of the Rod and Frame and the postural sway test (Guerraz et al., 2001; Roberts, Melo, Siddiqui, Arshad & Patel, 2016). In this test the observer must rotate a rod to the subjective visual vertical, but the visual references frame they are presented with is a rotating dot cloud – introducing visual motion into the test. The measures of visual dependence used in Chapter 4 isolate the effects of visual motion and the neural representation of verticality. This is a common approach that isolates the manifestation of an individual's underlying dependence on vision (see Pavlou Bronstein & Davies, 2013; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004; Van Ombergen et al., 2016). However, this introduces a key limitation to the work collected in Chapter 4: PPPD symptoms may correlate with this alternative test where visual motion and verticality are assessed together. Future research should replicate the studies reported in Chapter 4, substituting the traditional laboratory measures of visual dependence with the Rod and Disk.

4.4.2. The Wii Board as a Tool to Measure Postural Sway

The research reported in Chapter 4 quantified postural sway using the Wii board and the BrainBlox software (Cooper, Siegfried & Ahmed, 2014 – see Chapter 3 Methods section). Although this is a more accessible and affordable technology that has been used in previous PPPD research Guerrez at al., (2001) the use of this device may explain why the studies failed to establish a significant correlation. The device itself uses Bluetooth technology and is a substitute for the wider Sensory Organisation Test (SOT: Umphred & Lazaro, 2012). Thus, had the more advanced postural kinematics tools been used findings may have been different. Unfortunately, it was not possible for access such equipment due to the large costs associated with ascertaining SOT. Future researchers

with access to such resources should look to replicate the correlational studies presented in this Chapter using postural data from the SOT in order to shed light on the relationship between PPPD and the postural and perceptual markers of visual dependence.

4.4.3. Sampling Students

Another key limitation of the researched conducted in Chapter 4 is the use of the student population. This group is notorious for colds/flus, alcohol consumption, poor sleep quality and or general mental/physical fatigue (Bewick et al., 2008; Davoren, Demant, Shiely & Perry, 2016; Langkamp-Henken et al., 2015; Lund, Reider, Whiting & Pritchard, 2010; Sievertsen, Gino & Piovesan, 2016; Singleton & Wolfson, 2009). Any and all of these extraneous variables that may have introduced some degree of unsystematic variance into the Rod and Frame and postural sway measures. This highlights the importance of replicating this research with a more heterogenous sample.

4.4.4. Bayesian Statistics: Seeking Support for the Null Hypothesis

A strength of the work presented in Chapter 4 is the adoption of Bayesian Correlational analyses. Unlike traditional frequentist correlations, Bayesian statistics are not bound to an analysis model that seeks support for the alternate hypothesis only. Rather, the use of Bayesian models allows the data to show support for either the conceptual null hypothesis or alternative hypothesis. By employing such methods, a deeper understanding of the data is available. This has allowed the researcher to establish that parameters of interest fail to correlate and show support for the null hypothesis confirming initial correlational observations by offering interpretations of the data that go beyond a lack of statistical significance.

4.5. Chapter 4: Summary

The PPPD condition is thought to arise due to a maladaptive over-reliance on the visual cue for postural control and balance mechanisms (Bronstein, 1995). Symptoms of PPPD have been identified in the student population (Powell, Derry-Sumner, Rajenderkumar, Rushton and Sumner, 2020). However, it is currently unknown if the theory of visual dependence can explain the existence of symptoms in these people. In addition, there are two commonly used laboratory measures of visual dependence (the Rod and Frame test and the postural sway Vection Quotient) do not always correlate with one and other which may suggest that the measure do not actually measure the same underlying factor (Bronstein, 1995; Guerraz et al., 2001; Pavlou, Davies & Bronstein, 2006). The findings presented in Chapter 4 failed to find and significant correlation between PPPD symptoms and performance on the two traditional laboratory measures of visual dependence. This may suggest that

visual dependence may not be able to explain PPPD symptoms in the healthy and sub-clinical student population – though experimental data would be required to confirm this theory. In addition, the two measures of visual dependence did not significantly correlate with one and other. This may suggest one of three things. The first, that the measure may not actually measure visual dependence. The second, that if they do measure visual dependence, then visual dependence may not be one uniform entity. Or third, that one or both of the measures are compromised.

Chapter 5: General Discussion

5.1. General Overview

PPPD is a functional (non-organic), debilitating neuro-vestibular disorder characterised by chronic episodes of dizziness, disequilibrium, postural imbalance and non-spinning vertigo (Bronstein, 1995; Deitrich & Staab, 2017; Seemungal & Passamonti, 2018; Staab et al., 2017; Wurthman et al., 2017). Symptoms are triggered by situations of visuo-vestibular conflict, intense visual-environments and active and passive motion (Bronstein, 1995; Bronstein, 2004; Pavlou, 2010; Staab et al., 2017). Commonly reported triggers include cluttered visual environments, busy moving traffic and walking around the notorious supermarket aisle (McCabe,1975; Söheston, Bittar & Staab, 2016; Staab et al., 2017). The central aim of this Thesis was to understand the different symptoms, experiences and factors associated with PPPD and how they relate to one and other and other. This was achieved by setting and achieving specific empirical objectives, documented in Chapter 2, 3 and 4. An overview of each empirical Chapter of the Thesis is presented below.

5.1.1. Chapter 2: Overview

PPPD patients often present with secondary, or potentially precipitating, psychiatric illnesses which result in a debilitated patient with complex co-occurring illnesses that diminishes quality of life (Staab, Eckhardt-Henn, Horri, Jacob & Stupp, 2014; Zur et al., 2015). The aim of Chapter 2 was to explore how individuals with PPPD make sense of their symptoms and condition and to better understand the lived experiences of PPPD, including the psycho-social impacts of the condition. Qualitative findings suggested PPPD patients may experience identity loss, dismissal and non-belief, poor psychological well-being, out of body experiences and processes of sense-making. Shared themes included poor metal well-being, sensory overload and sleep impairments. PPPD also stated that television/movie watching did not affect their condition or symptoms. Poor sleep quality, poorer general mental well-being, somatic amplification, higher levels anxiety and depression, and lower levels of self-esteem were all shown to correlate with PPPD symptoms - though the magnitude of the linear relationship varied between the scales and constructs used. However, only anxiety and somatic amplification were significant predictors of PPPD when the PPPD is measured by the Visual Vertigo Analogue Scale (VVAS: Dannenbaum, Chilingaryan & Fung, 2011). When PPPD is measured by the Niigata PPPD Questionnaire (NPQ; Yagi et al., 2019) anxiety, somatic amplification and depression were significant predictors PPPD symptoms.

5.1.2. Chapter 3: Overview

Optokinetic stimulation has also been shown to produce changes in markers of visual dependence (Bronstein, 1995; Guerraz et al., 2001; Pavlou et al., 2012; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). The aim of Chapter 3 was test if short-term exposure to optokinetic stimulation can produce changes in markers of visual dependence. As little is known about the timeframes on which optokinetic stimulation on, the researcher also aimed to explore these timeframes. Chapter 3 also aimed to establish if engaging multi-media technology can be used to as optokinetic stimulation. The findings from Chapter 3 suggest that passively viewing movies with optokinetic properties are the most effective media-based tools for reducing the postural marker of visual dependence. These effects were, however, small and hard to replicate. The results suggest that the 'movie' must be visually intense and contain unpredictable motion patterns for results to be evident. The research reported in Chapter 3 were conducted with healthy student participants. As such, no conclusions can be drawn about these tools in relation to the PPPD patient group.

5.1.3. Chapter 4: Overview

PPPD manifests due to an over-dependence on the vision for postural control mechanisms (Bronstein, 1995; Bronstein, 2016). The aim of Chapter 4 was to explore the relationships between self-reported PPPD scores and performance on traditionally used laboratory measures of visual dependence, in the healthy student and sub-clinical PPPD student cohorts. These measures were the Rod and Frame test of the Subjective Visual Vertical (Asch & Wilkin, 1948a; Asch & Wilkin, 1948b) and the postural sway Vection Quotient (Barnes & Crutchfeild, 1990; Guerraz et al., 2001). Findings suggest that PPPD symptoms do not correlate with performance on the two traditional laboratory measures of visual dependence. This may suggest that visual dependence may not best explain symptoms in sub-clinical PPPD participants. The research reported in Chapter 4 also failed to find a significant correlation between the two measures of visual dependence. This may suggest that one, or both, of the measures were compromised or that visual dependence is not one uniform entity. Findings were consistent in the healthy and sub-clinical PPPD participants. Interpretation of the data may suggest that there is something different between PPPD patients and sub-clinical PPPD participants reporting similar levels of visually induced dizziness.

5.2. Phenomenological Interactions with Patients can Promote Candour and Disclosure of Deeply Personal Experiences and Symptoms

Previous research has explored the lived experiences of chronic dizziness and/or PPPD; however, these studies are limited (Mendel, Lutzen, Bergenius and Bjorvell, 1997; Sezier, Saywell, Terry, Taylor & Kayes, 2019). The aforementioned studies have used a range of qualitative techniques including thematic analysis, content analysis and grounded theory (Mendel, Lutzen, Bergenius and Bjorvell, 1997; Tinetti, Williams & Gill, 2000; Herdman, Evetovits, Everton & Murdin, 2020). However, no research has adopted the hermeneutic approach of Interpretative Phenomenological Analysis (IPA) to probe lived experiences PPPD in the patient community. This method has shown promise as a tool for helping people articulate complicated health compromising illnesses (Ashe, Furness, Taylor, Haywood-Small & Lawson, 2011; Flowers, Davis, Larkin, Church & Marriott, 2011; Fox & Diab, 2015; Strickland, Worth & Kennedy, 2015). A key strength of the research presented in this Thesis is the adoption of the IPA method applied to the PPPD community. This method was shown to promote candour in qualitative interactions with patients resulting in novel and personal themes emerging from the data. Using this method allowed the researcher to co-produce a rich data set that documents the implications of the PPPD condition across numerous domains of life.

In line with previous research, IPA has proven to be a successful tool to probe lived experiences of hard to articulate experiences such as PPPD (Ashe, Furness, Taylor, Haywood-Small & Lawson, 2011; Flowers, Davis, Larkin, Church & Marriott, 2011; Fox & Diab, 2015; Strickland, Worth & Kennedy, 2015). Furthermore, the tool has allowed for novel themes to be captured and associated with PPPD such as out of body experiences, the impact of the condition on identity and the guilt and shame that can be produced by experiencing PPPD symptoms and avoiding triggering scenarios. The IPA method also identified novel psycho-social factors that are associated with PPPD such impaired sleep, poor general psychological well-being, impaired self-esteem, depression and somatic amplification. These variables may not have been discovered had it not been for patients' candour during semi-structured interviews. Many of the themes discussed are deeply personal, potentially shameful, stigmatised, and/or hard to admit or disclose. Thus, this Thesis has shown the effectiveness of the IPA method in promoting candid disclosure of experiences. It must, however, be noted that the interpersonal

IPA promotes a commitment to the ideographic approach. Applying this tool to the PPPD patient groups allowed for the generate rich personalised accounts of the lived experiences of PPPD from each individual expert in their condition. Adopting the ideographic approach may have validated

patient experiences and promoted an ethical way to explore the condition: an approach rooted in the experiences of those who navigate daily life with the condition.

5.3. Psycho-Social Factors Can Account for Some of the Variance in Persistent Postural-Perceptual Dizziness Symptoms

In Chapter 2, novel psycho-social variables identified from interviews with PPPD patients were explored (quantitatively) in a relatively large data set, using health student participants. The psycho-social variables were shown to relatively explain a small amount of variance in the data; however, this is meaningful. The data suggests that PPPD is not a solely visual problem, as psycho-social factors can explain approximately 14.8% – 20.3% of the variance in symptoms in the healthy student population. This highlights the idea that psycho-social factors are also part of the PPPD condition. It is important to note that no claims about the PPPD patient group can be made based on this data. However, one may assume that these factors might explain a greater amount variance in symptoms in the clinically dizzy PPPD patient group. Future research should therefore look to replicate this study in the PPPD cohort where PPPD symptoms in patients may be explained by these factors.

5.4. Using Contemporary Diverse Multi-Media as Optokinetic Stimulation

In the case of managing the PPPD patient, Vestibular Based Rehabilitation Therapy (VRBT) is the 'gold standard' rehabilitation tool (Hannsson, 2007; Meldrum & McConn-Walsh, 2018; Umphred & Lazaro, 2012) but must involve optokinetic stimulation techniques (Bronstein, 2004; Bronstein, Lemptert & Seemungal, 2010; Pavlou, Lingeswaran, Davies, Gresty & Bronstein, 2004). To date, the stimuli used as optokinetic stimulation are monotonous and unengaging; which potentially explains patient non-adherence and attrition in rehabilitation programmes (Soto-Varela et al., 2017).

For clinically significant changes to occur in PPPD patients, 8 weeks of active vestibular rehabilitation that focuses on visual desensitisation via optokinetic stimulation (alongside more traditional head-rotation tasks) is required (Hansson, 2007; Horak, Jones-Rycewicz & Shumway-Cook, 1992). Although a wealth of literature has confirmed the clinical efficacy of VRBT with and without optokinetic stimulation techniques (Pavlou et al., 2011; Popkirov, Stone & Holle-Lee, 2018; Thompson, Goetting, Staab & Shepard, 2015; Whitney et al., 2006) no research has directly explored the short-term effects of optokinetic stimulation (visual desensitisation techniques) on the postural and perceptual markers of visual dependence. Furthermore, although optokinetic stimuli in clinical settings are starting to become more engaging, stimuli are still far from user-centred, enjoyable or

even engaging (Aharoni, Lubetzy, Arie & Krasovsky, 2017; Aharoni, Lubetzy, Wang, Goldman & Krasvosky, 2019; Meldrum et al., 2012; Pavlou et al., 2012; Popkriov, Stone & Holle-Lee, 2018).

In Chapter 3, proof of principle data that show that engaging optokinetic multi-media can be used as optokinetic stimulation to successfully reduce the postural, but not perceptual, marker of visual dependence. This was in line with suggestions from previous research (see Van Ombergen et al., 2016). Virtual video games, clips of videos and blockbuster movies were all shown to reduce have a transient effect on postural visual dependence; however, passively viewing movies were shown to be the most promising tools in the healthy student population.

5.5. The Potential for Gamifying Rehabilitation

Virtual video games may be useful tools that clinicians could use to administer optokinetic stimulation to rehabilitate PPPD. The gamification of rehabilitation may offer a more engaging process for the patient and could be supported by at home play that may encourage patient adherence to 'at home' tasks that support the rehabilitation process. Previous research has suggested that virtual video games may be effective tools in clinical settings (Wilkinson, Ang & Goh, 2008) and they have been successfully employed to support clinical treatment of a range of disorders such as Autism, amblyopia (a condition where the eyes and brain do coordinate correctly), anxiety and depression (Carassco, 2016; Gambacorta et al., 2018; Mazeruk & Englehart, 2013; Wijnhoven, Creemer, Engels & Granic, 2015). Given this success, virtual video games may be promising tools for rehabilitating PPPD however this excited avenue of research had yet to be explored.

The findings presented in Chapter 3 suggest that virtual video games can produce a transient short-term reduction in the postural marker of visual dependence. This effect is not sustained after exposure. Data do, however, suggest that passively viewing visual motion movies may be more effective than using virtual video games. This may be due to the optokinetic properties of the stimulus; when exposed to movies you cannot predict the motion coming from the stimulus in the same way that you can from actively playing a virtual video game. For example, when playing a virtual video game (such as Mario Kart 8; see Chapter 3) the user may be able to anticipate where the motion patterns originate from and predict coming motion as they are actively navigating the avatar and car. Previous research has established that prior information about the sensory world affects the perceptual representations humans form (Alberts, de Brouwer, Selen & Medendorp, 2016; Mamassian, Landy & Maloney, 2002; Shi, Church & Meck, 2013). Based on the findings documented in Chapter 3, actively interacting with an optokinetic stimulus may dilute the sensory recalibration effects of the stimuli.

5.6. Clarifying Visual Dependence: The Leading Theory of Persistent Postural-Perceptual Dizziness (PPPD)

Visual dependence is the leading theory of PPPD manifestation (Bronstein, 1995; Staab et al., 2017; Pavlou, 2010). However, the data from presented in Chapter 4 suggest that the theory cannot fully explain all experiences of chronic subjective dizziness outside of clinically dizzy populations. In Chapter 4 quantitative data were presented that show that symptoms of PPPD are not correlated with the postural nor perceptual marker of visual dependence. This finding was consistent in the healthy student population and sub-clinical PPPD student population. This finding is in contrast to theoretical expectations within the literature (Bronstein, 1995; Fetsch, Pouget, DeAngelis & Angelaki, 2011; Powell, Derry-Sumner, Rajenderkumar, Rushton & Sumner, 2020; Staab et al., 2017).

PPPD symptoms exist in approximately 10 percent of the population (Powell, Derry-Sumner, Rajenderkumar, Rushton & Sumner, 2020). It has been suggested that people who go on to develop PPPD already over rely on the visual cue for postural control mechanisms. The data presented in Chapter 4 may suggest this theory is not correct as in those without the disorder there is no evidence of a correlation between visual dependence and PPPD symptoms. This does not rule out the theory of visual dependence in those with PPPD. Rather, what the data may suggest is that if there is no relationship between the factors before the vestibular compromising illness, then the vestibular illness may be the catalyst for the development of an over-dependence on vision. The initial vestibular comprise must force a reorganisation of the senses facilitating postural control and balance mechanisms which upweights the visual cue extends beyond the degree to which it is needed. Thus, although the data may suggest that the theory of visual dependence may not explain PPPD symptoms in the generally healthy population, the theory may still be correct for actual PPPD patients. This tells us something important about the sub-clinical PPPD student population – they are not the same as PPPD patients. There is therefore need for future research to replicate this study with the PPPD community to better understand the relationships between PPPD and visual dependence. However,ascertaining a large patient sample size would be needed. This may prove difficult for future researchers.

5.7. Passively Viewing Television and Movies Does Not Seem to Exacerbate or Trigger Visually Induced Dizziness

If one were to apply the theory of visual dependence (Bronstein, 1995; Bronstein, 2005; Cousins et al., 2014) to actual real-world visual behaviours, the theory would suggest that people who report higher PPPD symptoms should avoid visual stimuli and behaviours that result in sensations of

vection, vertigo or disequilibrium (Bronstein & Lempert, 2007; Bronstein & Lempert, 2010; Pavlou, Davies and Bronstein, 2009). Visually induced dizziness should, theoretically, discourage people from engaging with recreational motion activities that contain visual motion. This may include daily behaviours/activities such as television and movie watching or virtual video game playing. In Chapter 3, this question was posed to PPPD patients qualitively qualitatively with actual PPPD patients. Accounts suggest that passively viewing television and/or movies did not trigger their symptoms when viewed in the home setting. This goes against many assumptions within the literature and may therefore call the validity of PPDP scales into question as all commonly used questionnaire-based tools that assess PPPD contain items exploring tolerance to television/movies with high motion content (see VVAS: Dannenbaum, Chilingaryan & Fung, 2011, presented in Appendix 1.1: SSQ: Kennedy, Lane, Berbaum & Lilenthal, 1993, presented in Appendix 4.1). This criticism of the validity of assessment may also be a fair criticism of the research in Chapter 2 and 4, where such scales were employed. Future research should explore PPPD patients' ability to tolerate visual motion from television/movies and virtual video game. Depending on findings this may warrant the development of a new psychometric tool that captures PPPD experiences without considering a tolerance to television/movies and games.

5.8. Using Healthy and Sub-Clinical Participants to Explore Persistent Postural-Perceptual Dizziness

In clinical research it is not always possible, nor ethical, to continually access patient groups for research purposes. What becomes clear from taking a holistic view of the Thesis is that healthy participants free from PPPD, and sub-clinical PPPD participants, can be used as research participants. However, their use should be dependent on the research design and methods chosen. Recent research by Powell, Derry-Sumner, Rajenderkumar, Rushton and Sumner (2020) identified that in the generally healthy population, approximately 11% of people self-report PPPD symptoms that are equal to, or greater than, those symptoms reported by PPPD patients. They successfully probed dizziness using questionnaire-based methods (see the section 1, Chapter 1). Data presented in Chapter 2 and 4 support the existence of PPPD symptoms in the healthy student population. Data also suggest that the use of the healthy student and sub-clinical student PPPD are useful participants pools to explore self-reported PPPD symptoms.

One general implication of this Thesis is that the use of the healthy student population as a sample to explore visual dependence may not be promising. The experiments presented in Chapter 3 suggest that whilst significant changes in the postural marker of visual dependence can be identified as result of exposing participants to optokinetic stimulation, effect sizes are often small and hard to replicate.

Taken together, these experiments lead to the conclusion that exploring visual dependence in healthy student participants are not a promising avenue for research. This may be due to this group not presenting with an over-dependence on vision. It is important that future researchers be aware that participants with typical levels of visual dependence may not be a scientifically promising sample for empirical investigations that aim to reduce markers of visual dependence. No comments can be made about the promise of sub-clinical participants in laboratory experiments measuring visual dependence.

5.9. General Strengths and Limitations of the Thesis

This section of the discussion Chapter provides the reader with general strength and limitations of the Thesis. Specific strength and weaknesses have been highlighted within each individual Chapter of the Thesis; however, this section deals with general higher-level critiques on the research methodologies and paradigms employed to fulfil the doctoral Thesis.

5.9.1. The Pragmatists Mixed-Methods Approach

The epistemological position of this Thesis aligns with the pragmatists approach (Feilzer, 2010; Howe, 1988). The pragmatists approach to scientific endeavours accepts all ontological approaches. It accepts that knowledge is constructed with one single truth that can be measured and detected; this is known was the positivist approach (Hudson & Ozanne, 1988). However, it also accepts that knowledge and can be a fallacy and reflect the lived experiences and constructed social reality humans operate in. Realties are therefore multiple and relative; this is known as the interpretivist position (Lincoln & Guba, 1988). Aligning with one approach can bind the researcher to tools only available to one ontological stance. Thus, taking the pragmatists approach allows for a researcher appreciate the strengths and weaknesses of qualitative and quantitative research methods and apply the most appropriate research methods to the most appropriate research questions. Thus, a key strength of this Thesis is therefore its reflexive pragmatic approach that uses mixed methods to probe PPPD in the most appropriate manner.

5.9.2. Poor Representation of Actual PPPD Patients

Much of the research collected in this Thesis explored PPPD symptoms in the healthy student population and/or sub-clinical student populations. As such, a key limitation of the research conducted is the poor representation of PPPD patients in the studies conducted. The research studies do, however, offer data to that explore PPPD symptoms outside of patient populations. This is a research area neglected, to date. Thus, this research has provided value knowledge in regard to PPPD

symptoms in the healthy student population; however, findings may not be applied to the PPPD patient group without the replication of these studies with the PPPD cohort.

5.9.3. Promoting a Sensitivity to Context

A sensitivity to context refers to generating and analysing data and making interpretations of findings that is sensitive to the social context and complex psycho-social factors that may affect data interpretations (Yardley, 1993). Sensitivity also refers to the generation to the interpretations of data that have given a deeper understanding to a complex phenomenon (Yardley, 1993). The empirical works produced as part of this doctoral Thesis have broadened the scope of PPPD assessment by exploring psycho-social factors that may affect PPPD symptoms and by exploring symptoms outside of clinical communities. These investigations have provided the scientific community with a more rigorous understanding of the PPPD condition showing that there are unexplored psycho-social factors that are associated with PPPD. Findings have also suggested that some assumptions within the literature do not best explain PPPD experiences. For example, it is assumed that PPPD would produce an intolerance or avoidance to television/movies and gaming (see Dannenbaum, Chilingaryan & Fung, 2011; Yagi et al., 2019). Data presented in Chapter 2 suggest this may not be the case. Furthermore, PPPD is best explained by visual dependence. Data presented in Chapter 4 suggest that this theory may not account for the existence of symptoms outside of clinical populations. Thus, holistically this Thesis has provided the scientific community with a greater sensitivity to context in relation to the PPPD condition.

5.10. Future Research Directions

Throughout this Thesis suggestions for future research have been made, either explicitly or implicitly. In the final section of Chapter 5, future research directions are posited. Recommendations are based on the empirical works collected in this Thesis.

5.10.1. Explaining Persistent Postural-Perceptual Dizziness Symptoms by Accounting for Psychosocial Factors

The theory of visual dependence may not be able to explain the symptoms in all PPPD patients as approximately 15% of patients report an initial psychological illness rather than vestibular (see Staab et al.). Furthermore, data from Chapter 4 suggests that this theory may not explain symptoms in non-clinical samples. Data from Chapter 2, does however suggest that psycho-social factors associated with PPPD symptoms can explain approximately 14.8% – 20.3% of the variance in symptoms. A figure which may be heightened in the PPPD community. Future research should look to establish the

degree to which psycho-social factors can explain PPPD symptoms in the clinical PPPD group and sub-clinical PPPD population. This may allow for a more comprehensive understanding of how psycho-social factors can explain PPPD symptoms. This may also provide the scientific community with a viable alternative to the theory of visual dependence to explain symptoms in the healthy student and sub-clinical student populations.

In addition, in Chapter 2 novel psycho-social factors have been associated with PPPD. Such factors include sleep, somatic amplification, depression and out of body experiences. Future research should look to better understand how these factors interact with the PPPD condition.

5.10.2. Gamifying Rehabilitation for Persistent Postural-Perceptual Dizziness

The optokinetic stimuli used in the rehabilitation and management of PPPD may be considered monotonous and unengaging, (see stimuli presented in: Meldrum et al., 2012; Whitney et al., 2006). Although the research collected in Chapter 3 explored the functional role of video games for reducing markers of visual dependence this was not trialled with the PPPD patient cohort. As such, virtual video games may be promising rehabilitation tools for those with PPPD.

Future research should look to conduct a randomised control trial to test if exposure to optokinetic stimulation (in the form of active and/or passive video games) are effective rehabilitation tools and how they perform in relation to typical clinical stimuli. This exploration would establish the promise of active and passive virtual video games in the management of PPPD. This study should be conducted over the typical clinical timeframe of 8-16 weeks (Umphred & Lazaro, 2012). Measures should be taken throughout the program. This would also help clarify the timeframes of any effects. No claims of the efficacy of virtual video games as rehabilitation tolls for the PPPD patient group can be made until such an enquiry has been conducted. Note, such a paradigm should also measure self-reported vestibular and PPPD symptoms and psychological state; in line with RCTs in the field (Pavlou, Bronstein & Davies, 2013).

5.10.3. Immersive Movies as Rehabilitation Tools

Immersive movies, if selected carefully, may offer the same optokinetic properties as currently available rehabilitation tools. As such, immersive movies that are high in visual motion, enjoyable and engaging may be a beneficial alternative to monotonous and unengaging rehabilitation tools for PPPD. The research reported in Chapter 3 suggests immersive movies show promise at reducing the underlying dependence on vision. However, these studies were conducted with a non-clinical samples. There is a need to conduct research hat the establishes whether or not such movies are clinically

effective tools. Future research should look to replicate the studies collected in Chapter 3 with the PPPD patient community. There is also need to explore whether or not more engaging stimuli distract patients, allowing for longer exposure times during rehabilitation.

5.10.4. Correlating Persistent Postural-Perceptual Dizziness Symptoms with Performance on the Rod and Disk Test

The findings reported in Chapter 4 suggest that, in the non-clinical and sub-clinical student population, performance on traditional measures of visual dependence do not correlate with PPPD symptoms. There is however another measure of visual dependence, predominantly used in clinics: Rod and Disk test (Guerraz et al., 2001; Roberts, Melo, Siddiqui, Arshad & Patel, 2016). The Rod and Disk test is a measure of visual dependence that works as a symbiosis of the Rod and Frame and the postural sway test. In order to corroborate the findings presented in Chapter 4 future research should look to correlate scores on this alternative measure of visual dependence with PPPD symptoms. This work is required to be able to be confident that in the student non-clinical and sub-clinical samples, visual dependence is not related to PPPD symptoms.

5.10.5. Hermeneutics in Clinical Interactions with Persistent Postural-Perceptual Dizziness Patients

The IPA method operates based on hermeneutic principles of interpretation (Smith, Flowers & Osborn,1997, Smith, Jarman & Osborn, 1999). Chapter 2 highlights the success of the method for promoting candid disclosure of experiences associated with PPPD. As the diagnosis of PPPD is made based on, fundamentally qualitative, interactions with patients, applying the principles of IPA may benefit clinical assessments and diagnosis. Future research should look to explore this using a mixed-methods design with clinicians. One potential paradigm could include randomly allocating clinicians to a typical assessment group or hermeneutic based assessment

5.10.6. Increasing Qualitative Sample Sizes by Data Mining Lived Experiences

Qualitative data from PPPD patients is difficult to ascertain due to multiple contraints such as the need for NHS ethical clearance, limited access to patients, and the need for in depth qualitative research training – often neglected within psychological science training (Daher, Carre, Jaramillo, Olivares & Tomicic, 2017; Henwood & Pidegon, 1992). The digital age has, however, changed the way people communicate, share experiences and seek support from others (Graham, 2015; Katz, Nissan & Moyer, 2004; Moreno, Navarro, Tench & Zerfass, 2015; Wood & Smith, 2004). Social media has become a daily used tool (for some) for mediated social interactions and therefore houses a great deal of readily available qualitative data that is accessible to the public. Future research should

aim to expand on the qualitative enquires presented in Chapter 2 by mining social media data on the PPPD condition. A key limitation of the qualitative research presented in Chapter 2 is the small patient sample size. Data mining qualitative accounts of the lived experience of PPPD may therefore allow the results to be verified in a substantially larger diverse cohort. Furthermore, digital qualitative accounts would be free from any potential bias from power dynamics between researcher and participant. Qualitative data mining as a social research method is, however, is still in its infancy. Thus, this may require researchers to manually source online qualitative data; a task which may be time-consuming and laborious. Finally, to date, there are no clear guidelines documenting what constitutes as ethical practice when mining data from online platforms. This should be considered, or developed, before qualitative enquiry begins.

5.11. Summary

Persistent Postural-Perceptual Dizziness (PPPD) is a functional (non-organic), debilitating neuro-vestibular condition characterised by chronic episodes of visually induced dizziness (Bronstein, 1995; Staab et al., 2017). Symptoms are triggered by situations of visuo-vestibular conflict, intense visual-environments and active and passive motion (Bronstein, 2004; Pavlou, 2010). PPPD is thought to develop due to an over reliance on vision for postural control mechanisms (Bronstein, 1995). The aim of this Thesis was to explore factors associated with PPPD. The aim of Chapter 2 was to explore how individuals with PPPD make sense of their symptoms and condition and to better understand the lived experiences of PPPD, including the psycho-social impacts of the condition. Findings suggest identity loss, dismissal and non-belief, poor psychological well-being, out of body experiences and processes of sense-making are factors associated with PPPD. Shared themes included poor metal well-being, sensory overload, sleep impairments and PPPD not affecting television/movie watching. The aim of Chapter 3 was to test if short-term exposure to optokinetic stimulation can produce changes in markers of visual dependence and to establish the promise of multi-media technology for producing recalibration effects. Findings suggest that passively viewing movies are the most effective optokinetic stimulus for recalibration effects. The aim of Chapter 4 was to explore the relationships between self-reported PPPD symptoms and performance on traditionally used laboratory measures of visual dependence in the non-clinical student and sub-clinical student populations. Findings suggest that PPPD symptoms do not correlate with levels of visual dependence. Furthermore, traditional measures of visual dependence also failed to correlate. Taken together, this Thesis provides the scientific community with a deeper understanding of factors associated with PPPD in clinical and non-clinical communities.

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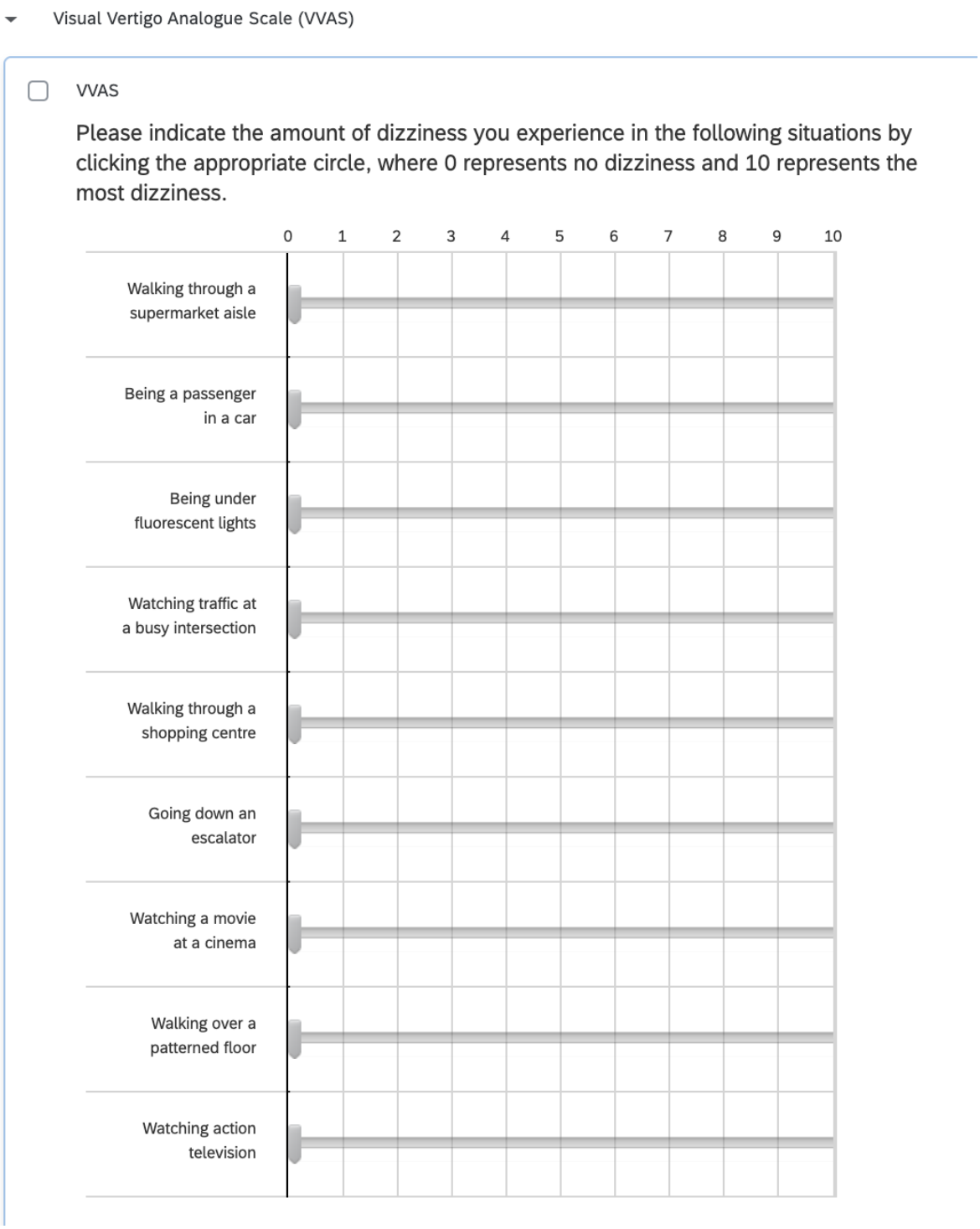
Appendices

Appendix 1.1. The Visual Vertigo Analogue Scale (VVAS).



Appendix 1.2. The Simulator Sickness Questionnaire (SCQ).

Simulator Sickness Questionnaire

Q89

Please rate the following issues/symptoms you experience when exposed to simulated motion from a multi-media technology (television, movies, video games, virtual reality exposure).

	None	Slight	Moderate	Severe
General discomfort	○	○	○	○
Fatigue	○	○	○	○
Headache	○	○	○	○
Eyestrain	○	○	○	○
Difficulty focusing	○	○	○	○
Increased salivation	○	○	○	○
Sweating	○	○	○	○
Nausea	○	○	○	○
Difficulty concentrating	○	○	○	○
Fullness of head	○	○	○	○
Blurred vision	○	○	○	○
Dizziness (eyes open)	○	○	○	○
Dizziness (eyes closed)	○	○	○	○
Vertigo	○	○	○	○
Stomach awareness	○	○	○	○
Burping	○	○	○	○

Appendix 2.1. The Simulator Sickness Questionnaire (SCQ).

Supplementary Chapter S2.1: Persistent Postural-Perceptual Dizziness (PPPD) and Cyber-Sickness: Understanding the Relationship Between the Two Sister-Conditions

Introduction

Cyber Sickness is defined as a unique form of motion sickness that results from the stationary observer being exposed to virtual visual motion patterns that give a compelling sensation of self-motion and/or vection (LaViola, 2000). From this definition it is clear that Cyber Sickness appears to be a similar condition to PPPD but relegated to the virtual world and the by-product of engaging with simulated, rather than real-world motion. As such, the syndrome is often given the term 'Simulator Sickness' (Kennedy, Lane, Berbaum & Lilenthal, 1993); however, the appropriateness of this is debated within the literature (see Stanney & Kennedy, 1997). Given the similarities between the conditions, some suggest that Cyber Sickness is not a unique form of visually induced dizziness, rather a general visually induced dizziness from motion patterns that are virtual in their nature (Mazloumi, Walker, Hodgson & Nalivaiko, 2018). This may suggest that the condition is the same as PPPD, simply bound to the virtual world. However, it should be noted that Cyber Sickness involves factors such as eyestrain due to convergence, accommodation cues, fixed focal demands, and, where relevant the 'fit' of head mounted displays. These do not appear to have obvious equivalents in PPPD. However, this does not mean that the two conditions are unrelated and there may be some unifying factors that explain both conditions. Regardless of the interpretations of what constitutes Cyber Sickness and how it is conceptualised, what is clear is that the two forms of visually induced dizziness may be related and may be explained by shared factors.

Although the conditions are similar, the questionnaire-based tools that capture PPPD and Cyber Sickness measures probe vertiginous experiences differently. The Visual Vertigo Analogue Scale (VVAS: Dannenbaum, Chilingaryan & Fung, 2011), is a commonly used measure of PPPD (see Chapter 4 Method section) that explores the dizziness across visual scenarios known to elicit symptoms. Whereas, Cyber Sickness is traditionally measured using the Simulator Sickness Questionnaire (SSQ: Kennedy, Lane, Berbaum & Lilenthal, 1993). The SSQ explores a range of diverse symptoms associated with vertigo (dizziness, eyestrain, sweating etc.) post exposure to an

optokinetic stimulus. These scales probe visually induced dizziness differently. The VVAS is concerned with solely with dizziness, whilst the SSQ explores a more diverse range of symptoms associated with vertigo. Thus, if used in conjunction these two measures could help us better understand the symptoms of PPPD, probing not only dizziness but a diverse range of symptoms associated with vertiginous experiences.

Aims and Objectives

In order to better inform the qualitative enquiries conducted in Chapter 2, supplementary research was conducted to better explore PPPD experiences. The aim of supplementary Chapter S2.1 was twofold. First, the researcher aimed to better understand PPPD symptoms and their relationship with Cyber Sickness. Currently available measures of PPPD probe activities such as television and cinema viewing, assuming that these forms of visual motion should trigger symptoms. However, initial conversations with patients suggest this may not be the case. Second, symptoms of PPPD and Cyber Sickness overlap; however, they explore experiences differently. PPPD measures assess the level of dizziness an individual experiences in a specific visual scenario. In contrast, Cyber Sickness measures assess a diverse range of symptoms associated with vertiginous experiences in one given scenario. Thus, the first objective of supplementary Chapter S2.1 was to establish if PPPD and Cyber Sickness symptoms are correlated. The second objective was to establish if diverse symptoms cluster meaningfully in order to better shape questions around the lived experiences of PPPD in the qualitative enquiries of Chapter 2.

Exploring the Relationships Between Persistent Postural-Perceptual Dizziness (PPPD) and Cyber Sickness

The aim of this supplementary research was to assess if PPPD symptoms correlate with Cyber Sickness and if experiences could be explained by shared factors. Thus, the first objective of Study 1 was to correlate scores on the VVAS with scores on the SSQ in the healthy student population. It was hypothesised that the data would show a positive correlation between the two scales. The second objective was to establish if there were shared factors that could explain the conditions. It was hypothesised that symptoms may cluster meaningfully representing factors that could be further probed in the qualitative enquires of Chapter 2.

Method

Study 1: Participants

Two-hundred and ninety-five participants (258 females; 37 males) completed a short ten-minute online study for course credit. Information about the study was administered online and consent taken digitally. Data was collected during an imposed lockdown. No wider demographic details were collected. No exclusion criteria were applied. These students were recruited from the sample undergraduate psychology participant pool. Participants may therefore be the same as participants in Chapter 3 and 4.

Materials

The Visual Vertigo Analogue Scale (VVAS)

The Visual Vertigo Analogue Scale (VVAS: Dannenbaum, Chilingaryan & Fung, 2011) quickly identifies whether an individual shows the characteristic symptoms of PPPD. See Chapter 4, section 4.2.1.2.2, for the psychometric properties of the scale. See Appendix 1.1 for the VVAS scale.

Simulator Sickness Questionnaire (SCQ)

The Simulator Sickness Questionnaire (SSQ: Kennedy, Lane, Berbaum & Lilenthal, 1993) is a 16-item questionnaire that captures physiological responses to virtual motion. Respondents are given 16 symptoms and asked to state the degree to which exposure to virtual motion has induced each symptom. Participants are free to respond with a value of 0 - 3 where 0 represents 'no experience of that symptom' and 3 represents 'severe symptoms'. The survey captures three unique composites of Cyber Sickness symptoms: nausea, oculomotor and disorientation. Seven items capture symptoms that fall under the factor of nausea such as 'general discomfort', 'sweating' and 'difficulty concentrating', seven capture oculomotor symptoms such as 'eye-strain' and 'blurred-vision' and seven capture disorientation such as 'vertigo', dizzy with eyes open' and 'dizzy with eyes closed'– note items are allowed to represent more than one underlying factor and thus only 16 items are used but some are represented in numerous sub-scales. For example, 'nausea' represents both nausea and disorientation. Scores are summed and then multiplied by a varimax factor weight which operates as a constant scaling factor. The weighting system and associated constants are necessary for two important reasons: first, they allow the data to represent no report of symptoms as 0. Second, the standard deviation of the sample provided by Kennedy et al. is 15 for the total sample of approximately 1200 respondents. Due to the large sample size and appropriate standard deviation formed, the data provided in the paper can be used as a population norm group to compare newly acquired data against. The nausea sub-scale score is calculated by summing all relevant items and multiplying this

value by 9.54. The oculomotor sub-scale is formed by the sum of the seven relevant items multiplied by 7.58 and the dizziness sub-scale by the sum of disorientation items multiplied by 13.92. The overall SSQ score is computed by taking the sum of all scores and multiplying the value by 3.74. Total SSQ scores may range between 0 – 179.52. Missing scores were awarded 0. See Appendix 4.1 for the SSQ.

Procedure

Participants were invited to take part in a short online study aiming to assess the relationships between visually induced dizziness in the physical and virtual world(s). Participants were free to complete the survey on any internet connected device. Consent was taken digitally and upon completion of the study participants were, digitally, debriefed and awarded course credit.

Results

Descriptive Statistics

Eight participants did not complete the SSQ leaving N = 287. The Median Absolute Deviation (MAD) or Modified Z (Iglewicz & Hoaglin, 1993) to detect outliers on the variables of interest. Any score +/- 3.5*MAD were removed from the analysis. The mean scores on the scales were as follows: VVAS = 14.59 (12.13), SSQ = 35.56, Nausea SSQ Sub-Scale = 20.48 (18.74), Oculomotor SSQ Sub-Scale = 47.60 (29.09) and Disorientation SSQ Sub-Scale = 49.50 (46.19).

Correlating Symptoms of Persistent Postural-Perceptual Dizziness and Cyber Sickness

Data are depicted in Figure S2.1. Visual Vertigo Analogue Scale (VVAS) scores were shown to correlate significantly with Simulator Sickness Scores ($r = 0.59$, $p = <.001$) with the relationship being of moderate strength. When VVAS scores were correlated with each of SSQ sub scales evidence of a moderate correlation between VVAS and the a) nausea ($r = 0.48$, $p = <.001$), b) oculomotor ($r = 0.56$, $p = <.001$) and c) disorientation ($r = 0.56$, $p = <.001$) sub-scales were detected. The data suggest there are moderate significant correlations between PPPD and general Cyber Sickness experiences and with each of the three unique sub-scales that make up the wider SSQ.

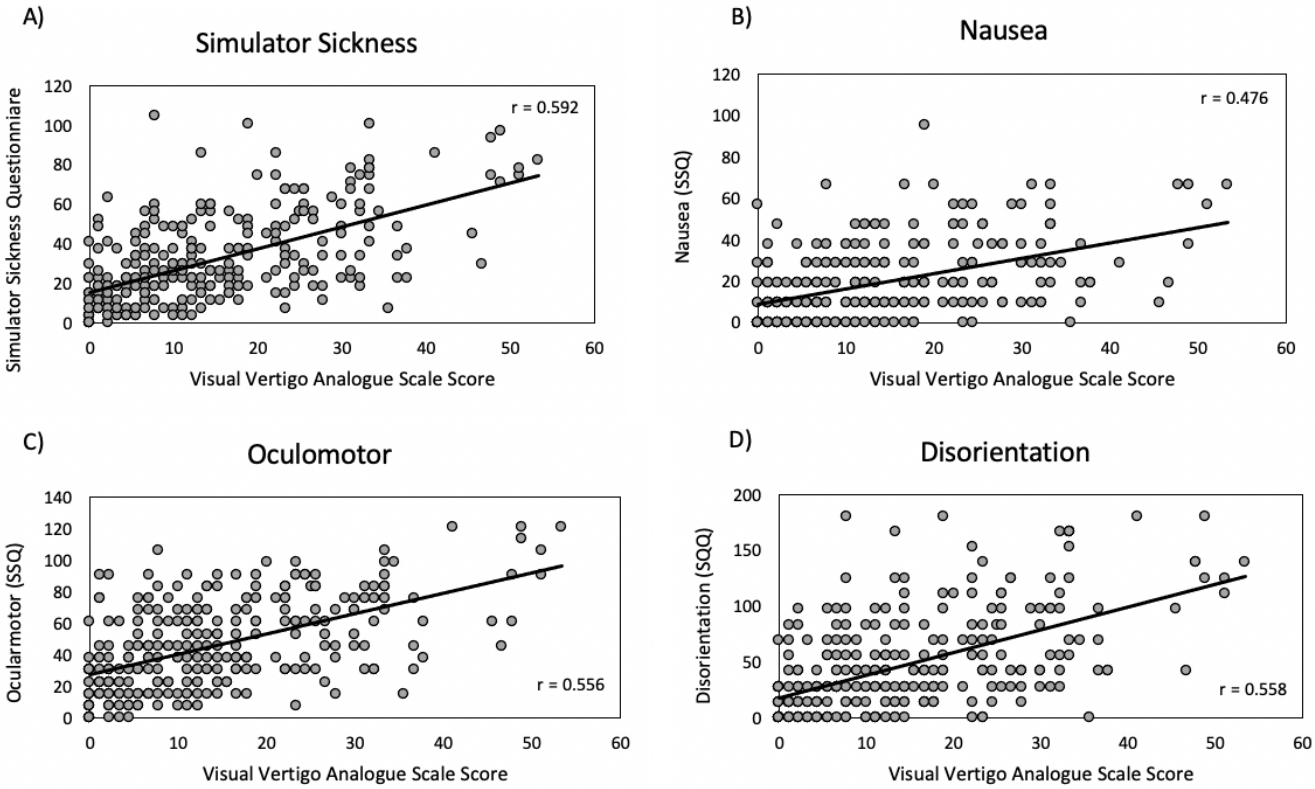


Figure S2.1. Scatterplot of Visual Vertigo Analogue Scale (VVAS) scores and Simulator Sickness Questionnaire (SSQ) scores. A) The relationship between VVAS scores and the general Simulator Sickness Questionnaire (SSQ) Score. B) The relationship between VVAS scores and the Nausea SSQ sub-scale. C) The relationship between VVAS scores and the Oculomotor SSQ sub-scale. D). The relationship between VVAS scores and the Disorientation SSQ sub-scale.

Are There Shared Underlying Factors that Explain both Persistent Postural-Perceptual Dizziness and Cyber-Sickness?

The second objective of this study as to establish if there were shared factors that could explain the conditions. This would also allow for a better understanding of symptoms to inform question design for Chapter 2. One useful statistical tool explore underlying factors and symptom clustering is Factor analysis.

Planned Statistical Analyses: Introducing Factor Analysis

Factor analysis is a set of multivariate statistical methods for data reduction to allow for a more parsimonious understanding of a given set of measured variables to establish the number and nature of common factors that best explain the patterns of observed correlations between items (Fabringer & Wegender, 2011; Fabrigar, Wegener, MacCallum & Strahan, 1999; Pearson, 1901). Exploratory

Factor Analysis (EFA) is unique form of factor analysis that can be used to establish underlying factors when little to no relationships or theoretical clusters between variables are known (Cudeck, 2000a; Cudeck, 2000b; Lorenzo-Seva & Ferrando, 2006). It is therefore considered a first 'port of call' to establish clusters of variables that best explain the factors that underlie and unify variables with the aim of establishing the most parsimonious explanation of the data. Thus, for initial EFA models the nature of factors is more important than traditional 'goodness of fit' indices. I do however report these nonetheless for context and completeness.

There a several commonly used indicators of the goodness of fit of a factor analysis model. Key indicators include the Root Mean Square Error of Approximation (RMSEA), Bayesian Information Criterion (or Schwartz Information Criterion; Schwartz, 1978) and the Tucker-Lewis Index. For the RMSEA a value of >0.05 is considered good, 0.05 – 0.008 is considered acceptable and anything >0.08 is considered poor (Fabrigar, Wegener, MacCallum & Strahan, 1999; Xia & Yang, 2019) For TLI, Bentler and Bonnett (1980) suggest that a value of >0.90 represents a good fit. Finally, for BIC a value below 2 is too small to be statistically significant in comparison to the alternative or null hypothesis. A value of 2 – 6 represents that the alternative model is positive, whereas a value between 6-10 represents evidence for the best model (Claeskens & Hjort, 2008; Schwartz, 1978). As the value increases above 10 this represents an increase in the strength of the model. For more on fit indices see Xia and Yang (2019) or Claeskens and Hjort (2008).

Exploratory Factor Analysis (EFA) Model

Data were analysed by an Exploratory Factor Analysis (EFA) model using scores on the 9 items of the VVAS and the 16 items of the SSQ. Due to the most common response on VVAS and SCQ items being 0, raw data was used for the analysis without removing outliers that were 3.5*MAD as the median for multiple items is 0 which would result in any item >0 considered an outlier.,

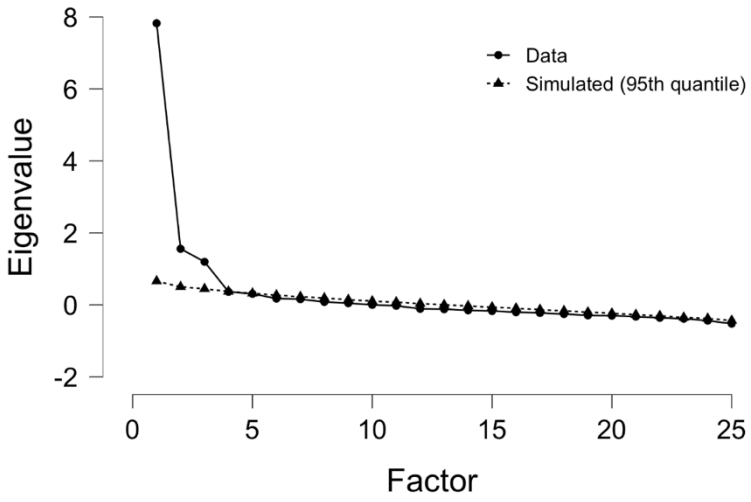


Figure S2.2. Scree plot for the Exploratory Factor Analysis (EFA) model showing the representation of the data and simulated data eigenvalues.

The EFA model indicated the presence of three unique factors that unified items on the two scales of interest. The Scree plot and Table of factor loadings are presented in Figure S2.2 and Table S2.1. The Scree plot indicates a three-factor model to explain the relationships and cluttering patterns within the data. Complimenting this, items appear to load onto three unique factors. Items that load onto any factor at the threshold of 0.40 were interpreted as an indicative measure of the underlying factor.

Bartlett's test of sphericity indicated that the correlational structure is adequate for factor analysis: χ^2 (300) = 3197.74, p = <.001. The minimum residual factor analysis with a cut-off point of 0.40 using the parallel analysis method with oblique rotations meaning that factors could correlate (Hayton, Allen & Scarpello, 2004) yielded a three-factor solution as the best fit of the data, accounting for 43.8% of the variance. Factor loadings are presented in Table S2.1 and are visually depicted as a Path diagram in Figure S2.3.

Factor 1 comprised eight items. These eight items explained 17% of the variance in thus specific factor with factor loadings ranging from 0.41 – 0.78. The second factor compromised 7 items and these 7 items explained 14.9% of the variance in the factor. Factor loadings ranged from 0.466 – 0.881. The third and final factor, factor 3, comprised 7 items which explained 11.9% of the variance. These loadings ranged from 0.51 – 0.66. Cumulatively, the factorial model explains 43.8% of the total variance.

The model's goodness of fit indices were as follows: (RMSEA) = 0.07; RMSEA 90% Confidence = 0.06 – 0.07; TLI = 0.87; Bayesian Information Criterion (BIC or Schwartz Information Criterion;

Schwartz, 1978) = -792.79. Broadly, these statistics suggest the model is a fairly good or moderate fit, however these statistics are not required for EFA. Statistics are provided for completeness.

Interpretations

The EFA model can be interpreted as partial support for the original factor analysis presented by Kennedy, Lane, Berbaum and Lilenthal (1993) that suggested the SSQ captures three unique factors of Cyber Sickness; nausea, disorientation and oculomotor symptoms. The EFA offers evidence of the disorientation (green) and the nausea factor (yellow) see Table S2.1. However instead of the oculomotor factor the items from the VVAS, representing dizziness (blue), cluster together. Inspection of the factor loadings presented in Table S2.1 and the path diagram for the analysis in presented in Figure S2.3 show that the model partially supports the factor loadings of the simulator sickness questionnaire where the disorientation factor (green) and the nausea factor (yellow) are evident. However, the items from the VVAS probing dizziness, and thus dizziness factor (blue), clusters separately. No evidence of the third SSQ factor (oculomotor) was present. The data do however provide a better understanding of how symptoms manifest and this information can be used to inform questions that probe different experiences differently in Chapter 2. For example, the data suggests that qualitative enquires should probe dizzy, nauseating and disorienting symptoms separately.

Table S2.1

Study 1 Exploratory Factor Analysis loadings for the SSQ and VVAS.

	Study 1		
Items	Factor 1	Factor 2	Factor 3
Visual Vertigo Analogue Scale (VVAS)			
Walking through a Supermarket	-0.166	**0.861**	0.086
Being a passenger in a car	0.334	0.288	0.025
Being under fluorescent lights	0.250	**0.565**	-0.167
Watching traffic at a busy intersection	0.221	**0.575**	-0.110
Walking through a shopping centre	-0.055	**0.881**	0.010
Going down an escalator	0.177	**0.414**	0.142
Watching a movie at a cinema	0.275	0.390	0.127
Walking over a patterned floor	0.139	**0.553**	0.013
Watching action television	0.284	**0.466**	0.022
Simulator Sickness Questionnaire (SSQ)			
General discomfort	**0.408**	0.081	0.322
Fatigue	**0.461**	0.081	0.045
Headache	**0.601**	-0.38	0.063
Eyestrain	**0.773**	0.011	-0.031
Difficulty focusing	**0.775**	-0.044	-0.052
Increased salivation	-0.045	0.064	**0.561**
Sweating	-0.097	-0.064	**0.619**
Nausea	0.213	0.115	**0.554**
Difficulty concentrating	**0.654**	0.075	0.001
Fullness of head	0.394	0.185	0.223
Blurred vision	**0.661**	-0.037	0.155
Dizziness (eyes open)	**0.447**	0.093	0.365
Dizziness (eyes closed)	0.251	0.100	**0.512**
Vertigo	0.026	-0.046	**0.655**
Stomach awareness	-0.005	0.010	**0.617**
Burping	-0.063	0.016	**0.608**

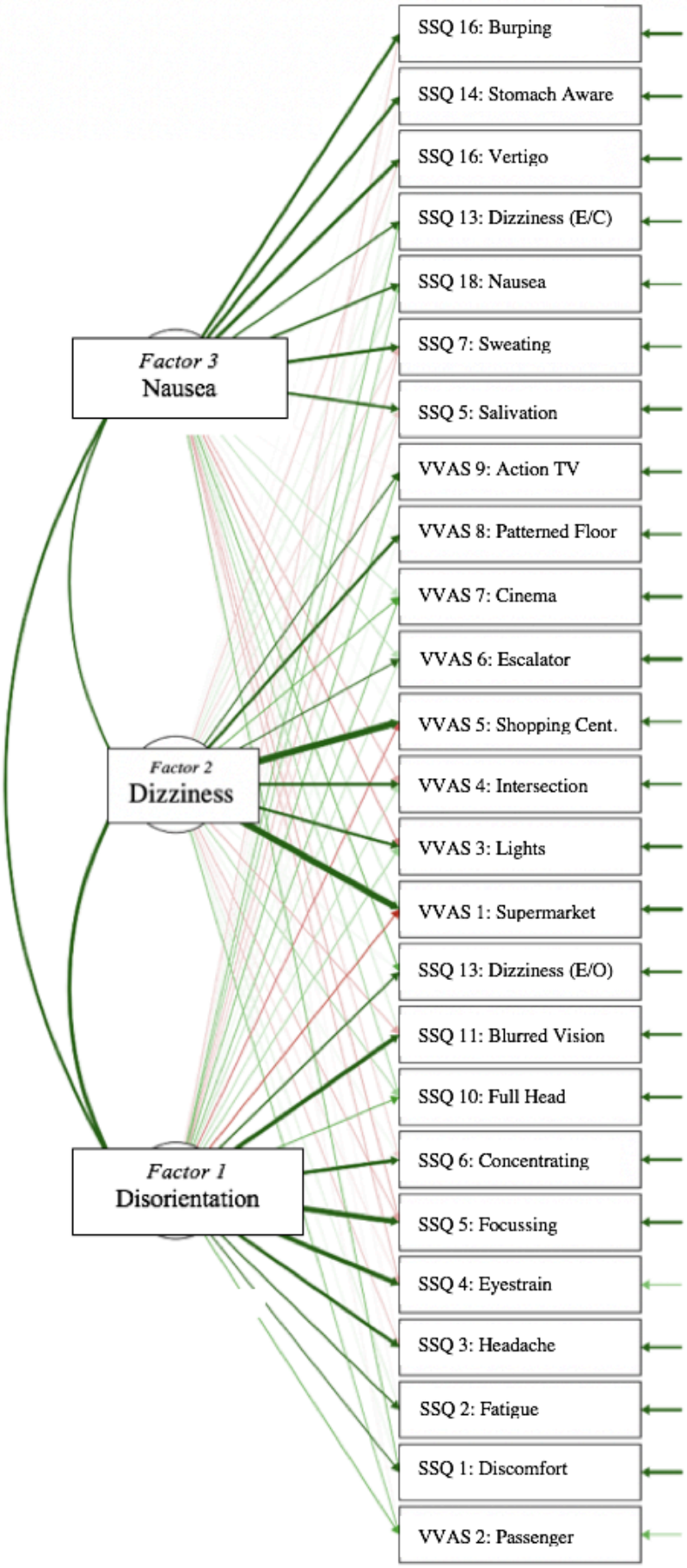


Figure S2.3. Path diagram of factors identified within the Exploratory Factor Analys

Supplementary Chapter S2.1: Discussion

Characteristic symptoms of PPPD over-lap with those of Cyber Sickness (Mazloumi, Walker, Hodgson & Nalivaiko, 2018). As such, theorists have suggested that Cyber Sickness is not a unique form of visually induced dizziness, rather a general visually induced dizziness from motion patterns that are virtual in their nature (Mazloumi, Walker, Hodgson & Nalivaiko, 2018). However, the way these two similar conditions are explored using psychometrics differ greatly. In order to better inform the qualitative enquiries conducted in Chapter 2, supplementary research was conducted to better explore PPPD experiences. The aim of this supplementary Chapter S2.1 was to better understand the relationship between PPPD and Cyber Sickness and explore the clustering factors that underpin conditions. The findings from supplementary Chapter S2.1 suggest that PPPD and Cyber Sickness symptoms are moderately correlated and that symptoms cluster around those that produce dizzy symptoms, nauseating symptoms or disorientation. These findings could be used to better inform qualitative enquires probing the lived experiences of the PPPD condition.

The Manifestation of Visually Induced Dizziness

Experiences of subjective dizziness seemed to manifest in tandem with vertiginous/sickly vestibular episodes, as well as the disorienting light-headedness. This is in line with previous research that suggests that chronic subjective dizziness is not just dizziness but manifests with nauseating vertigo and/or confusing, discombobulating experiences (Baloh, 1995, Barraclough & Bronstein, 2009, Bronstein, 1995; Bronstein, 2004; Bronstein, 2013; Ruckenstein & Staab, 2009). The data presented in Chapter S2.1 suggest that the manifestation of dizziness is not always clear however three unqiue types of experience and/or symptoms manifest when exposed to triggers of PPPD. What this suggests is that dizziness is a complex sensory experience that usually manifests with sickly or confusing experiences - both of which can be debilitating and highly uncomfortable. This information has isolated experiences of PPPD which may be used to infrom the qualitative enquiries of Chpater 2.

Failing to Replicate All Three Factors of the Simulator Sickness Questionnaire (SSQ)

The factor analysis presented in supplementary Chapter S2.1 shows partial support for the initial factor analysis presented in Kennedy, Lane, Berbaum and Lilenthal's (1993) seminal paper on simulator sickness. The analyses did however fail to establish the third oculomotor factor evident in the Kennedy et al. paper. This may suggest that the oculomotor factor is not present in the student population and/or in contemporary experiences. This would in turn require either empirical validation and/or an update to psychometric properties of the scale, respectively. Although to some degree dated,

the SSQ has been well validated and used in research for over 20 years (see Balk, Bertola & Inman, 2013) which would suggest that the tool itself is operational and functioning according to its design. A The oculomotor factor may not have been visible due to adding items from the VVAS and/or changing the way in which the SSQ is typically administered. Historically, the tool is used immediately after simulated motion. I asked participants to reflect on simulated motion rather than actually administer visual motion. Removing the active exposure to motion may have caused the psychometric tool to operate under different conditions and may therefore explain why the current work failed to fully replicate the factors in the SSQ.

Note, previous research has challenged the factorial composition of the SSQ suggesting that there are actually only two specific factors when used with specific user groups (Bouchard, Robillard & Renaud, 2007). Therefore, the user group in question may affect the factors which explain visually induced dizziness. Future research should establish what factors emerge from the SSQ in the student population and PPPD patient group and if factors differ from healthy control users. For completeness research should also look to re-establish the psychometric properties of the SSQ to see if 20 years later norm group data would show the same patterns and the same factorial composition. This is crucial as the SSQ is currently the leading psychometric tool for capturing Cyber Sickness in both research and applied sciences (Bimberg, Weissker & Kulik, 2020).

The Administration of the SSQ

It is also important to note that traditionally the SSQ is administered immediately post simulated/immersive experience such as a flight simulator (Kennedy, Lane, Berbaum & Lilenthal, 1993; Kennedy, Lane, Lilenthal, Berbaum & Hettinger, 1992; Kennedy, Lilenthal, Berbaum, Baltzley & McCauley, 1989; Pausch, Crea & Conway, 2017). In my studies I simply asked respondents to state how ‘simulated motion’ made them feel. As no controlled stimuli was administered the experiences the respondent may have been ruminating on could be extremely diverse. Furthermore, people’s mental models of what simulated motion is may not converge idea and thus may not represent the same sensory experience. In short, different people may be thinking about different types of virtual motion. Future research should look to replicate this work but administer the SSQ immediately after virtual motion.

Chapter S2.1: Summary

Some theorists have suggested that Cyber Sickness is not a unique form of visually induced dizziness, rather a general visually induced dizziness from motion patterns that are virtual in their nature (Mazloumi, Walker, Hodgson & Nalivaiko, 2018). Given this criticism it is therefore fair to suggest that Cyber Sickness and PPPD may correlate and be explained by some shared unifying factors. This Chapter aimed to explore if these two conditions were correlated and could be explained some shared factors. This research was conducted in order to support the development of an interview guide for qualitative enquires into the lived experiences of PPPD (see Chapter 2). The findings presented supplementary Chapter S2.1 suggest that although the conditions are moderately correlated and have overlapping symptoms, they are not the same condition and were not explained by the same underlying factors. Findings suggest that qualitative enquires should specifically probe different experiences of PPPD such as those that result in dizziness, nausea and disorientation.

Appendix 2.2. Semi-Structured Interview Guide and Questions.

IPA Interview Guide: Exploring the Lived Experiences of Persistent Postural-Perceptual Dizziness

Interview type:

PPPD patients: Patients referred by Hannah-Derry Sumner and Dr Deepak Rajenderkumar at the University Heath Hospital (Department of Audiology) or other PPPD patients who raised their interest in taking part.

Prior to interview (15 minutes)

1. Introduction
2. Introduction of researcher, the project and each participant
3. Ask visual vertigo analogue scale (VVAS) questions
4. Consent for the interview to be recorded – record on both Ryan and Kev's computers
5. Back up audio recording using Dictaphone
6. Outline the plan and nature of the interview (sense making, experiential, setting expectations such as establishing if break will be needed, time limitations, remind them about the study being recorded etc.)

Time (minutes)	Topics for discussion
15	Start up 1. Introduction Re-cap: introduce both Ryan Gamble and Dr Kev Wilson-Smith to help with the participant telling us about themselves in the interview. 2. Briefly outline the study and explain its purpose. We want to understand your 'clinical diagnosis', how you make sense of that and the psychological impact is having. 3. Introduce the participant to the topic of discussion and what is of particular interest 4. Let each participant briefly explain about their dizziness (onset, duration, triggers, management etc.)
45	Consultation questions The questions asked should help to identify the symptoms the participant experiences, and gain information about how these symptoms affect the patient's psycho-social wellbeing.
	• Firstly, could you tell us a little about yourself. What are you like as a person? Are you logical, irrational, emotional, calm etc? What do you do for a living? How do you identify? What do you do in your spare time? • Can you tell me about the first time you ever experienced dizziness? Where were you and who were you with? Would your experience have changed if you were somewhere else or with someone else? • How would you describe how you experience dizziness and vertigo? How would you explain it and describe it to a friend? • When you think back to the first time you experienced dizziness what was happening before the episode? Or have you always been dizzy? Was there a 'triggering' event? Anything important happening in your life when dizziness began? What were you thinking and feeling?

	• When this began did the experience scare or excite you? In what ways? Is that the same now? • What impact did this have on you today? Do you think it affected you at all? Health, happiness, mental health, relationships? • What were your relationships, health, mental health like before dizziness? Did they change after? Could you give us examples? What are they like now? • Do you think anything brings on your dizziness/ How do you feel just before an episode? Think of scenarios – what was happening around the time of the attacks? What were your thoughts, what was going on in life? • Are any parts of your life impaired? Were they initially and are they now? Are there any things that you can/cannot do now due to your dizziness? Can you give examples and explain why you think dizziness may be affecting it? Can you use transport? Does this affect your mood/well-being? Is there anything that you never would have tried but now do- yoga, mindfulness? • Is there anything surprising from your dizziness? Anything you did not expect to experience? Are there any specific experiences/episodes of vertigo that made you particularly emotional? If you have given a negative, can you think of a positive? • How do you manage your dizziness? Have your social interactions changed because of this– how do you know facilitate your relationships (same as always, telephone, social media etc)? Do people understand your dizziness? • Has dizziness affected your mental health and well-being? How would you describe your mental health before dizziness? Was anything going on? What did that look like after dizziness started and what is that like now? Could you tell me about an experience when you noticed your mental health particularly suffer from dizziness? And now please tell us about an experience where you think your mental health became stronger? • Romantic relationships – are they affected by dizziness? If so, why do you think so? In what ways are your romantic relationships no longer the same. Dating, marriages etc? • How do you feel as a person who has dizziness? Are you happy, anxious, stressed, depressed? Do you believe your mood and happiness is related to the PPPD? • How do you manage your symptoms? Can you access or manage anything that helps your symptoms? • Have you ever visited a doctor- could you tell me about that experience? Did you find it useful? In what ways did you feel supported or not supported? Did this affect you and/or your dizziness? Do you feel supported by medical staff? What do you need to feel supported? Have you ever sought any other help with your dizziness? If yes, then with who? If no, then why not? • In an ideal world, what would happen to your dizziness and related symptoms? Could you picture the perfect scenario of your life if you no longer experienced dizziness? What would you do, where would you be, who would you be with and how would you feel?
10	Exit question: • Is there anything else you would like to share or discuss? Is there anything you feel that people would benefit from knowing in regard to vertigo, PPPD and psycho-social impacts? These can be both positive and/or negative.
5	Close of the interview, thank participant, and remind them that if they would like to receive a copy of the findings that this will not be available for the next 18 months or so and establish with participant what the best way is for them to receive the information (email findings or face to face meeting?). All participants to be paid sent a £15 Amazon Voucher from Ryan Gamble. Stop Recording.

Appendix 2.3. The Pittsburgh Sleep Quality Index (PSQI) Psychometric Properties and the Sub-Components of Sleep.

The Seven Sub-Components of Sleep: The PSQI creates 7 component scores which reflect different dimensions of sleep quality. Subjective sleep quality is measured by question 9 which asks, 'during the past month how would you rate your overall sleep quality?' with responses ranging from very good (0) – Very bad (3) on a 4-point scale. Sleep latency is calculated by questions 2 and 5a where participants are asked 'during the past month, how long (in minutes) has it usually taken you to fall asleep?' where they are free to place any number in their response box. Any response of <15 minutes is coded as a score of 0, 13-30 minutes as 1, 31-60 minutes as 2 and >60 minutes as 3. They are also asked 'during the past month, how often have you had trouble sleeping because you cannot get to sleep within 30 minutes. To this second item participants must respond on a 4-point scale where 'not during the past month' represents 0, less than once a week represents 1, once or twice a week represents 2 and three or more times a week represents 3.

The sleep latency score is the sum of these 2 items where any total score of 0 is coded as 0, 1-2 as 1, 3-4 as 2 and 5-6 as 3. Sleep duration is measured by question 4 which asks the respondent, 'during the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed)' where again participants are free to enter the number of hours of actual sleep they have had. An answer of >7 hours is coded as 0, 6-7 hours as 1, 5-6 hours as 2 and <5 hours as 3. Sleep efficacy is measured by questions 1, 3 and 4 where participants are asked 'during the past, what time have you usually gone to bed at night?', 'during the past month, what time have you usually gotten up in the morning' and 'during the past month, how many hours of actual sleep did you get? (this may be different than the numbers of hours spent in bed)'. Sleep efficacy is expressed as (hours slept/hours in bed) x 100 to give an efficacy of sleep score that can range from 0% - 100%. A sleep efficacy score of >85% is coded as 0, 75-84% as 1, 65-74% as 2 and <65% as 3.

Sleep disturbance is captured by questions 5b – 5j where participants are asked items like 'during the past month, how often have you had trouble sleeping because you…', 'wake up in the middle of the night', 'have to get up to use the bathroom' and 'cannot breathe comfortably'. Responses to each of the 8 items range from 'not during the past month' (0) – 'three or more times a week' (3). The sleep disturbance component score is the sum of these 8 items and then coded. Any sum of 0 is coded as 0, a score of 1-9 as 1, 10-18 as 2 and 19-27 as 3. The use of sleep medication is captured using question six, which asks, 'during the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?' where again 'not during the past month' = 0 and 'three or more times a week' = 3. Finally, daytime dysfunction is measured by questions 7 and 8 which asks, 'during the past month, how often have you had trouble staying awake while driving, eating meals or engaging in social activity?' where a response of 'not during the past month' is 0 and 'three or more

times a week' is 3 and 'during the past month, how much of a problem has it been for you to keep enough enthusiasm to get things done?'. Answers to this item range from 'no problem at all' (0) – a very big problem (3). Scores on these two items are summed and any score of 0 is coded 0, 1-2 is coded as 1, 3-4 as 3 and finally 5-6 as 3.

Appendix 2.4. Additional Regression Models for the Niigata PPPD Questionnaire (NPQ) Sub-Scales.

NPQ Upright Posture/Walking Sub-Scale: A stepwise multiple linear regression to the data to estimate the ability of anxiety, depression, self-esteem, general mental well-being, sleep and somatic amplification in predicting PPPD symptoms, as measured by the NPQ sub-scale upright posture/walking. The model suggests that anxiety, somatic amplification and mental well-being could significantly predict participants scores on the NPQ. The regression model could explain 13.5% of the variance and that this model was a significant predictor of PPPD symptoms: $F(3, 31) = 16.11$, $p = <.001$. The model suggests that anxiety ($\beta = 0.10$, $p = 0.00$), somatic amplification ($\beta = 0.05$, $p = <.0.01$) and mental well-being ($\beta = -0.03$, $p = 0.03$) were the only predictors that significantly contributed to the model. The final predictive model was therefore, NPQ Upright Posture/ Walking = 0.43 + (0.53*Anxiety) + (0.27*Somatic Amplification) + (0.41*Mental Well-Being).

NPQ Movement Sub-Scale: A second stepwise multiple linear regression to the data to estimate the ability of anxiety, depression, self-esteem, general mental well-being, sleep and somatic amplification in predicting PPPD symptoms, as measured by the NPQ sub-scale movement. The model suggests that anxiety, somatic amplification and depression could significantly predict participants scores on the NPQ. The regression model could explain 19.8% of the variance and that this model was a significant predictor of PPPD symptoms: $F(3, 353) = 28.78$, $p = <.001$. The model suggests that anxiety ($\beta = 0.19$, $p = <.001$), somatic amplification ($\beta = 0.16$, $p = <.001$) and depression ($\beta = 0.173$, $p = 0.01$) were the only predictors that significantly contributed to the model. The final predictive model was therefore, NPQ Movement = -1.95 + (0.19*Anxiety) + (0.16*Somatic Amplification) + (0.17*Depression).

NPQ Visual Stimulation Sub-Scale: A third stepwise multiple linear regression to the data to estimate the ability of anxiety, depression, self-esteem, general mental well-being, sleep and somatic amplification in predicting PPPD symptoms, as measured by the NPQ sub scale visual stimulation. The model suggests that anxiety, somatic amplification and depression could significantly predict participants scores on the NPQ sub scale. The regression model could explain 11.7% of the variance and that this model was a significant predictor of PPPD symptoms: $F(3, 339) = 14.88$, $p = <.001$. The model suggests that anxiety ($\beta = 0.12$, $p = 0.01$), somatic amplification ($\beta = 0.10$, $p = <.001$) and depression ($\beta = 0.12$, $p = 0.04$) were the only predictors that significantly contributed to the model. The final predictive model was therefore, NPQ Movement = -1.53 + (0.12*Anxiety) + (0.10*Somatic Amplification) + (0.12*Depression).