Visuo-vestibular contributions to anxiety and fear

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Abstract

The interactive roles of the visual and vestibular systems allow for postural control within boundaries of perceived safety. In specific circumstances, visual vestibular and postural interactions act as a cue that trigger fear, similarly to what occurs in motion sickness. Unusual patterns of visuo-vestibular interaction that emerge without warning can elicit fear, which can then become associated to a certain stimuli or situation, creating a CS-US association, (i.e., phobia), or can emerge without warning but also without becoming associated to a particular concomitant event (i.e., panic). Depending on the individual sensitivity to visuo-vestibular unusual patterns and its impact in postural control, individuals will be more or less vulnerable to develop these disorders. As such, the mechanism we here propose is also sufficient to explain the lack of certain fears albeit exposure. Following this rationale, a new subcategory of anxiety disorders, named visuo-vestibular fears can be considered. This model brings important implications for developmental and evolutionary psychological science, and invites to place visuo-vestibular fears in a particular subtype or specification within the DSM-5 diagnostic criteria.

Keywords: acrophobia; anxiety; driving; hypophobia; motion sickness; panic; phobias; postural stability; sensory conflict; vestibular system.

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1 - Introduction

Presently, the DSM-5 (APA, 2013) maintains the classification of specific phobias according to five subtypes: animal, natural environment, blood-injection-injury, situational, and other. Animals such as rodents, insects, arachnids and reptiles usually trigger animal fears. Natural environment fear involves objects or situations found in nature, the examples being heights, storms, water, and the dark. The blood-injection-injuries subtype (BII) includes situations that involve blood or injuries. The situational phobia subtype concerns circumstances involving driving, tunnels, bridges, enclosed places, or flying. The additional fifth type “other” is a residual category comprising all the fears that are not better classified by the previous sub-categories, including situations such as loud noises or choking.

The Diagnostic and Statistical Manual of Mental Disorders (DSM) created these descriptive subtypes according to signs, symptoms and their statistical and clinical patterns and not according to their etiology. However, identifiable pathophysiologic etiologies are most desired when it comes to providing a diagnosis as well as a treatment (Kupfer and Regier, 2011). A comprehensive and clustered analysis of prevalence, age of onset, gender, comorbidity and other risk factors is used in the absence of a better explanation. However the different subgroups of fears are not yet clearly separated. With the exception of BII phobias, the physiologic pattern of panic attacks driven either by a non-identified stimulus (panic attack) or by a clear trigger (e.g., fear of spiders) the symptomatic profile is assumed to be the same (Craske, 1991). BII phobia is an exception in which a vasovagal response is commonly present, and for which a distinct treatment is available (Ost et al., 1991; Ost et al., 1984). This particular subtype is well differentiated from the other subtypes. The animal subtype is also relatively well characterized (Davey, 2011) but the inability to distinguish a
situational fear from a natural environment fear in most of its dimensions is more evident, and some findings support grouping these two types in a single category (e.g., Fredrikson et al., 1996; Muris et al., 1999).

Although a recent review argued cogently that more research is needed to compare different phobias within types (LeBeau et al., 2010), we propose a less conventional line of inquiry. For the purpose of this review, we address a growing number of findings related with etiological factors that can play a determinant role in the cause of fear responses to a group of stimuli. These stimuli are not grouped according to the present DSM-5 groups, but according to the fact that all of these fears have a visuo-vestibular cue or trigger implicated in their genesis and elicitation. Here we address these factors, which can trigger fear of heights, driving and panic attacks. We also argue that the same underlying mechanisms that cause these fears might also inhibit fear. When in those situations; in line with Marks and Nesse (1994) we call this hypophobia.

Fear seems to follow a continuum, from the extreme lack of fear that causes subjects to engage in potentially dangerous situations, to an extreme fear associated with phobias and other anxiety disorders. Two main arguments are currently used to explain why some people do not acquire fear in spite of adversity. One possible explanation for the lack of fear is latent inhibition (LI) (Bond and Siddle, 1996; Lubow, 1973). The LI theory states that previous benign experience with a neutral stimulus (NS), such as climbing trees, reduces the likelihood of fear later being conditioned in a similar situation, for instance decreasing the probability of the act of climbing becoming a conditioned stimulus (CS). This means that the experience with a stimulus protects the person against fear development when it is later linked to an aversive non-conditioned stimulus (US), which would be the equivalent of fearing falling in the above-
mentioned example. In his way, LI provides an explanation as to why people do not necessarily develop a phobia following a traumatic experience. Doogan and Thomas (1992) observed a significant number of subjects with fear of dogs who reported having less contact with dogs before the emergence of the fear, as compared to fearless subjects. Subjects with previous experience are more attuned to how dogs behave, perceiving their behavior as predictable and controllable. Similarly Kent (1997) and ten Berge and colleagues (2002) noted that the acquisition of dental fear was less likely after a history of non-invasive dental visits.

The other explanation often used to clarify why some people do not develop or learn fear, in spite of aversive encounters, is the non-associative theory. This theory argues that the majority of members of a species will show fear (or not) to a set of stimuli, from the first encounter, independent of associative learning experiences. This perspective states that the continuity between normal and abnormal anxiety is no different from that which exists between normal and abnormal sugar levels in the blood. Given that medical and psychological disorders come from the same type of vulnerabilities, the evolutionary perspective applies equally well in both cases (e.g., see Nesse, 1999).

Neither theory is comprehensive. Firstly, LI seems insufficient to explain some previously obtained results. For example, participants in a study about fear of water were evaluated as similar to subjects without this fear in several aspects. They learned to swim at the same age, had a similar amount of water exposure, and their first water-related accident occurred at the same age (Poulton et al., 1999). By contrast, the non-associative theory does not explain to what specific elements of the phobic stimuli persons who have phobia react. Using the water as an example, it is not clear whether they fear the water itself, as they can use it to drink, bathe or water their plants (Kleinknecht, 2002). This model is also quite broad, in the sense that it does
not clarify the mechanisms leading to fear, simply stating that some fears are innate and need no previous experience for their manifestation.

The two theories offer different explanations for fear of heights. The LI theory would predict that subjects who do not develop fear after severe injuries caused by falls must have a history of previous benign experience of heights. The non-associative model would state that evolutionary pressures caused terrestrial animals to fear heights, and that fear of heights was shaped by natural selection to create an adaptive response (freezing) (e.g., Marks and Nesse, 1994).

Given the prevalence and impact of specific phobias (SP), progress in setting diagnostic criteria is of the utmost importance. The 12-month and lifetime prevalence for SP, defined by the Diagnostic and Statistical Manual of Mental Disorders criteria (DSM-IV, APA, 1994), is 7.1% and 9.4% respectively (Stinson et al., 2007). However, these estimates vary significantly not only across studies but even across settings. For example, Boyd and colleagues (1990) estimated one month’s prevalence for SP in the USA to vary significantly between cities such as St. Louis (1.5%), New Haven (2.7%) and Baltimore (5.6%). The United States National Comorbidity Study estimated a lifetime prevalence of 20.0% for adolescents (ages 13-17) and of 13.8% in adults (ages 18-64) (Kessler et al., 2012). Because fear is a common and necessary emotion in the normal population, as long as manifested within acceptable limits, these differences may reflect the lack of a clear division between what is normal and pathological fear in different settings (e.g., inter-rate variability in subjective diagnosis). This ambiguity causes significant difficulties in estimating prevalence (Chapman, 1997).
The accurate provision of a diagnosis requires awareness about both the unique and shared characteristics of each disorder. Etiology, prevalence, age of onset, gender, focus of fear, neurobiology, physiology, genetic factors, specificity of feared stimulus, response to treatment and comorbidity are all assessed when portraying a differential diagnostic of SP (e.g., LeBeau et al., 2010). When creating SP sub-categories, researchers have grouped different fears according to their triggering stimuli. Although these categories sought to respect the significance of the abovementioned factors, for instance the focus of fear, this taxonomy resulted in an endless terminology with questionable value (Marks, 1987).

The traditional psychological view regarding fears states that people have interpretation, attention and judgment biases that lead to danger overestimation, and a lack of confidence in coping with anxiety in certain situations (e.g., Arntz et al., 1995; Steinman and Teachman, 2011). A maladaptive schema causes these misinterpretations. Schemas are cognitive structures or core beliefs that organize experience and behavior (Beck et al., 2004) in this case increasing anxiety and promoting avoidance. Schemas seem to play a central role in the maintenance of chronic problems regardless of their etiological root (Padesky, 1994). One criticism of such models is that they can seem somewhat circular: “I’m anxious because I fear, and I fear because I’m anxious.” There is a considerable functional overlap between the generation of fear and anxiety (e.g., McNaughton and Corr, 2004), and as such one (anxiety) should not be used in order to explain the other (fear) and vice-versa.

An alternative approach holds that fears don’t need to be learned, as they are responses to evolutionarily prepared threat stimuli. This approach likewise falls into a circular argument: “the fear is common because it is prepared, and is thought to be prepared because it is common.” The main problem with either approach is that it is largely hung up on the question of what is learnt and what inherited, leaving the precise causes of phobia unknown (Coelho & Wallis, 2010). A common fear is seen as being prepared, and is
thought to be prepared because it is common. This is a circular argument as frequently feared stimuli are argued to be as such due to its survival relevance but there is no independent criteria that defines its phylogenetic survival relevance (e.g., de Jong & Merckelbach 1997).

In this paper, we propose that visuo-vestibular conflicts are able to act as cues for certain fears, namely: heights, driving and panic attacks. The lack of sensibility to visuo-vestibular information can also be responsible for the lack of fear displayed by some subjects (e.g., no fear of heights or no fear of high speeds while driving). The present model does not preclude the previous ones (classical conditioning and latent inhibition) but is more explicit in explaining specific factors involved in creating a sense of fear in such environments. Anxiety and vestibular disorders are known to co-occur (e.g., Balaban, 2002; Kalueff et al., 2008; Levinson, 1989). Further, the role in different individual of interactions among an anxious temperament, visual motion sensitivity, and presence of objective signs of balance dysfunction (including postural sway) are well-recognized in the literature on comorbid anxiety and balance disorders (e.g., Balaban et al., 2011). Dizziness, motion sickness and vection also can provoke emotional reactions (Carmona et al., 2009), but clinical psychologists and other healthcare professionals seldom consider these as fear cues, and often not consider them in treatment plans for panic or phobias. The aim of this paper is to clarify the role of these factors as important intrinsic mediators of fear and as potential treatment targets.

**Visuo-vestibular cued phobias**

Although phobias are highly comorbid (e.g., Magee et al., 1996), the scarcity of epidemiologic studies relating panic, acrophobia and/or fear of driving likely reflects the unawareness of the possible links between these disorders. Most epidemiological studies in the field of anxiety
compare or relate comorbidity data between men and women (Essau et al., 2000; Fredrikson et al., 1996; Kessler et al., 2005); between phobias and depression (Choy et al., 2007; Goodwin, 2002; Kendler et al., 1993; Wittchen et al., 2000); or between phobia and the risk of alcohol use disorders (Becker et al., 2007; Stinson et al., 2007; Zimmermann et al., 2003). However, studies relating similarities and differences within phobia subtypes are limited and in need of further research (LeBeau et al., 2010). To our knowledge only Davey Menzies and Gallardo (1997) addressed possible links between acrophobia and agoraphobia. In a population of 100 university students (45 female), they reported that measures of acrophobia were highly associated with a tendency to interpret ambiguous bodily feelings as threatening. These characteristic was also found by Coelho and Wallis (2010) which used the same bodily feelings questionnaire, the body sensations questionnaire (BSQ) from Chambless and colleagues (1984), and found it to be associated with fear of heights (along with the other features discussed bove). Davey and colleagues argue that the observed comorbidity between acrophobia and agoraphobia can be linked to similar biases in the interpretation of bodily feelings. We argue here that the emergence of these body feelings can be originated by a visuo-vestibular conflict.

Acrophobia

Acrophobia, or fear of heights, typically involves the avoidance of apartments and offices located in high buildings, bridges and elevators, thereby restricting daily activities (Menzies, 1997). This disorder has a high prevalence (e.g., Boyd et al., 1990; Essau et al., 2000; Kessler et al., 1994). In the Epidemiologic Catchment Area (ECA) Study, 4.7% of the subjects met the criteria for a diagnosis of acrophobia (e.g., Ardic and Atesci, 2006; Balaban, 2002; Kalueff et al.,

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1 The Epidemiologic Catchment Area (ECA) a large psychiatric epidemiological study done in five states of the United States (New Haven, Connecticut; Baltimore, Maryland; St. Louis, Missouri; Los Angeles, California; and Durham/Piedmont, North Carolina). It comprises 20,000 participants (14,429 assessed in relation to phobias) each of whom underwent a detailed diagnostic interview.
2008; Levinson, 1989), and a recent mental health incidence study (Depla et al., 2008) in the Netherlands found fear of heights to be the most prevalent phobia (4.9%).

Huppert, Grill and Brandt (2013) propose the use of three terms to distinguish the different states: ‘‘physiological visual height imbalance of posture,’’ which concerns everyone; ‘‘visual height intolerance,’’ which occurs in one in three and is more or less distressing (Huppert et al., 2013) and ‘‘acrophobia or fear of heights’’ which affects about 4% (Brandt et al., 2012) or 5% (Depla et al., 2008) of the population.

A normal fear related to heights develops during the onset of crawling (Campos et al., 1978; Gibson and Walk, 1960; Scarr and Salapatek, 1970; Schwartz et al., 1973). Dahl and colleagues (2013) have presented experimental evidence consistent with the hypothesis that ‘‘visual proprioception’’ (the ocular-based perception of self-movement) is a developmental determinant of wariness of heights. When infants learn peripheral lamellar optic flow (PLOF) to control body sway, the perceptual content of the PLOF is attenuated by the postural responses when one encounters depth at an edge. The wariness is the result of a mismatch between visual proprioceptive information and other sensory signals (vestibular, proprioceptive and autonomic), which are associated with self-motion.

The emergence of this fear is a normative experience of development, and most individuals later retain a degree of healthy wariness about heights that is functional and adaptive to survival. At present, it is unclear if acrophobia is related to a failure to achieve this developmental milestone (Coelho and Purkis, 2009; Coelho et al., 2009), but what is striking about the origins of fear of falling is an intimate connection with self-movement. It seems evident from the many experiments done in developmental science that crawling (the common PLOF generator) is necessary to originate heights related fears (Dahl et al., 2013).
The limited number of studies in adult populations done so far seems to support the hypothesis that fear of heights is modulated by self-generated postural adjustments to motion. When participants with acrophobia were tested in a virtual reality environment, Coelho and Colleagues (2008a) found that they experienced elevated anxiety not only as height increased, but also when required to move laterally at a fixed height. When self-motion was involved, anxiety levels were significantly higher than those elicited by viewing the fear-evoking scene while immobilized. The authors also noted that the virtual reality exposure therapy protocol (VRET) was more effective than an in vivo exposure protocol (Coelho et al., 2008a; Coelho et al., 2008b) and a meta-analysis from Powers & Emmelkamp (2008) found VRET to be significantly more effective than exposure in vivo. One must caution that this effect might be due to an artifact of the experiments. Specifically, owing to the usual delay between head movements and visual presentation of the display, the system delay might have been responsible for a visuo-vestibular recalibration that ultimately helped the participants to deal with the elevated environments where similar unusual combinations of multimodal sensory information occur. This possible artifact or confound is nonetheless important since it suggests the link between a multimodal sensory recalibration and the origins, cues or triggers for certain fears. As children learn to detect threats to balance and find compensatory strategies to regain posture (Adolph, 2002) when in disequilibrium, acrophobic subjects learn how to move in height-related situations (Coelho et al., 2009). As such, similar to motion sickness, fear of heights might be triggered by a conflict between the visual, somatosensory and vestibular senses (Brandt et al., 1980).

A visual-vestibular conflict has long been regarded as a factor related to motion sickness. In fact motion sickness is often caused by unusual types of movement (e.g., (Hettinger et al., 1990; Kennedy and Fowlkes, 1992; Lawson et al., 2002; Stanney and Hash, 1998; Yardley, 1992) and is often characterized by nausea and vertigo, cold sweats and general discomfort,
occasionally accompanied by vomiting (Money, 1970; Money et al., 1996; Tyler and Bard, 1949).

Postural instability has been examined as contributing to motion sickness (Owen et al., 1998; Riccio and Stoffregen, 1991). The visual-vestibular conflict theory (Stoffregen and Riccio, 1988; Stoffregen and Riccio, 1991; Stoffregen and Smart, 1998) suggests that the visuo-vestibular induced motion sickness occurs when the movement signaled by vision is discrepant with the pattern of signals expected by the vestibular sense based on previous experience (Cobb, 1999; Reason and Brand, 1975; Schiffman, 1990). The postural instability model argues that vestibular-ocular conflicts are common and do not necessarily cause motion sickness (Stoffregen and Smart, 1998). Instead, the authors suggest postural instability as a prerequisite to produce motion sickness.

The contribution of sensory conflict and postural instability to motion sickness is a consequence of the roles of the vestibular, visual and somatosensory systems in gravitoinertial sensation, balance control and autonomic function. Balance is an “implicit” sense; it is perceived indirectly through its influences in normal daily activities (Balaban and Jacob, 2001; Balaban and Thayer, 2001). Because the vestibular and visual sensors (1) give information from the frame of reference of the head and (2) act via reflexes to maintain postural and gaze stability, the performance of postural and ocular adjustments influences both sensory integration and instantaneous postural stability. From this perspective, it seems reasonable to regard postural instability and sensory conflict as two potential elements of a sufficient set of conditions for motion sickness. Stoffregen and Smart (1998) showed that subjects standing in a room exposed to an oscillating optical flow (sometimes not noticed by subjects) revealed increases in postural sway before the onset of subjective motion
sickness symptoms. This study is particularly interesting as it shows that people will not attribute their symptoms to their sense of balance. Because visuo-vestibular and postural cues act outside awareness (Angelaki and Cullen, 2008), discrepancies between the stimulation of different perceptual systems do not necessarily need to be interpreted as sensory conflict (Stoffregen and Riccio, 1991). We next argue that the overall discomfort caused in the Stoffregen and Smart (1998) experiment can be similar to the one caused in other situations, such as in heights.

The Stoffregen and Smart (1998) approach shares the perspective of the Coelho and Wallis (2010) acrophobia model. This fear of heights model states that fear will occur as follows: “When exposed to heights the lack of visual cues creates a natural increase in postural sway aimed at reactivating visual control. In visual field-dependent individuals with poor nonvisual postural control, the lack of visual cues will result in postural instability and a feeling of being off balance resulting in a moderate level of fear. For those individuals who tend to feel disoriented and sick through such instability more fear will result” (Coelho & Wallis, 2010, p.869). In the particular case of acrophobia, the increased reliance on vision (Jacob et al., 1995; Jacob et al., 1993; Redfern et al., 2001) seems to be promoting disequilibrium.

People with acrophobia are more reliant on optic flow for postural control (Hüweler et al., 2009). When preference is given to visual inputs, the individuals are said to be visually dependent, and more sensitive to perturbations in their visual surroundings. But when close to a roof edge, for example, there is an absence of motion parallax cues. The greater one’s distance from a visual target is (e.g., height above a ground plane), the smaller is the motion parallax cue provided by movement of the head. At this point the observer needs to rely mainly on vestibular and somatosensory inputs to maintain balance (Simeonov et al., 2003). Hence, the production of increased postural sway in elevated locations (Davis et al., 2009) can be viewed as an active
sensory sampling strategy (Gibson, 1966) to enhance visual feedback (Bles et al., 1980; Brandt et al., 1980; Gatev et al., 1999; Whitney et al., 2005). Increased postural sway is not equivalent to decreased stability, and subjects without acrophobia in laboratory studies often stabilize static posture by minimizing muscle activation (Kiemel et al., 2011). Persons with acrophobia might have more difficulty to achieve this relaxation due to fear added to elevated sway in balance control, which is more frequent in individuals with acrophobia even when not exposed to heights (Boffino et al., 2009). As the person with acrophobia actively avoids high places, it becomes harder to achieve the experience required to calibrate the new action-perception system to that specific environment. By practicing movements in the elevated environment the person creates new visuo-vestibular expectations. The central nervous system learns to predict the body’s next position, thereby allowing an appropriate corrective sway. This prediction requires experience to define the control variables of new systems of action-perception and to allow this calibration. In acrophobia, a discrepancy between the actual and the expected feedback creates a “sensory conflict”, very similar to the one explained in motion sickness literature.

Predictability and synchronization between visual and vestibular information is also a common feature shared by acrophobia and motion sickness. In virtual environments, for example, the predictability of future movement reduces or eliminates the sensory conflict (Kolasinski, 1995). Stanney and Hash (1998) noted that users adapt to sensory conflicts by building conditioned expectations through repeated interactions in the virtual world (e.g., when the person’s head turns, he or she learns to expect the visual field to change a few milliseconds later). If the virtual environmental motion is determined by the virtual system and users have no knowledge of the direction in which they will move in any given moment, motion sickness symptoms will more easily arise. Similarly, the random and unpredictable oscillatory motion of
waves might pose challenges, even to familiarized sailors. Exposure leads to adaptive compensations, which brings about an adequate performance and relieves the symptoms. Similarly motion sickness occurs less frequently with pilots and drivers than with co-pilots and passengers (Pausch et al., 1992).

Expectations might also be involved in lack or absence of fear (hypophobia). People with extensive experience of disorienting activities or environments, such as some athletes and roof-workers, are able to ignore discrepant visual or vestibular information (Brandt et al., 1980), having a higher risk of falling due to extreme confidence and more exposure. In 2005, the American Bureau of Labor Statistics reported 770 fatalities in United States industry due to falls. In particular, falls from roofs account for about one third of all fatal fall incidents in the construction industry each year (Simeonov et al., 2003). Although these falls are not necessarily due to hypophobia, it alerts to the fact that a lack of fear of heights and its possible consequences should be addressed in these potentially fatal situations. It is remarkable that subjects with no fear frequently experienced the worst traumatic experiences (see Coelho and Purkis, 2009 for a review), suggesting that some individuals fail to learn fear. For example, subjects with no fear of heights often have a past history of more falls and more severe outcomes when compared to subjects with phobias (e.g., Poulton et al., 1998; Poulton and Menzies, 2002). Failing to learn fear can have multiple causes, and one of the risk factors for the inability to learn fear can be related with visuo-vestibular integration conflicts. As this conflict can be measured, it is possible to test participant’s susceptibility to its variations.

Corroboratory data of the hypothesis relating vision, motion and fear of heights also came from vestibular rehabilitation treatment programs (Andersson et al., 2006; Beidel and Horak, 2001). Vestibular rehabilitation relies on the use of exercises to stimulate vestibular
compensation and improve balancing capabilities. These exercises are designed to facilitate processes termed adaptation, substitution, and desensitization (Herdman and Whitney, 2007). Adaptation exercises train a patient to either ignore or repair the components of balance that are functioning poorly. Substitution refers to a process of learning to substitute other sensory information or activities to improve balance performance. Desensitization exercises train individuals to tolerate an abnormal response. On a practical level, vestibular physical therapy procedures focus on vestibulo-ocular reflex, cervico-ocular reflex, depth perception, somatosensory retraining, dynamic gait exercises, and improved aerobic function (Balaban et al., 2012; Herdman and Whitney, 2007; Whitney and Sparto, 2011). Exercises that promote awareness of somatosensory inputs also enhance a participant’s confidence in height situations (Whitney et al., 2005). These findings strongly support the idea that the construction of new visuo-vestibular expectations is an important factor in acrophobia etiology and treatment. As such, the treatment of this fear should be addressed differently from other fears such as animals, storms or social fears.

Driving

The situational phobia subtype is relative to several circumstances, including driving. Studies examining driving phobia have reported similarities with fear of accidents (specific phobia), fear of anxiety (panic disorder), and fear of embarrassment (social phobia) (Ehlers et al., 1994). Taylor, Deane and Podd (2000) point out that there are many different possible foci for this fear, leading to many relevant questions, such as: *Is the fear specific to driving on the highway? Is the fear of the actual anxiety symptoms? Is it ultimately of having an accident or causing injury to self or others? Is it of being negatively evaluated and criticized by other people*
on the road? Or is it a combination of all of these fears (i.e., the entire chain of events)? If it is the latter, then how much weight does each of these individual fears contribute to the overall fear? (Taylor et al. 2000, p.455). Taylor and Deane (Taylor and Deane, 2000) found no significant differences between participants who had experienced a motor vehicle accident (MVA) and those who had not, in terms of fear severity. These findings suggest that those who have not experienced an MVA also express danger expectancies (see also Taylor et al., 2002 for a review).

Our present model is consistent with the idea that some components of driving phobia may have a common underlying vulnerability to space and motion discomfort (Jacob et al., 2009; Jacob et al., 1993) and panic (Ehlers et al., 2007), rather than to MVA. For example, the twenty items on the Space and Motion Discomfort-1 (SMD-1) subscale include 11 items about riding as a passenger in a car, riding in a bus, or traveling through a tunnel, and a high SMD-1 score is associated with a somatosensory dependence for balance control (Jacob et al., 2001). It seems reasonable, therefore, to consider the role of learned action-perception expectations particular to the driving experience in development of a phobia. Considering Seligman’s (1971) preparedness model, which argues that certain stimuli are evolutionally predisposed to evoke fear responses, it would be natural that humans show fear of driving and an extreme difficulty to overcome it, since humans were not biologically prepared to “run” at the speed that cars allow travel. However the opposite seems to occur, because most people easily overcome the initial fear of driving. This fact suggests that fear responses are recalibrated to avoid impeding adaptability of the visuo-vestibular and postural systems to many novel space and motion environments. This adaptability can potentially permit subjects to drive at high speeds and along winding mountain roads without sensing fear.

Although humans often adapt to unusual situations, the
opposite can also occur when sensory inputs provide false, ambiguous or unreliable information. Deficient or distorted vestibular, visual or somasthetic sensory information can result in a misleading or fictive sense of movement or disorientation, which ultimately leads to somatic symptoms such as nausea, sweating, anxiety or panic (Page and Gresty, 1985). Sub-clinical disturbances of vestibular input can also cause a mismatch between the various systems (vestibular, visual and somatosensory) that can lead to symptoms in particular situations (Godemann et al., 2005).

Many variables may contribute to development of a fear of driving. Because every tissue in the body has mass (including blood), linear acceleration (including gravity) is implicit in sensory signals from baroreceptors, visceral pressure receptors and somatic proprioceptors for functions such as respiratory control. These different sensory sources are combined and contribute simultaneously to an integrated activity involving vestibular, visual, proprioceptive and visceral mechanisms. While in a car, vestibular, visual and proprioceptive signals have a different weight when compared to other situations. The driver experiences gravitoinertial accelerations that have differential effects on a large number of receptors in the muscles, cardiovascular system, and abdominal viscera (Balaban and Yates, 2004). The pressure on the skin and deep tissues, as well as temperature gradients on the body surface, gives information about gravity. For example, pressure and warmth on the buttocks give a sense of direction while seated. Regional blood distribution, movements of the abdominal viscera within the peritoneal cavity relative to the torso (e.g., traction on mesenteries, contact with the parietal surface of the peritoneum and stretch due to pressure on the diaphragm), proprioceptive information, and changes in intraocular and intracranial pressure are also influenced by gravitoinertial stimuli (Balaban and Yates, 2004). Hence, driving may be an activity with a relatively high demand for somesthetic information.
processing for action-perception coupling during accelerating, braking and turning.

An experienced driver creates new expectations after practice and becomes able to foresee the future movement of the car and adjust the posture. In fact, the driver is effectively the locomotor sensor and controller for the vehicle. The driver’s anticipatory adjustments reduce or eliminate the sensory conflict (Kolasinski, 1995), which reduces motion sickness susceptibility (Pausch et al., 1992) and the overall sensory input that can give rise to a potential fear cue. The driver is able to build conditioned expectations through repeated interactions with the car, similar to what occurs in virtual worlds (Stanney and Hash, 1998). The lack of control felt by the passengers does not allow the stabilization of these expectations given that the users have no knowledge of the direction in which they will move at any given moment (i.e., the course is determined by the driver). Motion sickness does not occur as frequently with self-produced movements, such as running or driving (Schiffman, 1990). In contrast, the same head movements can be extremely stressful if not self-generated, meaning that subjects automatically account for them within the context of executing voluntary movements (Durlach and Mavor, 1994).

The sensitivity of the vestibular system to angular and linear acceleration has an important perceptual implication: the vestibular nerve signal is identical when we are at rest (zero velocity) and when we are traveling at constant velocity. By contrast, constant velocity motion can be sensed from optic flow information. The otolithic organs detect the direction of the constant linear acceleration due to gravity (Lysakowski and Goldberg, 2004). The linear acceleration sensitivity is due to the fact that the kinocilia and stereocilia of the otolith organ hair cells are embedded in an otoconial membrane that is covered with calcite crystals that grow in the presence of the primary otoconial matrix protein, otoconin 90 (Lu et al., 2010). The mass of the otoconia and membrane confers linear acceleration sensitivity to the hair cells (Wilson and
Melvill Jones, 1979). The force produced by the action of gravity on the mass of the otoliths generates a static position signal by moving the tips of the kinocilia and stereocilia toward the ground. When the head is upright, this static position signal is driven by the saccule and the anterior portion of the utricle. If the head is tilted forward or backward, the location of activation shifts to regions with aligned hair cells on both organs. Lateral static roll of the head is similarly detected by the utricular hair cells. The inertia of the otoliths is overcome as they accelerate to reach the same velocity as the temporal bone, and after the initial acceleration, only the optic flow provides information during motion at constant velocity. At this point in time, the vestibular system detects no acceleration. Because the visual flow velocity in a car is much above human running speed, it may be perceived as unusual in terms of prepared learning (Seligman, 1971), since humans are not biologically prepared to travel at these speeds while seated and not moving. Previously, we addressed acrophobia and the importance of the peripheral lamellar optic flow (PLOF). In elevated environments a loss of information from nearby periphery objects is absent (Dahl et al., 2013). In driving situations the inverse occurs, since PLOF is enhanced, albeit with different spatial and temporal properties (e.g., the seated frame of reference in the car and higher speeds). For example, people with fear of heights might be very sensitive to the absence of motion parallax cues, while people with fear of driving might be more sensitive to conflicts to optic flow over large portions in the visual field, without the usually simultaneous vestibular stimulation (Schmäl, 2013). It seems to be possible that people more susceptible to this conflict are more prone to fear of driving, and others less sensitive to it show hypophobia and a lack of fear of driving at high speeds. In 2007, the estimated annual number of deaths caused by motor vehicle crashes was 1,230,000 (World Health Organization 2009); perhaps dangerous driving behaviors might be motivated by a lack of fear.
The limited utility of vestibular information for judgments of spatial vertical and orientation was well known in the earlier psychophysical literature (Asch and Witkin, 1992; Guedry Jr, 1974; Mittelstaedt, 1996) including judgment errors during centrifugation (Schoene, 1964) and water immersion (Nelson, 1968; Ross et al., 1969) in the absence of visuospatial cues. The perceptual consequences of these sensory phenomena are a very significant factor in aviator spatial disorientation (Headquarters, Department of the Army 2000), which requires consideration of aggravating and ameliorating interactions between vestibular, visual, autonomic and proprioceptive information in maintaining spatial awareness and orientation. Similar training could be used to either help participants to overcome fear of driving, as well as to train participants to become more aware of speeding.

**Panic**

Although panic disorder is not a specific phobia, it shares clinical features with specific phobias (Craske, 1991). Because of the physiological overlap between these disorders, predictability is the most important variable in the differential diagnosis between a specific phobia and panic disorder (Coelho et al., 2010). This unawareness of the cue in a panic attack is in accordance with a vestibular trigger, as we will explain.

An increasing body of evidence now links anxiety to vestibular functions (e.g., (Andersson et al., 2006; Bles et al., 1980; Brandt et al., 1980; Redfern et al., 2001; Simeonov et al., 2005; Whitney et al., 2005). As we have previously addressed, preservation of posture requires vestibular and proprioceptive input, especially when visual information is ambiguous or unavailable (Horak et al., 1990; Nashner et al., 1982). Vestibular and other information regarding balance control also exerts a significant influence on ascending pathways that
are involved in interoception (sense of the physiological well-being of the body) and anxiety (Balaban, 1999; Balaban and Porter, 1997; Balaban and Thayer, 2001). The vestibular nuclei also project to the parabrachial nucleus in rabbits (Balaban, 1996), rats (Porter and Balaban, 1997) and monkeys (Balaban, 2002). The parabrachial nucleus has descending connections to both rostral and caudal aspects of the vestibular nuclei, which are distributed more extensively than the sites of origin of the vestibuloparabrachial pathway (Balaban, 2004). It also has reciprocal connections with the central amygdaloid nucleus, infralimbic cortex and hypothalamus (Fulwiler and Saper, 1984; Herbert et al., 1990; Moga et al., 1990). In addition, the caudal parabrachial nucleus (i.e., vestibulo-recipient region) projects to several midline and intralaminar nuclei, including the centromedian nucleus, ventromedial nucleus and ventroposterior nucleus (Krout and Loewy, 2000) providing potential integrative input with visceral pathways to the insular, anterior cingulate and medial prefrontal cortices.

Due to its important role in the formation of conditioned fear and anxiety responses, the parabrachial nucleus has been widely cited as a substrate for panic and anxiety disorders (Charney and Deutch, 1996; Goddard and Charney, 1997; Gorman et al., 2000; Ressler and Nemeroff, 2000). This view is consistent with the emerging concept in the pain literature that the parabrachial nucleus is an important integrative component of pathways for interoception and associated affective and emotional responses (Craig, 2003; Craig, 2002; Gauriau and Bernard, 2002; Zylka, 2005). These ideas are strikingly similar to the proposed role of the vestibulo-recipient parabrachial region in the link between balance and anxiety (Balaban and Thayer, 2001). The caudal parabrachial nucleus includes neurons that respond to both linear and angular whole body motion in darkness (Balaban et al., 2002; McCandless and Balaban, 2010). The primary axis of linear acceleration sensitivity is either in the vertical semicircular canal or the interaural plane,
and the angular acceleration sensitivity can be either for rotation around the linear acceleration axis or in the plane of the linear acceleration axis. The guidance sensors may play a role in detecting stability of movement trajectories and the instability accompanying loss of balance. The mammalian vestibular nuclei receive noradrenergic input from a distinct pathway that originates in the pole of the locus coeruleus and the adjacent nucleus subcoeruleus (Schuerger and Balaban, 1993, 1999). This locus coeruleus pathway has been implicated both as an initiator of anxiety responses and panic attacks (Charney and Deutch, 1996; Gorman et al., 2000; Pratt, 1992) and as a modulator of vestibular function (Schuerger and Balaban, 1999). Four quantitatively distinct regional densities of noradrenergic innervation have been identified. The superior and lateral vestibular nuclei receive the densest innervation; a dense plexus also fills the rostral aspect of the nucleus prepositus hypoglossi in monkeys. The innervation is less dense in the medial vestibular nucleus, and the inferior vestibular nucleus receives minimal innervation. The terminals may affect vestibular nucleus neurons via post-synaptic alpha (alpha 1 and alpha 2) and beta adrenergic mechanisms (reviewed in Schuerger and Balaban, 1999). The density differences in coeruleo-vestibular projections to different vestibular nuclei are likely to mediate increases in postural sway and altered vestibular-evoked eye movements during anxiety and changes in alertness (Schuerger and Balaban, 1999).

Serotonergic innervation of the vestibular nuclei arises from two sources, the dorsal raphe nucleus and the nuclei raphe pallidus et obscurus (Halberstadt and Balaban, 2003). These nuclei contain both serotonergic and non-serotonergic neurons, which appear to project in parallel to their efferent targets. The non-serotonergic neurons utilize a variety of transmitters. The 5-HT negative cells in the dorsal raphe nucleus include populations that express GABA (Bagdy et al., 2000; Stamp and Semba, 1995), dopamine (Stratford and Wirtshafter, 1990; Yoshida et al.,
1989), excitatory amino acids (Clements et al., 1987), and neuropeptides (Lechner et al., 1993; Petit et al., 1995). Approximately equal numbers of serotonergic and nonserotonergic dorsal raphe nucleus neurons project to the vestibular nuclei (Halberstadt and Balaban, 2003, 2006).

Single dorsal raphe nucleus axons often project via collaterals to multiple sites (Kazakov et al., 1993; Kirifides et al., 2001; Petrov et al., 1994). Halberstadt and Balaban (2006) demonstrated that populations of both serotonergic and non-serotonergic neurons from the dorsal raphe nucleus send collateralized projections to the vestibular nuclei and the central amygdaloid nucleus. Approximately one quarter of the dorsal raphe neurons that project to the vestibular nuclei also have ascending collaterals to the amygdala. This finding provides strong support for the hypothesis that there is coordinated serotonergic modulation of vestibular and central amygdaloid nucleus information processing, which may contribute to coordinated activation in association with anxiogenic contexts. For example, a collateral co-modulation of the vestibular nuclei and central amygdaloid nucleus could contribute to increased postural sway in response to optic flow in patients with anxiety disorders and the tendency for a persistence of increased sway in a post-experimental period (Jacob et al., 1995). A similar argument may be made for fear of heights.

The vestibular system also influences cardiovascular and visceral control (Balaban and Yates, 2004; Spiegel and Sommer, 1944; Uchino et al., 1970; Yates and Miller, 1994; Zakir et al., 2000). Vestibular sensations can include palpitations, “queasiness” or even nausea, and other more vaguely defined visceral sensations. These visceral aspects of vestibular sensation occur without ready attribution (Balaban and Yates, 2004; Balaban, 1999).

It is simple for someone with fear of dogs to identify a dog and claim that the dog is clearly associated with the felt fear. Nonetheless, even for such a clear event, it can be difficult for the subject, once questioned, to determine exactly what makes the dog fearful. It can be
disgust, for example, or fear of being attacked. In a panic attack, which has no apparent fear motif, it seems even more difficult for the sufferer to associate the panic attack with a visuo-vestibular trigger. Most people will not attribute their symptoms to their sense of balance or to the lack of it. Furthermore the cues or precipitating factors are unclear to the person (e.g., vection). As visuo-vestibular and postural cues act outside awareness, a visuo-vestibular conflict might trigger fear in particular situations without the person’s awareness of the cue. The extensive and continuous multimodal convergence of vestibular stimulation with information from muscles, joints, skin, and eyes does not give rise to a separate and distinct conscious sensation (Angelaki and Cullen, 2008). This can help to explain why the person does not explicitly acknowledge these cues as fear cues, as it occurs in specific phobias. This kind of unconscious influence is a possible trigger to a panic attack and an analysis to human emotional responses to vection could give us important information regarding this issue.

According to Jacob and Colleagues (Jacob et al., 2009; Jacob et al., 1993) “space and motion discomfort” (SMD) may result in phobic patterns of fear and avoidance in particular situations, and might be the basis for some cases of panic disorder and agoraphobia. In fact, panic disorder is one of the best-studied anxiety disorders and the one most often linked to a vestibular/balance dysfunction. A major interaction between vestibular/balance systems and panic disorder is well documented (e.g., Asmundson et al., 1998; Clark et al., 1994; Eagger et al., 1992; Frommberger et al., 1994; Furman and Jacob, 1997; Godemann et al., 2006; Godemann et al., 2005; Gordon et al., 1998; Hoffman et al., 1994; Jacob et al., 1996a; Jacob et al., 1996b; Jacob et al., 1985; Perna et al., 2001; Simon et al., 1998; Sklare et al., 1990; Szirmai et al., 2005; Tecer et al., 2004; Yardley et al., 1995). Furman & Jacob (2001)
provide a reasonable framework for understanding psychiatric manifestations more broadly in the context of balance function (see their Figure 2). Anxiety proneness and comorbid non-anxiety conditions (e.g., OCD and depression) can be considered as predisposing factors for SMD and situationally specific postural sway to be manifested as space and motion phobia or anxiety disorder features. The association of panic (or fear) with vertigo was noted repeatedly in ancient medical texts (Balaban and Jacob, 2001). Levinson (1989) reported that Benedikt suggested in 1870 that vestibular dysfunction caused agoraphobia. It is worth noting that agoraphobia was named by Westphal (1871) to describe symptoms of dizziness, palpitations and trembling, experienced by patients walking in open spaces, this being close to what is now described as panic (see Boyd and Crump, 1991).

It is also critical, particularly in order to help explaining panic attacks which occur during the night, to consider how the vestibular and respiratory systems are linked (Perna et al., 2001). The locus coeruleus, the raphe nuclei and the ventrolateral medulla have been proposed as contributors to panic etiology (Bellodi and Perna, 1998); these regions are interconnected with the vestibular nuclei and related pathways (Balaban, 1996; Balaban and Thayer, 2001). The vestibular pathways are likely to contribute to maintaining circulatory homeostasis during movement and postural changes, as well as affecting control of both ‘pump’ and upper airway muscles involved in respiration (reviewed by Balaban and Yates, 2004). Indeed, the associations of hyperventilation, dizziness and vertigo are well-known in the literature (e.g., Furman and Jacob, 2001; Sakelleri and Bronstein 1997, Sakelleri et al, 1997; Theunissen et al., 1986). Extensive studies by Pompeiano and co-workers during the late 1960s (e.g., Lenzi et al., 1968) showed that central vestibular transmission occurs during sleep. As such, it has been proposed that
dysfunction of the vestibular system may contribute to abnormal respiratory performance and sensations, which in turn may predispose fear in response to motion or space discomfort (Gorman et al., 2000; Perna et al., 2001). Moreover, phobic situations increase arousal and hyperventilation, further destabilizing the balance system and creating the well-known perpetuating panic cycle during which anxiety leads to more anxiety and eventually panic (Perna et al., 2001), nonetheless more evidence is required to confirm this view.

Conclusion

Following the arguments presented in this paper, it seems plausible that visuo-vestibular interactions with postural stability might act together to elicit fears related to particular environments. These factors were already showed to be significantly associated with a person’s level of acrophobia (Coelho and Wallis, 2010), and are very closely related to panic and driving.

The identification of visuo-vestibular and postural dimensions relevant to a number of anxiety disorders raises the issue of whether to place these specific phobias in a particular subtype or specification within the DSM-5 diagnostic criteria. Vestibular and postural cues have been neglected as potential fear signaling cues in favor of a more descriptive categorization. In this review, we have defended these factors as potential causes of fear and, as such, as contributors to certain fears which could therefore be grouped in a new subtype category. Rehabilitation (and re-education) of vestibular and balance information processing can reduce some forms of anxiety; cognitive behavioral therapy for anxiety can also improve balance (e.g., (Brown et al., 2006; Holmberg et al., 2006; Jacob et al., 2001; Yardley and Redfern, 2001).
interdependence argues for the inclusion of visuo-vestibular fears in diagnostic manuals, which would highlight additional treatment options. As Mallan and colleagues (2013) recently argued: “preferential fear learning may reflect on a number of underlying mechanisms which we need to identify if we want to gain a more complete understanding of its causation and derive effective interventions that can limit the negative consequences of such preferential learning” (Mallan et al. 2013, p.1177-1178).

A visuo-vestibular subtype or specification of a specific phobia should also be considered in large-scale longitudinal and cohort studies that often include thousands of participants. These studies usually use classical batteries of questionnaires that strive to find correlations between specific phobias but also often do not address postural and visuo-vestibular symptoms. Similar to blood-injection-injuries, a subtype of specific phobias based in the etiology and phenomenology of the disorder would represent a step in the advance of psychological science. Parallel considerations in neurology have resulted in the recognition of the vestibular migraine as a clinical entity (Furman et al., 2013).

Given the multiple etiology and complexity of the disorders under discussion, one must exercise caution that placing them under a common visuo-vestibular banner not be misleading. Hence, a terminological qualification seems prudent. For example, obsessive-compulsive disorder may be qualified by co-existing factors such as poor insight, as recognized previously in axis V (Global Assessment of Functioning). A similar recognition of “visuo-vestibular” performance could lead the clinician to a better diagnosis and treatment of such debilitating and prevalent conditions and co-morbidities. The recent DSM-5 no longer uses the same multiaxial system of diagnosis. The DSM-4-TR (APA, 2000) used the specifier “with poor insight” for obsessive-compulsive disorder. This specifier is now present in the DSM5 as: 1) good, 2) poor,
or 3) absent or delusional insight. And similar specifiers were included for other disorders. Similarly, a specifier stressing the existence, or not, of visuo-vestibular vulnerability would be of great use for the clinician to be aware of how the fear should be treated.

As there is no single cause for anxiety, and an appropriate etiological model must comprise multiple genetic and environmental factors, which can either be of a predisposing or risk precipitating nature according to a risk factor model. The current etiological models of anxiety disorders recognize the necessity to consider individual differences that can function as vulnerability factors (Mineka and Oehlberg, 2008). These factors are often relatively general. For example personality traits such as trait anxiety, anxiety sensitivity, or emotion regulation, as well as stressful life events, early learning experiences or unpredictable stress, all can be significant diatheses for a variety of anxiety disorders (e.g., Tull et al., 2009). The interactions among an anxious temperament, visual motion sensitivity, and presence of objective signs of balance dysfunction (including postural sway) are well-recognized in the literature on comorbid anxiety and balance disorders (e.g., Balaban et al., 2011). Here we focus on a more general contribution of visuo-vestibular factors to anxiety risk factor models. Visuo-vestibular (VV) contributions should be included among more discrete conditioning experiences for future studies in research related to the causes of anomalous anxiety responses.

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well-recognized in the literature on comorbid anxiety and balance disorders (e.g., Balaban et al., 2011). Disorders such as social phobia (Schneier et al., 1992) and depression (Roy-Byrne et al., 2000) can act as modulators or facilitators of panic attacks. Depressed mood on its own, can increase anxiety in response to danger (e.g., Robinson et al., 2012) and this should not be related with any kind of visuo-vestibular trigger. In fact, any situation that can cause a phobic response can also ultimately cause a panic response and panic attacks are always cued, although the individual is not aware of the cue (Craske, 1991). Here we focus on a more general contribution of visuo-vestibular factors to anxiety risk factor models. Visuo-vestibular (VV) contributions should be included among more discrete conditioning experiences for future studies in research related to the causes of anomalous anxiety responses.

Additional research is also necessary to compare and separate fears triggered by visuo-vestibular conflicts from those cued by other well-known factors, such as blood-injection-injuries, disgust or visual characteristics. This is particularly important since it was suggested that other fears, such as enclosed spaces (Faugloire et al., 2007) and public speaking (Levinson, 1989), might also be cued by similar imbalance mechanisms. There is a scarce amount of studies relating claustrophobia symptoms and its possible relation with postural movements and motion sickness incidence. More research about this topic might bring important information about the multimodal sensory integration and psychological disorders. We opted not to discuss social fears since their relation with visuo-vestibular triggers is not as obvious and, at least for public speaking, other explanatory theories seem far more adequate (e.g., Hofmann et al., 2004).

The multiplicity of sensory inputs related to spatial orientation is processed by the central nervous system to produce a single non-ambiguous, perception of body orientation and movement - when under natural unperturbed conditions (e.g., Angelaki and Cullen, 2008). During most of our daily routine tasks and practices, different sensory information is integrated
and perceived as coherent (DiLuca et al., 2009). The process by which different sensory signals are compared with one another is known as multisensory or multimodal integration (MI) (e.g., DiLuca, Machulla, and Ernst, 2009). MI logic is plastic and modifiable (e.g., Reason, 1978) depending on changes in expectations, either from 1) an efferent copy of active movement undertaken by the subject that would normally produce a given feedback, or 2) the expected continuation of a pattern of movement such as a swing oscillation (Young, 1984). Although the perception of coherence (or unity assumption) between sensory modalities can differ between individuals (Liu, 2011), it varies within a measurable amplitude windows. This window has been better studied in relation to the audio-visual integration. In this case, an asynchronous sound relatively to a visual stimulus is likely interpreted as signaling different events. For example, while observing people talking, seeing a person moving the lips, and concurrently hearing a voice that does not match the movement of the lips, the observer concludes that two persons talk. But when discrepancies arise from VV stimuli, it is difficult to explain what is happening and give it a meaning, as both senses are integrated by default and usually corroborate redundantly with each other. In fact, when under natural, unperturbed conditions, the vestibular information does not even give raise to conscious awareness (Angelaki, 2008). Nonetheless, the extent to which a given visual stimuli contributes to perception is influenced by the concurrent vestibular stimulation (e.g., Young, 1984). Perceptual, postural, and eye-movement responses to identical sensory patterns show habituation to repeated stimuli and adaptation to rearranged sensory signals, and the perception of simultaneity is affected by the flexibility each individual adapts to asynchronous stimuli.

An accurate and reliable processing of visuo-vestibular stimuli is a prerequisite for a variety of daily-required occurrences that involve motion and balance, as well to accurately
perceive verticality to make judgments about the orientation of an object in the gravitational field and to keep postural stability. The expected patterns of motion, whether grounded on continuation of an existing pattern, or in the memory of the motion from previous exposures to similar situations, are of vital importance in determining the manner in which multiple sensory inputs are combined to yield a single perception of spatial orientation and movement. Special cases of ambiguous stimuli resulting in confusion about spatial orientation may result in vertigo, motion sickness, fear of heights, panic and driving fears, as well as hypophobia.

In sum, an unusual combination of visual and vestibular stimuli may exceed the capacity to integrate multimodal information, creating a sensory conflict that either generates the typical motion sickness physiological responses, such as nausea, vomiting and palpitations or, in different circumstances (and in different persons) might give rise to a fearful or phobic response.

Hypophobia (reduced fear) is also a characteristic that should be addressed in future research. As we have seen, some individuals seem to fail to learn fear, even after experiencing serious traumatic experiences. This was already reported in acrophobia, where a past history of falls and severe outcomes is common in fearless participants, and this feature should be taken into account when considering to driving as well. In relation to traffic accidents, the mutual influence between attentional performance and balance control may play a significant role. Similarly to what was found by (Boffino et al., 2009) in individuals with acrophobia, also individuals with driving fear might show altered sensitivity to both visual-vestibular and balance-cognition interactions.

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Dear Professors Verity Brown and Liang Li

Editor and Associate Editor of *Neuroscience and biobehavioral reviews*

**Highlights:**

We show new methods to measure psychophysiological correlates underlying an integrated sense of balance;

We contribute to a taxonomy that reflects a diagnostic conceptualization based on the etiology;

We explore balance-related sensory integration its role within mechanisms of fear and anxiety;

We explore possible new mechanisms to address hypophobia (lack of fear).

Yours sincerely,

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