Stress, Cerebral Asymmetry, and the Vestibular System: Rethinking the Neurobiology of Dizziness

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Abstract: Stress may be defined as the perceived threat to an individual's physical, emotional, social, economic, or bodily integrity, eliciting a coordinated set of adaptive neurobiological responses. In right-handed individuals, interoceptive self-awareness is predominantly represented in the right anterior insula, a region critically involved in integrating internal bodily states with higher-order regulatory functions. Emerging evidence indicates that threat-related stress can induce lateralized changes in right insular activity, thereby modulating the vestibular system. Such modulation may produce vestibular functional asymmetry, resulting in dizziness, postural imbalance, and a subjective sense of instability. This review synthesizes current evidence on stress-induced lateralized insular modulation and the structural and functional connectivity between the insular cortex and vestibular nuclei. We propose an integrative neurobiological model linking these mechanisms to alterations in bodily self-experience during states of threat.

Keywords: Vestibular system, Lateralization, Stress, Vestibular asymmetry, Dizziness.

INTRODUCTION

Stress is broadly defined as the perception of threat to physical, emotional, social, economic, or bodily integrity, activating a large-scale adaptive neurobiological response involving the hypothalamic–pituitary–adrenal axis and salience networks.[1] Among the cortical hubs recruited during stress, the anterior insula plays a crucial role in interoception, self-awareness, and integration of autonomic and affective information [2, 4]. In right-handed individuals, interoceptive representation is predominantly lateralized to the right anterior insula, which is central to the subjective monitoring of bodily states and the detection of salient internal signals [1, 2, 5]. Conversely, stress and threat states can modify insular activity in a lateralized fashion, particularly reducing or dysregulating right-insular function [6, 8].

The insular cortex is anatomically and functionally connected to the vestibular system, with direct and indirect projections to the vestibular nuclei in the brainstem, as well as multisensory vestibular cortical areas [9, 12]. Neurophysiological and neuroimaging studies have shown that vestibular stimulation recruits posterior and anterior insular sectors, and that direct electrical stimulation of the insular cortex can evoke vestibular sensations [13, 15]. The available anatomical, electrophysiological and neuroimaging evidence indicates that cortical vestibular fields — including the posterior insular / parieto-insular vestibular cortex (PIVC) — are not merely passive recipients of ascending labyrinthine signals but also participate in top-down modulation of brainstem vestibular nuclei. Tract-tracing and stimulation studies in non-human primates and other models have identified corticovestibular projection neurons in temporal, parietal and insular cortices that can alter firing in vestibular nucleus neurons, consistent with direct or short-latency polysynaptic corticofugal control of vestibular processing [11, 16]. Functional imaging and lesion/stimulation studies in humans further support a functional corticofugal pathway: cortical activation in temporo-parietal and insular regions correlates with changes in vestibular perception and oculomotor/vestibulo-ocular control, and focal disruption of these cortical areas produces lateralized vestibular symptoms [13, 14].

Functional imaging and lesion/stimulation studies in humans further support a functional corticofugal pathway [12, 14].

More recently, experimental tract-tracing and physiological work has emphasized that the insular cortex possesses descending outputs [17, 21]. Capable of modulating brainstem sensory and autonomic nuclei (for example parabrachial nucleus, periaqueductal gray and related targets), providing a mechanistic substrate by which stress-related insular lateralization could bias vestibular nucleus activity and thereby produce asymmetric vestibular output, dizziness or imbalance [17, 18]. In sum, converging data from neuroanatomy, neurophysiology and human neuroimaging indicate that the insular / parieto-insular cortical complex can exert modulatory, and sometimes lateralized, descending influences on vestibular brainstem circuits — a pathway plausibly mediating the translation of stress-related cortical asymmetry into vestibular dysfunction.

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Given the insula's modulatory control over vestibular pathways, stress-induced lateralized insular changes could plausibly influence vestibular nucleus activity, producing vestibular asymmetry and symptoms such as dizziness, imbalance, and instability [6, 7, 16]. While stress–vestibular interactions have been described clinically, an integrated neurobiological model linking stress-induced lateralized insular modulation with vestibular dysfunction has not yet been fully articulated. The aim of this review is to synthesize current evidence supporting such a mechanism and to discuss its implications for bodily self-perception and dizziness under threat.

MATERIALS AND METHODS

This work was conducted as a narrative, integrative review, focusing on neurobiological evidence regarding stress-induced hemispheric lateralization of the insular cortex and its downstream vestibular implications.

Searches were performed in PubMed, Scopus, and Web of Science using terms including "insula," "anterior insula," "lateralization," "stress," "threat," "vestibular nuclei," "vestibular cortex," "interoception," "right hemisphere," "functional connectivity."

Inclusion criteria included

1. Empirical studies describing stress-related changes in insular activity [2, 8];
2. Studies examining structural or functional connections between insular regions and vestibular pathways [9, 15].
3. Vestibular research relevant to dizziness, balance regulation, or central modulation [11, 12, 16].

Exclusion criteria included purely peripheral vestibular studies without central correlates, absence of hemispheric specificity, or non-peer-reviewed reports.

DATA SYNTHESIS

Findings were classified into:

a) Evidence of lateralized insular responses under stress,

b) Evidence of insulo-vestibular connectivity,

c) Evidence linking vestibular asymmetry with altered interoception or stress.

Search Period and Study Selection

The search period covered studies published between January 1980 and December 2024. The initial search yielded 1,247 records: 684 from PubMed, 392 from Scopus, and 171 from Web of Science. After removal of duplicates, 812 records remained. Screening of titles and abstracts resulted in the exclusion of 676 studies that did not meet inclusion criteria. A total of 136 full-text articles were assessed, of which 62 met criteria for inclusion in this narrative synthesis. A PRISMA-style flow description has been added for transparency, although formal systematic review procedures were not applied.

DISCUSSION

The evidence reviewed indicates that stress induces lateralized modulation of right anterior insular activity, particularly reducing or destabilizing activity associated with interoceptive awareness and salience processing [1, 4, 6, 8]. Since the right anterior insula plays a dominant role in referencing internal bodily states for threat detection [1, 2], stress-related downregulation of this region may impair accurate bodily self-awareness and generate altered interoceptive predictions.

Concurrently, the insula is a key node in the vestibulo-insular network, receiving vestibular afferents and sending modulatory outputs to vestibular nuclei and multisensory vestibular cortical areas[9-13] Studies with direct electrical stimulation of the insula demonstrate that this region can evoke vestibular sensations, including motion perception and imbalance [13, 14]. Likewise, functional neuroimaging consistently identifies insular activation during vestibular stimulation through caloric, galvanic, or motion cues [12, 13].

The lateralized reduction of right-insular function under threat may thus produce asymmetric modulation of vestibular nuclei—either via altered descending projections or via imbalance in hemispheric integration of vestibular input. This mechanism is consistent with clinical presentations of dizziness and imbalance during stress and aligns with observations in persistent postural-perceptual dizziness (PPPD), where disrupted insular-vestibular connectivity and increased threat vigilance coexist [6, 15, 19].

Although convergent evidence supports the plausibility of this pathway, direct causal studies linking stress-induced right-insular modulation to vestibular asymmetry remain limited. Further research combining lateralized neuromodulation (*e.g.* focal transcranial stimulation), functional neuroimaging with directional connectivity analyses, and detailed vestibular physiology (*e.g.* asymmetric caloric/rotational testing

with concurrent imaging) is needed to empirically validate the proposed mechanism.

Individual Variability in Stress Responses, Vestibular Sensitivity, and Insular Function

The proposed model must be interpreted in light of substantial individual variability in stress reactivity, vestibular responsivity, and insular network function. Multiple biological factors—including age, sex, trait anxiety, prior stress exposure, and neuroplastic adaptations—can shape both the magnitude and direction of stress-induced insular modulation and its vestibular consequences.

Age-Related Differences

Aging affects vestibular hair cell density, otolith and semicircular canal signalling, and central vestibular compensation, increasing susceptibility to imbalance and dizziness. Age-related structural and functional changes in the insula—including reduced gray matter volume, altered salience network connectivity, and diminished interoceptive accuracy—may amplify the vulnerability of older adults to stress-induced vestibular asymmetry. Thus, stress-related right-insular downregulation may have disproportionately large vestibular effects in aging individuals due to reduced compensatory capacity [22, 24].

Sex Differences

Evidence from neuroimaging and psychophysiology indicates that women show stronger stress-related activation in limbic-salience and interoceptive networks, greater cortisol reactivity in certain paradigms, and higher prevalence of functional dizziness syndromes (including PPPD). Sex hormones modulate insular excitability and vestibular processing, suggesting that men and women may differ in their susceptibility to stress-induced biased vestibular modulation. Such differences may partially account for the higher clinical presentation of stress-related dizziness in female patients [25, 27].

Trait Anxiety and Affective Predisposition

Individuals with elevated anxiety traits or heightened interoceptive sensitivity show increased baseline insular activity and greater salience network reactivity to threat. These predispositions can potentiate stress-induced disruption of right-insular functioning, increase the likelihood of biased vestibular integration, and promote maladaptive predictions about bodily states. This aligns with evidence from PPPD and chronic dizziness populations, where anxiety traits and hypervigilance are key modulators of symptom persistence [28, 31].

Neuroplasticity and Prior Stress Exposure

Repeated stress induces structural remodeling and functional reorganization in insular, prefrontal, and limbic circuits. Such neuroplastic adaptations can either enhance resilience or, conversely, predispose individuals to exaggerated stress-induced insular dysfunction. In the context of vestibular control, prior vestibular insults or chronic compensation demands may similarly sensitize central vestibular pathways, rendering them more reactive to descending modulatory influences during stress [32, 34]. This interaction between stress-related cortical plasticity and vestibular plasticity may explain interindividual differences in vulnerability to stress-related dizziness.

Taken together, these factors highlight that stress-induced right-insular modulation is not uniform across individuals. Age, sex, trait anxiety, and neuroplastic history likely shape both the neural expression of stress and the degree to which descending insulo-vestibular modulation manifests as vestibular asymmetry. Incorporating these sources of variability enhances the ecological and clinical relevance of the proposed neurobiological model.

Psychophysiological Mechanisms Linking Stress and Dizziness

Psychophysiological models provide an essential framework for linking the proposed neurobiological mechanism with real-world clinical presentations of dizziness. Three domains are particularly relevant:

(1) Threat-hypervigilance. Stress increases salience network reactivity and biases sensory processing toward threat-relevant cues. Hypervigilance to internal sensations may amplify perception of minor vestibular discrepancies arising from stress-induced insular modulation, promoting maladaptive predictions such as perceived instability [35].

(2) Autonomic dysregulation. The insula plays a central role in autonomic control. Stress-induced right-insular disruption may impair integration of baroreceptive, vestibulo-cardiac, and respiratory-autonomic signals, producing symptoms like lightheadedness, tachycardia, and “dizziness-like” bodily discomfort. These autonomic perturbations interact bidirectionally with vestibular nuclei, which receive direct sympathetic and parasympathetic influences [36, 38].

(3) Somatosensory amplification. Individuals with high interoceptive sensitivity or trait anxiety may overinterpret normal vestibular fluctuations. Stress-related insular dysregulation could heighten

somatosensory amplification, reinforcing dizziness during threat contexts [39, 40].

Together, these psychophysiological mechanisms bridge the gap between the neurobiological model and clinical symptomatology, particularly in disorders such as PPPD, anxiety-related dizziness, and stress-triggered imbalance episodes.

Role of the Posterior Insula and PIVC

Although much of the stress literature emphasizes the anterior insula, the posterior insula and the parieto-insular vestibular cortex (PIVC) constitute key vestibular processing hubs [10, 41, 42]. These regions integrate multisensory vestibular inputs and contribute to spatial orientation, self-motion perception, and the bodily sense of verticality.

Structural and electrophysiological studies show that the PIVC contains dense corticofugal projections to vestibular nuclei and participates in both bottom-up and top-down control [43, 44]. Posterior insular regions may therefore act as intermediaries through which stress-induced cortical asymmetry influences vestibular brainstem circuits. Under stress, altered integration between anterior insula (interoceptive-salience) and posterior insula/PIVC (vestibular-spatial) may distort the predictive balance between expected and actual motion cues, contributing to dizziness and instability.

A more balanced framework thus requires considering interactions across the anterior–posterior insular axis and their combined corticofugal outputs.

Corticofugal Pathways: Mechanistic Clarification

Multiple corticofugal streams may mediate top-down modulation of vestibular nuclei under stress:

Direct insulo-vestibular projections from posterior and retroinsular cortices, demonstrated in tract-tracing studies [50-53].

Multisynaptic projections via the parabrachial nucleus, nucleus tractus solitarius, periaqueductal gray, and dorsal motor nucleus of the vagus, allowing integration of vestibular, autonomic, and interoceptive states [54].

Temporo-parietal corticofugal pathways, including those originating in the PIVC and temporo-parietal junction, influence vestibulo-ocular and vestibulospinal reflexes [55].

Stress-induced right-insular downregulation may modify these descending pathways by altering efferent signaling strength, altering gain, or biasing hemispheric asymmetry of brainstem vestibular outputs. This provides a mechanistic substrate consistent with clinical observations of lateralized vestibular dysfunction during stress.

Translational Clinical Evidence

Integrating clinical syndromes strengthens the translational relevance of the model:

Persistent postural-perceptual dizziness (PPPD) features hypervigilance, altered insulo-limbic connectivity, threat-driven postural control, and increased reliance on visual prediction—all compatible with the proposed insulo-vestibular mechanism [7, 15].

Anxiety-related dizziness occurs in the absence of peripheral deficits and is strongly associated with interoceptive bias and salience network hyperreactivity [56-58].

Stress-triggered episodic imbalance is reported in panic disorder, generalized anxiety, and trauma-exposed populations [48, 49], suggesting a shared pathway involving threat-modulated vestibular integration.

This convergence supports that stress-induced insular modulation can manifest clinically as dizziness even without structural vestibular injury.

Lateralization Variability in Left-Handed and Ambidextrous Individuals

Although the review focuses on right-handed individuals (approximately 85–90% of the population), lateralization patterns differ in left-handed and ambidextrous individuals. Studies indicate:

Interoceptive and salience-network lateralization may be less pronounced or even reversed [45, 47].

Vestibular cortical dominance can shift to the left hemisphere in a subset of left-handers.

Asymmetry in vestibulo-ocular reflex suppression and PIVC activation patterns varies with handedness [59].

Thus, stress-induced right-insular downregulation may not uniformly predict vestibular asymmetry across all individuals. Future research should explicitly stratify participants by handedness to clarify hemispheric variability in the proposed mechanism.

Limitations

This review is narrative in nature and therefore may be subject to interpretive bias. The lack of a formal systematic search increases risk of publication bias, and the absence of quantitative synthesis limits

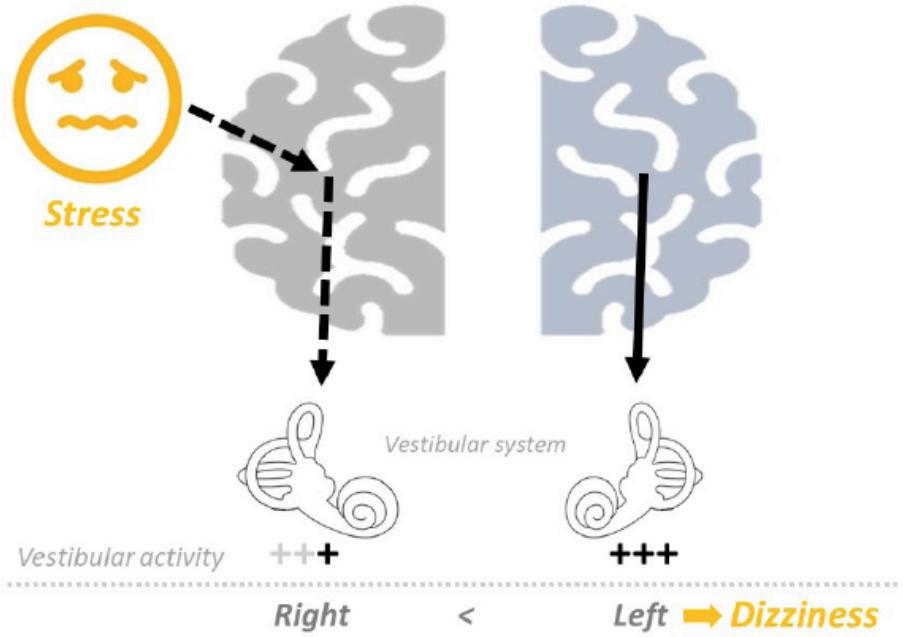

Figure 1: Proposed neurobiological mechanism of vestibular imbalance/dizziness in stress; lateralized changes of neuronal activity in parieto-insular vestibular cortex involved in self- awareness result in lateralized bottom-down regulation of the vestibular system.

objectivity regarding effect sizes. Another limitation is hemispheric variability across individuals, particularly left-handers, which the current literature often underreports. Finally, most available evidence is correlational; causal pathways are inferred rather than demonstrated empirically.

Future Directions

Future research should adopt experimental designs capable of testing the proposed causal pathway. Promising approaches include:

Lateralized neuromodulation (*e.g.*, TMS, tDCS, or focused ultrasound) targeting right anterior or posterior insula with concurrent vestibular physiological testing.

Simultaneous fMRI–vestibular stimulation combining caloric/rotational testing with effective connectivity analyses to determine directionality of insulo-vestibular influence.

High-resolution tractography to map corticofugal pathways from anterior and posterior insular sectors to brainstem vestibular nuclei.

Longitudinal stress paradigms assessing how acute and chronic stress modify insulo-vestibular coupling over time.

Stratified analyses separating right-handers, left-handers, and ambidextrous participants to clarify hemispheric variability.

These methodologies would provide direct empirical validation of the proposed model.

CONCLUSIONS

Stress modulates interoception, autonomic regulation, and bodily self-awareness through lateralized influences on the insular cortex, particularly the right anterior and posterior regions. Given the robust reciprocal connections between insular networks and vestibular pathways, such stress-induced lateralization may bias vestibular nucleus activity and produce functional vestibular asymmetry. This review proposes a novel integrative neurobiological model linking stress-related insular modulation with dizziness, bridging psychophysiology, vestibular neuroscience, and clinical syndromes such as PPPD and anxiety-related dizziness.

Although direct causal evidence in humans is lacking, the convergent literature strongly motivates targeted experimental studies. Clarifying these mechanisms may ultimately improve diagnosis, patient stratification, and neuromodulation-based treatments for stress-related dizziness and imbalance.

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AMS elaborate the paper, CA review the article.

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