Brain Interoception: The Missing Organ on the Interoceptive Map

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Abstract

Interoception — the sensing of the body's internal state — is catalogued across cardiac, pulmonary, gastrointestinal, and other organ systems. The brain is absent from this catalogue. No published paper proposes "brain interoception" as a defined concept, despite substantial evidence that the brain senses its own tissue state. This review argues that brain interoception is a coherent category forming a single continuum. At the pathological end, patients detect brain-state changes via meningeal nociceptors (headache), cortical spreading depression (migraine aura), focal neural discharge (epileptic aura), and intracranial hemorrhage (thunderclap headache) — phenomena accepted in clinical neurology but never unified as interoception. In the middle range, universal experiences of sleepiness, brain fog, mental fatigue, and caffeine withdrawal reflect the detection of metabolic brain-state changes through established mechanisms: central chemoreceptors (explicitly called interoception in respiratory physiology), astrocyte-derived adenosine signaling, and microglial surveillance. At the high-precision end, some individuals report discriminating subtle variations in cognitive readiness with sufficient resolution to guide task selection. Drawing an analogy to skeletal muscle — which has both proprioception (spindles, Golgi tendon organs) and interoception (Group III/IV metabolic afferents) — we propose that the brain similarly has both metacognition (monitoring cognitive processes) and interoception (monitoring tissue state), and that these are phenomenologically distinguishable. The classification gap reflects an unexamined Cartesian inheritance in which the brain is treated as the interpreter of interoceptive signals rather than as an organ whose state is itself interocepted. Six testable predictions are offered.

Keywords: brain interoception, interoception, metacognition, brain fog, mental fatigue, meningeal afferents, central chemoreception, predictive processing

Introduction

A migraine sufferer reports that she can feel an attack coming hours before the pain begins. A sleep-deprived researcher notices that his thinking is unreliable today and decides to postpone the analytical work he had planned. A meditation practitioner reports detecting subtle variations in cognitive readiness and adjusting her practice accordingly. A caffeine drinker, four hours past his

usual dose, senses a familiar heaviness in the head and recognizes it as withdrawal. These reports are so routine that they pass without comment. Yet none of them appear in the interoception literature.

Interoception — defined by Craig (2002, 2003) as the sense of the physiological condition of the body — catalogues the heart, the lungs, the gastrointestinal tract, the bladder, the vasculature. The Khalsa et al. (2018) roadmap published out of the 2016 NIH Interoception Summit enumerates cardiovascular, pulmonary, gastrointestinal, genitourinary, nociceptive, chemosensory, osmotic, thermoregulatory, visceral, immune, and autonomic systems. The brain is absent from this list. Garfinkel et al.'s (2015) influential tripartite model of interoceptive accuracy, sensibility, and awareness was developed and validated through heartbeat detection tasks; the brain appears only as the processor of these signals, never as their source. A systematic search of the published literature yields no paper in which "brain interoception" or "cerebral interoception" is proposed as a defined concept.

The exclusion is peculiar for several reasons. First, the brain is anatomically an organ: it has tissue, vasculature, metabolic demands, and internal states that vary over time. Second, clinical neurology has long accepted that patients sense changes in their own brain state. Headache, migraine aura, epileptic aura, the thunderclap headache of subarachnoid hemorrhage, and the prodromal sensations preceding stroke are all cases of subjective detection of intracranial events. Third, converging lines of basic research have, in recent years, identified mechanisms by which the brain monitors its own tissue state. Central respiratory chemoreceptors have been explicitly characterized as a form of interoception (Guyenet & Bayliss, 2023). Meningeal trigeminal afferents, previously understood only as nociceptors, have been shown to respond to non-noxious mechanical conditions and have been proposed as "intracranial interoceptors under physiological conditions" (Blaeser et al., 2022). Migraine has been modeled as an allostatic reset triggered by unresolved interoceptive prediction errors (Sedley et al., 2024). Astrocyte-derived adenosine has been shown to mediate the brain's sensing of its own accumulated activity, producing the homeostatic drive to sleep (Halassa et al., 2009).

These findings accumulate in parallel across fragmented literatures — headache neuroscience, respiratory physiology, sleep research, migraine theory, ecstatic epilepsy — but they have not been unified. No synthesis has yet proposed that these phenomena belong together, nor that the brain, like the heart and the gut, generates interoceptive signals about its own state that the organism can sense.

This review advances three claims. First, brain interoception is a coherent category: the brain senses and signals its own tissue state through established mechanisms, and these signals reach conscious awareness under a range of conditions. Second, brain interoception forms a continuum. At one end sits pathological brain-state sensing — headache, aura, thunderclap, prodrome — which is clinically accepted and filed under "neurological symptoms." In the middle sits general brain-state sensing — sleepiness, brain fog, mental fatigue, caffeine withdrawal — which is experienced universally and filed under "fatigue" or "subjective state." At the other end sits high-precision brain-state discrimination — the detection of subtle variations in cognitive readiness, processing speed, and integration capacity — which is filed under "introspection" or

dismissed as vague subjective impression. The labels differ but the underlying phenomenon is one: the sensing of changes in brain metabolic and functional state. Third, the classification gap that has kept these phenomena separate reflects an unexamined inheritance of Cartesian dualism. The heart, lungs, and gut belong to the body and are therefore candidates for interoception. The brain has been tacitly assigned to the mind, and its self-sensing has been parceled out into neurology (when pathological), fatigue research (when general), and introspection (when high-precision). Naming brain interoception as a single phenomenon allows the fragments to connect.

The argument proceeds as follows. Section 2 surveys the major interoception frameworks and documents the consistent absence of the brain as a source organ. Section 3 reviews the clinically established end of the continuum: headache, migraine, epileptic aura, and related phenomena. Section 4 reviews the mechanistic evidence for brain self-sensing: central chemoreceptors, glucosensing and osmoreceptive neurons, astrocytic adenosine signaling, microglial surveillance. Section 5 addresses the counter-evidence — chronic sleep deprivation, silent strokes, the meditation paradox, and the parenchymal innervation limit — and argues that these findings constrain rather than refute the continuum model. Section 6 introduces the decisive theoretical move: muscle has both proprioception and interoception; no one questions that an athlete can feel "today my legs are heavy"; the parallel for the brain is available but has not been drawn. Section 7 formalizes the continuum model and connects it to predictive processing frameworks. Section 8 offers a single-case phenomenological illustration of the high-precision end of the continuum, drawing on the documented observations of one individual with unusually acute brain-state discrimination. Section 9 considers clinical, research, and theoretical implications and sets out testable predictions. Section 10 concludes.

The Brain's Absence from Interoception Frameworks

The modern science of interoception can be traced through four landmark contributions. In each, the brain is positioned as the organ that receives and processes interoceptive signals. In none is it treated as an organ whose own physiological state is itself interocepted. The omission is never defended; it is simply architecturally assumed.

2.1 Craig's reconceptualization (2002–2009)

Craig (2002) reconceptualized interoception as "the sense of the physiological condition of the body," moving it beyond the classical viscероceptive definition of Sherrington (1906). Craig's framework centers on a specific ascending pathway: thin-fiber (Aδ and C) afferents from body tissues project via lamina I of the spinal cord to the ventromedial posterior nucleus of the thalamus, and from there to the posterior insula. In the posterior insula, a primary interoceptive representation of the body's physiological state is constructed; this representation is then re-represented in the anterior insula, which Craig (2009) proposed as the neural substrate of subjective awareness itself. The signals catalogued include pain, temperature, itch, sensual touch, muscular and visceral sensations, vasomotor activity, hunger, thirst, and the need to breathe. The framework is defined by its directionality: signals travel from body tissues to the brain. This entails that the brain is the terminus of the interoceptive pathway, never its origin. The possibility that the

brain's own tissue — its metabolic state, its neurochemical milieu, its vascular dynamics — might generate interoceptive signals about itself is not raised and not excluded; it is simply outside the frame. Craig's (2003) update specifies that the lamina I system represents "the physiological condition of all tissues of the body," a formulation capacious enough to include brain tissue, yet never applied to it.

2.2 Garfinkel et al.'s three-dimensional model (2015)

Garfinkel, Seth, Barrett, Suzuki, and Critchley (2015) introduced a tripartite distinction that has become standard: interoceptive accuracy (objective behavioral performance on detection tasks), interoceptive sensibility (self-evaluated trait-level assessment), and interoceptive awareness (the correspondence between accuracy and confidence — a metacognitive dimension). The model was developed and validated primarily using heartbeat detection paradigms. The heart serves as the canonical interoceptive organ because it produces a discrete, countable signal whose detection can be objectively verified.

The third dimension, interoceptive awareness, deserves particular attention. It measures the degree to which an individual's confidence in their own interoceptive judgments tracks their actual accuracy. This is, by definition, a metacognitive capacity: the brain monitoring and evaluating the quality of its own interoceptive processing. Yet it is not framed as brain interoception. Instead, it is positioned as a higher-order evaluative process superimposed on first-order body-signal detection. The possibility that the brain might also be a first-order source of interoceptive signals — that one might detect changes in one's own cognitive state with accuracy, sensibility, and awareness analogous to cardiac interoception — is not considered. The omission is structurally revealing: the metacognitive capacity required to evaluate one's own heartbeat detection already involves the brain sensing aspects of its own processing, but this self-referential dimension is absorbed into the evaluative framework rather than recognized as interoception.

2.3 The Khalsa et al. roadmap (2018)

Khalsa et al. (2018), writing from the 2016 NIH Interoception Summit, defined interoception as "the process by which the nervous system senses, interprets, and integrates signals originating from within the body." Their roadmap includes a comprehensive table of interoceptive domains: cardiovascular, pulmonary, gastrointestinal, genitourinary, nociceptive, chemosensory, osmotic, thermoregulatory, visceral, immune, and autonomic systems. The table is organized by organ system, and the brain does not appear as an entry. By structuring the field around peripheral organ systems, the roadmap reinforces the assumption that interoception is an outside-to-inside process: signals arise in the body's periphery and travel to the brain for processing. The brain's role is interpretive and integrative. That the brain itself might generate signals about its own tissue state — and that these signals might reach conscious awareness — lies outside the taxonomy.

2.4 The Chen et al. NIH workshop (2021): a partial opening

Chen et al. (2021), summarizing the 2019 NIH Blueprint for Neuroscience Research Workshop on Interoception, represents a transitional moment. The paper's primary definition maintains the outside-to-inside framing: "'Sensing' denotes communication from physiological systems outside of

the central nervous system (CNS) to the CNS." However, the same paper also acknowledges that "chemical interoceptors located on neurons inside the brain most likely receive interoceptive signals through non-neural pathways such as the circulatory or lymphatic systems." This is a significant concession. It recognizes that the brain contains interoceptors — molecular sensors that detect internal physiological states — while simultaneously maintaining a definitional frame in which interoception is fundamentally about peripheral-to-central communication. The tension is unresolved. If the brain contains interoceptors that detect the brain's own chemical environment, then some interoceptive sensing is not peripheral-to-central but local to the CNS itself. Chen et al. identify this possibility without fully reckoning with its implications.

2.5 The gap and its significance

A search of PubMed and Google Scholar for "brain interoception," "cerebral interoception," and "interoception of brain state" yields no papers in which these terms are proposed as defined concepts. Barrett and Simmons (2015), in their Embodied Predictive Interoception Coding (EPIC) model, come closest: they propose that interoceptive experience largely reflects limbic predictions about expected body state, a framework that could in principle accommodate brain-state predictions. Seth and Tsakiris (2018), in their "beast machine" account, argue that embodied selfhood arises from interoceptive inference about the body's essential variables; the computational architecture is modality-agnostic and could accommodate brain-tissue variables, but the extension is not made. Across the field, brain interoception remains a concept for which the evidence exists, the theoretical machinery is available, and the name is missing.

The absence demands explanation. It is not the result of an argument: no published paper contends that the brain cannot or should not be considered an interoceptive organ, nor that brain-state sensing is fundamentally different from cardiac or gastric interoception. Nor is it a consequence of missing evidence, as the following sections will show. The most parsimonious explanation is historical: interoception was defined, from Sherrington (1906) through Craig (2002) to Khalsa et al. (2018), within a framework that presupposes a sensing body and an interpreting brain. This division mirrors the Cartesian partition between res extensa and res cogitans — the material body and the thinking mind. The brain, as the organ of thought, was placed on the mind side of the partition and thereby exempted from the category of body organs whose states are interoceptively sensed. The partition was never argued for; it was inherited.

Clinical Brain-State Sensing Is Already Established

If the brain is absent from interoception frameworks, it is ubiquitous in clinical neurology. Patients routinely report sensing changes in their own brain state — and these reports are taken seriously, acted upon, and used diagnostically. The phenomena reviewed in this section occupy the pathological end of the continuum proposed in this paper. They are included not because they are novel findings, but because they have never been collectively recognized as instances of brain interoception.

3.1 Headache and meningeal interoception

The brain parenchyma itself lacks nociceptors. Headache pain arises from pain-sensitive intracranial structures: the meninges (dura mater and pia-arachnoid), the cerebral blood vessels, and the venous sinuses, all of which are innervated by trigeminal Aδ and C fibers constituting the trigeminovascular system (Messlinger & Ellrich, 2001; Levy & Moskowitz, 2023). This much is textbook neuroscience. What is not widely appreciated is that these same meningeal afferents respond to non-noxious conditions. Blaeser et al. (2022), using two-photon calcium imaging in awake mice, demonstrated that a substantial subset of meningeal trigeminal afferents are activated during ordinary locomotion. These afferents encode meningeal expansion, compression, shearing, and displacement along the dorsoventral axis, with directional tuning and graded responses to mechanical deformation. Blaeser et al. explicitly proposed that these mechanosensitive afferents may function as "intracranial interoceptors under physiological conditions," guiding cranial interoception in addition to headache pain.

This finding is pivotal for two reasons. First, it demonstrates that the meningeal innervation is not exclusively nociceptive: it provides the brain with information about intracranial mechanical conditions under normal, non-pathological circumstances. Second, it bridges the neurology literature and the interoception literature in a single paper, using the term "interoceptor" for an intracranial structure. The bridge has not yet been widely crossed. Blaeser et al. focus on meningeal mechanics rather than on the broader theoretical implications for how interoception is defined. The present paper argues that these implications are substantial: if the meninges contain interoceptors that signal non-noxious intracranial conditions, then headache — the activation of these same structures under pathological conditions — is the noxious end of a meningeal interoceptive continuum.

3.2 Migraine: interoception's strongest foothold in the brain

Migraine offers the most developed connection between interoception and brain-state sensing. Migraine aura is produced by cortical spreading depression (CSD), a slowly propagating wave of neuronal depolarization (3–6 mm/min) first described by Leão (1944) and confirmed in human fMRI by Hadjikhani et al. (2001). CSD is a massive, coordinated change in cortical state — a disturbance that patients detect subjectively as visual, sensory, or language phenomena minutes before headache onset. The detection of CSD by the patient is, in a straightforward sense, the brain sensing a change in its own cortical activity.

More significant for the present argument is the migraine prodrome. Distinct from aura, the prodrome consists of symptoms appearing hours to days before the headache phase: fatigue, concentration difficulty, neck stiffness, mood changes, food cravings, photophobia, yawning. Schwedt et al. (2025), in the PRODROME trial, found that qualifying prodromal events were followed by headache within one to six hours at a mean rate of 84.4% per participant, with 76.9% of participants achieving this consistency at least 75% of the time, establishing the prodrome as a reliable if imperfect predictive signal. Importantly, prodromal symptoms are not localized to a single neural system; they reflect a distributed change in brain state, likely originating from hypothalamic and brainstem circuits (Maniyar et al., 2014). Patients who learn to recognize their prodrome are, in effect, performing brain interoception: detecting a global shift in central nervous system state from internal cues.

The theoretical integration has already begun. Sedley et al. (2024) proposed that migraine is an allostatic reset triggered by the accumulation of unresolved interoceptive prediction errors. In this model, the brain's predictive processing system continuously generates predictions about internal body state; when the mismatch between predictions and actual afferent signals (the prediction error) exceeds a threshold for too long, the migraine attack serves as a corrective reset. The framework is explicitly built on predictive interoception (Seth, 2013; Barrett & Simmons, 2015) and positions migraine as a disorder of interoceptive regulation. The prodrome, in this account, reflects the brain's detection of escalating prediction error — a signal that the system is approaching the reset threshold. Jones et al. (2025) found that migraineurs show normal heartbeat detection accuracy but altered self-reported interoceptive behavior, suggesting that migraine involves changes in interoceptive processing style rather than global interoceptive ability. A 2026 fMRI study reported altered dorsal anterior insula functional connectivity related to interoceptive awareness in migraineurs, further localizing the interoceptive dimension of the disorder.

3.3 Epileptic aura and ecstatic epilepsy

Epileptic auras, classified under current ILAE terminology as focal aware seizures, are subjective experiences preceding seizure generalization. In temporal lobe epilepsy, common auras include rising epigastric sensation, fear, déjà vu, jamais vu, olfactory and gustatory hallucinations, and a range of experiential phenomena (Foldvary-Schaefer & Unnwongse, 2011). These experiences are generated by abnormal focal neural activity, and patients detect them as signals of impending seizure. The aura is the patient sensing a change in their own brain's electrical activity — a capacity so clinically routine that its theoretical significance is overlooked.

One subtype of epileptic aura has received sustained attention in the interoception literature. Ecstatic epilepsy, a rare condition in which seizures produce feelings of bliss, mental clarity, and profound certainty, was first linked to the anterior insular cortex by Picard and Craig (2009). Craig's involvement is notable: the same researcher who defined the modern interoception framework also co-authored the proposal that ecstatic auras arise from the interoceptive cortex. Subsequent work by Picard et al. (2013), using SPECT and stereo-EEG, confirmed discharge propagation to the anterior insula during ecstatic seizures. Nencha et al. (2022) achieved the first evocation of an ecstatic experience through direct electrical stimulation of the dorsal anterior insula in a patient without ecstatic epilepsy, demonstrating that the experience is producible by insular activation. Picard et al. (2023) proposed the most complete neurocognitive account: the ecstatic state arises from the suppression of interoceptive prediction errors in the dorsal anterior insula, producing the subjective impression of a perfectly predicted world — certainty, clarity, and bliss as the experiential correlate of zero prediction error.

The ecstatic epilepsy literature thus already connects a subjective brain-state experience directly to interoceptive processing in the anterior insula. What it has not done is generalize: if abnormal insular activity during a seizure produces a dramatic subjective experience of brain-state change, then normal insular activity during ordinary cognitive work may produce subtler but functionally analogous signals — the everyday sense of cognitive clarity, fog, or readiness.

3.4 Subarachnoid hemorrhage and stroke

The thunderclap headache of subarachnoid hemorrhage (SAH) — sudden-onset, maximal-intensity headache caused by aneurysm rupture and blood entering the subarachnoid space — is clinically distinctive in that patients almost universally report an immediate sense that something is catastrophically wrong (Beck et al., 2006). Sentinel headaches, milder precursory headaches occurring days to weeks before major SAH, are reported in 10 to 43% of cases (Viarasilpa et al., 2020). These sentinels represent the detection of minor intracranial bleeding via the same meningeal afferents that Blaeser et al. (2022) identified as intracranial interoceptors. Yet the SAH literature has never engaged with the interoception framework. The thunderclap headache — quite literally the acute interoceptive detection of intracranial hemorrhage — remains classified as a neurological emergency rather than recognized as what it also is: the most dramatic example of brain interoception.

Stroke presents a more complex picture. Pre-stroke interoceptive sensing is poorly supported: silent strokes outnumber symptomatic ones by approximately ten to one, transient ischemic attack symptoms are frequently misattributed, and pre-stroke cognitive trajectories show no consistent subjective decline. The stroke literature's engagement with interoception has focused exclusively on post-stroke deficits. Garcia-Cordero et al. (2016) documented impaired cardiac interoceptive accuracy in patients with fronto-insular lesions, demonstrating that insular damage compromises interoception. This finding supports the centrality of the insula to interoceptive processing but does not address whether stroke patients sense the onset of their own cerebrovascular event. The contrast with SAH is informative: SAH produces an unmistakable signal (blood activating meningeal nociceptors), while ischemic stroke typically does not activate nociceptive pathways, resulting in clinical presentation through focal deficits rather than interoceptive alarm. This pattern is consistent with, rather than contrary to, the brain interoception continuum: the detectability of a brain-state change depends on whether it activates interoceptive pathways, just as the detectability of a cardiac event depends on whether it produces signals accessible to cardiac interoceptors.

Mechanistic Evidence for Brain Self-Sensing

The clinical phenomena reviewed in Section 3 establish that patients detect brain-state changes under pathological conditions. This section asks the prior question: through what mechanisms does the brain sense its own tissue state? The answer, drawn from respiratory physiology, metabolic neuroscience, glial biology, and neuroanatomy, is that several such mechanisms are already well characterized — and at least one has been explicitly identified as interoception.

4.1 Central chemoreceptors: interoception by name

The strongest existing precedent for brain interoception comes from respiratory physiology. Central chemoreceptors are neurons and astrocytes within the brainstem that detect changes in CO■, H■, and pH in the brain's interstitial fluid and cerebrospinal fluid. The retrotrapezoid nucleus (RTN), medullary raphe serotonergic neurons, and locus coeruleus noradrenergic neurons have all been identified as distributed chemoreceptor sites (Nattie & Li, 2012). RTN neurons possess intrinsic pH sensitivity mediated by TASK-2 potassium channels and GPR4 proton-sensing receptors, supplemented by paracrine signaling from nearby astrocytes that detect CO■ and relay

the signal via ATP release (Guyenet & Bayliss, 2023). The system is exquisitely sensitive: small changes in arterial CO■ — within the range produced by ordinary variations in metabolic rate — produce graded adjustments in ventilatory drive.

What makes this literature decisive for the present argument is its terminology. Guyenet and Bayliss (2023) describe central respiratory chemoreception as "a type of interoception." Bayliss et al. (2023), proposing formal criteria for identifying central respiratory chemoreceptors, characterize the system as "an interoceptive homeostatic system." These are not metaphorical uses of the term. The authors are stating, within the conventions of their field, that the brain detecting the chemical composition of its own tissue environment is interoception. The brain is both the sensing organ and the organ being sensed. The implication — that other forms of brain self-sensing might similarly qualify as interoception — is available but has not been pursued.

Central chemoreception also exhibits the continuum structure proposed in this paper. Under normal conditions, small pH fluctuations produce unconscious adjustments to breathing — the healthy baseline of brain interoception. Under moderate perturbation (e.g., rebreathing, high altitude), air hunger emerges as a conscious interoceptive signal. Under severe perturbation (e.g., respiratory failure, CO■ narcosis), the signal becomes overwhelming. The gradient from unconscious homeostatic regulation through conscious discomfort to pathological alarm mirrors the continuum from high-precision discrimination through general awareness to pathological brain-state sensing proposed for brain interoception more broadly.

4.2 Glucosensing and osmoreception: the brain's metabolic windows

The brain monitors its own metabolic environment through specialized neurons that use glucose as a signaling molecule. Levin et al. (2004) established that glucosensing neurons, using glucokinase as the rate-limiting sensor, form a distributed network across the ventromedial hypothalamus, arcuate nucleus, lateral hypothalamus, and brainstem. These neurons come in two functional types: glucose-excited neurons that increase firing as glucose rises, and glucose-inhibited neurons that increase firing as glucose falls. Together they provide bidirectional monitoring of the brain's fuel supply. Routh et al. (2014) further characterized how these neurons integrate glucose signals with other metabolic and hormonal inputs.

The circumventricular organs (CVOs) — the subfornical organ (SFO) and the organum vasculosum of the lamina terminalis (OVLT) — constitute the brain's most direct interface with the body's internal milieu. Lacking a blood-brain barrier, these structures expose specialized neurons directly to circulating blood. Single SFO neurons can integrate multiple simultaneous signals: osmolarity, glucose concentration, angiotensin II, and ghrelin (Ferguson, 2014). The CVOs thus function as multimodal interoceptors operating within brain tissue itself. Their output drives thirst, hunger, and vasopressin secretion — homeostatic responses that are among the most familiar examples of interoception, yet their anatomical location within the brain is rarely noted in the interoception literature.

4.3 Astrocyte-derived adenosine: the brain sensing its own fatigue

Perhaps the most compelling case of brain interoception in the non-pathological range comes from sleep neuroscience. Astrocytes, long regarded as passive support cells, are now understood to actively monitor neural activity through glutamate uptake and to coordinate metabolic support through the astrocyte-neuron lactate shuttle (Pellerin & Magistretti, 1994; Magistretti & Allaman, 2018). In a landmark study, Halassa et al. (2009) demonstrated that genetically inhibiting SNARE-dependent gliotransmission in astrocytes attenuated the accumulation of sleep pressure and prevented the cognitive deficits normally produced by sleep deprivation. The mediating molecule is adenosine, a byproduct of ATP metabolism that accumulates during sustained wakefulness and acts on neuronal A■ receptors to promote sleep.

The adenosine sleep-pressure system constitutes brain interoception in a precise sense: the brain detects the cumulative metabolic cost of its own activity (via adenosine accumulation), generates a homeostatic signal (sleep pressure), and this signal reaches conscious awareness as the subjective experience of sleepiness. Nadjar et al. (2013) showed that inflammation-induced sleep pressure operates through the same astrocytic adenosine mechanism, extending the system to immune-mediated brain-state sensing. Caffeine exerts its wake-promoting effect by blocking adenosine A■ and A■A receptors (Huang et al., 2005), and caffeine withdrawal symptoms — headache, fatigue, concentration difficulty, measurable increases in cerebral blood flow velocity and EEG theta power (Jones et al., 2000; Sigmon et al., 2009) — represent the subjective detection of adenosine receptor upregulation. That caffeine withdrawal is experienced as a change in brain state, and that this experience tracks objectively measurable neurophysiological changes, supports the characterization of the adenosine system as generating interoceptive signals about brain tissue state.

4.4 Microglia and the blood-brain barrier: surveillance systems

The brain possesses additional mechanisms for monitoring its own internal environment. Microglia, once described as the brain's "resting" immune cells, are now recognized as continuously active surveyors of the neural microenvironment. Hanisch and Kettenmann (2007) demonstrated that microglia constantly extend and retract processes to sample the surrounding tissue. Badimon et al. (2020) showed that microglia sense neuronal activity through P2Y12 purinergic receptors and provide negative feedback via adenosine acting on neuronal A■ receptors — directly detecting hyperactive neurons and suppressing their firing. This constitutes a local feedback loop in which the brain senses and regulates its own activity state at the cellular level.

The blood-brain barrier (BBB) itself functions as a signaling interface. Its endothelial cells express pattern recognition receptors including Toll-like receptors and inflammasome components, enabling the detection of circulating inflammatory signals (Varatharaj & Galea, 2017). Systemic inflammation can thus be sensed at the BBB and transduced into central signals that alter brain function — producing sickness behavior, cognitive slowing, and the subjective experience of brain fog. Greene et al. (2024) demonstrated BBB disruption in patients with long-COVID cognitive impairment, with subjective brain fog correlating with objective neurobiological changes including elevated serum markers persisting for up to a year.

4.5 Sotnikov's hypothesis: primary interoceptive neurons in brain parenchyma

Sotnikov (2005, 2006) proposed the most anatomically direct form of brain interoception: primary interoceptive sensory neurons residing within the brain parenchyma itself. His analysis catalogued several candidate cell types — ciliated neurons, supraependymal plexuses and intraependymal neurons contacting cerebrospinal fluid, Cajal-Retzius neurons in the cortical boundary layer, paravasal neurons of Dolgo-Saburov in the brain and spinal cord, Lugaro cells in the cerebellum, and NO-positive cortical neurons whose asynaptic dendrites extend into the precapillary space. Sotnikov hypothesized that these neurons participate in short autonomic reflex arcs controlling local metabolism, analogous to the intramural metasympathetic sensory neurons described by Dogiel in visceral organs.

This hypothesis must be treated with caution. The editorial board of the publishing journal noted that "many of the views and conclusions expressed by Professor O.S. Sotnikov are not generally accepted and are controversial" (Chumasov, 2006). The identified structures — CSF-contacting neurons, paravasal neurons — are independently documented, but their function as interoceptive sensory neurons has not been validated by subsequent independent investigation. The hypothesis remains provocative and unconfirmed. It is mentioned here for completeness and because it represents the only published attempt to identify brain-intrinsic interoceptive neurons by explicit analogy with visceral interoceptors. The broader argument of this paper does not depend on Sotnikov's hypothesis: the mechanisms reviewed in Sections 4.1 through 4.4 — central chemoreceptors, glucosensing neurons, circumventricular organs, astrocytic adenosine signaling, microglial surveillance, and BBB-mediated immune sensing — are independently established and collectively sufficient to support the concept of brain interoception.

Counter-Evidence and Limits

A responsible proposal of brain interoception must reckon with the evidence that brain-state sensing is often inaccurate, absent, or systematically biased. This section reviews four categories of counter-evidence and argues that they constrain the continuum model without refuting it. The parallel with cardiac interoception is instructive throughout: the existence of poor heartbeat detectors does not invalidate cardiac interoception as a phenomenon; it establishes that interoceptive accuracy varies across individuals and conditions. The same logic applies to the brain.

5.1 Chronic sleep deprivation: the brain's blind spot

The most damaging evidence against naive claims of accurate brain interoception comes from sleep research. Van Dongen et al. (2003) restricted healthy adults to four or six hours of sleep per night for fourteen days and found that subjective sleepiness ratings plateaued after the first few days, while objective cognitive performance — measured by the psychomotor vigilance task — continued to decline linearly throughout the study period. Participants were, in the authors' words, "largely unaware of these increasing cognitive deficits." Van Dongen et al. (2004) subsequently demonstrated stable, trait-like individual differences in vulnerability to sleep deprivation, but subjective insight remained poor across the sample.

This finding is a genuine challenge. It shows that under conditions of chronic, gradual brain-state change, subjective monitoring fails: people do not accurately sense the progressive degradation of their own cognitive capacity. However, two qualifications are important. First, the failure is specific to chronic, incremental change. During acute total sleep deprivation, subjective sleepiness tracks performance decline more closely in the short term, suggesting that the brain-state signal is available but adapts to a new baseline under chronic conditions — an interoceptive recalibration analogous to sensory adaptation in other modalities. Second, the Van Dongen finding concerns accuracy of magnitude estimation (how impaired am I?), not detection (is something different?). Many chronically sleep-deprived individuals do report a sense that something is off, even when they underestimate the degree of impairment. The distinction between detection and calibration is well established in the cardiac interoception literature (Garfinkel et al., 2015) and applies here: the brain may detect a state change without accurately estimating its magnitude.

5.2 Silent strokes: interoception requires a pathway

Silent cerebral infarctions outnumber symptomatic strokes by approximately ten to one (Vermeer et al., 2007). Most small-vessel ischemic events produce no subjective experience whatsoever, despite causing measurable tissue damage. This finding appears to contradict the claim that the brain senses its own state.

The resolution lies in mechanism specificity. Brain interoception, like all interoception, requires an afferent pathway. Cardiac interoception depends on baroreceptor and cardiac afferent activation; cardiac events that fail to engage these pathways (such as painless myocardial infarction, which occurs in roughly 20–25% of cases) go undetected. Silent strokes are the cerebral equivalent: small-vessel occlusions in white matter or deep gray matter do not activate meningeal nociceptors, do not trigger cortical spreading depression, and do not produce the metabolic perturbation sufficient to engage central chemoreceptors or astrocytic signaling at a conscious level. The absence of detection reflects the absence of pathway engagement, not the absence of brain interoceptive capacity. Large-vessel strokes involving cortical territory are far more likely to be subjectively experienced — precisely because they disrupt neural activity on a scale that engages detectable metabolic and electrical changes. The pattern is consistent with the continuum model: brain interoception detects changes that are large enough, fast enough, and localized to regions with sufficient interoceptive innervation.

5.3 The meditation paradox

Meditation practice is widely reported to enhance interoceptive sensitivity. Long-term meditators show neuroplastic changes in interoceptive brain regions, particularly the insula and anterior cingulate cortex (Farb et al., 2013; Sevinc et al., 2018), and report heightened awareness of bodily sensations. However, Khalsa et al. (2008) found that experienced meditators did not outperform non-meditators on heartbeat detection accuracy — the gold standard measure of objective interoceptive performance. This dissociation between subjective sensibility and objective accuracy has been replicated across several studies.

The meditation paradox poses a nuanced challenge. It does not show that meditation fails to alter brain processing of interoceptive signals; it shows that the enhancement operates on sensibility and attention rather than on detection threshold. In Garfinkel et al.'s (2015) terms, meditation may improve interoceptive sensibility (the disposition to attend to body signals) without improving interoceptive accuracy (the ability to detect a specific signal correctly). For brain interoception, this distinction matters: an individual who meditates may become more attentive to cognitive state variations without becoming more accurate in discriminating them. Enhanced attention is not the same as enhanced detection — a conclusion that should inform the interpretation of self-reported brain-state sensing in high-awareness populations.

5.4 Anatomical constraints: the parenchymal limit

A persistent anatomical objection to brain interoception concerns the innervation of brain tissue. The brain parenchyma — the neural tissue itself — lacks conventional sensory nerve endings. Peripheral sensory fibers innervating intracranial structures (meninges, blood vessels) lose their autonomic and sensory fiber coverage as vessels penetrate into brain parenchyma beyond the Virchow-Robin perivascular space. The brain therefore cannot sense its own tissue state through the same mechanism by which the gut senses distension or the skin senses temperature: there are no peripheral sensory neurons embedded in the neural tissue.

This objection is anatomically correct but conceptually incomplete. The mechanisms reviewed in Section 4 do not depend on peripheral sensory innervation. Central chemoreceptors are neurons within the brainstem that are themselves chemosensitive. Glucosensing neurons detect glucose through their own glucokinase activity. Circumventricular organs use their own blood-brain barrier-free exposure. Astrocytes detect neural activity through glutamate uptake at their own synaptic contacts. Microglia sense neural activity through their own purinergic receptors. In every case, the sensing element is intrinsic to the central nervous system, not projected from the periphery. The brain's mode of self-sensing is different from the gut's or the heart's — it relies on chemosensitive neurons, metabolically responsive glia, and barrier-free vascular interfaces rather than on afferent sensory nerve endings — but the functional outcome is analogous: the detection of tissue-state changes and the generation of signals that can reach conscious awareness. The absence of conventional sensory innervation in brain parenchyma is a constraint on mechanism, not a barrier to function.

5.5 Summary: limits define the shape of the continuum

The counter-evidence reviewed in this section does not undermine the concept of brain interoception. Rather, it specifies the conditions under which brain interoception succeeds and fails. Acute, large-scale, rapidly developing brain-state changes (migraine aura, SAH thunderclap, acute sleep deprivation) are reliably detected. Chronic, gradual, small-scale changes (chronic sleep restriction, silent infarction) are poorly detected. Enhanced attention to brain states (meditation) improves subjective sensibility but not necessarily objective accuracy. And the mechanism of brain self-sensing differs from peripheral interoception, relying on intrinsic chemosensitivity and glial signaling rather than afferent nerve endings.

This pattern mirrors the broader interoception literature. Cardiac interoception accuracy ranges from near-zero to near-perfect across individuals (Schandry, 1981). Painless myocardial infarction demonstrates that even cardiac events can go undetected. Gastric interoception is less accurate than cardiac interoception, and respiratory interoception is modulated by attention and anxiety. No organ system offers perfect, universal interoceptive coverage. The brain is no exception — it is simply the organ whose interoceptive profile has not yet been characterized.

The Muscle Dual-Sensing Analogy

The theoretical move proposed in this section is simple in structure but has not, to the authors' knowledge, been made in the published literature. Skeletal muscle possesses two distinct sensory systems: proprioception, which monitors position, length, and tension through muscle spindles and Golgi tendon organs; and interoception, which monitors metabolic state through thin-fiber Group III and IV afferents. The brain, we argue, possesses an analogous duality: metacognition, which monitors cognitive processes; and brain interoception, which monitors the brain's metabolic and functional state. The muscle analogy makes the concept of brain interoception intuitive because no one doubts that muscle interoception exists.

6.1 Muscle: two sensing systems, one organ

The proprioceptive system of skeletal muscle is well characterized. Muscle spindles, innervated by large-diameter Group Ia and II afferents, detect muscle length and velocity of stretch. Golgi tendon organs, innervated by Group Ib afferents, detect force. Together, these receptors support position sense, kinesthesia, and the coordination of voluntary movement. The pathway is fast, myelinated, and projects through the dorsal column-medial lemniscal system to the somatosensory cortex.

Alongside this proprioceptive system, skeletal muscle has an interoceptive system that is less widely appreciated outside exercise physiology. Group III (thinly myelinated Aδ) and Group IV (unmyelinated C) muscle afferents detect the metabolic state of the tissue. Jankowski et al. (2013), using comprehensive phenotyping of mouse muscle afferents, identified two functional subtypes among these thin-fiber neurons: metaboreceptors, which respond to innocuous levels of metabolites produced during normal exercise (lactate, ATP, protons at physiological concentrations), and metabonociceptors, which respond only to noxious metabolite levels produced during ischemic or injurious conditions. The metaboreceptor-metabonociceptor distinction maps directly onto a continuum: under normal exercise, metaboreceptors provide graded feedback about muscular effort and metabolic cost; under pathological conditions, metabonociceptors generate pain and alarm.

Wilson, Andrew, and Craig (2002) demonstrated that muscle contraction activates lamina I spinothalamic neurons — the same neurons that Craig (2002) identified as the core of the interoceptive afferent pathway. This places muscle metabolic sensing squarely within the interoceptive system. Amann et al. (2015) established that Group III/IV afferents both optimize exercise performance through interoceptive feedback and contribute to central fatigue by signaling the brain that the muscle's metabolic reserves are depleting.

The resulting picture is one of dual sensing: muscle spindles and GTOs tell the brain where the muscle is and how much force it is producing (proprioception); Group III/IV afferents tell the brain what metabolic condition the muscle is in (interoception). The athlete's report that "today my legs feel heavy" or "my power is fine but my coordination is off" reflects the integration of these two streams. No one treats these reports as vague subjectivity or dismisses them as introspective confabulation. They are recognized as what they are: the sensing of an organ's state.

6.2 The brain: one organ, two sensing systems?

The parallel to the brain is available but has not been drawn in the published literature. Metacognition — the monitoring and control of cognitive processes (Flavell, 1979; Nelson & Narens, 1990) — is functionally analogous to proprioception. It tells the brain about its own cognitive operations: how difficult a task is (feeling of difficulty), whether a memory is stored (feeling of knowing), whether learning has occurred (judgment of learning), whether processing is fluent or effortful. These metacognitive signals report on cognitive process, just as proprioceptive signals report on motor process.

Brain interoception, as proposed in this paper, would be functionally analogous to muscle interoception. It would tell the brain about its own metabolic and functional state: how well-rested the tissue is, how efficiently fuel is being supplied, whether neuroinflammation is present, whether the neurochemical milieu supports optimal processing. The mechanisms reviewed in Section 4 — central chemoreceptors, glucosensing neurons, astrocytic adenosine signaling, microglial surveillance — are the brain's equivalents of Group III/IV muscle afferents: they detect tissue-state variables rather than information-processing variables.

The distinction matters because metacognition and brain interoception can dissociate. An individual might metacognitively detect that a task is difficult (high processing demand) without interoceptively detecting that the brain is fatigued (depleted metabolic resources). Conversely, an individual might interoceptively sense brain fog (altered metabolic state) without metacognitively identifying which cognitive processes are impaired. The athlete's "my coordination is off but my power is fine" has a cognitive parallel: "something feels wrong with my thinking today, but I can't pinpoint which function is affected." The two systems report on different aspects of the same organ.

6.3 Why the analogy has not been drawn

If the parallel is as straightforward as it appears, why has it not been proposed? Three factors may contribute. First, the muscle dual-sensing literature sits in exercise physiology, while metacognition sits in cognitive psychology; the two fields rarely engage with each other. Second, the embodied cognition movement, which might have been expected to bridge the gap, has focused on the influence of body states on cognition rather than on the brain sensing its own tissue state — the direction of influence is typically body-to-mind rather than brain-to-brain. Third, and most fundamentally, the Cartesian inheritance described in Section 2 sorts muscle into "body" (eligible for interoception) and brain into "mind" (exempt from interoception). The muscle analogy makes the double standard visible: if we accept that the athlete senses the metabolic state of her quadriceps, we should accept that the same organism senses the metabolic state of her prefrontal

cortex. The mechanisms are different (Group III/IV afferents versus central chemoreceptors and astrocytic signaling), but the functional principle is the same: an organ monitoring its own tissue condition and making that information available to the organism.

McMorris, Barwood, and Corbett (2018) came closest to drawing this parallel. In their paper proposing an interoceptive model of central fatigue during endurance exercise, they explicitly bridge Noakes' central governor concept with Craig's interoception framework. However, their analysis focuses on the brain sensing the body's muscular state during exercise — the brain as recipient of peripheral muscle interoceptive signals — rather than the brain sensing its own state. The final step — from the brain sensing muscle fatigue to the brain sensing brain fatigue — was available but not taken.

The Brain Interoception Continuum

The preceding sections have established that the brain is excluded from interoception frameworks (Section 2), that clinical brain-state sensing is well documented (Section 3), that mechanistic substrates for brain self-sensing exist (Section 4), that the counter-evidence constrains rather than refutes the concept (Section 5), and that the muscle analogy provides a compelling precedent (Section 6). This section formalizes the proposed continuum model and connects it to predictive processing.

7.1 The continuum

Brain interoception, as proposed here, is the sensing of the brain's own metabolic, neurochemical, vascular, and functional state by mechanisms intrinsic to the central nervous system, producing signals that can — under appropriate conditions — reach conscious awareness. A terminological clarification is warranted. The phenomena reviewed in this paper span two anatomically related but distinct categories: intracranial interoception (sensing of conditions within the cranial cavity, including meningeal and vascular events) and brain-tissue interoception (sensing of conditions within the neural parenchyma itself, including metabolic state, neurochemical milieu, and functional capacity). Headache and SAH thunderclap headache involve meningeal and vascular structures that are intracranial but not brain parenchyma; central chemoreception and astrocytic adenosine signaling involve brain tissue proper. The continuum model encompasses both, on the grounds that both inform the organism about the state of the organ housed within the cranium. The distinction matters mechanistically — meningeal interoception uses conventional trigeminal afferents, while parenchymal sensing uses chemosensitive neurons and glia — but the functional principle is shared: the detection of intracranial conditions that the organism can act upon.

At the pathological end, large-magnitude, rapid-onset brain-state changes engage nociceptive or high-threshold interoceptive pathways and produce vivid conscious experiences. Headache arises from meningeal nociceptor activation. Migraine aura is the conscious detection of cortical spreading depression. Epileptic aura is the subjective detection of focal abnormal neural discharge. Thunderclap headache in SAH is the acute detection of intracranial hemorrhage via meningeal interoceptors. These phenomena are classified as neurological symptoms and are managed by neurologists.

In the middle range, moderate-magnitude, typically gradual brain-state changes engage metabolic and homeostatic interoceptive pathways and produce familiar but less precisely articulated conscious experiences. Sleepiness reflects adenosine accumulation detected through astrocytic signaling. Brain fog during illness reflects neuroinflammation sensed through BBB-mediated and microglial pathways. Mental fatigue reflects the depletion of cognitive resources, integrated as an interoceptive cost signal in the anterior insula and anterior cingulate cortex (Müller & Apps, 2019; Boksem & Tops, 2008). Caffeine withdrawal symptoms reflect adenosine receptor upregulation producing measurable cerebrovascular and electrophysiological changes (Jones et al., 2000). These phenomena are classified as fatigue, subjective states, or mood and are typically managed — to the extent they are managed — by sleep hygiene, lifestyle adjustment, or pharmacology.

At the high-precision end, small-magnitude variations in brain functional state are detected with sufficient resolution to guide behavioral decisions. An individual senses that cognitive processing is slightly slower today, that integration across domains is less fluid, that a particular type of work is feasible while another is not. These discriminations resemble the athlete's sense that "today my legs are heavy" — a detection of organ state that is subjectively clear, behaviorally consequential, and not yet measured by any standard research instrument. These phenomena are classified as introspection, metacognition, or subjective self-assessment, and they are frequently dismissed as unreliable.

The labels — neurological symptom, fatigue, introspection — differ, but the underlying phenomenon is one: the brain sensing changes in its own tissue state. The continuum model proposes that these three ranges are not categorically distinct phenomena requiring separate explanatory frameworks; they are regions of a single continuum, distinguished by magnitude, rate of change, and pathway engagement. The classification boundaries that currently separate them reflect the disciplinary boundaries between neurology, sleep medicine, and cognitive psychology — and, at a deeper level, the Cartesian partition between body-as-machine and mind-as-observer.

7.2 Connection to predictive processing

The continuum model is compatible with, and strengthened by, the predictive processing account of interoception. Seth (2013) proposed that subjective feeling states arise from actively inferred generative models of the causes of interoceptive afferents. Barrett and Simmons (2015) developed this into the Embodied Predictive Interoception Coding (EPIC) model, in which the brain continuously generates predictions about expected body state, and interoceptive experience is constituted by the prediction error — the discrepancy between predicted and actual interoceptive input. Seth and Tsakiris (2018) extended this framework to propose that embodied selfhood is grounded in interoceptive inference about essential physiological variables.

These frameworks are computationally modality-agnostic. The predictive architecture does not specify which organ's signals it processes; it generates predictions and computes prediction errors for any interoceptive input. If the brain generates predictions about its own metabolic state — anticipated glucose availability, expected adenosine levels, predicted neuroinflammatory state — then mismatches between these predictions and actual brain-tissue signals would constitute brain

interoceptive prediction errors. Sedley et al. (2024) have already instantiated this logic for migraine: the migraine attack is the consequence of accumulated interoceptive prediction errors exceeding a threshold. Picard et al. (2023) have instantiated it for ecstatic epilepsy: the ecstatic experience is the subjective correlate of suppressed interoceptive prediction errors in the anterior insula.

The extension to non-pathological brain interoception follows naturally. The everyday sense that "my thinking is clear today" or "something feels off" may reflect the brain's interoceptive prediction error regarding its own functional state — the mismatch between the predicted level of cognitive performance and the actual metabolic and functional signals. When the mismatch is small, processing feels smooth and effortless (low prediction error). When the mismatch is large, processing feels foggy or unreliable (high prediction error).

This formulation invites a reexamination of what the metacognition literature calls "feeling of difficulty" (Efklides, 2006, 2011). Existing accounts treat feeling of difficulty as a unitary metacognitive signal about task demands. The brain interoception framework suggests it may contain two distinguishable components: a task-derived component (this problem is hard — a report on the processing object) and a tissue-state-derived component (my brain is heavy — a report on the processing organ). These are phenomenologically distinct: one can encounter a difficult problem while feeling cognitively sharp, or encounter an easy problem while feeling cognitively dull. The former is pure metacognition; the latter is brain interoception. In practice, Efklides's "feeling of difficulty" likely conflates both. The dual-sensing model proposed in this paper (metacognition ≈ proprioception of cognitive process; brain interoception ≈ interoception of brain tissue state) predicts that individuals with high brain interoceptive precision should be able to separate these two components, reporting task difficulty and brain-state quality as independent dimensions — just as an athlete can independently report "this movement is technically demanding" and "my muscles are fatigued."

A Single-Case Phenomenological Illustration

The continuum model developed in the preceding sections predicts the existence of individuals at the high-precision end — people who detect subtle variations in their own brain state with sufficient resolution to guide behavioral decisions. This section presents a phenomenological illustration drawn from one such individual, referred to as Participant A.

Participant A is a male adult in his forties, self-identified as twice-exceptional (2E). He has a documented history of unusually acute interoceptive sensitivity across multiple modalities and has engaged in systematic self-observation of his cognitive and bodily states over several decades. The observations reported here were collected through structured phenomenological interview and are supplemented by one session of consumer-grade EEG recording (Muse S headband) conducted during ordinary cognitive work. The purpose of this section is illustrative, not confirmatory: it aims to show what the high-precision end of the brain interoception continuum looks like in practice, grounded in the specific observations of one individual whose case is consistent with the continuum model. It does not establish prevalence, typicality, or causal mechanism. Ethics: Participant A provided informed consent for the use of anonymized data. The

observation protocol was self-initiated and compliant with the principles of the Declaration of Helsinki for self-experimentation; no institutional ethics review was required.

8.1 General interoceptive precision

Participant A reports the ability to estimate his own blood pressure within approximately ±5 mmHg. During one documented hospital visit, his measured systolic blood pressure was 72 mmHg; he had identified the hypotensive state prior to measurement and had attributed his earlier loss of consciousness to it. While a single verification event does not establish sustained accuracy, this level of cardiovascular interoceptive precision, if confirmed, would place Participant A at the extreme high end of the documented range of individual differences in the heartbeat detection literature (Schandry, 1981; Garfinkel et al., 2015). It suggests high interoceptive sensitivity in at least one measurable modality.

Participant A also reports the ability to detect fascial tissue state changes — specifically, distinguishing myofascial densification from other tissue states following percussive stimulation. His description of the sensation ("the stimulus entered the fascial layer; the tissue has been in a constrained range of motion for too long") was consistent with the clinical concept of fascial densification as characterized by Stecco et al. (2011), involving hyaluronic acid viscosity changes in deep fascial layers.

8.2 Brain-state discrimination

The observation most directly relevant to the present paper is Participant A's report of discriminating subtle variations in brain functional state. He describes this as detecting differences in cognitive readiness — a composite sense that encompasses processing speed, the capacity to integrate information across domains, and the degree to which cognitive operations feel fluid versus effortful. Critically, he reports using these discriminations to make practical decisions: selecting which type of work to undertake (analytical, creative, editorial, routine), adjusting expectations for performance, and recognizing when a particular cognitive mode is unavailable.

The phenomenological structure of this discrimination is worth specifying. Participant A does not report a single scalar signal (e.g., "brain at 80%"). Rather, he reports a multidimensional sense in which different aspects of cognitive function can vary independently — analogous to the athlete who reports that power is available but coordination is impaired, or that endurance is present but reaction time is degraded. On some occasions, he reports that conceptual integration across domains is fluid while sequential analytical processing is slow; on others, the pattern is reversed. The discrimination is not introspective in the sense of deliberate self-examination; it is experienced as a direct, pre-reflective sense of cognitive state, similar to the way one senses hunger or cold without having to check.

The relationship to general brain-state variables is apparent to Participant A. Sleep deprivation produces recognizable degradation patterns. Caffeine state (presence, absence, withdrawal) is tracked as a modulator. Post-rest recovery is sensed and calibrated against expected recovery trajectories. These reports are consistent with the mechanisms reviewed in Section 4: adenosine-mediated sleep pressure, caffeine-adenosine receptor dynamics, and metabolic

recovery processes generate signals that Participant A appears to detect with unusual precision.

8.3 Developmental trajectory: perceptual calibration

A striking feature of Participant A's case is the developmental trajectory through which his brain-state discrimination appears to have been established. At approximately age twelve, he participated as a subject in an EEG research study in which he received real-time feedback about his own brain-wave states. Through this biofeedback experience, he acquired the ability to voluntarily shift between alpha-dominant and beta-dominant states and, more importantly, to recognize the subjective correlates of each state from the inside. This constitutes a documented instance of perceptual calibration: the alignment of internal subjective experience with externally verified physiological measurement, producing a stable internal reference frame for brain-state discrimination.

At approximately age fifteen, Participant A experienced a traumatic brain injury resulting in loss of consciousness and a near-death experience. During recovery, he retrospectively interpreted the subjective phenomena of the experience — light, tunnel-like visual patterns, vivid landscape-like imagery — not as mystical or transcendent, but as the perceptual consequences of disordered neural activity: random firing in the visual cortex producing tunnel-like patterns consistent with the retinal-cortical mapping described by Blackmore and Troscianko (1989), and temporal lobe hyperactivation producing emotionally charged visual imagery consistent with the mechanisms described by Blanke and Dieguez (2009). The critical point is not the accuracy of this interpretation but the cognitive stance it reveals: Participant A treated the subjective experience of a brain-state crisis as data about the brain's functional state, rather than as a revelatory or ineffable event. This stance — the treatment of subjective brain-state experience as informative signal rather than noise — appears to have been enabled by the prior EEG calibration experience, which established the principle that subjective states correspond to identifiable neural states.

8.4 Neural correlate: EEG during cognitive work

During a 42-minute session of demanding cognitive work (simultaneous engagement with two AI systems on philosophical argumentation and self-referential brain-state analysis), Participant A's EEG was recorded using a Muse S headband with data captured via the Mind Monitor application. After quality filtering (HSI ≤ 2 for the primary channel of interest), 2,304 valid data points were retained.

The most salient finding was elevated theta power (1.48 dB relative to baseline) at the TP10 electrode (right temporal), approximately double the theta power recorded at the contralateral TP9 (0.75 dB) and dramatically higher than frontal theta (AF7: 0.00 dB; AF8: –0.03 dB). TP10 also showed the highest gamma power across channels (0.81 dB). Heart rate remained stable throughout (mean 72.4 bpm, range 65.4–89.1), indicating parasympathetic predominance despite the cognitive demand.

The co-elevation of theta and gamma at TP10 is consistent with theta-gamma coupling, a neural mechanism associated with working memory maintenance and cross-frequency information integration (Lisman & Jensen, 2013; Canolty & Knight, 2010). While consumer-grade EEG lacks

the resolution to confirm phase-amplitude coupling directly, the sustained co-elevation across a 42-minute session is notable: it suggests a tonic rather than phasic pattern of theta-gamma co-activation during sustained cognitive work. Whether this pattern is related to the sustained brain interoceptive monitoring that Participant A reports is an open empirical question that would require investigation with research-grade equipment and formal experimental protocols. The present observation is offered as an empirical anchor for future investigation, not as evidence for a specific neural mechanism.

8.5 Summary

Participant A illustrates the high-precision end of the brain interoception continuum. He detects variations in his own cognitive state with sufficient resolution to guide task selection; he tracks modulators (sleep, caffeine, recovery) as state variables; and his developmental history includes a documented calibration event (EEG biofeedback at age twelve) that appears to have established a stable internal reference frame for brain-state discrimination. His case is consistent with the continuum model but does not, by itself, establish it. The continuum model was derived from the evidence reviewed in Sections 2 through 7; Participant A's case demonstrates that the model's predictions are realizable in at least one individual.

Implications and Predictions

The brain interoception continuum, if accepted, has implications for clinical practice, research methodology, and theoretical integration. It also generates testable predictions that distinguish it from the status quo, in which brain-state sensing is distributed across unconnected literatures.

9.1 Clinical implications

Reclassifying brain fog, mental fatigue, and subjective cognitive complaints as brain interoception — rather than as vague subjective states or psychiatric symptoms — opens new assessment and intervention pathways. Brain fog in long COVID, chemotherapy-related cognitive impairment, and post-concussion syndrome are currently characterized primarily by the mismatch between patient complaints and objective neuropsychological test performance. If brain fog is understood as a brain interoceptive signal — analogous to the discomfort of air hunger or the heaviness of fatigued muscles — then the patient's report gains informational status. It becomes a signal to be characterized (What brain-state variable is being detected? Through which pathway?) rather than a complaint to be validated or dismissed.

The migraine prodrome offers an existing clinical model. Patients who learn to recognize prodromal signals and intervene early (e.g., with triptans or behavioral modifications) achieve better outcomes than those who wait for the headache phase (Schwedt et al., 2025). This is, in effect, a clinical application of brain interoception: the patient detects a brain-state change and acts on the information. Extending this logic, training patients to detect and act on subtler brain-state signals — fatigue onset, concentration decline, post-exertional cognitive malaise — could become a component of cognitive rehabilitation.

9.2 Research implications: toward a brain interoception task

The cardiac interoception literature advanced rapidly once Schandry (1981) introduced the heartbeat counting task — a simple, standardized behavioral measure of interoceptive accuracy. No equivalent task exists for brain interoception. The development of such a task is a priority implied by the continuum model.

Candidate tasks include: blood pressure estimation (the participant estimates systolic blood pressure; this is verified against measurement), cognitive load detection (the participant rates current cognitive load during tasks of known difficulty; the rating is compared against objective load parameters), caffeine state discrimination (the participant identifies whether they are in a caffeinated, baseline, or withdrawal state under blinded conditions), and sleep-debt estimation (the participant estimates cumulative sleep debt; this is compared against actigraphic data). Each of these tasks probes a different aspect of brain-state sensing, reflecting the likely organ-specificity of interoceptive accuracy (Banellis et al., 2026). A multitask battery, analogous to the multisensory interoception batteries now being developed for body interoception (Murphy et al., 2020), would enable the characterization of individual brain interoceptive profiles.

9.3 Theoretical implications

The continuum model connects three literatures that currently operate in isolation. The metacognition literature (Flavell, 1979; Efklides, 2006, 2011; Fleming, 2024) studies the brain's monitoring of its own cognitive processes. The interoception literature (Craig, 2002; Garfinkel et al., 2015; Seth, 2013) studies the brain's monitoring of the body's physiological state. The fatigue literature (Müller & Apps, 2019; Boksem & Tops, 2008; McMorris et al., 2018) studies subjective energy and effort allocation. Brain interoception sits at their intersection: the brain's monitoring of its own physiological state. By naming this intersection, the continuum model enables cross-fertilization. Metacognitive feelings (feeling of difficulty, feeling of fluency) may be partly constituted by brain interoceptive signals. Interoceptive prediction errors may contribute to cognitive performance monitoring. Fatigue may be reframed as the brain's interoceptive detection of its own resource depletion.

9.4 Testable predictions

The continuum model generates predictions that distinguish it from the current framework in which brain-state sensing is either ignored or treated as unreliable introspection:

1. **Organ-specificity prediction**: Brain interoceptive accuracy should show some but not total correlation with cardiac interoceptive accuracy, consistent with the finding that interoceptive accuracy is partially organ-specific (Banellis et al., 2026). High heartbeat detectors should not automatically be high brain-state detectors, and vice versa.

2. **Migraine-generalization prediction**: Patients with high migraine prodrome detection accuracy should show above-average performance on other brain interoceptive tasks (e.g., caffeine state discrimination, cognitive load detection), reflecting a general brain interoceptive sensitivity rather than a migraine-specific skill.

3. **Meditation-dissociation prediction**: Meditation training should improve brain interoceptive sensibility (self-reported awareness of cognitive state variations) without necessarily improving brain interoceptive accuracy (objective performance on brain-state detection tasks), paralleling the cardiac meditation paradox (Khalsa et al., 2008).

4. **Sleep-deprivation-dissociation prediction**: Chronic sleep restriction should produce a characteristic pattern of preserved brain interoceptive detection (the person senses that something is different) but impaired brain interoceptive calibration (the person underestimates the magnitude of cognitive decline), consistent with Van Dongen et al. (2003) reinterpreted within the detection-calibration framework.

5. **Developmental-calibration prediction**: Individuals with prior EEG neurofeedback experience — in which brain-state measurements are fed back to the individual in real time, enabling the calibration of internal subjective states against externally verified neural activity — should show enhanced brain interoceptive accuracy compared to matched controls. This is distinct from biofeedback modalities that calibrate non-brain signals (e.g., HRV biofeedback calibrates cardiac interoception, not brain interoception). The perceptual calibration mechanism illustrated by Participant A involves specifically brain-to-brain calibration: learning the subjective correlates of one's own neural states through external measurement.

6. **Dual-component dissociation prediction**: If feeling of difficulty contains both a metacognitive component (task demand) and a brain interoceptive component (tissue state), then these should be experimentally separable. Participants performing tasks of varying difficulty under varying brain-state conditions (e.g., rested vs. sleep-deprived, caffeinated vs. withdrawal) should show independent main effects of task difficulty and brain state on subjective difficulty ratings. Individuals with high brain interoceptive precision should show a larger brain-state main effect and should be able to report task difficulty and brain-state quality as independent dimensions.

Conclusion

The brain is the body's most metabolically demanding organ, consuming roughly twenty percent of the body's energy at two percent of its mass. It has vasculature, neurochemistry, immunological activity, and a tissue environment that fluctuates with activity, fatigue, disease, and recovery. It possesses mechanisms — central chemoreceptors, glucosensing neurons, circumventricular organs, astrocytic adenosine signaling, microglial surveillance, meningeal interoceptors — through which it monitors its own tissue state. These mechanisms generate signals that, under a range of conditions, reach conscious awareness: as headache, as aura, as sleepiness, as brain fog, as mental fatigue, as the everyday sense that thinking is clear or cloudy.

And yet, when interoception scientists catalogue the organs whose internal states the body senses, the brain is not on the list.

This review has argued that the omission is neither justified by evidence nor defended by argument. It is an inheritance — from Sherrington's original partition between interoceptive and

exteroceptive, through Craig's reformulation of interoception as the sense of the body's physiological condition, to the NIH roadmaps that enumerate every organ system except the one doing the enumerating. The brain was placed on the "mind" side of the Cartesian partition and thereby made invisible as a body organ whose state could be interoceptively sensed.

The remedy is not a new theory but a reclassification. The evidence already exists: it is scattered across headache neuroscience, respiratory physiology, sleep research, migraine theory, epilepsy, metabolic neuroscience, and glial biology. What has been missing is the name. Brain interoception — the sensing of the brain's own tissue state — is a coherent category, a single continuum, and a phenomenon that the field of interoception is now equipped to study. The muscle analogy makes the concept intuitive, the mechanistic evidence makes it plausible, the clinical literature makes it undeniable, and the predictive processing framework makes it theoretically tractable. What remains is the empirical work: the development of standardized tasks for measuring brain interoceptive accuracy, the characterization of individual differences, and the investigation of clinical populations in whom brain interoception may be impaired or enhanced. The brain has been hiding in plain sight. It is time to put it on the map.

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