

The GABAergic system in motion sickness: a narrative review from the vestibular molecular brake to clinical evidence

O sistema GABAérgico na fisiopatologia e no tratamento da cinetose: uma revisão narrativa do freio molecular vestibular à evidência clínica

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ABSTRACT

Background: Motion sickness is a prevalent and temporarily incapacitating condition, traditionally managed with anticholinergics and antihistamines that act downstream in the emetic cascade. The GABAergic system — the main inhibitory pathway of the central nervous system — has received growing attention as a rational therapeutic target but remains underrepresented in reviews of the field.

Objective: To critically review the neurophysiological and pharmacological basis underlying the role of gamma-aminobutyric acid (GABA) in the pathophysiology and treatment of motion sickness.

Methods: Narrative review of the literature, synthesizing evidence from vestibular neurophysiology, clinical pharmacology, and controlled trials.

Results: The brainstem velocity storage mechanism, indexed by the time constant of the angular vestibulo-ocular reflex (TVOR), correlates directly with motion sickness susceptibility and is modulated by GABA-B receptors on vestibular-only neurons. Agents that enhance GABAergic tone — from baclofen, as mechanistic proof of concept, to benzodiazepines — raise the motion sickness threshold. Recent randomized clinical evidence demonstrated that a combination of GABA and neuroactive cofactors was superior to ginger extract in the management of chronic complaints consistent with motion sickness.

Conclusion: The GABAergic system is a mechanistically grounded and clinically promising therapeutic target for motion sickness, supporting the development of next-generation modulators and combination strategies.

Keywords: Motion Sickness; gamma-Aminobutyric Acid; Vestibular Nuclei; Reflex, Vestibulo-Ocular; Cerebellum.

RESUMO

Introdução: A cinetose é uma condição prevalente e temporariamente incapacitante, tradicionalmente manejada com anticolinérgicos e anti-histamínicos que atuam a jusante na cascata emética. O sistema GABAérgico — principal via inibitória do sistema nervoso central — tem recebido atenção crescente como alvo terapêutico racional, mas permanece sub-representado nas revisões da área.

Objetivo: Revisar criticamente as bases neurofisiológicas e farmacológicas que fundamentam o papel do ácido gama-aminobutírico (GABA) na fisiopatologia e no tratamento da cinetose.

Métodos: Revisão narrativa da literatura, com síntese de evidências de neurofisiologia vestibular, farmacologia clínica e ensaios controlados.

Resultados: O mecanismo de armazenamento de velocidade do tronco cerebral, indexado pela constante de tempo do reflexo vestibulo-ocular angular (TVOR), correlaciona-se diretamente com a susceptibilidade à cinetose e é modulado por receptores GABA-B em neurônios vestibulares. Agentes que reforçam o tônus GABAérgico — do baclofeno, como prova de conceito mecanicista, aos benzodiazepínicos — elevam o limiar de cinetose. Evidência clínica randomizada recente demonstrou que uma combinação de GABA e cofatores neuroativos foi superior ao extrato de gengibre no manejo de queixas crônicas compatíveis com cinetose.

Conclusão: O sistema GABAérgico constitui um alvo terapêutico mecanicamente fundamentado e clinicamente promissor para a cinetose, justificando o desenvolvimento de moduladores de nova geração e de estratégias combinadas.

Palavras-chave: Enjoo devido ao Movimento; Ácido gama-Aminobutírico; Núcleos Vestibulares; Reflexo Vestibulo-Ocular; Cerebelo.

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Conflict of Interest Statement

The corresponding author (H.T.R.) is an employee of Laboratório Gross S.A., the manufacturer of the GABA and cofactor formulation (Organoneuro Cerebral®) discussed in this review, and is a co-author of the clinical trial by Nunes et al. (2023) cited herein. Laboratório Gross S.A. funded that clinical study and is its direct commercial beneficiary. The remaining authors (M.V.N.S. and W.B.G.B.), affiliated with Farmanguinhos/Fiocruz, declare no competing interests. This review presents a critical appraisal of the published literature; the clinical results referenced were independently peer-reviewed prior to publication.

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Author Contributions (CRediT)

H.T.R.: Conceptualization, Investigation, Writing – original draft. M.V.N.S.: Supervision, Writing – review & editing, Methodology. W.B.G.B.: Supervision, Writing – review & editing. All authors approved the final version and agree to be accountable for all aspects of the work.

Declaration on the Use of Artificial Intelligence

In accordance with the journal's editorial policy, the authors declare that an AI-assisted writing tool was used during the preparation of this manuscript for language structuring, academic writing support, and formatting. All scientific content, reference selection and verification, and final validation were performed entirely by the authors, who take full responsibility for the accuracy and integrity of the manuscript.

INTRODUCTION

Motion sickness, or kinetosis, is among the most prevalent yet most neglected conditions in clinical practice. An estimated 25% to 40% of the population develops symptoms under moderate exposure conditions, with even higher proportions under intense provocation, such as sea travel, flights in turbulence, or the use of virtual reality environments.¹ Although usually benign and self-limiting, motion sickness is a source of considerable distress for individuals with chronic complaints and can impair the occupational performance of sailors, pilots, drivers, and habitual travelers.²

The pathophysiology of motion sickness rests on the sensory conflict theory: the mismatch among vestibular, visual, and proprioceptive information — or between current sensory experience and the central nervous system's predictive internal model — triggers an autonomic cascade that culminates in nausea and vomiting.^{1,3} From this understanding, a pharmacological armamentarium developed over decades has come to be dominated by two classes: anticholinergics, represented by scopolamine, and first-generation antihistamines. Both classes act by blocking excitatory signaling downstream in the emetic cascade.⁴

Less widely discussed, yet mechanistically closer to the origin of the phenomenon, is the approach that seeks to reinforce central nervous system inhibition through the GABAergic system. Gamma-aminobutyric acid (GABA) is the main inhibitory neurotransmitter of the mammalian brain, and evidence accumulated over the past three decades indicates that GABAergic modulation of the vestibular nuclei and cerebellum raises the motion sickness threshold.^{5,6} Nevertheless, GABA is usually treated only in passing in reviews of the field, which favor the classical agents. This narrative review proposes to invert that axis: to place the GABAergic system as the guiding thread, from molecular mechanism to clinical evidence, critically reviewing why — and on what grounds — the inhibitory pathway constitutes a rational therapeutic target for motion sickness.

NEUROPHYSIOLOGICAL BASIS OF MOTION SICKNESS

The perception of self-motion results from the continuous integration of three sources of information: the vestibular system of the inner ear, which detects linear and angular accelerations through the semicircular canals and otolith organs (saccul and utricle); the visual system, which provides optic flow; and the somatosensory and proprioceptive system, which reports body position and contact.^{1,3} These inputs converge on the vestibular nuclei of the brainstem and on the cerebellum, where they are

compared with a prediction generated by an internal model. When sensory experience diverges from that prediction — as occurs when reading inside a moving vehicle, a situation in which the vestibular system signals displacement while the visual system signals stillness — the sensory conflict that characterizes motion sickness ensues.³

To maintain gaze stability and the integration of head and eye movements, the central nervous system relies on the so-called velocity storage mechanism, a compensatory system located in the vestibular nuclei that prolongs and integrates slow-phase eye velocity signals.⁶ This mechanism sets the time constant of the angular vestibulo-ocular reflex (TVOR). Studies by Cohen, Raphan, Dai, and colleagues demonstrated that the TVOR is directly correlated with motion sickness susceptibility: when velocity storage is excessively active and prolonged, the conflict signal is amplified over time, increasing the probability of triggering symptoms.^{6,7} This finding carries a direct therapeutic implication — shortening the velocity storage time constant should decrease motion sickness susceptibility.

From the vestibular nuclei, the conflict signal, when sufficiently intense, projects to the so-called vomiting center — comprising the nucleus tractus solitarius (NTS) and the medullary reticular formation — and, through connections with the hypothalamus and the chemoreceptor trigger zone, elicits the autonomic manifestations of motion sickness: salivation, cold sweating, pallor, nausea, and vomiting.^{2,4} The conscious experience of nausea further involves insular and anterior cingulate processing. It is within this circuit — the vestibular nuclei, the cerebellum, and their brainstem connections — that GABAergic modulation exerts its effect (Figure 1).

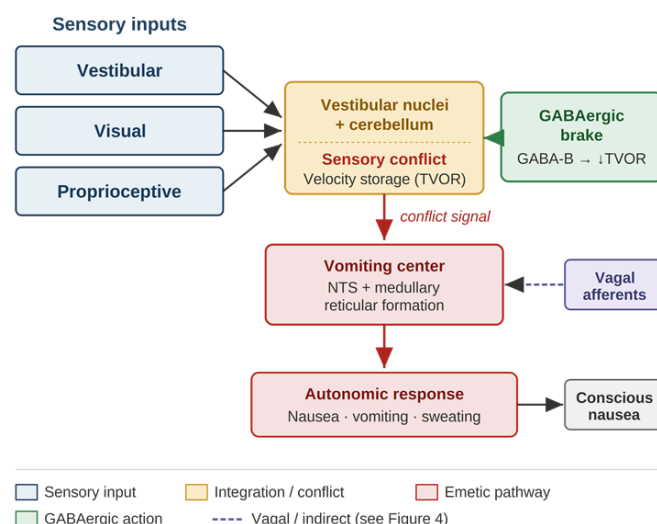


Figure 1. Neurophysiological circuit of motion sickness, showing sensory convergence, the velocity storage mechanism, the site of GABAergic inhibition, and the downstream emetic pathway.

THE GABAERGIC SYSTEM AS A VESTIBULAR MOLECULAR BRAKE

GABA is the main inhibitory neurotransmitter of the mammalian central nervous system, synthesized from glutamate by the enzyme glutamic acid decarboxylase (GAD), which depends on pyridoxal-5-phosphate — the active form of vitamin B6 — as an essential cofactor.⁸ Its action is mediated by two receptor types: GABA-A receptors, pentameric ion channels permeable to chloride that produce rapid inhibition through hyperpolarization and shunting; and GABA-B receptors, metabotropic and G-protein-coupled, which mediate slow and prolonged inhibition (Figure 2).^{8,9}

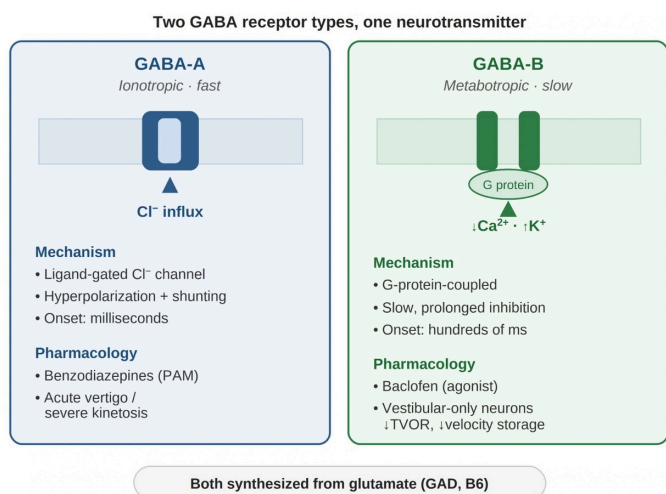


Figure 2. Contrast between the ionotropic GABA-A receptor (fast inhibition; benzodiazepine target) and the metabotropic GABA-B receptor (slow inhibition; baclofen target).

The inhibitory anatomy of the motion sickness circuit is remarkably GABAergic. The Purkinje cells of the cerebellum — whose output is purely inhibitory to the deep cerebellar and vestibular nuclei — are quintessential GABAergic neurons, and the vestibular nuclei of the brainstem receive dense GABAergic inhibition, both from cerebellar sources and from local interneurons.⁵ Holstein and colleagues demonstrated, by immunocytochemistry, the presence of baclofen-sensitive GABA-B binding sites in the medial vestibular nucleus.¹⁰

The decisive mechanistic link between GABA and motion sickness was established by the localization of GABA-B receptors on specific vestibular neurons (vestibular-only neurons), whose axons constitute the substrate of the velocity storage mechanism.^{6,7} Stimulation of these GABA-B receptors reduces the TVOR and the velocity storage time. This yields a clear and testable prediction: any intervention that increases cerebellar and vestibular GABAergic inhibitory tone should raise the motion sickness threshold — that is, make the individual more resistant to sickness.^{5,6} Also noteworthy is the functional complementarity between GABA and histamine in the vestibular nuclei: histamine, via

H1 receptors, excites vestibular neurons and amplifies the gain of the response to conflict, whereas GABA inhibits them. The balance between these two systems determines the resting gain of the vestibular nuclei, which explains why both histaminergic blockade and GABAergic reinforcement converge on the same functional effect — reduction of vestibular excitability — through distinct molecular mechanisms.⁴

FROM THE MECHANISTIC HYPOTHESIS TO GABAERGIC PHARMACOLOGY

The pharmacological agents relevant to motion sickness can be mapped onto the emetic cascade according to where they act: GABAergic agents upstream, near the origin of the conflict signal, and the classical agents downstream, along the emetic pathway (Figure 3).

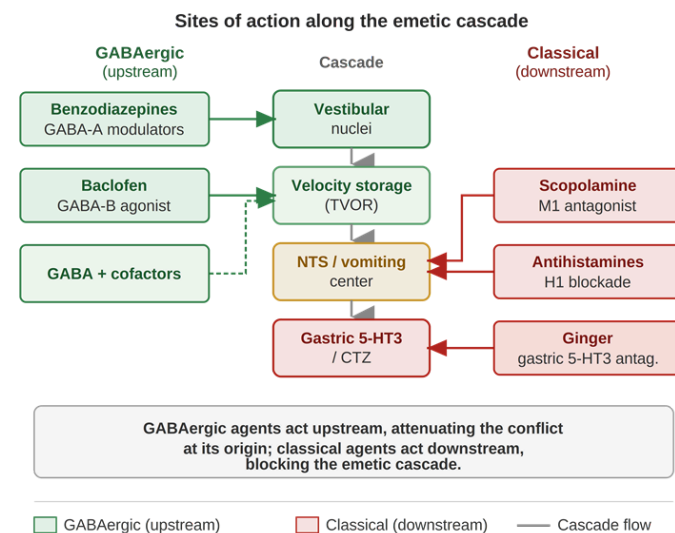


Figure 3. Sites of pharmacological action along the emetic cascade: GABAergic agents act upstream, near the origin of the conflict, whereas classical agents act downstream.

Baclofen — the proof of concept. Baclofen, a specific GABA-B receptor agonist used clinically as a muscle relaxant for spasticity, holds a singular position in the pharmacology of motion sickness. It was the agent that most directly demonstrated, in experimental models, the GABAergic mechanism of vestibular conflict suppression: Cohen and colleagues showed that baclofen reduces the TVOR and velocity storage time in primates through action on GABA-B receptors on vestibular neurons, experimentally decreasing motion sickness susceptibility.^{6,7} In clinical studies with volunteers, oral baclofen reduced motion sickness in standardized stimulation paradigms, with efficacy comparable to that of antihistamines, but with sedation and orthostatic hypotension as limiting effects.⁵ Its routine use is not established; its value is that of an irreplaceable proof of concept that the GABAergic pathway is pharmacologically accessible and clinically effective for motion sickness.

Benzodiazepines. Benzodiazepines — diazepam, clonazepam, lorazepam — act as positive allosteric modulators of GABA-A receptors, increasing the frequency of chloride channel opening in response to endogenous GABA and potentiating GABAergic inhibition in the vestibular nuclei and cerebellum.⁹ They have an established use in acute vertigo and in severe episodes of incapacitating motion sickness, providing rapid relief. Notably, sub-sedative doses also reduce motion sickness, suggesting that the antiemetic effect is at least partly independent of sedation and mediated by vestibular GABAergic potentiation.⁵ The main limitation for prophylactic use is the risk of dependence, which is incompatible with regular use.

GABA and neuroactive cofactors. The oral administration of GABA, accompanied by the metabolic cofactors of its synthesis and function, represents the most recent frontier of the GABAergic approach. Its logic differs fundamentally from that of the classical agents: rather than blocking excitatory signaling downstream, it seeks to favor the tone of the inhibitory system further upstream in the cascade, attenuating the conflict signal before it fully triggers the emetic response in the NTS.⁵ The rationale for combining GABA with cofactors — rather than administering it alone — stems from the recognition that the efficacy of the inhibitory system depends on the entire metabolic chain that sustains it: glutamate as substrate, pyridoxine (B6) as cofactor of glutamic acid decarboxylase, thiamine (B1) in neuronal energy support, cyanocobalamin (B12) in axonal and myelin integrity, and calcium in the modulation of neurotransmitter release through voltage-dependent channels and calmodulin.^{8,11,12} As discussed below, this composition also has direct implications for the question of GABA penetration into the central nervous system.

Non-pharmacological interventions with a GABAergic basis. The same mechanistic rationale illuminates non-pharmacological interventions: vagal stimulation and slow, controlled breathing potentiate parasympathetic and GABAergic tone in the brainstem, and contemplative practices such as yoga raise measurable brain GABA concentrations by magnetic resonance spectroscopy, as demonstrated by Streeter and colleagues.¹³ The connection between these practices, GABA, and motion sickness remains one of the most promising and least explored areas of current research.

CLINICAL EVIDENCE: THE GABA + COFACTOR COMBINATION VERSUS GINGER

The hypothesis that oral supplementation of GABA and cofactors could produce a clinically measurable anti-motion-sickness effect was tested in a randomized, double-blind, self-paired clinical trial published by Nunes and

colleagues in 2023, which constitutes the most robust clinical evidence available for this approach.¹⁴ For the sake of transparency, it is declared that one of the authors of the present review (H.T.R.) is among the authors of that trial and is affiliated with the manufacturer of the study formulation.

The study compared a combination of GABA tartrate (100 mg), glutamic acid (100 mg), dibasic calcium phosphate (50 mg), thiamine nitrate (25 mg), pyridoxine hydrochloride (10 mg), and cyanocobalamin (5 µg) (Group A, n = 170) versus *Zingiber officinale* (ginger) extract 160 mg, corresponding to 8 mg of gingerols (Group B, n = 165), in 335 patients with chronic motion sickness in the state of Rio de Janeiro, between August 2021 and September 2022. The study was approved by an ethics committee and registered at ClinicalTrials.gov (NCT05221892). Efficacy was measured with the Motion Sickness Assessment Questionnaire (MSAQ), a validated 16-item instrument assessing gastrointestinal, central nervous system, peripheral, and sopite-related manifestations, with scores ranging from 16 (no symptoms) to 144 points.^{14,15}

For the primary endpoint — the proportion of patients with a reduction of 20 or more points in the total MSAQ score across trips 2 to 4 relative to the initial unmedicated trip — Group A was significantly superior at every trip: 54.1% versus 43.6% at trip 2; 62.3% versus 50.9% at trip 3; and 66.4% versus 55.1% at trip 4. The mean absolute score per patient per trip was 31.3 points in Group A versus 36.2 in Group B, a statistically significant difference representing a substantially lower symptom burden with the GABAergic combination.¹⁴

Subjective assessments corroborated the objective data: the investigating physician's assessment scores and patients' willingness to continue treatment (8.2 versus 6.3 on a 10-point scale) were significantly higher in Group A.¹⁴ The tolerability profiles reflected the distinct mechanisms of action. Group A presented 45 adverse events — predominantly mild neurological ones, such as drowsiness, consistent with central action — whereas Group B presented 77 events, predominantly gastrointestinal, such as bloating, flatulence, and a burning sensation on the tongue, consistent with the peripheral action of ginger. No serious adverse events and no withdrawals for safety reasons occurred in either group.¹⁴

The blood-brain barrier question and the plausible mechanism

A mechanistic objection must be addressed explicitly. The entire circuit reviewed in the preceding sections is central — GABA-B receptors on vestibular-only neurons, cerebellar Purkinje cells, velocity storage in the brainstem. However, orally administered GABA is classically considered to cross the blood-brain barrier (BBB) poorly, being a polar molecule that is charged at physiological pH.^{16,17} This poses an apparent paradox: how would an oral

intervention produce a clinical effect on a central target if its eponymous component does not reach the brain parenchyma in relevant amounts? The same principle — that only agents with central penetration are effective in motion sickness — is illustrated by the ineffectiveness of second-generation antihistamines, which were designed precisely not to cross the BBB.⁴

Resolving this paradox does not weaken the GABAergic hypothesis; on the contrary, it refines it. The observed efficacy does not require that ingested GABA cross the BBB, and it is better explained by two convergent axes. The first is the metabolic support of endogenous GABA synthesis within the central nervous system itself: unlike GABA, its synthesis cofactors — pyridoxine, thiamine, and the precursor glutamate — do cross the BBB and sustain glutamic acid decarboxylase activity *in situ*, which explains why the effective formulation is not GABA alone but GABA combined with cofactors.^{8,16} The second axis is gut-to-brainstem signaling through the vagus nerve: luminal and circulating GABA can influence the central nervous system by activating the enteric nervous system and vagal afferents, a mechanism whose dependence on vagal integrity has been demonstrated in animal models and which, in humans, is associated with elevated cerebrospinal-fluid GABA after vagal stimulation.^{16,18} This axis is particularly relevant to motion sickness because vagal afferents converge precisely on the NTS — the same nucleus in which vestibular conflict triggers the emetic cascade — so that vagal modulation of the NTS offers an anatomically coherent route to attenuate the emetic response without requiring trans-barrier passage of the neurotransmitter.^{2,4} This is, admittedly, a mechanism not yet definitively demonstrated for the motion sickness endpoint, and its experimental confirmation — for example, through vagal-dependence studies or cerebellar GABA spectroscopy — remains an open research agenda (Figure 4).

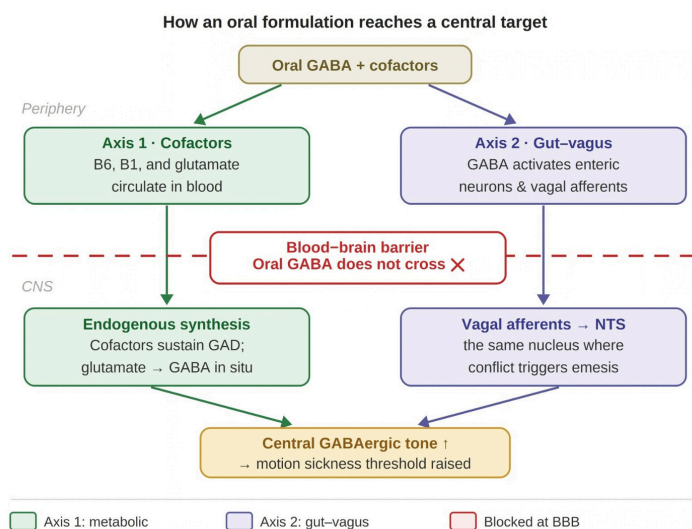


Figure 4. Proposed mechanism by which the oral formulation acts centrally without GABA crossing the blood-brain barrier, via cofactor-supported endogenous synthesis and vagal gut-to-brainstem signaling.

The trial finding is consistent with the entire chain of evidence reviewed: the superiority of an intervention that favors GABAergic inhibition over an agent whose principal action — the antagonism of gastric 5-HT₃ receptors by ginger — occurs peripherally, downstream of the origin of the conflict.^{4,14} It should be noted as a limitation that the comparator was ginger, not an antihistamine or scopolamine, which are more potent reference standards; future trials confronting the GABAergic combination with those classes, or testing their combined use, will be needed to position it definitively within the therapeutic armamentarium.

FRONTIERS AND PERSPECTIVES

Next-generation GABAergic modulators. The demonstration that GABA-B receptors on vestibular-only neurons and cerebellar Purkinje cells constitute valid and effective targets has opened the way for the development of second-generation GABA-B agonists with greater subtype selectivity and less sedation than baclofen. Compounds with selectivity for predominantly presynaptic subtypes could, in theory, suppress vestibular hyperactivity with a better tolerability profile — one of the most rationally grounded lines in the pharmacology of motion sickness.⁶

Susceptibility biomarkers. Objective susceptibility candidates converge with the GABAergic hypothesis. Resting heart rate variability, a marker of vagal tone, correlates negatively with susceptibility; the TVOR time constant, directly linked to velocity storage, is a plausible mechanistic predictor; and magnetic resonance spectroscopy of cerebellar GABA offers the most direct marker of local inhibitory tone, albeit at high cost.^{1,6,13} Their validation could underpin risk stratification and personalized prophylaxis.

Motion sickness in autonomous vehicles and virtual reality. The spread of autonomous vehicles has created an emerging motion sickness problem: by removing the act of driving, the passenger loses the anticipatory visual cues that once reduced the visual-vestibular mismatch, with prevalence estimates two to three times higher than in conventional vehicles.¹ Similarly, virtual and augmented reality environments have become both a cause and an experimental model of motion sickness. These contexts amplify the relevance of interventions acting on the central threshold — among them the GABAergic ones — and reinforce the demand for transdisciplinary knowledge connecting engineering, vestibular neuroscience, and pharmacology.

CONCLUSION

The review of the neurophysiological, pharmacological, and clinical literature converges on a coherent thesis: the GABAergic system is a mechanistically grounded therapeutic target, not merely an adjuvant one, in the management of motion sickness. The chain of evidence begins with the correlation between the velocity storage mechanism and motion sickness susceptibility, proceeds through the demonstration that GABA-B receptors on vestibular-only neurons modulate this pathway, finds proof of concept in baclofen and the benzodiazepines, and culminates in randomized clinical evidence that a combination of GABA and cofactors outperforms a traditional comparator. Unlike the classical agents, which block signaling downstream, the GABAergic approach acts upstream, on the origin of the conflict signal. Explicit recognition of this axis — currently underrepresented in reviews of the field — justifies both the development of next-generation GABAergic modulators and the design of trials testing combination strategies, pointing toward a more rational and mechanistically oriented pharmacology of motion sickness.

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