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Thought Suppression as a Psychological Amplifier of Motion Sickness: A Contextual Literature Review

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Abstract: This literature review examines whether thought suppression may intensify motion sickness and visually induced motion sickness. The central claim is deliberately cautious. The recent literature does not yet provide a large body of direct experiments showing that suppressing thoughts about nausea reliably worsens motion sickness. However, several adjacent evidence streams make the claim theoretically plausible and empirically testable. Contemporary motion sickness research defi nes motion sickness as a physiological response to provocative physical or visual motion that includes nausea, dizziness, thermoregulatory disruption, altered arousal, headache, and ocular strain. Recent work also emphasizes individual susceptibility, cognitive load, interoceptive processing, anxiety, expectation, autonomic regulation, and gut-brain communication. Contemporary thought-suppression research has also become more nuanced. Suppression does not always cause rebound, but suppression can become maladaptive when it creates continuous monitoring, high cognitive demand, threat appraisal, and rigid avoidance of internal experience. This review integrates these strands to propose a contextual model. Thought suppression may worsen motion sickness when it increases monitoring of nausea-related sensations, competes with limited cognitive resources, strengthens negative expectancy, and amplifies autonomic or gastrointestinal feedback loops. The review concludes that future research should test suppression, acceptance, distraction, and reappraisal instructions during motion-sickness exposure using symptom ratings, physiological measures, eye tracking, and longitudinal habituation designs.

Keywords: Thought Suppression; Motion Sickness; Visually Induced Motion Sickness; Interoception; Cognitive Load; Nausea

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1. Introduction

Motion sickness is an old problem, now dressed in new technology. People felt ill on ships long before they got queasy in VR headsets, but the glossy interface does not make the underlying issue go away. It remains a serious obstacle—for travel comfort, maritime and aviation performance, automated-vehicle adoption, immersive entertainment, clinical simulation, and virtual training. Recent diagnostic work treats motion sickness and visually induced motion sickness as normal physiological responses, though they can reach disorder-level severity when recurrence, avoidance, or functional impairment become substantial (Cha et al., 2021). That distinction matters. This is not a weak stomach or a failure of will. It is a complex body-brain response involving sensory mismatch, autonomic regulation, cognitive appraisal, and a cluster of symptoms: nausea,

dizziness, sweating, fatigue, headache, and ocular discomfort.

This review narrows the lens to one theoretically sharp question: can thought suppression intensify motion sickness? A simple answer is tempting. A person notices nausea, tries hard not to think about it, monitors whether the thought has vanished, notices the nausea again, grows more distressed, and feels sicker. Psychologically plausible—but perhaps too tidy. The current literature does not contain a large, direct, and settled experimental base showing that thought suppression reliably worsens motion sickness across contexts. So a careful review should not pretend that the field has already settled the matter. Stretching evidence does not improve academic writing.

A more demanding position seems warranted. Thought suppression may worsen motion sickness under specific conditions: when the suppression target is nausea-related, when the individual is already anxious or highly susceptible, when the motion stimulus creates strong sensory conflict, when spare cognitive resources are scarce, and when suppression turns into continuous self-monitoring. This claim aligns with recent motion-sickness research, which increasingly emphasises individual susceptibility, physiological markers, cognitive load, visual devices, and gut-brain pathways (Keshavarz & Golding, 2022; Park et al., 2022; Talsma & de Winkel, 2025). It also fits contemporary thought-suppression work, which has moved beyond the crude notion that suppression always produces rebound. Recent findings indicate that suppression outcomes vary with training, intention, control demands, target material, and whether the process becomes defensive monitoring rather than flexible regulation (Mamat & Anderson, 2023; Mamat et al., 2024).

Treating thought suppression as an amplifier rather than a universal cause matters. An amplifier does not create nausea from nothing. It acts on an existing signal—a mild wave of dizziness or stomach discomfort. The person tries to force that sensation out of awareness. That effort consumes attention, increases self-monitoring, and reduces the capacity to adapt to the motion environment. The result can be a vicious loop: the body signals distress, the mind attempts to suppress it, monitoring keeps the signal active, and the body responds with heightened arousal. Human design, it seems, rarely comes with a user manual.

The issue has practical teeth. Existing countermeasures range from medication and habituation to environmental adjustments, music, airflow, and other behavioural techniques (Keshavarz & Golding, 2022; Rahimzadeh et al., 2023). Yet people often rely on private mental strategies during an episode. They tell themselves not to think about vomiting, avoid looking at the horizon, scan their stomach, repeatedly check whether they are worsening, or catastrophise about public embarrassment in a car, classroom, clinic, ship, or VR lab. These strategies could alter symptom trajectories, but standard experiments rarely measure them precisely. Given how much of motion sickness is subjective experience, that gap is almost absurd. Researchers meticulously record head movement, heart rate, EEG, and questionnaire scores, while the participant's active attempt to manage awareness often goes unexamined.

This review has five concrete aims. First, clarify the core constructs: motion sickness, visually induced motion sickness, cybersickness, and thought suppression. Second, summarise recent work on diagnosis, individual susceptibility, physiological monitoring, and behavioural countermeasures. Third, connect thought-suppression research to motion-sickness mechanisms. Fourth, highlight methodological controversies and unresolved challenges. Fifth, outline an agenda for direct experimental tests of whether suppressing nausea-related thoughts worsens symptoms during motion exposure. I adopt a narrative, integrative format over a systematic one. The direct literature linking suppression to motion sickness is too sparse for a credible meta-analysis. That limitation is not a problem if stated openly. It becomes a problem only when disguised by unwarranted certainty.

2. Theoretical Background

2.1 Motion Sickness, Visually Induced Motion Sickness, and Cybersickness

Motion sickness refers to a cluster of symptoms that emerge during or after exposure to provocative physical motion. Visually induced motion sickness refers to similar symptoms caused primarily by visual motion cues, such as immersive displays, moving visual scenes, simulators, and virtual reality. Cybersickness is often used for sickness symptoms caused by virtual reality and related immersive media. These concepts overlap, but they are not interchangeable. The Bárány Society consensus criteria distinguish motion sickness, visually induced motion sickness, motion sickness disorder, and visually induced motion

sickness disorder in order to improve diagnostic clarity (Cha et al., 2021). This diagnostic work is important because the same word, sickness, can hide different triggering environments, symptom profiles, and measurement problems.

Recent reviews describe motion sickness as a multi-system response that includes nausea, dizziness, cold sweating, pallor, fatigue, drowsiness, headache, ocular strain, and changes in arousal (Cha et al., 2021; Keshavarz & Golding, 2022). The symptom cluster is broad because provocative motion does not disturb one isolated channel. Visual, vestibular, proprioceptive, autonomic, gastrointestinal, and cognitive systems all participate. A person in a car may feel nausea when reading because the visual system receives a stable page while the vestibular system receives acceleration cues. A person in virtual reality may see self-motion without matching vestibular motion. A person in a simulator may experience conflict between expected movement, visual flow, and bodily feedback. The brain must interpret this mess in real time, because apparently evolution had other appointments.

The dominant explanatory family remains sensory conflict and related mismatch accounts. These accounts suggest that sickness arises when sensory inputs about motion and orientation conflict with one another or with expected internal models. This does not mean that sensory conflict alone explains everything. Keshavarz and Golding (2022) summarize current concepts and management issues by emphasizing current trends in mechanisms, risk factors, and mitigation. Recent work also highlights individual differences, visual-display factors, autonomic pathways, and behavioral context. Sensory conflict provides a backbone, but it does not explain why two people exposed to the same stimulus can have dramatically different symptom courses. It also does not explain why anxiety, attention, expectation, and coping strategies appear to shape susceptibility and severity.

2.2 Individual Susceptibility and Measurement

Motion sickness emerges during or after exposure to physical motion. Visually induced motion sickness stems from visual cues alone—head-mounted displays, simulators, or large-field moving scenes. Cybersickness is the common term for VR-related discomfort. The three terms overlap but are not interchangeable. The Bárány Society consensus criteria separate motion sickness, visually induced motion sickness, and their “disorder” counterparts (Cha et al., 2021) to improve diagnostic clarity. That distinction is practical: “sickness” can hide very different triggers, measurement approaches, and environmental contexts.

The symptom profile is wide: nausea, dizziness, cold sweat, pallor, fatigue, headache, and eyestrain (Cha et al., 2021; Keshavarz & Golding, 2022). The breadth is unsurprising, because provocative motion never stays within a single channel. Visual, vestibular, proprioceptive, autonomic, and gastrointestinal systems all contribute. Reading in a moving car—visual input is stable, but the vestibular system detects acceleration. In VR, the user sees self-motion without matching vestibular signals. In a simulator, expected movements, visual flow, and bodily feedback can contradict each other. The brain must reconcile these conflicting signals in real time.

Sensory conflict and internal model mismatch remain the leading explanatory framework. Yet the framework is not all-powerful. Keshavarz and Golding (2022) provide a comprehensive review of current mechanisms, risk factors, and mitigation strategies. Still, conflict theory struggles to explain why two individuals exposed to the same stimulus show such divergent symptom courses, nor does it account for how anxiety, attention, expectation, and coping strategies modulate susceptibility and severity.

2.3 Cognitive Load and Limited Processing Capacity

Cognitive load offers a clean bridge between thought suppression and motion sickness. Park et al. (2022) examined HMD-induced visually induced motion sickness using heartbeat-evoked potentials within a cognitive-load framework. Their data suggest that motion sickness relates to information-processing capacity, and that symptom onset coincides with reduced cognitive performance. That matters because suppression is costly. It demands attention, monitoring, and repeated checking—notice the target, block it, verify it is gone. In an environment where the nervous system already wrestles with sensory conflict, that is a poor bargain.

The logic is straightforward. Motion generates conflict. The brain spends resources on interpreting it, updating orientation, maintaining posture, and regulating arousal. Suppression draws on some of those same resources. If the suppressed content

is nausea or dizziness, the person also monitors the very symptoms they intend to ignore. So the outcome is not mere distraction. It is recurrent attention to a threat cue. Hence, not all attentional strategies are equivalent. External distraction may reduce symptom focus; internal suppression may increase symptom checking. The semantic difference is modest, but the functional difference is large.

This distinction also clarifies why some distracting tasks reduce motion sickness while others worsen it or impair performance. Gentle external engagement may lower nausea monitoring. A demanding task that competes with orientation processing may exacerbate symptoms. Suppression can do both at once—divert awareness from nausea while forcing the individual to keep checking whether nausea remains. That internal contradiction is the psychological equivalent of inspecting a fire every ten seconds to see if it is still alight.

2.4 Thought Suppression and Ironic Monitoring

Thought suppression is the deliberate exclusion of unwanted thoughts, images, memories, sensations, or urges from awareness. The simple view that suppression is always harmful no longer holds. Mamat and Anderson (2023) found that training people to suppress fearful thoughts improved mental-health outcomes and did not trigger the classic rebound effect often assumed inevitable. Mamat, Levy, and Bayley (2024) similarly call for a reconsideration of suppression and ironic processing in clinical settings. These findings guard against a blanket condemnation.

But nuance has limits. Suppression may be less harmful when it is trained, brief, controlled, and free of anxious self-monitoring. It may turn harmful when it is untrained, prolonged, desperate, and attached to a feared bodily state. Motion sickness fits the latter profile. The target is not abstract—it is visceral. The body keeps generating nausea, and the motion stimulus keeps provoking it. Suppression thus risks becoming a monitoring loop rather than a discrete cognitive act.

Consider pain. Kreddig, Hasenbring, and Keogh (2022) compared suppression with focused distraction in the context of pain-related attentional biases. They did not find a universal rebound effect; instead, they suggested that suppression can offer short-term benefits under specific conditions. That is relevant because pain and nausea both involve bodily threat signals. Brief suppression may temporarily aid function; rigid, repeated suppression paired with anxious checking may impair it. The motion-sickness case therefore calls for a conditional theory, not a moral verdict against suppression.

2.5 Interoception, Nausea, and Gut-Brain Pathways

Motion sickness is not purely vestibular or visual. It is also a problem of bodily awareness. Nausea is felt in the body, appraised by the mind, and regulated via autonomic and gastrointestinal routes. Talsma and de Winkel (2025) argue that motion-sickness models should incorporate gut-brain communication—afferent anatomical, hormonal, immune, and extracellular pathways. This expands the field beyond a unidirectional account where the brain detects conflict and sends symptoms downward. The body, too, may relay information upward, shaping nausea and subjective experience.

That two-way path matters for suppression. Suppressing nausea is not like suppressing a random image. Nausea is interoceptive; bodily input feeds it continuously. If suppression sharpens attention to internal cues, it may heighten nausea's conscious salience. If it elevates stress arousal, autonomic shifts could amplify gastrointestinal distress. This does not imply a mystical mind-over-stomach effect—only a plausible feedback loop linking attention, appraisal, autonomic state, and visceral sensation.

Rahimzadeh et al. (2023) reviewed nutritional and behavioural countermeasures, including music and breathing. These strategies can modify attention, arousal, and parasympathetic tone. Li et al. (2025) used EEG to examine music's mitigating effect on motion sickness; joyful and soft music reduced symptoms more than other conditions. Those findings do not test suppression directly. They do confirm that emotional and attentional states influence motion-sickness outcomes. If music and breathing shift these trajectories, suppression might as well—possibly in the opposite direction when conditions are maladaptive.

3. Current Research

3.1 Recent Diagnostic and Conceptual Advances

Cha et al. (2021) sharpened the diagnostic picture. They offered criteria for motion sickness, visually induced motion sickness, and their disorder-level counterparts. That separation matters: it distinguishes transient responses from clinically

significant patterns. It also acknowledges that physical and visual motion can both trigger sickness, and that symptoms may accumulate over time. For a review on suppression, this has implications. Suppression could affect short-term symptom severity. It could also reinforce long-term avoidance, if the entire context becomes threatening through repeated suppression of nausea-related thoughts.

Keshavarz and Golding (2022) reviewed current concepts and management. They stress that motion sickness remains relevant in transport, simulators, and virtual environments. Management requires multiple approaches, because mechanisms and contexts differ. Medication is effective but has side effects and is not always practical for training, driving, or everyday tasks. Non-pharmacological strategies thus deserve attention. Suppression is a common spontaneous strategy—people often tell themselves: "Don't think about being sick," "Ignore the nausea," "Stop checking your stomach." The question is whether such instructions help or harm.

Motion sickness is multidimensional. A study that measures only nausea may miss dizziness, fatigue, eyestrain, thermoregulatory changes, or altered arousal. A single post-exposure score may hide the time course. A single global rating may obscure the difference between gastric symptoms and visual strain. Future studies on suppression should use multiple symptom domains and track them over time. Suppression might intensify nausea and anxiety without affecting eyestrain. It might raise perceived threat before any physiological change appears. Crude measurement would flatten these possibilities.

3.2 Visually Induced Motion Sickness and Cybersickness

Visually induced motion sickness has grown in practical importance. Screens, simulators, and VR systems now appear in education, gaming, therapy, medicine, military training, and engineering design. A technology can be immersive and impressive—and still leave users wanting to lie on the floor. Biswas, Mukherjee, and Bhattacharya (2024) reviewed cybersickness research and documented rapid growth in the field. Their review covers the range of symptoms, causes, measures, and mitigation strategies. For the present review, this body of work is useful because virtual environments allow researchers to manipulate both visual motion and psychological instructions experimentally.

Cybersickness also illustrates why suppression may depend on context. In a moving car, one can close one's eyes, look out the window, or stop reading. In VR, social pressures often intervene—finishing a task, completing a game, avoiding embarrassment. Removing the headset may feel like failure. Suppression may then interact with presence, workload, visual flow, and self-consciousness. In clinical settings, patients may tolerate discomfort for therapeutic goals. In gaming, rewards may encourage persistence through symptoms. In research labs, participants may try to be "good subjects." Social performance can thus turn a vestibular challenge into a cognitive-emotional one.

Measurement tools have evolved to address this complexity. Kourtesis, Linnell, Amir, Argelaguet, and MacPherson (2023) developed and compared a cybersickness questionnaire for VR against established measures. Their work reflects a broader shift toward instruments suited to immersive settings. Questionnaire refinement is not merely technical. It determines whether studies can detect the psychological mechanisms at work. If suppression mainly increases nausea-related checking and subjective distress, a scale must capture those dimensions. If it alters fatigue or disorientation later in exposure, the scale must track the time course. The field needs measures sharp enough to reveal the mechanism—not merely broad enough to produce a number.

3.3 Physiological and Neurocognitive Markers

Physiological markers are gaining traction in motion-sickness research. Park et al. (2022) used heartbeat-evoked potentials during HMD exposure and linked motion sickness to cognitive processing changes, proposing indicators with some classificatory value. Other studies have applied EEG, functional connectivity, pupillometry, cardiovascular signals, and machine learning to detect or predict visually induced sickness (Shen et al., 2024; Yang et al., 2022). These tools matter for suppression research because suppression may leave traces beyond self-report. If it increases cognitive load, physiological arousal, or interoceptive monitoring, multimodal measurement should detect at least some of those changes.

Li et al. (2025) used EEG to examine music's mitigating effects on motion sickness in simulated driving. Joyful and soft music reduced symptoms more than other conditions, though the mechanisms remain unclear. The finding supports a broader principle: emotional and attentional context can influence sickness. That principle is central here. Suppression is an

attentional and regulatory context. It does not alter the visual motion input, but it may alter how the person processes bodily signals during that input. The same sensory conflict may thus feel different under acceptance, distraction, catastrophizing, suppression, or calm external engagement.

Physiological measures could help resolve a central question. Suppose suppression worsens reported symptoms but leaves physiological markers unchanged. That would suggest a mainly interpretive or reporting effect. Suppose it alters autonomic activity, gastric rhythms, heartbeat-evoked potentials, pupil dynamics, or EEG indices. Then the mechanism likely involves deeper body-brain coupling. Suppose suppression has no effect in trained individuals but worsens symptoms in anxious or highly susceptible ones. Then individual risk moderates the effect. Without physiological and behavioural data, the field will remain anchored to subjective reports and plausible speculation.

3.4 Anxiety, Expectation, and Emotional State

Anxiety connects suppression to motion sickness in a particularly direct way. Alcantara-Thome, Miguel-Puga, and Jauregui-Renaud (2021) found that anxiety and motion sickness susceptibility may affect horizontal-plane orientation updating. Their study does not demonstrate that anxiety causes sickness through suppression. It does suggest that anxiety-related processes intertwine with spatial updating and susceptibility. That is enough to motivate an integrated model. A highly anxious person may detect bodily sensations earlier, interpret them as threatening, and attempt suppression more forcefully. That combination may produce stronger symptoms than motion alone.

Expectation also matters. If a person expects sickness, early bodily cues may serve as confirmation. Suppression may then act as a nocebo amplifier. The person tries not to think about vomiting because it seems likely. That effort confirms that the thought is dangerous. Each symptom becomes a test result. This can occur without conscious drama—noticing the stomach, checking the throat, scanning for dizziness, comparing the present with past episodes. The body provides noise; the mind turns it into a narrative.

Prior severe episodes may intensify this pattern. They provide vivid memories. The person may suppress not only current nausea but also memories of previous vomiting, embarrassment, or loss of control. The target is emotionally loaded. Recent suppression research confirms that target content and training matter (Mamat & Anderson, 2023; Mamat et al., 2024). Suppressing a neutral phrase is not the same as suppressing a feared bodily memory while seated in a moving vehicle. The latter combines sensory provocation, autobiographical memory, threat appraisal, and ongoing bodily signals.

3.5 Thought Suppression In Bodily-Symptom Contexts

Direct studies of thought suppression during motion-sickness exposure appear rare. That gap is the main justification for this review. But related literatures on bodily symptoms offer clues. Kreddig et al. (2022) showed that suppression and focused distraction have different effects on pain-related attention. Their findings caution against assuming that suppression always triggers rebound. They also highlight the need to distinguish suppression from distraction. In motion sickness, that distinction is essential. Reading a neutral story may reduce symptom focus. Telling oneself “don’t think about nausea” may keep nausea at center stage, because the forbidden target must be monitored.

Bodily symptoms differ from purely cognitive targets in a critical way. Suppress a word, and it may disappear until memory or monitoring retrieves it. Suppress nausea, and the body keeps sending signals. The motion continues—the car turns, the boat rocks, the VR scene moves. Motion sickness is thus a uniquely difficult target. The person is not suppressing a thought; they are suppressing awareness of an ongoing bodily process. The body does not accept administrative instructions from consciousness.

Targets also differ among themselves. One person may suppress “I will vomit.” Another may suppress stomach sensations. A third may suppress memory of a previous episode. These are not identical. Suppressing catastrophic interpretations might reduce fear if done briefly and effectively. Suppressing interoceptive sensations may increase monitoring. Suppressing memories may affect expectancy. Future studies should manipulate the suppression target rather than treating suppression as a single strategy. Otherwise, inconsistent results will appear mysterious when they are merely underspecified.

3.6 Countermeasures and the Indirect Evidence for Attentional Regulation

Behavioral countermeasure research offers indirect support for the attention-regulation link. Rahimzadeh et al. (2023)

reviewed nutritional and behavioral approaches, including music and diaphragmatic breathing. Breathing may influence autonomic regulation. Music may shift attention and emotion. Li et al. (2025) added EEG evidence that music type alters motion-sickness reduction patterns. These studies do not prove that suppression worsens symptoms. They do show that psychological context matters.

That point is central. If external, calming, or emotionally positive strategies reduce symptoms, then internal, effortful, threat-focused strategies might plausibly increase them. The mechanism need not be symmetrical, but it invites direct testing. Researchers could compare suppression with acceptance, external distraction, breathing, and a neutral control. Does suppression produce more symptom escalation, greater physiological arousal, less habituation, or stronger avoidance after exposure? That design would move the topic from elegant speculation to empirical testing.

Habituation adds another layer. Repeated exposure reduces motion sickness for many people. But habituation may require tolerating sensations long enough for the nervous system to update predictions. Suppression may interfere if it frames sensations as intolerable threats. Acceptance or mindful strategies might support habituation by allowing sensations to be noticed without panic or avoidance. This remains hypothetical for motion sickness, but it fits broader regulatory logic. The body may learn safety when symptoms are experienced without catastrophic control; it may learn threat when symptoms are constantly suppressed and monitored.

4. How Thought Suppression May Exacerbate Motion Sickness

4.1 Monitoring of Nausea-Related Sensations

The first and most direct pathway is monitoring. Suppression requires knowing whether the unwanted thought has returned. When the target is nausea, monitoring becomes bodily scanning. The person checks the stomach, throat, head, balance, sweat, and salivation. Each check can amplify minor sensations. This is especially likely when the person believes nausea is dangerous, embarrassing, or uncontrollable. Suppression thus gives nausea a privileged position in awareness. It says, in effect: “Do not think about nausea, but keep checking that you are not thinking about nausea.” Even poorly designed software would reject this loop.

Monitoring may amplify motion sickness because symptom perception depends partly on attention. A mild sensation may remain background noise when attention is elsewhere. The same sensation may become evidence of deterioration when attention locks onto it. A person who suppresses may therefore experience greater subjective severity without any immediate change in the provocative stimulus. Over time, increased attention may also affect autonomic arousal and behaviour. The person may tense muscles, alter breathing, reduce head movement, close eyes, stop engaging with the environment, or withdraw from adaptive strategies. Monitoring is not passive observation. It can change the system being observed.

4.2 Cognitive-Load Competition

The second pathway is cognitive-load competition. Motion sickness requires the nervous system to manage conflicting sensory information. Suppression adds a second task: inhibit unwanted content, monitor for its return, and regulate distress. Park et al. (2022) linked visually induced motion sickness to cognitive processing capacity via heartbeat-evoked potentials. If sickness itself reduces cognitive processing, then suppression during sickness may create a vicious cycle. The person needs resources to adapt to motion but spends them trying not to think about being sick.

This pathway predicts that suppression should be most harmful under high-load conditions. It may matter more in immersive VR, reading in a moving vehicle, complex simulator tasks, demanding navigation, or socially evaluative contexts where hiding symptoms matters. It may matter less during mild, passive exposure with ample resources and low threat. The prediction is testable. Researchers can manipulate task load and suppression instructions simultaneously. If suppression worsens sickness mainly under high load, a contextual model gains support. If suppression worsens sickness equally across low and high load, then monitoring or expectancy may be more important.

4.3 Threat Appraisal and Negative Expectancy

Threat appraisal is the third pathway. Suppression can signal that the target is dangerous. Desperately avoid thoughts of vomiting, and vomiting gains psychological weight. The very need for control makes the thought seem significant. This strengthens negative expectancy. The person expects sickness, watches for it, and interprets ambiguous cues as its onset.

Anxiety rises. Increased anxiety may further disrupt spatial updating, consistent with findings linking anxiety, susceptibility, and orientation updating (Alcantara-Thome et al., 2021).

Expectancy also operates via memory. People with severe prior episodes enter motion environments with a stored script: early discomfort signals another bad episode. Suppression may activate that script by marking the thought as forbidden. Acceptance or reappraisal might weaken the script—treating the thought as a mental event, not a prediction. This is a mechanistic claim about appraisal and feedback, not a call for optimism.

4.4 Autonomic and Gut-Brain Feedback

Autonomic and gastrointestinal feedback constitute the fourth pathway. Symptoms include nausea, sweating, pallor, and arousal shifts. Talsma and de Winkel (2025) argue that gut-brain communication is underappreciated in motion sickness. If gut afferents shape sickness, then cognitive states affecting autonomic regulation may influence severity. Suppression can be stressful. Stress alters breathing, vagal tone, sympathetic activity, and gastrointestinal sensation. These shifts may feed back into nausea.

Testing this pathway is difficult but feasible. Researchers could combine motion exposure with heart rate variability, electrodermal activity, gastric rhythm, respiration, and symptom ratings, comparing suppression against acceptance, distraction, or breathing. If suppression increases sympathetic markers or reduces adaptive parasympathetic regulation while symptoms worsen, the autonomic pathway gains support. If symptoms change without physiological shifts, the effect may be perceptual. Both outcomes would advance the field.

4.5 Interference with Adaptive Countermeasures

Behavioral interference is the fifth pathway. People reduce sickness by looking at stable references, controlling head movement, adjusting posture, using airflow, or stopping exposure. Suppression may interfere. Absorbed in avoiding nausea, a person might miss orientation cues. Ashamed of symptoms, they might hide discomfort instead of adjusting. Interpreting symptoms as failure, they might persist too long, forming a stronger aversive memory.

This pathway is especially relevant in VR, transport, training, and classrooms. A student may suppress symptoms to avoid embarrassment. A trainee may suppress due to performance evaluation. Here, suppression is socially shaped—the environment rewards endurance and penalises disclosure. Future research should measure perceived pressure, embarrassment, and willingness to stop. Social contexts make nausea a performative issue.

4.6 A Contextual Taxonomy for the Suppression Hypothesis

The proposed mechanism becomes clearer when contexts are separated. A low-risk context involves mild visual motion, low nausea expectancy, good user control, and a non-bodily suppression target. Suppression may have little effect or offer brief control. A moderate-risk context involves noticeable motion, mild symptoms, and early checking for sickness. Suppression may increase symptom salience. A high-risk context involves strong sensory conflict, high susceptibility, prior vomiting memories, social pressure, and a target like “don’t think about throwing up.” Suppression is most likely to amplify symptoms here.

This taxonomy avoids lazy universal claims. It also generates testable predictions. The suppression effect should be stronger for nausea-related targets than neutral ones. It should be stronger among high-susceptibility individuals than low-susceptibility individuals. It should be stronger during active monitoring than during trained direct suppression. It should be stronger when users cannot easily stop exposure. These predictions are narrow enough to test and broad enough to matter.

The taxonomy also explains why some everyday advice backfires. Telling someone “don’t think about being sick” may sound supportive but may push them toward internal monitoring. Orienting externally, breathing slowly, looking at a stable reference, or engaging with a non-threatening task may work through different pathways. The distinction seems obvious once stated—which is why it is frustrating that it has not been measured more often. Motion-sickness research is technically sophisticated but still under-measures the self-instructions people generate during sickness.

4.7 Methodological Implications for Future Experiments

The taxonomy has methodological consequences. Future experiments should not merely ask whether participants tried to suppress. They should assign precise regulatory instructions and measure whether those instructions changed monitoring,

effort, expectancy, and symptom-focused attention. A suppression condition: keep nausea-related thoughts out of mind. An acceptance condition: notice nausea-related sensations without fighting them. A distraction condition: attend to an external auditory or visual task. A reappraisal condition: interpret nausea as a temporary, non-dangerous response to sensory conflict. A neutral control: no special instruction. This design would separate mechanisms that everyday language mashes together.

The same design should stratify by susceptibility. The strongest test would recruit participants across low, moderate, and high VIMS susceptibility, expose them to the same visual-motion stimulus, and collect repeated symptom ratings, physiological indices, gaze behaviour, and post-exposure recovery data. The crucial outcome is not whether suppression always increases sickness. It is whether suppression increases sickness under the predicted high-risk conditions. That is the kind of question that can survive peer review.

5. Research Challenges and Controversies

5.1 The Direct-Evidence Gap

The biggest challenge is straightforward: direct evidence is sparse. Motion sickness, cybersickness, cognitive load, anxiety, interoception, and thought suppression all have strong literatures. But few studies directly manipulate suppression during motion exposure. So any review claiming "thought suppression worsens motion sickness" as settled overreaches. The defensible conclusion is that suppression is a plausible, under-tested amplifier. That is not timid—it is precise.

This evidence gap also affects reference selection. Many adjacent studies examine mechanisms rather than the full causal chain. Park et al. (2022) support a cognitive-load account of visually induced sickness. Mamat et al. (2024) support a nuanced view of suppression. Talsma and de Winkel (2025) support gut-brain expansion of motion-sickness models. Rahimzadeh et al. (2023) and Li et al. (2025) support behavioural and emotional countermeasures. These sources build the mechanism but do not substitute for direct experiments. A strong review should make that boundary explicit.

5.2 Suppression Versus Distraction

A second challenge is frequent confusion between suppression and distraction. Suppression says: "Don't think about X." Distraction says: "Attend to Y." The two can look similar from the outside—both reduce direct engagement. Internally, however, suppression often requires monitoring X, while distraction may not. In motion sickness, this difference may determine outcomes. External distraction may reduce nausea focus. Suppressing nausea may intensify it. If studies fail to distinguish these processes, findings become hard to interpret.

This problem also affects intervention design. Telling someone to "take your mind off it" is not the same as "don't think about nausea." The first may encourage external engagement. The second may trigger internal policing. Researchers should therefore write instructions carefully and check whether participants used the intended strategy. Manipulation checks should assess monitoring, effort, perceived control, emotional tone, and target content. Without these, a suppression condition may secretly become distraction, rumination, acceptance, or panic with better wording.

5.3 Short-Term Relief Versus Long-Term Cost

A third controversy involves time scale. Suppression may reduce discomfort briefly. A passenger who suppresses nausea for two minutes may feel in control. That short-term benefit does not guarantee long-term adaptation. The person may rebound later, develop avoidance, or show poorer habituation across exposures. Conversely, brief rebound does not mean suppression is always harmful. Mamat and Anderson (2023) show trained suppression can be beneficial in some mental-health contexts. The field needs time-sensitive models.

Motion sickness suits time-scale studies because symptoms unfold across exposure. Researchers can collect minute-by-minute ratings, continuous physiology, and follow-up reports. They can examine onset latency, peak severity, recovery speed, and later avoidance. Suppression may affect one phase more than another—delaying symptom reporting but increasing peak severity, or reducing anxiety during exposure while slowing recovery. It may help experienced users but harm novices. A single post-exposure score cannot answer such questions.

5.4 Heterogeneity Across Motion Contexts

Motion sickness does not occur in a single environment. Cars, boats, aircraft, simulators, VR headsets, rotating chairs, and visual displays generate distinct sensory conflicts. Physical and visually induced sickness share symptoms but differ in

triggers. Cybersickness adds immersion, presence, latency, field of view, interface demands, and user interaction. Suppression may operate differently across these settings. On a ship, suppression may centre on gastrointestinal monitoring. In VR, it may involve visual strain, disorientation, and performance pressure. In automated driving, it may combine reading, loss of control, and anticipatory anxiety.

This heterogeneity does not make the topic impossible. It makes broad generalisations impossible. Studies should specify the motion context, symptom domain, suppression target, and participant group. A model that works in VR gaming may not work on ships. A model that works in adolescents may not work in experienced sailors. A model that works for nausea may not work for headache. Researchers should stop treating "motion sickness" as one blob simply because the phrase is convenient.

5.5 Measurement and Reporting Bias

A fifth challenge is measurement bias. Suppression may affect what people report rather than what they feel. Some participants may underreport because suppression ties to self-control. Others may overreport because monitoring makes sensations salient. Social desirability also matters. In group travel, classrooms, military training, or laboratories, people may hide symptoms. In clinical settings, they may report more carefully. These dynamics can mimic or mask genuine physiological effects.

Multimodal measurement can reduce this problem. Symptom scales should pair with physiological markers, behavioural signs, task performance, and recovery indices. But physiological markers also have limits. Autonomic activity is not specific to motion sickness. Heart rate variability can reflect anxiety, effort, posture, breathing, and medication. EEG indices can reflect attention, fatigue, and artefact. Machine learning can classify patterns, but classification accuracy does not explain mechanism. The point is not to replace self-report with machines. It is to triangulate, because one measure alone is a lonely monarch with too much power.

5.6 Cultural and Developmental Considerations

A final challenge concerns culture and development. Coping with motion sickness may be shaped by social norms around endurance, emotional control, embarrassment, and symptom disclosure. Suppression may be more common where expressing discomfort is discouraged. Development also matters. Children and adolescents may have different susceptibility, emotion-regulation skills, and willingness to disclose. Most current studies rely on adults, students, or convenience samples. This limits generalisation.

Future work should examine whether suppression has stronger effects among individuals with high trait anxiety, high susceptibility, strong embarrassment concerns, high interoceptive vigilance, or low emotion-regulation flexibility. It should also examine whether training changes the effect. Suppression as a learned attentional skill may differ from suppression as panic control. The recent suppression literature makes this distinction unavoidable (Mamat & Anderson, 2023; Mamat et al., 2024). The motion-sickness literature now needs to catch up.

6. Conclusion and Future Directions

This review argues that thought suppression may exacerbate motion sickness—but only contextually. The evidence does not justify a simple universal statement. Direct literature is too sparse. However, recent research provides a coherent mechanism. Motion sickness involves sensory conflict, individual susceptibility, cognitive load, autonomic regulation, and body-brain communication. Suppression can create monitoring, consume cognitive resources, increase threat appraisal, strengthen expectancy, and interfere with adaptive coping. When the target is nausea-related, suppression may make bodily signals more salient and harder to regulate. That is the central argument.

The strongest case is for a conditional amplification model. Suppression should worsen motion sickness most strongly under five conditions: (a) moderate to high susceptibility; (b) noticeable bodily signals from the motion stimulus; (c) a symptom-related target (nausea, dizziness, vomiting imagery); (d) anxiety, embarrassment, or performance pressure; (e) ongoing monitoring rather than trained, brief, direct attentional control. Under these conditions, suppression may turn a physiological disturbance into a self-monitoring loop.

Future research should test this model directly. A basic experiment could randomly assign participants to suppression, acceptance, external distraction, breathing, or neutral control during standardised visually induced motion. Researchers

should measure symptoms repeatedly, not just at the end. They should include nausea, dizziness, ocular strain, fatigue, thermoregulatory symptoms, and arousal. They should measure thought intrusions, suppression effort, monitoring, expectancy, anxiety, and perceived control. They should collect physiological data—heart rate variability, electrodermal activity, respiration, pupil size, eye movements, and, where feasible, gastric or EEG markers. They should also test recovery speed and willingness to re-enter the motion environment.

A stronger design would include repeated exposures. If suppression worsens habituation, symptoms should decline more slowly across sessions. If suppression only affects momentary perception, symptoms may rise during exposure but not alter later adaptation. If suppression helps some participants, moderators must be identified. The goal is not to prove suppression is bad. It is to determine when it is bad, when harmless, and when useful. That is how mature science behaves without slogan-driven distraction.

Clinical and practical implications should remain cautious until direct evidence improves. It would be premature to tell all sufferers never to suppress. It would also be careless to ignore suppression as a common coping habit. Practitioners and designers can reasonably encourage strategies that reduce threat monitoring and support external orientation, calm breathing, gradual exposure, and adaptive stopping rules. VR developers, transport designers, and researchers should also reduce contexts that pressure users to hide symptoms. If people feel forced to suppress discomfort, the environment may be helping create the problem it later tries to measure.

The main gap is clear. Motion-sickness studies need to include cognitive coping variables; suppression studies need to enter bodily-symptom environments. Each field studies one side of the loop. Motion-sickness researchers study the body-brain response but often under-measure private regulation. Suppression researchers study cognitive control but often use targets without continuous bodily input. Motion sickness offers a powerful test case—the unwanted experience is embodied, dynamic, and consequential. If suppression amplifies symptoms there, the finding would refine both motion-sickness and emotion-regulation theory.

The final conclusion is simple but not simplistic. Suppression may exacerbate motion sickness when it becomes effortful monitoring of feared bodily sensations under sensory conflict. It is not a universal villain. It is a strategy whose function depends on target, context, susceptibility, timing, and control style. The literature now needs direct experiments that respect this complexity. Until then, the best answer is not "suppression causes motion sickness." It is: "Suppression may make motion sickness worse through identifiable mechanisms, and the field has enough evidence to test that claim properly." That sentence is less flashy. It is also far less overblown.

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