Vestibular Stimulation and Neuroplasticity: Evidence, Boundaries, and the Phase 1 Research Question

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Document Status

This scientific white paper was prepared by SeaKing Solace as an evidence-based review of the published scientific literature. It has not been peer reviewed or published by an academic journal. Its purpose is to establish the scientific and evidentiary framework used to inform the SeaKing Solace research program. The underlying studies cited throughout the paper include peer-reviewed publications; their inclusion should not be interpreted as independent validation of SeaKing Solace's hypotheses or proposed applications.

1. Introduction

1.1 Scientific Question and Rationale

The question here is not whether vestibular stimulation can alter the nervous system, that premise is already supported by a substantial experimental literature. The question is whether established forms of vestibular plasticity, together with evidence on learning, sensory integration, state dependence, and repeated exposure, provide sufficient biological and translational rationale to investigate repeated natural multidimensional motion as a distinct experimental stimulus.

Direct human evidence establishes that vestibular processing is modifiable by experience. Controlled visuo-vestibular mismatch can rapidly alter vestibulo-ocular reflex (VOR) gain, with the magnitude of adaptation influenced by visual context.[1] Vestibular perceptual learning has been demonstrated in older adults exposed to repeated passive motion training, with improvements in trained self-motion thresholds and selected postural and gait measures remaining detectable at least one day afterward.[2] Human vestibular systems therefore retain measurable capacity for experience-dependent change across sensorimotor and perceptual domains.

Natural motion cannot be assumed to reproduce laboratory conditions, however. Measurements during everyday human movement show that natural vestibular input is structured, multidimensional, and dependent on axis, behavior, and self-motion dynamics.[3] At the neural level, combined canal and otolith inputs are not processed as a simple linear sum; central vestibular neurons integrate them subadditively and frequency-dependently.[7] Because a moving vessel produces concurrent, temporally varying vestibular inputs rather than a single controlled rotation, translation, or electrical perturbation, natural multidimensional motion is a stimulus class requiring direct study, not an ecological substitute for an already characterized laboratory intervention.

Evidence connecting vestibular stimulation with learning is conditional rather than uniform. In healthy adults, noisy galvanic vestibular stimulation (nGVS) enhanced acquisition of a complex functional-mobility task, with improved performance persisting briefly after stimulation ended; the same study found no significant advantage for a manual-control task, and retention was assessed only over a short post-stimulation interval.[4] In a separate repeated-training experiment using a simulated lunar-rover navigation task, participants improved with practice but nGVS did not accelerate the rate of learning.[6] Vestibular stimulation can therefore influence some learning processes under defined conditions, with direction and magnitude depending on task and paradigm.

Reproducible physiological responsiveness must also be distinguished from neuroplasticity. Repeated exposure to a fixed galvanic stimulus can produce highly individual yet comparatively reproducible eye-movement patterns within the same person.[5] Such within-person stability demonstrates that vestibular responses contain reliable individual structure; it does not establish adaptation, learning, or a stable neuroplastic phenotype. Reproducibility describes the consistency of a response, while plasticity requires evidence that the system has changed as a consequence of experience.

Together, these findings support a bounded framing for SeaKing. Repeated natural multidimensional motion is scientifically plausible as a candidate stimulus for producing measurable vestibular and physiological responses and, potentially, experience-dependent change, a plausibility that follows from direct evidence that human vestibular systems are plastic, that passive externally imposed input can participate in learning, that responses show meaningful within-person structure, and that natural motion provides a complex stimulus not captured by single-axis paradigms.[1–5] It does not follow that natural boat motion has been demonstrated to enhance neuroplasticity, improve learning, or produce therapeutic benefit; no direct human study in the present evidence base establishes those outcomes. SeaKing is therefore approached here as an evidence-generating research program rather than a proven intervention. The literature provides substantial basis for asking whether repeated, quantitatively characterized natural multidimensional motion produces reproducible acute responses, exposure-dependent changes, or physiological states relevant to later learning studies; direct research determines which of those possibilities actually occur.

1.2 Why Neuroplasticity Requires Evidentiary Discipline

That plausibility is not itself a license to treat "neuroplasticity" as a single, uniformly positive outcome. The term is frequently used as though synonymous with improvement, but scientifically it is not: neural systems can change without producing learning; learned performance can improve without durable retention; retained changes can fail to generalize beyond the trained task; and persistent changes can be beneficial, harmful, or functionally indeterminate. These distinctions govern the claims made throughout this review.

A translational hierarchy therefore separates at least eight outcomes: immediate performance or physiological response, acquisition, consolidation, retention, near transfer, far transfer or generalization, spontaneous real-world behavioral change, and meaningful functional or clinical benefit. Evidence at one level does not establish the levels above it.

Human learning studies show these dissociations directly. Motor-sequence performance can change during an offline interval after practice, so what is acquired during training and what is expressed after consolidation are not identical phenomena.[9, 17] Conversely, different instructional methods

can produce similar immediate performance yet differ substantially in later maintenance and generalization: in a randomized trial involving individuals with acquired brain injury, systematic instruction produced no clear immediate accuracy or fluency advantage but yielded better 30-day maintenance and better generalization of trained skills to interactions with other people.[10] The later consequences of learning cannot always be inferred from end-of-session performance.

Transfer requires equally careful treatment. Controlled cognitive-training studies show that substantial improvement on a practiced task can occur without corresponding superiority on near-transfer, far-transfer, or everyday functional outcomes,[13, 14] consistent with a broader motor-learning and rehabilitation literature emphasizing that trained-task improvement is not generalized functional recovery.[18] Transfer is possible, but must be demonstrated rather than presumed.

The same discipline applies to vestibular adaptation. Sensory reweighting can be functionally adaptive: individuals with vestibular impairment may redistribute reliance toward other sensory information as vestibular reliability declines, and healthy postural control dynamically adjusts sensory weighting as environmental conditions change.[11, 12] These processes show that changes in sensory weighting can contribute to compensation and effective behavior, not that all vestibular change is beneficial or that adding vestibular stimulation to an intact system will improve function.

Persistent vestibular phenomena provide the complementary boundary. Persistent postural-perceptual dizziness (PPPD) involves chronic dizziness or unsteadiness associated with upright posture, motion, and visually complex environments,[15] and motion-triggered mal de débarquement syndrome (MdDS) can involve persistent rocking, bobbing, or swaying after passive-motion exposure.[16] Neither establishes a single maladaptive-plasticity mechanism, and neither should be used to portray normal vestibular adaptation as hazardous. What they demonstrate is more fundamental: persistence alone has no positive functional valence, and a change can endure and still be undesirable. Persistent changes are therefore classified here as adaptive, maladaptive, or functionally indeterminate according to demonstrated consequences, a framework developed in Section 2.4.

The consequences for SeaKing follow directly from this hierarchy. An acute change in heart rate variability, pupillary dynamics, or vestibular responsiveness would establish a measurable response, not neuroplasticity; systematic change across repeated exposures could support exposure-history dependence, but not learning; improved acquisition would not establish consolidation; retention would not establish generalization; and generalization would not establish meaningful benefit in daily life. These are not semantic restrictions. They define the experiments required to move from biological plausibility to functional evidence, and they let a positive early result stand without being asked to prove more than it measured.

1.3 Scope and Exclusions

Applying that discipline requires specifying what kinds of evidence this review draws on and how they relate to one another. It examines evidence across several interconnected levels: direct vestibular adaptation and perceptual learning; natural and experimentally controlled self-motion; human learning and retention; hippocampal and spatial-network interactions; neuromodulatory and state-dependent influences on learning; developmental considerations; and the distinction between persistent neural change and meaningful functional outcome.

One methodological rule applies throughout: stimulation modalities are informative but not interchangeable. Natural multidimensional motion, passive mechanical motion, active self-motion, isolated rotation or translation, galvanic and noisy galvanic stimulation, caloric stimulation, implanted and transcutaneous auricular vagus nerve stimulation (taVNS), and nonvestibular neuromodulation each engage the nervous system through different physical inputs, anatomical routes, temporal structures, and experimental contexts. This matters most when translating laboratory vestibular findings to natural motion. Human self-motion produces structured inputs across multiple dimensions,[3] central vestibular encoding differs according to whether movement is actively generated or externally imposed,[19] and concurrent canal and otolith signals interact nonlinearly

rather than additively.[7] The effects of natural multidimensional motion cannot be predicted by summing findings from isolated pitch, roll, translation, rotation, or electrical paradigms.

Electrical vestibular stimulation remains valuable because it provides controlled causal evidence about vestibular responsiveness and selected learning processes, nGVS enhanced acquisition in one functional-mobility paradigm while failing to improve another sensorimotor task.[4] Such findings inform the biological possibility that vestibular input interacts with learning without establishing that mechanically generated natural motion will do the same. The same restriction applies outside the vestibular system: taVNS has enhanced acquisition of selected novel letter-sound associations in healthy adults under a defined protocol,[20] relevant to state-dependent modulation and learning but not evidence that multidimensional motion recruits the same neuromodulatory pathways. Neural changes associated with nonvestibular interventions cannot be transferred to SeaKing merely because downstream brain regions or theoretical mechanisms overlap.[8, 20]

Animal studies are used where they provide mechanistic information not presently obtainable in humans, particularly for cellular, circuit-level, hippocampal, and neuromodulatory questions. Their role is to establish biological possibility and constrain hypotheses, not to serve as direct proof of human SeaKing effects; the same principle applies to evidence from vestibular disease, rehabilitation, and electrical stimulation models. The review does not assume that an observed neural or physiological change is therapeutic, nor does it treat adverse persistent vestibular syndromes as evidence that repeated natural motion is generally harmful, both errors collapse functional interpretation into the mere presence of change.

Within this framework, existing evidence establishes multiple component capabilities that make repeated natural multidimensional motion a credible stimulus to investigate. It does not establish that SeaKing-like motion enhances neuroplasticity or produces therapeutic benefit. That unresolved step is precisely why direct experimental study is warranted, and it is the question the next section's evidentiary framework is built to keep in view.

2. Conceptual and Evidentiary Framework

2.1 Operational Meanings of Plasticity, Adaptation, Habituation, and Learning

The evidentiary discipline just established depends on terminology that distinguishes different forms of change rather than treating them as interchangeable. Throughout this review, neuroplasticity is an umbrella term for experience-dependent change in nervous-system processing or organization. Where evidence demonstrates only a behavioral, perceptual, physiological, or systems-level effect, the phenomenon is described at that measured level rather than attributed to an unmeasured cellular or synaptic mechanism.

Adaptation is a change in system output that improves or recalibrates performance in relation to altered sensory, motor, or environmental conditions. Human VOR research shows such recalibration can be shaped by the temporal structure of training: repeated VOR training blocks separated by consolidation intervals produced differences in subsequent retention, so repeated exposure is not simply additive and the organization of practice influences what persists.[21] Adaptation can also be response specific, repeated exposure to an experimentally unreliable galvanic signal produced durable changes in postural behavior detectable months later while other vestibular responses showed no corresponding adaptation.[24] A persistent adaptive effect need not represent global change across the vestibular system.

Habituation describes attenuation of a response with repeated exposure rather than recalibration toward a new sensory or motor relationship. Repeated low-frequency rotational stimulation can reduce selected dynamics of the human VOR, although the degree of retention varies substantially between individuals.[22] Habituation can therefore represent genuine experience-dependent change without implying improved performance, generalized learning, or therapeutic benefit.

Learning is reserved for evidence that performance, discrimination, prediction, or another task-relevant capability changes as a consequence of experience. Repeated passive roll-tilt discrimination training improved self-motion discrimination under the tested conditions, but with substantial task specificity and limited transfer.[23] The finding establishes vestibular perceptual learning while demonstrating why learning should not be equated with generalized enhancement.

These distinctions separate acquisition (change expressed during or immediately following practice) from consolidation (processes stabilizing or modifying the acquired state afterward) and retention (what remains when the system is tested after an interval). Motor-learning research shows performance can change during an offline period after training,[9] conceptual work on memory formation distinguishes acquisition from consolidation rather than treating learning as instantaneous,[17] and VOR research provides the practical analogue: the interval between training blocks influences retained adaptation even when immediate training conditions are similar.[21]

Compensation is related but distinct. Following vestibular loss, postural control can redistribute reliance across remaining sensory channels, maintaining stability despite degraded vestibular information;[11] healthy postural control likewise reweights sensory information when environmental reliability changes.[12] These establish the nervous system's capacity for context-sensitive sensory reorganization, not that externally increasing vestibular stimulation in an intact system will improve function.

None of these terms is synonymous with benefit. Vestibular compensation can support improved function;[11] PPPD and motion-triggered MdDS show that persistent vestibular-related states can instead be adverse;[15, 16] and measurable neural change may occur without clinical advantage.[8] Section 2.4 develops this classification.

The operational rule is therefore straightforward: evidence is labeled according to what was actually demonstrated. Adaptation is not called learning; habituation is not called adaptation; acquisition is not treated as retention; retention is not treated as transfer; persistent change is not presumed beneficial merely because it persists. If repeated natural motion produces a progressively altered physiological or vestibular response in Phase 1, that establishes exposure-dependent change; whether to classify it as adaptation, habituation, learning, or compensation depends on the design and the behavior of the measured endpoint, and its functional meaning requires the further evidentiary step developed below.

2.2 Evidence Hierarchy and Intervention-Specific Inference

Distinguishing forms of change is only half the discipline; the other half is distinguishing what kind of evidence supports each claim. Evidence relevant to vestibular stimulation and neuroplasticity spans several experimental levels that are complementary but not interchangeable, and rigorous synthesis requires asking not merely whether a finding exists, but what kind of inference it can support.

Direct human experimental evidence provides the strongest basis for claims about humans under a defined intervention. Functional neuroimaging during caloric vestibular stimulation shows that controlled vestibular stimulation recruits the human hippocampus within a distributed vestibular network.[25] This is direct evidence for regional engagement, not for hippocampal long-term potentiation (LTP), neurogenesis, improved memory, or therapeutic neuroplasticity, none of which were measured.

Direct mechanistic animal evidence addresses questions that cannot ordinarily be tested invasively in humans. In rats, passive whole-body rotation facilitates electrically induced CA1 LTP through a cholinergic septohippocampal mechanism.[26] Under the conditions of that experiment, vestibular input interacts with cellular mechanisms of hippocampal synaptic plasticity, without establishing that natural human motion generates hippocampal LTP, that boat motion produces the same effect, or that the mechanism improves human learning.

Human translational evidence occupies another level. Quantification of vestibular inputs during ordinary behavior shows natural self-motion contains structured, multidimensional acceleration and

rotation whose statistical properties vary across axes and activities.[3] This bears directly on the physical stimulus SeaKing seeks to characterize and establishes that natural motion differs meaningfully from an isolated laboratory perturbation, without establishing that those statistics generate neuroplasticity or benefit.

Evidence that challenges a mechanistic narrative matters equally. In an animal study using galvanic vestibular stimulation, changes in hippocampal cell-proliferation and neurogenesis-related measures occurred without corresponding change in spatial-memory performance.[27] The protocol used high-amplitude stimulation under anesthesia and cannot be generalized to natural human motion, but its contribution is important: biological change within a learning-related structure does not guarantee behavioral improvement.

Framework and review evidence serves another purpose. Systematic review of MdDS supports recognition of a persistent adverse motion-related phenotype while showing its causal neural mechanism remains unresolved.[28] Neuroimaging reviews of PPPD identify altered patterns within sensory and brain networks but cannot establish whether individual findings represent cause, compensation, predisposition, or consequence.[29] Such literature defines the state of knowledge, identifies convergence, and exposes uncertainty; it cannot convert associative or heterogeneous evidence into causal proof.

These four classes are not ranked by universal scientific value; each answers a different question. The restriction is that inference cannot silently cross between them: animal evidence cannot establish a human SeaKing mechanism, electrical stimulation cannot establish an effect of natural mechanical motion, neuroimaging activation cannot establish synaptic plasticity, and a review of a clinical syndrome cannot prove its causal mechanism. This discipline does not weaken the rationale for SeaKing; it makes the rationale testable. The literature can establish that vestibular systems are plastic, that vestibular information reaches learning-related networks, that natural motion provides a complex multidimensional stimulus, and that vestibular input can interact with plasticity-related biological processes under defined conditions.[3, 25, 26] It cannot presently establish that repeated SeaKing-like motion produces those downstream effects in humans, that question remains empirical.

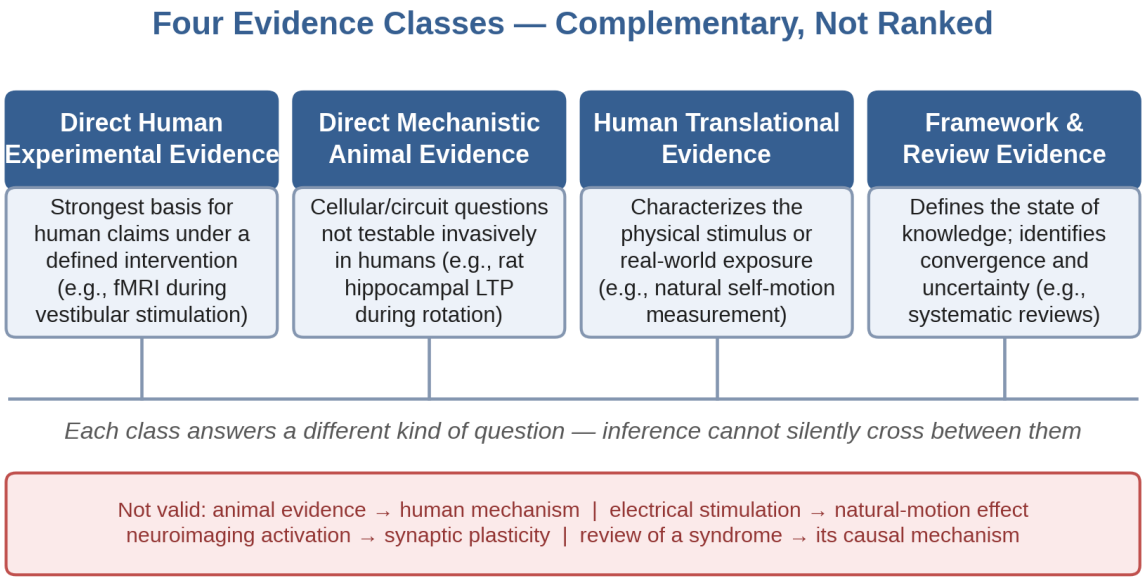

Figure 1. Four classes of evidence relevant to vestibular stimulation and neuroplasticity. Each class answers a different kind of question; inference cannot silently cross between them.

2.3 Translational Outcome Hierarchy

Evidence classes and outcome levels are two sides of the same discipline: just as inference cannot silently cross between evidence classes, outcomes must be organized according to what they actually demonstrate rather than assumed from one another. The progression used here runs:

neural or physiological response → training performance → acquisition → consolidation → retention → near transfer → far transfer or generalization → spontaneous real-world behavior → meaningful functional or clinical benefit

The arrows indicate a research progression, not an assumption of causality. Each transition requires its own evidence, and none follows automatically from the stage before it.

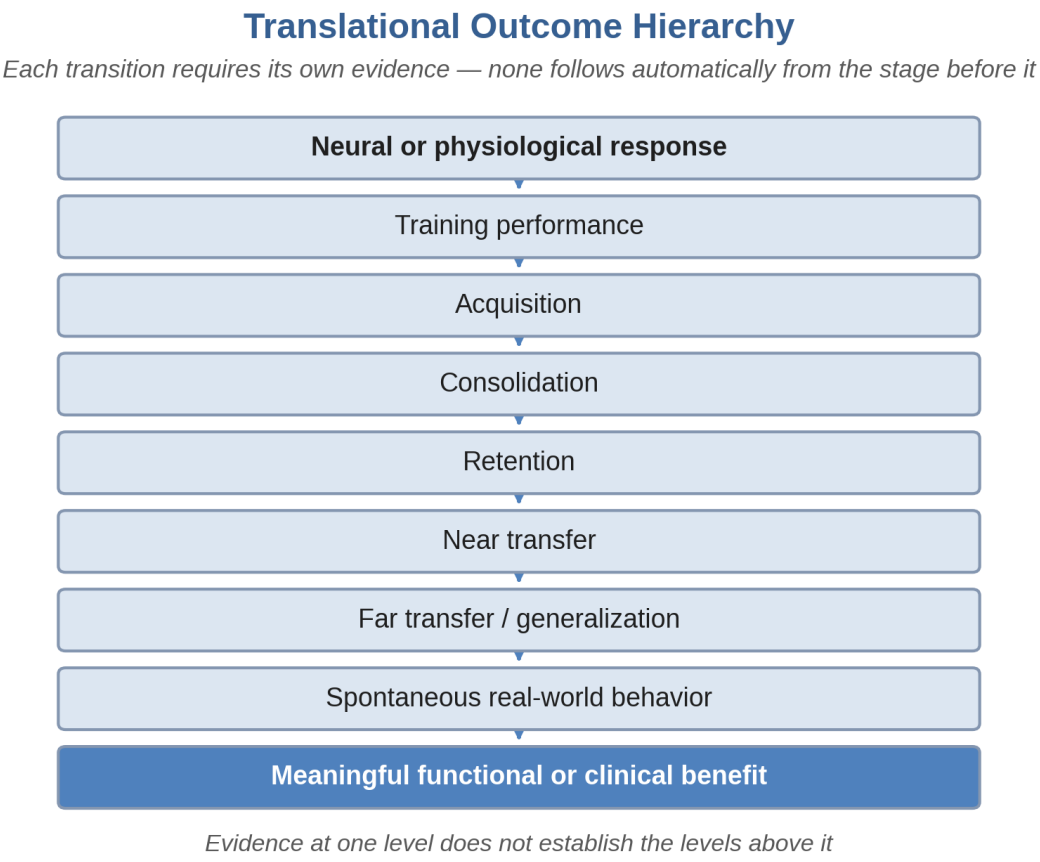

Figure 2. The translational outcome hierarchy used throughout this review. A finding at one stage is evidence only for that stage; stronger claims require independent evidence at each subsequent level.

A neural or physiological response establishes only that an intervention measurably affects the organism, important when reproducible within individuals or systematically related to stimulus parameters, but not learning or adaptation. Training performance describes what occurs during exposure; acquisition requires evidence that capability changes through experience; consolidation concerns stabilization or offline modification afterward, which motor-learning experiments show can continue changing performance during a post-training interval;[9] and retention asks whether the change survives a delay.

Transfer concerns whether improvement extends beyond the trained condition, and the evidence is conditional in both directions. Following task-specific motor training after stroke, improvement on one practiced task did not necessarily transfer fully to a distinct motor task,[30] yet robot-assisted

rehabilitation has demonstrated generalization to an untrained movement within the same workspace.[31] Learning can generalize, but degree and type depend on task and context.

The distinction sharpens for far transfer and real-world behavior. An intervention that appears unremarkable at the end of training can differ in the durability or portability of what was learned.[10] In autism research, longitudinal analysis shows autistic children can display social-communication abilities across different people, settings, and time points, though directionally and context dependently rather than uniformly, an observation that cannot attribute every generalized behavior to the parent intervention, particularly where that trial showed no effect on the relevant primary measure, but that does demonstrate generalization can be measured rather than assumed.[32] Controlled cognitive-training studies supply the complementary boundary: gains on a trained domain can occur without superior effect on real-world function[13] or on separate near- and far-transfer outcomes.[14] Transfer is not impossible; it must be an independent endpoint.

At the highest level lies meaningful function or clinical benefit. A change becomes clinically meaningful only when connected to an outcome that matters in the relevant population, as the randomized PPPD study illustrates, where an intervention-related perfusion difference did not translate into superiority over sham on the primary dizziness endpoint.[8]

This hierarchy lets early SeaKing studies produce important findings without prematurely requiring proof of efficacy: a reproducible acute response would establish biological responsiveness, systematic change across repeated exposures could establish exposure-history dependence, persistence after an interval could support retention, and extension to an untrained task would address transfer. Only linkage to prespecified real-world outcomes would justify a claim of meaningful benefit.

2.4 Functional Classification of Persistent Change

The hierarchy above answers how far an effect extends; a separate question is whether an effect that persists is good, bad, or neither. Persistence describes duration. It does not determine whether a change is beneficial, harmful, or neutral. Persistent neural, physiological, perceptual, or behavioral changes are therefore classified here as adaptive, maladaptive, or functionally indeterminate according to demonstrated consequences.

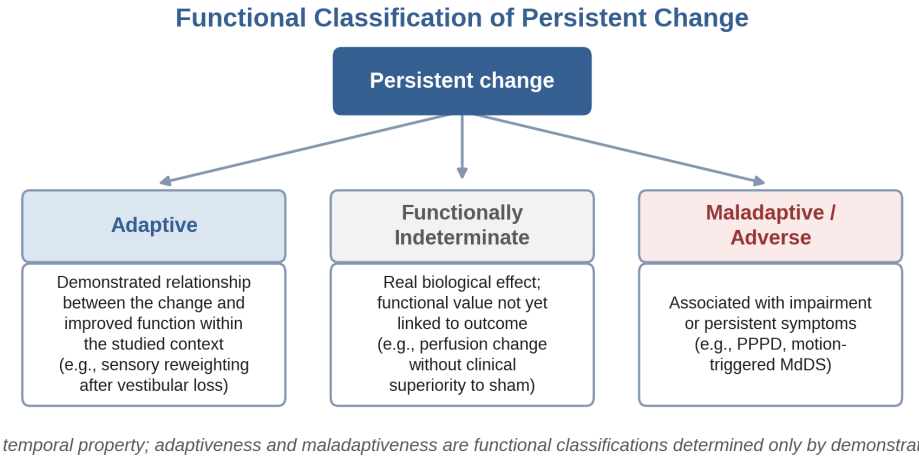

Figure 3. Functional classification of persistent change. Persistence is a temporal property; whether a persistent change is adaptive, maladaptive, or functionally indeterminate depends on demonstrated consequence, not duration.

Adaptive change is supported by the vestibular-compensation literature. Individuals with vestibular loss can alter multisensory weighting in ways that support postural stability despite degraded vestibular information,[11] healthy adults dynamically reweight sensory inputs when reliability

changes,[12] and vestibular-rehabilitation frameworks describe mechanisms through which adaptation, substitution, habituation, and compensation contribute to improved function after impairment.[33] The boundary matters: compensation after pathology does not prove that additional stimulation improves an intact system. Adaptive classification depends on a demonstrated relationship between the change and improved function within the studied context.

Maladaptive or adverse persistent change is well established at the phenotype level. PPPD is characterized by chronic dizziness or unsteadiness with sensitivity to upright posture, motion, and visually complex environments;[15] motion-triggered MdDS can involve persistent rocking, bobbing, or swaying after passive-motion exposure.[16, 28] These conditions demonstrate that persistent vestibular-related states can be functionally adverse, but not one settled neural-plasticity mechanism. Neuroimaging findings in PPPD show persistent differences in sensory and network organization that cannot by themselves determine whether a feature is causal, compensatory, predisposing, or consequential.[29] Likewise, the association between passive-motion exposure and MdDS establishes a clinically meaningful phenotype without determining a SeaKing-specific incidence, dose-response relationship, or causal cellular mechanism.[28]

Functionally indeterminate change is equally important. A persistent biological effect may be real while its functional value remains unknown, as in the sham-controlled PPPD trial, where treatment-related regional cerebral-perfusion differences occurred without significant active-versus-sham superiority on the primary dizziness outcome.[8] The correct interpretation is neither that the finding was meaningless nor that it demonstrated benefit.

This category will matter particularly in early SeaKing research, where a persistent change in autonomic physiology, vestibular responsiveness, pupillary behavior, or another biomarker should initially be classified as functionally indeterminate unless linked prospectively to a relevant functional endpoint. That is not a negative conclusion: it indicates a biological effect has been identified and that the next question is whether it predicts, mediates, or accompanies meaningful behavior. The framework prevents two opposite errors, reading persistence as proof of therapeutic neuroplasticity, and treating uncertainty about functional consequence as evidence that the finding lacks value.

3. Direct Human Evidence for Vestibular Plasticity

With that evidentiary framework in place, the review turns to the evidence itself. The strongest foundation for any relationship between vestibular stimulation and neuroplasticity is direct evidence that the human vestibular system changes as a consequence of experience. That evidence is substantial, and it establishes an equally important second principle: vestibular plasticity is conditional, its magnitude, persistence, and transfer depending on stimulus parameters, sensory context, training structure, and the outcome measured. Human vestibular function is therefore neither fixed nor invariably modifiable by repetition. Section 4 develops the parameter dependence; this section establishes what has been demonstrated.

3.1 Human Vestibulo-Ocular and Sensorimotor Adaptation

Because the VOR stabilizes gaze during head movement, investigators can impose a controlled mismatch between head motion and visual information and measure whether the nervous system recalibrates. It does, rapidly, and by an amount that depends on the sensory environment in which the error occurs: incremental visuo-vestibular mismatch during passive head impulses produced significant VOR gain increases under two visual conditions, with greater adaptation in an enriched than a uniform visual environment (approximately 32.5% versus 24.1%).[1]

Training structure alters both acquisition and persistence. Three five-minute VOR training blocks separated by 20-minute rest intervals produced greater adaptation than a single block, but by less than linear accumulation would predict, and the spaced protocol left acquired gain comparatively stable across the subsequent hour.[21] Repetition interacted with temporal structure rather than functioning as an arithmetic increase in stimulus quantity.

Longitudinal training separates two components of the response. Across 10 adaptation sessions over 12 days followed by 19 further days of testing, each session produced approximately 10% short-term adaptation while repeated exposure gradually produced a smaller baseline shift that saturated at approximately 5% above initial baseline and remained partially detectable during subsequent testing.[34] Acute adaptation and persistent baseline change were distinguishable. That study also found adaptive drive weakened once residual gain deviation and associated retinal image slip fell below a relatively small range,[34] which argues against assuming every increment of vestibular error produces proportionate adaptation, though those values arose from a specific VOR paradigm and establish no universal threshold transferable to natural motion, cognition, or SeaKing dosing.

Three further findings establish that the effective stimulus is not captured by exposure quantity. Sinusoidal training centered at 1.3 Hz or spanning approximately 0.5 to 2 Hz produced measurable adaptation while a 0.5-Hz protocol did not, and adaptation transferred incompletely between sinusoidal and higher-frequency head-impulse responses.[35] Systematically varying training time, impulse number, repetition rate, and visual-target exposure, adaptation was influenced more strongly by total training time and visual-error exposure than by increasing the number or rate of impulses.[36] And when training demand varied substantially during a manual gaze-stabilization paradigm, adaptation was markedly smaller than under tightly controlled conditions, with participants with unilateral vestibular hypofunction showing response-specific differences.[38]

Error organization also matters. Comparing incremental visuo-vestibular training with a conventional constant-demand approach, both altered VOR gain, but incremental training produced larger gain changes in healthy participants and larger active-response changes in participants with unilateral hypofunction; adaptation acquired during active training was also expressed during passive testing.[37]

The human VOR literature therefore establishes a strong but selective capability claim: the adult vestibular sensorimotor system can recalibrate in response to controlled experience, repeated exposure can produce changes extending beyond the training period, and training structure alters acquisition and retention, while frequency, error magnitude, visual context, exposure duration, variability, and structure all constrain the result.[1, 21, 34–38] This supports the premise that repeated vestibular experience can change human sensorimotor processing. It does not support assuming any particular natural-motion exposure will produce adaptation, nor does VOR adaptation establish changes in cognition, affective regulation, autonomic function, or clinical outcome.

3.2 Vestibular Perceptual Learning

Controlled experiments also demonstrate perceptual learning, experience-dependent improvement in the ability to discriminate or judge self-motion, with a consistent pattern of genuine improvement and limited transfer.

Klaus and colleagues trained blindfolded healthy adults using passive 0.2-Hz roll-tilt direction discrimination. After approximately nine hours and 1,800 trials, thresholds for the trained condition decreased by approximately one-third with no comparable improvement in untrained controls. Improvement did not clearly transfer to pitch tilt or interaural translation, and an apparent change at an untrained higher frequency also appeared in controls.[23]

Wagner and colleagues assigned 30 healthy adults to passive roll-tilt training at 0.2 or 0.5 Hz or to no training, across approximately 1,300 trials over five days. The 0.2-Hz group improved significantly at both 0.2 and 0.5 Hz; the 0.5-Hz group improved at 0.2 Hz with a borderline result at its nominal training frequency; neither improved at the untrained 1-Hz condition. The experiment also detected reduced postural sway under selected foam-standing conditions in one training group[39], limited functional transfer rather than generalized benefit, since it established neither broad behavioral improvement, long-term retention, nor clinical efficacy.

Fitze and colleagues extend the finding into later adulthood and add a time-course observation. Forty healthy adults aged 70 to 88 were randomized to repeated passive roll-tilt or interaural-translation discrimination training on a six-degree-of-freedom motion platform, completing seven sessions totaling 2,800 trials across approximately two weeks. Thresholds improved for the trained motion and selected postural and gait measures also improved, with final assessment one day after training. Improvement was not detectable after three sessions but emerged after the full protocol, and cross-motion transfer was again limited.[2] The time course of perceptual learning can therefore differ from rapid VOR adaptation, which cautions against reading an early null as evidence that a stimulus cannot produce learning.

A negative study defines the boundary. Hartmann and colleagues gave healthy participants intensive passive yaw-rotation or interaural-translation discrimination training in darkness. Despite 3,360 trials across six or twelve days, thresholds did not significantly improve; where visual information was available performance could improve, but this did not transfer to the purely vestibular condition.[40] The contrast with the positive roll-tilt studies argues against a rule that enough repeated trials will inevitably improve thresholds: motion geometry, combined canal and otolith engagement, sensory context, task demands, feedback, and training frequency may all determine whether learning occurs.

Vestibular perceptual learning is therefore real, measurable, and reproducible under appropriately structured conditions, but is not a generic consequence of repeated exposure.[2, 23, 39, 40] Positive results establish capability, not universality.

Table 1. Human vestibular perceptual-learning studies discussed in Section 3.2. All used passive, externally imposed motion; transfer across axes and frequencies was consistently limited or absent.

Study	Population	Paradigm	Key Finding	Transfer
Klaus et al.[23]	Healthy adults	Passive 0.2-Hz roll-tilt discrimination, ~1,800 trials	Thresholds decreased ~33% for trained condition	Not to pitch tilt or interaural translation
Wagner et al.[39]	Healthy adults (n=30)	Passive roll-tilt at 0.2 or 0.5 Hz, ~1,300 trials	Improvement at trained and some untrained frequencies	Not to untrained 1-Hz condition
Fitze et al.[2]	Adults aged 70–88 (n=40)	Passive roll-tilt or interaural translation, 7 sessions	Thresholds improved; selected postural/gait gains	Limited across motion type
Hartmann et al.[40]	Healthy adults	Passive yaw/translation in darkness, 3,360 trials	No significant threshold improvement	Visual-condition gains did not transfer to darkness

3.3 Passive Externally Imposed Motion Can Support Learning

The active-passive distinction matters for SeaKing because much of the vestibular stimulation aboard a moving vessel is externally generated. A potential objection would be that meaningful vestibular learning requires self-generated motion; direct human evidence does not support that restriction.

Mahfuz and colleagues compared active self-generated with passive externally imposed head-impulse training during incremental VOR adaptation. Both produced approximately equivalent short-term adaptation under matched visual-error conditions, with no significant retention difference attributable to how the head movement was generated, and with post-training visual exposure influencing retention more strongly than training mode.[41] This does not make active and passive movements neurologically identical, active self-motion engages motor commands, efference copies, and prediction that externally imposed motion does not. It establishes something narrower and directly relevant: passive vestibular motion is not inherently incapable of driving measurable adaptation when an appropriate learning signal is present. The perceptual-learning studies in 3.2

reinforce this, since all the positive roll-tilt paradigms used passive externally imposed movement.[2, 23, 39]

The boundary condition is equally important: passive motion by itself is not sufficient. Hartmann and colleagues also used repeated passive stimulation, yet thousands of trials failed to improve pure-vestibular thresholds.[40] The relevant difference cannot be reduced to passive versus active. Passive vestibular input can serve as a substrate for learning when combined with an effective task structure, error signal, sensory context, and stimulus geometry; the nervous system neither requires all learning signals to be self-generated nor automatically transforms repeated externally imposed motion into learning.

This sharpens the SeaKing rationale. Natural vessel motion is a legitimate experimental vestibular stimulus because passive inputs support human plasticity, but natural boat motion differs from VOR error training and laboratory discrimination protocols in stimulus geometry, predictability, behavioral context, duration, and the presence or absence of explicit task feedback. The appropriate inference is experimental plausibility, not established efficacy.

3.4 Lifespan Retention of Vestibular Plasticity

Developmental neuroscience demonstrates that age influences plasticity: sensitive periods exist, and the efficiency or form of experience-dependent change differs across developmental stages.[42] It would nevertheless be incorrect to convert developmental sensitivity into a claim that meaningful plasticity disappears after childhood.

The Fitze result is direct evidence against an early-life-only model: significant vestibular perceptual learning in healthy adults aged 70 to 88, with effects detectable one day after training rather than confined to the training trials themselves.[2] It does not establish that magnitude, speed, mechanism, or durability are identical across ages, developmental research argues strongly against age invariance, with different systems showing different periods of heightened sensitivity,[42] and adolescence increasingly understood as a period of continuing and, in selected domains, heightened experience sensitivity rather than the end of childhood plasticity.[43]

These frameworks establish general principles about developmental timing, not an autism-specific or SeaKing-specific treatment window, and neither they nor the vestibular evidence justifies an exact age cutoff.[42, 43] Age should be treated as a biological moderator rather than an exclusion criterion. If repeated vestibular stimulation is eventually shown to produce meaningful neuroplastic effects, age may influence dose, learning rate, retention, or the nature of the response; current evidence does not support assuming the relevant capacity disappears outside a narrow developmental window.

3.5 What Null and Limited-Transfer Findings Mean

Several of the strongest studies above contain both positive and null results, frequency-selective VOR adaptation,[35] reduced adaptation under variable training demand,[38] failed threshold improvement despite thousands of discrimination trials in darkness,[40] and roll-tilt learning that did not transfer across axes, motion types, or frequencies.[23, 39] These findings do not contradict vestibular plasticity. They define it.

Two interpretive errors are available: using a positive finding to claim that vestibular stimulation generally enhances neuroplasticity, or using a null result from one stimulus to claim it cannot produce learning. The Hartmann-Klaus contrast shows why neither follows. Both used extensive passive discrimination training; one improved thresholds substantially and one did not.[23, 40] The scientific question is not whether vestibular stimulation was repeated, but which stimulus was delivered, under what sensory conditions, at what frequency and magnitude, with what error or feedback structure, for how long, and against which outcome.

Limited transfer carries a related lesson in both directions. Task specificity should not be mistaken for absence of plasticity, improvement confined to a trained judgment is still genuine learning. Equally,

limited transfer prevents a trained improvement from being called generalized vestibular enhancement: a decrease in roll-tilt discrimination threshold establishes improved roll-tilt discrimination under the tested conditions, not improved spatial cognition, autonomic regulation, emotional regulation, or clinical function.[23, 39]

For SeaKing these findings are constructive. Natural multidimensional motion should not be conceptualized as a single exposure that is either effective or ineffective; its amplitude, frequency content, temporal variability, axis combinations, duration, sensory context, and repeated-exposure history are experimental variables. A Phase 1 study can therefore be informative even where some motion conditions or endpoints show little exposure-dependent change: a null effect under one profile would constrain the parameter space, a reproducible response under another would identify a candidate region for further study, a response changing across sessions would motivate testing retention, and evidence persisting beyond exposure would justify subsequent questions about transfer and functional significance.

The direct human literature thus provides a strong but bounded foundation. The conclusion is not merely that the vestibular system is plastic, but that it is experimentally tractable as a learning system whose responses depend on the structure and history of stimulation.[1, 2, 21, 23, 34–43]

4. Parameter, Task, and Stimulus Dependence of Vestibular Plasticity

The studies in Section 3 were organized by what they demonstrate. Read instead by what governs them, they converge on a single principle: vestibular plasticity is parameter, task, and context dependent. The nervous system does not respond according to a rule in which more stimulation produces proportionately more plasticity.

4.1 Vestibular Dose Is Multidimensional

The VOR findings in 3.1 identify at least six distinct dimensions of an effective stimulus, none reducible to the others.

Frequency is selective: 1.3-Hz and 0.5-to-2-Hz training produced adaptation where 0.5-Hz training did not, with incomplete transfer across response frequencies.[35] Error magnitude interacts with system state: adaptive drive weakened once residual retinal-slip error fell below a small range, so the relationship between stimulus error and response was not indefinitely linear and the effective stimulus depends partly on the state of the system receiving it.[34] Duration outweighed repetition count, with adaptation influenced more by total training time and visual-error exposure than by more or faster head impulses, arguing directly against a repetition-count model of neuroplasticity.[36] Spacing affected acquisition and retention differently, three spaced blocks producing more adaptation than one but far less than three times as much, while leaving retention comparatively stable.[21] Consistency mattered independently: variable adaptive demand produced markedly smaller adaptation than tightly controlled training, even though the nominal task still involved repeated vestibular and visual stimulation.[38] And error organization shaped the outcome, with incremental training outperforming constant-demand training in both healthy participants and those with unilateral hypofunction.[37]

Reducing vestibular dose to a single quantity such as minutes of exposure, number of movements, or acceleration magnitude would therefore discard variables already known to affect human adaptation.[21, 34–38]

A different neuromodulatory system supplies a conceptual control against assuming more stimulation must produce more plasticity. In rats undergoing vagus-nerve-stimulation-paired motor training, moderate-intensity stimulation produced greater task-relevant cortical reorganization than either lower or higher intensities.[44] This is not a vestibular dose-response curve and its intensity values cannot be translated to natural motion. Its relevance is narrower: a plasticity-promoting intervention can have an intermediate effective range rather than a more-is-better relationship, a conclusion the human vestibular findings reach independently.

Neither the presence nor absence of a response at one dose therefore defines the capability of the system. A null condition identifies an ineffective region of parameter space; a positive condition establishes that the system can respond under those parameters; neither establishes the optimum.

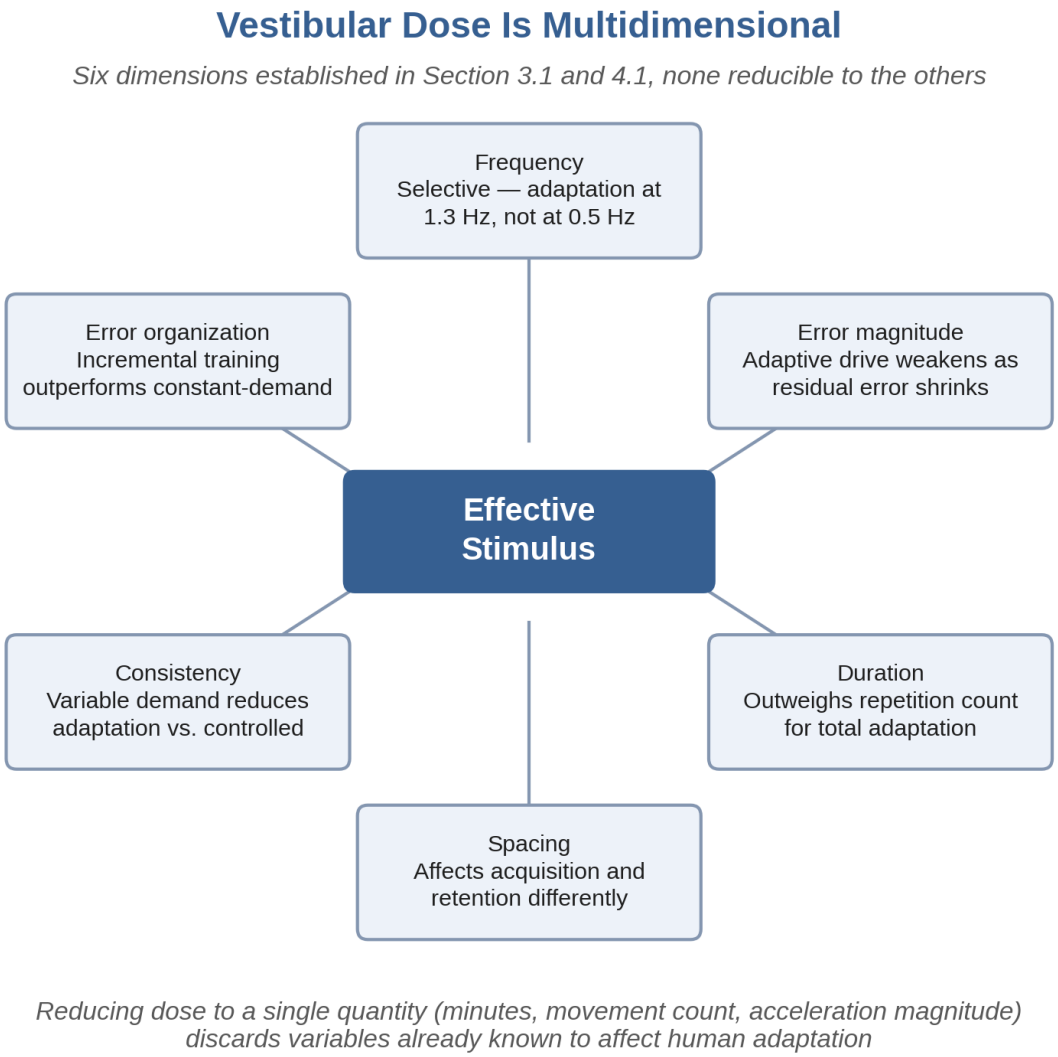

Figure 4. Vestibular dose as a multidimensional construct. Six parameters established in the human VOR literature each independently shape the effective stimulus; none can be reduced to the others.

4.2 Task and Sensory-Context Dependence

The physical stimulus does not act alone. The same visuo-vestibular mismatch produced greater adaptation in an enriched than a uniform visual environment,[1] and the Hartmann-Klaus contrast in 3.2 cannot be explained by repetition, since both paradigms involved extensive practice, motion geometry, sensory combination, visual context, task requirements, and feedback structure determined whether learning emerged.[23, 40] Task structure likewise governed the gaze-stabilization result: describing an activity broadly as VOR training did not predict its physiological effect, because how the task delivered the adaptive error was critical.[38]

Context can even become part of the learned state. Shelhamer and colleagues trained healthy adults to develop opposing VOR gains associated with different vertical gaze positions; afterward, participants expressed different gains depending on whether they looked upward or downward.[45]

The nervous system had learned context-linked response states rather than one globally expressed adapted gain. The study involved three participants and should not be generalized into a broad theory of state-dependent learning, but it provides direct human vestibular evidence that an adapted response can become conditional on a contextual cue.

This is especially relevant to natural motion. A person aboard a moving vessel receives vestibular stimulation alongside changing visual horizons, optic flow, proprioceptive information, pressure through the feet and body, auditory information, active postural corrections, expectation of movement, and behavioral activity. Whether those signals strengthen, weaken, redirect, or merely accompany an exposure-dependent response cannot be assumed from laboratory studies, and their known influence on controlled vestibular learning means they should not be treated as irrelevant noise.[1, 23, 38, 40, 45] The implication is methodological: motion should be quantified alongside its context wherever feasible, because a physical motion trace alone may not describe the effective neural stimulus.

4.3 Natural Multidimensional Motion Is Its Own Stimulus Class

Laboratory research necessarily simplifies motion, isolating rotation, translation, tilt, frequency, or a particular visual-vestibular mismatch to address causal questions under control. Natural motion differs in three ways that matter.

First, its structure. Measurements of head movement during ordinary behavior show the vestibular organs normally experience structured combinations of rotational and translational accelerations across multiple axes and frequencies, with statistical properties varying by dimension and behavior, and with the motion reaching the organs reflecting both active movement and the biomechanical filtering properties of the body.[3] This cannot be represented by a single sinusoidal rotation or translation.

Second, its central processing. Vestibular neural responses depend partly on whether movement is actively generated or passively imposed: in alert macaques, central vestibular neurons responded differently during matched active and passive translations, with attenuation during active movement depending on the relationship between predicted and actual proprioceptive feedback.[19] Two physically similar movements can be represented differently depending on how they were generated.

Third, its integration. Comparing neural responses to isolated and combined rotational and translational motion, Carriot and colleagues found central vestibular neurons integrated canal and otolith signals subadditively rather than as an arithmetic sum, with weighting varying by frequency.[7] At the level of the recorded neurons:

response to combined rotation and translation ≠ response to rotation alone + response to translation alone

This does not establish the behavioral consequence of every multidimensional stimulus. It establishes that component-by-component extrapolation can fail at the first stages of central vestibular integration. Suppose isolated roll, heave, and pitch each produced a known physiological response in the laboratory: it would still be unjustified to add those three effects and claim their simultaneous occurrence aboard a vessel would produce the summed response. Components may interact, weighting may depend on frequency, one dimension may alter the interpretation of another, and active postural and head movements may modify central processing of the externally imposed stimulus.[3, 7, 19]

The same non-equivalence separates intervention classes. nGVS can influence human task acquisition under selected conditions[4] and taVNS can enhance acquisition on selected learning measures under a different paradigm,[20] but GVS perturbs vestibular afferent signaling electrically, natural motion stimulates the organs through physical angular and linear acceleration, and taVNS targets an anatomically distinct peripheral pathway. Their effects cannot be transferred across classes merely because all eventually influence the central nervous system.[4, 20] Within mechanical stimulation the same applies: isolated yaw rotation is not roll tilt, translation is not

combined canal-otolith motion, and active head movement is not neurally identical to passive whole-body movement.[3, 7, 19]

A vessel compounds this, its motion evolving continuously through combinations of heave, pitch, roll, yaw, surge, and sway with changing relative contributions. This does not make natural motion scientifically uninterpretable; it makes direct characterization necessary. Natural-motion discovery therefore cannot begin from a simulation assembled from assumed effective component waveforms. A simulator may eventually reproduce profiles shown empirically to be relevant, but the multidimensional stimulus and its associated responses must be characterized first, which gives on-water measurement a mechanistic purpose in addition to an ecological one.

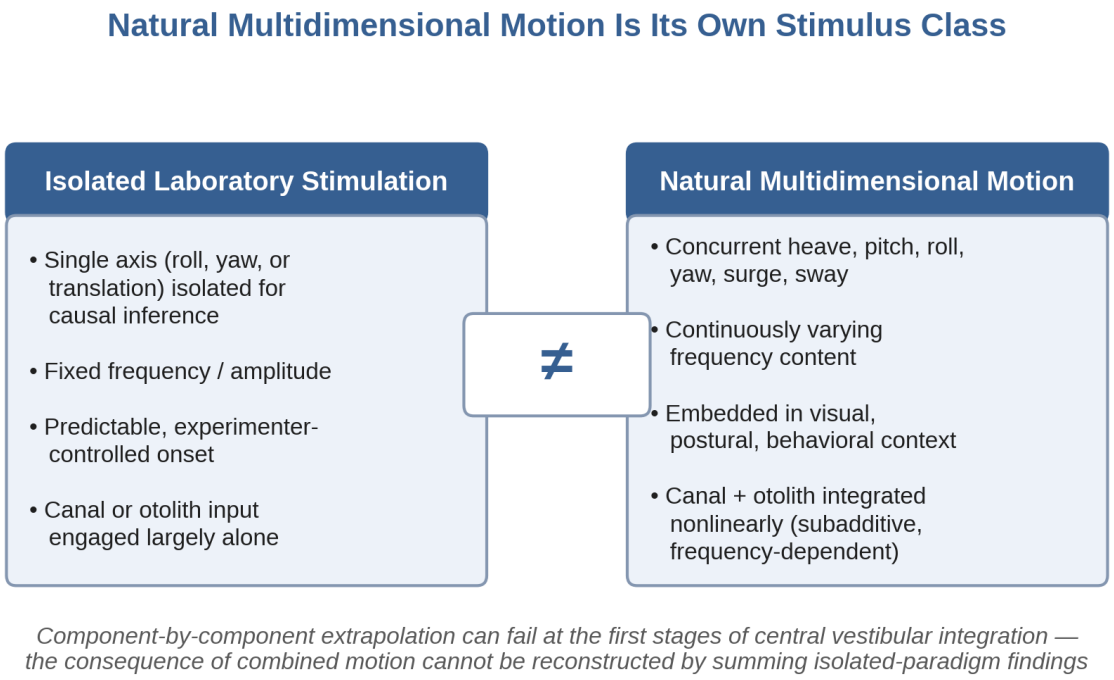

Figure 5. Natural multidimensional motion versus isolated laboratory vestibular stimulation. The two are not interchangeable stimulus classes; findings from one cannot be assumed to transfer to the other.

4.4 SeaKing Dose and Geometry Remain Empirical Variables

No existing evidence establishes an ideal combination of heave, pitch, roll, yaw, sway, or surge; no published study defines the optimal amplitude, frequency spectrum, exposure duration, repetition schedule, or intersession interval for SeaKing-like motion; and nothing establishes that increasing motion intensity will monotonically increase a desirable response. What the literature supplies instead is the list of variables that must be treated empirically.

Two further findings extend that list. Balter and colleagues measured galvanic-induced postural responses across repeated sessions and observed a decline after initial exposure followed by a relatively stable response, with intervals from one day to two weeks producing no material difference in the stabilized response within that protocol.[46] This is direct human evidence that exposure history can affect subsequent measurements while showing that a universal spacing advantage cannot be assumed, it does not establish that one-day and two-week intervals are equivalent for natural motion, vestibular learning generally, or SeaKing outcomes, but it demonstrates why each participant's exposure count and prior history belong in the experimental description.

Separately, MacDougall and colleagues found substantial between-individual differences in three-dimensional eye-movement responses to a standardized galvanic stimulus while repeated responses within individuals were considerably more stable.[5] The repeated-session component involved only two participants and cannot establish a stable responder phenotype, but it illustrates a design possibility that matters for discovery research: meaningful between-person heterogeneity can coexist with within-person structure. A group mean can be small even when individuals display substantial reproducible responses in different directions, and conversely, apparent individual differences in one session may disappear on repeated testing. Longitudinal sampling is required before stable response characteristics can be inferred.

Dose should therefore be treated as a multidimensional construct rather than a scalar, with candidate components including rotational and translational acceleration; axis composition and simultaneous multi-axis geometry; frequency spectrum; temporal variability; cumulative exposure duration; within-session sequence; number of repeated sessions; interval between sessions; participant movement and postural behavior during exposure; environmental sensory context; and prior exposure history. The relevance of each remains to be determined empirically.

This creates a clear role for an initial discovery phase. Rather than beginning from a claim that a particular vessel-motion profile is therapeutic, the study can characterize the motion environment continuously and determine whether specific features covary with measurable physiological responses within and between participants. Repeated sessions then make a second question possible: does the relationship between motion and response change systematically with exposure history? If acute responses are reproducible, they become candidate physiological signatures; if they change across sessions, they become candidates for exposure-dependent adaptation or habituation; if those changes persist beyond exposure, retention can be tested. Only then would it become appropriate to ask whether the resulting state influences learning, transfer, or meaningful function.

The resulting proposition is both narrower and stronger than a therapeutic claim: natural multidimensional motion is a biologically complex vestibular stimulus whose effective dose cannot be inferred from motion intensity or isolated laboratory components alone, and whose geometry, temporal structure, exposure history, individual response characteristics, and physiological consequences must be mapped empirically. That requirement is not a weakness in the SeaKing hypothesis. It is the central justification for the discovery experiment.

5. Vestibular Access to Hippocampal and Spatial-Navigation Systems

Sections 3 and 4 established that the vestibular system is plastic and parameter dependent. A separate question is whether vestibular information reaches neural systems relevant to spatial representation, memory, and learning. That narrower question is well supported: human neuroimaging, human vestibular-loss studies, and mechanistic animal experiments converge on the conclusion that vestibular information materially influences hippocampal and spatial-navigation networks.[25, 47–50]

The boundary is equally important. Access to the hippocampus does not establish hippocampal plasticity; altered hippocampal physiology does not establish enhanced learning; loss-of-function evidence does not show that increasing vestibular stimulation above normal improves cognition; and direct demonstrations of vestibular-induced hippocampal long-term potentiation (LTP) or neurogenesis in humans remain absent. The evidence supports a network-access and mechanistic-plausibility argument, not a claim that vestibular stimulation is a general hippocampal or cognitive enhancer.

5.1 Vestibular Access to Hippocampal and Spatial-Navigation Networks

Direct human neuroimaging demonstrates that controlled vestibular stimulation engages the hippocampus within a wider distributed network. Suzuki and colleagues used functional MRI during caloric vestibular stimulation in healthy adults and observed group-level activation involving the hippocampus alongside the insula, intraparietal sulcus, superior temporal cortex, cingulate cortex,

and thalamus.[25] Because the measure was blood-oxygen-level-dependent activity, this establishes regional recruitment but not synaptic potentiation, learning, neurogenesis, or therapeutic neuroplasticity.

Human loss-of-function evidence is complementary. Brandt and colleagues studied adults with chronic complete bilateral vestibular loss following bilateral vestibular neurectomy. Compared with controls, they showed impaired virtual spatial-navigation performance together with an approximately 16.9% reduction in hippocampal volume, while other forms of memory were not comparably impaired, a pattern specific to spatial processing rather than a generalized amnestic syndrome.[47] The population was highly selected, with profound bilateral deafferentation after surgery. The result establishes that intact bilateral vestibular input is relevant to normal hippocampal and spatial-navigation function. It does not establish the reverse proposition that increasing vestibular stimulation enlarges the hippocampus or improves spatial memory in neurologically intact people.[47]

Mechanistic animal work strengthens the network-level inference. Temporarily disrupting peripheral vestibular function in rats disrupted the normal location-specific firing of hippocampal place cells and the directional firing of head-direction cells, with spatial firing recovering as vestibular function returned;[48] an earlier experiment found vestibular lesions abolished normal directional firing in anterior-thalamic head-direction cells despite continued behavior.[49] Vestibular input is therefore functionally important to the systems representing position and direction in space, which is not evidence that additional stimulation enhances those representations beyond their normal state.

The human structural findings are heterogeneous, and usefully so. Kremmyda and colleagues studied patients with bilateral vestibulopathy retaining residual function, finding delayed spatial learning, greater spatial anxiety, and region-specific reductions in mid-hippocampal and posterior parahippocampal areas.[50] But Hüfner and colleagues found no significant hippocampal atrophy years after unilateral vestibular deafferentation and no clear spatial-navigation deficit after left-sided loss, with some evidence of laterality-dependent differences following right-sided loss,[51] and Göttlich and colleagues found no general reduction in total hippocampal gray-matter volume in a clinically representative group with incomplete bilateral vestibular failure, with reduced CA3 gray matter instead associated with greater clinical impairment.[52] The relationship between vestibular function and hippocampal structure is therefore not all-or-none: severity, bilaterality, residual function, disease duration, laterality, compensation, and analytical method may all shape the observed phenotype.[47, 50–52]

The conclusion supported by this evidence is that vestibular information is functionally integrated with hippocampal and spatial-navigation systems in both humans and animals.[25, 47–52] What remains unestablished is whether increasing natural vestibular input in humans improves those systems, whether repeated multidimensional motion changes hippocampal plasticity, or whether any resulting neural change would translate into better learning or function.

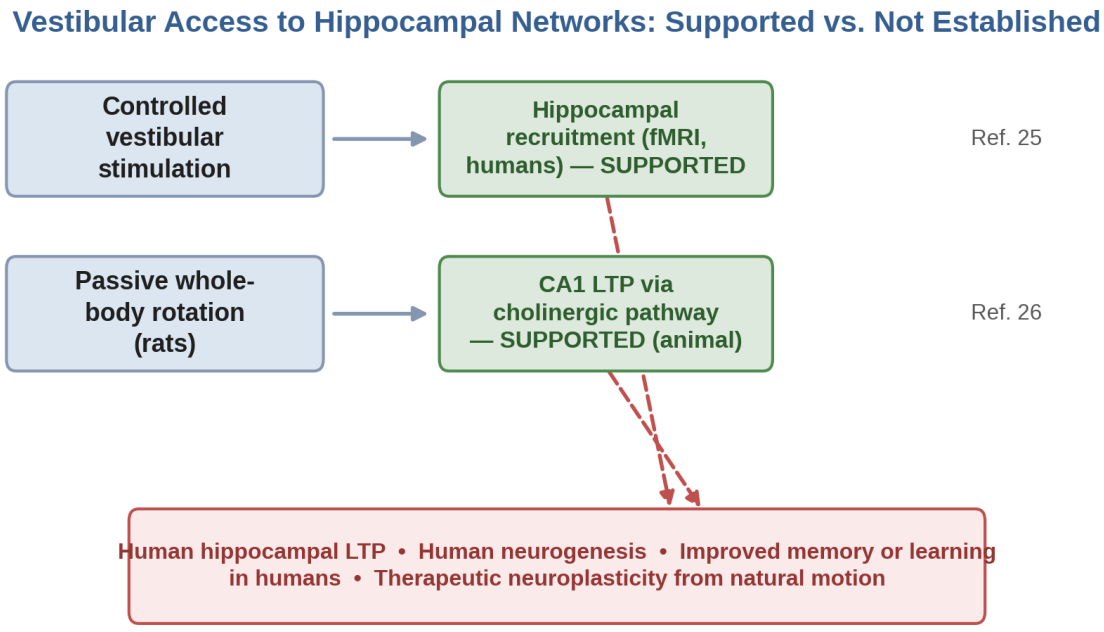

Figure 6. Vestibular access to hippocampal networks. Regional recruitment and animal LTP are directly supported; human hippocampal LTP, neurogenesis, and improved memory from natural motion remain unestablished.

5.2 Human Translational Evidence From Stimulation and Vestibular Loss

The stimulation and loss-of-function literatures in 5.1 approach the same relationship from opposite directions, and both stop short of a stimulation claim: demonstrating that normal vestibular information is required for normal spatial-network function does not demonstrate that increasing vestibular input beyond ordinary levels will further enhance it.

Clinical stimulation studies provide a more direct translational bridge while introducing a further distinction, vestibular stimulation may influence cognition differently when the nervous system is recovering from vestibular injury than when it is neurologically intact. Oh and colleagues randomized 83 adults with acute unilateral peripheral vestibulopathy to ten daily sessions of near-threshold galvanic vestibular stimulation or sham. The active group showed greater improvement on several visuospatial outcomes, including measures of spatial-learning recovery, over the four-week study period.[53] This is important human evidence that a defined vestibular intervention can influence the trajectory of selected cognitive outcomes. It is also a recovery study: acute unilateral vestibular injury creates an altered biological state involving compensation and reorganization, so the result cannot establish that the same stimulation would enhance cognition in healthy participants or that natural mechanical motion would engage the same processes.[53]

The appropriate inference across this literature is therefore not that vestibular stimulation improves the hippocampus, but that vestibular input is biologically relevant to human spatial-network function and that altering vestibular input can, under defined circumstances, alter outcomes linked to those networks.[25, 47, 50–53] That provides a credible translational foundation for asking whether natural multidimensional motion interacts with learning-related systems. It does not answer the question in advance.

5.3 Animal Evidence for Hippocampal Physiology and Plasticity-Related Biology

Animal studies permit direct physiological and cellular measurements generally unavailable in human vestibular experiments, and they supply the strongest mechanistic evidence connecting vestibular input with hippocampal biology.

Cholinergic signaling. Electrical stimulation of the peripheral vestibular system increased hippocampal acetylcholine release in rats measured by in vivo microdialysis, rising to approximately 152% of baseline under one stimulation condition.[54]

Synaptic potentiation. Tai and Leung examined CA1 LTP in freely behaving rats while an electrical LTP-induction train was delivered either during passive whole-body rotation or during immobility. LTP was greater when induction occurred during rotation, and that enhancement was abolished by muscarinic blockade and by selective lesions of medial-septal cholinergic neurons.[26] This is direct causal mechanistic evidence within its experimental system: passive vestibular rotation can facilitate electrically induced CA1 LTP through a cholinergic septohippocampal mechanism. The qualification is essential, passive rotation did not independently produce spontaneous LTP. Electrical stimulation induced the potentiation, with rotation modifying the magnitude of that evoked process. The study supports a state-dependent interaction between vestibular input and synaptic-plasticity mechanisms in rats. It does not establish that motion generally causes LTP, that natural boat motion induces human hippocampal LTP, or that enhanced LTP improves behavior.

Oscillatory state. Chronic bilateral vestibular lesions in rats disrupted normal hippocampal theta characteristics, including reduced theta power and frequency under tested conditions,[55] while passive whole-body rotation elicited continuous hippocampal theta in behaviorally immobile rats, with theta peak frequency increasing with rotational velocity and the effect depending substantially on septohippocampal cholinergic signaling.[56] The velocity dependence reinforces the parameter dependence established in Section 4. Theta modulation should not, however, be converted into a mechanism for cognitive benefit: Neo and colleagues tested whether artificially restoring hippocampal theta after bilateral vestibular lesions would rescue the accompanying cognitive and emotional deficits, and found medial-septal stimulation restored theta power (at a higher frequency than normal) without repairing the measured behavioral deficits.[57] This does not establish that theta is irrelevant; it demonstrates that restoring one oscillatory feature is insufficient to replace the distributed consequences of normal vestibular input, and that vestibular influence on hippocampal function is unlikely to reduce to a single electrophysiological variable.

Cellular measures. Zheng and colleagues examined hippocampal cell proliferation and neurogenesis-related markers after galvanic vestibular stimulation in rats. Under a suprathreshold anesthetized protocol, GVS reduced BrdU-labeled cell proliferation bilaterally and produced a more restricted reduction in doublecortin labeling, while spatial-memory performance did not significantly differ from sham.[27] The direction matters: vestibular stimulation altered hippocampal biology, but the change was not an increase in neurogenesis and produced no corresponding behavioral change. Vestibular input can therefore alter processes associated with hippocampal plasticity without the presence of such an alteration indicating its behavioral meaning.

Reviews of vestibular influence on hippocampal theta describe multiple candidate routes by which vestibular information may reach the hippocampal formation and emphasize that the precise multisynaptic pathways remain incompletely resolved.[58] Taken together, the animal literature establishes substantial mechanistic plausibility across acetylcholine release, theta dynamics, synaptic-potentiation conditions, and proliferation-related measures[26, 27, 54–58], effects that are experimentally real but not uniform in direction or functional consequence. For SeaKing this justifies mechanistic hypotheses. It does not justify stating that natural multidimensional motion induces hippocampal LTP, increases neurogenesis, or improves memory in humans.

5.4 Human Hippocampal LTP and Neurogenesis Remain Unproven

Direct human evidence that vestibular stimulation induces hippocampal LTP or neurogenesis is absent from the canonical evidence base of this review. The point requires explicit statement

because the findings above could otherwise be assembled into an appealing but unsupported mechanistic narrative: human vestibular stimulation activates hippocampal regions,[25] rat passive rotation enhances electrically induced LTP through cholinergic mechanisms,[26] rat GVS alters proliferation and neurogenesis-related markers,[27] and reviews supply plausible connecting pathways.[58] None of these constitutes direct evidence of vestibular-induced human hippocampal LTP or neurogenesis.

The methods themselves impose the limit. Functional MRI measures hemodynamic responses associated with neural activity, not synaptic potentiation. Behavioral improvement can demonstrate learning or performance change without identifying LTP as the mechanism. Structural MRI cannot demonstrate the formation of new neurons. Animal histology and invasive electrophysiology answer mechanistic questions that cannot automatically be projected onto humans.

This matters particularly for SeaKing because the proposed exposure is further removed from those experiments than they are from each other. The LTP study involved electrically induced CA1 potentiation paired with controlled passive rotation;[26] the neurogenesis-related study involved suprathreshold galvanic stimulation under anesthesia.[27] Neither resembles awake human exposure to natural multidimensional vessel motion. The defensible statement is that vestibular input has access to human hippocampal networks and can modify hippocampal plasticity-related biology in animal models,[25–27, 54–58] while whether natural vestibular stimulation induces corresponding synaptic or cellular changes in the human hippocampus remains unknown.

Preserving this gap identifies a future research question rather than merely exercising caution. If later SeaKing studies demonstrate reproducible physiological responses, exposure-dependent changes, and relevant learning effects, increasingly direct measures of hippocampal-network function could then be justified. The mechanism should follow the evidence rather than being assumed at the outset.

5.5 Cognition Can Move in Positive, Null, or Adverse Directions

If vestibular information reaches learning-related systems, the next question is whether controlled vestibular stimulation changes human cognitive performance. It can, and the direction of change is not uniform.

On the positive side, subsensory galvanic stimulation applied during memory retrieval shortened reaction times for correctly recalled face-name associations without reducing accuracy;[59] because stimulation occurred during retrieval, this demonstrates modulation of memory performance rather than improved acquisition or consolidation. Hilliard and colleagues reported that subsensory noisy GVS improved overall spatial learning during a virtual-navigation paradigm in healthy young adults, though effects on different spatial representations depended on sex, task experience, information type, and baseline spatial-working-memory ability, so even within a positive experiment, enhancement was not uniform across participants or outcomes.[60] A subsequent preliminary study of noisy GVS during virtual spatial-memory encoding also reported better navigation performance in the stimulated group, with the caveats that it involved only 32 participants, used a between-subject design, and contained potential differences in virtual-reality motion sickness relevant to interpreting the performance effect.[61] Clinical evidence is likewise positive but context specific, since the four-week GVS recovery study discussed in 5.2 involved an injured and compensating nervous system rather than healthy cognition.[53]

Direct counterevidence rejects a generic enhancement model. Dilda and colleagues randomized 120 healthy participants to different GVS intensities and found suprathreshold stimulation increased errors on match-to-sample, perspective-taking, and Stroop performance, while other tasks were unaffected or changed in different directions and mental-rotation performance improved under both subthreshold and suprathreshold conditions.[62] The same vestibular intervention could therefore be associated with improvement, impairment, or no detectable effect depending on task and intensity. Hu and colleagues conducted three within-subject experiments examining visual working memory during sinusoidal GVS and found no significant enhancement in any of them despite varying

exposure duration and stimulus orientation[63], repeated nulls demonstrating that a plausible cognitive target does not necessarily respond. And Schöne and Mast found high-current GVS impaired working-memory span in healthy adults, while lower-current stimulation did not significantly affect executive performance and neither intensity materially altered several other executive measures.[64]

The supported conclusion is therefore narrower and more useful than generic enhancement: controlled vestibular stimulation can modulate selected aspects of human cognitive performance, but direction and magnitude depend on the stimulation paradigm, task, participant state, and outcome measured.[53, 59–64] The hippocampal and spatial-network literature provides a credible biological reason to investigate cognition and learning; it does not justify a directional assumption that natural motion will improve them. Future SeaKing cognitive experiments should therefore use prespecified outcomes and direction-neutral hypotheses, with improvement, absence of change, and impairment all remaining legitimate possible results.[25–27, 47–64]

6. Human Cognitive Modulation and Learned-Performance Acquisition

Section 5 establishes that vestibular stimulation can alter selected aspects of human cognitive performance. The next question is more demanding: can it influence the acquisition of learned performance?

The distinction is central. Faster retrieval during stimulation, better performance on a spatial task, or an altered perceptual threshold demonstrates cognitive or perceptual modulation. It does not demonstrate that the nervous system learned more effectively. Evidence for acquisition requires change across practice attributable to the intervention; evidence for retention additionally requires that the acquired change remain after the intervention or training period. If vestibular stimulation can alter the processing state in which a task is performed, it becomes biologically plausible that it could sometimes influence how rapidly a task is acquired, but plausibility is not demonstration, and the acquisition question must be examined directly.

6.1 Direct Human Evidence for Learned-Performance Acquisition

The most direct healthy-human evidence comes from Putman and colleagues.[4] Thirty-six healthy adults learned either a complex multisensory functional-mobility task involving navigation through an obstacle environment or a seated manual roll-control task. During repeated learning trials they received low-amplitude noisy GVS, a higher-amplitude random GVS condition, or sham; afterward they transitioned blindly to sham for an immediate post-stimulation phase.[4]

The functional-mobility result was positive: participants receiving nGVS demonstrated a significantly larger learning metric than sham, approximately 171% greater after adjustment for the experimental comparison.[4] The importance extends beyond improvement observed while stimulation was active, because performance gains remained detectable during the immediate post-stimulation testing period after transition to sham[4], distinguishing the result from a purely online effect existing only while current is delivered. The retention interval was nevertheless brief, approximately 15 minutes,[4] so the experiment demonstrates short post-stimulation persistence of acquired performance, not durable consolidation over days, weeks, or months.

The second task supplies an equally important boundary: the same study found no significant nGVS learning advantage on the seated manual-control task.[4] Even within a single protocol, vestibular stimulation enhanced acquisition in one task while failing to produce a significant benefit in another. That contrast materially strengthens the interpretation, since the positive result cannot be translated into the proposition that nGVS generally increases the capacity to learn. The functional-mobility paradigm required integration of vestibular, visual, proprioceptive, and motor information during whole-body movement, while the manual-control task involved a different sensorimotor computation; it is plausible that the former interacted more directly with vestibular processing, but that remains mechanistic inference rather than a demonstrated explanation.[4]

The strongest supported claim is therefore specific: low-amplitude noisy vestibular stimulation can enhance acquisition of a complex multisensory functional-mobility task in healthy adults under defined laboratory conditions, and the acquired performance can remain measurable briefly after stimulation ends.[4] The study does not establish durable consolidation, broad transfer, far generalization, or universal enhancement of sensorimotor learning. It is nevertheless a meaningful result: under at least one controlled human condition, vestibular stimulation altered the trajectory of learning itself rather than merely altering cognition or perception during exposure.

6.2 Online Facilitation Versus True Acquisition

Improved performance during stimulation can arise through mechanisms that do not require lasting learning. An intervention might temporarily increase signal detectability, alter arousal, change sensory weighting, or modify attention, improving performance only while present. If performance returns to baseline immediately when stimulation stops, the experiment demonstrates acute facilitation rather than retention of an acquired state.

Keywan and colleagues provide a direct vestibular example. Thirteen healthy adults underwent individually optimized imperceptible nGVS while vestibular roll-tilt discrimination thresholds were measured. Ten of thirteen showed improved thresholds during active stimulation, but when stimulation ended after approximately 30 minutes, thresholds returned toward baseline immediately with no meaningful aftereffect.[65]

This does not contradict the Putman result; the experiments measured different phenomena:

online facilitation: stimulation → improved performance during stimulation → return toward baseline when stimulation stops

acquisition with short retention: stimulation during repeated practice → altered learning trajectory → acquired performance remains detectable after stimulation stops

These patterns are experimentally different even though both produce superior performance during active stimulation. The retrieval findings in 5.5 illustrate the same point: subsensory GVS accelerated retrieval of associations acquired before stimulation, demonstrating retrieval-specific modulation rather than enhanced acquisition or consolidation.[59] Learning requires analysis of change across repeated practice rather than comparison of performance at a single time point.

For later SeaKing experiments this determines endpoint timing. An outcome measured only during motion can establish acute modulation; measurements after exposure ends are required to determine whether any change persists; and repeated testing across days is required to distinguish brief persistence from longer consolidation and retention. An acute SeaKing-associated change in cognition, perception, autonomic physiology, or task performance should not be described as learning merely because performance improves during motion. This does not diminish the importance of an online effect, which could itself be biologically informative and could identify the state in which later learning experiments should occur. It answers a different question.

6.3 Replication, Task Specificity, and Null Evidence

Three features constrain inference from the positive acquisition result. First, the effect was task specific within the original experiment, with no significant enhancement in the manual-control paradigm.[4]

Second, an independent five-day experiment produced a null intervention effect. Sherman and colleagues assigned 24 healthy adults to sham, auditory white noise, nGVS, or combined sensory-noise conditions during five daily sessions of a virtual lunar-rover navigation task. Participants improved through practice, but treatment condition did not significantly affect the rate of learning, and participants rated nGVS as more distracting than sham.[6] The study was relatively small and used a different task, so it cannot negate the Putman result. It does establish that repeated nGVS does not inevitably accelerate acquisition even during a complex operational task.

Third, the targeted evidence review identified no independent direct replication demonstrating the same functional-mobility acquisition advantage together with durable days-to-weeks retention. This absence should be interpreted carefully: lack of independent replication is not evidence that the original result is false, but confidence in its generality must remain lower than it would be after replication across investigators, tasks, and participant groups.

The evidence therefore forms a coherent pattern rather than a positive-versus-negative dispute, a complex functional-mobility task showed enhanced acquisition and brief retention during nGVS while a manual-control task in the same investigation did not;[4] a separate five-day operational task showed clear practice-related learning without nGVS acceleration;[6] and acute vestibular perceptual enhancement in another experiment disappeared immediately after stimulation ended.[65] The appropriate classification is supported but conditional: enough direct evidence to justify the hypothesis that vestibular stimulation can facilitate learning under selected conditions, not enough to claim it generally improves acquisition or that an effective paradigm has been defined for broad translation.

Table 2. Direct human evidence on vestibular stimulation and learned-performance acquisition. The positive result is task specific and has not been independently replicated.

Study	Intervention	Task	Result
Putman et al.[4]	Low-amplitude nGVS	Complex functional-mobility task (vs. manual-control task)	Acquisition enhanced ~171% on mobility task; no effect on manual-control task; advantage persisted ~15 min post-stimulation
Sherman et al.[6]	nGVS, 5 daily sessions	Simulated lunar-rover navigation	Clear practice-related learning; nGVS did not accelerate acquisition rate
Keywan et al.[65]	Individually optimized nGVS	Vestibular roll-tilt discrimination	Thresholds improved during stimulation; returned to baseline immediately after stimulation ended

This changes the purpose of future learning experiments. A SeaKing learning study would not be designed to confirm an established effect, but to test whether a new vestibular stimulus class reproduces, extends, or fails to reproduce an effect demonstrated under more controlled electrical-stimulation conditions. A positive result would be genuinely informative; so would a null result, particularly if motion parameters and acute physiological responses were measured well enough to identify the conditions under which learning did or did not change.

6.4 Natural Multidimensional Motion Remains an Untested Learning Intervention

The direct human learning evidence above involves galvanic vestibular stimulation, not natural vessel motion, and that distinction cannot be collapsed. As established in Section 4, natural motion produces multidimensional input whose statistical properties vary across axes, frequencies, and activities;[3] central processing differs between externally imposed and self-generated motion;[19] and simultaneous canal and otolith inputs can be integrated subadditively rather than as a simple sum.[7] The neural stimulus generated by natural multidimensional motion cannot be assumed to equal the stimulus generated by electrical vestibular modulation. The operational-learning null reinforces the boundary from the other direction: even within the nGVS modality, repeated stimulation did not improve acquisition in every complex task,[6] so it would be particularly unjustified to assume a substantially different intervention must produce a positive learning effect.

No direct verified human study in the present evidence base demonstrates that SeaKing-like natural multidimensional boat motion enhances acquisition, consolidation, retention, or generalization of learned performance. That absence defines a genuine empirical gap, and the rationale for

investigating it is substantial: human vestibular systems are capable of sensorimotor and perceptual plasticity, passive externally imposed motion can participate in vestibular learning, that plasticity depends on stimulation parameters and context, vestibular information reaches hippocampal and spatial-navigation systems, controlled stimulation can alter selected human cognitive outcomes in positive, null, or adverse directions, and controlled nGVS has enhanced acquisition of at least one complex human task.[1–7, 19, 21–23, 25, 34–41, 47–65]

The unresolved link is whether repeated natural multidimensional motion can influence learned-performance acquisition. That question should be tested rather than answered by analogy. A rational progression would first characterize the natural motion exposure and determine whether it produces reproducible physiological responses; repeated exposures could then determine whether those responses change with experience; and once the stimulus-response relationship is sufficiently characterized, a dedicated learning experiment could compare acquisition during defined motion conditions against an appropriate control. Such a study would need to distinguish performance during exposure from acquisition across trials, include post-exposure testing, use longer follow-up to determine whether any acquired advantage consolidates, and include separate untrained outcomes to address transfer.

The direct human literature has already crossed an important threshold: under at least one controlled condition, vestibular stimulation altered human learned-performance acquisition rather than merely engaging learning-related brain regions.[4] The effect is credible enough to justify further study, while the task-specific nulls and limited replication make direct testing essential rather than redundant.[4, 6, 65] The resulting SeaKing hypothesis is appropriately prospective: repeated natural multidimensional motion may create vestibular and physiological conditions capable of modifying human learning, but whether it enhances acquisition, how long any effect persists, and whether it generalizes beyond the trained task remain experimentally unresolved.

7. Consolidation, Retention, and the Temporal Durability of Learning

Demonstrating that vestibular stimulation can influence acquisition does not establish that the resulting change will endure. Performance can improve during training, remain altered for minutes after an intervention, stabilize across longer periods, or disappear once the facilitating condition is removed, different outcomes representing different levels of evidence.

The literature contains an important asymmetry. Long-lasting vestibular adaptation is established, with some forms persisting for months[24] and persistent behavioral effects observed after selected clinical vestibular interventions.[68, 69] Durable vestibular-enhanced trained-skill retention is not: the most direct healthy-human study demonstrating vestibular-enhanced acquisition assessed retention for only about 15 minutes.[4] That asymmetry defines one of the most important unresolved questions for any future SeaKing learning study.

7.1 Acquisition, Consolidation, and Retention Are Distinct Stages

What a participant can do at the end of a training session is not necessarily what will remain hours, days, or weeks later. Acquisition is improvement developing through practice. Consolidation refers to post-acquisition processes through which the learned state may stabilize, reorganize, become more resistant to interference, or change in later expression. Retention is demonstrated when the acquired capability remains measurable after an interval in which the participant is no longer continuing the original training episode.

Human motor learning provides direct experimental evidence for the separation. Walker and colleagues trained healthy adults on a sequential finger-tapping task and compared performance across intervals containing either wakefulness or sleep; performance improved substantially following the sleep-containing interval while an equivalent waking period did not produce the same offline improvement.[9] The point for this review is not that sleep is required for vestibular learning, but that performance can change after training has ended, so the state measured at the completion of acquisition cannot be assumed to represent the final retained memory. Neuroimaging extends

this: sleep-associated behavioral improvement was accompanied by changes in recruitment across distributed motor-memory networks,[66] indicating that post-training change can involve altered neural organization rather than preservation of the activity pattern present during practice. Reviews of human learning and motor-memory consolidation reach the same conceptual conclusion, and note that different types of learning stabilize, interfere, and generalize differently, which argues against treating all persistent performance changes as one universal consolidation process.[17, 67]

The distinction matters most when immediate performance is similar across interventions. Powell and colleagues randomized adults with acquired brain injury to two methods of learning assistive-technology skills; immediate post-training accuracy and fluency did not differ significantly, yet systematic instruction produced better 30-day maintenance and greater generalization to interactions with people other than the original instructor.[10] Measuring acquisition alone can therefore miss differences that emerge during retention, and a condition producing superior immediate performance may fail to maintain that advantage once the intervention is removed.

The direct vestibular evidence illustrates the current boundary. The nGVS functional-mobility advantage remained detectable during an immediate post-stimulation phase (stronger evidence than an online effect alone), but the retention interval lasted only approximately 15 minutes.[4] That establishes brief post-stimulation retention of learned performance, not long-term consolidation.

A future vestibular learning experiment seeking to demonstrate durable neuroplasticity would therefore require temporally separated measurements, giving SeaKing a clear evidentiary progression:

acquisition during exposure → immediate post-exposure retention → delayed retention → longer-term stability

A positive acquisition result would be important even if longer-term retention were unknown; a positive immediate-retention result would strengthen the inference by showing the effect does not depend entirely on continued motion; and delayed testing would determine whether the learned state survives meaningfully beyond the experimental session. The central rule is that these stages cannot be collapsed.

7.2 Brief Aftereffects Are Not Durable Retention

Temporal terminology matters in vestibular research because measurable changes can continue for minutes after stimulation without demonstrating durable learning. The Putman result and the Keywan result illustrate the two patterns respectively, acquisition whose advantage briefly outlives stimulation,[4] and acute perceptual facilitation that disappears the moment stimulation ends.[65] The second is not a contradiction of the first; they are different temporal phenomena, and neither establishes days-to-weeks retention.

The distinction prevents a common interpretive error: describing any post-stimulation response as evidence of consolidation. An aftereffect lasting minutes may reflect transient persistence within the stimulated system, whereas consolidation refers specifically to stabilization or transformation of a learned state after acquisition,[17, 67] on timescales that differ across forms of learning.[67]

This review therefore imposes no arbitrary threshold at which a response becomes long-term. Duration is reported explicitly: a 15-minute effect is described as approximately 15-minute retention, a 24-hour effect as 24-hour retention, a months-long effect as such. This matters for SeaKing because early studies may identify changes at several timescales simultaneously, an autonomic response returning to baseline immediately after motion stops, another endpoint remaining altered for tens of minutes, a baseline shift from repeated exposure detectable on a later day, and a trained behavioral task that may or may not retain an acquisition advantage. These should not be forced into one category merely because all represent change over time.

For an eventual SeaKing learning experiment, immediate post-motion testing would establish whether performance remains altered without concurrent motion; testing later the same day would

characterize short-term persistence; testing on subsequent days would address delayed retention; and longer follow-up would be required before claims of durable learning. The key distinction is temporal as well as mechanistic: an aftereffect demonstrates persistence of an effect, while durable retention demonstrates persistence of an acquired capability.

7.3 Durable Vestibular Adaptation Is Not Durable Trained-Skill Consolidation

Other vestibular-related changes do persist for long periods. Dilda and colleagues had ten healthy adults undergo repeated pseudorandom galvanic vestibular stimulation during dynamic posturography over 12 weeks. Early exposures substantially disrupted posture, but performance progressively adapted until participants maintained approximately baseline postural stability despite continued perturbation, and the adapted behavioral response was still detectable six months after training.[24] The adaptation was response specific, since the roll VOR response to the GVS perturbation did not undergo corresponding attenuation, which the authors interpreted as central sensory reweighting rather than uniform habituation of vestibular responsiveness.[24]

This is direct evidence that repeated vestibular perturbation can produce remarkably durable human behavioral adaptation. It does not establish durable consolidation of an independently trained skill. Participants learned to function in the presence of an experimentally unreliable vestibular signal, and the persistent outcome was adaptation of postural behavior to that perturbation, relevant to vestibular neuroplasticity, but not equivalent to vestibular stimulation enhancing long-term retention of a separate learned task.

Clinical studies supply further persistence examples with the same limitation. Gutkovich and colleagues randomized chronically seasickness-susceptible adults to a structured intervention combining GVS with inverse-phase rotatory-chair stimulation or to sham; the active group showed reduced severe seasickness, a shorter vestibular time constant, and lower subsequent use of motion-sickness medication and clinical services at three-month follow-up.[68] Because GVS and rotatory-chair stimulation were combined, the contribution of either component cannot be isolated, and the result establishes no general learning-enhancement effect. Schmidt and colleagues studied patients with chronic tactile extinction following right-hemisphere brain injury, finding repeated GVS produced improvements in tactile identification not seen during sham, with improvement still detectable at a planned follow-up approximately 2.8 months later and persistent improvement reported in five treated participants at a later second follow-up.[69] The persisting outcome was not a conventional vestibular endpoint, which is notable; but the study was small, the persistence mechanism was not measured directly, and the authors' discussion of LTP-like mechanisms should be treated as hypothesis rather than evidence that GVS induced human long-term potentiation.[69]

Controlled null evidence prevents these findings from becoming a generalized claim. Volkening and colleagues randomized 24 adults with right-hemisphere stroke and spatial neglect to repeated left-cathodal GVS, right-cathodal GVS, or sham alongside standard rehabilitation across 10 to 12 sessions, with outcomes measured immediately after treatment and again at two and four weeks. The trial did not demonstrate a reliable GVS advantage on neglect outcomes across follow-up.[70]

Two conclusions follow simultaneously. Vestibular-related plasticity is capable of lasting well beyond the stimulation period, with six-month postural adaptation in healthy adults[24] and months-long effects in selected clinical contexts.[68, 69] And persistence is endpoint and intervention specific: durable adaptation to vestibular perturbation does not establish durable consolidation of an unrelated trained skill, persistent improvement in a clinical deficit does not establish generalized learning enhancement, and a repeated vestibular intervention can produce no significant durable clinical advantage.[24, 68–70]

For SeaKing this means the two results cannot substitute for one another. SeaKing could produce durable vestibular or physiological adaptation without enhancing long-term skill retention, or influence acquisition without producing durable consolidation. These are different biological effects and should be analyzed independently.

7.4 The Direct Vestibular Long-Term Retention Gap

The canonical evidence base contains four relevant findings: nGVS can enhance acquisition of a complex functional-mobility task with performance persisting approximately 15 minutes after stimulation;[4] repeated vestibular perturbation can produce postural adaptation lasting six months;[24] vestibular-intervention-associated behavioral changes can persist for months in selected clinical conditions;[68, 69] and repeated GVS can fail to produce durable neuropsychological improvement.[70]

What it does not contain is direct evidence that vestibular stimulation enhances acquisition of a trained skill in healthy humans and that the resulting learning advantage remains superior to control across a delayed retention interval of days to weeks. That gap should remain explicit, and it cannot be filled by substitution: six-month postural adaptation is a different construct from consolidation of a separately trained skill;[24, 67] persistent clinical improvement is recovery or modulation of a deficit rather than enhanced acquisition and retention of a newly learned skill;[68, 69] nonvestibular motor-learning literature establishes general principles of consolidation rather than vestibular enhancement of it;[9, 17, 66, 67] and the approximately 15-minute retention phase cannot be relabeled as long-term memory.[4]

This is a high-value experimental question rather than a minor gap. The acquisition evidence establishes that a vestibular intervention can alter the learning trajectory of at least one complex human task,[4] and the durable-adaptation literature separately establishes that repeated vestibular exposure can produce changes persisting for months.[24] Together these make long-term learning retention biologically plausible without demonstrating it.

A staged design follows directly. A SeaKing learning experiment could first determine whether defined natural multidimensional motion alters acquisition relative to an appropriate control; immediate post-exposure testing could determine whether the effect survives removal of motion; a delayed test approximately 24 hours later would provide a first direct assessment of retained learning beyond the session; and additional follow-up across subsequent days or weeks could determine whether any advantage remains stable. Critically, a delayed test should assess the trained capability itself rather than whether a physiological biomarker remains altered. This permits four scientifically distinct outcomes, improved acquisition with no post-exposure retention; improved acquisition with brief but not delayed retention; improved acquisition with durable delayed retention; or no acquisition advantage despite measurable vestibular or physiological responses, each of which constrains the mechanism differently.

The same principle applies if repeated motion alters a participant's response across sessions. Progressive reduction, amplification, or reorganization of an acute physiological response could demonstrate adaptation, habituation, sensitization, or another exposure-dependent process, but would not establish memory consolidation unless a learned behavioral capability was also measured.

The literature therefore supports a precise progression, vestibular responsiveness → exposure-dependent adaptation → acquisition → immediate retention → delayed retention → transfer → functional consequence, in which not every experiment must traverse the entire sequence. Each stage can be tested separately, and a positive finding at an earlier stage remains scientifically important while later stages remain unresolved. The retention gap consequently strengthens rather than weakens the rationale for further research: the existing evidence establishes the biological capabilities on either side of the unanswered question, and whether they intersect to produce durable vestibular-enhanced retention of newly acquired human skill is a clear, falsifiable, and experimentally accessible question.

8. Transfer, Generalization, and Functional Meaning

Retention establishes that a learned change persists. It does not establish that the change extends beyond the condition in which it was acquired, and translational value depends increasingly on what

happens outside the trained task. A participant may improve substantially on the exact behavior practiced while showing little improvement on a similar untrained task; improvement may generalize to another task but not another environment; a skill demonstrated during structured testing may not be used spontaneously in everyday life; and even broad behavioral change may fail to produce clinically meaningful improvement in symptoms, participation, independence, or quality of life.

The human literature contains examples at each level. Training can generalize under defined conditions,[10, 31, 32, 71] and substantial trained-task improvement can also occur without measurable near transfer, far transfer, or real-world functional advantage.[13, 14, 30, 72, 73] Transfer is therefore neither rare nor expected. It is an independent outcome whose distance and functional meaning must be measured directly.

8.1 Near Transfer and Far Transfer Are Different Claims

Near transfer generally refers to improvement on an untrained task sharing important elements with the trained task, similar sensory information, movement dynamics, cognitive operations, or environmental demands. Far transfer requires extension to a more distinct task or capability. As the distance between trained and untrained conditions increases, the scientific claim becomes progressively stronger.

Motor-learning studies illustrate both outcomes. Schaefer and colleagues examined whether upper-extremity training after stroke transferred from a practiced task to a distinct untrained motor task; participants improved on the trained behavior, but transfer was limited.[30] Dipietro and colleagues provide the complementary result, with kinematic changes following robot-assisted rehabilitation expressed not only in the trained movement but also in an untrained movement performed within the same workspace.[31] These are not contradictory: transfer depends on the relationship between trained and untrained behaviors, so learning can generalize where sufficient common structure exists but should not be assumed merely because two tasks belong to the same broad functional category.[30, 31]

Powell and colleagues add generalization across people, systematic instruction produced better generalization when participants performed learned skills with individuals other than the original instructor.[10] The trained capability extended across an interpersonal context while remaining fundamentally the same learned skill, which is stronger than performance restricted to the original instructor but differs from far transfer to a fundamentally different ability. Modern controlled evidence makes the distinction sharpest: Zhong and colleagues conducted a preregistered, double-blind randomized trial of adaptive visuospatial working-memory training under different schedules, and longer training produced larger improvement on the practiced n-back task without a corresponding advantage on separate near-transfer executive-function measures or a far-transfer fluid-intelligence measure.[14] Substantial improvement in a trained cognitive task can coexist with null near and far transfer.

The hierarchy is therefore trained-task improvement → near transfer → far transfer, and these are not different labels for the same phenomenon. Motor-learning frameworks used in neurorehabilitation have long emphasized the same point: improvement through practice can be highly task specific, and gains on the practiced behavior should not be interpreted as generalized recovery.[18]

For SeaKing, the distance between endpoints must be specified rather than described generically as generalization. A SeaKing-associated improvement in one trained sensorimotor task would not establish generalized learning capacity; conversely, restricted transfer would not invalidate the original learning effect but would define its range. Direct evidence that vestibular-enhanced learning produces broad far transfer in humans remains insufficient; the literature supports the broader principle that transfer is possible and measurable but highly conditional.[10, 14, 30, 31]

8.2 Generalization Can Occur Under Defined Conditions

Learned changes can generalize across tasks, contexts, and people. Beyond the robot-assisted and instructional examples above,[10, 31] autism research provides directly relevant evidence.

Rosenblau and colleagues randomized high-functioning autistic adults to three months of social-cognitive training or a non-social active-control intervention. Participants receiving the social-cognitive intervention improved on both close and more distant social-cognitive generalization measures, with behavioral improvement accompanied by changes in functional activity and cortical thickness within prefrontal regions.[71] This establishes that adult autistic participants were capable of intervention-associated behavioral learning, that improvement was not restricted entirely to the exact trained material, and that measurable neuroimaging changes accompanied the behavioral effects. The boundaries remain: the population consisted of high-functioning autistic adults, the intervention targeted social cognition specifically, the findings establish no generalized neuroplastic enhancement across autism phenotypes or learning domains, and neuroimaging change does not independently establish clinical benefit.

Carruthers and colleagues provide a different form of evidence. Across repeated assessments of 248 autistic children, social-communication abilities demonstrated directional generalization across people and environments, including relationships from home measures to later school and unfamiliar research settings; generalization was not uniform, and the analysis concerned developmental cross-context relationships rather than demonstrating that the parent intervention caused every observed change.[32] Autistic children can therefore generalize capabilities across contexts, which rejects the opposite categorical claim that improvement in one environment necessarily transfers everywhere.

Cuneo and colleagues supply the counterexample at the level of language learning. In a large comparison involving autistic and neurotypical adults, autistic participants performed well learning novel word meanings that did not require flexible extension but showed greater difficulty when familiar meanings had to be extended flexibly into new contexts.[72] Acquisition and generalization dissociated within the same population, which matters for interpreting intervention studies, because failure of far transfer does not necessarily indicate failure to learn. It may indicate that the acquired representation is narrower, more context dependent, or less flexibly deployed.

Human learning can therefore generalize beyond the exact trained task, including across movements, people, cognitive tasks, and social contexts, with degree and direction depending on the learned material and the relationship between training and transfer conditions.[10, 31, 32, 71, 72] For SeaKing this makes generalization a legitimate empirical target: if future research demonstrates that natural multidimensional motion modifies acquisition or retention, it would be reasonable to test whether the effect remains restricted to the original task or extends to related untrained tasks, progressively increasing transfer distance. What would not be justified is assuming in advance that a demonstrated learning effect must generalize because the intervention is hypothesized to influence neuroplasticity broadly.

8.3 Training Gains May Fail to Transfer

A substantial literature demonstrates that trained-task improvement often exceeds transfer, including at ecologically meaningful endpoints. Beyond the stroke motor-training and n-back results above,[14, 30] Landínez-Martínez and Grisales-Aguirre examined whether adaptive working-memory training transferred to instrumental activities of daily living in adults with chronic stroke. Both adaptive training and the active control improved on several measures, but adaptive training did not outperform control on the overall real-world instrumental-activity outcome despite a domain-specific working-memory advantage.[13] Improvement in a targeted cognitive process did not automatically generate superior everyday functional independence.

Null transfer also appears where the theoretical rationale was strong. Fleming and colleagues randomized adults with traumatic brain injury to compensatory prospective-memory rehabilitation with or without additional metacognitive-skills training; adding the metacognitive component did not significantly improve the prespecified everyday prospective-memory or psychosocial-integration outcomes relative to the comparison approach.[73] This does not mean metacognitive training is universally ineffective. It demonstrates that adding a theoretically plausible learning component does not guarantee superiority at the level of everyday function.

The successful generalization studies remain essential to interpreting these nulls.[10, 31] The evidence supports no categorical conclusion that training fails to transfer; it establishes that transfer is an additional empirical achievement beyond acquisition and retention. A training intervention can therefore produce at least five distinguishable patterns: trained-task improvement without transfer; improvement with narrow near transfer; broader transfer to untrained tasks or contexts; transfer on structured measures without measurable everyday functional advantage; or transfer accompanied by meaningful functional improvement. These are not equivalent evidence of neuroplasticity.

For SeaKing the implication is sharp, because a mechanism proposed to affect neural plasticity could tempt investigators to expect broad transfer, yet even established learning interventions frequently produce task-specific improvement.[13, 14, 30, 72, 73] A positive SeaKing acquisition result should be interpreted first as evidence for the trained outcome, with transfer then tested using deliberately selected untrained endpoints rather than inferred from the acquisition effect. If no transfer occurs, the original learning effect remains valid but restricted; if near transfer occurs, the next question is whether the effect survives greater contextual or task distance; if far transfer occurs, ecological and functional consequences remain to be established separately. This staged logic prevents the concept of neuroplasticity from becoming a shortcut around actual translational measurement.

8.4 Spontaneous Real-World Behavior Is a Higher Translational Level

Demonstrating transfer on a structured test does not establish that a learned capability will be used spontaneously in everyday life. Experimental tasks are administered by investigators, performed under explicit instructions, and often structured to cue the participant to use the learned strategy, whereas real-world environments provide fewer prompts, more competing demands, greater variability, and different motivational and social contexts.

Autism research illustrates the distinction from both directions. The Carruthers longitudinal finding provides strong evidence against a categorical assumption that autistic learning remains trapped within one environment.[32] Yet systematic review of social-communication interventions in young autistic children found that generalization beyond the intervention context was inconsistently measured and inconsistently demonstrated,[74] so treatment gains could not be taken as proof of equivalent gains in everyday settings. A meta-analysis of group social-skills interventions for autistic youth found modest overall effects with null teacher-report outcomes, while stronger youth self-report effects appeared to reflect increased social knowledge more than demonstrated enactment of social behavior.[75]

That knowledge-performance distinction is fundamental. A participant may understand a strategy, demonstrate it when prompted, and perform it in a structured transfer test, yet still fail to deploy it spontaneously during ordinary life. The Cuneo language findings reinforce the same principle from another direction: a stored representation and the flexible deployment of that representation are not the same capability.[72] The chronic-stroke working-memory result provides the parallel outside autism, where domain-specific cognitive gains did not translate into superior overall instrumental activities of daily living.[13]

The translational progression therefore expands to trained performance → structured near transfer → structured far transfer → cross-context performance → spontaneous real-world enactment. The boundaries are not absolute, but as experimental structure decreases and ecological complexity increases, the outcome becomes more informative about actual daily function.

For SeaKing this has direct implications. If a later study demonstrates that motion-associated training improves performance on an untrained laboratory measure, that would represent evidence of transfer without establishing that participants spontaneously use the capability in school, work, home, or social settings. Claims involving everyday regulation, participation, social behavior, independence, or quality of life require ecologically meaningful endpoints. This applies to autism, where structured laboratory performance should not be treated as a proxy for spontaneous real-world behavior merely because both involve the same broad domain,[32, 72, 74, 75] and equally to

POTS, where improvement in a laboratory physiological or cognitive measure would not by itself establish improved ability to attend school, work, exercise, perform activities of daily living, or tolerate upright activity. The scientific burden increases as the claim becomes more meaningful. This is not a reason to undervalue controlled laboratory outcomes, which are essential for establishing causal effects and identifying mechanisms where confounding can be minimized; it is a reason to move from controlled evidence toward real-world evidence rather than substituting one for the other.

8.5 Clinical or Functional Benefit Must Be Measured Directly

At the highest level the question is no longer whether the nervous system changed, whether a task was learned, or whether that learning transferred, but whether the intervention produced an outcome that materially matters to the individual.

Several lines of evidence show why this step must be measured independently. The traumatic brain injury and chronic stroke trials above found that a plausible learning mechanism did not guarantee superiority on meaningful daily-life endpoints, and that improvement in a component cognitive process was not equivalent to superior functional independence.[13, 73] Powell and colleagues demonstrate that functional translation can nevertheless occur, with systematic instruction producing better 30-day maintenance and greater generalization even though immediate post-training performance did not significantly differ[10], immediate laboratory performance can therefore underestimate later functional differences just as laboratory improvement can overestimate them.

Neurobiological evidence requires the same discipline. In the sham-controlled PPPD trial, active tDCS produced treatment-related differences in regional cerebral blood flow involving temporal and hippocampal regions, yet was not significantly superior to sham on the primary dizziness outcome.[8] The neural change was measurable; clinical superiority was not. Systematic rehabilitation evidence reaches the same conclusion more broadly: meta-analysis of robot-assisted upper-limb therapy after stroke found statistically significant but generally small improvement in selected impairment-level measures without uniform translation into proportionately larger activity or functional effects,[76] and a Cochrane review of repetitive task training found modest functional benefits in selected outcomes with substantial dependence on the task, limb, endpoint, and durability assessed.[77]

These findings do not diminish the importance of biological or behavioral effects. They define what those effects mean. A biomarker may reveal mechanism; a trained-task improvement may establish learning; delayed performance may establish retention; an untrained task may establish transfer; a daily-life measure may establish ecological generalization. A clinically meaningful endpoint is required to establish benefit.

Early-stage SeaKing studies therefore need not prove the entire chain simultaneously. A Phase 1 discovery study can be scientifically successful by identifying reproducible relationships between quantified natural motion and physiological responses, with repeated sessions determining whether those relationships change with exposure history. Subsequent learning studies can determine whether a defined motion state influences acquisition and retention; transfer experiments can determine whether any learning advantage extends beyond the trained task; and only later efficacy studies should be expected to determine whether those effects produce meaningful improvement in symptoms, participation, everyday behavior, or functional capacity. A measurable Phase 1 physiological response does not need to be called therapeutic to be important, it establishes the biological responsiveness necessary to justify the next experiment. For eventual autism studies, meaningful outcomes might include ecologically valid measures of communication, participation, adaptive behavior, or regulation; for POTS, symptom burden, orthostatic tolerance, activity, or participation. The specific endpoints would require independent justification at the protocol stage.

The complete hierarchy developed across Sections 6 through 8 can therefore be summarized as:

acute modulation → acquisition → consolidation → retention → near transfer → far transfer → cross-context generalization → spontaneous real-world behavior → meaningful functional or clinical benefit

Every transition represents a stronger claim and requires its own evidence. The literature demonstrates that humans are capable of moving through this hierarchy under some training conditions[10, 31, 32, 71] and that progression can stop at virtually any stage.[8, 13, 14, 30, 72, 73, 76, 77] Natural multidimensional motion should therefore not be declared broadly neuroplastic or therapeutic because it produces a physiological response, nor should a narrow learning effect be inflated into generalized functional improvement. Clinical significance cannot be reverse-engineered from a biomarker.

9. Exposure History and Metaplasticity

Repeated exposure raises a question distinct from adaptation, habituation, acquisition, or retention: can the nervous system's previous activity alter its capacity to undergo a later plastic change? This phenomenon is termed metaplasticity, used narrowly here, not simply plasticity occurring more than once, nor a response that changes across repeated sessions, but specifically a change in the inducibility, magnitude, or direction of a later plastic response as a consequence of prior neural activity or prior plasticity induction.

Human vestibular studies clearly demonstrate exposure-history effects, including habituation, persistent sensory reweighting, changing postural responses, and differences between early and later exposures.[24, 46, 85–87] These show that the state entering a later vestibular session can differ from the state entering the first. They do not, by themselves, establish metaplasticity, because most do not measure whether a subsequent plasticity-inducing process has itself become easier, harder, larger, smaller, or directionally different. This distinction matters for SeaKing Phase 1: repeated-session data revealing stable responses, progressive adaptation, habituation, or no systematic change would all be scientifically meaningful outcomes, but calling them metaplasticity would require an additional experiment specifically designed to test altered inducibility of later plasticity.

9.1 Direct Human Evidence That Prior Activity Changes Later Plasticity Induction

Human motor-cortex experiments demonstrate the phenomenon directly. Jung and Ziemann manipulated cortical plasticity before participants underwent rapid thumb-flexion motor training; different paired-associative-stimulation priming conditions altered subsequent learning, with direction and magnitude depending on the sign and timing of the preceding cortical change.[78] Prior induced plasticity therefore influenced later learning rather than merely creating an independent earlier response. Murakami and colleagues demonstrated the principle more directly at the level of subsequent plasticity induction: without priming, intermittent theta-burst stimulation (TBS) increased corticospinal excitability while continuous TBS decreased it, but same-direction priming suppressed the later response and opposite-direction priming enhanced aspects of it.[79] The critical variable was not whether stimulation had occurred, but what had occurred beforehand. Bocci and colleagues extended the principle to human visual cortex, where preconditioning occipital excitability with transcranial direct-current stimulation changed both the strength and, under selected combinations, the direction of the response produced by later repetitive transcranial magnetic stimulation.[80]

Repeated-induction experiments add a spacing dimension. Goldsworthy and colleagues found spaced repetition of continuous TBS produced a larger and more prolonged reduction in corticospinal excitability than a single block.[83] Tse and colleagues found an even stronger interval-dependent effect: a fifteen-minute interval between two intermittent TBS blocks increased motor-evoked-potential amplitude for up to an hour, a five-minute interval decreased it, and a single block produced no significant group-level change[84], changing the interval between two otherwise related events changed the direction of the response, demonstrating why repeated stimulation cannot be treated as a simple cumulative dose. Individual variability remained substantial in both studies, with

only a subset of participants expressing the dominant group-level direction, which reinforces the need to distinguish a population-level history effect from a universal individual rule.[83, 84]

Across these experiments, metaplasticity can be conceptualized as prior neural activity or induced plasticity → altered response to a later plasticity-inducing event, fundamentally different from repeated exposure → progressively smaller or larger response, which may instead represent habituation, adaptation, sensitization, practice, or physiological drift. The distinction becomes evidence of metaplasticity only when an experiment shows that prior activity altered the capacity for a later plasticity-inducing event to produce change.

This evidentiary threshold matters because plasticity-induction paradigms are themselves variable. Boucher and colleagues examined intermittent TBS, continuous TBS, and sham across repeated visits and found intermittent TBS facilitation at the first visit matched by sham, continuous TBS failing to generate expected suppression, and no strong intra-individual reproducibility across visits.[81] A more recent sham-controlled study of modified continuous TBS likewise failed to reproduce the expected suppressive effect, with motor-evoked potentials increasing over time in both real and sham conditions.[82] These do not invalidate metaplasticity; the controlled priming experiments above demonstrate that prior neural state can causally alter later induction responses[78–80], but they establish a necessary boundary: unexplained session-to-session variability in a nominal plasticity measure cannot itself be relabeled metaplasticity.[81, 82] A metaplasticity claim requires a defined first manipulation, a defined subsequent plasticity-induction challenge, and evidence that the response to the second differs because of the first.

9.2 Prior Vestibular Exposure Alters Later Vestibular Response

Direct human vestibular evidence strongly supports the proposition that previous exposure influences later exposure, though the relevant experiments use terms like habituation, adaptation, postural learning, and sensory reweighting rather than metaplasticity, appropriately, since they measured changes in the vestibular or behavioral response itself rather than altered inducibility of a separate subsequent plasticity process.

As established in Section 7.3, Dilda and colleagues found postural responses to repeated GVS over 12 weeks progressively adapted to approximately baseline stability, with the adapted response detectable six months later while the roll VOR response to GVS did not show corresponding attenuation[24], behavioral control changed while another vestibular response remained comparatively stable. Clément and colleagues found a complementary pattern with repeated yaw angular-velocity steps across ten sessions followed for as long as eight months: VOR peak slow-phase velocity, time constant, and perceived self-rotation all progressively habituated during training, then recovered toward baseline over approximately one month, while other VOR measures did not change in the same manner.[85] Temporal structure influenced the magnitude of that habituation directly, in a subsequent experiment, reductions in selected VOR measures were larger when velocity steps occurred after nystagmus reversal than before it, so the timing of a new vestibular input relative to the ongoing response affected the magnitude of later habituation.[86]

Balter and colleagues provide an important counterexample to the assumption that longer intervals necessarily change the later response. In 40 healthy adults across five repeated GVS measurements, the galvanic-induced body-sway response declined after the first exposure and then remained comparatively stable, with absolute later responses not differing significantly across tested intervals from one day to two weeks.[46] Prior exposure clearly mattered (the initial decline demonstrates that), but within this protocol, interval duration across that range did not. Tjernström and colleagues add evidence that adaptation can accrue between sessions rather than only during them: postural responses to repeated galvanic perturbation over five consecutive days declined strongly day to day, and the lower response level reached after training remained evident at three-month follow-up.[87]

These studies collectively establish strong vestibular exposure-history effects: prior stimulation can alter later postural, reflexive, and perceptual responses; some changes persist for months; some

recover toward baseline; some are response specific; and some appear insensitive to the tested intersession interval.[24, 46, 85–87] Exposure history does not guarantee change, however. MacDougall and colleagues found substantial between-individual differences in three-dimensional eye movements during a fixed GVS stimulus, while each participant's response pattern remained comparatively stable across five repeated sessions, with within-person variability considerably smaller than between-person differences.[5] Only two participants contributed the repeated-session data, but the study provides an important conceptual counterweight: repeated vestibular exposure can reveal stable individual response structure rather than progressive adaptation.

None of these patterns establishes vestibular metaplasticity in the strict sense defined above. That would require showing that prior vestibular exposure changes the ability of a later plasticity-inducing intervention or learning event to produce plastic change, an open question.

9.3 Reproducibility, Adaptation, and Metaplasticity Must Remain Separate

A difference between session 1 and session 5 in a repeated-session study can arise for at least six reasons: a reproducible individual response; adaptation or habituation caused by prior exposure; practice or familiarity with the measurement procedure; nonspecific temporal or physiological fluctuation; measurement error; or altered inducibility of a later plastic response, which alone meets the stricter definition of metaplasticity. The MacDougall study illustrates the first, stability across sessions is scientifically informative but is not evidence of adaptation or neuroplasticity.[5] The Dilda and Tjernström studies illustrate the second, where the response itself changed systematically with exposure history.[24, 87]

Even systematic longitudinal improvement requires controls. Nooristani and colleagues randomized 28 healthy young adults to 30 minutes of noisy GVS or sham and assessed postural control before, immediately after, and one hour later; both active and sham groups improved relative to baseline with no significant between-group difference.[88] Apparent longitudinal improvement was therefore compatible with repeated testing or task familiarity rather than a specific nGVS aftereffect, a participant can improve over time without the experimental stimulus being the cause. The same issue appears in cortical plasticity research, where Boucher and colleagues found poor intra-individual reproducibility and sham-sensitive changes across repeated visits, and a subsequent study found a previously reported cortical-excitability effect failed under sham-controlled replication with no reliable person-level moderators.[81, 82] The governing rule: variability does not prove state dependence, and longitudinal change does not prove metaplasticity. A post hoc explanation can almost always be constructed for why one participant responded and another did not; such explanations become evidence only when the relevant state variable is measured prospectively and predicts the outcome under an appropriate design.

The categories therefore require separate operational tests, reproducibility (does the same stimulus produce a similar response when measured again?), adaptation or habituation (does the response itself change systematically with repeated exposure?), retention (does that changed response remain after an interval?), state dependence (does a prospectively measured biological or contextual state alter the response?), and metaplasticity (does prior activity or prior plasticity induction alter the capacity of a later plasticity-inducing event to produce change?), and each can occur without the others: a response may be highly reproducible with little adaptation,[5] may adapt substantially and remain altered months later,[24, 87] may show apparent improvement equally under active and sham conditions,[88] or may vary substantially from visit to visit without reliable state explanation.[81, 82]

9.4 Phase 1 Exposure-History Opportunity

This evidence creates a strong rationale for repeated-session analysis in Phase 1, since direct vestibular studies already demonstrate several scientifically distinct outcomes under repeated exposure: highly individual but comparatively reproducible responses,[5] durable central adaptation,[24] an early response decline followed by stabilization,[46] and progressive between-session

postural adaptation persisting months later.[87] Repeated natural-motion sessions can therefore provide substantially more information than repeated copies of a single acute exposure.

The primary Phase 1 question should remain bounded to whether physiological responses associated with quantified natural motion differ as a function of prior exposure history, and whether individual response patterns and baseline-exposure-recovery trajectories are reproducible or change systematically across sessions. Several outcomes would all be interpretable: a stable repeated response would support within-person reproducibility without demonstrating neuroplasticity;[5] a progressive reduction could be compatible with habituation or adaptation, for which vestibular studies provide direct precedent;[24, 46, 87] progressive reorganization in which some endpoints change while others remain stable would parallel the Dilda dissociation between postural recovery and persistent VOR response;[24] between-session accumulation of change, as Tjernström observed,[87] would justify modeling session number and exposure history rather than treating sessions as independent; and no systematic longitudinal change would still constrain the hypothesis and identify endpoints better interpreted as acute rather than adaptive. The Nooristani null is a direct caution here: improvement over repeated measurements cannot automatically be assigned to the vestibular intervention, so Phase 1 analyses should distinguish exposure-related trajectories from effects plausibly arising through repeated testing, familiarity, sensor drift, or ordinary physiological fluctuation.[88]

A useful Phase 1 model would retain session number, cumulative previous exposure, interval since the preceding session, characteristics of previous motion exposure, baseline physiological state, and prior response trajectory, tested against the acute response observed during the current exposure. Such analyses can answer whether history matters. They cannot answer whether prior natural motion makes the nervous system more or less capable of undergoing subsequent neuroplastic change, that stronger question requires a dedicated induction experiment, for example comparing acquisition of a defined learning task before and after different motion-exposure histories.

Human neuroscience establishes that prior neural activity can modify later plasticity induction;[78, 79, 83, 84] human vestibular research independently establishes that prior vestibular exposure can alter later vestibular and postural responses.[24, 46, 85–87] These two evidence streams make repeated-session history scientifically important while remaining distinct, and whether they intersect for natural motion remains an open, directly testable question.

10. Spacing, Timing, and the Structure of Repeated Exposure

Repeated stimulation is defined not only by what is delivered but by when successive exposures occur. A later stimulation event does not necessarily act on the same biological system that received the first, effects of the earlier event may still be developing, may have stabilized, may be recovering toward baseline, or may have altered the response to subsequent stimulation. Two protocols delivering the same nominal amount of stimulation can therefore produce substantially different outcomes if the intervals between exposures differ.

Direct human experiments outside the vestibular system demonstrate that spacing can alter the magnitude, persistence, and even direction of experimentally induced plasticity,[83, 84, 89–91] and vestibular studies independently show that rest periods and stimulus timing can influence adaptation or habituation, while another controlled GVS study found no meaningful difference across intersession intervals ranging from one day to two weeks once the response had stabilized.[21, 46, 86] The combined evidence establishes that timing matters without establishing a universal schedule: its effect is protocol specific and nonmonotonic. For SeaKing, no available evidence establishes that daily, weekly, or 7- to 14-day sessions are biologically optimal, so session spacing should remain an empirical design variable rather than being justified retrospectively from studies using different stimulation paradigms.

10.1 Spacing Can Alter Induced Plasticity, Including Its Direction

The clearest evidence comes from direct human cortical-stimulation experiments, several already introduced in 9.1. Beyond Goldsworthy's spaced-versus-single cTBS comparison[83] and Tse's opposite-direction result at five versus fifteen minutes,[84] Monte-Silva and colleagues found that two cathodal tDCS periods produced different effects depending on when the second stimulation occurred relative to the aftereffect of the first, repetition during an ongoing aftereffect could strengthen or prolong inhibition, while later repetition could attenuate or abolish the expected response.[89] The finding does not identify a transferable interval; it demonstrates that a second stimulation event interacts with the physiological state created by the first, so identical stimulation delivered at different times need not have identical consequences. A subsequent repeated anodal-tDCS study similarly found selected intersession timing could produce markedly prolonged excitability enhancement while other timing and pharmacological conditions attenuated, abolished, or reversed expected effects.[90] Bastani and Jaberzadeh extended the principle by manipulating repetition count and interval jointly, finding a 25-minute interval produced more persistent effects than a 5-minute interval in selected repeated conditions, with effects extending to approximately 24 hours in some cases.[91]

It would be tempting to read the Bastani-Jaberzadeh result as evidence that longer intervals are inherently superior. The broader evidence rejects that: Tse found a 15-minute interval produced facilitation while a 5-minute interval produced suppression,[84] and Monte-Silva found later repetition could weaken or abolish a response strengthened when stimulation occurred during an earlier physiological state.[89] The direction of the interval effect depends on the protocol and the state produced by the first intervention.[83, 84, 89–91] The numerical intervals themselves are not transferable to vessel-motion sessions, these interventions act through different physical inputs, anatomical pathways, intensities, and timescales. What transfers is structural: repeated exposure should not be modeled as dose 1 + dose 2 + dose 3, because the effect of dose 2 may depend on the state produced by dose 1. The more appropriate representation is first exposure → evolving biological state → later exposure acting on that altered state, and whether that interaction strengthens, weakens, reverses, or fails to alter a later response must be determined empirically.

10.2 Vestibular Timing and Rest Effects

Direct human vestibular experiments demonstrate the same principle. Mahfuz and colleagues found three five-minute VOR training blocks separated by twenty-minute rest intervals increased VOR gain by approximately 7.1%, against approximately 3.1% for a single block, greater than the single-block effect but substantially smaller than threefold, and the acquired gain did not decline significantly over the subsequent hour when consolidation intervals were included.[21] Repeated training is therefore not linearly additive, and rest between blocks was associated with stabilization of the resulting adaptation, though the study does not establish twenty minutes as an optimal interval or a schedule for natural-motion exposure.

The Clément timing result from 9.2 parallels this at a conceptual level: the relevant variable was not the interval measured by a clock but the timing of the next stimulus relative to the physiological state generated by the preceding one.[86] Balter's interval-insensitivity finding provides the direct counterexample, prior exposure clearly mattered because the initial response declined, but once that change occurred, varying the tested interval from one day to two weeks did not produce a detectable difference in the stabilized response.[46] The correct conclusion is not that timing always matters equally, but that its importance depends on the endpoint, stimulus, and stage of adaptation. Session interval should therefore be recorded and analyzed in SeaKing, with no current scientific basis for assuming a particular interval will maximize a desired effect.

10.3 Why Longer Spacing Is Not Inherently Better, and No Schedule Is Yet Justified

The intuitive assumption that more recovery time between sessions is progressively better is directly contradicted by the evidence above: more spacing does not automatically mean more plasticity, and

less spacing does not automatically mean greater cumulative stimulation; the relevant relationship is interaction, not accumulation.[84, 89] Vestibular evidence adds an independent boundary, since Balter found no significant difference in stabilized response across a one-day-to-two-week range;[46] this does not show that one day and two weeks are interchangeable for all forms of vestibular training, but it does show that a broad rule cannot be inferred. The same logic applies to daily versus weekly training: daily sessions might help one outcome because the effect of a prior session is still present when the next occurs, while that same overlap could attenuate or interfere with another process, and a longer interval could permit useful consolidation in one paradigm while allowing an adaptive state to decay in another.

Without direct data these remain hypotheses, and the literature does not justify claims that daily, weekly, or 7-to-14-day spacing is inherently superior, each assigns a directional biological effect to a schedule that has not been directly tested in the relevant intervention. This is especially true for SeaKing because natural motion acts continuously through sensory pathways over extended periods rather than through the brief pulses used in the cortical-stimulation literature.

No SeaKing session schedule is therefore established as optimal, and that conclusion should remain explicit because isolated findings could otherwise be selectively cited to justify almost any preferred interval, VOR adaptation benefiting from consolidation periods,[21] stability across a one-day-to-two-week GVS range,[46] spaced cortical stimulation strengthening effects,[83, 91] a different interval reversing that direction,[84] aftereffect-dependent interaction,[89] and nonmonotonic neuromodulatory intensity[44] were obtained using different interventions, neural targets, durations, and outcomes, and none establishes whether SeaKing sessions should occur daily, weekly, or at longer intervals. This does not prevent an initial protocol from selecting a practical schedule (every study requires predetermined session timing), but the schedule should be understood as an experimental sampling framework, not an established optimal neuroplasticity dose. Repeated measurements can then determine whether interval matters, retaining time since the preceding exposure, cumulative previous exposures, cumulative motion exposure, characteristics of the preceding session, baseline state, and the magnitude and direction of the previous response.

Phase 1 need not identify an optimal schedule to succeed. Similar responses across sessions would support reproducibility under the tested schedule; diminishing responses could support habituation or adaptation; larger responses would identify sensitization requiring investigation; a relationship between motion and physiology that changes with interval would make timing a dose-finding variable for later experiments; and no detectable interval association would still be informative, paralleling Balter's interval-insensitive range.[46] All of these outcomes are interpretable without assuming beforehand what spacing should accomplish, and later studies could deliberately randomize or compare schedules if Phase 1 identifies a plausible interval-dependent signal, a far stronger design than selecting one preferred schedule and explaining its supposed optimality afterward.

10.4 Dose and Spacing Interact With Protocol

The spacing literature reinforces a principle from Section 4: biological effect cannot be predicted from a single dose variable. Repeated-stimulation experiments demonstrate interactions among intensity, timing, repetition number, and temporal pattern, Monte-Silva showed identical cathodal tDCS exposures interacting differently depending on timing,[89] and Bastani and Jaberzadeh showed repetition count and interval jointly contributing to magnitude and persistence.[91]

Vagus nerve stimulation (VNS) provides an independent mechanistic example of the same nonlinear principle, extending the intensity finding introduced in Section 4.1. Beyond the nonmonotonic intensity-response relationship already noted,[44] Hays and colleagues compared VNS delivered in temporal association with successful rehabilitation trials after experimental stroke against an equivalent amount of VNS delivered two hours after training. Paired stimulation produced greater recovery, and delivering several-fold more VNS during training did not improve outcome and produced less benefit than the standard paired protocol[92], two independent failures of a simple dose model, since the same amount of stimulation produced different effects depending on when it

was delivered, and more stimulation did not produce more recovery. Addo and colleagues showed temporal structure can matter independently of total pulse count: several moderate pulse-frequency conditions produced similar task-relevant cortical reorganization, while burst stimulation and another pulse-count-matched pattern failed to produce the same enhancement.[93] These VNS studies are mechanistic analogies rather than evidence about vestibular motion, but they demonstrate how a neural intervention can depend jointly on intensity, timing, repetition, and temporal organization[44, 92, 93], the same principle the direct human cortical-stimulation literature establishes by different methods.[84, 89, 91]

For SeaKing, a natural-motion dose therefore cannot be represented adequately by a variable like "two hours on the water." Two sessions of equal duration could contain different distributions of heave, pitch, roll, translational acceleration, frequency content, temporal variability, and stillness, and the second session could occur while effects of the first remain present or after they have returned toward baseline. The relevant exposure is more realistically a multidimensional function, motion geometry × amplitude × frequency content × temporal variability × duration × repetition history × intersession interval × participant state, not implying every variable will ultimately matter, but identifying why the effective parameters should be discovered empirically rather than assumed, and why an early discovery study should avoid prematurely compressing exposure into one summary number. The appropriate research goal is therefore not to maximize dose, but to identify the relationship between stimulus structure, timing, and measurable response.

11. State Dependence, Baseline State, and Candidate Biomarkers

A further variable beyond stimulus parameters, exposure history, and temporal structure is the biological and behavioral state of the participant at the time an intervention is delivered. Human experimental neuroscience provides direct evidence that plasticity induction is state dependent, attention, recent motor activation, sleep-wake history, acute stress, and circulating cortisol can each alter the magnitude or direction of subsequently induced cortical plasticity, and the same nominal protocol can produce substantially different outcomes under different physiological states.[94–99]

This is highly relevant to repeated vestibular research, but risks overinterpretation: state dependence existing does not mean any convenient physiological signal identifies the relevant state. The current evidence base does not validate heart-rate variability as a biomarker of a human "neuroplasticity window," nor does pupil dilation constitute specific evidence of locus-coeruleus norepinephrine activity. For SeaKing, the opportunity is prospective, not diagnostic: baseline physiology, exposure history, and behavioral context can be measured and tested against subsequent responses, and states that reliably predict later effects may become useful candidate stratifiers, a relationship to be demonstrated, not assumed.

11.1 Human Plasticity Is State Dependent

Attention provides one of the clearest causal examples. Kamke and colleagues manipulated visual-spatial attention while healthy participants underwent paired-associative stimulation designed to produce either LTP-like or LTD-like motor-cortex plasticity; directing attention toward the region associated with the stimulated hand enhanced the LTP-like response while reducing the LTD-like response relative to attention directed elsewhere.[95] The same nominal induction protocol therefore produced different effects according to where participants directed attention, establishing not that attention to vestibular motion will enhance vestibular plasticity specifically, but the general principle that behavioral state during stimulation can modify induced plasticity.

Recent physical activity can also alter a later induction response. Goldsworthy and colleagues found continuous TBS produced comparatively consistent LTD-like suppression when participants remained relaxed beforehand, while a preceding voluntary muscle contraction substantially increased response variability and altered the expected suppressive effect.[96] Sleep-wake state produces still stronger effects. Kuhn and colleagues found sleep deprivation increased cortical excitability, partially occluded the LTP-like response normally produced by paired-associative

stimulation, and impaired declarative-memory encoding.[97] Salehinejad and colleagues extended this: sleep deprivation increased cortical facilitation, reduced inhibition, abolished the expected LTP-like aftereffect of anodal tDCS, and converted the normally LTD-like effect of cathodal tDCS into an excitatory response, with motor-sequence learning, working memory, and attention also impaired.[98] This is particularly strong evidence of state dependence because the effect was not merely quantitative, under sleep deprivation, the same nominal intervention produced an effect in the opposite direction from the rested condition.

Hormonal and stress state contribute as well. Sale and colleagues found PAS-induced motor-cortex plasticity was more readily expressed when circulating cortisol was lower, while experimentally increasing cortisol reduced the effectiveness of the induction protocol[99], a causal relationship between endocrine state and plasticity inducibility within that paradigm. Infortuna and colleagues exposed healthy adults to a socially evaluated cold-pressor stressor before testing a cortical metaplasticity protocol combining tDCS priming with rTMS, and acute stress altered the subsequent cortical metaplastic response relative to control.[94]

Attention, recent motor activation, sleep-wake history, acute stress, and endocrine state can therefore each influence human plasticity induction,[94–99] without identifying one universal variable that determines whether the brain is "plastic." The reliability literature from Section 9 provides an important counterweight: Frieske and colleagues failed to reproduce the expected suppressive effect of a modified continuous TBS protocol under sham control, and candidate moderators including sex, age, and time of day did not reliably distinguish expected from paradoxical responders.[82] This does not contradict the causal state manipulations above; it establishes that unexplained variability cannot be converted retrospectively into evidence of state dependence simply by searching for a plausible moderator after the fact. The strongest conclusion is therefore both positive and restrictive: human plasticity induction is demonstrably state dependent, but the relevant state variables must be manipulated or measured prospectively rather than inferred from response variability alone.[82, 94–99]

11.2 Vestibular Response Expression Can Depend on Context and Prior State

Direct vestibular research demonstrates that response expression can depend on context and prior exposure history. As established in Section 4.2, Shelhamer and colleagues trained healthy adults to express different VOR gains linked to gaze direction, showing that an adapted vestibular response need not be expressed identically across contexts[45], direct evidence, from three participants, of learned contextual expression. Prior vestibular history changes the state entering a later session more broadly: the Dilda postural-adaptation and Clément habituation results from Sections 7 and 9 both show that a participant exposed to the same nominal stimulus after repeated training is not physiologically equivalent to that participant at first exposure.[24, 85] At the same time, MacDougall and colleagues found repeated response patterns within individuals were substantially more consistent than the differences between individuals,[5] demonstrating that between-person heterogeneity can coexist with within-person reproducibility.

These findings identify three distinct forms of state or context dependence that should not be collapsed into one mechanism: learned contextual expression, in which the response varies with a trained cue;[45] exposure-history dependence, in which previous stimulation changes later responses;[24, 85] and stable within-person response structure, in which individuals differ but a person's own response remains comparatively reproducible.[5] Context-specific VOR expression is not necessarily evidence of systemic physiological state; long-term habituation is not automatically metaplasticity; stable individual responses are not automatically evidence of neuroplasticity. What these studies collectively establish is that the effective vestibular response cannot always be predicted from the physical stimulus alone; the participant's history and context can matter[5, 24, 45, 85], which is directly relevant to natural multidimensional motion, where identical motion is unlikely to occur under identical behavioral and physiological conditions across sessions.

11.3 Candidate SeaKing Baseline-State Associations

Two evidence streams justify asking whether the state in which a participant begins a SeaKing exposure predicts what happens during it: prospectively manipulated states such as stress, sleep deprivation, attention, and endocrine condition alter later plasticity induction,[94–99] and repeated vestibular experiments show individual responses can contain reproducible structure while exposure history alters later behavior.[5, 24, 45, 85] Neither stream identifies which SeaKing baseline variables will prove useful, candidates could include autonomic physiology, pupil characteristics, recent sleep or fatigue, previous exposure history, motion-sickness symptoms, or subjective arousal, with an initially exploratory or stratifying rather than diagnostic role.

The correct question is not "is this participant in a neuroplasticity window?" but "does a prospectively measured baseline state predict the magnitude, direction, reproducibility, or longitudinal evolution of the response that follows?" A state-response association, a moderator effect on longitudinal trajectory, and independent replication would each strengthen confidence progressively, but none of these stages alone would establish that the baseline measure identifies enhanced capacity for neuroplasticity. The reliability literature from Section 9 provides an additional safeguard: poor test-retest reliability, strong sham/time effects, and moderators that fail to explain response patterns under controlled replication all demonstrate why apparent subgroups should not be defined solely by post hoc analysis of noisy physiological data.[81, 82]

Repeated within-person measurement is particularly valuable here, since a single measurement cannot easily distinguish a stable participant characteristic from a transient state, while multiple sessions allow asking whether changes in a participant's own baseline state associate with changes in that same participant's response, a question partially controlling for fixed individual differences. Phase 1 can therefore legitimately search for candidate baseline-response relationships. It cannot legitimately designate those relationships as biomarkers of plasticity until they predict an independently measured plasticity or learning outcome.

11.4 HRV Is Not a Validated Neuroplasticity-Window Biomarker

Heart-rate variability is relevant to SeaKing because it can provide useful information about autonomic physiology and within-person responses during natural motion. That legitimate value must be separated from a stronger claim the current evidence does not support: no evidence in the frozen Review 04 base validates HRV as a biomarker of a human neuroplasticity window or a direct measure of cortical plasticity gating. This does not mean HRV lacks scientific value, cannot correlate with states that influence learning, or could never be linked to a plasticity outcome in later SeaKing research, it means that link has not already been established sufficiently to use HRV prospectively as proof the nervous system has entered a plasticity-enhancing state.

The state-dependence evidence above explains why such a biomarker would be attractive, sleep, stress, recent activation, attention, and endocrine conditions can alter induced plasticity,[94–99] and a noninvasive signal reliably indexing those conditions would be useful. But a useful biomarker requires validation against the target construct. HRV is an autonomic measure; neuroplasticity is not an autonomic variable. A correlation between HRV and motion exposure would establish an autonomic response to motion, and even a repeatable HRV pattern across sessions would establish physiological reproducibility, not plasticity, precisely the lesson of the MacDougall ocular-response finding, where reproducibility did not turn the response into a biomarker of learning.[5] Likewise, measurable physiological or neural change cannot substitute for an independent functional endpoint, as the sham-controlled PPPD study demonstrates directly: treatment-related cerebral-perfusion differences coexisted with no primary-outcome advantage over sham.[8] Biological measurability does not establish behavioral meaning.

For Phase 1, HRV can appropriately quantify autonomic responses during motion, contribute to baseline-exposure-recovery modeling, be tested for within-person reproducibility, and be retained as a candidate predictor or stratifier. What it cannot currently do is establish that a participant is in a state of enhanced neural plasticity, that would require prospective linkage between the HRV pattern

and an independent plasticity-related endpoint such as acquisition or retention. Until such evidence exists, the appropriate language is candidate state correlate, not neuroplasticity-window biomarker.

11.5 Pupil Response Does Not Prove LC-Norepinephrine Engagement

Pupil diameter is influenced by multiple interacting autonomic and central processes and has been used as a noninvasive correlate of arousal and neuromodulatory state. Because locus-coeruleus norepinephrine (LC-NE) signaling is strongly implicated in arousal and learning, pupil dilation has sometimes been treated as indirect evidence of LC-NE engagement. Human vagus-nerve-stimulation studies demonstrate why that inference requires caution.

Sharon and colleagues delivered brief transcutaneous vagus nerve stimulation (taVNS) bursts to healthy adults while measuring pupillary and EEG responses; active stimulation produced transient pupil dilation and attenuation of EEG alpha activity relative to control.[100] Lloyd and colleagues attempted replication and reproduced the pupil-dilation effect but not the alpha-power reduction[101], partial rather than complete replication of the original signature. These positive findings are meaningful but do not directly measure LC firing or norepinephrine release.[100, 101]

Other controlled studies provide substantial counterevidence against treating pupil response as a universal or specific taVNS biomarker. Keute and colleagues found no significant effect of active stimulation on resting pupil size, event-related dilation, or behavioral performance during rest and an auditory oddball task.[102] D'Agostini and colleagues tested continuous taVNS against sham in 66 healthy adults using pupillometry, salivary alpha-amylase, and cortisol, finding no significant effect on any tested marker.[103] Ludwig and colleagues paired brief real or sham stimulation bursts with memory encoding and found both conditions associated with pupil dilation and improving memory performance over time, with no clear taVNS-specific advantage for either outcome[104], direct demonstration that phasic sensory events and sham stimulation themselves can produce pupil responses.

The combined evidence supports a nuanced conclusion: brief taVNS can produce reproducible pupil dilation under some protocols,[100, 101] but pupil dilation is neither universally produced by taVNS nor specific evidence that LC-norepinephrine signaling has been engaged.[102–104] These studies involve taVNS rather than vestibular motion and cannot determine whether natural motion affects pupil behavior; their relevance to SeaKing is methodological. If pupil diameter changes reproducibly during natural motion, that establishes a pupillary response; if it correlates with motion parameters, it may become a useful physiological signature; if baseline pupil characteristics predict later response, they may become candidate state variables. None of those findings alone would prove LC-NE activation, and this boundary matters because mechanistic narratives can become circular, if motion causes pupil dilation, pupil dilation is assumed to prove LC activation, and LC activation is then invoked to explain neuroplasticity: the same indirect observation has effectively been used to establish both the mechanism and the outcome the evidence does not support that chain. Pupillometry remains highly useful precisely because it can be measured continuously and noninvasively; its value does not depend on treating it as anatomically specific.

11.6 A "Plasticity Window" Requires Prospective Validation

The concept of a plasticity window is scientifically plausible in general terms: human experiments clearly show the same plasticity-inducing intervention can behave differently according to attention, prior activation, sleep, stress, and endocrine state,[94–99] so it is reasonable to hypothesize that some measurable states may be more permissive than others for particular forms of learning. What has not been established is a validated SeaKing biomarker that identifies such a state, and neither a reproducible physiological response nor an association with motion is sufficient to establish one.

Consider a hypothetical Phase 1 finding in which a particular combination of HRV and pupil behavior repeatedly appears during a specific motion profile. That the state is measurable, associated with the motion condition, reproducible within participants, and worth investigating further would all be justified. That the state represents a window of enhanced neuroplasticity would not be, validating

that claim requires the candidate state to predict an independent plasticity-related outcome. A prospective pathway would need to proceed in stages: identify reproducible candidate states associated with motion, test whether a dedicated learning experiment shows different acquisition when training occurs during or following those states, test whether any acquisition advantage is retained on delayed testing, and replicate the state-performance relationship in an independent sample. Only after that sequence would it become reasonable to discuss a physiological pattern as a predictive plasticity biomarker.

The pupillometry literature demonstrates why this progression is necessary, a pupil effect can replicate under one phasic protocol,[100, 101] disappear under another,[102, 103] or occur similarly under active and sham conditions.[104] The cortical-stimulation reliability literature makes the same point, since apparent person-level or state-level response patterns can fail under sham-controlled replication.[81, 82] And the PPPD study demonstrates that an objective neural difference can coexist with a null primary clinical comparison.[8] Physiological responsiveness does not guarantee mechanistic specificity, and biological measurability does not establish behavioral meaning.

The evidentiary sequence for a SeaKing plasticity-window claim is therefore: motion-associated physiological state → reproducibility → prospective prediction of independent acquisition or plasticity → delayed retention → replication. This standard is demanding, but Phase 1 does not need to prove the entire chain, its repeated within-person design can provide the first step by determining whether natural motion produces identifiable states and whether those states vary systematically with motion, history, and baseline condition. If no reproducible state emerges, the hypothesis narrows; if reproducible states emerge but fail to predict later learning, their value remains physiological rather than neuroplastic; if a state predicts acquisition but not retention, it may represent acute facilitation rather than a durable window; if it predicts both across independent samples, the mechanistic argument becomes substantially stronger. This prospective structure prevents biomarkers from being defined by the outcome they are later invoked to explain.

Human neuroplasticity is demonstrably state dependent, and human vestibular responses can depend on context and prior exposure history. These findings justify prospective measurement of SeaKing baseline and exposure states. They do not presently validate HRV, pupil diameter, or any other Phase 1 physiological signal as a biomarker of enhanced neuroplasticity. Phase 1 can identify candidate states; later experiments must determine what those states mean.

12. Neuromodulatory Gating of Experience-Dependent Plasticity

Vestibular plasticity depends on stimulus characteristics, exposure history, temporal spacing, and biological state. A complementary mechanistic question is how neural activity occurring during an experience becomes selectively reinforced rather than producing indiscriminate change throughout the nervous system. Neuromodulatory systems provide one experimentally established solution: direct animal studies show that basal-forebrain cholinergic pathway activation or vagus nerve stimulation (VNS) can promote experience-specific cortical reorganization when neuromodulatory activity is temporally associated with selected sensory or motor events,[105–107] with mechanistic experiments further implicating cholinergic, noradrenergic, and serotonergic systems in selected forms of VNS-directed plasticity.[109–113] The effect is not simply proportional to stimulation amount or intensity, VNS-directed plasticity depends on intensity, timing relative to behavior, pairing interval, and temporal structure.[44, 92, 93, 120]

Human taVNS studies provide partial translational support: taVNS has improved acquisition or delayed memory under some protocols,[20, 116, 117] while other controlled studies have found behavioral null effects, dissociations between neural change and performance, or no memory benefit.[104, 118, 119] The human evidence supports conditional neuromodulatory effects rather than generic enhancement. For SeaKing, this literature is mechanistic and hypothesis-generating: natural multidimensional vestibular motion has not been demonstrated to recruit the same cholinergic, noradrenergic, serotonergic, or locus-coeruleus mechanisms established for implanted VNS, and natural motion and VNS act initially through different peripheral pathways.[3, 7, 19, 105–

107, 112, 113] The VNS literature can establish that temporally structured neuromodulatory activity is capable of gating experience-specific plasticity. It cannot be used to assert that natural motion already produces such gating.

12.1 Neuromodulators Can Gate Experience-Specific Cortical Plasticity

A central challenge for any theory of neuroplasticity is selectivity: plasticity would be poorly targeted if every rise in global arousal or neuromodulator release strengthened all active neural representations equally. Kilgard and Merzenich provided a foundational demonstration using the basal forebrain: in adult rats, repeated electrical activation of the nucleus basalis paired with a selected auditory stimulus produced large, progressive, stimulus-specific reorganization of auditory cortical receptive fields, while exposure to the sensory stimulus without equivalent neuromodulatory pairing did not.[105] The resulting reorganization was related to the sensory information present during the neuromodulatory event, establishing that neuromodulatory activation can reinforce neural representations associated with concurrent experience rather than producing indiscriminate cortical change.

A different intervention reaches a similar result. Porter and colleagues repeatedly paired implanted VNS with execution of a selected forelimb movement in rats; cortical representation increased preferentially for the paired movement rather than expanding nonspecifically throughout the motor map.[106] Bowles and colleagues examined this with greater circuit-level resolution: VNS paired with skilled motor practice enhanced learning and altered task-relevant cortical activity, with cholinergic reinforcement contributing to selective modification of circuits associated with the practiced behavior, even though VNS initially produced broader neural effects.[107] Together these experiments demonstrate that neuromodulatory activity can function as a gating or reinforcement signal changing the probability that activity around a behavior or sensory event becomes incorporated into longer-lasting cortical reorganization.[105–107]

Four restrictions are essential: these are animal experiments; nucleus basalis stimulation and cervical VNS are not interchangeable interventions; the studies do not establish a generic whole-brain "plastic state," since the effects were selective and related to specific events; and greater neuromodulatory activation does not necessarily imply greater learning. Carroll and colleagues tested VNS paired with training across several tasks in healthy rats, including skilled motor learning and sensory discrimination, and VNS failed to improve learning rate or performance across the tested tasks[108], important because it demonstrates that even an intervention with well-established capacity to direct cortical plasticity does not automatically improve every form of learning. The correct principle is therefore that neuromodulatory systems can gate experience-specific cortical plasticity, but successful recruitment of a neuromodulatory pathway does not guarantee generalized or task-specific behavioral enhancement under every condition.[105–108] Even if future experiments demonstrate that natural motion engages a neuromodulatory system, that finding alone would identify a potential mechanism, not established improved learning; behavioral consequences would still require independent measurement.

12.2 Paired VNS and Nucleus-Basalis Evidence

The VNS literature provides an unusually detailed mechanistic model because investigators have manipulated both the timing of neuromodulatory stimulation and the neurotransmitter systems required for the resulting cortical changes. Evidence for cholinergic involvement is particularly strong: Hulsey and colleagues selectively depleted basal-forebrain cholinergic innervation before pairing VNS with motor training, and while paired VNS produced expected reorganization in intact animals, cholinergic depletion prevented that VNS-directed map reorganization[111], intact cholinergic innervation was necessary within this paradigm. Noradrenergic signaling is also implicated: Hulsey and colleagues directly recorded LC neurons during implanted VNS and found increased activity whose magnitude and temporal profile depended on stimulation parameters,[112] while Roosevelt and colleagues measured extracellular norepinephrine during graded VNS and found intensity- and region-dependent increases, a 0.5-mA condition increased hippocampal

norepinephrine while 1.0 mA increased it in both hippocampus and cortex, with the lower tested intensity not producing equivalent changes.[113] These findings reinforce a theme developed throughout this review: nominal stimulation does not create one uniform biological response, and the response depends on parameters and neural target.

Causal depletion studies strengthen the mechanistic inference further. Selective depletion of either norepinephrine or serotonin prevented the expected VNS-directed motor-cortex reorganization that intact animals showed,[109] and local α2-adrenergic receptor signaling within motor cortex was required for VNS-induced motor-map plasticity in a separate rat paradigm.[110] Necessity must be interpreted carefully, however: Morrison and colleagues administered clinically relevant drugs affecting cholinergic, adrenergic, or serotonergic systems during VNS-paired training, and none significantly blocked the expected motor-map plasticity[114], profound pathway removal and partial pharmacological alteration are fundamentally different manipulations, so this does not negate the depletion studies. Brougher and colleagues similarly found that depletion of cortical dopamine did not prevent VNS-mediated task-relevant motor-map reorganization,[115] demonstrating that not every neuromodulator involved in ordinary motor learning is necessarily required for VNS-directed plasticity.

Together these experiments support a distributed but selective neuromodulatory model, paired VNS can recruit the locus coeruleus and increase norepinephrine;[112, 113] cholinergic signaling is necessary for selected forms of VNS-directed motor-cortical plasticity;[107, 111] noradrenergic and serotonergic systems can also be necessary under defined conditions;[109, 110] cortical dopamine was dispensable for one VNS-directed effect;[115] and partial pharmacological modulation of several implicated systems did not reproduce the effects of profound depletion.[114] This is considerably more nuanced than saying vagal activation releases acetylcholine and norepinephrine and therefore causes neuroplasticity. The evidence instead supports a timing-sensitive, pathway-specific, task-related process in which several neuromodulatory systems contribute to selective reinforcement of active neural circuitry.[105–115] For SeaKing, if natural motion were eventually shown to recruit analogous neuromodulatory processes at behaviorally relevant times, such mechanisms could provide one plausible route through which a motion-associated physiological state interacts with learning. That mechanistic bridge has not yet been demonstrated.

12.3 Human taVNS Learning and Memory Evidence Is Mixed

Human taVNS research provides a translational test of whether noninvasive vagal stimulation can influence learning and memory, and the evidence is mixed. Thakkar and colleagues randomized healthy young adults to active taVNS or several control conditions while participants learned novel Hebrew letter-sound relationships across five training lessons; active taVNS enhanced acquisition on selected learning measures relative to controls.[20] This is direct human evidence that taVNS can facilitate acquisition under a defined language-learning protocol, important because it involved learning across training rather than transient performance during stimulation, without establishing generalized enhancement, identifying the responsible neurotransmitter, demonstrating broad transfer, or showing equivalence to implanted cervical VNS. More recent episodic-memory evidence is also positive: Mary and colleagues found 24-hour free recall significantly greater following active taVNS overall in a randomized within-subject crossover design across 90 healthy young adults, with the benefit not uniquely restricted to one specific memory stage.[117]

Ventura-Bort and colleagues provide a more complex result across two studies examining emotional memory: neural effects associated with taVNS were reproducible under selected conditions, but behavioral memory enhancement was not consistent across both experiments, with one study reproducing electrophysiological effects without a corresponding memory-performance benefit and the other showing improved longer-term memory.[116] This neural-behavioral dissociation is informative, a measurable neural effect associated with taVNS does not guarantee measurable behavioral enhancement. Other controlled studies are null: Mertens and colleagues found no significant improvement in immediate verbal recall or delayed recognition memory from tVNS in younger and older healthy adults.[119] Nakagawa and Osu found motor performance on a dexterous

two-ball rotation task improved similarly whether participants received active taVNS or sham, even though motor-cortex representation expanded to a greater extent in the active condition[118], direct demonstration of neural plasticity-related change without additional behavioral improvement, since the cortical-mapping result does not become evidence of enhanced learning merely because the map changed. Ludwig and colleagues, discussed in Section 11.5, provide an additional control: brief real and sham stimulation bursts during memory encoding both produced pupil dilation and improving memory performance over time, without a clear taVNS-specific advantage.[104]

The combined human evidence cannot support the proposition that taVNS is a generic learning or memory enhancer. It supports a narrower conclusion: taVNS can influence human learning, memory, and neural-plasticity-related measures under selected protocols, but positive behavioral effects are task and protocol dependent and coexist with controlled null findings[20, 104, 116–119], precisely the pattern expected from a state-dependent neuromodulatory intervention rather than a universally effective cognitive enhancer. The human results also weaken any attempt to infer behavioral benefit from mechanistic biomarkers alone: a cortical map can expand without additional performance improvement,[118] physiological responses can occur during both real and sham stimulation,[104] and neural effects can replicate even when behavioral memory benefits do not.[116] For SeaKing, these findings provide translational precedent for the possibility that an experimentally induced neuromodulatory state can sometimes influence learning. They do not establish that natural vestibular motion generates the same state.

12.4 Timing and Intensity Are Nonlinear

The mechanistic VNS literature provides direct evidence against a simple "more stimulation equals more plasticity" model, extending the dose-response findings introduced in Sections 4 and 10. As established there, moderate-intensity VNS enhanced task-relevant motor-cortex reorganization while lower and higher intensities did not,[44] and VNS paired with successful rehabilitation events produced greater recovery than the same amount delivered two hours after training, with substantially more VNS during training failing to improve outcome.[92] Borland and colleagues add a further dimension: varying the interval between VNS-tone pairings changed the extent of auditory-cortex map reorganization without supporting a universal monotonic rule in which shorter or longer intervals simply generated progressively greater plasticity.[120] Addo and colleagues, also introduced in Section 10.4, found several moderate pulse-frequency conditions produced similar increases in representation of the paired movement, while burst stimulation and a pulse-count-matched pattern failed to produce the same plasticity[93], pulse count alone was insufficient to define the effective stimulation.

These experiments converge with the spacing and metaplasticity literature from Sections 9 and 10: neural interventions can depend simultaneously on intensity, timing, temporal pattern, behavioral pairing, and the physiological state created by preceding stimulation. The relevant model is not more neuromodulation → more plasticity, but neuromodulatory parameters × timing × concurrent neural activity → selective plasticity outcome, and even this remains simplified because the result can vary by task, circuit, organism, and previous state. Carroll and colleagues' behavioral null, noted in 12.1, provides an important boundary: successful cortical-plasticity mechanisms do not ensure measurable behavioral enhancement under every training condition.[108]

For SeaKing, this has a direct methodological implication: if later research finds evidence that natural motion recruits a candidate neuromodulatory mechanism, it would be scientifically inappropriate to assume that larger motion, longer exposure, stronger autonomic change, or larger pupil response represents a more favorable condition. The VNS literature instead predicts nonlinear relationships, a moderate signal may be more effective than a stronger one, temporal alignment with experience may matter more than total amount, similar cumulative stimulation delivered in different temporal patterns may produce different outcomes, and a physiological response may occur without improved behavior.[44, 92, 93, 108, 118] These principles reinforce the need for empirical motion-response mapping rather than maximal stimulation.

12.5 Natural Motion Has Not Been Shown to Recruit the VNS/LC Gating Mechanism

The mechanistic strength of the VNS literature creates a tempting translational shortcut that must be rejected. Natural multidimensional vestibular motion and vagus nerve stimulation are not the same intervention: natural self-motion generates structured rotational and translational accelerations across multiple dimensions,[3] central vestibular responses depend on whether motion is actively generated or externally imposed,[19] and simultaneous canal and otolith inputs are integrated nonlinearly.[7] Implanted VNS instead begins through electrical stimulation of the cervical vagus nerve, with a demonstrated mechanistic chain including direct recruitment of locus-coeruleus activity,[112] changes in extracellular norepinephrine,[113] dependence of selected cortical-plasticity effects on cholinergic, noradrenergic, and serotonergic systems,[109–111] and circuit-selective reinforcement associated with cholinergic signaling.[107] No evidence in the present canonical literature demonstrates that natural multidimensional vestibular motion reproduces this pathway. The following chain is therefore not established: natural vessel motion → vagal activation → locus-coeruleus/norepinephrine recruitment → cholinergic reinforcement → enhanced neuroplasticity. Individual links may be biologically plausible or supported in different experimental systems; the complete intervention-specific chain has not been demonstrated.

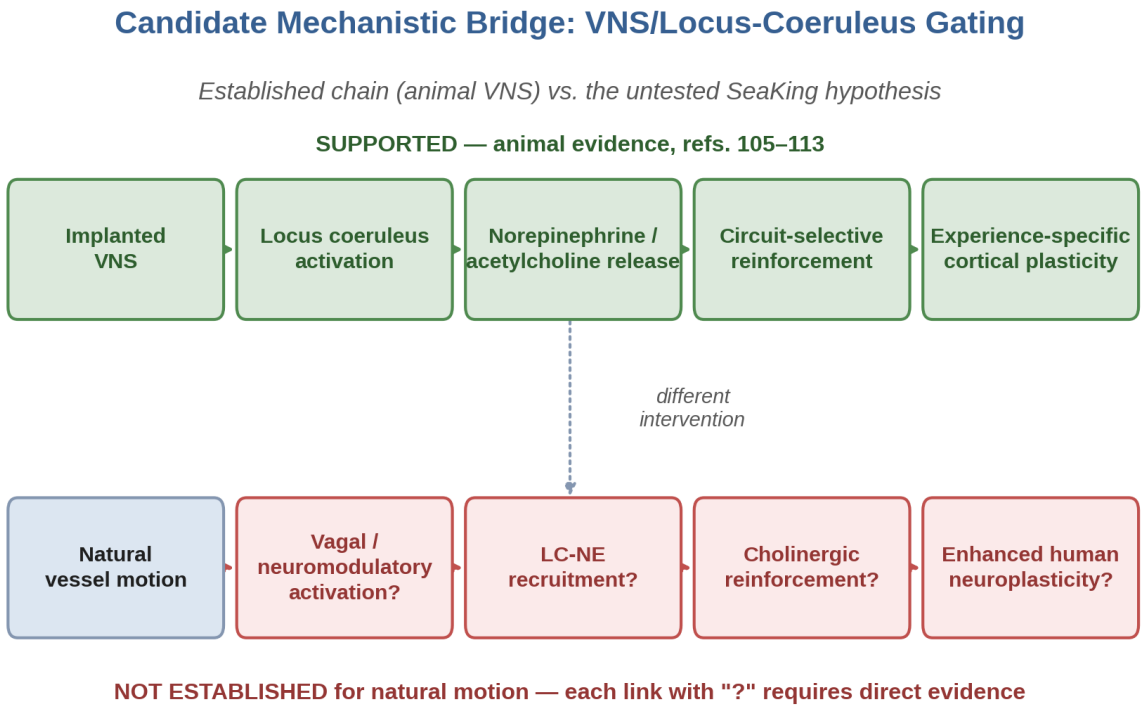

Figure 7. Candidate mechanistic bridge between vagus-nerve-stimulation-directed cortical plasticity (established in animals) and the SeaKing hypothesis (not established for natural motion). Dashed arrows mark unresolved inferential steps.

This is more than a distinction between animal and human evidence, it is a distinction between different inputs into the nervous system. The vestibular literature establishes that natural motion has complex, nonlinear central representation;[3, 7, 19] the VNS literature establishes that direct vagal stimulation can engage specific neuromodulatory systems and, when appropriately paired with experience, direct cortical plasticity.[105–113] Those two evidence streams do not automatically connect. An autonomic response during natural motion would not close the gap: a change in heart

rate, HRV, respiration, or another autonomic endpoint could establish that natural motion alters autonomic physiology, not that the cervical VNS mechanism has been reproduced. A pupil response would also be insufficient, as Section 11.5 established, since pupillary changes are indirect and nonspecific and controlled taVNS studies show both positive and null pupillary results.[102–104] Even evidence that natural motion changes learning would not by itself establish the VNS mechanism, since a behavioral effect can occur through multiple neural routes. The VNS literature establishes a plausible model of neuromodulatory gating. It does not establish the mechanism of natural multidimensional vestibular motion. That remains an empirical question, one this boundary makes specifically testable rather than irrelevant.

12.6 What a Later Mechanistic Bridge Study Would Need to Show

Moving from mechanistic analogy to SeaKing-specific evidence requires an intervention-specific bridge: candidate neuromodulatory effects must be linked directly to natural multidimensional motion and then related prospectively to an independent plasticity or learning outcome. Human taVNS studies illustrate why no single surrogate measure is sufficient. The Nakagawa motor-cortex expansion occurred without additional behavioral improvement,[118] so a neural plasticity-related measure did not automatically predict superior task acquisition. Capone and colleagues conducted a randomized double-blind crossover study examining short-latency afferent inhibition, a TMS measure sensitive to cholinergic circuitry, after real and sham taVNS, and found no significant alteration relative to sham[121], not proof that taVNS fails to engage cholinergic systems, but demonstration that an animal mechanism cannot simply be assumed to appear in every human indirect biomarker. The Keute and D'Agostini null findings on pupil, salivary alpha-amylase, and cortisol reinforce the same point.[102, 103] Even when an intervention has a biologically plausible neuromodulatory mechanism, indirect human markers may remain unchanged, may lack specificity, or may dissociate from behavioral outcomes.[102, 103, 118, 121]

A future SeaKing mechanistic bridge study would therefore need to address several questions together, not any one in isolation: whether quantified natural motion reliably changes the candidate physiological or neural measure relative to an appropriate control (without which there is no effect to explain); whether the candidate measure is sufficiently specific to support the proposed mechanism (pupil dilation alone would not establish LC activation, HRV alone would not establish vagus-mediated cholinergic or noradrenergic gating); whether variation in the candidate mechanism predicts an independent learning or plasticity outcome (a physiological effect unrelated to acquisition or retention may be biologically interesting without functioning as the hypothesized gating mechanism); whether the temporal relationship makes mechanistic sense, given that the VNS literature shows pairing neuromodulatory events with specific behavioral activity determines the resulting cortical reorganization;[105–107] and whether the relationship can be reproduced, since a bridge inferred from one exploratory dataset requires prospective replication before being treated as established.

The conceptual progression is therefore natural motion → intervention-specific physiological or neural change → demonstrated association with independently measured plasticity or learning → temporally coherent mechanism → replication. Only after this chain is supported would analogy to VNS become a mechanistic bridge rather than a theoretical comparison, and such a study need not reproduce every feature of implanted VNS, since natural motion may influence learning through a partially overlapping pathway, a different neuromodulatory pathway, vestibular-cortical mechanisms independent of VNS, or interactions among several systems, with direct data determining which model is most appropriate.

The VNS and basal-forebrain evidence establishes that experience-specific cortical plasticity can be selectively gated by temporally associated neuromodulatory signals, with the resulting effect depending on neurotransmitter systems, timing, intensity, and behavioral context.[44, 92, 93, 105–115, 120] Human taVNS research further demonstrates that noninvasive neuromodulation can influence learning or memory under some conditions while producing null or dissociated neural and behavioral outcomes under others.[20, 104, 116–119, 121] These findings make neuromodulatory

gating a legitimate candidate mechanism for future investigation. They do not establish that natural multidimensional vestibular motion recruits it. Phase 1 can characterize motion-associated physiological states without assigning them prematurely to a specific neuromodulatory pathway; later studies can determine whether any reproducible state predicts learning; and only then should increasingly mechanistic experiments ask whether cholinergic, noradrenergic, vagal, locus-coeruleus, or other systems contribute to the observed effect.

13. Developmental Timing and Sensitive Periods

Neuroplasticity changes across development. Some neural systems exhibit periods during which particular experience exerts unusually strong or enduring effects, and developmental stage can alter the efficiency, direction, or consequences of learning and neural reorganization.[42, 122] This establishes developmental timing as an important biological variable. It does not establish a single age at which meaningful neuroplasticity closes: sensitive periods differ across neural systems and forms of experience,[42, 122] adolescence can itself contain periods of substantial developmental reorganization rather than representing the simple termination of childhood plasticity,[43, 123] and direct human evidence demonstrates continuing learning, training-associated neural change, and structural development during adolescence and adulthood, including in autistic populations.[71, 124–129]

This distinction matters for SeaKing. Developmental stage should influence study design, stratification, and interpretation; it should not become an unsupported mechanistic exclusion threshold. In particular, the evidence reviewed here does not support a universal claim that meaningful neuroplasticity in autism disappears at approximately age 14. The defensible position is developmental rather than deterministic: age can modify plasticity, but chronological age alone does not identify whether an individual nervous system remains capable of meaningful experience-dependent change.

13.1 Sensitive Periods Are Real but Domain Specific

Knudsen defined sensitive periods as developmentally limited intervals during which experience exerts particularly strong and enduring effects on behavior and neural organization[122], not merely that younger nervous systems are plastic, but that the influence of a given experience varies according to when it occurs. Ismail and colleagues reviewed evidence across developing neural systems and reached the same conclusion, with periods of heightened responsiveness that are domain specific rather than governed by one global timetable: language, motor learning, multisensory integration, association-cortex development, and vestibular adaptation need not share the same developmental trajectory.[42, 122]

It is therefore scientifically inappropriate to describe the brain as possessing one global plasticity window that opens in childhood and closes at a specific age. Different neural circuits mature at different rates, different experiences interact with those circuits at different stages, and a reduction in the efficiency of one form of plasticity does not imply disappearance of all plastic capacity.[42, 122] A sensitive period identifies a developmental interval of particular responsiveness; it does not imply that meaningful learning becomes impossible afterward, as the vestibular, motor, cognitive, and rehabilitation-related learning demonstrated throughout this review in adults, including older adults, already shows.[2, 9, 10, 24]

Adolescence complicates any simple early-childhood-only model further. Fuhrmann and colleagues reviewed evidence that adolescence may itself represent a period of heightened or distinctive sensitivity for selected cognitive and social processes,[43] and Larsen and Luna described prolonged maturation of prefrontal and association-cortex systems, arguing that critical-period-like mechanisms may influence higher-order cognition during adolescence.[123] Neither review establishes adolescence as one universal sensitive period; both demonstrate that developmentally meaningful reorganization continues well beyond early childhood.[43, 123] This has direct consequences for interpreting autism: the existence of early developmental differences does not

identify the age at which those differences become immutable, and evidence that early intervention can be valuable does not establish that intervention after childhood is biologically incapable of producing change. The developmental evidence supports the claim that timing matters; it does not support a universal closure age.[42, 43, 122, 123] For SeaKing, developmental stage should be treated as a possible moderator of response, not an arbitrary upper age limit derived from a generalized conception of childhood neuroplasticity.

13.2 Adolescence and Adulthood Are Not the End of Meaningful Plasticity, Including in Autism

Direct human evidence contradicts the idea that adolescence represents a general endpoint for meaningful plasticity. Foxe and colleagues found younger autistic children showed marked impairment in audiovisual speech integration relative to neurotypical peers, but the group difference was no longer evident by early adolescence under the tested conditions;[124] because the study was cross-sectional it cannot establish within-person normalization or an intervention mechanism, but it demonstrates that an autism-associated multisensory phenotype observed earlier in development need not remain fixed. Ainsworth and Bertone similarly found audiovisual temporal-binding windows narrowed with increasing age in autistic participants,[125] again age-associated rather than interventional, but demonstrating continuing developmental change in multisensory temporal processing across childhood and adolescence.

Longitudinal structural evidence extends the trajectory further. Wallace and colleagues followed autistic and neurotypical participants aged approximately 14 to 24 using repeated structural MRI and found autistic participants continued to show measurable cortical development across adolescence and young adulthood, including regionally accelerated cortical thinning relative to controls while surface-area trajectories were more comparable.[128] Structural maturation is not synonymous with therapeutic neuroplasticity, cortical thinning cannot be interpreted as improvement, deterioration, or treatment responsiveness, but the finding establishes something basic and directly relevant: autistic cortical development remains dynamic beyond age 14 and into young adulthood.

Direct learning and intervention evidence matters more, since developmental change alone does not establish capacity for experimentally induced modification. Hayes and colleagues found intact sensorimotor learning in autistic adults, though the relationship between learning and associated visual perception differed from neurotypical participants[126], demonstrating retained sensorimotor learning capacity alongside differently organized learning-related processes. Bölte and colleagues found autistic participants undergoing facial-affect-recognition training improved behavioral performance and showed increased bilateral neural responses during an affect-processing task following training,[127] demonstrating intervention-associated behavioral and neuroimaging change beyond early childhood. Rosenblau and colleagues provide particularly strong adult evidence: high-functioning autistic adults randomized to three months of social-cognitive training, versus a non-social active control, improved on both close and more distant social-cognitive generalization measures, accompanied by functional and structural neuroimaging changes involving prefrontal regions.[71]

These findings do not establish globally typical plasticity in autism, that every intervention remains equally effective at every age, or that developmental timing is irrelevant. They establish something narrower but decisive: meaningful learning and intervention-associated neural and behavioral change remain demonstrable after childhood, including in autistic adults.[71, 126, 127] The developmental reviews are consistent with this, adolescence may contain distinctive periods of experience sensitivity,[43] and higher-order association systems continue prolonged development through this stage[123], arguing against treating puberty or early adolescence as the end of biologically meaningful modifiability. A more accurate model is that the form, magnitude, mechanisms, and efficiency of plasticity may change with age while plastic capacity persists, with translational consequences: a later developmental stage may require different training intensity, task structure, exposure duration, or endpoints than an earlier stage, and these possibilities should be

studied as age-dependent differences in response rather than compressed into a binary distinction between "plastic" children and "nonplastic" adolescents or adults.

13.3 The Autism Age-14 Cutoff Is Unsupported

No verified source in the canonical evidence base establishes age 14 as a biological endpoint beyond which autistic individuals lose meaningful capacity for learning, neural modification, or intervention-associated change. The direct findings in 13.2 contradict such a claim from multiple independent angles: autistic cortical development remains measurable from adolescence into young adulthood, incompatible with development becoming static at 14;[128] autistic adults demonstrate experimentally measurable sensorimotor learning;[126] training-associated behavioral and neural change has been demonstrated beyond childhood;[71, 127] and multisensory developmental trajectories continue changing through and beyond adolescence rather than reaching an experimentally established hard stop at 14.[124, 125] A fifth line of evidence adds direct support at the level of induced cortical modulation itself: Oberman and colleagues used theta-burst stimulation to examine motor-cortex excitability in adults with Asperger disorder and found the autistic group showed unusually prolonged TBS-associated corticospinal effects relative to matched controls, with response duration showing substantial discriminatory value in a second cohort.[129] Prolonged TBS aftereffects do not prove globally enhanced, beneficial, or representative plasticity across all autistic adults, but the finding directly contradicts the proposition that experimentally inducible neural modulation disappears after adolescence.

The combined evidence rejects the age-14 cutoff for two independent reasons: there is no verified direct evidence establishing it, and there is direct evidence of continued development, learning, cortical modulation, and training-associated change at and beyond that age.[71, 124–129] This does not make age irrelevant, an intervention may be more effective at one developmental stage than another, particular neural systems may become less modifiable with maturation, and learning rate, retention, generalization, and response to neuromodulation may all vary by age. What is unsupported is converting those possibilities into the categorical rule that meaningful autism neuroplasticity ends at approximately age 14. For SeaKing, age 14 should therefore not be used as a mechanistic exclusion threshold; any age restrictions in future studies should instead arise from the scientific question, safety considerations, protocol requirements, population definition, and evidence relevant to the specific outcome being studied.

13.4 Developmental Stage Should Be Modeled Rather Than Presumed

Rejecting a universal cutoff does not mean age is biologically unimportant, it should be modeled as a potential moderator of response. Direct cross-system experimental evidence shows why. Opie and colleagues compared a priming theta-burst protocol in young and older healthy adults and found priming enhanced subsequent motor-cortex plasticity in younger participants but not in the older group under the tested protocol[130], an age-dependent difference in one specific metaplasticity paradigm, not evidence that older adults lack plasticity generally. Indeed, direct vestibular evidence reviewed in Section 3 demonstrates substantial vestibular perceptual learning in adults aged 70 to 88.[2] The two findings are entirely compatible if plasticity is understood as system and protocol dependent rather than one global biological quantity: age can alter one plasticity-induction response while leaving meaningful learning capacity demonstrable in another system. Chronological age is therefore a moderator, not a direct biomarker of plastic capacity.

For future SeaKing studies, developmental stage should enter analysis as a variable whose relationship with response is tested rather than assumed. Younger and older participants might show similar acute responses but different adaptation rates across sessions, different acute responses but similar learning outcomes, faster acquisition in one group with equivalent eventual retention in the other, or developmental differences appearing in one endpoint and absent in another, none of which should be predetermined by a generic developmental-plasticity narrative rather than measured directly.

This is particularly important if SeaKing progresses into autism research: autism is heterogeneous and developmental trajectories are not uniform, so evidence from high-functioning adolescents or adults cannot automatically generalize to all autistic individuals, any more than evidence from younger children defines the capabilities of every adolescent or adult.[71, 124–129] Future studies should therefore avoid both early-age determinism, in which later participants are presumed biologically incapable of meaningful change, and age indifference, in which developmental stage is assumed to have no effect; the evidence supports neither. Developmental stage should instead be incorporated prospectively into study design, stratification, and interaction testing where sample size permits, asking not simply whether older and younger participants differ but which endpoints differ, under which stimulus conditions, and with what consequences for acquisition, adaptation, retention, or function.

This has an important consequence for the SeaKing hypothesis: the project does not require the existence of an early-childhood-only therapeutic window. Human vestibular plasticity persists into later adulthood,[2] autistic adults demonstrate measurable learning and intervention-associated neural and behavioral change,[71, 126, 127, 129] and autistic neurodevelopment continues beyond early adolescence.[128] SeaKing should not, at the same time, assume the magnitude or form of those responses will be identical across developmental stages. The scientifically supported position is that development changes plasticity without imposing a universal age at which meaningful plasticity disappears, preserving both sides of the evidence: sensitive periods are biologically real and can make developmental timing important,[42, 43, 122, 123] yet direct human evidence demonstrates continuing modifiability well beyond childhood, including within autism.[71, 124–129] For SeaKing, age should be studied rather than presumed.

14. Autism-Specific Plasticity: Atypical Regulation, Retained Capacity, and Evidentiary Boundaries

Section 13 establishes that meaningful learning and neural change continue beyond childhood, including in autistic adolescents and adults. A more difficult question is whether autism itself is associated with a characteristic alteration in neuroplasticity. The direct evidence does not support a simple answer: TMS, paired-associative stimulation, and visual plasticity paradigms have reported both reduced and exaggerated plasticity-like responses in autistic participants,[129, 131–137] with no convergence on one uniform direction, some cohorts show attenuated induction, others prolonged or exaggerated responses, and the measured effect varies with age, protocol, genotype, medication exposure, circuit, and study population.[129, 131, 132, 135–139] Autism should therefore not be characterized as globally hypoplastic or globally hyperplastic. The stronger proposition is that regulation of plasticity can be atypical and heterogeneous, with direction and magnitude dependent on what is measured and in whom.

This distinction is especially important for SeaKing. A hypothesis in which natural motion simply "restores deficient plasticity" or "normalizes excessive plasticity" would require a unitary autism-plasticity abnormality the evidence does not establish. The more defensible research question is whether autistic participants show measurable, reproducible, and potentially phenotype-dependent responses to a defined motion exposure, and whether those responses relate to learning or function.

14.1 Why Global Hypoplasticity and Hyperplasticity Models Both Fail

As introduced in 13.3, Oberman and colleagues found TBS-induced motor-cortex modulation persisted significantly longer in autistic adults with Asperger disorder than in matched controls[129], robust and prolonged induced cortical modulation, though probing one motor-cortical response in one historically defined, cognitively able cohort, not globally enhanced plasticity. Other motor-cortex experiments point the opposite direction. Pedapati and colleagues examined intermittent TBS in autistic and typically developing youth aged 11 to 18 and found the autistic group showed less facilitatory response, including a significant group difference at one post-stimulation time point[131], a nine-versus-nine pilot, but direct evidence that an experimentally induced LTP-like response can

be reduced in an autistic cohort. Jung and colleagues similarly found significantly less PAS-induced potentiation in participants with high-functioning autism or Asperger syndrome than in matched controls.[132] These findings directly contradict a universal hyperplasticity model, just as Oberman's contradicts a universal hypoplasticity model.

Visual-plasticity paradigms add a further layer of disagreement within the same broad sensory system. Wilson and colleagues interpreted high-frequency visual stimulation results as showing greater persistent visual-cortical potentiation in autistic adults,[133] while a later study by Ellis and colleagues reported the opposite: reduced short-term, LTP-like visual-evoked-potential potentiation relative to neurotypical controls.[134] Theta-burst studies provide further complexity: Jannati and colleagues found more facilitatory cumulative cTBS responses in autistic children and adolescents aged 10 to 16, varying with age within the autistic cohort,[135] while a larger prospective and pooled adult analysis found post-cTBS motor-evoked-potential responses differed systematically between autistic and neurotypical participants, moderated by BDNF and APOE genotype[136], an atypical cortical response, not one universal direction.

Desarkar and colleagues reported still another pattern: both LTP-like and LTD-like motor-cortical responses were greater in autistic adults than controls, and subsequent active rTMS attenuated the excessive LTD-like response while leaving the LTP-like component unmodified[137], difficult to reconcile with either a global deficiency or a global-excess model, since both potentiation-like and depression-like responses could be exaggerated within the same cohort while an intervention differentially modified one component. A theoretical hyperplasticity framework has also been proposed in the autism literature;[138] such models can generate hypotheses, but the source is explicitly theoretical rather than direct experimental evidence and cannot establish that autism as a whole is hyperplastic or that putative hyperplasticity is beneficial.

The direct evidence therefore supports a constrained conclusion: autism is associated with atypical responses on several experimental plasticity assays, but the direction is not uniform across studies, protocols, circuits, or populations.[129, 131–137] This is not literature inconsistency to be averaged away; the contradictions are themselves informative, since a system whose regulation varies by circuit, induction protocol, developmental stage, genetic background, or phenotype would be expected to generate precisely this heterogeneity. For SeaKing, the scientific rationale should not depend on a claim that autistic brains need a generic increase or decrease in plasticity; existing evidence does not identify such a universal deficit.

Table 3. Direction of effect across human autism cortical-plasticity assays. Studies span reduced, prolonged, and exaggerated responses depending on paradigm, age, and cohort, evidence for heterogeneous rather than uniformly hypo- or hyperplastic regulation.

Study	Paradigm	Population	Direction of Effect
Oberman et al.[129]	Theta-burst stimulation (motor)	Adults, Asperger disorder	Prolonged modulation vs. controls
Pedapati et al.[131]	Intermittent TBS (motor)	Youth 11–18 (pilot, n=9/9)	Reduced facilitatory response
Jung et al.[132]	Paired-associative stimulation	High-functioning autism / Asperger	Reduced LTP-like potentiation
Wilson et al.[133]	High-frequency visual stimulation	Adults	Greater persistent VEP potentiation
Ellis et al.[134]	High-frequency visual stimulation	Adults	Reduced LTP-like VEP potentiation
Jannati et al.[135]	Continuous TBS (motor)	Children/adolescents 10–16	More facilitatory cumulative response; age-related
Jannati et al.[136]	Continuous TBS (motor), pooled	Adults	Systematically different response; moderated by BDNF/APOE genotype
Desarkar et al.[137]	Paired-associative stimulation	Adults	Both LTP-like and LTD-like responses exaggerated

14.2 Atypical and Heterogeneous Regulation Is the Stronger Proposition

The evidence in 14.1 supports heterogeneous rather than directionally uniform regulation on at least four grounds. Direction changes across assays, reduced facilitation and potentiation in some studies,[131, 132] prolonged or exaggerated responses in others.[129, 135, 137] Disagreement occurs even within one sensory system, as the opposite-direction visual-plasticity findings show.[133, 134] Developmental stage alters the measured phenotype: Oberman and colleagues separately found the duration of cTBS-induced modulation increased with age across autistic boys aged 9 to 18,[139] and Jannati found comparable age relationships within pediatric cTBS responses[135], not one universal developmental trajectory, but evidence that age interacts with experimentally probed plasticity rather than serving as a demographic descriptor. And person-level biological moderators matter, since the relationship between post-stimulation cortical response and autism diagnosis in the larger Jannati adult cohort was moderated by BDNF and APOE genotype[136], direct evidence that a measured plasticity phenotype can depend partly on biological characteristics within or across groups.

Together these support a model in which autism-related plasticity differences may vary across neural circuit, induction paradigm, developmental stage, genotype, medication exposure, sensory or motor domain, participant phenotype, and previous neural or experimental state.[129, 131–139] This does not mean every contradictory study is equally correct or that heterogeneity explains all disagreement (small samples, measurement variability, and imperfect replication also contribute), but the existing direct evidence is incompatible with a one-direction whole-brain autism-plasticity phenotype. This changes the research question: instead of asking whether SeaKing motion "increases autism neuroplasticity," a later autism study should ask whether defined motion exposure changes specific measured responses and whether those responses differ systematically across participants, sessions, developmental stages, or phenotypic characteristics.

A participant whose response differs from the group mean should not automatically be classified as a failed responder, the difference may itself be meaningful if reproducible and prospectively associated with measurable baseline characteristics, while subgroup interpretations must still be validated

rather than invented retrospectively. The strongest autism-specific proposition available is therefore that plasticity remains experimentally inducible in autism, but its regulation appears atypical and heterogeneous rather than uniformly increased or decreased[129, 131–139], more scientifically accurate and more useful for SeaKing study design than a generic normalization model.

14.3 Autistic Adolescents and Adults Retain Meaningful Learning Capacity, Including Training-Associated Neural Change

Abnormal results on cortical-plasticity assays do not imply absent behavioral learning. Sharer and colleagues found autistic participants successfully acquired a motor sequence while manipulating visual and proprioceptive information, though the relative influence of sensory information on learning differed from neurotypical participants[140]; the learning outcome was present, and what differed was the sensory weighting supporting it. Travers and colleagues similarly demonstrated motor-linked implicit learning in autistic adolescents and young adults,[141] establishing that meaningful implicit acquisition remains demonstrable beyond early childhood without claiming every form of implicit learning is intact in autism. The Hayes sensorimotor-learning finding from 13.2 extends this into adulthood, with preserved acquisition again coexisting with atypically organized underlying processes.[126]

Two training studies already introduced in 13.2 add the neural dimension directly. Bölte and colleagues found facial-affect-recognition training improved the trained behavioral outcome and changed task-related brain responses,[127] and Rosenblau and colleagues found three months of social-cognitive training, against an active non-social control, improved both close and distant social-cognitive generalization measures with corresponding functional and structural neuroimaging change involving prefrontal regions.[71] These establish a direct capability claim: autistic adolescents and adults retain measurable capacity for experience-dependent learning and training-associated behavioral improvement.[71, 126, 127, 140, 141] That conclusion should not be inflated, learning on one task does not establish globally typical plasticity, does not mean every intervention will work, does not establish retention or broad generalization, and evidence from cognitively able cohorts cannot automatically transfer to the entire autism spectrum (addressed directly in 14.5).

Its importance is that it separates cortical-plasticity differences from behavioral incapacity: an autistic participant can show atypical TMS or sensory-plasticity responses while remaining capable of meaningful learning, consistent with the broader argument that plasticity is not one scalar biological quantity. For SeaKing, later studies involving autistic adolescents or adults would not begin from a biological presumption that meaningful learning is unavailable; the evidence demonstrates learning remains experimentally accessible. Whether natural multidimensional motion changes that learning remains unknown.

Several restrictions on interpreting Bölte's and Rosenblau's neural findings are necessary: functional activation change is not synonymous with synaptic potentiation, cortical-thickness change does not prove neurogenesis or a specific cellular mechanism, an association between imaging change and behavioral improvement does not establish that the imaging change caused the improvement, and these training-specific results do not establish generalized therapeutic neuroplasticity across all autistic adults.[71, 127] Even so, the studies answer directly that adulthood does not preclude experimentally measurable training-associated neural change in autism, a stronger evidentiary foundation than developmental anatomy alone, since longitudinal structural maturation demonstrates the brain remains developmentally dynamic[128] while intervention studies demonstrate that a defined experience can be associated with measurable behavioral and neural modification.[71, 127] That finding supports the feasibility of later mechanistic SeaKing research in adolescents and adults. It does not establish that natural motion will generate similar neural change.

14.4 Mechanistic Evidence Is Not Representative of the Full Autism Spectrum

The direct autism-plasticity literature contains an important external-validity limitation: nearly all of the mechanistic evidence above, the Oberman adult and pediatric TBS studies,[129, 139] the Pedapati and Jung TMS/PAS studies,[131, 132] the Wilson and Ellis visual-plasticity studies,[133,

134] the Jannati pediatric and adult cTBS studies,[135, 136] and the Rosenblau training study[71], comes from relatively small, cognitively able, verbally fluent, or historically "high-functioning" autism cohorts, selected because the procedures required extended participation in TMS, psychophysical, electrophysiological, or training protocols. These samples provide direct experimental access to plasticity mechanisms that would otherwise remain difficult to investigate, but they are not representative of the full autism spectrum, and cannot automatically be generalized to autistic individuals with moderate or severe intellectual disability, limited spoken language, substantial sensory intolerance, complex co-occurring medical conditions, or higher support needs. This matters because SeaKing's long-term translational importance may include precisely those underrepresented populations.

The correct response is neither to assume mechanistic findings from cognitively able cohorts apply unchanged, nor to infer that higher-support-needs individuals lack plastic capacity; the evidence supports neither inference. Representativeness must instead become part of the research design: participant phenotype should be characterized carefully, findings reported according to the population actually studied, and generalization across cognitive, communicative, developmental, or support-needs strata attempted only when data permit it. This is particularly important because heterogeneity is already evident even within the comparatively selected cohorts used in mechanistic studies[129, 131–139], a more heterogeneous clinical population should not be expected to produce a more uniform response.

Underrepresentation creates uncertainty; it does not establish biological incapacity. Direct mechanistic plasticity evidence in autistic individuals with substantial intellectual disability remains sparse within the canonical evidence base, and this limitation should remain explicit. Available research nonetheless demonstrates that more inclusive participation is feasible when procedures are adapted appropriately. Langdon and colleagues conducted a feasibility study of an adapted behavioral anxiety intervention in 28 autistic adults with moderate or severe intellectual disability, supported by carers and therapists, and concluded the adapted intervention and research procedures were feasible and acceptable[142], not designed to prove efficacy and not a measure of neural plasticity, but methodologically relevant in demonstrating that a population underrepresented in conventional experimental neuroscience is not inherently incapable of participating in structured intervention research. Tan and colleagues provide a second example with randomized evidence: a multicentre masked sham-controlled trial of accelerated cTBS in 200 autistic children aged 4 to 10, enrolling children with full-scale IQ from 50 upward and stratifying above and below IQ 70, found active stimulation produced greater improvement than sham on the primary social-responsiveness measure immediately after treatment and at one-month follow-up.[143] This trial did not directly measure neuroplasticity, an IQ threshold of 50 does not represent individuals with the most profound intellectual disability, and a behavioral response to cTBS does not establish a shared neural mechanism across participants, but it demonstrates that controlled neuromodulation research and measurable intervention response are not restricted to autistic participants with average or above-average intellectual ability.

Together these studies support a crucial boundary: the scarcity of direct mechanistic plasticity data in higher-support-needs autism reflects an evidence gap, not evidence that meaningful plasticity or intervention responsiveness is absent.[142, 143] For SeaKing, later research should neither simply extrapolate mechanistic findings from high-functioning cohorts to higher-support-needs participants, nor exclude those participants on the unsupported assumption that they lack capacity to respond. Future protocols may instead require adapted communication, sensory accommodations, caregiver involvement, modified behavioral measures, tolerance-based procedures, and endpoints appropriate to the population, a problem of measurement and representation, not a pre-established biological absence of plasticity.

14.5 Natural Motion Has Not Been Shown to Reopen or Normalize Autism Plasticity

The autism-specific evidence creates a final and essential boundary for the SeaKing hypothesis. No direct study in the canonical evidence base demonstrates that natural multidimensional motion reopens an autism sensitive period, restores a developmentally closed plasticity process, or normalizes an autism-specific cortical-plasticity abnormality. Several evidence streams make such a hypothesis biologically conceivable in the abstract, natural self-motion provides structured multidimensional vestibular input,[3] central vestibular processing depends on the relationship among rotational, translational, active, and passive motion components,[7, 19] human autism research demonstrates that learning and training-associated neural change remain possible beyond childhood,[71] and developmental neuroscience establishes that sensitive periods exist with domain-specific timing[42, 122] within an adolescence that can retain heightened experience sensitivity and active cortical maturation.[43, 123] These findings do not establish the combined chain: natural multidimensional vestibular motion → reopening of a sensitive period → normalization of autism plasticity → improved function. Each arrow would require its own evidence, whether natural motion alters a plasticity-relevant state in autistic participants, whether that state changes acquisition or retention, whether any learning effect generalizes, and whether the resulting change produces meaningful function.

There is currently no evidentiary basis for referring to SeaKing as a sensitive-period-reopening intervention, and no unitary autism-plasticity abnormality for such an intervention to "normalize", as 14.1 and 14.2 demonstrate, direct human assays produce both reduced and exaggerated responses depending on experimental context.[129, 131–137] This makes normalization an especially problematic endpoint unless the relevant physiological abnormality is defined prospectively for a specific assay and population: a participant with a prolonged TBS response does not necessarily need that response shortened, and a participant with reduced PAS potentiation does not necessarily need it increased, since neither measure has been established as a universal causal determinant of autism symptoms or functional outcome.[129, 131, 132] The SeaKing rationale should therefore avoid a repair model in which natural motion is presumed to push an autistic nervous system toward a single neurotypical plasticity state.

A more testable framework is available. Autistic nervous systems remain capable of measurable learning and training-associated neural change;[71, 126, 127, 140, 141] plasticity regulation appears heterogeneous rather than globally absent;[129, 131–139] and natural motion is a complex vestibular stimulus whose human physiological consequences can be quantified.[3, 7, 19] These component findings justify asking whether natural multidimensional motion interacts with learning or physiological regulation in autism. They do not determine the answer. A later SeaKing autism study could test whether quantified motion produces reproducible physiological responses, whether repeated exposure changes those responses, whether participant characteristics explain meaningful heterogeneity, and whether a defined motion condition modifies acquisition or retention relative to control, with mechanistic experiments, if such effects are demonstrated, then determining whether they involve vestibular sensory reweighting, neuromodulatory gating, state-dependent learning, altered cortical excitability, or another pathway. The mechanism should be discovered from the data rather than selected in advance because it creates the most attractive narrative.

Autism is not characterized by an experimentally established global loss of neuroplasticity, nor by a consistently demonstrated global excess. Direct human evidence instead supports atypical and heterogeneous regulation together with retained capacity for meaningful learning and training-associated neural change beyond childhood.[71, 126, 127, 129, 131–141] The principal limitation is representativeness, since much of the mechanistic evidence derives from cognitively able cohorts while higher-support-needs populations remain substantially less characterized.[71, 129, 131–139, 142, 143] For SeaKing, these findings support investigation rather than normalization claims: natural multidimensional motion has not been shown to reopen a sensitive period, correct an autism-specific plasticity defect, or produce therapeutic neuroplasticity. What the literature establishes is that the

relevant nervous system remains modifiable, that its plasticity regulation is heterogeneous, and that the proposed intervention-specific effect remains an empirical question.

15. Adaptive, Maladaptive, and Functionally Indeterminate Vestibular Plasticity

The existence of neuroplasticity does not determine whether the resulting change is beneficial. This is particularly important in vestibular neuroscience because experience-dependent change can support compensation and improved function under some conditions while persistent vestibular-related states can be disabling under others,[11, 12, 15, 16] and a third possibility must be preserved: neural or physiological change may be demonstrable while its functional significance remains unknown.[8] These categories should not be confused with duration, persistence is a temporal property, while adaptiveness and maladaptiveness are functional classifications. A change should be classified as adaptive when evidence links it to improved function within the measured context, maladaptive or adverse when associated with impairment or persistent symptoms, and functionally indeterminate when measurable change exists without sufficient evidence establishing benefit or harm.

This framework matters directly for interpreting future SeaKing data. A physiological response that becomes reproducible or persists across repeated natural-motion exposures would be scientifically important, but persistence alone would not justify describing the effect as therapeutic neuroplasticity, functional meaning must be demonstrated independently.

15.1 Persistence Is Not Synonymous With Benefit

Persistent change is often treated implicitly as stronger evidence of beneficial neuroplasticity than transient change. Scientifically, persistence establishes durability, not value. Human vestibular compensation provides examples of persistent change with beneficial functional meaning: individuals with bilateral vestibular dysfunction can redistribute sensory weighting toward more reliable visual and proprioceptive information as vestibular reliability declines,[11] and healthy postural control dynamically adjusts sensory weighting when environmental conditions change.[12] Persistent vestibular-related states can equally be adverse; PPPD is characterized by chronic dizziness or unsteadiness exacerbated by upright posture, motion, and complex visual environments,[15] and MdDS can involve persistent rocking, bobbing, or swaying after passive-motion exposure, sometimes lasting far beyond the motion event that preceded it.[16] Persistence itself therefore carries no inherent positive functional direction, and the same conclusion applies to neural measurements: in the randomized, sham-controlled PPPD tDCS trial introduced earlier, active treatment produced regional cerebral blood-flow differences involving the right superior temporal and left hippocampal regions, yet did not significantly outperform sham on the primary dizziness outcome.[8] The neural difference was real; the clinical benefit was not established, illustrating why a persistent or reproducible biological effect cannot be assigned therapeutic meaning solely because it is measurable. The opposite error is also possible: an adverse persistent state should not automatically be read as evidence of irreversible neural damage, since the functional classification describes what the state is doing to the person, not whether the underlying neural processes are permanent.

The appropriate framework is therefore: persistence plus improved function is evidence compatible with adaptive change; persistence plus impairment is evidence of an adverse or maladaptive functional state; and persistence without demonstrated functional consequence is functionally indeterminate change. For SeaKing, suppose repeated natural motion produces an autonomic, pupillary, vestibular, or other physiological response that remains altered after exposure, that establishes persistence. If the persistent change is subsequently associated prospectively with improved task performance or another prespecified functional endpoint, an adaptive interpretation could become justified; if associated with worsened symptoms, an adverse interpretation would be justified; if neither relationship is demonstrated, the correct classification remains functionally

indeterminate. This rule protects the scientific meaning of an early positive physiological result, it does not need to be called therapeutic to be valuable, since a durable biological response would itself establish that repeated natural motion can alter the measured system over time. The question of benefit is a later and stronger evidentiary step.

15.2 Adaptive Vestibular Compensation

Vestibular neuroscience provides substantial evidence that experience-dependent reorganization can be functionally adaptive. Alberts and colleagues studied people with genetically defined progressive bilateral vestibular dysfunction and found participants increasingly relied on other sensory information as vestibular function declined, compensatory redistribution rather than a uniform collapse of postural control.[11] Assländer and Peterka found healthy adults dynamically reweighted sensory contributions to postural control within seconds when the reliability and magnitude of sensory perturbations changed experimentally, with the transition depending partly on the direction of the environmental change.[12] Sensory weighting is therefore not fixed, in either an impaired or an intact system.

Longitudinal vestibular-perturbation studies, already introduced in Sections 7.3 and 9.2, demonstrate that adaptive change can also develop over much longer periods. Dilda and colleagues found postural stability progressively recovered to approximately baseline over 12 weeks of repeated GVS exposure despite continued perturbation, with the adapted response detectable six months later, while the VOR response to the same stimulus did not show corresponding attenuation[24], suggesting the nervous system had not become globally less responsive, but that postural control had reorganized to operate effectively despite the unreliable signal. Tjernström and colleagues found a related pattern with repeated galvanic perturbations over five consecutive days, where the lower postural-response state reached after training remained evident three months later.[87] Vestibular rehabilitation frameworks place these observations within a broader clinical context, describing compensation after vestibular loss as involving adaptation, substitution, altered sensory weighting, and changes in postural strategy that collectively improve function.[33]

The scientific conclusion is strong: vestibular plasticity can be functionally adaptive. The boundary is equally important, these studies largely concern compensation for vestibular loss or adaptation to experimentally unreliable vestibular information,[11, 24, 33, 87] and do not demonstrate that increasing vestibular stimulation in an already intact system automatically improves function. A nervous system responding successfully to degraded or misleading vestibular information demonstrates plastic capability; it does not establish that additional natural motion will push an already intact system toward a superior functional state. For SeaKing, the relevance lies in capability rather than proof of benefit: the adult human vestibular system can reorganize across repeated exposure, that reorganization can persist for months, and the resulting change can improve functional performance under the condition to which the nervous system adapted.[24, 87] This makes adaptive change a legitimate prospective SeaKing outcome without establishing that it will occur; functional endpoints will be required to determine whether any longitudinal modification observed should be classified as adaptive.

15.3 Persistent Adverse Vestibular States, and Why They Do Not Prove One Causal Mechanism

The same vestibular system capable of adaptive reorganization can also be associated with persistent adverse states. Two clinical syndromes are particularly informative. PPPD is defined by persistent dizziness, unsteadiness, or non-spinning vertigo present on most days for at least three months and exacerbated by upright posture, movement, and complex visual stimuli.[15] Storm and colleagues found people with PPPD showed an altered sensory profile on visual and vestibular motion-perception tasks relative to healthy controls, including poorer processing of complex visual motion[144], supporting persistent disruption or reorganization of visual-vestibular processing rather than a simple model of peripheral vestibular loss. Neuroimaging adds network-level evidence: Li and colleagues identified altered within- and between-network functional connectivity in PPPD involving

visual and default-mode regions and areas implicated in monitoring the environment and regulating posture,[146] and a larger subsequent study found altered connectivity and network-topological characteristics across visual, precuneus, sensorimotor, multisensory vestibular, and cerebellar regions, with reduced network efficiency and connectivity associated with greater dizziness-related handicap.[147]

MdDS provides a complementary example because motion-triggered MdDS follows passive-motion exposure specifically. The Bárány Society diagnostic criteria define it as persistent oscillatory self-motion perception, typically rocking, bobbing, or swaying, beginning after passive-motion exposure.[16] Cha and colleagues used FDG-PET and resting-state fMRI in individuals with persistent motion-triggered MdDS (median symptom duration 17.5 months) and found differences in metabolism and functional connectivity involving entorhinal, amygdalar, visual, vestibular, and prefrontal regions[145], a persistent adverse self-motion-perception state remaining associated with measurable brain-network differences long after the original motion exposure. Systematic review confirms MdDS as a recognizable persistent motion-related clinical phenotype while emphasizing heterogeneity in proposed mechanisms and historically limited treatment evidence.[28]

These findings are highly relevant to the functional interpretation of vestibular neuroplasticity: they demonstrate that persistent alterations in vestibular-related perception and network function can accompany clinically significant disability.[15, 16, 144–147] They do not establish that ordinary or therapeutic vestibular exposure commonly causes these syndromes; PPPD can arise following multiple precipitating conditions and is not defined as caused by repeated natural motion,[15] and while MdDS has a more direct relationship with passive-motion exposure, the syndrome's existence does not establish its incidence after any specific type, intensity, duration, or pattern of motion.[16, 28]

Nor do these associations reveal the syndromes' complete neural mechanism, and this distinction matters because neuroimaging findings can appear mechanistically persuasive while remaining fundamentally correlational. The PPPD and MdDS network differences above are important because they demonstrate that the persistent clinical state is accompanied by measurable differences in brain organization, but they cannot determine whether those differences caused the disorder, predisposed the individual to it, emerged as a consequence of chronic symptoms, represent unsuccessful or successful compensation to some other process, or reflect interactions with anxiety, attention, or visual dependence.

Reviews of the PPPD neuroimaging literature reach the same boundary, noting that interpretation is complicated by clinical heterogeneity and potential contributions from anxiety, depression, personality characteristics, and preceding vestibular abnormalities.[29] The PPPD tDCS dissociation from 15.1 reinforces the same point directly: even when an intervention changes a measured neural variable, the resulting difference cannot automatically be interpreted as correction of the pathological mechanism.[8] For this review, maladaptive should therefore remain primarily a functional classification; a persistent state producing dizziness, disequilibrium, or impaired function is justifiably described as functionally adverse, while assigning a specific cellular or network mechanism requires additional causal evidence. If a SeaKing participant developed persistent adverse symptoms following natural-motion exposure, the occurrence would be clinically important even if the neural mechanism remained unknown; conversely, a persistent imaging, autonomic, pupillary, or electrophysiological change without symptoms would not justify labeling that change maladaptive. Mechanism and functional consequence must remain separate.

Correct interpretation is safety- and monitoring-relevant for SeaKing: persistent adverse vestibular states exist, and motion-triggered persistent adverse states specifically exist, so studies of repeated natural motion should prospectively monitor persistent dizziness, rocking, swaying, disequilibrium, visual-motion sensitivity, and nausea rather than assuming all durable motion-associated changes are desirable. This does not imply SeaKing-like exposure is expected to cause these syndromes; it means adverse persistence is biologically and clinically possible and should be measured.

15.4 Persistent Adverse States Can Be Modifiable

Classifying a state as maladaptive does not imply irreversible neural damage. MdDS provides direct evidence. Dai and colleagues treated 24 patients with persistent motion-triggered MdDS using a protocol pairing passive head movement with rotating optokinetic visual stimulation; 17 participants experienced complete or substantial recovery lasting four months or longer, six initially improved and relapsed, and one did not respond.[148] The study was uncontrolled and cannot establish efficacy relative to natural history or confirm the proposed vestibulo-ocular mechanism, but it demonstrates that substantial improvement can occur after symptoms have become persistent.

Mucci and colleagues subsequently evaluated a related protocol with a partial sham-control phase: sham exposure did not significantly change symptoms or posturography, while active treatment produced improvement in a subset of participants, with overall success less uniform than in the original uncontrolled report and response varying across clinical subgroups.[149] This preserves both sides of the evidence: persistent symptoms can improve, and improvement is not universal. A later randomized open non-inferiority trial comparing a virtual-reality implementation of the optokinetic readaptation protocol against the established booth approach found both groups improved without a significant difference between delivery methods;[150] lacking an untreated or sham arm it cannot independently establish treatment-effect size, but it further demonstrates modifiability of the persistent phenotype in at least some patients. Longitudinal evidence extends this: Dai and colleagues followed 141 people with classic motion-triggered or spontaneous-onset MdDS after an individualized four- to five-day readaptation protocol, finding initial improvement greater than 50% in 78% of the classic motion-triggered group and 48% of the spontaneous group, with significant improvement remaining at one year in 52% and 48% respectively[151], again lacking an untreated control, but demonstrating that persistent adverse symptoms can improve substantially and durably in a subset.

PPPD provides independent evidence of modifiability. A randomized, single-blind, sham-controlled trial of repetitive TMS in 66 adults with PPPD found greater improvement in dizziness and anxiety measures following active treatment than sham at post-treatment follow-up, with differences persisting through three months.[152] This does not establish that rTMS corrects one known PPPD plasticity mechanism; it establishes that a persistent adverse clinical state can be modified in a controlled human experiment. Not every neuromodulatory treatment produces superiority, however, the PPPD tDCS study found neural/perfusion differences but no significant active-versus-sham advantage on the primary dizziness outcome.[8] The combined evidence supports a balanced conclusion: persistent adverse vestibular states can be modifiable, but response is heterogeneous and treatment-specific mechanisms remain incompletely established.[8, 148–152] Conceptually, this means that where "maladaptive" is functionally appropriate terminology, the state it describes can itself remain plastic; persistence does not imply inevitable permanence. Both observations should be retained for SeaKing safety interpretation: MdDS demonstrates that persistent motion-related adverse symptoms can occur, and the treatment literature demonstrates that persistence does not imply irreversibility.

15.5 Functionally Indeterminate Change

A third category is necessary because many scientifically meaningful biological changes cannot immediately be classified as either beneficial or harmful. Adaptive vestibular compensation provides one endpoint of the spectrum, where sensory reweighting preserves or improves function when vestibular information becomes unreliable;[11, 12] persistent clinical vestibular syndromes provide the other, where PPPD and MdDS are associated with chronic symptoms and functional impairment.[15, 16] Between these lies a large class of observations in which physiology or neural activity changes but functional significance has not yet been demonstrated, the PPPD tDCS study is the direct controlled example, where regional cerebral blood-flow differences occurred following active stimulation without significant clinical superiority to sham.[8] The correct interpretation is neither that the neural difference was false nor that it was beneficial; its functional meaning was unresolved within that experiment.

This category is especially important in early-stage mechanistic research because physiological endpoints are often more sensitive than behavioral endpoints. Repeated natural motion might alter HRV, pupil dynamics, vestibular responsiveness, produce a baseline shift across sessions, or generate a physiological state detectable after exposure, any of which could represent important evidence that the intervention affects the nervous system, without their functional valence being known until linked prospectively to relevant function. The term functionally indeterminate should not be read as dismissive; it identifies a real biological effect whose meaning requires additional study, and it prevents two opposite mistakes: therapeutic inflation (measurable change → neuroplasticity → benefit) and adverse inflation (persistent change → abnormality → harm).

Neither sequence is scientifically justified without functional evidence. For SeaKing Phase 1, functionally indeterminate change should therefore be the default classification for persistent physiological effects unless benefit or harm is measured directly, a reproducible longitudinal HRV or pupil change, or a change in responsiveness across repeated exposures, would be physiologically meaningful and should be described according to what it demonstrates. If later experiments show such a state predicts improved acquisition, better retention, or reduced symptoms, its interpretation can advance in the adaptive direction; if it predicts adverse symptoms, in the opposite direction. Until then, indeterminate is the rigorous category.

15.6 SeaKing-Specific Adverse Risk Remains Unquantified

PPPD and motion-triggered MdDS establish that persistent adverse vestibular states are biologically and clinically possible, not the probability that SeaKing-like motion will produce them. This is particularly important for MdDS because passive-motion exposure is part of the diagnostic definition of the classic motion-triggered syndrome,[16] longitudinal treatment research confirms patients can experience persistent symptoms requiring specialized intervention,[151] and systematic review identifies prolonged passive motion, including travel by boat, aircraft, and automobile, as a common preceding context.[28] None of these sources provides a SeaKing-specific incidence rate: the literature does not establish the probability of persistent adverse symptoms following the motion characteristics, session duration, repeated-exposure count, heave/pitch/roll/yaw/surge/sway combination, participant population, or session spacing specific to the SeaKing research vessel. It would therefore be scientifically inappropriate either to assign a numerical risk based on the MdDS literature or to claim the risk is negligible because the syndrome is uncommon in ordinary clinical practice. The correct classification is that SeaKing-specific persistent adverse risk is currently unknown.

That uncertainty is experimentally manageable. Phase 1 can prospectively monitor acute tolerability and post-exposure persistence, with relevant outcomes including dizziness, nausea, rocking or swaying sensations, disequilibrium, visual-motion sensitivity, headache, and motion-associated anxiety. Follow-up beyond the immediate post-session period matters particularly because a persistent syndrome cannot be detected solely by asking how participants feel while aboard the vessel, and stopping criteria can be defined prospectively for individuals who develop unexpected or prolonged symptoms. These measures do not imply investigators expect a high incidence of persistent harm; they reflect the appropriate response to an unquantified risk for which relevant clinical phenotypes are known to exist. The treatment literature provides additional context, since persistent MdDS symptoms can improve substantially in some individuals[151] and PPPD is modifiable under some controlled treatment conditions[152], the adverse-state literature supports monitoring without requiring a deterministic view of harm. Natural motion is not established to be dangerous as a neuroplastic intervention, and it is also not entitled to a presumption that every persistent effect will be adaptive; the scientific standard is to measure both.

For SeaKing, the same repeated-session architecture that makes longitudinal physiological discovery possible also creates an opportunity for unusually careful safety characterization: acute tolerability, recovery after each exposure, changes across repeated sessions, and persistent post-exposure effects can all be quantified alongside motion and physiological data, allowing the study to distinguish acute tolerability from repeated-exposure adaptation from persistent adverse response

from persistent functionally indeterminate change from, eventually, persistent adaptive change. Plasticity is neither inherently beneficial nor inherently harmful; its functional meaning depends on what changes and what that change does. Human vestibular research demonstrates both durable adaptive compensation and persistent adverse clinical states,[11, 12, 15, 16, 24, 87, 144–152] and demonstrates that measurable neural change can occur without corresponding clinical benefit.[8] The appropriate SeaKing goal is therefore not to maximize neuroplasticity as an abstract quantity, but to determine whether repeated natural multidimensional motion produces measurable experience-dependent change, characterize the direction and durability of that change, and ultimately establish its functional meaning.

16. SeaKing-Specific Synthesis and Phase 1 Scientific Rationale

The preceding sections establish a substantial scientific foundation for studying repeated natural multidimensional motion while defining the limits of what can presently be claimed. Human vestibular systems are demonstrably plastic;[1, 2] natural motion produces structured multidimensional vestibular input rather than isolated single-axis stimulation;[3] controlled vestibular stimulation can influence acquisition of learned performance under selected conditions;[4] repeated vestibular responses can contain meaningful within-person structure;[5] vestibular input reaches human hippocampal and spatial-navigation networks;[25] repeated vestibular perturbation can produce durable adaptation;[24, 87] and the broader literature demonstrates that neural plasticity depends on stimulation parameters, timing, exposure history, context, developmental stage, and biological state.

At the same time, the evidence repeatedly rejects a simple progression from vestibular stimulation to therapeutic neuroplasticity: stimulation does not enhance every learning task,[6] combined vestibular inputs are not processed as the linear sum of isolated components,[7] acute facilitation can disappear when stimulation ends,[65] physiological or neural change can occur without clinical superiority,[8] learning can remain task specific and fail to transfer,[14] and candidate physiological measures do not automatically establish a specific neuromodulatory mechanism.[102, 103] The resulting SeaKing hypothesis should therefore be neither weak nor inflated: the literature provides strong biological and experimental rationale for determining whether repeated natural motion produces reproducible physiological responses and exposure-dependent changes that can later be tested against learning, without establishing those effects in advance. Phase 1 is the experiment that begins to close that gap.

16.1 The Strongest Defensible Hypothesis

The strongest current SeaKing hypothesis: repeated natural multidimensional motion is a biologically plausible candidate stimulus for producing measurable, reproducible, and potentially experience-dependent changes in human vestibular and physiological responses, creating a scientifically justified foundation for later direct tests of learning, retention, transfer, and functional outcome.

Five evidence streams support this. The adult vestibular system is capable of experience-dependent modification through both controlled VOR adaptation[1] and passive discrimination training that produces perceptual learning into later adulthood.[2] Natural human motion is a structured, quantifiable stimulus with distinct statistical properties across axes and activities, not an undefined environmental exposure.[3] Vestibular stimulation can interact with learning: nGVS enhanced acquisition of a complex functional-mobility task, with the advantage briefly outlasting stimulation.[4] Repeated vestibular responses can carry within-person structure that group averages obscure, marked between-person variation in ocular responses to standardized GVS alongside comparative within-person stability.[5] And vestibular input reaches systems relevant to spatial cognition, with caloric stimulation recruiting the hippocampus within a distributed vestibular network, establishing neural access, not hippocampal LTP or enhanced memory.[25]

These converge on plausibility, not efficacy, and three constraints keep the distinction sharp. Central vestibular neurons integrate canal and otolith inputs subadditively and frequency-dependently,[7] so the consequence of combined natural motion cannot be reconstructed by summing isolated

paradigms. A separate repeated-nGVS study found substantial practice-related learning with no stimulation advantage,[6] so vestibular modulation is not a universal learning enhancer. And a sham-controlled PPPD study produced measurable regional perfusion change without superiority on the primary dizziness outcome,[8] so biological change is not benefit.

The defensible hypothesis is therefore not that natural motion causes beneficial neuroplasticity, nor that it activates the hippocampus and vagus and thereby improves learning. It is that natural multidimensional motion is a biologically credible and experimentally measurable vestibular stimulus whose acute and repeated human effects warrant direct investigation. SeaKing does not begin from an arbitrary environmental exposure; it begins from a stimulus class connected to established vestibular plasticity, learning-related neural networks, demonstrated human learning effects, and repeated-response phenomena.[1–5, 25] What remains unknown is what this particular stimulus does.

16.2 What the Literature Does Not Establish

No verified human study demonstrates that SeaKing-like vessel motion produces neuroplasticity, and each supporting finding stops short of that claim. Natural-motion measurements establish the physical properties of self-motion exposure but not learning, adaptation, or benefit;[3] nonlinear canal-otolith integration establishes that combined motion is neurally distinct from a sum of components but not which vessel motions, if any, are optimal;[7] controlled nGVS enhanced one complex task but does not establish that natural mechanical motion reproduces the effect;[4] and individually structured GVS responses demonstrate repeatability under a standardized electrical stimulus but not stable physiological signatures during vessel motion.[5]

Mechanistic claims stop equally short. Passive whole-body rotation facilitates electrically induced hippocampal LTP through cholinergic septohippocampal mechanisms, and galvanic stimulation alters hippocampal proliferation and neurogenesis-related measures, both in animals, neither demonstrated in humans during natural motion, and neither established as beneficial.[26, 27] Acute vestibular facilitation can vanish the moment stimulation ends;[65] trained-task improvement can occur without superior near or far transfer;[14] and pupil responses are not specific evidence of locus-coeruleus engagement, with null taVNS findings across several protocols.[102, 103]

The literature therefore does not establish that SeaKing motion induces human hippocampal LTP or neurogenesis, enhances acquisition or consolidation, produces durable learning, produces near or far transfer, improves autism or POTS symptoms, reopens developmental sensitive periods, normalizes an autism-specific plasticity abnormality, activates a VNS-like locus-coeruleus or cholinergic gating pathway, creates a measurable "plasticity window," has an established optimal motion dose or session schedule, requires closed-loop physiological control, or produces clinical benefit. These are not weaknesses that invalidate the research program. They are the unanswered questions that define it; the literature has established enough component biology to justify asking them, without having answered them for the specific intervention proposed.

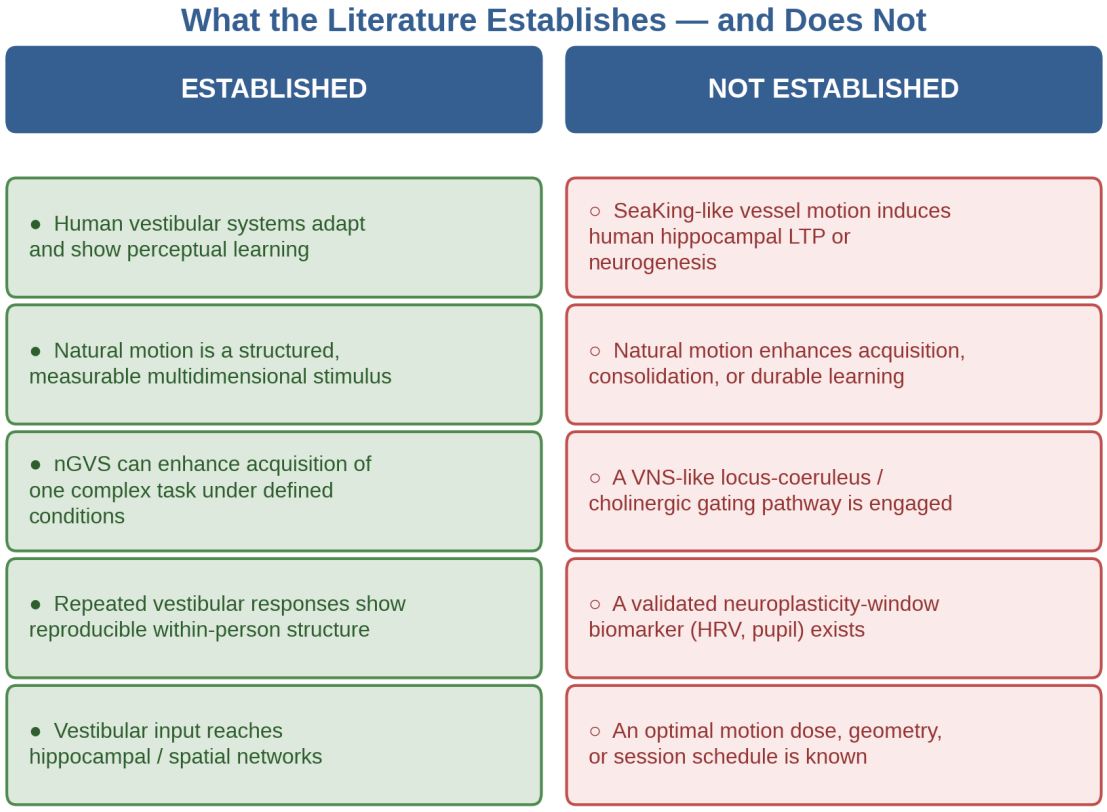

Figure 8. A synthesis of what the canonical evidence base establishes and does not establish about SeaKing's proposed mechanism. Both columns are drawn from the same body of literature reviewed in Sections 3 through 15.

16.3 What Phase 1 Can Validly Discover

Phase 1 can answer four questions directly with substantial scientific value. First, whether natural multidimensional motion produces measurable within-person physiological response structure, direct precedent exists in the MacDougall finding that GVS responses vary strongly between individuals while remaining comparatively stable within one.[5] Second, whether responses change with exposure history: Balter found postural sway declining after initial exposure and then stabilizing;[46] Tjernström found progressive between-session reduction persisting three months;[87] Dilda found more prolonged postural adaptation detectable six months later;[24] and Clément found long-lasting but reversible habituation.[85] These establish several plausible Phase 1 longitudinal patterns, stable repeatability, progressive attenuation, adaptation, habituation, between-session accumulation, persistence, recovery toward baseline, or different trajectories across different endpoints. Third, whether response expression depends on context or baseline state, for which the small but direct Shelhamer finding on context-linked VOR gains provides precedent.[45] Fourth, whether an apparent longitudinal effect exceeds nonspecific repeated-testing effects. Nooristani found both active nGVS and sham groups improved on repeated postural testing without a significant between-group difference,[88] demonstrating that repeated measurement itself can produce apparent improvement that Phase 1 must be designed to distinguish from motion-associated change.

The legitimate scientific output of Phase 1 is therefore not "proof of neuroplasticity." It is a quantitative description of the relationship among natural motion → acute physiological response →

recovery → repeated-session response trajectory → exposure history → participant-level heterogeneity, a relationship not yet characterized for SeaKing-like motion.

16.4 Why Phase 1 Is Likely to Yield Useful Information, Even From Null or Heterogeneous Results

A discovery study can produce valuable knowledge with direction-neutral hypotheses. A stable response is informative because it establishes reproducibility;[5] an early reduction followed by stabilization is informative because it establishes an exposure-history trajectory;[46] progressive adaptation is informative because it demonstrates longitudinal change;[24, 87] a null active-versus-control difference is informative because it constrains the intervention-specific hypothesis and may expose practice effects;[88] and substantial between-person heterogeneity with reproducible within-person responses is informative because it argues for individualized rather than purely group-mean analysis.[5] The broader plasticity literature adds an important boundary: candidate plasticity-induction measures can themselves show poor repeatability or substantial sham/time effects, as Boucher and Frieske both demonstrated in repeated theta-burst-stimulation paradigms.[81, 82] Phase 1 scientific yield therefore depends not merely on collecting large amounts of physiological data but on measurement quality, sufficient motion-exposure variation, reliable and synchronized sensors, explicit baseline/exposure/recovery periods, prespecified primary analyses, and separation of confirmatory from exploratory work.

Scientific success in Phase 1 should not be defined solely as a statistically significant directional shift in one candidate biomarker. A reproducible group-level response would establish a consistent physiological effect across participants; reproducible individual responses with a small group mean would indicate meaningful heterogeneity justifying participant-level modeling;[5] progressive longitudinal change would establish exposure-history dependence;[24, 46, 87] stable responses across repeated exposure would establish repeatability and argue against substantial longitudinal adaptation in that endpoint; a null motion-response association would identify an endpoint that does not merit priority under the tested conditions; and change also present under control or repeated testing would identify a nonspecific effect requiring the study to distinguish it from motion-associated change.[81, 82, 88] All of these outcomes reduce uncertainty.

A null result becomes scientifically weak only when the study cannot determine whether the absence of effect reflects inadequate measurement rather than genuinely little biological response, which a well-characterized design, measuring the motion stimulus directly and repeatedly sampling the same participants, is built to avoid. Phase 1 should therefore be evaluated on whether it maps response structure, not whether every candidate biomarker moves in a preferred direction. Stable heterogeneity can become a scientific finding; unstable heterogeneity remains noise until evidence shows otherwise.

16.5 What Phase 1 Cannot Claim

Phase 1 has a clear interpretation ceiling: even a highly successful physiological discovery study would not establish therapeutic neuroplasticity. Keywan's disappearing acute effect demonstrates that a response present during stimulation does not establish persistent change;[65] Nakagawa's motor-cortex expansion without behavioral improvement demonstrates that a neural plasticity-related effect does not automatically establish enhanced learning or function;[118] the null taVNS pupil and noradrenergic-marker findings demonstrate that a Phase 1 pupillary response could not establish locus-coeruleus engagement merely because the mechanism is theoretically plausible;[102, 103] and the PPPD tDCS dissociation demonstrates that measurable neural or perfusion change can occur without significant clinical superiority.[8]

Phase 1 cannot by itself prove beneficial neuroplasticity, enhanced learning, consolidation or long-term retention, near or far transfer, clinical efficacy, autism- or POTS-specific benefit, a validated neuroplasticity window, locus-coeruleus or VNS-like neuromodulatory engagement, hippocampal LTP or neurogenesis, an optimal therapeutic motion dose or intersession schedule, or a requirement for closed-loop control. Even a persistent physiological response would initially remain functionally

indeterminate unless linked prospectively to an independent functional outcome; an association between baseline state and subsequent response could identify a candidate moderator without validating that state as a biomarker of enhanced plasticity; and reproducible motion-response relationships may later justify testing whether real-time closed-loop control improves reproducibility or tolerability, without Phase 1 needing closed-loop control to prove such control necessary. These limitations define the study rather than diminish it, a discovery experiment should not be judged by whether it answers questions belonging to later efficacy and mechanism studies. Its responsibility is to establish the empirical foundation those later questions require.

16.6 Scientific Role of Phase 1 in the Staged Program

Phase 1 occupies a specific position: the literature already establishes several component capabilities, human vestibular systems adapt and learn,[1] natural motion can be characterized quantitatively and differs from simplified laboratory stimulation,[3] controlled vestibular stimulation can alter acquisition under selected conditions,[4] repeated vestibular responses can contain reproducible individual structure,[5] and repeated exposure can produce persistent adaptation[87], but what is missing is the direct bridge between these component findings and the SeaKing intervention itself. Without Phase 1, later experiments would need to choose motion parameters largely by analogy to isolated-axis, electrical, clinical, or animal paradigms; with Phase 1, later experiments can instead be based on empirically observed motion-response relationships.

If a reproducible acute response is discovered, subsequent work can ask whether that state affects learning; if repeated exposure produces adaptation, later studies can ask whether the adapted state alters acquisition or retention; if particular motion features consistently produce stronger or more tolerable responses, those features become candidates for controlled simulation; if response patterns differ strongly among participants, later studies can test whether phenotype or baseline state explains that heterogeneity; and if no important longitudinal physiology emerges, the program can redirect toward acute effects or alternative motion parameters. Each experiment determines what the next experiment is entitled to ask.

The central SeaKing proposition at the end of this synthesis is therefore precise: the literature provides strong evidence that the biological systems SeaKing proposes to engage are capable of experience-dependent change, that natural multidimensional motion constitutes a distinct and measurable vestibular stimulus, and that vestibular manipulation can influence human learning under selected conditions.[1, 3–6, 24, 87, 88] What remains unknown is whether repeated SeaKing-like natural motion produces reproducible physiological states or exposure-dependent changes relevant to later learning. Phase 1 is the appropriate direct experiment for answering that question. A positive result would not prove therapy, it would convert the SeaKing hypothesis from a literature-derived inference into a directly characterized human stimulus-response phenomenon. A null or heterogeneous result would materially constrain the hypothesis. Either outcome advances the program, because Phase 1 is designed to determine which features of the proposed mechanism, if any, are actually present in humans exposed to quantified natural multidimensional motion, not to confirm a predetermined therapeutic mechanism.

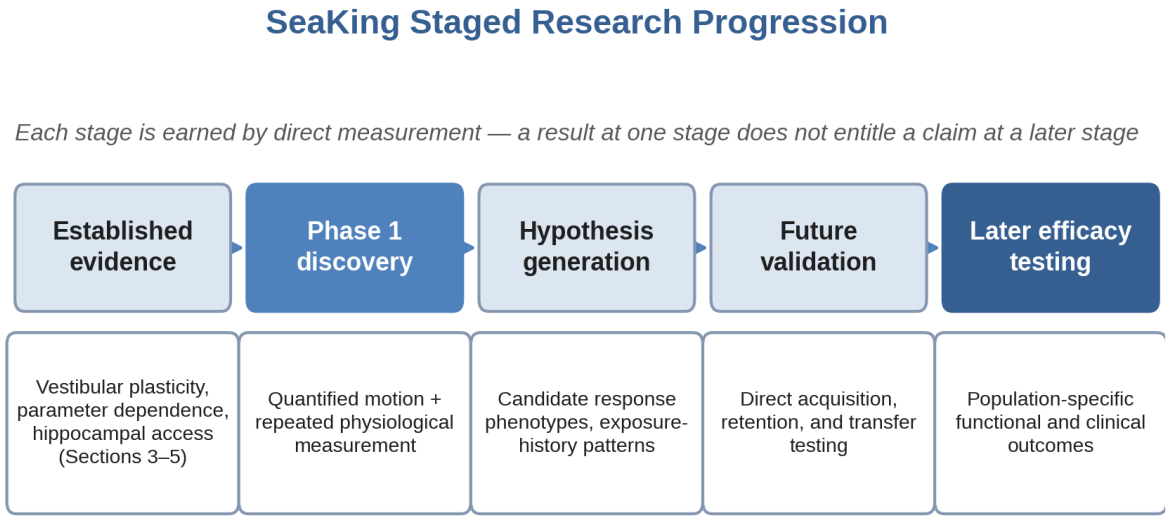

Figure 9. The staged SeaKing research progression. Each stage must be earned by direct measurement before the next scientific claim becomes appropriate; Phase 1 corresponds to the discovery stage only.

17. Staged Endpoint Architecture for Later SeaKing Studies

The evidence reviewed throughout this paper supports a staged rather than collapsed model of SeaKing research. A measurable physiological response is not equivalent to learning; learning during exposure is not equivalent to retained learning; retention is not equivalent to transfer; transfer on a structured task is not equivalent to spontaneous real-world behavior; and none of these, independently, establishes meaningful clinical benefit. Sections 6 through 8 already demonstrated each dissociation directly: vestibular stimulation enhanced acquisition under one task condition while failing under another,[4] motor learning continued to change after practice ended,[9] training methods produced similar immediate performance but different later maintenance,[10] trained-task improvement occurred without near or far transfer,[14] domain-specific cognitive improvement occurred without superior real-world outcome,[13] and measurable neural change occurred without superiority on a primary clinical endpoint.[8] The resulting architecture is sequential, physiological response → acquisition → delayed retention → near transfer → far transfer → spontaneous real-world behavior → meaningful functional or clinical outcome, not a claim that every SeaKing effect will progress through every stage, but a framework for determining how far any observed effect actually extends, with each downstream claim requiring its own directly measured endpoint.

17.1 The Eight-Stage Architecture

Stage 1 is biological responsiveness: Phase 1 can determine whether motion is associated with reproducible physiological responses that change across repeated exposures, building on the MacDougall precedent that fixed stimuli can produce highly individual yet reproducible within-person patterns[5], establishing response structure, not learning. Stage 2 is exposure-dependent change or adaptation, per Section 16.3.

Stage 3 is acquisition, where the Putman nGVS finding establishes that vestibular intervention can influence learning itself, though its task specificity prevents treatment as general learning enhancement.[4] Stage 4 is retention and consolidation: since performance can continue to change after practice ends[9] and training conditions can produce later differences not evident immediately,[10] an effect measured during or immediately after SeaKing exposure requires delayed testing before durable learning can be claimed. Stages 5 and 6 are near and far transfer, neither impossible

nor guaranteed, improvement can generalize beyond the trained condition[10, 71] just as controlled training can produce large practiced-task gains without corresponding transfer.[14] Stage 7 is spontaneous real-world behavior, where a participant may demonstrate a capability under explicit testing while failing to use it consistently in ordinary settings, and where rehabilitation and autism evidence shows generalization across people and contexts can occur but must be measured rather than assumed.[10, 71] Stage 8 is meaningful functional or clinical benefit, where Landínez-Martínez's domain-specific gains without instrumental-activity superiority[13] and the PPPD tDCS dissociation[8] both stand as direct boundaries.

The arrows represent increasingly strong research questions, not an assumed causal progression: a result at Stage 1 does not entitle a claim at Stage 3, a result at Stage 3 does not entitle Stage 5, and a result at Stage 5 does not establish Stage 8. This framework is valuable for SeaKing precisely because early physiological results may be highly informative before clinical efficacy is tested, Phase 1 does not need to establish therapeutic benefit to succeed scientifically; its role is to determine whether the first stages of the proposed biological chain exist, so later studies can test the next stages directly.

17.2 Direct Learning, Retention, and Transfer Tests

Once natural-motion response characteristics are sufficiently defined, the next question is whether a selected motion condition alters acquisition of a defined learned capability. The Putman precedent provides direct rationale, and its own manual-control null, together with Sherman's independent finding that nGVS did not accelerate learning on a lunar-rover navigation task despite clear practice-related improvement[4, 6], supplies the design warning: a future SeaKing acquisition experiment should not assume any sufficiently complex task will respond to motion, but should select the task prospectively, measure acquisition across repeated practice rather than single-exposure performance, use an appropriate control (since participants can improve substantially simply through practice[6]), prespecify the learning outcome, and continue testing after the motion condition ends, the last point directly motivated by Keywan's finding that nGVS-improved thresholds returned toward baseline the moment stimulation stopped.[65] The correct acquisition question is therefore "does practice under a defined motion condition alter the trajectory of learning relative to an appropriate comparison?" rather than "does performance look better while in motion?"

Any acquisition effect should be followed by prespecified delayed retention testing, since the state at the end of training need not represent the state expressed later, as both the Walker/consolidation-network evidence[9, 66] and the Powell 30-day maintenance finding[10] established in Sections 7.1 and 8.5. For vestibular-enhanced learning specifically, Putman's retention interval was only approximately 15 minutes,[4] establishing short retention, not durable consolidation over days or weeks. A useful temporal architecture distinguishes immediate post-exposure, later same-day, next-day, and longer delayed retention, with duration reported directly rather than described vaguely as "long-term neuroplasticity", permitting mechanistically informative dissociations (enhanced acquisition that fades by the next day; no immediate advantage but better delayed retention; both improved; or physiological response without any learning effect) that the present literature does not resolve in advance.

If a learning advantage persists, transfer should be tested by explicit distance rather than one broad "generalization" endpoint: trained task, closely related untrained task (near transfer), a meaningfully different capability (far transfer), a different person or environment (cross-context generalization), and ordinary behavior without explicit prompting (spontaneous generalization). Section 8 already established both directions of this evidence, Schaefer's limited motor transfer against Dipietro's workspace generalization,[30, 31] Carruthers' directional cross-context generalization in autistic children against Cuneo's acquisition-generalization dissociation,[32, 72] and Landínez-Martínez's and Zhong's null transfer and near/far-transfer findings despite genuine trained-task gains.[13, 14] A failure at one stage does not invalidate earlier stages: task-specific learning without near transfer, bounded-range effects with near but not far transfer, and capability-without-independent-deployment when cross-context performance improves but spontaneous behavior does not are all informative

outcomes rather than failed experiments. This structure matters most for later autism and POTS/rehabilitation studies, where laboratory performance and everyday participation may diverge.

17.3 Meaningful Functional Outcomes, Moderators, and Biomarker Validation

The highest translational threshold is meaningful function, not neural change or even generalized learning. Section 8.5 already demonstrated this distinction directly: Fleming's null metacognitive-component trial after traumatic brain injury,[73] Powell's positive counterpart where superior maintenance emerged despite similar immediate performance,[10] Landínez-Martínez's domain-specific-without-instrumental-activity result,[13] and the PPPD tDCS dissociation[8] together with the broader rehabilitation literature[76, 77] establish that physiological response is not clinical benefit, neural change is not clinical benefit, trained-task improvement is not clinical benefit, and transfer is not automatically clinical benefit. A claim of benefit requires direct evidence at the level of function or symptoms, for future autism research, validated measures of adaptive function, communication, participation, or regulation; for POTS research, validated symptom burden, orthostatic tolerance, or daily activity, with precise measures determined at protocol development. This hierarchy protects later SeaKing studies from both overclaiming and underclaiming: an early mechanistic result remains valuable before efficacy is demonstrated, and a later functional result becomes more convincing because the intervening steps were measured rather than assumed.

Repeated SeaKing studies should treat exposure history, biological state, development, and phenotype as prospectively testable moderators, not unexplained noise, but not automatic evidence of personalized response either. Section 11 already established the state-dependence evidence,[94, 97, 99] Section 13 the developmental evidence,[124, 126, 129] and Section 14 the autism-phenotype and representativeness evidence,[71, 142] all directly relevant here. The distinguishing requirement, per Sections 9 and 11's reliability literature, is that a candidate moderator must be measured before the outcome, predict the subsequent response, and replicate, variability alone does not establish personalization, and a plausible moderator identified retrospectively is not automatically a biological mechanism.[81, 82] The repeated-measures structure of SeaKing is well suited to this, since each participant contributes multiple baseline-state and response observations, permitting between-person and within-person analyses to remain distinct rather than conflated.

Biomarker validation follows the same logic developed in Section 11.4–11.6: reproducibility, as MacDougall demonstrated,[5] is necessary but not sufficient; the pupillometry literature shows a physiological measure can be responsive under one protocol, null under another, affected by nonspecific sensory events, or only indirectly related to the proposed mechanism,[100–104] and the PPPD tDCS study shows even a sophisticated neural measure cannot validate itself as a marker of benefit.[8] A rigorous sequence therefore runs candidate signal → test-retest reproducibility → prospective association with an independent endpoint → replication → only then, consideration as a validated biomarker. HRV cannot be validated as a neuroplasticity biomarker because HRV changed; pupil dilation cannot be validated as an LC biomarker because the pupil dilated; a physiological state cannot be validated as learning-enhancing unless learning is independently measured.

17.4 Safety and Functional Valence Must Remain Explicit

Every later SeaKing protocol should preserve the adaptive/adverse/functionally-indeterminate distinction developed in Section 15 throughout: adaptive sensory reweighting,[11, 12] persistent adverse states including PPPD and MdDS,[15, 16, 144] and physiological change without established functional consequence, exemplified again by the PPPD tDCS dissociation,[8] are all live possibilities requiring separate measurement, did a change occur, did it persist, was it associated with improved function, was it associated with adverse symptoms, or did its functional significance remain unresolved. This prevents investigators from assuming persistence is beneficial merely because the project seeks neuroplastic effects, and from labeling persistent change harmful without evidence of adverse consequence. Prospective monitoring should include acute tolerability, recovery after exposure, longitudinal symptom trajectories, and post-session persistence, with particular

attention to rocking or swaying, dizziness, disequilibrium, nausea, and visual-motion sensitivity, the existence of MdDS and PPPD establishes that persistent adverse vestibular phenotypes are possible without establishing a SeaKing-specific incidence,[15, 16] justifying surveillance without a deterministic view of harm. At later efficacy stages, safety and benefit should remain parallel domains: a study should ask not only whether participants improved, but whether a subgroup experienced persistent worsening, and a directionally desirable physiological endpoint should not be presumed beneficial unless its functional consequence is measured directly.

The evidence-derived architecture for later SeaKing research is therefore deliberately staged: Phase 1 characterizes motion, acute response, reproducibility, exposure history, and candidate moderators; a subsequent bridge study tests acquisition directly; delayed assessments determine retention; separate untrained outcomes test near and far transfer; cross-context and spontaneous-behavior measures establish whether learning extends outside the laboratory; and population-specific efficacy studies ultimately determine meaningful functional or clinical outcomes. Throughout, candidate biomarkers must be validated against independent endpoints, heterogeneity must be modeled prospectively, and persistent changes must be assigned functional meaning from demonstrated consequences rather than from direction or duration alone. This structure does not assume SeaKing will succeed at every stage. It ensures that if an effect is discovered, the research program can determine what the effect actually is, how long it lasts, how far it generalizes, for whom it occurs, and whether it meaningfully helps.

18. Conclusions and Research-Program Implications

The central question of this review was whether the scientific literature provides a credible basis for investigating repeated natural multidimensional motion as a candidate stimulus relevant to neuroplasticity. The answer is yes, with important boundaries.

What the literature establishes is a substantial chain of experimentally demonstrated biological capabilities, developed across Sections 3 through 15: human vestibular systems adapt and learn;[1, 2] natural motion generates structured multidimensional vestibular input;[3] vestibular stimulation can modify acquisition of learned performance under selected conditions;[4] repeated vestibular responses can contain reproducible within-person structure;[5] vestibular input reaches human hippocampal and spatial-processing networks;[25] experience-dependent cortical plasticity can be selectively shaped by neuromodulatory activity;[105] and learning-related neural and behavioral change remains demonstrable beyond childhood, including in autistic adults.[71, 129, 131] At the same time, the literature consistently demonstrates that these processes are conditional, stimulation does not enhance every learning task,[6] some cognitive outcomes remain unchanged,[63] biological changes do not always produce behavioral benefit,[8] human neuromodulation studies contain controlled null findings,[119] and autism-related plasticity shows no universal direction.[129, 131] These findings constrain the SeaKing hypothesis without removing its scientific rationale. Section 16.1 states the resulting hypothesis in full: natural multidimensional motion is a biologically credible and experimentally measurable vestibular stimulus whose acute and repeated human effects warrant direct investigation, plausible, not yet demonstrated, and testable rather than assumed. Section 16.2's inventory of what remains unknown or rejected, generic cognitive enhancement, human hippocampal neurogenesis, durable plasticity from acute facilitation, a validated dose-response rule, a validated neuroplasticity biomarker, a single autism-plasticity direction, and neural change standing in for functional benefit, defines the research agenda rather than undermining it: each item indicates exactly what future experiments must measure rather than infer.

Phase 1 is the appropriate next scientific step for the reasons Section 16.3 through 16.6 developed directly: it is positioned not to demonstrate efficacy but to determine whether the first SeaKing-specific links in this chain exist, and the direct vestibular evidence, stable individual response structure,[5] habituation followed by stability,[46] between-session adaptation persisting months,[87] and more prolonged postural reorganization persisting six months[24], establishes that repeated vestibular measurement is capable of revealing several scientifically meaningful trajectories under

well-controlled conditions. The resulting research progression is therefore staged: characterize motion and physiological response; establish reproducibility and exposure-history effects; test acquisition directly; test delayed retention; test near and far transfer; test cross-context and spontaneous behavior; test population-specific functional and clinical outcomes; and only then investigate mechanisms, moderators, biomarkers, and optimization, with the next claim earned by direct measurement at every stage. This structure allows the program to remain scientifically ambitious without requiring unsupported certainty.

The literature already establishes that the nervous systems under consideration possess the relevant capacities for experience-dependent change. The unanswered question is whether repeated natural multidimensional motion can recruit those capabilities in a useful and reproducible way, a question no literature review can answer, and only direct experimentation can. Phase 1 does not presume that natural vessel motion is therapeutic, does not require that every physiological endpoint respond, and does not require that all participants respond in the same way. It asks a more fundamental question: whether a quantitatively characterized natural motion environment produces reproducible human physiological phenomena that justify progressively stronger tests of adaptation, learning, retention, transfer, mechanism, and benefit. If reproducible motion-response relationships are found, the SeaKing hypothesis advances from biological plausibility to direct human evidence; if repeated exposure produces systematic change, longitudinal adaptation becomes directly testable; if stable individual differences emerge, participant-level response phenotypes can be investigated prospectively; and if no meaningful response is found under the tested conditions, the parameter space and mechanistic model narrow. Each outcome improves the scientific model.

The conclusion of this review is therefore deliberately stronger than mere speculation and deliberately narrower than an efficacy claim: the published evidence establishes a credible, multi-level biological foundation for investigating repeated natural multidimensional vestibular motion as a candidate modulator of human physiological state and later learning. The intervention-specific effects remain unproven, but the unanswered questions are experimentally tractable. A carefully designed Phase 1 repeated-measures study is scientifically justified and has a strong likelihood of generating the information required to determine whether the SeaKing hypothesis should advance toward direct tests of neuroplasticity, learning, retention, generalization, and clinical function.

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