The SeaKing Solace Hypothesis: A Consolidated Scientific Rationale for Investigating Natural Multidimensional Motion

Scientific White Paper, SeaKing Solace Hypothesis Paper

Document Status

This scientific white paper consolidates the evidence and boundaries established across SeaKing Solace Scientific Reviews 01 through 05 into a single canonical hypothesis and research architecture. It has not been peer reviewed or published by an academic journal. It does not present new primary literature review; it synthesizes propositions already established in the five prior reviews, supplemented selectively by direct citation to original studies where a specific study-level claim requires it. The underlying studies cited throughout this paper include peer-reviewed publications; their inclusion should not be interpreted as independent validation of SeaKing Solace's hypotheses or proposed applications.

PART I. FOUNDATIONS

1. Scientific Question

1.1 Define the Exact Question

Externally applied physical motion is a structured biological vestibular stimulus rather than a binary motion or no-motion exposure [1]. Natural vestibular exposure is multidimensional and time-varying, biologically distinct from a simple sum of isolated axes: concurrent canal and otolith signals interact nonlinearly and contextually, and their relevance can depend on frequency, amplitude, temporal structure, orientation, cross-axis relationships, behavior, and exposure history [1, 2]. These two established properties define the physical input at the center of this paper's central scientific question.

The exact question is this. Does quantified natural multidimensional motion, delivered under real or closely approximated maritime conditions, contain reproducible, temporally structured physiological input-output relationships in humans, and do those relationships show recovery, exposure-history dependence, or stable individual heterogeneity? This is a discovery question about measurable physiological structure. It is not a question about therapeutic benefit, and it does not presuppose a specific mechanism, disease application, or clinical outcome.

1.2 Why Current Literature Does Not Answer It

The existing human literature contains substantial precedent that motion-rich exposure can produce measurable outcomes, but that literature typically does not isolate the physical motion stimulus from the surrounding intervention [2]. Evidence for a whole intervention and evidence for motion itself are different claims. Causal resolution increases progressively from package-level efficacy, to active-comparator designs, to component manipulation, to direct physical-stimulus manipulation, and even high-resolution motion manipulation remains specific to the tested protocol and population [2]. Physical motion can be measured quantitatively, yet the actual physical stimulus is incompletely characterized across much of the motion-rich clinical literature; this measurable-but-unmeasured gap directly justifies prospective quantitative characterization of natural multidimensional motion [2, 1]. The gap does not identify an optimal waveform, and it does not establish efficacy for any specific intervention [2].

This means the existing literature, including the human vestibular plasticity and learning evidence developed in Review 04, can motivate the present question without answering it [3]. No source identified across Reviews 01 through 05 establishes that natural, quantified, maritime-relevant multidimensional motion produces reproducible physiological structure in humans, because that specific exposure has not been measured and tested directly as an experimental variable within the evidence base synthesized here.

1.3 The Immediate Research Gap

Phase 1 is a discovery and early system-identification study designed to determine whether measured natural head-centered motion contains reproducible, temporally structured physiological input-output relationships, and whether those relationships show recovery, exposure-history dependence, or stable heterogeneity [1, 4, 5, 3, 2]. This has not yet been demonstrated, and Phase 1 does not itself establish learning or neuroplasticity even if a positive result is obtained.

The immediate research gap is therefore narrow and specific: whether natural multidimensional motion, measured with adequate temporal and spatial resolution at or near the participant's head, produces physiological responses that recur within participants, relate systematically to features of the delivered motion, and behave in scientifically interpretable ways across repeated exposure. This gap defines what Phase 1 is designed to close, and it is addressed in full in Part V of this paper.

2. Why This Hypothesis Is Necessary

2.1 Convergence of Five Prior Reviews

The SeaKing program is evidence-gated but not a single strictly linear ladder. One branch develops stimulus causality, moving from characterization through association, replication, challenge of alternative explanations, controlled reproduction, reduction and manipulation, to dose-response mapping. A linked outcome branch tests whether validated physiological states affect acquisition, retention, transfer and generalization, and real-world or clinical function. Disease-specific testing, personalization, and adaptive control occur only after the prerequisite evidence for the relevant branch exists [1, 4, 5, 3, 2]. This architecture, developed across the five prior reviews in this series, provides the backbone for the hypothesis presented in this paper.

Each prior review contributes a distinct and complementary component. Review 01 establishes the physical-input problem: how natural motion produces structured, multidimensional, head-centered

vestibular stimulation. Review 02 establishes the vestibular-autonomic interface, including direct human evidence that vestibular stimulation can modulate sympathetic outflow. Review 03 establishes the architecture and evidentiary boundaries of autonomic measurement itself. Review 04 establishes human vestibular plasticity and its conditional relationship with learning. Review 05 establishes that motion-rich human interventions produce measurable outcomes while leaving the physical motion stimulus largely uncharacterized. This paper does not re-review each of these five papers in detail; it consolidates their established propositions into one coherent scientific hypothesis and research architecture.

2.2 The Measurable-but-Unmeasured Gap

Physical motion can be measured quantitatively, yet the actual physical stimulus is incompletely characterized across much of the motion-rich clinical literature [2]. This measurable-but-unmeasured gap is not a claim that motion cannot be quantified; controlled laboratory and biomechanical studies demonstrate that it can [2, 1]. It is a claim about what has and has not been done in the existing intervention literature, where dose values for natural multidimensional motion remain unknown even when session structure, duration, and population are well described.

This gap is the central methodological justification for the SeaKing hypothesis. If natural motion could not be measured with adequate rigor, direct characterization would not be a tractable research step. Because it can be measured, and because existing intervention research largely has not measured it, prospective quantitative characterization of natural multidimensional motion becomes both feasible and necessary.

2.3 Why Inference Must Become Experiment

A candidate natural motion-response relationship becomes mechanistically stronger only if it survives controlled reproduction, in which the candidate exposure is delivered under controlled conditions and the physiological response recurs [1, 4, 5]. Mechanical replay alone does not guarantee biological reproduction; the physiological response itself must be shown to recur under control. Because naturalistic measurement cannot eliminate all confounding or competing mechanisms, including respiration, visual context, posture, cardiovascular state, motion sickness, and participant behavior, direct controlled reproduction is required to move beyond naturalistic association [2].

This is the scientific argument for why the SeaKing program cannot remain at the level of literature synthesis or naturalistic observation. Association observed during real maritime exposure, however carefully measured, cannot by itself establish that motion is the causally relevant variable. Only controlled, direct experimental testing can determine whether the associations this paper hypothesizes are real, reproducible, and attributable to motion rather than to a co-occurring factor. This paper does not anticipate the results of that testing; it establishes why the testing is the necessary next scientific step.

3. Scope and Boundaries

3.1 What This Paper Is

This paper is an evidence-derived scientific hypothesis and research architecture [2, 1]. It consolidates propositions already established across Reviews 01 through 05 into a single canonical statement of what is known, what remains uncertain, and what a staged research program would need to demonstrate at each stage to justify progression. It defines a falsifiable central hypothesis for Phase 1, an explicit set of boundaries on what that hypothesis does and does not claim, and a staged evidentiary architecture connecting physiological discovery to eventual learning and clinical translation.

This paper is not a protocol, an efficacy paper, or a product claim, and it draws on the autonomic, plasticity, and human intervention evidence developed across Reviews 02 through 04 only to the

extent that each contributes to the consolidated hypothesis and boundaries stated here [4, 3, 5]. It does not specify exact statistical thresholds, sample sizes, or measurement instrumentation; those belong in a separate statistical analysis plan. It does not report new experimental data. Its function is to state, as precisely as the existing evidence allows, what scientific question the SeaKing program is asking and why that question is worth asking now.

3.2 What This Paper Does Not Claim

No current evidence establishes that SeaKing-like natural motion induces human hippocampal long-term potentiation or neurogenesis, reopens developmental sensitive periods, creates a validated plasticity window, has an optimal neuroplastic dosing schedule, or improves autism or postural orthostatic tachycardia syndrome (POTS) through neuroplasticity [3]. Disease-specific physiology and clinically meaningful outcomes are separate evidence gates, and healthy-adult Phase 1 findings cannot establish benefit in POTS, autism, or any other population [4, 5, 2, 1, 3]. Reproducible physiological phenotypes are not automatically therapeutic, plasticity, or surrogate biomarkers; responsiveness or prediction alone does not establish mechanistic or surrogate validity [3, 4].

This subsection functions as the primary global claim firewall for the entire paper. No current evidence establishes therapeutic neuroplasticity, clinical benefit, or SeaKing efficacy of any kind. Every hypothesis, evidence gate, and research-architecture statement that follows in this paper is subordinate to this boundary, and no later section should be read as relaxing it.

3.3 Epistemic Rule

A measured signal changing during a defined exposure is an observation, and it must not be presented as though it were already a validated physiological inference, a multimodal physiological pattern, a mechanistic finding, or a functional or clinical benefit. These are five distinct claim levels, each requiring its own evidence: observation, validated physiological inference, multimodal physiological inference, mechanistic inference, and functional or clinical benefit. Prediction does not establish mechanism, and statistical, machine-learning, clustering, latent-variable, or composite outputs remain data-derived constructs until independently validated, since high predictive accuracy can still exploit correlated noncausal variables [4].

This claim ladder is stated once here and is not re-derived in later sections; where a later section makes or evaluates a claim, it should be read against the five levels defined in this subsection. It follows directly from the evidentiary discipline developed in Reviews 03 and 04, and the ladder itself is developed further, with SeaKing-specific examples, in Part IV [5, 3].

PART II. BIOLOGICAL FOUNDATION

4. Motion as a Biological Stimulus

4.1 External Motion as Vestibular Input

Externally applied physical motion is a structured biological vestibular stimulus rather than a binary motion or no-motion exposure [1]. This is an established physical and physiological principle rather than a SeaKing-specific finding, and it applies before any downstream physiology is considered: the vestibular organs transduce head acceleration and orientation continuously, so any externally imposed motion delivers a graded, information-bearing input rather than a simple on or off condition.

This subsection establishes the physical input alone. It does not address whether that input reaches autonomic circuitry, whether it interacts with learning, or whether it produces any measurable downstream effect; those questions are addressed in Sections 5 and 6.

4.2 Natural Multidimensional Structure

Natural vestibular exposure is multidimensional and time-varying, biologically distinct from a simple sum of isolated axes. Concurrent canal and otolith signals interact nonlinearly and contextually, and their relevance can depend on frequency, amplitude, temporal structure, orientation, cross-axis relationships, behavior, and exposure history [1, 3]. This property directly justifies direct, high-resolution characterization of natural multidimensional motion rather than inference from single-axis laboratory paradigms, since a single-axis stimulus cannot be assumed to represent the physiological consequences of combined, naturally occurring motion.

The SeaKing-relevant combination of features within this multidimensional structure remains unknown. Establishing that natural motion is multidimensional does not identify which combination of axes, frequencies, or temporal patterns is physiologically important; it establishes only that the exposure cannot be adequately described by a single scalar quantity, and this paper makes no superiority claim for any one motion profile over another.

4.3 Head-Centered Exposure

Received head motion is a more proximal description of vestibular exposure than vessel or platform motion alone, and it may differ from platform motion because of posture, stabilization, voluntary movement, and participant behavior [1, 4]. A vessel's own motion is not identical to the motion experienced at a participant's head, since posture and active postural correction can amplify, dampen, or redirect the platform's motion before it reaches the vestibular organs.

Whether head-level measurement improves physiological prediction relative to platform-level measurement is an empirical question rather than an established fact. This subsection therefore creates a falsifiable Phase 1 exposure-specificity hypothesis: that head-centered motion measurement adds explanatory value beyond vessel motion alone. Section 15.1 and Evidence Gate 2 in Part IV return to this question directly.

5. Vestibular Access to Autonomic Systems

5.1 Anatomical and Direct Human Bridge

Vestibular nuclei have anatomical access to autonomic and cardiovascular circuitry, and vestibular stimulation can modify directly recorded sympathetic activity in humans [4]. Direct human microneurography provides the strongest form of this evidence. Sinusoidal galvanic vestibular stimulation has been shown to modulate muscle sympathetic nerve bursts in human subjects [6], and vestibular and pulse-related modulation of skin sympathetic nerve activity has been demonstrated using the same sinusoidal galvanic paradigm [7]. A more mechanically relevant human bridge exists as well: low-frequency physiological activation of the vestibular utricle modulates muscle sympathetic nerve activity in awake humans without requiring electrical stimulation [8].

Together these three sources provide the core physiological bridge from motion to autonomic response that this hypothesis depends upon. They establish that a defined vestibular pathway has direct, recorded access to human sympathetic outflow. They do not establish that natural maritime motion produces beneficial autonomic effects, and the electrical and low-frequency mechanical paradigms used in these studies are not natural multidimensional vessel motion.

5.2 State-, Stimulus-, and Effector-Dependence

Vestibulo-autonomic responses are stimulus-, state-, and effector-dependent rather than uniformly directional [4, 5]. The same vestibular input can produce different sympathetic outcomes depending on which effector is measured, and depending on the physiological or behavioral state of the participant at the time of stimulation. This conditional pattern is consistent across the direct human evidence reviewed in Section 5.1, where muscle and skin sympathetic responses to related vestibular stimuli were not identical [6, 7, 8].

The SeaKing-relevant state variables and response functions that would govern this dependence in the context of natural maritime motion remain unknown. This subsection therefore supports state-dependent modeling as a Phase 1 analytic requirement and supports later, evidence-gated tests of conditional personalization; it does not itself specify which states matter or how they should be modeled.

5.3 Multidimensional Autonomic Interpretation

Autonomic control is distributed, regional, dynamic, and effector-specific rather than a single global sympathetic-parasympathetic axis [5]. Global scalar labels for autonomic state are not supported by routine biomarkers. This principle has a long foundation in autonomic physiology: the autonomic nervous system is organized with substantial regional specificity to support precise, effector-specific regulation of homeostatic and protective functions rather than uniform whole-body activation [9], and classic work on autonomic space explicitly rejects a single reciprocal sympathetic-parasympathetic dimension in favor of independently varying autonomic modes [10].

No single practical noninvasive biomarker measures the autonomic nervous system as a whole. Heart rate variability, electrodermal activity, pupil diameter, blood pressure, respiration, pre-ejection period, and related signals each retain domain-specific meaning, and each carries distinct confounds and limited mechanistic specificity [5, 4]. Heart rate variability in particular can inform cardiac parasympathetic modulation under appropriate conditions, but it is not a direct measure of whole-vagus activity or global autonomic health, and its interpretation requires respiratory, heart-rate, posture, duration, and state context; internationally recognized measurement standards make this same interpretive requirement explicit [11]. Multimodal convergence across non-equivalent channels can strengthen evidence that a real multidomain physiological response occurred, and divergence across channels can be physiologically informative in its own right rather than representing measurement failure; neither convergence nor divergence alone establishes mechanism or benefit [5, 4].

This subsection defines how SeaKing physiological outputs must be interpreted throughout the remainder of this paper. No later section should collapse multiple physiological channels into a single global autonomic score, and no later section should treat heart rate variability alone as evidence of whole-vagus engagement.

6. Human Capacity for Adaptation and Plasticity

6.1 Human Vestibular Plasticity

Human vestibular systems retain measurable capacity for experience-dependent adaptation and perceptual learning across sensorimotor and perceptual domains [3, 1]. Direct human evidence supports this capacity at two levels. Controlled visuo-vestibular mismatch can rapidly potentiate incremental vestibulo-ocular reflex adaptation [13], demonstrating that the human vestibular sensorimotor system can recalibrate in response to controlled experience. Separately, passive vestibular perceptual learning has been demonstrated to improve self-motion perception, posture, and gait in older adults, showing that this capacity for experience-dependent change persists into later adulthood rather than being confined to a narrow developmental window [14].

This capacity is a basic biological prerequisite for the SeaKing hypothesis. It does not establish SeaKing-specific plasticity, and it does not establish that any particular natural motion exposure will produce adaptation or learning; it establishes only that the human vestibular system is, in principle, capable of the kind of experience-dependent change the SeaKing hypothesis proposes to investigate.

6.2 Vestibular Interaction With Learning

Vestibular stimulation can modify human learning acquisition under selected conditions, but controlled null findings show that it is not a universal learning enhancer [3]. Noisy galvanic vestibular

stimulation has been shown to enhance acquisition of a functional-mobility learning task while producing no corresponding effect on a manual control nulling task, within the same study [15]. A separate controlled, repeated-training experiment using a lunar rover navigation task found clear evidence of learning through practice, without vestibular sensory-noise augmentation providing an additional learning advantage across repeated sessions [16].

Together these sources establish a conditional, task-dependent relationship between vestibular stimulation and learning rather than a general-purpose enhancement effect. Task, protocol, timing, and participant state determine whether an effect appears. This conditional relationship supports later, direct learning experiments in the SeaKing research architecture without assuming generalized enhancement, and it must not be read as establishing hippocampal long-term potentiation, neurogenesis, or any other specific neural mechanism; vestibular stimulation can engage human hippocampal and spatial-processing networks, but this regional engagement does not itself establish those downstream mechanisms [3].

6.3 Persistence and Functional Valence

Persistence is a temporal property, not a value judgment. Persistent change may be adaptive, maladaptive or adverse, or functionally indeterminate, and functional meaning requires demonstrated consequences rather than duration alone [3, 5]. Physiological change is not physiological improvement: a candidate beneficial response requires context-appropriate dynamics, appropriate recovery or reconfiguration, reproducibility, tolerability, and association with meaningful functional or clinical benefit [5, 3].

This principle prevents a persistent Phase 1 physiological or behavioral change from becoming an automatic benefit claim. A response that recurs across repeated exposures, or that remains altered after exposure ends, has established only that it is persistent; whether that persistence is adaptive, adverse, or simply undetermined requires the additional evidence specified in Part IV and Part V of this paper.

7. Human Motion-Rich Intervention Precedent

7.1 Human Intervention Precedent

Multiple independently studied human motion-rich intervention classes can produce measurable physiological, behavioral, impairment-level, or functional outcomes, and null or mixed results also occur across this same literature [2]. A randomized controlled trial of therapeutic horseback riding in children and adolescents with autism spectrum disorder demonstrated measurable behavioral improvement relative to a control condition, providing direct human precedent that a complex intervention containing sustained rhythmic movement can be associated with clinically measurable change in a population of direct interest to this research program [30].

This precedent does not establish a common mechanism across motion-rich interventions, a universal positive effect direction, or SeaKing efficacy. The therapeutic horseback riding intervention combines horse-generated movement with active postural correction, animal interaction, instructor contact, and social engagement, and the whole-package effect demonstrated in this trial cannot be attributed specifically to motion [2, 30].

7.2 Controlled Passive-Motion Precedent

Quantitatively specified, passive, externally imposed motion can alter selected human physiological, neural, sleep, or memory-related outcomes under defined controlled protocols [2]. Whole-night continuous rocking, delivered under a quantitatively specified lateral rocking protocol, has been shown to entrain spontaneous neural oscillations and produce measurable benefits for sleep and memory relative to a stationary control condition [21]. This is a direct human bridge showing that physical motion itself, isolated from any surrounding intervention package, can be a biologically consequential experimental variable.

These effects are protocol-specific, and null findings also occur within the same general class of controlled motion manipulation. A double-blind randomized controlled study of whole-body vibration in chronic stroke found no specific benefit of a higher-amplitude vibration condition relative to a low-amplitude placebo condition delivered at the same frequency [22]. This null result does not contradict the rocking finding; it demonstrates that the mere presence of a stronger physical motion parameter does not guarantee a superior outcome, and it establishes that this entire evidence class must be interpreted protocol by protocol rather than as a uniform "motion works" principle.

7.3 Translational Meaning

Human precedent, taken together from Sections 7.1 and 7.2, makes motion a serious experimental variable worth direct investigation [2, 1]. The existing human motion-rich literature supports the biological and experimental plausibility of direct motion research and the feasibility of manipulating motion as an experimental variable. It does not establish SeaKing efficacy, a common mechanism across the interventions reviewed, or an optimal SeaKing waveform [2].

This subsection provides the final translational ceiling carried forward from Review 05 into the present hypothesis. Every subsequent section of this paper that references human intervention precedent operates within this ceiling: engineering and efficacy remain empirical questions, not conclusions this paper is entitled to draw from the evidence reviewed here.

PART III. THE CENTRAL PROBLEM

8. The Natural Motion Problem

8.1 The Physical-Stimulus Characterization Gap

Consistent with the measurable-but-unmeasured gap established in Section 2.2, physical motion can be measured quantitatively, yet the actual physical stimulus remains incompletely characterized across much of the motion-rich clinical literature [2, 1]. That gap identifies a central unmet measurement problem that this paper's hypothesis is built to address, not an optimal waveform or an efficacy claim.

This gap is not a criticism of the studies that leave it unresolved. Clinical and intervention studies are typically designed to answer clinical questions, not to characterize physical exposure with engineering precision. The gap simply means that, as of this paper, no adequately characterized account of natural multidimensional maritime motion as a biological stimulus exists in the literature this program can draw on directly.

8.2 Candidate Motion-Dose Dimensions

Motion dose is multidimensional and may include axis composition, frequency, amplitude, acceleration, temporal structure, duration, and cumulative exposure. Formal dose-response characterization belongs after the relevant components are identified under control, and responses may be nonlinear such that more stimulation is not necessarily better [1, 4, 2, 3]. No universal or therapeutic dose is currently established, and parameters drawn from heterogeneous interventions, such as whole-body vibration frequency, equine trunk kinematics, or controlled rocking displacement, are not interchangeable with one another or with natural vessel motion.

This subsection lists candidate dimensions rather than committing to any of them as necessary or sufficient. Which of these dimensions matter for natural multidimensional motion, and in what combination, is precisely the empirical question Phase 1 and the later evidence gates in Part IV are designed to address.

8.3 State and Active Engagement Alter Effective Exposure

Nominal platform or vessel motion is not sufficient to define the exposure actually received by a participant [1, 4, 2, 5]. Received head motion is a more proximal description of vestibular exposure than vessel or platform motion alone, and may differ because of posture, stabilization, voluntary movement, and participant behavior. Because vestibulo-autonomic responses are stimulus-, state-, and effector-dependent rather than uniformly directional, the same nominal exposure delivered to two participants, or to the same participant at two different times, cannot be assumed to constitute the same effective physiological stimulus.

This finding provides a strong methodological rationale for participant-level sensing and contextual measurement. A vessel-only architecture would leave an important ambiguity as to whether an apparent null reflected little biological relationship or inadequate characterization of received exposure; head-centered motion remains a more proximal description of vestibular input than vessel motion, and Phase 1 can test directly whether head-centered measurement adds explanatory value beyond vessel motion alone.

9. Why Existing Intervention Literature Cannot Answer the Question

9.1 Intervention Wrapper Versus Physical Stimulus

Whole-package efficacy cannot be assigned automatically to motion [2]. Evidence for a whole intervention and evidence for motion itself are different claims, and this distinction is the primary home for the wrapper argument developed throughout this paper. A randomized, stratified, single-blind trial of a hippotherapy simulator in children with cerebral palsy illustrates the underlying causal-resolution problem directly: comparing a moving simulator condition against a nonmoving simulator condition improves causal resolution relative to comparing live riding against an unrelated control, because it holds much of the intervention wrapper constant while varying motion specifically [23].

This subsection establishes the conceptual distinction; Section 9.3 develops its methodological consequence. The point here is narrow: an intervention producing a measurable outcome, however well-designed the trial, does not by itself tell us that motion caused that outcome unless the study design was structured to isolate motion from the rest of the intervention.

9.2 Non-Equivalence of Motion Classes

Horse riding, rocking, vibration, rehabilitation exercise, sailing, and electrical vestibular stimulation are not interchangeable motion classes, and cross-domain parameter equivalence between them is prohibited by this paper [1, 3, 2]. Natural vestibular exposure is multidimensional, time-varying, and biologically distinct from a simple sum of isolated axes, and received head motion is a more proximal description of vestibular exposure than vessel or platform motion alone; both of these established properties apply differently to each motion class, since a rocking bed, a vibration platform, a horse, and a sailing vessel each deliver physically distinct combinations of axis, frequency, amplitude, and predictability.

This non-equivalence is a direct consequence of the physical principles established in Section 4 and the translational boundary established in Section 7.3, applied specifically to the comparison problem: a finding from one motion class cannot be assumed to generalize to another simply because both involve externally imposed physical movement.

9.3 Need for Direct Isolation and Manipulation

Causal resolution requires controlled reproduction and component manipulation [1, 4, 2]. A candidate natural motion-response relationship becomes mechanistically stronger only if the relevant received exposure can be reproduced under controlled mechanical conditions and the physiological response recurs, and after controlled reproduction, stimulus components should be removed, isolated, varied, and recombined to test necessity, sufficiency, interaction, and the minimum sufficient stimulus. The cerebral palsy simulator trial again provides a concrete precedent for this kind of direct manipulation,

since it isolated the presence or absence of motion as an experimental variable while holding much of the surrounding intervention constant [23].

This subsection should not be read as implying that any existing natural intervention package, including the hippotherapy simulator trial itself, has already proven motion causality for the SeaKing hypothesis. It establishes the methodological principle, illustrated by existing precedent, that direct isolation and manipulation are required, and that this requirement is what the SeaKing program's controlled-reproduction and reduction stages, developed in Part IV, are designed to satisfy.

10. The Missing Variable

10.1 What Is Missing

The missing variable is time-resolved, participant-received natural multidimensional motion, measured with sufficient resolution to relate systematically to synchronized physiological data [2, 1, 4, 5, 3]. This is the distilled problem statement for the entire paper: not a missing theory, not a missing population, and not a missing clinical outcome, but a missing measurement. Every proposition established in Parts I and II, spanning the physical-input problem, the vestibular-autonomic bridge, human plasticity, and human intervention precedent, converges on this same unaddressed variable.

Stating the missing variable this precisely is what allows the hypothesis in Part IV to be falsifiable. A vague claim that "motion matters" cannot be tested; a specific claim that time-resolved, participant-received natural multidimensional motion relates reproducibly to synchronized physiological responses can be tested directly, and can fail.

10.2 Why Synchronization Matters

Motion, physiology, state, and context, together with recovery, require a common temporal framework [4, 5]. Physiological regulation should be characterized across baseline, exposure or challenge, within-exposure dynamics, recovery or reconfiguration, and repeated sessions, and no single response direction or recovery speed is intrinsically beneficial. Because no single biomarker captures the autonomic nervous system as a whole, as established in Section 5.3, synchronized multimodal measurement is required to interpret any candidate motion-physiology relationship correctly, and that synchronization must extend to the motion signal itself, not only across physiological channels.

Timing alone does not constitute causal proof. A physiological response that changes at approximately the same time as a motion feature has established temporal association, which is a necessary but not sufficient condition for the controlled-reproduction and mechanistic evidence gates developed in Part IV.

10.3 What Characterization Enables

Characterization can reveal reproducibility, heterogeneity, recovery, and exposure-history structure that would otherwise remain invisible [4, 5, 3]. A scientifically meaningful Phase 1 result can consist of a reproducible exposure- or motion-feature-related, temporally structured, well-tolerated physiological phenotype; stable repeatability, systematic longitudinal change, reproducible heterogeneity, informative nulls, and identified practice or nonspecific effects can all reduce scientific uncertainty. Candidate motion-response relationships must recur within participants, and where appropriate across participants, before the program advances to later evidentiary stages.

This subsection closes Part III by stating explicitly what characterization is and is not. It is still discovery, not therapy. A well-characterized, reproducible physiological phenotype is a scientific finding worth reporting; it is not, on its own, a claim of learning, adaptation, or benefit, and Part IV exists specifically to keep that distinction explicit as the SeaKing hypothesis is stated in full.

PART IV. THE HYPOTHESIS

11. Canonical Hypothesis

The four hypothesis statements below, together with their explicit falsification condition, constitute the canonical SeaKing Solace hypothesis. Each is stated as a testable proposition rather than as an established fact, consistent with the claim ladder defined in Section 3.3.

11.1 H1: Physiological Responsiveness

Quantified natural multidimensional motion may produce reproducible physiological responses in humans. This is the primary hypothesis, and it is a hypothesis about response, not benefit. It follows directly from the physical-input, vestibular-autonomic, and multidimensional-interpretation propositions established in Parts I and II: externally applied motion is a structured vestibular stimulus [1], vestibular nuclei have anatomical access to autonomic circuitry with direct human evidence of modulated sympathetic activity [4], autonomic responses are stimulus-, state-, and effector-dependent [5], and no single biomarker captures the full autonomic response, so physiological responsiveness must be assessed through appropriately interpreted multimodal measurement [3].

H1 is deliberately narrow. It does not specify which physiological channel will respond, in which direction, or by how much. It asserts only that quantified natural multidimensional motion is a plausible candidate to produce a reproducible, scientifically interpretable physiological response, and that this possibility is worth testing directly.

11.2 H2: Exposure-History Dependence

Repeated exposure to quantified natural multidimensional motion may produce systematic, exposure-history-dependent response change [1, 3]. Repeated exposure can change motion sensitivity and physiological response, and habituation, adaptation, learning, sensitization, fatigue, tolerance, and ordinary variability are competing interpretations of that change that must be distinguished from one another rather than assumed. Direct human vestibular precedent for this kind of change exists: repeated galvanic vestibular perturbation has produced both short-term and longer-term postural learning to withstand the perturbation across multiple sessions [18]. Human VOR adaptation can also be expressed in a context-specific manner, demonstrating that learned contextual cues can influence the expression of an adapted vestibular response [20].

This subsection must not be read as calling exposure-history change learning or beneficial plasticity. H2 asserts only that repeated exposure may produce systematic change of some kind; which of the competing interpretations, if any, applies to a given SeaKing finding is a separate question requiring the evidence gates developed in Sections 14 and 15.

11.3 H3: Stable Response Phenotypes

Repeated measurement may identify stable, participant-specific response structure [4, 5, 3]. This is a hypothesis about heterogeneity rather than about central tendency: rather than assuming all participants respond identically to a given motion exposure, H3 proposes that individual differences in response may themselves be stable and reproducible. Direct human evidence for this kind of structure exists outside the SeaKing context: between-subject variability in the human eye-movement response to bilateral galvanic vestibular stimulation can be substantial, while within-subject reliability across repeated testing sessions can remain comparatively high [17].

Personalization is not yet justified by H3 alone. Identifying that stable individual differences exist is a necessary but not sufficient condition for personalized measurement or intervention; individualization becomes scientifically justified only once the additional criteria developed in Section 18.1 are met.

11.4 H4: Bridge to Later Controlled Learning and Mechanism Studies

Characterized physiological phenotypes, if identified, can justify subsequent controlled tests of mechanism, acquisition, and downstream translational questions [1, 3]. If a reproducible physiological or exposure-history-dependent state is identified through Phase 1, later controlled studies may test whether that state alters acquisition of a prespecified learned capability relative to an appropriate practice or control condition. Phase 1 discovery is a scientifically necessary precursor for this kind of downstream test; without a characterized, reproducible physiological response to build on, a learning or mechanism study would have no defined state to test.

H4 does not presume that those later effects exist. A physiological state identified in Phase 1 is not yet a learning-enhancing state, and this bridge hypothesis will only be worth pursuing for phenotypes that satisfy the reproducibility and interpretability standards developed throughout this paper.

11.5 Explicit Falsification Condition

If adequately measured natural motion does not produce a reproducible, scientifically meaningful motion-linked physiological response under adequate exposure and data-quality conditions, the premise required for progression should weaken, redirect, or stop [1, 4, 5, 3]. An independently justified smallest-effect-size-of-interest should be used for the primary analysis where one can be defensibly derived from external reliability, measurement-error, reliable-change, test-retest, or other appropriate literature; where no independently defensible threshold exists, the endpoint should remain exploratory rather than receive an arbitrary threshold.

Under this framework the primary analysis can reach one of three outcomes. A supportive result requires that the effect meet the prespecified scientific threshold and reproducibility criteria with adequate precision. An inconclusive result occurs when the uncertainty interval still spans both scientifically meaningful and negligible effects, or when prespecified data-quality or exposure conditions prevent a valid decision. A non-supportive result requires that the study achieve adequate measurement and exposure conditions and that the resulting precision exclude effects at or above the independently justified threshold. Failure to reject a conventional null hypothesis is not, by itself, evidence of no scientifically meaningful effect, and a primary analysis that fails to support H1 should be classified according to this three-outcome framework rather than reported simply as a failure. No post hoc rescue by exploratory endpoints is permitted under any of the three outcomes: the research program should redirect or narrow according to which of the three outcomes was reached, rather than searching the data for an alternative endpoint that happens to reach significance.

12. Explicit Hypothesis Boundaries

12.1 Response Is Not Benefit

Physiological response, learning, retention, transfer, and benefit remain separate evidentiary claims [5, 3, 2]. Physiological change is not physiological improvement; a candidate beneficial response requires context-appropriate dynamics, appropriate recovery or reconfiguration, reproducibility, tolerability, and association with meaningful functional or clinical benefit. This distinction is not unique to the SeaKing hypothesis; it follows established rejection of a simple one-dimensional model of autonomic control, in which observed change can move in more than one physiologically valid direction without any single direction representing intrinsic improvement [10].

This is the primary benefit firewall for the hypothesis stated in Section 11. Every one of H1 through H4 concerns physiological response, exposure-history-dependent change, stable phenotypes, or a bridge to later testing; none of them, individually or collectively, constitutes a claim of benefit.

12.2 Mechanisms Not Yet Established

No current evidence establishes SeaKing-specific vagal activation, locus coeruleus recruitment, hippocampal long-term potentiation or neurogenesis, or any other specific neural mechanism [5, 3]. No current evidence establishes that SeaKing-like natural motion induces human hippocampal long-

term potentiation or neurogenesis, reopens sensitive periods, creates a validated plasticity window, has an optimal neuroplastic dosing schedule, or improves autism or POTS through neuroplasticity. Heart rate variability can inform cardiac parasympathetic modulation under appropriate conditions, but it is not a direct measure of whole-vagus activity or global autonomic health, and internationally recognized measurement standards make this same interpretive boundary explicit [11].

This is the mechanistic boundary for the hypothesis. Vestibular stimulation can engage human hippocampal and spatial-processing networks in principle, but this regional engagement does not establish any of the specific downstream mechanisms this subsection excludes, and no SeaKing finding should be described using mechanistic language this boundary prohibits until that mechanistic evidence is independently established.

12.3 Clinical and Control Claims Not Yet Established

No Phase 1 result can establish autism or POTS efficacy, an optimal treatment dose, or closed-loop control readiness [1, 4, 5, 3, 2]. Disease-specific physiology and clinically meaningful outcomes are separate evidence gates, and healthy-adult Phase 1 findings cannot establish benefit in POTS, autism, or any other population; direct target-population studies are required before any clinical claim becomes appropriate. Closed-loop or adaptive stimulation should be considered only after a validated target, controllable stimulus-response dynamics, adequate signal quality and latency, tolerability bounds, and superiority to simpler fixed or open-loop control have been demonstrated, and technical feasibility alone is insufficient to justify that step.

POTS and autism are each justified as future mechanistic populations on independent grounds developed further in Part V, but neither population's plausibility should be read as existing treatment evidence. POTS has mechanistic rationale from vestibular participation in orthostatic cardiovascular regulation together with a direct motion-rich treatment evidence gap, and autism has autonomic and sensory-autonomic rationale together with a largely untested direct mechanical vestibular-autonomic coupling; in neither case does this rationale establish causation, universality, or treatment benefit. This is the clinical and engineering boundary for the entire hypothesis paper, and it governs every reference to POTS, autism, or adaptive control anywhere else in this document.

PART V. RESEARCH ARCHITECTURE

13. Phase 1 Discovery Study

13.1 Primary Purpose

The primary purpose of Phase 1, defined in Section 1.3, is to discover reproducible motion-linked physiological structure: whether measured natural head-centered motion contains reproducible, temporally structured input-output relationships, and whether those relationships show recovery, exposure-history dependence, or stable heterogeneity [1, 4, 5, 3]. Phase 1 has no efficacy endpoint of any kind.

This purpose statement follows directly from the hypothesis stated in Part IV. H1 through H4 define what Phase 1 is testing; this subsection states, in operational terms, what Phase 1 is for. A successful Phase 1 is one that answers the discovery question precisely, whether the answer is positive, negative, or heterogeneous, not one that produces a favorable-looking result.

13.2 Repeated-Measures Logic

Repeated sessions test recurrence, longitudinal change, and stable heterogeneity [1, 4, 5, 3]. Candidate motion-response relationships must recur within participants, and where appropriate across participants, before advancing to later evidentiary stages, and biological reproducibility does not require identical waveforms across exposures. Direct human precedent supports the value of this repeated-measures design: between-subject variability in response to a standardized vestibular

stimulus can be substantial even while within-subject reliability across repeated sessions remains comparatively high, which means a single-session design could mistake genuine individual structure for noise [17].

Repeated observations within a participant are not independent samples, and Phase 1's statistical treatment must respect that nested structure. A participant who contributes ten sessions has not thereby contributed ten independent data points; the appropriate hierarchical treatment of this nesting is developed further in Section 13.3.

13.3 Statistical Governance

Phase 1 requires prespecified primary structure, multiplicity control, nested models, and precision or smallest-effect-size-of-interest thresholds where defensible [1, 5, 3]. Discovery and confirmation must be separated, with prespecified primary structure, multiplicity control, held-out or later validation, scientifically meaningful effect thresholds where defensible, and hierarchical models that respect nested participant, session, and exposure structure.

Exact protocol numbers, including specific sample sizes, exact statistical thresholds, and detailed analysis code, belong in a separate statistical analysis plan rather than in this hypothesis paper. This subsection establishes the governing principles that any such plan must satisfy; it does not itself constitute that plan.

13.4 Competing Models

Candidate relationships must compete against respiratory, visual, sickness, and behavioral alternative explanations [4, 5]. Respiration, visual context, posture, cardiovascular state, motion sickness, participant behavior, environment, expectancy, and prior exposure can all alter or mimic a candidate motion-associated physiological response, and naturalistic measurement cannot eliminate all such confounding by design alone. Direct precedent for the need to rule out nonspecific explanations exists in the human vestibular literature: a randomized, sham-controlled experiment questioning the lasting effect of galvanic vestibular stimulation on postural control found that an apparent longitudinal improvement could not be distinguished from a nonspecific or practice-related effect under controlled comparison [19], and context-specific adaptation of the vestibulo-ocular reflex gain demonstrates that the same underlying adaptation can express differently depending on contextual cues, which is itself a competing explanation that must be considered before a response is attributed to motion alone [20].

Prediction does not establish mechanism. Statistical, machine-learning, clustering, or latent-variable outputs remain data-derived constructs until independently validated, since high predictive accuracy can still exploit correlated, noncausal variables rather than the motion signal itself. Phase 1 analyses must therefore explicitly test candidate motion-response relationships against these competing models rather than assuming that a statistical association with motion is sufficient evidence of a motion-specific effect.

14. Discovery Outcomes

14.1 Positive or Reproducible Pattern

A reproducible motion-feature-related phenotype is meaningful discovery [4, 3, 5]. A scientifically meaningful Phase 1 outcome can consist of a reproducible exposure- or motion-feature-related, temporally structured, well-tolerated physiological phenotype. This outcome is not therapeutic. It establishes that a defined relationship between motion and physiology recurs under the tested conditions, which is a necessary precondition for every later stage of the SeaKing research architecture, without itself constituting evidence of learning, adaptation, or benefit.

14.2 Stable Heterogeneity

Stable individual differences can justify prospective moderator or phenotype studies [4, 3, 5]. As direct human precedent already demonstrates, between-subject variability in vestibular response can coexist with high within-subject reliability [17], and Phase 1 may reveal a comparable pattern for natural multidimensional motion: not a single population-average response, but a set of stable, participant-specific response phenotypes.

This is not yet personalized dosing. Identifying stable heterogeneity supports the hypothesis that individualization may eventually be justified, but the criteria for that justification, developed in Section 18.1, require considerably more than the observation that individuals differ.

14.3 Longitudinal Change or Stability

Adaptation-like, habituation-like, stable, or recovery trajectories all reduce uncertainty about the nature of a candidate response [1, 3]. Repeated exposure can change motion sensitivity and physiological response, and the classification of any observed change depends on the specific endpoint and design used to measure it. Human vestibular precedent shows that repeated exposure to a controlled perturbation can produce both short-term and longer-lasting postural learning to withstand it across sessions [18], illustrating one of several trajectories, among habituation, sensitization, and simple stability, that a longitudinal Phase 1 analysis would need to distinguish.

Classification of a longitudinal Phase 1 finding as adaptation, habituation, sensitization, or stability depends entirely on the endpoint measured and the design used to measure it. No single longitudinal pattern should be assumed in advance of the data.

14.4 Null or Nonspecific Result

A precise null or practice or nonspecific effect is scientifically informative [5, 3]. Direct precedent exists for exactly this kind of informative null: a randomized, sham-controlled study questioning the lasting effect of galvanic vestibular stimulation on postural control found that apparent improvement occurred similarly regardless of active stimulation, meaning the improvement could not be attributed specifically to the stimulation itself [19]. A comparably precise Phase 1 null, obtained under adequate measurement and exposure conditions, would be equally informative.

Post hoc rescue by exploratory endpoints is not permitted. If the prespecified primary Phase 1 analysis returns a non-supportive or nonspecific result, that result should be reported as such, consistent with the falsification condition stated in Section 11.5, rather than reinterpreted favorably by searching among secondary or exploratory endpoints for a positive finding.

15. Evidence Gates

15.1 Controlled Reproduction

Candidate natural exposure must reproduce physiology under control [4, 5, 1]. This is the critical association-to-causal-strengthening gate in the entire SeaKing research architecture: a candidate natural motion-response relationship becomes mechanistically stronger only if the relevant received exposure can be reproduced under controlled mechanical conditions and the physiological response recurs. Naturalistic association observed during real maritime exposure, however carefully measured, does not by itself satisfy this gate.

Mechanical replay alone does not guarantee biological reproduction. A controlled apparatus that reproduces the recorded motion waveform has not thereby proven that it reproduces the biologically relevant features of that waveform; the physiological response itself, not merely the motion signal, must recur under control before this gate is satisfied.

15.2 Reduction and Component Testing

After controlled reproduction, stimulus components should be removed, altered, and varied to test necessity and sufficiency [1, 4]. Components should be removed, isolated, varied, and recombined to determine which are necessary, which are sufficient, which interact, and what the minimum sufficient stimulus might be. Natural complexity should not be preserved by default once controlled reproduction has been achieved; the goal of this stage is simplification guided by evidence, not fidelity to the original naturalistic exposure for its own sake.

15.3 Dose-Response

Map the multidimensional response surface within tolerability limits [2, 3]. Motion dose is multidimensional and may include axis composition, frequency, amplitude, acceleration, temporal structure, duration, and cumulative exposure, and formal dose-response characterization belongs after the relevant components have been identified under control at the reduction stage. Responses may be nonlinear, and more stimulation is not necessarily better; direct precedent for this nonlinearity exists in a double-blind randomized study of whole-body vibration, in which a higher-amplitude condition produced no specific benefit relative to a low-amplitude placebo condition delivered at the same frequency [22].

No universal or therapeutic dose is currently established for any motion class examined in this paper, and parameters drawn from heterogeneous interventions are not interchangeable with one another or with natural multidimensional maritime motion.

15.4 Target Validation

Only validated, functionally meaningful phenotypes can become intervention targets [5, 3]. A reproducible physiological phenotype is not automatically a therapeutic, plasticity, or surrogate biomarker; validation requires test-retest reproducibility, prospective association with an independent relevant endpoint, replication, and considerably stronger evidence before causal mediation or surrogate status can be claimed. Internationally recognized heart rate variability measurement standards illustrate why this validation burden is necessary even for well-established physiological signals, since standardized measurement guidance exists precisely because such signals are easily over-interpreted without it [11]. More broadly, biomarkers and surrogate endpoints in clinical trials require a specific, demonstrated relationship to the clinical outcome of interest before they can substitute for that outcome in any inferential sense [12].

Reproducibility alone is insufficient. A physiological phenotype that recurs reliably across sessions has satisfied only the first of several requirements this subsection establishes; it has not yet been validated as a meaningful target for intervention, biomarker use, or surrogate endpoint status.

16. Learning Translation

16.1 Acquisition Test

Consistent with the H4 bridge hypothesis stated in Section 11.4, a selected controlled motion state identified through Phase 1 can be tested for its effect on a prespecified learning task, relative to an appropriate practice or control condition [3, 2]. Direct human precedent shows that this kind of test is both feasible and informative: noisy galvanic vestibular stimulation has enhanced acquisition of a functional-mobility learning task while producing no corresponding advantage on a manual control nulling task within the same study [15], and a separate controlled repeated-training experiment found clear evidence of learning through practice on a lunar rover navigation task without vestibular sensory-noise augmentation adding further benefit across sessions [16].

No generic cognitive enhancement claim is permitted here. Vestibular stimulation can modify human learning acquisition under selected conditions, but controlled null findings, including the manual control and lunar rover results just described, show that it is not a universal learning enhancer; task, protocol, timing, and participant state determine whether an acquisition effect appears at all.

16.2 Retention or Consolidation

Delayed testing is required before durable learning is claimed [3, 2]. An acquisition advantage must survive prespecified delayed testing before durable learning or consolidation can be claimed, and immediate or within-stimulation performance is insufficient on its own. Acute response, exposure-dependent change, acquisition, consolidation or retention, near transfer, far transfer or generalization, spontaneous real-world behavior, and meaningful functional or clinical benefit are distinct outcome levels that require separate evidence, and evidence at one stage does not establish the next.

A future SeaKing acquisition finding, however strong, would establish only that a defined motion state altered performance during or immediately after training. Retention would remain a separate, subsequently tested claim, and failure at the retention stage would not erase the validity of a genuine earlier acquisition effect.

16.3 Transfer or Generalization

Near, far, cross-context, spontaneous, and real-world transfer require distinct evidence [3, 2]. Retained learning or proximal improvement must be tested separately for near transfer, far transfer, cross-context generalization, spontaneous behavior, and real-world function, and none of these can be presumed from trained-task gains alone. This staged requirement mirrors the outcome hierarchy established throughout this paper: each level of generalization is a stronger and more valuable claim than the one before it, and each requires its own dedicated evidence.

Transfer failure does not erase acquisition. If a future controlled SeaKing learning study demonstrates acquisition and retention but finds that the trained advantage does not generalize to an untrained task or context, that outcome does not invalidate the acquisition and retention findings; it simply establishes the boundary of what those findings support.

17. Clinical Translation

17.1 Disease-Specific Physiology Before Efficacy

Target populations must be studied directly after a controllable stimulus-response relationship is established [1, 4, 5, 3, 2]. Disease-specific physiology and clinically meaningful outcomes are separate evidence gates, and healthy-adult Phase 1 findings cannot establish benefit in POTS, autism, or any other population. Direct target-population studies are required before this program can make any disease-specific claim, and those studies presuppose the controlled stimulus-response relationship developed in Parts IV and V of this paper.

This subsection governs the two disease-specific branches developed in Sections 17.2 and 17.3. Neither branch is permitted to substitute mechanistic plausibility for direct target-population evidence, and neither branch may treat a healthy-adult Phase 1 finding as though it already applied to POTS or autism.

17.2 POTS Branch

POTS has mechanistic rationale together with a direct motion-rich treatment evidence gap [4, 2]. POTS is a justified future mechanistic population because vestibular pathways participate in orthostatic cardiovascular regulation, and preliminary disease-specific otolith-autonomic associations exist; at the same time, controlled motion-rich POTS treatment evidence remains an identified evidence gap. Expert consensus on the diagnosis and treatment of postural tachycardia syndrome establishes POTS as a heterogeneous orthostatic disorder requiring individualized clinical characterization rather than a single uniform treatment approach [24]. Direct disease-specific evidence supports a preliminary link between otolith function and vestibulo-sympathetic interaction in POTS specifically, providing a mechanistic bridge between the vestibular-autonomic physiology developed in Part II and this clinical population [25]. Review 05's examination of the randomized-treatment landscape summarized by a systematic review of randomized POTS treatment trials,

which found an established pharmacologic and nonpharmacologic treatment literature, did not identify a controlled motion-rich treatment within that landscape [2, 26].

No current treatment-efficacy claim is permitted for POTS. The evidence gap identified by this systematic review is not evidence of ineffectiveness; it identifies an unresolved and directly testable clinical question rather than a basis for extrapolated efficacy, association does not establish vestibular causation, and this branch remains mechanistic rationale plus an explicit evidence gap rather than existing treatment evidence.

17.3 Autism Branch

Autism has autonomic and sensory rationale together with human motion-rich intervention precedent [4, 2, 3]. Autism is a justified future mechanistic population because autonomic differences and altered sensory-autonomic reactivity are reported in the literature, while direct controlled mechanical vestibular-autonomic coupling remains largely untested in this population. A meta-analysis of heart rate variability in individuals with autism spectrum disorders supports group-level differences in cardiac autonomic measures, without establishing a universal low-vagal-tone phenotype, causation, or treatment implication [27]. A systematic review of resting-state autonomic function in autism supports heterogeneity and context dependence of these findings rather than one consistent autism autonomic phenotype [28]. Direct human experimental evidence shows altered autonomic reactivity during sensory stimulation in children with autism spectrum disorder, though sensory stimulation of this kind is not equivalent to isolated mechanical vestibular stimulation [29].

ASD additionally has a substantive human motion-rich intervention literature, including randomized equine and horse-riding studies and mechanical analogues with measurable but heterogeneous outcomes [2], among them the therapeutic horseback riding trial already introduced in Section 7.1 [30]. Because these are multimodal interventions, the contribution of physical motion is not isolated, and package effects are not motion-specific; this precedent adds direct human translational context for the autism branch without claiming that motion itself was the causally responsible component.

17.4 Meaningful Clinical Outcomes

Clinical benefit requires direct, prespecified symptoms, function, or participation outcomes [12]. As established in Section 15.4, a biomarker or surrogate endpoint requires a specific, demonstrated relationship to the clinical outcome of interest before it can substitute for that outcome; this requirement applies with equal force to any future POTS or autism SeaKing outcome, where a favorable physiological or biomarker signal would not itself constitute evidence of clinical benefit.

This subsection closes Part V's clinical translation discussion by restating, in disease-specific terms, the benefit firewall first established in Section 12.1. Biomarkers cannot substitute for prespecified clinical outcomes without independent validation, regardless of how promising the underlying physiological finding appears.

18. Personalization and Adaptive Control

18.1 Moderator Validation

Moderators must be prospective, predictive, replicated, and add value [5, 3]. Individualization becomes scientifically justified only when candidate moderators, such as baseline state, developmental stage, phenotype, or exposure history, are measured prospectively, predict subsequent response, replicate across samples, and demonstrate that participant-specific information improves prediction or outcome beyond simpler population-level or stratified approaches.

Heterogeneity alone is insufficient. Section 14.2 already established that Phase 1 may reveal stable individual differences in response; this subsection states the additional, considerably more demanding criteria that must be satisfied before those differences justify moderator-based individualization of any kind.

18.2 Individualized Stimulation

Participant-specific selection is justified only if it outperforms simpler population-level approaches [1, 4, 5, 3]. This criterion follows directly from Section 18.1: even a validated, replicated moderator does not automatically justify individualized stimulation unless individualized selection can be shown to outperform a simpler fixed or stratified population-level approach using the same moderator information.

No personalization assumption is made anywhere in this paper. Individualized stimulation remains a downstream possibility contingent on evidence this program has not yet generated, not a design feature this hypothesis presumes will ultimately be adopted.

18.3 Closed-Loop Control

Closed-loop control requires a validated target, stable dynamics, and signal quality. Closed-loop or adaptive stimulation should be considered only after a validated target, controllable stimulus-response dynamics, adequate signal quality and latency, tolerability bounds, and demonstrated superiority to simpler fixed or open-loop control have all been established.

Technical feasibility is not scientific necessity. That a closed-loop system could, in principle, be engineered does not by itself justify building one; this subsection represents the furthest downstream and most conditional hypothesis in this paper, and it is reached only after every earlier evidence gate in Part V has been satisfied.

PART VI. CONCLUSIONS AND RESEARCH PROGRAM

19. Scientific Conclusions

19.1 What Is Established

Motion is a measurable vestibular stimulus. Externally applied physical motion is a structured biological vestibular stimulus rather than a binary motion or no-motion exposure [1]. Vestibular-autonomic coupling is directly demonstrated in humans: vestibular nuclei have anatomical access to autonomic and cardiovascular circuitry, and vestibular stimulation can modify directly recorded sympathetic activity [4]. Vestibular plasticity is established: human vestibular systems retain measurable capacity for experience-dependent adaptation and perceptual learning across sensorimotor and perceptual domains [3]. Human motion-rich intervention precedent is established: multiple independently studied intervention classes can produce measurable physiological, behavioral, impairment-level, or functional outcomes, and quantitatively specified passive externally imposed motion can alter selected human outcomes under defined controlled protocols [2].

These four propositions introduce no new claims. Each has been developed in full, with its supporting evidence and explicit boundaries, in Parts I through III of this paper, and this subsection restates them together as the foundation on which the hypothesis in Part IV was built.

19.2 What Is Plausible or Testable

Natural multidimensional motion may contain reproducible physiological structure worth direct investigation [1, 4, 5, 3]. Phase 1 is a discovery and early system-identification study designed to determine whether measured natural head-centered motion contains reproducible, temporally structured physiological input-output relationships, and whether those relationships show recovery, exposure-history dependence, or stable heterogeneity. If a reproducible physiological or exposure-history-dependent state is identified, later controlled studies may test whether that state alters acquisition of a prespecified learned capability.

The hypothesis stated in Part IV remains unresolved. H1 through H4 are testable propositions, not established findings, and this paper does not anticipate what Phase 1 will find. What is plausible is

that the discovery question itself is worth asking; whether the answer is positive, negative, or heterogeneous is precisely what direct experimentation must determine.

19.3 What Is Not Established

No SeaKing efficacy, optimal waveform, disease benefit, or therapeutic neuroplasticity is established by any evidence reviewed in this paper [1, 4, 5, 3, 2]. No current evidence establishes that SeaKing-like natural motion induces human hippocampal long-term potentiation or neurogenesis, reopens sensitive periods, creates a validated plasticity window, has an optimal neuroplastic dosing schedule, or improves autism or POTS through neuroplasticity. The existing human motion-rich literature supports the biological and experimental plausibility of direct motion research and the feasibility of manipulating motion as an experimental variable; it does not establish SeaKing efficacy, a common mechanism across the interventions reviewed, or an optimal SeaKing waveform. Disease-specific physiology and clinically meaningful outcomes are separate evidence gates, and healthy-adult Phase 1 findings cannot establish benefit in POTS, autism, or any other population.

This is the final boundary statement of the scientific conclusions in this paper. It restates, in consolidated form, the claim firewall first established in Section 3.2 and reinforced throughout Parts IV and V, and it governs the research program architecture presented in Section 20.

20. SeaKing Research Program

20.1 Stimulus and Causality Branch

The stimulus and causality branch of the SeaKing research program proceeds through characterization, association, replication, challenge of alternative explanations, controlled reproduction, reduction or manipulation, and dose-response mapping [1, 4, 5, 3, 2]. This sequence is the primary final research architecture developed across Parts III through V of this paper. The SeaKing program is evidence-gated but not a single strictly linear ladder: this branch specifically develops stimulus causality, moving from measuring what natural motion is, to associating it with physiological response, to replicating that association, to ruling out competing explanations, to reproducing the response under controlled conditions, to isolating its necessary and sufficient components, and finally to mapping how physiological response varies across the multidimensional motion-dose space.

Each stage in this branch corresponds to one or more of the evidence gates described across Parts III through V. A finding that does not survive one stage should narrow or redirect the branch rather than be carried forward as though it had already satisfied a later stage.

20.2 Learning and Clinical Branch

The learning and clinical branch proceeds from physiological discovery to acquisition, to retention, to transfer or generalization, and to real-world or functional benefit [3, 2, 5]. Acute response, exposure-dependent change, acquisition, consolidation or retention, near transfer, far transfer or generalization, spontaneous real-world behavior, and meaningful functional or clinical benefit are distinct outcome levels that require separate evidence at each stage. An acquisition advantage must survive prespecified delayed testing before durable learning is claimed, and retained learning or proximal improvement must be tested separately for near transfer, far transfer, cross-context generalization, spontaneous behavior, and real-world function; none of these can be presumed from trained-task gains alone.

Not every branch must progress. A physiological discovery that does not translate into a testable learning state remains a valid scientific finding in its own right, and this branch's failure to progress beyond an early stage does not retroactively invalidate the physiological discovery that preceded it.

20.3 Conditional Personalization and Control

Personalization and adaptive control occur only after prerequisites are validated in the relevant upstream branch [1, 4, 5, 3]. Individualization becomes scientifically justified only when candidate moderators are measured prospectively, predict subsequent response, replicate, and demonstrate that participant-specific information improves prediction or outcome beyond simpler population-level approaches. Closed-loop or adaptive stimulation should be considered only after a validated target, controllable stimulus-response dynamics, adequate signal quality and latency, tolerability bounds, and demonstrated superiority to simpler fixed or open-loop control are all established.

This is the final conditional endpoint of the SeaKing research program as presented in this paper. Both personalization and adaptive control sit downstream of every other evidence gate described in Parts III through V, and neither should be pursued, described, or implied as a near-term deliverable of Phase 1.

20.4 Falsifiability and Stop Rules

Each stage of the SeaKing research program can weaken, redirect, simplify, or stop the relevant branch [1, 4, 5, 3]. If adequately measured natural motion does not produce a reproducible, scientifically meaningful motion-linked physiological response under adequate exposure and data-quality conditions, the premise required for progression should weaken, redirect, or stop. Discovery and confirmation must remain separated throughout, with prespecified primary structure, multiplicity control, held-out or later validation, and hierarchical models that respect nested participant, session, and exposure structure; exact numerical thresholds belong in a separate statistical analysis plan rather than in this paper.

The SeaKing program is evidence-gated, not inevitable. This principle closes the hypothesis paper as it opened: every proposition in this document is subordinate to the evidence that Phase 1 and its successors actually produce, and no stage of this research program is entitled to proceed on the strength of plausibility alone.

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