

Volume 2, Issue 1

Research Article

Date of Submission: 27 August, 2026

Date of Acceptance: 21 September, 2026

Date of Publication: 01 October, 2026

Bhaskare's Neuroplastic Reserve–Maturity Model: A Conceptual Framework Linking Age, Neuroplastic Capacity, and Rehabilitation-Induced Adaptation

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Citation: Bhaskare, G. M. (2026). Bhaskare's Neuroplastic Reserve–Maturity Model: A Conceptual Framework Linking Age, Neuroplastic Capacity, and Rehabilitation-Induced Adaptation. *J Brain Sci Ment Health*, 2(1), 01-10.

Abstract

Neuroplasticity, the nervous system's capacity for adaptive structural and functional change, has become a unifying construct in neuroscience, neurology, and rehabilitation medicine. Yet the literature has not consistently reconciled two observations that appear, on their surface, to be in tension: plastic capacity is generally described as declining across the lifespan, while the neural mechanisms that instantiate plasticity become progressively more organized and specialized with maturation. This systematic review synthesizes evidence bearing on two theoretical constructs proposed by the author to resolve this tension, collectively termed Bhaskare's Principles of Neuroplasticity: Bhaskare's Principle I, the Neuroplastic Reserve Principle, describing the intrinsic, largely age-graded capacity of the nervous system for adaptive modification, and Bhaskare's Principle II, the Neuroplastic Maturity Principle, describing the shift from broad, developmentally driven plasticity toward specialized, experience-dependent plasticity. A narrative-systematic search of PubMed, Google Scholar, and allied databases identified literature spanning developmental critical-period neuroscience, cognitive and brain reserve theory, synaptic aging and neurotrophic signaling, tumor-associated cortical reorganization, and dose-, timing-, and specificity-dependent principles of neurorehabilitation. Across these domains, converging evidence indicates that reserve and maturity are dissociable but interacting constructs, and that externally applied rehabilitation stimuli characterized by specificity, intensity, repetition, salience, appropriate timing, and sufficient sensory and motor engagement can recruit residual plastic capacity even in mature and aged nervous systems. These findings are integrated into a proposed Bhaskare's Neuroplastic Reserve–Maturity Model, which frames functional recovery as the product of an interaction between a constrained but non-zero biological reserve, an increasingly specialized but organizationally efficient neural architecture, and a rehabilitation stimulus capable of directing residual capacity toward functionally meaningful reorganization. Clinical implications for stroke rehabilitation, traumatic brain injury, pediatric neurology, and neuro-oncological surgery are discussed, alongside the conceptual and methodological limitations of a framework that remains, at this stage, illustrative rather than empirically parameterized.

Keywords: Neuroplasticity, Neuroplastic Reserve, Cognitive Reserve, Critical Period, Cortical Reorganization, Neurorehabilitation, Brain Tumour Plasticity, Aging, Synaptic Plasticity

Introduction

Neuroplasticity refers to the capacity of the nervous system to modify its structure, connectivity, and function in response to experience, injury, learning, or disease. The construct has moved from a peripheral curiosity in twentieth-century neuroscience to a central organizing principle across developmental neurobiology, cognitive aging research, stroke and traumatic brain injury rehabilitation, and neuro-oncological surgery [1].

Despite this centrality, the field has not fully reconciled two well-supported but seemingly opposing observations. The first is that the intrinsic capacity of the nervous system for large-scale adaptive change appears to be greatest early in

life and to decline, in a graded rather than binary fashion, across subsequent developmental and aging stages [2,3]. The second is that the neural mechanisms available for plastic change do not simply disappear with maturation; rather, they become increasingly specialized, network-organized, and experience-dependent, favoring targeted, well-directed forms of reorganization over the broad, exploratory plasticity characteristic of early development [4,5].

This review addresses that tension directly by examining two theoretical constructs proposed by the author as organizing principles for the field, together designated Bhaskare's Principles of Neuroplasticity: Bhaskare's Principle I, the Neuroplastic Reserve Principle, and Bhaskare's Principle II, the Neuroplastic Maturity Principle. Bhaskare's Principle I holds that neuroplastic reserve, understood as the intrinsic capacity of the nervous system to undergo adaptive structural, synaptic, and functional modification, is generally greater during early development and becomes progressively more constrained with maturation and aging, while emphasizing that chronological age alone does not fully determine an individual's neuroplastic reserve. Bhaskare's Principle II holds that, in parallel with this decline in raw reserve, the character of neuroplasticity itself changes: developmental plasticity is relatively broad and driven by intrinsic maturational programs, whereas mature and aged plasticity is more specialized, more organized around established networks, and more dependent on specific, structured experience. Taken together, these two principles generate a central hypothesis of practical importance to rehabilitation medicine and neuro-oncology alike: that individuals with greater available neuroplastic reserve may possess a correspondingly greater capacity for neural adaptation, that maturation and aging constrain this reserve while simultaneously increasing neural specialization, and that appropriately timed and sufficiently specific rehabilitative or therapeutic stimulation can nonetheless recruit and direct whatever residual plastic capacity remains available across the lifespan.

The clinical stakes of this question are considerable. In stroke and traumatic brain injury rehabilitation, the assumption that plasticity is age-limited has historically been used to justify more conservative therapeutic goals in older patients, even though dose-response and timing data suggest that appropriately structured intervention can produce meaningful gains well outside early developmental windows [6,7]. In pediatric neurology, the long-standing assumption that a younger, less mature brain necessarily recovers better from focal injury than an adult brain, an idea historically attributed to Margaret Kennard, has itself been substantially revised, with evidence indicating that very early or highly diffuse injury can produce outcomes that are equivalent to or worse than comparable adult injury, particularly for functions requiring prolonged maturation [8-10].

In neuro-oncology, the slow-growing nature of diffuse low-grade gliomas appears to permit a degree of cortical and subcortical functional reorganization that is rarely seen with acute lesions such as stroke, raising the possibility that tumor-induced plasticity, timing, and surgical planning interact in ways that a reserve-maturity framework could help formalize [11,12]. The present systematic review has three aims. First, to synthesize the empirical and theoretical literature bearing on neuroplastic reserve as a construct, drawing on parallel developments in cognitive and brain reserve theory, developmental critical-period neuroscience, and the neurobiology of synaptic aging. Second, to synthesize the literature bearing on neuroplastic maturity, including the developmental trajectory from broad plasticity to specialized, experience-dependent plasticity, and the clinical evidence, both supportive and contradictory, for age-related changes in the character rather than merely the amount of plastic capacity. Third, to synthesize the literature on the properties of external rehabilitation or therapeutic stimuli, including specificity, intensity, repetition, salience, task orientation, sensory input, motor practice, and timing, that determine whether residual plastic capacity is successfully recruited toward functional recovery. These three bodies of evidence are then integrated into a proposed Neuroplastic Reserve-Maturity Model intended to generate testable predictions for future rehabilitation and neuro-oncological research.

Method Design

This review followed a narrative-systematic design. Because the two organizing constructs under review, neuroplastic reserve and neuroplastic maturity, are theoretical rather than single measurable clinical outcomes, a quantitative meta-analysis with pooled effect sizes was not appropriate. Instead, a structured and reproducible search strategy was used to identify literature across the several subfields that bear on the constructs, followed by qualitative synthesis organized around the two principles and the properties of rehabilitation stimuli that link them to functional outcome. This approach is consistent with narrative-systematic reviews used elsewhere in neurorehabilitation science to synthesize mechanistically heterogeneous literatures that do not lend themselves to a single quantitative outcome metric.

Information Sources and Search Strategy

Literature was identified through structured searches of PubMed/MEDLINE, Google Scholar, Semantic Scholar, and publisher databases including Nature Publishing Group, Elsevier ScienceDirect, Springer, Frontiers, and Wiley Online Library. Search terms were organized around five conceptual clusters: (a) neuroplastic reserve and cognitive/brain reserve terminology (e.g., "neuroplastic reserve," "brain reserve," "cognitive reserve," "neuroplastic potential"); (b) developmental and critical-period plasticity (e.g., "critical period plasticity," "experience-dependent plasticity," "Kennard principle"); (c) synaptic and molecular aging (e.g., "BDNF aging synaptic plasticity," "long-term potentiation aging"); (d) tumor-associated cortical reorganization (e.g., "glioma cortical plasticity," "low-grade glioma functional reorganization," "awake mapping neuroplasticity"); and (e) rehabilitation stimulus parameters (e.g., "dose-response stroke rehabilitation," "constraint-induced movement therapy," "specificity of motor training," "timing of rehabilitation after stroke"). Searches

were not restricted by a fixed publication date range, given that several foundational theoretical papers predate the 2000s, but priority was given to identifying the most current empirical work within each conceptual cluster.

Eligibility Criteria

Eligible sources included peer-reviewed primary research articles, systematic and narrative reviews, and theoretical or consensus papers published in indexed neuroscience, neurology, neurosurgery, and rehabilitation medicine journals. Both human clinical and preclinical animal-model literature were included, given that several of the mechanistic claims underlying the reserve and maturity constructs, including motor map reorganization, synaptogenesis, and the post-injury timing window for rehabilitative efficacy, rest substantially on rodent and non-human primate models that are not yet fully replicated in human trials. Sources were excluded if they were not available in English, were not peer-reviewed, or did not bear conceptually on reserve, maturity, or rehabilitation-stimulus parameters as defined above.

Synthesis Approach

Given the theoretical nature of the two organizing constructs, data were synthesized narratively rather than statistically. Findings were grouped first under the Neuroplastic Reserve Principle, then under the Neuroplastic Maturity Principle, and finally under the moderating role of external rehabilitation stimulus characteristics, before being integrated into a proposed interactive model. Where studies used differing terminology for closely related constructs, for example, "brain reserve" in the aging and dementia literature versus "neuroplastic potential" in the neuro-oncological surgical literature, the conceptual correspondence between terms is made explicit in the synthesis rather than assumed.

Results and Synthesis

Bhaskare's Principle I: The Neuroplastic Reserve Principle

Neuroplastic reserve, as synthesized across the literature reviewed here, can be defined as the intrinsic capacity of the nervous system to undergo adaptive structural, synaptic, and functional modification in response to experience, learning, injury, or therapeutic stimulation. This reserve is generally described as greater during early development and as becoming progressively more constrained across middle and older adulthood, although the literature is consistent that chronological age alone is an imperfect predictor of an individual's available reserve.

Conceptual Foundations in Reserve Theory

The closest existing analogue to neuroplastic reserve in the clinical neuroscience literature is the construct of brain reserve, most systematically formalized within cognitive aging and dementia research. Brain reserve is generally treated as a passive, structurally grounded form of capacity, reflecting premorbid neural resources such as synaptic density and brain volume, whereas cognitive reserve is treated as a more active construct describing the brain's ability to recruit alternative networks or strategies to cope with accumulating pathology [13]. Individuals with comparable structural brain reserve can nonetheless differ substantially in functional outcome depending on their cognitive reserve, which suggests that reserve is not a single unitary quantity but a layered set of constructs operating at structural, functional, and strategic levels. A neuroplastic reserve construct, as proposed in the framework under review, sits conceptually closer to brain reserve in that it emphasizes the nervous system's intrinsic capacity for adaptive modification, but it differs from classical brain reserve theory in being explicitly dynamic: reserve in this sense is not a fixed premorbid quantity but a capacity that can, within limits, be recruited, directed, and partially replenished through appropriate stimulation.

Reserve as a Recrutable, Not Merely a Fixed, Quantity

Direct evidence for a recrutable and individually variable form of neuroplastic reserve comes from the neuro-oncological surgical literature, where the concept of "neuroplastic potential" has been operationalized using serial intraoperative and functional mapping. Herbet et al. (2016) demonstrated, using longitudinal awake mapping in patients undergoing surgery for diffuse low-grade glioma, that the capacity for functional reorganization is not distributed uniformly across the brain but is instead substantially constrained by white matter connectivity, such that subcortical connectomic architecture represents a primary limit on reorganization even when cortical reserve appears otherwise available [12]. This finding is important for a reserve-based framework because it demonstrates that neuroplastic reserve is not merely a function of chronological age or generic neural health, but is also anatomically and connectomically structured; some brain regions and networks retain substantially more capacity for reorganization than others, independent of the patient's age [12,14].

A closely related line of evidence comes from prehabilitation studies in neuro-oncology, in which non-invasive brain stimulation is used prior to surgery to actively induce cortical reorganization before tumor resection, with the explicit goal of relocating eloquent function away from tissue planned for removal. Rivera-Rivera et al. (2017) reported that repetitive transcranial magnetic stimulation applied preoperatively catalyzed measurable cortical plasticity that subsequently enabled more extensive resection of tumors situated in eloquent brain regions than would otherwise have been considered safe [15]. This finding is directly relevant to the reserve principle because it demonstrates that neuroplastic reserve can, at least in some circumstances, be actively recruited and directed by an external stimulus rather than existing only as a passive ceiling on adaptive capacity, a point taken up further in the discussion of rehabilitation stimulus characteristics below.

Age-Related Constraint of Reserve at the Synaptic and Molecular Level

At the molecular and synaptic level, the literature provides substantial support for the claim that neuroplastic reserve becomes more constrained with aging, even while cautioning against treating this decline as absolute or unmodifiable. Long-term potentiation, the principal cellular model of activity-dependent synaptic strengthening, is reliably attenuated in aged hippocampal tissue relative to young adult tissue, and this attenuation has been linked mechanistically to age-related reductions in brain-derived neurotrophic factor (BDNF) signaling and downstream kinase activity. Reduced expression of BDNF-regulated signaling components, including phosphorylated calcium/calmodulin-dependent protein kinase II and extracellular signal-regulated kinase, has been observed in aged hippocampal tissue and is associated with impaired maintenance of long-term potentiation, deficits that can be partially reversed pharmacologically, indicating that the underlying reserve, while reduced, is not entirely abolished. Human studies of non-invasive brain stimulation similarly find that older adults retain a capacity for stimulation-induced plasticity, but that this capacity is expressed differently than in younger adults, for example with a delayed rather than absent peak response to anodal transcranial direct current stimulation.

Reserve as Distinguishable from, but Related to, Structural Brain Health

The reserve and resilience literature emerging from consensus efforts such as the Alzheimer's Association's Reserve, Resilience and Protective Factors workgroup is instructive for a neuroplastic reserve construct because it explicitly distinguishes reserve, understood as a person's total neural or cognitive resources at a given point in time, from brain maintenance, understood as the relative preservation of neural structure and function over time [13]. This distinction maps usefully onto the distinction between neuroplastic reserve and neuroplastic maturity proposed in the framework under review: reserve describes how much adaptive capacity is available, while maturity, addressed in the next section, describes how that capacity is organized and what kind of stimulation is required to access it.

Summary of Principle I Evidence

Taken together, the reserve literature supports three claims central to Bhaskare's Neuroplastic Reserve Principle. First, neuroplastic reserve is generally, though not universally or linearly, greater in earlier developmental stages and becomes more constrained with maturation and aging, a pattern consistent across molecular, synaptic, and structural levels of analysis. Second, reserve is not a single global quantity but is regionally and connectomically structured, such that some circuits retain substantially greater reorganizational capacity than others regardless of the patient's age. Third, and most consequential for rehabilitation and neuro-oncological practice, residual reserve in mature and aged nervous systems is not inert; it can be recruited and, in some paradigms, actively primed by external stimulation, a finding that motivates the interaction between reserve, maturity, and rehabilitation stimulus developed later in this review.

Bhaskare's Principle II: The Neuroplastic Maturity Principle

Where the reserve principle addresses how much adaptive capacity is available, the maturity principle addresses how the character of that capacity changes across the lifespan. The central claim, drawn from developmental critical-period neuroscience, is that neuroplasticity transitions from relatively broad, developmentally driven plasticity toward increasingly specialized, organized, and experience-dependent plasticity as the nervous system matures, such that mature and older nervous systems may possess lower raw plastic reserve while retaining highly organized mechanisms capable of meaningful adaptation when appropriately stimulated.

Critical Periods as the Prototype of Maturation Change in Plasticity

The clearest experimental demonstration of maturity-dependent changes in the character of plasticity comes from the critical-period literature, most extensively developed in the visual system. Hensch (2005) showed that critical periods are not simply windows that are open in youth and closed thereafter, but are actively regulated by the maturation of excitatory-inhibitory circuit balance: plasticity is triggered by the maturation of inhibitory, particularly parvalbumin-positive interneuron, circuitry, and is subsequently closed by molecular "brake" factors, including perineuronal net formation, myelin-associated inhibitors, and epigenetic regulators, that stabilize the resulting circuitry [16]. This mechanistic account is significant for the maturity principle because it demonstrates that the shift away from broad developmental plasticity is not a passive loss of capacity but an active, adaptive process: the same maturational signals that close the critical period also consolidate and stabilize learned circuitry, which is functionally necessary for reliable perception, memory, and identity across the lifespan [3,4].

Critically, this same body of work demonstrates that critical-period plasticity is not simply lost with maturation but can be reactivated under specific conditions. Reducing cortical inhibition pharmacologically, for example, has been shown to reopen ocular dominance plasticity in adult animals that would otherwise be well outside the normal critical-period window, and conversely, artificially increasing inhibition in juvenile animals can prematurely close a critical period [3,5]. This bidirectional controllability of plasticity timing provides a strong mechanistic argument for a maturity construct that is graded and modifiable rather than a fixed developmental cliff, and it directly supports the claim, central to the framework under review, that appropriately targeted stimulation may recruit adaptive capacity that is not accessible under ordinary conditions in a mature nervous system.

Specialization of the Motor System With Learning and Maturation

A parallel pattern of increasing specialization is evident in the motor system, where the nature of experience-driven

plasticity depends closely on the specific demands of the motor behavior being trained. Motor skill training, endurance training, and strength training each produce distinct and largely non-overlapping forms of plasticity within motor cortex and spinal cord: skill training drives synaptogenesis and reorganization of movement representations within motor cortex, endurance training drives angiogenesis without altering cortical motor maps, and strength training alters spinal motoneuron excitability without producing cortical map reorganization [17]. This experience-specific patterning of plasticity is itself a maturational and specialization phenomenon: as the motor system consolidates a skill, cortical involvement becomes progressively more selective, with motor cortex playing an instructive role during early learning and becoming comparatively disengaged once a skilled movement is well established. Synaptogenesis and motor map reorganization, moreover, have been shown to emerge only during the later, slower phase of motor skill learning rather than during the initial rapid phase, indicating that structural plasticity in the mature motor system is temporally dissociated from early performance gains and reflects a more organized, consolidation-dependent process than the diffuse plasticity characteristic of early development.

The Kennard Principle and Its Revision: Maturity Is Not Simply Advantageous

A body of developmental neuropsychological literature complicates any simple assumption that lower maturity is uniformly advantageous for recovery from brain injury.

The so-called Kennard principle, generally summarized as the claim that earlier brain injury produces better functional outcomes than equivalent injury sustained in maturity, originates in the work of Margaret Kennard on motor recovery in infant versus adult primates following precentral cortical lesions [9,10]. However, historical and empirical reappraisal of this literature indicates that Kennard's own findings were considerably more nuanced than the eponymous "principle" that followed her, and that the relationship between age at injury and outcome is better characterized as curvilinear than linear [8].

Very early or highly diffuse injury, particularly injury that disrupts the formation of critical white matter tracts required for later-maturing, highly interconnected cognitive functions, can produce outcomes equivalent to or worse than comparable injury sustained in a more mature brain, a pattern sometimes termed the Kennard principle paradox. Preterm infants, for example, continue to show high rates of neurodevelopmental disability despite sustaining injury at a developmental stage when raw plastic reserve should, on a simple version of the reserve principle, be near its maximum [2].

This evidence is important for the maturity principle because it demonstrates that neuroplastic maturity is not a single dimension running from "more plastic and therefore better" to "less plastic and therefore worse." Rather, maturity describes a qualitative reorganization of how plasticity operates: functions that depend on extensive, prolonged intercortical communication may be more vulnerable, not less, to injury sustained before that communication has matured, even though the same young brain shows superior recovery for functions, particularly basic sensorimotor functions, that do not require the same degree of prolonged network maturation [2,8]. A reserve-only account, without a maturity dimension, cannot accommodate this pattern, which is one of the principal theoretical justifications for treating reserve and maturity as distinct, interacting constructs rather than a single lifespan trajectory.

Tumour-Associated Plasticity as a Natural Experiment in Maturity and Timing

Diffuse low-grade gliomas provide an unusually informative natural experiment on the interaction between maturity, timing, and reorganizational capacity, because their slow growth allows reorganization to occur over months to years rather than the seconds to days characteristic of stroke. Comparative work indicates that the degree of functional loss associated with a tumor is related more closely to the tumor's growth rate than to its volume, with fast-growing lesions producing more profound deficits than slow-growing lesions of comparable size, because slow growth affords time for progressive functional reorganization to consolidate [11]. Functional imaging and direct electrical stimulation studies of glioma patients show that this reorganization follows a patterned rather than random course, involving recruitment of perilesional tissue, homologous regions in the contralateral hemisphere, and domain-general association cortex, with the degree of reorganization varying by tumor grade and by the specific cognitive or motor domain under study [18]. Because this reorganization occurs within an already mature adult nervous system, it constitutes direct evidence that the specialized, network-constrained plasticity characteristic of the mature brain, as described by the maturity principle, is nonetheless sufficient to support substantial functional reorganization when the injury process itself unfolds slowly enough to engage it.

Summary of Principle II Evidence

The maturity literature supports three claims central to Bhaskare's Neuroplastic Maturity Principle. First, the transition from developmental to mature plasticity is an actively regulated, not merely a passively declining, process, governed by circuit maturation and molecular stabilization mechanisms that are themselves modifiable. Second, this transition involves genuine specialization: mature plasticity is more targeted, more consolidation-dependent, and more tied to specific experiential demands than the comparatively diffuse plasticity of early development. Third, greater maturity is not uniformly disadvantageous for recovery, and in some circumstances, particularly for functions requiring extensive network maturation, greater developmental maturity at the time of injury is protective rather than limiting, a finding that directly qualifies any simple reading of the reserve principle in isolation.

The Moderating Role of External Rehabilitation Stimulus

The reserve and maturity principles jointly predict that functional recovery should depend not only on how much plastic capacity is available and how that capacity is organized, but also on whether an external rehabilitation stimulus is delivered in a form capable of engaging it. The most influential synthesis of this stimulus-side literature remains Kleim and Jones's (2008) review of ten principles of experience-dependent neural plasticity, which identifies specificity, intensity, repetition, salience, task orientation, timing, and related dimensions of training as determinants of whether experience-driven plasticity occurs and whether it is functionally adaptive rather than maladaptive [1].

Specificity, Repetition, and Salience

Evidence that the nervous system responds to the specific demands of training, rather than to nonspecific activity, is well established. As reviewed above, skill, endurance, and strength training each engage largely distinct plastic mechanisms within the motor system, indicating that plasticity is dictated by the particular behavioral demands of a task rather than by physical activity in a generic sense [17]. Constraint-induced movement therapy provides a clinical instantiation of this specificity principle: by combining intensive, repetitive, task-specific training of the more-affected limb with techniques that discourage compensatory reliance on the less-affected limb, constraint-induced therapy has produced clinically meaningful motor gains in the substantial majority of treated stroke survivors, an effect attributed to a combination of overcoming learned non-use and inducing genuine use-dependent cortical reorganization [19].

Salience, the behavioral or motivational relevance of the trained task, appears to interact with repetition and specificity rather than substituting for them: reward-paired and behaviorally relevant motor training drives cortical map plasticity in ways that repetition alone, absent behavioral relevance, does not reliably produce.

Intensity and Dose

The dose-response relationship between rehabilitation intensity and functional outcome has been examined extensively in stroke rehabilitation, with a generally consistent, though not unlimited, positive relationship between training dose and recovery. Trials delivering very high cumulative doses of upper-limb therapy, on the order of tens to hundreds of hours, have produced substantially larger functional gains than trials delivering only a few dozen hours, and contemporary reviews argue that usual clinical care in most health systems delivers doses far below what preclinical and clinical evidence indicates is necessary to drive large, behaviorally meaningful reductions in impairment [6]. At the same time, the relationship between dose and outcome is not simply linear or universal: some randomized trials directly comparing higher- and lower-dose regimens in the chronic phase after stroke have failed to find a dose advantage for functional capacity, even though real-world retrospective analyses of routinely collected rehabilitation data have found a positive relationship between session intensity and recovery [7]. This apparent inconsistency is informative for a reserve-maturity framework: it suggests that intensity alone is insufficient without adequate specificity, salience, and correctly timed delivery, and that individual variation in available reserve may itself moderate how much a given dose of training can accomplish.

Timing: A Post-Injury Window of Heightened Responsiveness

Perhaps the most direct evidence for an interaction between reserve, maturity, and rehabilitation stimulus comes from preclinical work on the timing of post-stroke rehabilitative training. Biernaskie and Corbett (2001) demonstrated that enriched rehabilitative training, combining complex housing with daily skilled-reaching practice, produced substantially improved forelimb motor function and increased dendritic branching in perilesional and contralesional motor cortex following focal ischemic injury in rats [20]. A subsequent study from the same group showed that the efficacy of this same rehabilitative protocol declined sharply with the time elapsed since injury: training initiated five or fourteen days after stroke produced significantly better recovery, and correspondingly greater dendritic growth, than training delayed until thirty days post-injury, at which point rehabilitative training produced no more benefit than spontaneous recovery alone [21]. This finding indicates that the injury itself transiently increases neuroplastic reserve in perilesional and connected tissue, opening a time-limited window analogous to a developmental critical period, during which appropriately specific rehabilitative stimulation can be recruited with disproportionate effect; outside that window, the same stimulus produces markedly less benefit, even though some capacity for improvement, consistent with residual reserve, remains available [22].

This post-injury timing window has direct relevance for the maturity principle as well as the reserve principle, because it demonstrates that focal injury can transiently increase local plastic reserve independent of the organism's overall developmental or aging stage, effectively reopening a critical-period-like state within an otherwise mature nervous system. The clinical implication is that timing of rehabilitation delivery, not only its intensity or specificity, functions as an independent determinant of outcome, a point increasingly reflected in stroke rehabilitation guidelines that recommend against both excessively delayed and excessively premature intensive mobilization.

Priming Mechanisms: Exercise and Neurotrophic Signaling

A further stimulus-side mechanism relevant to the reserve principle is the capacity of acute aerobic exercise to transiently increase circulating BDNF and cortical excitability, thereby priming the nervous system for subsequent motor learning. Single bouts of high-intensity aerobic exercise have been shown to increase serum BDNF several-fold and to facilitate subsequent long-term-potential-like plasticity and implicit motor sequence learning in healthy adults, an effect

interpreted as evidence that acute exercise can transiently elevate the neurochemical substrate available for subsequent experience-dependent plasticity. Because BDNF signaling is also among the mechanisms implicated in age-related decline of synaptic plasticity, exercise-based priming represents a plausible route by which externally applied stimulation could partially offset age-related constraints on neuroplastic reserve, consistent with the reserve principle's emphasis on reserve as recruitable rather than fixed.

Summary: Stimulus Properties as the Bridge Between Reserve and Maturity

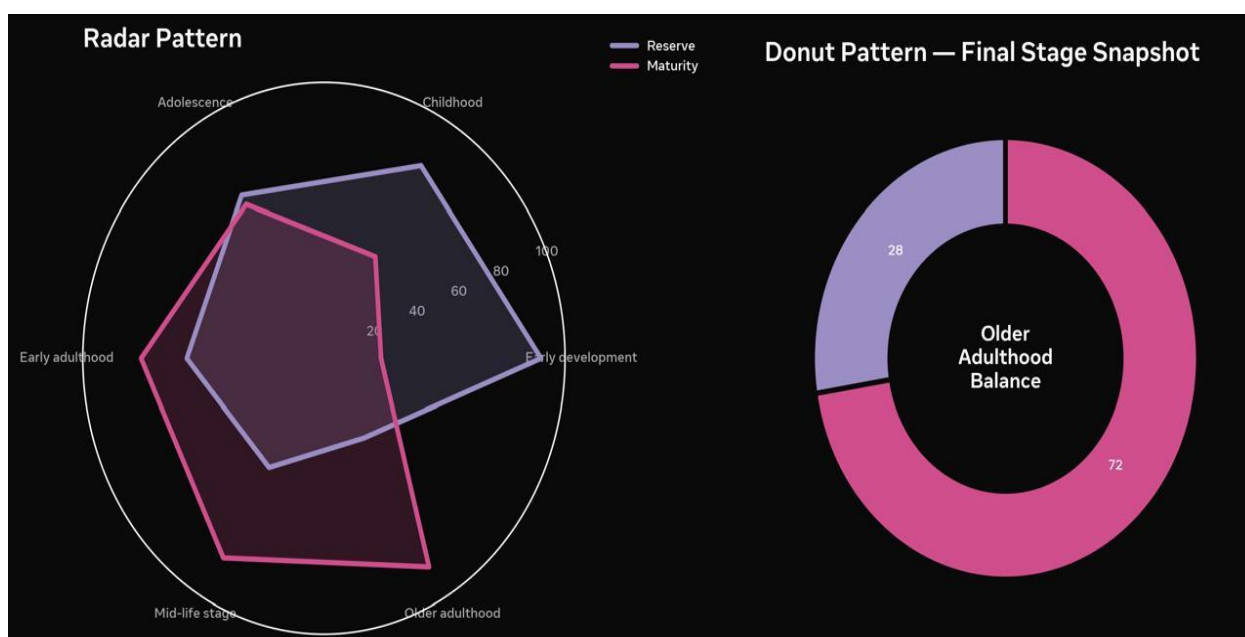
Across this literature, six stimulus properties recur as determinants of whether available neuroplastic reserve is successfully recruited toward adaptive, rather than maladaptive or absent, reorganization: specificity to the target function, sufficient intensity and dose, meaningful repetition, behavioral salience, correct timing relative to injury or developmental stage, and adequate sensory and motor engagement. None of these properties is sufficient in isolation, and their relative importance appears to vary with the maturity of the nervous system being treated: developmentally immature systems may respond to comparatively broad and unstructured stimulation, whereas mature and aged systems appear to require increasingly specific, salient, and correctly timed stimulation to achieve comparable functional gains, a pattern directly predicted by the interaction between the reserve and maturity principles.

Toward an Integrative Model: Bhaskare's Neuroplastic Reserve–Maturity Framework

The evidence synthesized above supports an integrative model in which functional neural adaptation is jointly determined by three interacting factors: the individual's available neuroplastic reserve, the maturity-related organization of that reserve, and the properties of any external rehabilitation stimulus applied. Neuroplastic reserve, drawn together from cognitive and brain reserve theory, neuro-oncological neuroplastic-potential mapping, and synaptic aging research, is proposed to be generally greater during early development and to become progressively more constrained across middle and older adulthood, while remaining substantially variable between individuals of the same chronological age and substantially structured by local connectomic architecture rather than uniformly distributed across the brain [12,13].

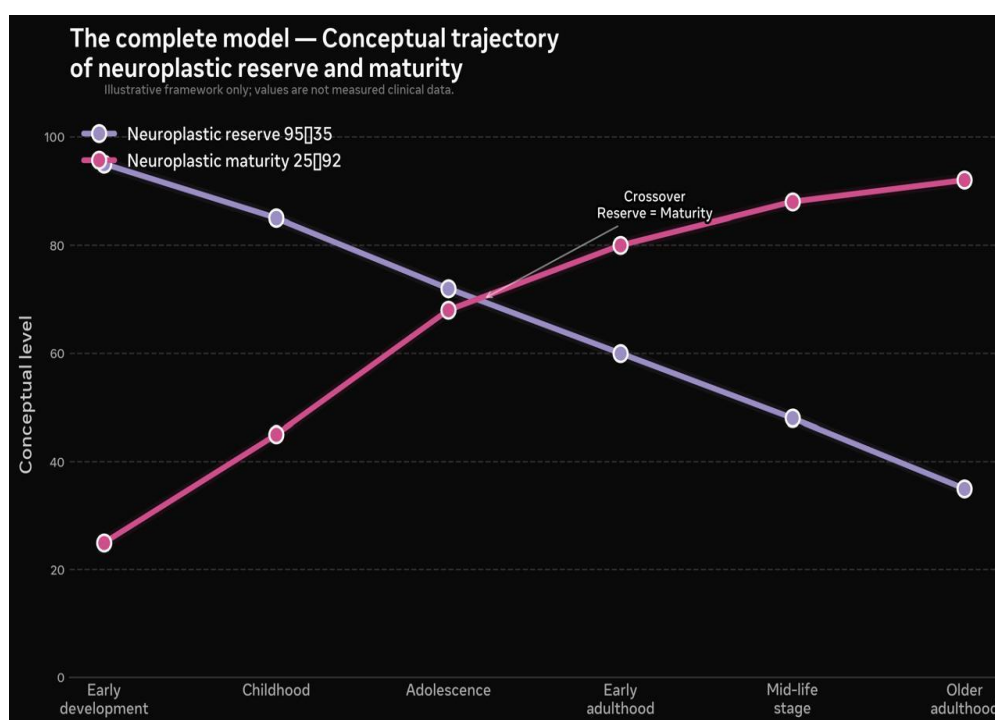
Neuroplastic maturity, drawn together from critical-period neuroscience, motor-learning specialization research, and the revised Kennard-principle literature, is proposed to increase across the same developmental trajectory along which reserve declines, reflecting a genuine shift from broad, developmentally programmed plasticity toward specialized, network-organized, experience-dependent plasticity, a shift that is itself actively regulated rather than a passive loss of capacity [3,8].

Bhaskare's Neuroplastic Reserve–Maturity Model's central hypothesis, consistent with the evidence reviewed, is that individuals with greater available neuroplastic reserve possess greater raw capacity for neural adaptation, that maturation and aging constrain this reserve while simultaneously increasing neural specialization and organizational efficiency, and that appropriately timed and sufficiently specific external rehabilitation stimuli can nonetheless recruit and direct whatever residual neuroplastic capacity remains available across the lifespan. This hypothesis accommodates findings that a reserve-only or maturity-only account cannot: it explains why post-injury rehabilitation windows exist even in adult, fully mature nervous systems (a transient, injury-induced increase in local reserve, consistent with); why slow-growing tumors permit substantially more functional reorganization than fast-growing lesions of similar size (time-extended engagement of specialized, maturity-organized reorganizational mechanisms, consistent with) [11,21].



why very early injury does not uniformly produce better outcomes than adult injury (functions dependent on prolonged network maturation may be more, not less, vulnerable to injury before that maturation is complete, consistent with) [8]. and why intensity or dose alone is an unreliable predictor of rehabilitation outcome (specificity, salience, and timing moderate how effectively a given dose engages available reserve, consistent with) [1,7].

Conceptually, reserve and maturity can be represented as two trajectories moving in largely opposite directions across the lifespan: reserve beginning high in early development and declining, with individual variation, into older adulthood, while maturity, understood here as the degree of specialization and organization of the neuroplastic mechanisms available, begins comparatively low and rises across the same period. Functional recovery capacity at any given point in the lifespan is proposed to reflect the interaction of these two trajectories with the properties of whatever rehabilitation stimulus is applied, rather than either trajectory in isolation. This framing generates directly testable predictions, discussed further below, including the prediction that measures of local neuroplastic reserve, such as connectomic integrity or neurotrophic signaling capacity, should predict rehabilitation responsiveness independently of chronological age, and the prediction that the optimal specificity and intensity of a rehabilitation stimulus should shift systematically with the maturity, rather than merely the age, of the nervous system being treated.



Discussion

This review set out to synthesize evidence bearing on two theoretical constructs proposed by the author, Bhaskare's Principle I (the Neuroplastic Reserve Principle) and Bhaskare's Principle II (the Neuroplastic Maturity Principle), and on the properties of rehabilitation stimuli that link them to functional recovery. Three broad conclusions follow from the synthesis. First, reserve and maturity are empirically dissociable constructs rather than two descriptions of the same underlying decline: reserve concerns how much adaptive capacity is available, while maturity concerns how that capacity is organized and what form of stimulation is required to access it, and treating them as a single lifespan trajectory obscures finding, such as the Kennard principle paradox and the post-injury rehabilitation window, that require both constructs to explain. Second, across every domain reviewed, from critical-period visual plasticity to tumor-associated cortical reorganization to stroke rehabilitation dose-response trials, residual plastic capacity in mature and even aged nervous systems is consistently found to be recruitable rather than simply absent, provided that the rehabilitation or therapeutic stimulus is sufficiently specific, salient, intensive, and correctly timed. Third, the properties of the rehabilitation stimulus itself, rather than functioning as a background variable, appear to interact directly with both reserve and maturity in ways that have direct clinical implications.

Clinical Implications

For stroke and traumatic brain injury rehabilitation, the reserve–maturity framework provides a principled argument against withholding intensive, specific rehabilitation from older patients on the assumption that plasticity is simply unavailable outside early adulthood; the evidence instead suggests that older nervous systems retain meaningful, if organizationally different, capacity for adaptation, and that the primary clinical lever available is the specificity, timing, and intensity of the stimulus applied rather than the patient's age per se. For pediatric neurology, the framework cautions against a simple “younger is always better” heuristic in prognostic counseling after early brain injury, particularly for functions that depend on prolonged network maturation, and supports individualized rather than age-based prognostic

estimates. For neuro-oncological surgery, the framework provides a conceptual basis for prehabilitation strategies, including preoperative non-invasive brain stimulation, that seek to actively recruit neuroplastic reserve before tumor resection rather than treating a patient's functional map as fixed, and it suggests that the slow growth rate of diffuse low-grade gliomas may itself be a clinically exploitable feature, since it affords the specialized, maturity-organized reorganizational mechanisms of the adult brain sufficient time to engage.

Testability and Future Directions

As presented, Bhaskare's Neuroplastic Reserve–Maturity Model is a conceptual synthesis rather than a quantitatively parameterized theory, and its central constructs, reserve and maturity, are not yet operationalized as measurable clinical variables with established psychometric or biological validity. Future work should prioritize the development of convergent biomarkers of local neuroplastic reserve, potentially integrating connectomic imaging, neurotrophic signaling assays, and non-invasive stimulation-response protocols, that could be tested prospectively as predictors of rehabilitation outcome independent of chronological age. Comparable work is needed to operationalize neuroplastic maturity as a measurable property of specific neural circuits rather than a global age-linked trait, potentially through indices of excitatory-inhibitory balance or network specialization derived from electrophysiological or connectomic data. Prospective trials that experimentally vary rehabilitation stimulus specificity, intensity, and timing while stratifying patients by independent measures of reserve and maturity, rather than by chronological age alone, would provide the most direct test of the model's central hypothesis.

Limitations

Several limitations qualify the conclusions of this review. First, the review is narrative-systematic rather than a quantitative meta-analysis; no pooled effect sizes are reported, and the strength of evidence varies considerably across the domains synthesized, from well-replicated cellular mechanisms of critical-period regulation to comparatively preliminary human neuro-oncological prehabilitation studies. Second, much of the mechanistic evidence for reserve decline and for the post-injury timing window derives from rodent models, and the degree to which specific timelines, such as the thirty-day post-stroke window identified in rat studies, translate directly to human rehabilitation timescales remains an open empirical question. Third, the reserve and maturity constructs as defined here are, at present, conceptual rather than operationalized; the review synthesizes literature that is consistent with these constructs but was not, in most cases, designed to test them directly, and alternative theoretical framings of the same underlying data remain possible. Finally, publication and citation patterns in this literature may favor positive findings of plasticity and recovery, and the review may accordingly understate the frequency with which rehabilitation stimuli fail to produce meaningful reorganization despite apparently adequate specificity, intensity, and timing.

Conclusion

The literature reviewed here supports Bhaskare's Principles of Neuroplasticity, a framework in which neuroplastic reserve (Bhaskare's Principle I) and neuroplastic maturity (Bhaskare's Principle II) are distinct but interacting determinants of the nervous system's capacity for adaptive change. Reserve, the raw intrinsic capacity for structural, synaptic, and functional modification, is generally greater in early development and becomes more constrained, though never entirely absent, with maturation and aging.

Maturity, the degree to which the mechanisms of plasticity have become specialized, organized, and experience-dependent, increases across the same developmental trajectory and is not simply the inverse of reserve but a qualitatively distinct organizational property with its own clinical consequences, including the Kennard principle paradox and the differential vulnerability of late-maturing cognitive functions to early injury. Critically, across stroke rehabilitation, tumor-associated cortical reorganization, and developmental critical-period research alike, residual plastic capacity in mature and aged nervous systems is consistently recruitable given a sufficiently specific, salient, intensive, and correctly timed external stimulus. This synthesis supports the central hypothesis that individuals with greater available neuroplastic reserve may possess greater capacity for neural adaptation, that maturation and aging constrain this reserve while increasing neural specialization, and that appropriately designed rehabilitation stimuli can nonetheless recruit and direct residual neuroplastic capacity across the lifespan. Translating this conceptual synthesis into a validated, clinically actionable model remains the principal task for future research in this area.

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