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Content

Review article

- Yuliia G. Serhiienko, Natalia O. Nekrasova, Yurii O. Kotlyarevskyi, Olena K. Riznychenko, Volodymyr O. Yurko*
Endothelial dysfunction as a predictor of post-reperfusion complications in acute ischemic stroke: a literature review 3-9
- Yuriy V. Flomin, Anastasiia L. Orlovska, Anastasiia V. Hrytsan, Maryna V. Gulyayeva, Mykola Y. Romanyshyn, Anastasiia V. Honchar, Iryna R. Havryliv, Maksym H. Tsariapkin*
Gait disorders in neurological diseases: clinical significance, assessment methods, and rehabilitation potential 10-18
- Vitaliy Y. Molotkovets, Vasyl V. Kondratyuk, Maria Y. Pobut*
Supramaximal resection of glioblastoma: from pathophysiological rationale to clinical standardization 19-27

Original article

- Yaroslav D. Bondarenko, Oksana I. Kauk, Iryna O. Hovbakh, Lidiya V. Cherkashyna, Olena K. Riznychenko*
A unified mechanism of cross-modal neuroplasticity: hub-based architecture, neurochemical and epigenetic regulation in sensory compensation and synesthesia 28-42
- Giussepe Condori-Jara, Jose Acha-Sanchez, Jose Amado-Tineo*
Predictive value of the Glasgow Coma Scale for postoperative functional outcomes in patients with aneurysmal subarachnoid hemorrhage: a retrospective study (2018–2021) 43-52
- Artem V. Rozumenko, Valentyn M. Kliuchka, Olexandr M. Lisianyi, Oleg O. Dyma, Tetyana A. Malysheva, Volodymyr D. Rozumenko*
A ventricular-wall topographic classification for periventricular high-grade gliomas: corridor selection and early surgical outcomes—a pilot single-center series 53-61
- Mykola O. Zorin*
A 25-year experience with percutaneous vertebroplasty 62-68
- Mark Y. Hlukhenkyi, Nikolay V. Kadzhaya, Alexander S. Gotin, Andriy P. Huk*
Cranioplasty during the intermediate postoperative period after gunshot wounds to the brain: clinical and organizational aspects of timely intervention 69-82
- Anton M. Dubrovka, Volodymyr O. Fedirko*
Intraoperative findings underlying pain recurrence after microvascular decompression for trigeminal neuralgia: experience with revision surgery 83-92
- Olga V. Guzun, Oleg S. Zadorozhnyy, Natalia V. Konovalova, Vira A. Vasyuta*
Neovascular glaucoma as a chronic neuroischemic optic neuropathy: the impact of cumulative burden on vision-related quality of life in a 36-month prospective study 93-101
- Vladyslav I. Kaliuzhka, Mykyta A. Markevych, Volodymyr K. Klochkov, Volodymyr Y. Prokopiuk, Volodymyr O. Piatykov*
Intrathecal cerium oxide nanoparticles improve functional recovery after experimental subarachnoid hemorrhage: a proof-of-concept study 102-115
- Natalia O. Mykhailovska, Iaroslav P. Zinkevych, Kostiantyn R. Kostyuk*
Analysis of the effectiveness of subthalamic nucleus deep brain stimulation in patients with advanced parkinson's disease: one-year follow-up from ukrainian experience 116-122

Case report

- Vladyslav I. Kaliuzhka, Mykyta A. Markevych, Mark I. Hlukhenkii, Iurii O. Babalian, Volodymyr O. Piatykov*
Endoscopic evacuation of intracerebral hemorrhage using mixed reality navigation: case report 123-126
- Anh Quynh Dang, Thuy Thi Hong Le, Hung Phi Nguyen, Huyen Thi Tran, Tuan Quoc Nguyen, Vy Thuc Nguyen, Phung Thi Kim Le, Nguyet Le Nhu Dang, Phi Duong Nguyen*
Spinal neurocysticercosis: a rare and challenging diagnosis in imaging 127-132

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A unified mechanism of cross-modal neuroplasticity: hub-based architecture, neurochemical and epigenetic regulation in sensory compensation and synesthesia

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Background: Synesthesia and sensory compensation have emerged as pivotal topics in contemporary neuroscience, elucidating the remarkable adaptability of the human brain. Both phenomena demonstrate the capacity for cross-modal integration and functional reassignment of neural networks, challenging the traditional modular view of sensory processing. Although synesthesia was long regarded as a rare anomaly and sensory compensation as a secondary adaptation to sensory loss, current evidence indicates that they represent convergent manifestations of the same universal principle: the brain's intrinsic ability to reorganize itself through neuroplasticity. Consequently, these processes illuminate not only the dynamics of perception but also broader mechanisms of cognition, creativity, and memory enhancement.

Objective: This study investigated the convergent neurobiological mechanisms underlying synesthesia and sensory compensation, focusing specifically on the role of plasticity universal convergence nodes (supernodes) —including the superior colliculus, intraparietal sulcus, superior temporal sulcus, and prefrontal projections—as coordinators of sensory remapping and cross-modal integration. It was hypothesized that these structures act as dynamic hubs capable of rerouting sensory flows under conditions of deprivation or enrichment, thereby enabling both compensatory and associative forms of neuroplasticity.

Methods: A systematic literature review was performed across PubMed, Scopus, Web of Science, and Google Scholar, encompassing studies published between 2000 and 2026. The search strategy employed Boolean logic and combined English and Ukrainian keywords related to “sensory compensation”, “synesthesia”, “neuroplasticity”, “multisensory integration”, and specific hub structures. Inclusion criteria encompassed experimental and clinical studies with neuroimaging (fMRI, MEG, PET, DTI), electrophysiological recordings, or molecular analyses in human participants and animal models. Review articles and meta-analyses providing integrative perspectives on multisensory plasticity were also included, while purely descriptive or metaphorical accounts were excluded.

Results: Evidence across modalities demonstrates that sensory reorganization is governed by a hub-based architecture rather than diffuse activation. Primary cortices, such as V1 and A1, exhibit functional reassignment when typical input is absent, while associative hubs coordinate the redistribution and integration of multisensory information. The superior colliculus enables rapid topographic integration of cross-modal signals; the intraparietal sulcus performs coordinate transformations and oscillatory gating; the superior temporal sulcus resolves temporal correspondences in audiovisual integration; and prefrontal projections provide top-down modulation and precision weighting. These structural roles are reinforced by molecular mechanisms: neurotrophin signaling (BDNF, NT-3, IGF-1) initiates plastic readiness, while epigenetic regulation (DNA methylation, histone acetylation, noncoding RNAs) ensures stabilization and long-term retention of novel sensory maps. Together, these mechanisms establish a dual-layer system of rapid reconfiguration and durable consolidation. Importantly, the same pathways that allow the blind to develop enhanced spatial hearing or the deaf to refine visuomotor communication also support the emergence of synesthetic cross-modal associations in individuals without sensory loss.

Conclusions: Synesthesia and sensory compensation should be interpreted not as exceptional deviations but as complementary expressions of a universal adaptive code of neuroplasticity. By activating supernodes, the brain dynamically reconfigures its sensory architecture in response to environmental demands, whether by necessity (compensation) or by enrichment (synesthesia). This framework has profound theoretical and practical implications. It advances our understanding of the brain as a dynamic network rather than a fixed modular system and provides translational insights into clinical neurorehabilitation, multisensory pedagogical strategies, and emerging fields of cognitive enhancement. Harnessing these mechanisms may enable new interventions—from targeted neuromodulation and neurotrophin-based therapies to educational methods that deliberately engage cross-modal correspondences—thus transforming the way we approach both deficits and potentials of the human mind.

Keywords: synesthesia; sensory compensation; neuroplasticity; cross-modal integration; plasticity supernodes; neurotrophins; epigenetic regulation; multisensory processing

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Introduction

Recently, synesthesia and sensory compensation have become major areas of investigation in neuroscience, demonstrating the brain's profound capacity to integrate and reorganize sensory maps [1, 2]. Synesthesia represents a stable neurological phenomenon in which stimulation of one sensory or cognitive channel automatically evokes an additional perception in another [3, 4]. Unlike illusions or metaphorical associations, these experiences have objective neurophysiological correlates, confirmed by neuroimaging studies [2-4]. A classical example is grapheme-color synesthesia, in which letters and numbers are associated with certain shades, or the music-color synesthesia, in which auditory stimuli elicit color perceptions [1, 3, 5-7]. Consequently, synesthesia is classified not as a pathology but as a stable feature of neuronal organization, thereby facilitating a deeper understanding of the fundamental principles of multisensory integration.

While the mechanisms of synesthesia remain debated, current hypotheses converge at several key levels [4-10]. First, increased functional or anatomical connectivity between sensory cortical areas has been documented in many synesthetes; specifically, activation of the color area V4 in response to graphemic or numerical stimuli [10-12]. Second, there is evidence of reduced efficiency of inhibitory mechanisms that normally segregate sensory streams, thereby allowing signals to access atypical channels [13-18]. Third, congenital forms of synesthesia may be determined by features of early neurodevelopment, when the boundaries between sensory systems have not yet rigidly established [13-18]. Genetic factors also play a critical role, influencing the formation of axonal connections and synaptic organization [19]. The diversity of synesthesia types underscores its multidimensional nature as a neurological phenomenon [8-19]. Grapheme-color synesthesia is the most common subtype; however, numerous additional forms have been described, including music-color, lexical-gustatory, spatial-numerical, and more unusual affective variants in which emotional states evoke sensory percepts [14-21]. Regardless of modality, all these forms demonstrate the brain's core feature: its capacity to establish stable cross-modal correspondences [21-23]. The cognitive profile of synesthetes has attracted increasing scientific attention [24]. Numerous studies have reported improved episodic and semantic memory, a high capacity for associative thinking, and increased creativity in artistic and scientific practices [24-26]. Neuroimaging data have indicated enhanced activity within parietal structures involved in multisensory integration, thereby supporting the systemic nature of this phenomenon [26-28]. Thus, synesthesia not only reveals hidden mechanisms of neuroplasticity but also demonstrates that the brain fundamentally functions not as a set of isolated modules but as a dynamic network with a high potential for integration [26-29]. The relevance of studying synesthesia and sensory compensation stems from their reflection of the brain's universal capacity for adaptive reorganization. Their comparison makes it possible to identify the basic principles of neuronal network organization, which is important not only for science but also for applied medicine and technology. The

central hypothesis is that the brain possesses specialized supernodes that coordinate the reassignment of sensory streams. Within this model, sensory deprivation or intensive training triggers the activation of supernodes, leading to the reconfiguration of sensory maps and the formation of new cross-sensory associations. Consequently, this process drives cognitive and sensory enhancement [24, 30].

Accordingly, synesthesia is interpreted not as a deviation but as a universal mechanism of adaptive neuroplasticity. At the applied level, this study posits that sensory deprivation triggers compensatory plasticity functionally similar to synesthesia: in blind individuals—auditory and tactile signals are integrated into visual maps; in deaf individuals—visual-motor stimuli activate the auditory cortex; whereas in synesthetes without sensory deficits, the same mechanisms manifest as "excess integration". The scientific novelty of this conceptual framework shifts the focus from viewing synesthesia as an exception to treating it as a universal code of brain adaptability. The practical significance covers a wide range of directions: from new approaches in neurorehabilitation and educational practices to modeling multisensory artificial intelligence systems and overcoming social stigmatization of atypical cognitive profiles.

Research aim: To identify the common neurobiological mechanisms of sensory compensation and synesthesia, with particular emphasis on the role of plasticity supernodes (superior colliculus, intraparietal sulcus, superior temporal sulcus, prefrontal projections) in the reorganization of sensory maps and the formation of cross-modal associations. This study aims to substantiate the concept that these phenomena represent not exceptional cases but manifestations of the brain's universal code of adaptation.

Materials and methods

A systematic literature search was conducted in the international scientific databases PubMed, Scopus and Web of Science, as well as in the open-access repository Google Scholar. The time range covered the period from 2000 to 2026 facilitating the integration of both classical and the most recent publications in the field of neuroplasticity. For the search, English- and Ukrainian-language keywords and their combinations were used: "sensory compensation AND neuroplasticity", "synesthesia AND cross-modal integration", "multisensory processing AND brain reorganization", "superior colliculus, intraparietal sulcus, superior temporal sulcus AND plasticity", as well as "Sensory compensation", "synesthesia", "neuroplasticity". Search strategies were formed using logical operators (AND, OR, NOT), as well as using filters by year of publication, document type and full-text availability. Inclusion criteria were defined as follows: (1) original experimental studies containing neuroimaging data (fMRI, MEG, PET, DTI), electrophysiology or molecular neurobiology; (2) investigations examining the mechanisms of sensory compensation or synesthesia in humans or in animal models; (3) reviews and meta-analyses summarizing current concepts of multisensory integration and neuroplasticity; (4) publications in peer-reviewed journals

indexed in major citation databases. Exclusion criteria covered: (1) descriptive articles without empirical or neurobiological data; (2) publications without full-text access or with limited information on methodology; (3) materials that considered the phenomena of sensory compensation or synesthesia exclusively in artistic, philosophical or metaphorical context; (4) works not related to the topic of neuroplasticity. After the initial search, all identified publications were checked based on the title and abstract. Eligible papers underwent the second stage of selection through full-text analysis. At each stage, articles failing to meet the inclusion criteria were excluded. Finally, a corpus of literature was formed, covering the results of experimental studies, neuroimaging works and theoretical generalizations, which became the basis for the conceptual integration of the phenomena of sensory compensation and synesthesia.

Results

Neuroimaging data

The most convincing evidence for the existence of neuroplasticity supernodes comes from neuroimaging studies involving individuals with sensory deficits, as well as from experiments with temporary sensory deprivation [24-34]. Functional magnetic resonance imaging (fMRI), magnetoencephalography (MEG), positron emission tomography (PET) and diffusion tensor imaging (DTI) data consistently demonstrate that sensory reorganization has a systemic character and is implemented through a limited set of structures capable of coordinating the redistribution of sensory streams [30-35]. The obtained data are schematically reflected in **Table 1**.

In the case of congenital or early-acquired blindness, the primary visual cortex (V1), as well as adjacent areas of the extrastriate cortex (V2-V4), are consistently activated in response to auditory and tactile tasks [36, 37]. Spatial auditory discrimination in such individuals is accompanied by activation of the occipital areas, and this activation correlates with the accuracy of task performance [37-39]. MEG data indicate that the latency of activation

peaks in the occipital cortex approaches the early sensory responses characteristic of visual information processing in sighted individuals [37, 39-41]. At the same time, DTI-tracing reveals enhanced connectivity between subcortical auditory structures and occipital cortical areas, creating an anatomical basis for functional reassignment [37, 41, 42]. Similar phenomena are recorded in congenital deafness [42-44]. The primary auditory cortex (A1) and adjacent areas of the superior temporal gyrus are activated in response to visual stimuli, including simple light stimuli [42-46]. These findings suggest that even primary auditory areas can function, as we propose, as multisensory nodes integrated into visuomotor networks [29, 40-47].

Studies with temporary sensory deprivation in healthy adults confirm that reassignment mechanisms can unfold within relatively short time intervals [29, 48-50]. After only several days of visual sensory isolation, the occipital cortex begins to activate during the performance of tactile tasks [50-53]. Similar results are also observed in the auditory modality, where restriction of auditory input signals is associated with increased activation in temporal multisensory areas during visual stimulation [44, 53-55]. These observations indicate that supernodes are the latent architectural basis of the brain, which is activated under conditions of sensory deficit [29, 55-57]. A synthesis of the findings reported in [37-58] allows for the classification of supernode organization into three distinct hierarchical levels. First, primary sensory areas (e.g., V1, A1) demonstrate the ability for functional reassignment and reception of atypical signals. Second, associative multisensory zones (for example, superior temporal sulcus, intraparietal sulcus) perform integrative and switching functions. Third, frontal areas (dorsolateral prefrontal cortex, anterior cingulate cortex) participate in cognitive control and targeted formation of new sensory maps. Consequently, neuroimaging data confirm that reorganization of sensory systems occurs not through diffuse activation but through the activity of specialized supernodes that coordinate adaptive reconfiguration of neuronal networks.

Table 1. Neuroimaging Evidence for Sensory Supernode Plasticity

Level / Region	Modality / Method	Key Findings
Primary visual cortex (V1), extrastriate cortex (V2-V4)	fMRI, MEG, PET, DTI	Activated by auditory and tactile tasks in congenital or early blindness; activation correlates with spatial hearing performance and enhanced connectivity with subcortical auditory structures
Primary auditory cortex (A1), superior temporal gyrus	fMRI, MEG, PET, DTI	Activated by visual stimuli including simple light perception in congenital deafness; supports multisensory integration
Occipital cortex in temporary sensory deprivation	fMRI, MEG	Rapid functional reassignment to tactile tasks after a few days of visual deprivation
Temporal cortex in temporary auditory deprivation	fMRI, MEG	Increased activation during visual stimulation when auditory input is restricted
Associative multisensory regions (superior temporal sulcus, intraparietal sulcus)	fMRI, MEG, DTI	Integrative and hub functions; coordinate sensory redistribution
Frontal regions (dorsolateral prefrontal cortex, anterior cingulate cortex)	fMRI, MEG	Cognitive control and formation of new sensory maps

Multisensory nodes as "switches"

Collectively, evidence from neuroimaging, electrophysiological, and causal interventions—including non-invasive stimulation methods in humans, optogenetics and microstimulation in animal experiments—indicates that adaptive reconfiguration of sensory maps is implemented not diffusely but through a limited set of nodes capable of performing the function of dynamic switching of flows [37-62]. These structures combine three key properties: (i) input convergence from different modalities, including subcortical and cortical sources; (ii) mechanisms of

rapid retuning of effective connectivity (millisecond-to-hour) through changes in excitability, gain, and interareal synchronization; (iii) molecular-synaptic conditions for long-term stabilization of new routes (days-weeks) through trophic and epigenetic cascades. Against this background, the superior colliculus (SC), intraparietal sulcus (IPS), superior temporal sulcus (STS), and prefrontal projections form a hierarchical switching system embedded in thalamo-cortical loops involving the thalamic reticular nucleus, pulvinar, and basal ganglia, which is further schematically reflected in **Fig. 1**, **Table 2** [54-65].

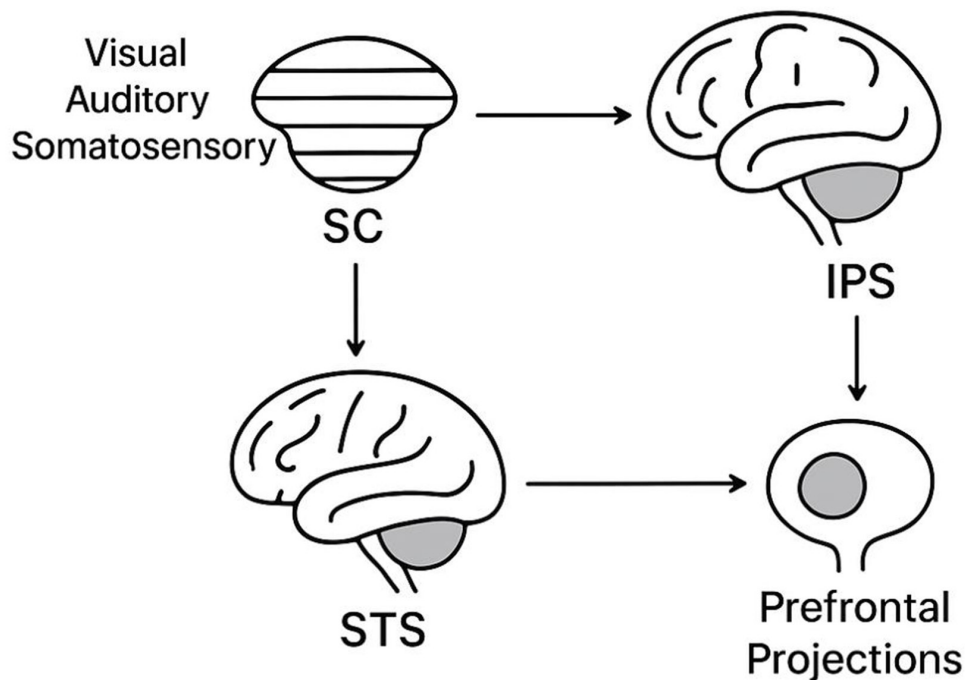


Fig. 1. Multisensory nodes as Switches

Table 2. Multisensory Hubs as Dynamic Switches in Adaptive Sensory Reorganization

Multisensory Hub	Evidence / Method	Key Properties / Functions
Superior colliculus (SC)	fMRI, MEG, PET, DTI, animal electrophysiology, optogenetics	Input convergence from multiple modalities; rapid reconfiguration of effective connectivity; molecular/synaptic support for long-term stabilization
Intraparietal sulcus (IPS)	fMRI, MEG, non-invasive stimulation, animal studies	Dynamic routing of sensory flows; integrative hub; participates in thalamo-cortical loops
Superior temporal sulcus (STS)	fMRI, MEG, DTI, causal interventions	Multimodal integration; fast network reorganization; synaptic plasticity for sustained map changes
Prefrontal projections	fMRI, MEG, non-invasive stimulation, animal experiments	Hierarchical control; cognitive modulation; guides adaptive sensory remapping
General mechanism	—	Adaptive sensory map reconfiguration via restricted, dynamic switch-like hubs rather than diffuse activation

Superior colliculus is a subcortical node with a clear laminar architecture: its superficial layers predominantly process visual input, while deeper ones integrate auditory and somatosensory signals. The spatial maps of these modalities are topographically aligned, facilitating the rapid (within tens of milliseconds) formation of a common peripersonal representation. The well-documented principles of multisensory superadditivity and “inverse effectiveness” (greater integrative gain for weak or partially uncertain stimuli) reflect the computational function of SC as a switch of the orienting response. Specifically, under visual insufficiency, the weights of auditory and tactile projections are increased, shifting the routing toward alternative channels with minimal delay [65-73]. Causal control over these shifts is exerted through loops with the basal ganglia (inhibitory output of substantia nigra pars reticulata) and through thalamo-cortical feedback (pulvinar), which determine whether local integration is transformed into a globally expressed orienting output [62-73]. At the microcircuit level, critical elements are GABAergic interneurons of the deep layers, which set “windows of excitability” for multisensory summation, while rapid plastic changes are implemented through spike-timing dependent plasticity (STDP) rules in convergent collaterals of auditory and somatosensory fibers [65-75].

Intraparietal sulcus acts as a cortical node of mapping and transformation of coordinates. Its neurons encode the position of stimuli in different reference frames—including retino-, head-, body-centered—performing the “stitching” of tactile and visual signals into a unified spatial map of actions. Through dense connections with temporal, occipital, and frontal fields, IPS implements topographic redirection: under modality deprivation it increases the efficiency of projections from preserved channels, maintaining the accuracy of localization and movement planning. At the level of network dynamics, IPS is a generator and retransmitter of alpha-beta rhythms that realize top-down channel selection, while the gamma range marks bottom-up transmission of sensory novelty. Temporary “coupling” (phase-amplitude coupling) between these ranges serves as a gain modulator, which is the mechanism of switching. At the molecular level these processes are sensitive to cholinergic and noradrenergic modulation: acetylcholine increases the precision of the sensory signal in local microcircuits, while noradrenaline optimizes global reactivity to unpredictable events [76-83].

Superior temporal sulcus functions as an associative integrator of temporally structured visual and auditory patterns such as biological motion, articulation, vocalizations. Its computational role lies in solving the “latent correspondence” problem: binding signals that differ in physical nature and temporal dispersion to a common causal event. To achieve this, STS implements integration windows in the range of tens to hundreds of milliseconds, where it aligns amplitude- and phase-specific features of different modalities [84-88]. Under conditions of sensory deprivation, the STS demonstrates “recoding”—a redistribution of neuronal populations from the dominant modality to preserved channels, in particular increased sensitivity to visual surrogates of the audio signal in speech processing tasks. At the microcircuit level, the key factor is the balance

between fast PV-interneurons (perisomatic inhibition) and SOM-interneurons (dendritic inhibition), which determines selectivity to multimodal coincidences and noise filtering; long-term stabilization of new patterns is supported by neurotrophin-mediated remodeling of dendritic spines and regulation of inhibitory tone [84-94].

Prefrontal projections—primarily dorsolateral prefrontal cortex and anterior cingulate cortex, in interaction with frontal eye fields—provide high-level control of switching through mechanisms of precision weighting and choice policy. In terms of predictive coding, these structures supply posterior expectations and precision estimates for different sensory channels, which directly determines gain in lower nodes (SC, IPS, STS) through thalamo-cortical loops and reticular inhibitory gating. Achievement of stable retuning depends on dopaminergic signaling (credit assignment and reinforcement of useful routes) and the cholinergic subsystem (enhancing the processing of behaviorally relevant features), while noradrenaline ensures adaptation to environmental volatility. Causal data show that temporary increase in excitability of prefrontal fields changes the direction of interareal effective connectivity in the fronto-parietal-temporal network, shifting the system into an alternative routing mode without the need for structural reorganization—and thus switching can be implemented as a “soft” retuning of weights and phase synchrony [95-99].

In addition to these four centers, thalamic nodes play a critical role—primarily the pulvinar and thalamic reticular nucleus—which function as relay and gating structures in the switching circuit. The pulvinar coordinates interareal synchronization in the delta-alpha-beta ranges, thereby establishing temporal windows for selective transmission between visual, parietal, temporal, and prefrontal areas. Concurrently, the reticular nucleus acts as an inhibitory shell that doses the flow of thalamo-cortical signals according to prefrontal priorities. At the white matter level, the key trajectories are pulvinar-cortical loop fibers, arcuate and superior longitudinal fasciculi (connectivity STS-IPS-PFC), as well as tectothalamic and tectobulbar pathways that provide subcortical routing [99-105].

The consolidated picture of this system can be outlined as follows: SC provides ultrafast multisensory integration and orienting switching; IPS performs topographic matching and coordinate transformation with rhythmic gain; STS integrates temporally extended multimodal patterns and performs “causal binding”; prefrontal fields determine policy and precision weighting, aligning local integration with global goals. Taken together, these structures implement a unified mechanism of dynamic routing, which on short time scales works through changes in excitability and synchrony, and on long ones is consolidated by synaptic, neurotrophin, and epigenetic reorganization [105-111]. Since the existence and functions of these supernodes are reproduced at the levels of systemic physiology, microcircuits, and molecular cascades, the following question arises: how do these dynamic switches manifest themselves in clinical and cognitive scenarios—from sensory compensation under deprivation to the phenomena of “excessive integration” characteristic of

synesthesia—and what specific switching parameters determine the success or limits of adaptation?

Sensory compensation and synesthesia

Sensory compensation is one of the most illustrative examples of how the brain manifests its capacity for systemic reorganization through the activation of switching nodes of neuroplasticity (**Fig. 2**).

In the case of congenital blindness, it is the occipital cortex, which normally specializes in processing visual signals, that takes on the functions of integrating auditory and tactile streams. This reorganization is not limited to passive activation: in such individuals, topographically organized spatial maps of sounds and touches are formed, which are subsequently integrated into parietal and hippocampal networks, thereby enhancing spatial orientation and episodic memory. Electrophysiological data indicate that the latency of V1 activation in response to auditory stimuli in blind individuals is comparable to the latency of visual responses in sighted individuals, which means reassignment of primary sensory streams at the level of early processing [112-119]. Morphological studies confirm an increase in cortical thickness and dendritic complexity in occipital areas, indicating long-term stabilization of new functions. Conversely, in the case of acquired blindness, the profile of plasticity appears different and clearly depends on the age at which vision loss occurred [29, 112-119]. If deprivation takes place during critical developmental periods, reorganization approximates the patterns characteristic of congenital blindness. In adulthood, however, activation of the

occipital cortex occurs more slowly and has a limited functional effect [112-119]. This demonstrates that neuroplasticity supernodes have phase sensitivity and realize their maximal potential precisely within windows of critical plasticity [29,112-119]. Nonetheless, even in adults, short-term visual deprivation can activate latent networks within a few days, indicating the presence of a hidden architecture of plasticity capable of reactivation through stimulation or pharmacological methods [112-119].

In the case of congenital deafness, the primary auditory cortex and adjacent associative areas, instead of reducing functions, integrate visual and motor stimuli, in particular those associated with articulation, facial expressions, and gestures [112-119]. This provides exceptional accuracy of lip-reading and sign communication, indicating active re-profiling of the auditory system toward visuo-motor integration. Neuroimaging studies confirm that the effectiveness of such reorganization directly correlates with cognitive and behavioral advantages, making compensatory processes an example of systemic adaptation [112-121]. Collectively, these findings indicate that sensory compensation is not an accidental phenomenon, but a forced activation of neuroplasticity supernodes, which reorganize brain maps and provide alternative routes of information processing [29,112-121]. Therefore, under conditions of sensory deprivation the brain retains a fundamental capacity to integrate lost maps into a new network architecture, maintaining cognitive efficiency and behavioral adaptation [112-121].

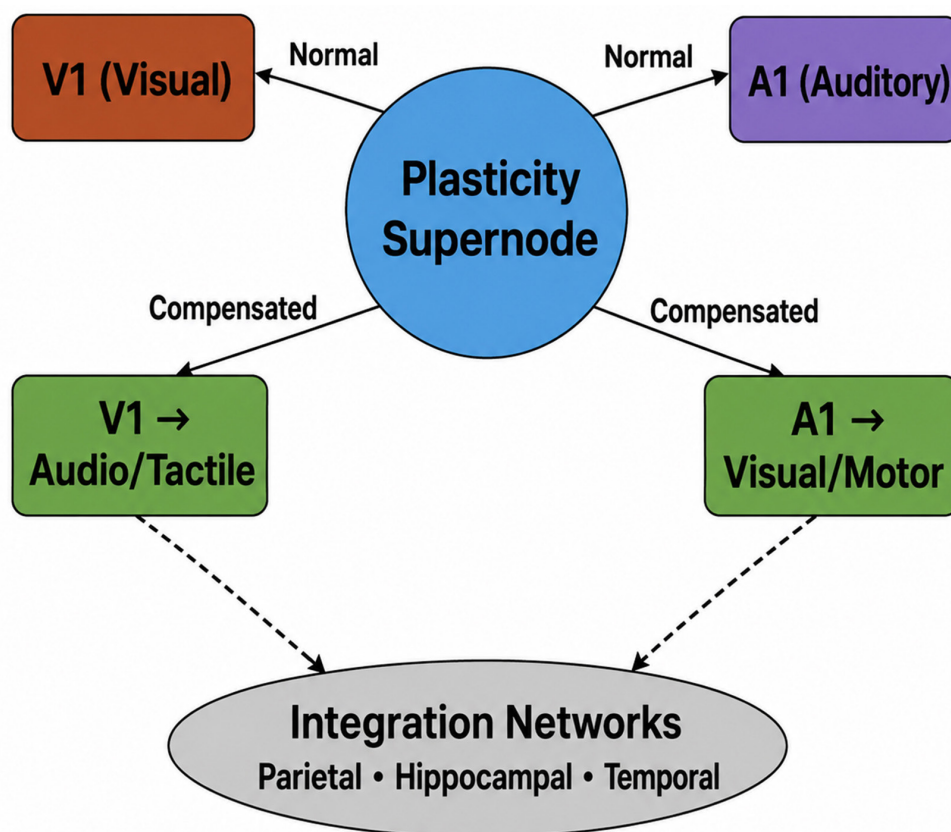


Fig. 2. Sensory Compensation: Neuroplasticity Supernodes

Neurochemical and epigenetic mechanisms

The functioning of neuroplasticity supernodes is determined by two complementary control circuits — neurochemical and epigenetic — which together set both the instantaneous variability of sensory routes and the long-term stabilization of new maps. At the neurochemical level, the key factor is the coordinated activation of neurotrophic and neuromodulatory systems in switching nodes (SC, IPS, STS, prefrontal fields, thalamic gates), which forms a key molecular inducer of plasticity: a vector of trophic and modulatory signals that increases the sensitivity of networks to learning (metaplasticity) and converts local changes in excitability into structural-functional reorganizations. Specifically, brain-derived neurotrophic factor (BDNF), through the TrkB receptor (tropomyosin receptor kinase B), triggers three main signaling cascades: PI3K-Akt-mTOR (phosphoinositide 3-kinase – protein kinase B – mechanistic target of rapamycin), MAPK-ERK (mitogen-activated protein kinase – extracellular signal-regulated kinase), and PLCγ-CaMK (phospholipase C gamma – calcium/calmodulin-dependent protein kinase). These concurrent pathways enhance local protein synthesis in dendrites, stabilize long-term potentiation (LTP), and promote maturation of dendritic spines [122-133]. At the same time, the balance between proBDNF/p75^{NTR} (precursor BDNF/p75 neurotrophin receptor) and mature

BDNF/TrkB finely shifts the equilibrium between long-term depression (LTD) and LTP, setting the direction of synaptic reorganization. Furthermore, neurotrophin-3 (NT-3), interacting with the TrkC receptor (tropomyosin receptor kinase C), regulates the specificity and accuracy of sensory projections, in particular thalamo-cortical inputs into associative cortical areas. Complementing this, insulin-like growth factor 1 (IGF-1), through the IGF1R receptor (insulin-like growth factor 1 receptor) and the mTORC1 (mechanistic target of rapamycin complex 1) and MAPK cascades, increases the metabolic readiness of neuronal ensembles for plastic changes and expands the “resource window” for the formation of new synaptic subnetworks [122-133].

Neuromodulatory systems—cholinergic (basal forebrain), noradrenergic (locus coeruleus), dopaminergic (mesocortical pathways), and serotonergic (raphe nuclei) — establish the point-specific weighting of sensory channels. Within this framework, acetylcholine increases selectivity and improves the signal-to-noise ratio in cortical microcircuits, noradrenaline broadly regulates sensitivity to novelty and environmental volatility, dopamine encodes reinforcement and credits the causal contribution of specific routes, while serotonin modulates temporal constants of integration and impulse control, together defining the switching policy in supernodes [122-137]. The obtained data are schematically shown in **Fig. 3**.

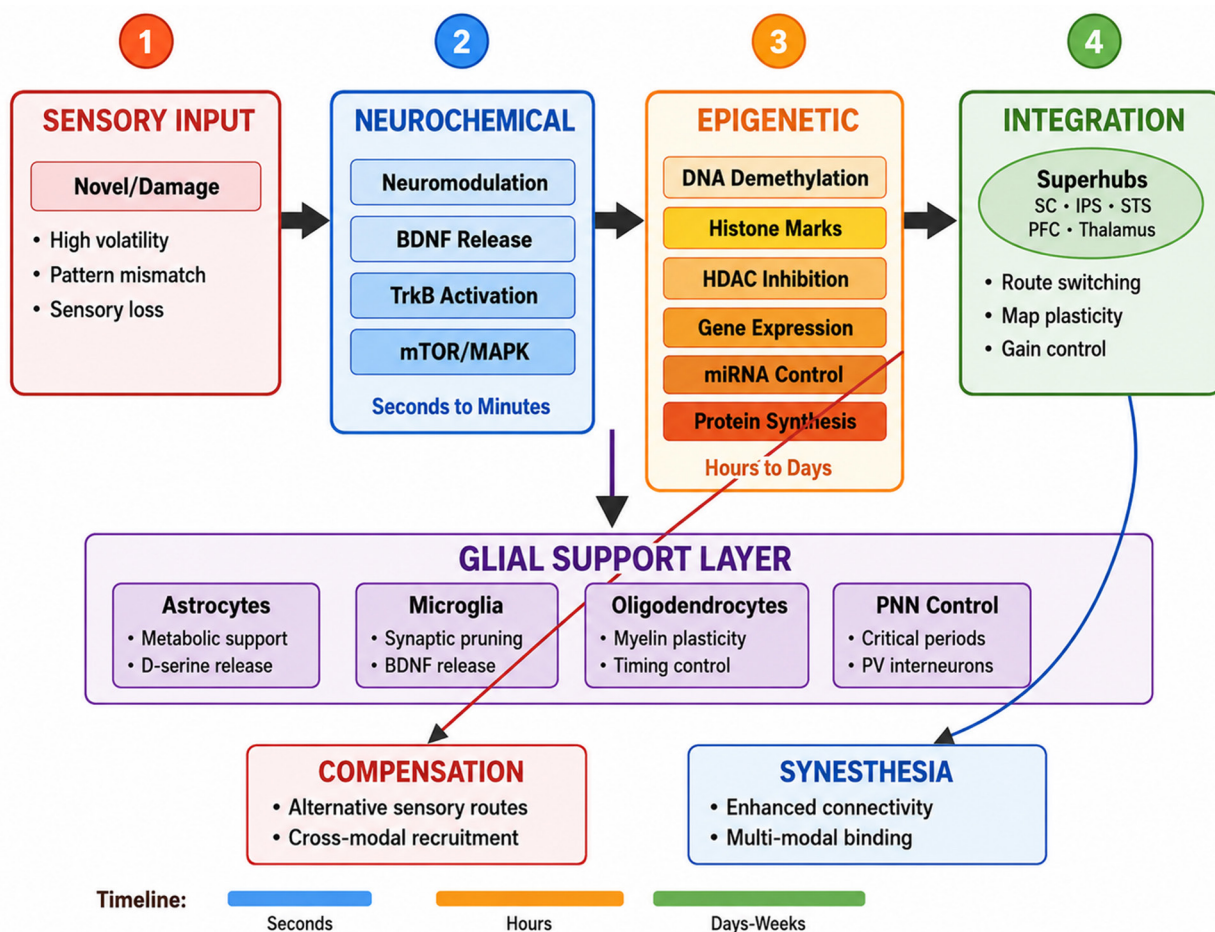


Fig. 3. Neuroplasticity control sequence

Glial cells are not a passive background of this process, but construct the “architecture of ensembles”. Astrocytes, forming the tripartite synapse, regulate the release of D-serine (co-agonist of NMDA receptors), potassium homeostasis, and GABA/glutamate balance, thereby opening or closing “plastic windows” in local microcircuits; through the astrocyte-neuron lactate shuttle they provide energetic support for mTOR-dependent protein synthesis during LTP consolidation. Simultaneously, microglia mediate synaptic “pruning” through complement-dependent mechanisms (C1q/C3), TNF α -induced synaptic scaling-up, and BDNF release, removing weak contacts and leaving highly efficient nodes in the new network configuration [122-142]. Oligodendrocytes and oligodendrocyte precursors provide activity-dependent myelin plasticity, finely adjusting conduction speed along the pathways connecting supernodes. These changes coordinate phase synchronization (alpha-beta for top-down gating, gamma for sensory “bottom-up”) and close the loop between rapid switching and “careful” structural tuning of networks [29, 122-142]. A critical regulator of the opening/closing of sensitive periods are perineuronal nets around PV-interneurons and the transfer of the homeoprotein Otx2; their remodeling, in particular under the influence of chondroitinase or activity changes, reduces inhibitory tone and opens a window for map reassignment in the adult brain [122-142].

The epigenetic circuit provides “rapid reprogramming” of gene expression required for transitions between sensory maps and makes these transitions manageable within a time range from hours to days without mandatory large-scale anatomical reconstruction. Activity-dependent demethylation and hydroxymethylation of promoters and enhancers (TET-dependent processes) combined with a decrease of DNMT-mediated repression increases accessibility of key LTP/LTD loci (e.g., *Arc*, *Egr1*, *Npas4*, *Bdnf*), chloride transporter KCC2, and NMDA receptor subunits, shifting the GluN2A/GluN2B balance and thereby retuning the temporal windows of STDP. In parallel, histone mark changes occur: increase of H3K27ac and H3K4me3 on active enhancers/promoters and weakening of repressive H3K9me2/H3K27me3, implemented through HAT (CBP/p300) activity and selective HDAC inhibition (especially HDAC2), known as the molecular lever for “reopening” plasticity in the mature cortex. Chromatin remodeling complexes (BAF/SWI-SNF) and topological proteins (CTCF and cohesin) reorganize the 3D genome architecture, bringing activity-dependent enhancer “clusters” together with promoters of plasticity genes; eRNA transcription at these enhancers serves as a marker and, likely, a modulator of stable activation. Non-coding RNAs, including miR-132 (enhancement of spine growth via p250GAP), miR-134 (restriction of excessive spinogenesis), and other activity-dependent microRNAs and long non-coding RNAs, finely tune local translation in dendrites and determine the degree and direction of reorganization. An additional level of regulation is provided by phosphorylation of eIF2 α and 4E-BP (elements of the ISR/mTOR pathway), which switches the network between a “scanning” mode (rapid switching without consolidation) and a “stabilizing” mode (scaled local protein synthesis and stabilization of new routes) [122-146].

In summary, the neurochemical key molecular inducer provides supernodes with a state of high plastic readiness and controls the instantaneous gain of sensory channels, whereas the epigenetic “rapid reprogramming” establishes molecular memory of the network state, allowing reversible and, when required, long-lasting transitions between maps [122-146]. Through the increased expression of KCC2 and correction of the chloride gradient, the inhibitory tone of PV-interneurons shifts, windows of gamma synchronization narrow or widen. Concurrently, the reorganization of NMDA receptor composition and AMPA-contact density redefines STDP rules in the nodes of IPS and STS; simultaneously BDNF/NT-3/IGF-1-driven cascades and glial mechanisms form and “clean up” ensembles, aligning the local microcircuit with the global policy of prefrontal control [122-152]. This dual-circuit organization explains how the brain can either compensate for the loss of a modality (forced inclusion of alternative routes) or manifest phenomena of “excessive integration” such as synesthesia. In the former scenario, the network undergoes an epigenetically sanctioned threshold shift and long-term stabilization of new maps. In the latter — the lowering of binding threshold and increase of precision gain allows temporary or stable co-activation of additional sensory representations without external necessity, forming adaptive, dynamic, rather than static synesthesia [122-157]. For this reason, neurochemical and epigenetic mechanisms are not merely auxiliary details of the larger scheme, but constitute its central control layer, which makes both the speed of switching and the stability of new sensory maps in the supernodes of neuroplasticity possible.

Conceptual integration

The consolidated empirical data facilitate the formulation of a unified conceptual framework that integrates sensory compensation and synesthesia within a universal mechanism of neuroplasticity [122-157]. The foundational premise of this framework is the recognition that the brain does not function as a set of isolated modules assigned to specific sensory or cognitive domains, but as an open dynamic system in which sensory maps can be reorganized depending on experiential conditions. Accordingly, sensory deprivation, as well as intensive cognitive or sensorimotor learning, trigger a unified mechanism of activation of switching nodes— the superior colliculus, intraparietal sulcus, superior temporal sulcus, and prefrontal projections. These structures function not only as integrators of multisensory streams but also as central regulators of neuroplastic reorganization: they determine which inputs should be strengthened, which routes should be redirected, and which networks synchronized with new contexts of behavioral demands. This conceptual model is illustrated in **Fig. 4**.

Activation of supernodes triggers a cascade of neurochemical changes centered around BDNF, NT-3, and IGF-1, alongside their interaction with local neuromodulatory systems. This cascade functions as a key molecular inducer creating a biochemical landscape of heightened plasticity: dendritic morphogenesis is enhanced, synaptic transmission is tuned toward long-term potentiation, and glial cells reorganize local microcircuits, preserving only those connections

that ensure optimal integration. In parallel, rapid epigenetic reprogramming unfolds, providing long-term stabilization of reorganized networks. Demethylation of plasticity-related gene promoters, histone modifications, and microRNAs activation establish a molecular memory of plastic states, thereby enabling the network to repeatedly enter highly flexible states or consolidate new configurations into a stable architecture [122-160].

This multilevel process drives the formal reorganization of sensory maps. During sensory deprivation, supernodes redirect information flow, ensuring that lost modalities are replaced by preserved channels. This mechanism facilitates not only deficit compensation but also, under specific conditions, functional advantages, including enhanced spatial memory in blind individuals or superior visuo-motor integration accuracy in deaf individuals. In synesthesia, the same mechanisms operate in a state of enhanced integration, where sensory maps are enriched with additional associative routes rather than being reorganized out of necessity, thereby enhancing cognitive associativity, creativity, and memory. In both scenarios, the mechanism remains convergent—activation of supernodes, initiation of the neurotrophin-epigenetic cascade, reorganization of sensory maps, and the formation of new cross-sensory associations [122-160]. The ultimate consequence of this architecture is cognitive enhancement. This process involves a systemic expansion of cognitive capacities achieved via the integration and optimization of sensory inputs

within the brain's dynamic network, rather than localized alterations in a single sensory modality. Hence, these findings indicate that compensation and synesthesia—traditionally regarded as separate phenomena (the former forced, the latter excessive)—represent variations of a universal principle embedded in the fundamental organization of the nervous system. Such conceptual integration not only unites disparate empirical observations but also proposes a novel paradigm for contemporary neuroscience.

Practical implications

The proposed conceptual model has a wide range of applied implications, encompassing medicine, education, and cognitive technologies. In medicine, it provides novel neurorehabilitation strategies for patients with sensory deficits. Given that sensory compensation is based on the supernode activation and reassignment of sensory maps, targeted stimulation of these structures may enhance functional recovery. This can be implemented via non-invasive neuromodulation technologies such as transcranial magnetic stimulation (TMS) or transcranial direct current stimulation (tDCS), aimed at occipito-parietal and prefrontal nodes. Pharmacological modulation of neurotrophic systems, particularly involving BDNF and IGF-1, may potentiate plasticity and increase the effectiveness of rehabilitation programs. In education, the proposed model suggests integrating synesthetic approaches as tools for enhancing cognitive efficiency. Since synesthesia demonstrates the potential of cross-modal associations to improve memory

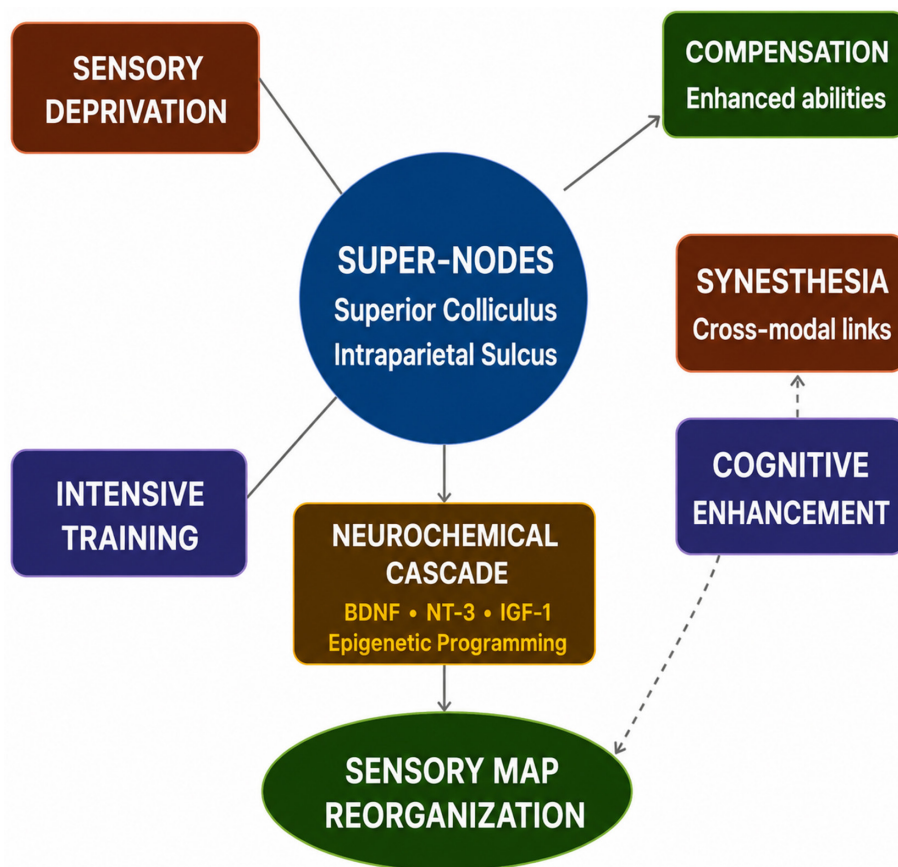


Fig. 4. Universal neuroplasticity mechanism

and associative thinking, employing multisensory educational methods can accelerate information assimilation and facilitate creativity processing. The combination of visual, auditory, and tactile stimuli in learning environments serves as a natural tool for strengthening associative networks, forming deeper and more durable knowledge. Within cognitive enhancement, controlled synesthesia induction represents a promising next-generation technology. Creating conditions for temporary or stable formation of additional cross-sensory associations may ensure expansion of cognitive capabilities beyond compensation of deficits — toward enhancement of memory, attention, creative thinking, and the integration of complex information structures. Thus, the practical implications of this model extend beyond therapeutic applications, providing a foundation for future transdisciplinary advances in medicine, education, and cognitive technologies.

Discussion

Current evidence suggests that sensory compensation and synesthesia represent convergent manifestations of a unified neuroplastic mechanism [29, 122-160]. However, this interpretation remains a theoretical-hypothetical model that requires further experimental validation. Neuroimaging and neurophysiological studies indicate that the reorganization of sensory maps occurs through a limited set of switching supernodes including the superior colliculus, intraparietal sulcus, superior temporal sulcus, prefrontal projections, supporting the concept of the brain as a dynamic and adaptive network [76-161]. In this context, Harvey (2013) posits that synesthesia serves as a model for understanding the principles of sensory system integration, by revealing mechanisms of cross-modal interaction that normally remain latent. Synesthetic experience is not an accidental anomaly, but rather the result of stable neural connections between high-level associative cortical areas, which provide flexible and multi-layered intertwining of sensory modalities [161]. Sensory compensation following deprivation demonstrates that primary sensory areas are capable of processing atypical signals, whereas synesthesia is an excessive variant of the same architecture, when cross-sensory routes are activated without external necessity. Parallel to these findings, Lee & Whitt (2015) demonstrate that cross-modal plasticity is realized through different mechanisms of synaptic reorganization: in deprived cortical areas homeostatic strengthening of intracortical connections occurs, while in preserved sensory areas — selective strengthening of thalamo-cortical pathways [162]. The authors emphasize that these processes may occur not only during critical developmental periods but also in the adult brain, offering significant opportunities for neurorehabilitation [162].

Neurochemical and epigenetic mechanisms, including neurotrophins BDNF, NT-3, IGF-1, glial cells, histone modifications, microRNAs) ensure rapid adaptation with long-term stabilization of new sensory maps [146, 163, 164]. These mechanisms may explain both the rapid activation of compensatory networks during temporary deprivation and the stable formation of cross-sensory associations in congenital or acquired loss of sensory functions. From an applied perspective, this

framework establishes a baseline for neurorehabilitation protocols and cognitive enhancement through supernode stimulation or integration of multisensory methods in learning. At the same time, questions remain open regarding switching parameters, the universality of supernode mechanisms, and the feasibility of inducing controlled synesthesia without disrupting sensory integration.

Conclusion

Sensory compensation and synesthesia may represent manifestations of shared mechanisms of brain neuroplasticity. Supernodes of plasticity including the superior colliculus, intraparietal sulcus, superior temporal sulcus, and prefrontal projections—play a central role in these processes, ensuring the reassignment of sensory maps and cross-modal integration. The scientific novelty of this work lies in the shift from interpreting these phenomena as exceptional to understanding them as a universal code of nervous system adaptation.

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Conflict of Interest

The authors declare that they have no conflicts of interest related to the research, authorship, or publication of this manuscript.

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