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ABHRAKA BHASMA AND THE NEURONAL EPIGENOME: A HYPOTHETICAL FRAMEWORK LINKING NANOSTRUCTURE, MECHANOTRANSDUCTION, AND LONG-TERM POTENTIATION

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ABSTRACT

Background: Abhraka Bhasma is a traditionally processed mineral preparation of Rasashastra with described Rasayana applications and associations with Majja Dhatu and cognitive functions. Contemporary physicochemical investigations have demonstrated that pharmaceutical processing can produce substantial changes in the mineral's elemental composition, crystallinity, particle characteristics, and morphology. These findings raise the possibility that the biological effects of Abhraka Bhasma may involve mechanisms beyond conventional chemical interactions. Mechanobiology provides a potential framework for investigating how the physicochemical characteristics of mineral-derived materials may interact with cellular mechanosensing, cytoskeletal signaling, nuclear regulation, and neuronal plasticity. **Aim and Objectives:** The aim of this review is to develop a hypothesis-driven framework linking the physicochemical characteristics of Abhraka Bhasma with mechanotransduction, epigenetic regulation, and neuronal synaptic plasticity, particularly long-term potentiation (LTP). The objectives are to review the Ayurvedic and pharmaceutical basis of Abhraka Bhasma; evaluate its contemporary physicochemical and nanoscale characteristics; examine evidence relating extracellular mechanics and mechanotransduction to neuronal function; explore the relationship between mechanical signaling, chromatin regulation, and neuronal

plasticity; and identify experimentally testable pathways connecting Abhraka-derived material properties with neuronal mechanotransduction and LTP. **Materials And Methods:** A structured narrative literature review was conducted using databases including PubMed, Scopus, Web of Science, Google Scholar, ScienceDirect, AYUSH Research Portal, and DHARA Online, along with relevant classical Ayurvedic literature. Literature concerning Abhraka Bhasma and Rasashastra, mineral nanomaterials, mechanobiology, mechanotransduction, neuroepigenetics, mechanical memory, neuronal plasticity, and LTP was identified and critically synthesized. Evidence from these domains was integrated to develop a conceptual Abhraka–neuronal epigenome framework. No primary experimental investigation was performed. **Observation And Results:** The reviewed literature indicates that Abhraka Bhasma is a complex processed mineral material whose physicochemical characteristics can vary according to pharmaceutical preparation and analytical methodology. Independent evidence from mechanobiology demonstrates that extracellular mechanical conditions can influence neuronal development, synaptic organization, and network activity through mechanosensing systems involving integrin-associated structures, mechanically activated ion channels, cytoskeletal signaling, and YAP/TAZ-mediated mechanotransduction. Mechanical signaling can also influence nuclear organization, chromatin regulation, and transcriptional programs, while mechanical memory has been demonstrated predominantly in non-neuronal cellular systems. In neuronal plasticity, activity-dependent calcium signaling, gene transcription, and structural synaptic modifications contribute to persistent changes associated with LTP. Integration of these findings provides a hypothetical mechanistic framework in which the physicochemical characteristics of appropriately prepared Abhraka Bhasma could potentially interact with neuronal or glial cell–material interfaces and influence mechanotransductive and epigenetic pathways relevant to synaptic plasticity. However, direct evidence demonstrating CNS delivery, neuronal mechanotransduction, epigenetic modification, or LTP induction by Abhraka Bhasma is currently lacking. **Conclusion:** The available evidence supports consideration of Abhraka Bhasma as a potential subject for interdisciplinary investigation at the interface of Rasashastra, materials science, mechanobiology, and neuroepigenetics. The proposed Abhraka–neuronal epigenome framework provides a testable hypothesis rather than an established mechanism, linking mineral-derived physicochemical characteristics with possible mechanotransduction, chromatin regulation, and neuronal plasticity. Experimental studies addressing material characterization, biological availability, cell–material interactions, mechanosensitive signaling, epigenetic changes, neuronal network activity, and LTP are required to evaluate

this proposed relationship.

KEYWORDS: Abhraka Bhasma; Rasashastra; Mechanobiology; Mechanotransduction; Neuroepigenetics; Mechanical memory; Synaptic plasticity; Long-term potentiation; YAP/TAZ; Mineral nanomaterials.

INTRODUCTION

Ayurveda conceptualizes the human body as a dynamic system involving the coordinated functioning of Dhatus, Doṣhas, and Srotas, with Majja Dhatu traditionally associated with important structural and functional attributes. The concept of Rasayana emphasizes preservation of tissue function, vitality, longevity, and cognitive faculties, providing a conceptual basis for exploring its effects through contemporary biological approaches.[1] Among mineral Rasayana preparations, Abhraka Bhasma, a traditionally processed mica-based formulation subjected to Shodhana and Maraṇa, has been attributed with rejuvenative and Dhatu supportive properties in Rasashastra. Contemporary characterization indicates that processing can alter the mineral's physicochemical properties, producing complex elemental composition, nanocrystalline features, and preparation-dependent morphological characteristics.[2] However, the molecular basis of its traditionally attributed neurological and cognitive effects remains inadequately understood.

Mechanobiology provides a potentially novel framework for investigating this knowledge gap. The brain is a mechanically active biological system in which mechanical forces and the physical properties of the extracellular environment can influence neuronal signalling, network organization, and plasticity.[3,4] Mechanical cues are detected through systems including integrin-associated adhesion complexes and mechanosensitive ion channels such as Piezo1/Piezo2.[5,6] while cytoskeletal signaling involving RhoA/ROCK can regulate nuclear mechanotransducers such as YAP/TAZ.[7] Experimental studies further demonstrate that substrate and matrix stiffness can influence neuronal maturation, synapse formation, network organization, and activity.[4,8] Mechanical signals can also interact with chromatin organization and epigenetic regulation, suggesting a potential pathway through which physical cellular environments may produce persistent changes in cellular phenotype[9.10] The concept of mechanical or “mechano-memory”, demonstrated particularly in stem-cell systems, further suggests that previous mechanical exposure can influence subsequent cellular behavior through pathways including YAP/TAZ, although its direct role in neuronal memory remains to be established.[11]

Long-term potentiation (LTP) is a major experimental model of activity-dependent synaptic strengthening underlying learning and memory, involving coordinated changes in neuronal excitability, calcium-dependent signaling, gene expression, and synaptic structure[12,13] These processes provide a conceptual intersection between mechanotransduction, epigenetic regulation, and neuronal plasticity. On this basis, the available evidence supports consideration of a hypothesis-driven Abhraka–neuronal epigenome framework in which the nanoscale morphology, mineral architecture, and surface characteristics of appropriately prepared Abhraka Bhasma may, under suitable biological conditions, interact with neuronal or glial cell material interfaces and influence mechanosensing pathways involving integrins, Piezo channels, cytoskeletal signaling, and YAP/TAZ. These signals could potentially affect chromatin regulation and the expression of plasticity associated genes such as BDNF, Arc, and c-fos, thereby modifying the cellular environment supporting synaptic plasticity and LTP. However, these mechanistic links remain hypothetical, and direct evidence that Abhraka Bhasma activates neuronal mechanotransduction or induces LTP is currently lacking.

Accordingly, this review integrates evidence from Ayurvedic pharmaceutics and Abhraka characterization, mechanobiology, neuroepigenetics, biomaterials science, and synaptic plasticity to develop and critically evaluate this proposed framework. Major unresolved questions include the physicochemical stability and bioavailability of Abhraka-derived material, systemic and CNS biodistribution, interaction with neuronal or glial extracellular interfaces, activation of mechanosensitive pathways, and subsequent epigenetic and functional effects. Rather than assuming a direct Abhraka-induced LTP mechanism, the proposed model identifies a sequence of experimentally testable relationships between mineral nanostructure → cell–material interaction → mechanotransduction → epigenetic regulation → activity-dependent neuronal plasticity → LTP. This hypothesis-generating framework therefore provides an interdisciplinary basis for investigating whether the biological effects traditionally attributed to Abhraka Bhasma can be scientifically explored through the emerging concepts of nano bio interfaces, mechanotransduction, epigenetic regulation, and cellular mechanical memory.

AIM

To critically evaluate and integrate Ayurvedic, materials-science, mechanobiological, and neuroepigenetic evidence to develop a testable hypothesis linking the physicochemical properties of Abhraka Bhasma with cellular mechanotransduction, epigenetic regulation, and

neuronal synaptic plasticity, particularly long-term potentiation (LTP).

OBJECTIVES

1. To examine the Ayurvedic rationale for Abhraka Bhasma as a Rasayana associated with Majja Dhatu and cognitive functions, and to critically evaluate its traditional pharmaceutical processing and contemporary physicochemical characterization.
2. To synthesize current evidence on the nanoscale morphology, mineral composition, surface characteristics, and other relevant physicochemical properties of Abhraka Bhasma that may influence its interaction with biological systems.
3. To critically review the evidence linking extracellular matrix mechanics, cellular mechanotransduction, cytoskeletal signaling, and nuclear mechanotransducers such as YAP/TAZ with neuronal development, synaptic organization, and plasticity.
4. To examine the emerging relationship between mechanotransduction, chromatin regulation, epigenetic mechanisms, and persistent cellular responses, including the concept of mechanical or mechano memory, in relation to neuronal plasticity.
5. To develop a conceptual and testable Abhraka neuronal epigenome framework in which the physicochemical characteristics of Abhraka Bhasma may potentially influence cellular mechanosensing, epigenetic regulation, and activity dependent synaptic plasticity, including LTP.
6. To identify the major experimental gaps and propose testable approaches for evaluating the proposed Abhraka mechanotransduction epigenetic axis, including studies of bioavailability, CNS biodistribution, cell material interactions, mechanosensitive signaling, epigenetic changes, and functional effects on neuronal networks.

MATERIALS AND METHODS

A structured literature search was conducted using PubMed, Scopus, Web of Science, Google Scholar, ScienceDirect, AYUSH Research Portal, and DHARA Online for publications from 1980 to 2025, along with relevant classical Ayurvedic texts. The search focused on five areas: Abhraka Bhasma and Rasashastra, mechanobiology, neuroepigenetics, biomaterials/nanostructures, and neuronal plasticity/LTP. Relevant peer reviewed studies and Ayurvedic literature available in English, Hindi, or Sanskrit with translations were included. Information regarding study characteristics, major findings, and their relevance to the proposed hypothesis was extracted and synthesized narratively. The available evidence was used to develop a hypothetical Abhraka neuronal epigenome framework linking the

physicochemical properties of Abhraka Bhasma with possible mechanotransduction, epigenetic regulation, and synaptic plasticity. No primary experiments were performed; potential experimental approaches for future validation were also identified.

Review of literature

1. Abhraka Bhasma: Ayurvedic Basis and Pharmaceutical Transformation

Abhraka Bhasma is an important mineral preparation of Rasashastra, traditionally subjected to a series of pharmaceutical processes such as Shodhana, Dhanyabhraka, Bhavana/Mardana, Maraṇa, and, in some preparations, Amṛutikaraṇa. Classical Rasashastra literature describes different varieties and pharmaceutical methods of Abhraka and provides variations in the media, number of Puta, and pharmaceutical procedures used for its preparation. Triphala Kwatha is frequently described as a Shodhana medium, while Kanji is commonly used during the Dhanyabhraka process. Different numbers and types of Puta are described depending on the method and intended therapeutic application.[14,15]

The pharmaceutical processing of Abhraka is considered important for transforming the raw mineral into a therapeutically suitable Bhasma. During Dhanyabhraka preparation, the purified Abhraka is processed to separate physical impurities and facilitate particle refinement. Marana involves repeated Bhavana, preparation of Chakrikas, Sharava Samputikarana, and controlled heating through Puta. Classical Bhasma examination includes characteristics such as Nishchandratva, Rekhapurnatva, Varitaratva, Unnama, Apunarbhavatva, Niruttha, and other parameters indicating appropriate processing and fineness.[2,14]

From a contemporary materials science perspective, these repeated processes of trituration, levigation, drying, and high-temperature treatment can substantially modify the physicochemical characteristics of the original mica mineral. Modern analytical investigations have demonstrated that Abhraka Bhasma is a complex mineral derived material containing elements such as silicon, aluminum, magnesium, iron, potassium, and other constituents. Structural and morphological investigations using techniques such as X-ray diffraction, FTIR, Raman spectroscopy, SEM, TEM, EDX, BET, DLS, and thermal analysis have demonstrated changes in crystallinity, morphology, particle characteristics, and surface properties following processing.[1]

Importantly, the physicochemical characteristics of Abhraka Bhasma are preparation dependent. Particle dimensions, crystalline phases, elemental composition, morphology, and

surface characteristics may vary according to the source of Abhraka, pharmaceutical method, Bhavana media, number of Puta, and analytical technique used. Therefore, Abhraka Bhasma should not be regarded simply as a source of a single mineral element. Rather, it represents a complex processed mineral material whose biological interactions may depend on its overall physicochemical and nanoscale characteristics.[1,15]

Preliminary biological investigations have explored antioxidant activity, cytotoxicity, and potential neuroprotective effects of Abhraka Bhasma. Studies in neuronal cell-line models have investigated its potential neuroprotective effects under specific experimental conditions, while experimental studies have also reported antioxidant and DNA-protective activities. However, these investigations have not established a mechanism involving neuronal mechanotransduction, epigenetic regulation, synaptic plasticity, or long-term potentiation. Thus, although preliminary biological evidence provides a basis for further investigation, the relationship between the physicochemical characteristics of Abhraka Bhasma and neuronal plasticity remains largely unexplored.[16,17]

2. Mechanobiology and Mechanotransduction in the Nervous System

The nervous system is traditionally viewed primarily as an electrically and chemically regulated system; however, contemporary neuroscience recognizes that mechanical forces and the physical properties of the cellular microenvironment can also influence neural function. The brain possesses distinct mechanical properties, and neurons, glial cells, and neural precursor cells continuously interact with their surrounding extracellular environment.[3,18]

Mechanotransduction refers to the process through which cells detect mechanical stimuli and convert them into intracellular biochemical and electrical signals. Mechanical information can be detected through cell–matrix adhesion complexes, integrins, the actin cytoskeleton, mechanically activated ion channels, and associated signaling molecules. Among the best-characterized mechanosensitive channels are Piezo1 and Piezo2, which can convert mechanical deformation into ionic currents.[5.6]

Evidence from neural stem-cell models demonstrates that mechanical sensing can influence neural cellular behaviour. Piezo1-mediated mechanosensing, for example, has been associated with changes in lineage determination in human neural stem cells. This provides evidence that mechanical stimuli can influence biological processes within neural cellular

systems, although such findings should not be directly extrapolated to mature neurons without appropriate experimental evidence.[19] Mechanical signals can also be transmitted through the actomyosin cytoskeleton and associated pathways such as RhoA/ROCK signalling. These pathways can regulate the activity and localization of the nuclear mechanotransducers YAP and TAZ. YAP/TAZ function as important regulators through which extracellular mechanical conditions can influence cellular transcriptional programs.[7]

The relevance of mechanical properties extends to neuronal maturation and synaptic organization. Experimental studies have demonstrated that variation in extracellular matrix stiffness can influence neuronal differentiation, neurite development, synapse formation, neuronal connectivity, and network activity. Changes in substrate stiffness have also been associated with alterations in synaptic density and electrophysiological characteristics of neuronal networks.[4,20]

These findings suggest that the physical microenvironment is not merely a passive structural component of neural tissue. Instead, mechanical information can potentially influence cellular organization, intracellular signalling, nuclear responses, and ultimately neuronal function. This provides an important conceptual basis for investigating whether the physicochemical properties of mineral derived nanoscale materials could interact with cellular mechanosensing systems.

3. Neuroepigenetics, Synaptic Plasticity and Long-Term Potentiation

Neuronal plasticity involves persistent changes in the structure and function of neurons in response to experience and activity. These changes are regulated by coordinated alterations in intracellular signalling, gene expression, synaptic structure, and neuronal connectivity. Epigenetic mechanisms provide an important molecular interface through which neuronal activity can produce relatively persistent changes in gene expression without altering the underlying DNA sequence.[21,22]

Major epigenetic mechanisms relevant to neuronal plasticity include DNA methylation, histone modifications, chromatin remodelling, and non-coding RNA-mediated regulation. These mechanisms can influence the accessibility of genes involved in neuronal development, synaptic function, learning, and memory.[23]

Activity-dependent regulation of genes such as brain-derived neurotrophic factor (BDNF)

represents an important example of the interaction between neuronal activity and epigenetic regulation. Changes in DNA methylation and chromatin organization can regulate the expression of plasticity-related genes, thereby contributing to long-lasting cellular adaptations associated with learning and memory.[24] Long-term potentiation (LTP) is one of the principal experimental models used to investigate the cellular and molecular basis of learning and memory. LTP involves a sustained increase in synaptic transmission following appropriate patterns of neuronal activity. Its induction and maintenance depend upon coordinated processes involving glutamatergic signalling, NMDA receptor-mediated calcium entry, intracellular kinase pathways, changes in gene transcription, structural modification of synapses, and altered neuronal excitability.[12,25]

The connection between epigenetic regulation and LTP is particularly important because long-lasting synaptic changes require sustained alterations in gene expression. Activity-dependent signalling can therefore influence transcriptional and chromatin states that support the persistence of synaptic modifications.[22]

Mechanobiology provides an additional potential layer of regulation. Mechanical signals transmitted through integrins, the cytoskeleton, nuclear structures, and mechanotransduction pathways may influence chromatin organization and epigenetic regulation. Thus, extracellular physical conditions could theoretically influence the transcriptional state of neurons and modify their capacity for activity-dependent plasticity.[3,26]

However, it is important to distinguish this established mechanistic background from the proposed Abhraka hypothesis. There is currently no direct evidence demonstrating that Abhraka Bhasma alters neuronal epigenetic regulation or directly induces LTP. LTP should therefore be considered a potential functional endpoint for future experimental investigation rather than an established effect of Abhraka Bhasma.

4. Mechanical Memory and the Proposed Abhraka–Neuronal Epigenome Axis

An emerging concept in mechanobiology is mechanical memory, or “mechano-memory,” referring to the ability of cells to retain information about previous mechanical environments and subsequently display altered responses even after the original mechanical stimulus has been removed. This phenomenon suggests that mechanical exposure can produce persistent changes in cellular state.[11]

Experimental evidence for mechanical memory has been particularly demonstrated in non-neuronal systems, including mesenchymal stem cells. Previous exposure to a stiff or soft extracellular environment can influence subsequent cellular behaviour through persistent mechanotransductive signaling involving YAP/TAZ and associated transcriptional programs. These findings indicate that mechanical signals can potentially produce cellular states that persist beyond the initial stimulus.[11] Recent work in mechanobiology further indicates that mechanical forces can influence chromatin organization and epigenetic regulation. Mechanical information can be transmitted from the extracellular environment through cell adhesion structures and the cytoskeleton to the nucleus, where changes in nuclear architecture and chromatin organization may alter gene accessibility and transcription.[10,26]

The concept of mechanical memory is therefore particularly interesting when considered alongside neuronal plasticity. Long-lasting neuronal adaptations associated with learning and memory also require persistent molecular and structural changes. However, neuronal mechano-memory has not yet been established as a direct mechanism of memory formation. The strongest evidence for mechanical memory currently comes from non-neuronal cellular systems. Its application to neuronal plasticity should consequently be regarded as a hypothesis requiring experimental validation.[11,12] Based on these observations, a novel conceptual connection can be proposed between the physicochemical characteristics of Abhraka Bhasma and neuronal mechanobiology. The proposed sequence is: Abhraka physicochemical characteristics → cell–material interaction → mechanotransduction → potential epigenetic regulation → activity-dependent neuronal plasticity → LTP.

In this proposed framework, the nanoscale morphology, layered mineral architecture, surface characteristics, and other physicochemical properties of appropriately prepared Abhraka Bhasma could potentially influence cellular interactions under suitable biological conditions. If Abhraka derived material were demonstrated to reach relevant biological compartments and interact directly with neuronal or glial extracellular interfaces, it could potentially influence mechanosensing mechanisms involving integrin-associated adhesion complexes, cytoskeletal organization, and mechanosensitive ion channels.

These mechanical signals could subsequently influence intracellular pathways such as RhoA/ROCK and YAP/TAZ, with possible downstream effects on nuclear organization and chromatin regulation. Changes in transcriptional programs involving neuronal plasticity-related genes could theoretically modify the cellular environment supporting synaptic

strengthening.

At the same time, chemical or ionic constituents released from Abhraka-derived material may potentially influence cellular signalling or neuronal activity. However, the precise dissolution, bioavailability, cellular uptake, and ionic behaviour of a particular Abhraka Bhasma preparation must be experimentally established before assigning such mechanisms.

The proposed Abhraka neuronal epigenome axis therefore represents a hypothesis generating model rather than an established biological pathway. Several critical questions remain unanswered, including whether orally administered Abhraka derived material reaches the central nervous system, in what physicochemical form it exists following digestion and systemic exposure, whether it can reach neuronal or glial extracellular interfaces, and whether it can influence mechanosensitive signalling or chromatin regulation.

Future studies would need to establish these intermediate steps systematically. Initial investigations could include detailed physicochemical characterization, gastrointestinal and serum stability studies, biodistribution analysis, cellular uptake studies, neuronal and glial cell–material interaction studies, assessment of mechanosensitive signalling pathways, analysis of chromatin and epigenetic changes, and finally functional evaluation using neuronal network activity and LTP models.

Thus, the existing literature supports several individual components of the proposed framework Abhraka Bhasma as a processed mineral nanomaterial, cellular mechanotransduction, mechanical regulation of neural cells, epigenetic regulation of neuronal plasticity, and LTP but the direct connection between these components and Abhraka Bhasma has not yet been experimentally demonstrated. The proposed framework consequently provides a testable interdisciplinary hypothesis connecting Rasashastra, mineral nanostructures, mechanobiology, neuroepigenetics, and synaptic plasticity.

DISCUSSION

The present review proposes a novel Abhraka mechanotransduction epigenetic framework in which the physicochemical characteristics of Abhraka Bhasma may potentially influence neuronal plasticity through mechanical and epigenetic pathways. Abhraka Bhasma undergoes extensive pharmaceutical processing involving Shodhana, Dhanyabhraka, Bhavana, Maraṇa and, in some preparations, Amṛutikaraṇa, resulting in substantial changes in its particle

characteristics, morphology, crystallinity, surface properties and elemental composition. These preparation-dependent physicochemical characteristics provide a rational basis for investigating Abhraka as a complex mineral-derived material rather than merely a source of individual mineral elements. Contemporary mechanobiology demonstrates that physical properties of the cellular microenvironment can influence neuronal development, synaptic organization and network activity through integrin-associated adhesion complexes, cytoskeletal signalling and mechanosensitive ion channels, while downstream pathways involving RhoA/ROCK and YAP/TAZ can connect mechanical stimuli with nuclear and transcriptional responses. Mechanical signals may further influence chromatin organization and epigenetic regulation, creating a potential interface between extracellular mechanical cues and persistent cellular changes. This is particularly relevant to neuronal plasticity because long-term potentiation (LTP), a major cellular model of learning and memory, requires coordinated calcium-dependent signalling, transcriptional regulation, structural synaptic modification and sustained changes in neuronal gene expression. On this basis, the present framework hypothesizes that, if Abhraka derived material reaches an appropriate biological compartment and interacts with neuronal or glial extracellular interfaces, its nanoscale morphology and surface characteristics could potentially influence cellular mechanosensing, cytoskeletal organization and downstream pathways such as YAP/TAZ, which may subsequently affect chromatin regulation and expression of plasticity-associated genes. The possible contribution of ionic constituents, particularly potassium, to neuronal excitability and activity dependent signalling is also of interest; however, the dissolution kinetics, bioavailability and biological effects of these constituents from Abhraka Bhasma remain to be established and should not be equated with experimentally induced depolarization. Similarly, although Piezo channels represent important mechanosensing systems and mechanical memory has been demonstrated in cellular models, their direct involvement in Abhraka-mediated neuronal plasticity and the existence of neuronal “mechano-memory” remain unproven. An important counterargument is the reported neurotoxicity of some engineered silica nanoparticles, which highlights that nanoscale silica-containing materials cannot automatically be considered beneficial; however, engineered silica nanoparticles and traditionally processed Abhraka Bhasma are physicochemically distinct materials, and their biological responses may differ according to composition, crystallinity, surface chemistry, morphology, dose and route of administration. Available Abhraka specific preclinical safety findings provide preliminary support for further investigation but do not establish neurological safety or efficacy. A major translational limitation is therefore the absence of

evidence demonstrating that orally administered Abhraka Bhasma or its relevant dissolution products reach the brain, particularly the hippocampal extracellular environment, in concentrations sufficient to influence mechanotransduction. Furthermore, the proposed pathway should not be interpreted as a direct molecular equivalent of Ayurvedic concepts such as Rasayana, Majja Dhatu or Srotas; rather, these concepts provide a traditional rationale from which experimentally testable biological questions can be generated. The central significance of the proposed hypothesis consequently lies in connecting otherwise independent fields Rasashastra pharmaceutics, mineral nanostructure, mechanobiology, neuroepigenetics and synaptic plasticity while clearly distinguishing established evidence from theoretical extrapolation. Experimental validation should therefore proceed sequentially through standardized physicochemical characterization and dissolution studies, assessment of gastrointestinal stability and biodistribution, neuronal and glial cell material interaction studies, evaluation of mechanotransduction markers such as YAP/TAZ and candidate mechanosensitive channels, analysis of epigenetic changes in plasticity related genes, and finally electrophysiological assessment of synaptic function and LTP. Comparative studies with amorphous and crystalline silica and differently prepared Abhraka Bhasma would further determine whether any observed biological effects are specific to the processed mineral preparation. Thus, the current evidence supports the plausibility of individual components of the Abhraka–mechanotransduction–epigenetic–plasticity pathway but does not yet establish the complete causal relationship, making the Abhraka Epigenome a hypothesis generating framework that warrants systematic experimental investigation rather than a demonstrated mechanism of action.

CONCLUSION

Abhraka Bhasma represents a complex, processed mineral preparation whose physicochemical characteristics provide a potential basis for exploring novel biological mechanisms. The proposed Abhraka mechanotransduction epigenetic axis offers a hypothesis driven framework linking mineral-derived nanostructure with cellular mechanosensing, epigenetic regulation, and neuronal plasticity, particularly LTP. Although these relationships remain experimentally unproven, the framework identifies important and testable mechanistic pathways and may provide a novel interdisciplinary approach for understanding the biological actions of Abhraka Bhasma and its traditional Rasāyana applications.

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