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INVITED REVIEW

The Hammock Hypothesis: An Integrative Conceptual Framework for Facial Aging

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ABSTRACT

Background: Facial aging is a multilayered and regionally heterogeneous process involving coordinated changes in the facial skeleton, fat compartments, retaining structures, fascial continuity, mimetic activity, and skin. Existing anatomical models have advanced understanding of age-related facial change, but may not fully explain how interactions across tissue layers and neighboring regions become expressed as recognizable surface phenotypes. This narrative review examines current concepts of facial aging and proposes the Hammock Hypothesis as an integrative structure–function framework.

Methods: Relevant literature was synthesized thematically across six domains: skeletal remodeling; superficial and deep fat-compartment change; retaining ligaments and septal support; superficial musculoaponeurotic system, fascial planes, and soft-tissue continuity; skin envelope and dermal aging; and dynamic influences. These concepts were organized into four interrelated dimensions of facial support behavior.

Results: The Hammock Hypothesis interprets visible facial aging as progressive disequilibrium within a mechanically coupled multilayer support system. The four proposed dimensions are: support base and foundational projection; anchoring and suspensory restraint; load transmission and dynamic redistribution; and surface deformation and visible expression. This framework relates whole-face support behavior to regional manifestations including tear trough prominence, midfacial flattening, nasolabial fold accentuation, jowl formation, marionette change, and temporal hollowing.

Conclusion: The Hammock Hypothesis is not intended to replace existing anatomical models, but to provide an integrative conceptual framework for clinical interpretation. It should be regarded as a heuristic and hypothesis-generating model requiring validation through imaging, biomechanical analysis, and phenotype-based clinical studies.

1 | Introduction

Facial aging is a complex, multilayered process involving progressive changes in the skin, soft tissues, retaining structures, fat compartments, the superficial musculoaponeurotic system (SMAS), and the underlying skeleton [1–32]. Advances in facial anatomy and esthetic medicine have substantially improved understanding of how these changes contribute to recognizable age-related facial features [6, 10–12, 15, 26, 33]. However, existing models do not always fully explain how aging becomes visibly expressed through interactions across tissue layers,

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structural interfaces, and neighboring facial regions [7, 10–12, 15, 17, 18, 27, 34].

Clinically, visible signs of aging often reflect coupled change rather than failure of a single structure in isolation [1–15, 17, 19, 23–27, 31–39]. Tear trough deformity, for example, may reflect not only local volume loss, but also alteration of the lid-cheek transition, reduced deep support, and tethering [7, 10–12, 34, 36, 37, 40–43]. Likewise, prominence of the nasolabial fold, jowl, or marionette region may not be adequately explained by descent or volumetric deficiency alone [1–15, 19, 35–38]. These observations suggest that facial aging may be understood as progressive disequilibrium within an interdependent support system rather than as the sum of isolated tissue changes.

This article reviews current concepts of facial aging and proposes the Hammock Hypothesis as an integrative conceptual framework for interpreting visible facial aging as the surface expression of progressive disequilibrium within a mechanically coupled multilayer support system, organized along four interrelated dimensions: support base and foundational projection, anchoring and suspensory restraint, load transmission and dynamic redistribution, and surface deformation and visible expression. The hypothesis is not intended to replace existing anatomical models, but to relate them within a broader structure–function framework that may support clinical interpretation.

2 | Conceptual Positioning of the Hammock Hypothesis in Relation to Existing Models

The Hammock Hypothesis is best understood in relation to established concepts of facial aging. Volume-loss and fat-compartment models emphasize compartment-specific changes in superficial and deep facial fat [1–4, 8, 9, 13, 31]. Skeletal remodeling and inside-out aging models emphasize loss of deep projection and altered soft-tissue drape [5, 6, 10–12, 15, 35, 36]. Retaining-ligament and fixed-mobile interface models clarify how tethering structures and septal boundaries shape transition zones, folds, and contour breaks [2, 3, 7, 14, 16, 17, 32, 38, 40–42]. SMAS, fascial-continuity, and biomechanical models further emphasize regional linkage, force transmission, and dynamic facial movement [17–20, 27, 28]. The conceptual gap addressed by the Hammock Hypothesis is not the absence of anatomical knowledge, but the need for a support-system vocabulary that links these established elements to visible clinical phenotypes. Rather than proposing a new anatomical structure, the hypothesis organizes existing anatomical and biomechanical concepts into four interdependent dimensions of support behavior.

For clarity, several terms require definition within this framework. “Support” refers to the capacity of skeletal, volumetric, fascial, and soft-tissue structures to maintain projection, contour, and positional stability under resting and dynamic conditions. “Restraint” refers to the limiting and organizing effect of retaining ligaments, septa, tethering structures, and fibrofascial boundaries on tissue displacement. “Anchoring efficiency” refers to the proposed ability of these restraining structures and adjacent tissue interfaces to preserve positional coherence and transmit tension across layers. “Support disequilibrium” is proposed to describe a mismatch among foundational projection, anchoring restraint, dynamic load distribution, and surface compliance that results in clinically visible contour change. At present, these terms should be regarded as conceptual descriptors rather than validated diagnostic or biomechanical constructs.

A distinct framework is therefore justified because clinicians frequently need to interpret how multiple known mechanisms combine to produce a specific visible phenotype. The relationship between the Hammock Hypothesis and prior facial-aging models is summarized in Table 1.

3 | Methods

This article is a narrative review examining the anatomical and biomechanical basis of facial aging in relation to the proposed Hammock Hypothesis. Relevant publications were identified through targeted PubMed searches, supplemented by manual screening of reference lists to capture foundational and clinically influential studies. Emphasis was placed on six literature domains considered most relevant to development of the proposed framework: skeletal remodeling; superficial and deep fat-compartment change; retaining ligaments and septal support; SMAS, fascial planes, and soft-tissue continuity; skin envelope and dermal aging; and dynamic influences on facial aging. Articles were selected on the basis of historical importance, conceptual relevance, anatomical contribution, and clinical interpretability. The literature was synthesized thematically to construct an integrated conceptual framework rather than to provide exhaustive evidence mapping. These thematic findings were then organized into four interrelated dimensions of facial support behavior.

4 | Current Concepts in Facial Aging

4.1 | Skeletal Remodeling

At the deepest level, the facial skeleton provides the foundational framework on which the facial support system depends [5, 10–12, 15, 18, 27]. Bony architecture shapes facial contour and projection and influences the mechanical behavior of the overlying soft tissues [5, 10–12, 15, 18, 27, 34]. With aging, skeletal remodeling reduces projection, alters support, and changes the drape of the soft tissues [1, 5, 6, 10–12, 15, 18, 27, 34, 35]. Enlargement of the orbital aperture, maxillary retrusion, changes in the pyriform region, and alteration of mandibular contour may collectively contribute to hollowing, retrusion, and regional contour instability [1, 5, 6, 10–12, 18, 34–36]. By diminishing the deep structural base on which both superficial and deep tissues rely, loss of bony projection may amplify the visible effects of soft-tissue attenuation [5, 10–12, 27, 35]. Skeletal aging, therefore, contributes not only to contour loss but also to progressive weakening of foundational support [1, 5, 6, 10–12, 15, 35, 36].

4.2 | Superficial and Deep Fat Compartment Changes

Facial fat aging occurs in a compartment-specific manner rather than as uniform global volume loss [1–4, 6, 8–13, 17, 18, 31]. Superficial and deep compartments may show different patterns

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TABLE 1 | Conceptual positioning of the hammock hypothesis in relation to existing facial-aging models.

Existing model or concept	Main concept	Limitations when used alone	Contribution of the Hammock hypothesis
Volume-loss and fat-compartment models	Facial aging is partly explained by compartment-specific changes in superficial and deep facial fat, resulting in hollowing, altered fullness, and contour imbalance.	These models explain important volumetric and compartmental changes, but may not fully describe how altered volume interacts with anchoring structures, dynamic loading, and surface compliance.	Reframes fat-compartment change as one contributor to support-base alteration and surface deformation within a broader multilayer support system.
Skeletal remodeling and inside-out aging models	Age-related bony remodeling changes the deep structural foundation of the face and alters soft-tissue drape, projection, and regional support.	These models clarify deep foundational change, but may underemphasize how soft-tissue restraint, fascial continuity, motion, and skin quality modulate visible expression.	Incorporates skeletal remodeling as the deepest support-base component while linking it to anchoring restraint, load redistribution, and visible surface change.
Retaining ligament and fixed-mobile interface models	Retaining ligaments, septa, and tethering structures define boundaries between relatively fixed and mobile tissues and influence folds, hollows, and contour breaks.	These models explain transition-zone behavior well, but are often regionally focused and may not fully integrate deep projection, dynamic force transmission, and dermal surface response.	Interprets retaining structures as part of an anchoring-and-suspensory dimension that interacts with foundational support, motion-related loading, and surface deformation.
SMAS and fascial-continuity models	The SMAS and related fascial planes provide structural continuity, distribute tension, and link adjacent facial regions.	These models emphasize fascial architecture and surgical anatomy, but may not provide a whole-face vocabulary connecting fascial tension to skeletal base, ligament restraint, dynamic motion, and skin expression.	Places SMAS-fascial continuity within the load-transmission and dynamic-redistribution dimension of a broader support-system framework.
Facial biomechanics and dynamic-aging models	Facial aging is influenced by tissue mechanics, repetitive movement, regional strain, and force redistribution over time.	These models provide important mechanistic insight, but may be difficult to translate into a clinically simple framework that also includes static anatomy and visible phenotypes.	Links dynamic force redistribution with static support elements and visible regional aging phenotypes in a clinically interpretable model.
Skin aging, photoaging, and dermal-quality models	Intrinsic and extrinsic skin aging reduce dermal resilience, elasticity, hydration, and resistance to wrinkling and laxity.	These models explain surface vulnerability, but may not fully account for how deeper structural imbalance determines where and how skin changes become clinically apparent.	Defines the skin and dermis as the surface-expression layer through which deeper support disequilibrium becomes visible.
Multilayer and anatomical-layering treatment models	Facial rejuvenation should consider multiple anatomical layers rather than treating isolated surface defects.	These models are clinically valuable but may remain treatment-oriented and may not explicitly define the mechanical relationship among support base, anchoring, load transmission, and surface expression.	Provides a structure-function vocabulary for interpreting multilayer aging before treatment selection, emphasizing diagnosis of support relationships rather than treatment layering alone.
Regional facial-aging models	Specific regions such as the periorbital area, midface, nasolabial fold, lower face, or temple are analyzed according to local anatomy and aging patterns.	Region-specific models may not fully explain how aging in one area affects adjacent facial units or whole-face balance.	Interprets regional phenotypes as local expressions of whole-face support disequilibrium and interregional coupling.

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of deflation, projection change, and altered relationships with adjacent tissues [2–4, 6, 8–13, 15, 17, 18, 27, 31, 35]. This compartment-based perspective has helped explain why facial aging often appears as a combination of hollowing and relative prominence rather than simple diffuse atrophy [1–4, 6, 8–13, 15, 27, 31, 37]. In some regions, compartmental decline leads to visible volume loss and reduced support, whereas in others an appearance of preservation or prominence may reflect contrast with neighboring areas of attenuation [1–4, 8–13, 31, 37]. Deep fat compartments are particularly important because they contribute to projection, contour foundation, and support of the overlying layers [2, 4, 8–13, 15, 27, 31]. Loss of this deeper support may accentuate hollowing, folding, and tethering, with consequences that extend beyond volume reduction alone [1–4, 6, 8–13, 15, 17, 27, 31, 35, 37].

4.3 | Retaining Ligaments and Septal Support

Retaining ligaments and related septal or tethering structures serve as key anchoring elements within the face, helping define boundaries between relatively fixed and relatively mobile tissues [2, 3, 6, 7, 9–19, 26, 32, 34, 38, 40–43]. Their role extends beyond simple fixation because they influence where age-related change becomes clinically apparent and how deformity is distributed across adjacent regions [2, 3, 7, 10–17, 19, 32, 34, 38, 40–43]. Contour alteration may be accentuated at fixed-mobile transitions, where anchored tissues meet more deformable structures [2, 3, 7, 9–19, 27, 32, 34, 38, 40–43]. Ligament-based models have therefore been particularly useful in explaining tear trough demarcation, nasolabial boundary behavior, jowl formation, and the topography of facial contour change [2, 3, 6, 7, 10–12, 14, 16, 17, 19, 27, 32, 34, 38, 40–43]. Even when retaining structures are not the primary site of age-related failure, they may still determine where imbalance becomes most evident [3, 7, 10–12, 14–17, 19, 32, 34, 37, 38, 40–43]. Emerging experimental evidence also suggests that age-related alteration of specific ligamentous structures, such as the zygomatic ligament, may further influence regional support behavior [7, 10, 12, 14, 16, 27, 32, 38].

4.4 | SMAS, Fascial Planes, and Soft-Tissue Continuity

The SMAS and related fascial-fibroseptal planes provide an important structural basis for continuity across the face [6, 7, 10–12, 15, 17–20, 26–28, 32, 34, 40]. Rather than functioning merely as a surgically defined layer, the SMAS helps organize relationships between superficial and deeper tissues and contributes to regional linkage and support coherence [6, 7, 9–12, 15, 17–20, 27, 28, 34]. Beneath the skin, the superficial fibroseptal-fascial layer also helps maintain continuity within the superficial soft tissues and distribute deformation across adjacent regions [2, 3, 7, 9–12, 15, 17, 19, 20, 28, 32]. These interconnected planes help explain why aging-related change may extend beyond a single focal site and why alteration in one facial region may influence contour behavior elsewhere [3, 7, 10–12, 15, 17, 19, 20, 27, 34, 40, 42]. With aging, reduced integrity of these fascial and fibroseptal structures may weaken support coherence and increase susceptibility to distortion, particularly where neighboring regions age at different rates [3, 7, 10–12, 17, 19, 20, 27, 28, 34].

4.5 | Skin Envelope and Dermal Aging

The skin and dermis form the visible outer boundary through which deeper structural changes become clinically apparent [6, 10–12, 15, 18, 21, 22, 26, 29–31, 43]. Age-related deterioration in collagen organization, elastin integrity, hydration, and dermal thickness reduces resilience and increases susceptibility to wrinkling, laxity, and redundant folding [6, 10–12, 15, 18, 21–24, 29–31]. Although cutaneous aging alone does not account for all age-related contour change, it strongly influences how deeper structural alteration is ultimately expressed on the surface [1, 6, 10–12, 15, 21–24, 29–31, 43].

4.6 | Dynamic Influences

Facial aging is shaped not only by structural alteration at rest, but also by the cumulative effects of repeated motion [6, 10–12, 15, 18, 26, 27, 30, 43]. Mimetic activity subjects facial tissues to recurrent mechanical loading, and over time, these forces act on structures whose resilience and support may already be altered by aging [10–12, 15, 18, 23–25, 27, 30, 39, 43]. Dynamic influences may therefore deepen expression-related lines, accentuate mechanically vulnerable regions, and contribute to the transition from temporary creasing to more persistent contour change [1, 6, 10–12, 15, 18, 29, 30, 35]. Because muscular activity, tissue restraint, and compliance vary across facial regions and among individuals, these effects are not distributed uniformly [11, 12, 18, 27]. Repetitive movement may thus amplify preexisting weakness in a patterned or asymmetrical manner, contributing to region-specific aging change [10–12, 18, 27].

Considered together, these concepts indicate that facial aging reflects multiple interacting anatomical and biomechanical processes rather than failure of a single tissue in isolation. However, current models do not always fully clarify how these processes interact across layers and regions as an interdependent support system. These interactions provide the rationale for the Hammock Hypothesis.

5 | The Hammock Hypothesis

The Hammock Hypothesis is proposed as an integrative conceptual framework for interpreting facial aging as progressive disequilibrium within a mechanically coupled, multilayer support system. In mechanical terms, a hammock is a suspended load-bearing structure whose form depends on the coordinated interaction of several elements: a support base and anchoring configuration that provide positional stability, suspensory components that maintain tension, transmission and redistribution of load across the suspended structure, and visible deformation of the central surface under stress. Its shape is therefore determined not by any single part in isolation, but by the balance among anchorage, tension, load, and material compliance. When one or more of these relationships is altered—through reduced support, weakened suspension, uneven loading, or diminished resistance within the suspended surface—the structure may sag, tilt, deform asymmetrically, or develop focal stress concentration (Figure 1). This mechanical logic provides a useful analogy for facial aging, in which the visible facial surface is likewise shaped by the coordinated

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behavior of multiple supporting elements acting across depth and region rather than by any one layer alone [23–27, 31, 39].

Although integrated models of midfacial aging and support-based concepts have previously appeared in more localized anatomical contexts—particularly in relation to skeletal–soft-tissue interaction, lower eyelid support, and ligament-based support of facial fat compartments—the present hypothesis applies the hammock concept in a broader and more explicitly integrative way [2–20, 26, 27, 31–38, 40–43]. Here, "hammock" does not refer to a single ligament, septum, or compartment-specific support structure. Rather, it denotes a whole-face conceptual model in which facial aging is interpreted as progressive imbalance within a multilayer support system.

From this perspective, visible contour change reflects not only age-related alteration of individual tissues, but also progressive disequilibrium among four interrelated dimensions of facial support behavior: support base and foundational projection, anchoring and suspensory restraint, load transmission and dynamic redistribution, and surface deformation and visible expression. These four dimensions are not proposed as anatomically independent compartments; rather, they represent interdependent analytical axes through which multilayer facial aging can be interpreted. Together, these four dimensions constitute the conceptual framework of the Hammock Hypothesis. The principal facial aging concepts reviewed above and their relevance to this framework are summarized in Table 2.

Several testable propositions follow from this framework. First, visible regional aging phenotypes should correlate more strongly with combined measures of foundational support, anchoring restraint, dynamic motion, and surface compliance than with any single anatomical variable alone. Second, transition-zone deformities, such as tear trough prominence or nasolabial fold accentuation, should show measurable relationships with both adjacent structural support and fixed-mobile boundary behavior. Third, dynamic imaging may reveal motion-related displacement patterns that are not fully apparent in static assessment. These propositions remain hypothetical and require validation.

6 | Facial Correlates of the Hammock Hypothesis

Building on the framework outlined above, this section relates the four dimensions of the Hammock Hypothesis to their principal facial correlates, which are summarized in Table 3.

6.1 | Support Base and Foundational Projection

At the deepest level, the facial support system depends on a stable structural base and adequate foundational projection [1, 5, 6, 10–12, 15, 18, 26, 27, 35]. These correlates correspond primarily to skeletal projection and deep volumetric buttressing, which together establish the foundational geometry on which more superficial support relies [2, 4–6, 8–13, 15, 31, 35, 36]. When age-related skeletal remodeling or loss of deep volumetric support weakens this base, the consequences extend beyond visible loss of projection alone [1, 2, 4–6, 8–13, 15, 26, 27, 31, 33, 35, 37, 43]. In the hammock analogy, weakening of the support base compromises balanced suspension and increases susceptibility to hollowing, contour instability, fold accentuation, and regional disharmony [4, 5, 8–13, 27, 31, 37, 43]. Foundational aging, therefore, reflects not only contour loss but also reduced efficiency of facial support.

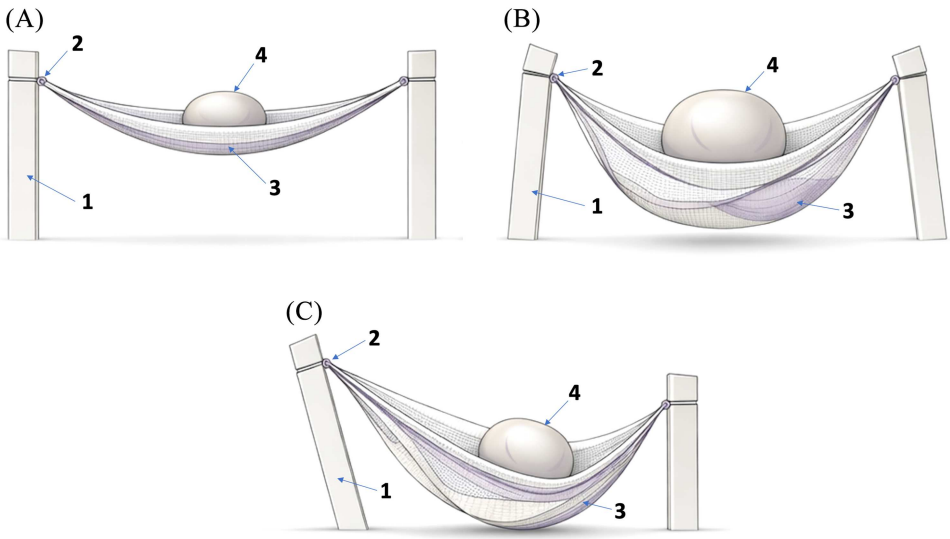


FIGURE 1 | Conceptual hammock model illustrating the four interdependent dimensions of the Hammock Hypothesis. (A) Balanced support, with stable anchoring and relatively even load distribution. (B) Progressive laxity/support loss, with reduced tension and increased sagging of the suspended surface. (C) Regional imbalance/asymmetric failure, with uneven anchoring, displacement, and asymmetric deformation. The figure illustrates how altered support relationships may produce visible deformation within a suspended multilayer system. Numbered labels indicate the four proposed dimensions of the framework: 1, support base and foundational projection; 2, anchoring and suspensory restraint; 3, load transmission and dynamic redistribution; and 4, surface deformation and visible expression. The figure is intended as a conceptual analogy rather than a direct biomechanical model.

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TABLE 2 | Current concepts in facial aging and their principal correspondences to the four dimensions of the Hammock hypothesis.

Literature domain	Principal anatomical emphasis	Core aging concept	Clinical significance	Correspondences to the Hammock hypothesis
Skeletal remodeling	Facial skeleton; orbital aperture; maxilla; pyriform region; mandible	Reduced projection; altered contour; weakened deep structural support	Hollowing; retrusion; prejowl deficiency; contour instability	Support base and foundational projection
Superficial and deep fat-compartment changes	Superficial and deep fat compartments; deep soft-tissue buttressing	Compartment-specific volume loss; altered contour; differential regional prominence	Nonuniform facial aging; coexistence of hollowing and relative fullness	Support base and foundational projection; surface deformation and visible expression
Retaining ligaments and septal support	Retaining ligaments; septa; tethering structures; fixed-mobile boundaries	Regulation of transition zones; concentration of visible imbalance	Tear trough demarcation; nasolabial boundary behavior; jowl formation; contour breaks	Anchoring and suspensory restraint
SMAS, fascial planes, and soft-tissue continuity	SMAS: superficial fibroepithelial-fascial layer; interfascial continuity	Distribution of tension, support, and deformation across regions	Regional linkage: influence of aging in one area on adjacent contour behavior	Load transmission and dynamic redistribution; anchoring and suspensory restraint
Skin envelope and dermal aging	Skin; dermis; collagen; elastin; hydration; dermal thickness	Reduced resilience; increased susceptibility to wrinkling, laxity, and redundant folding	Surface visibility of deeper structural imbalance	Surface deformation and visible expression
Dynamic influences	Mimetic musculature; motion; repetitive mechanical loading	Cumulative strain from repeated animation	Patterned, region-specific, and sometimes asymmetrical aging changes	Load transmission and dynamic redistribution; surface deformation and visible expression

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TABLE 3 | The four dimensions of the Hammock hypothesis, principal facial correlates, and potential measurable parameters.

Dimension of the Hammock hypothesis	Core definition	Principal facial correlate	Potential measurable parameters	Typical visible consequences
Support base and foundational projection	Deep structural support that establishes baseline facial geometry and projection	Facial skeleton; orbital rim; maxilla; pyriform region; mandible; deep fat compartments	CT or CBCT skeletal projection; 3D surface projection; regional convexity/concavity analysis; MRI-based deep fat volume; standardized facial-angle or contour measurements	Hollowing, loss of projection, retrusion, contour instability, fold susceptibility, and regional disharmony
Anchoring and suspensory restraint	Tissue restraint system that preserves positional coherence and limits displacement under load	Retaining ligaments; septa; tethering structures; fibrofascial interfaces; fixed-mobile boundaries	Ultrasound or MRI assessment of ligament-adjacent tissue; displacement at fixed-mobile interfaces; elastography-based stiffness; dynamic movement across tethered transition zones	Tear trough demarcation, nasolabial boundary accentuation, contour breaks, jowl concentration, visible transition-zone deformity
Load transmission and dynamic redistribution	Static and motion-related force propagation across a changing support system	SMAS; mimetic musculature; fascial planes; regional soft-tissue continuity	Dynamic 3D imaging; optical-flow or vector-displacement analysis; expression-related strain mapping; asymmetry of motion; finite-element simulation of load redistribution	Patterned deformation, asymmetry, mechanically vulnerable zones, progression from dynamic creasing to persistent contour change
Surface deformation and visible expression	Visible surface manifestation of deeper support imbalance and reduced tissue resilience	Skin; dermis; superficial soft-tissue envelope	3D wrinkle or fold-depth analysis; skin thickness; cutometry; elastography; surface texture analysis; photographic aging scales; patient-reported outcome measures	Wrinkling, laxity, redundant folding, contour interruption, asymmetry, clinically readable surface change

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6.2 | Anchoring and Suspensory Restraint

Positional coherence within the facial support system depends on retaining, tethering, and suspensory elements that limit deformation under load [2, 3, 7, 10–12, 14–19, 26, 32, 34, 38, 40–43]. In facial terms, these correlates are represented by retaining ligaments, septal and tethering structures, and the superficial fibroseptal-fascial support layer [2, 3, 7, 10–12, 14–17, 19, 20, 32, 34, 38, 40–42]. Together, they influence how support is maintained across adjacent regions and where transitions between relatively fixed and relatively mobile tissues become clinically important [3, 7, 10–12, 14–17, 19, 27, 32, 34, 38, 40–43]. In the hammock analogy, they correspond to the attachments and suspensory elements that preserve form under load [7, 12, 14–17, 19, 32, 34, 38, 40, 41]. As these relationships become less balanced with aging, anchored-mobile interfaces may become increasingly conspicuous, leading to accentuation of folds, hollows, and contour breaks [2, 3, 7, 10–12, 14, 16, 17, 19, 20, 27, 32, 34, 38, 40–43].

From a clinical perspective, this anchoring-and-suspensory dimension may help explain why treatment directed at ligamentous, septal, or sub-SMAS anchoring zones can produce clinically meaningful contour effects with relatively low filler volume [7, 16, 17, 27, 32, 44]. However, current evidence does not directly demonstrate that filler placement in these zones increases ligament tension, restores support behavior, or improves anchoring efficiency in a validated biomechanical sense. Therefore, within the Hammock Hypothesis, ligament-adjacent or sub-SMAS filler placement should be interpreted as a hypothesis-generating clinical implication rather than direct validation of the model.

6.3 | Load Transmission and Dynamic Redistribution

Unlike a static support structure, the face is repeatedly subjected to motion-related loading and force redistribution over time [6, 10–12, 15, 18, 26, 27, 43]. The SMAS, mimetic musculature, and related fascial planes constitute the principal structures through which force is generated, transmitted, and redistributed across the face [6, 10–12, 15, 17–20, 26–28, 34, 40]. Their significance lies not merely in moving tissue, but in determining how mechanical stress acts on a support system whose structural integrity may already be changing with age [16, 17, 19, 20, 27, 28, 38]. Recurrent animation may progressively expose weakness when tension is unevenly maintained or when support elements have become attenuated [10–12, 15–20, 27, 28, 34, 43]. In this way, facial aging may emerge as patterned regional deformation rather than as a uniform consequence of static tissue decline alone.

6.4 | Surface Deformation and Visible Expression

Changes visible at the facial surface are best understood as the outward expression of alteration within a deeper support system. In facial terms, this dimension is reflected in the way the skin and dermis register changes in deeper structures [6, 10–12, 15, 18, 21, 22, 29–31, 34, 43]. Although intrinsic cutaneous aging reduces resilience and increases susceptibility to wrinkling, laxity, and redundant folding, the Hammock Hypothesis emphasizes that visible surface change often reflects underlying disequilibrium in support, tension distribution, and interlayer coordination [1–24, 26, 27, 29–31, 35–38, 40, 41]. Surface hollowing, folds, asymmetry, and contour interruption may therefore be understood as clinically readable manifestations of deeper structural imbalance [4, 5, 7–13, 15–17, 19–22, 27–31, 34, 37, 38, 40–43].

7 | Regional Clinical Manifestations

A practical value of the Hammock Hypothesis lies in its ability to relate whole-face support disequilibrium to recognizable regional phenotypes. Within this framework, visible signs of aging are viewed as regional expressions of imbalance within a mechanically coupled facial support system rather than as isolated local defects. These interpretations should be regarded as hypothesis-generating rather than confirmatory. The principal regional manifestations are summarized in Table 4.

7.1 | Periorbital Region

The periorbital region is particularly prone to early and visible expression of support disequilibrium because it combines thin soft-tissue coverage, high mobility, and sharply defined fixed-mobile interfaces [6, 7, 10–12, 14, 16–18, 20, 30, 34, 37, 40–43]. Reduced skeletal contour and diminished deep support weaken the structural foundation of the lower eyelid and lid-cheek junction [1–13, 15, 17, 34–37, 40–43]. Ligamentous restraint and tethering then help determine where contour change becomes demarcated within this anatomically constrained region [3, 7, 10–12, 14, 16, 17, 32, 34, 37, 40–43]. Frequent movement repeatedly loads tissues that may already be less stably supported [18, 20, 30, 34, 40–43]. These changes become clinically evident as tear trough prominence, lid-cheek transition change, and exaggerated tissue contrast across a thin surface envelope [1–13, 15, 17, 18, 21, 22, 29–32, 34, 36, 37, 40–43]. The periorbital region, therefore, illustrates how even modest imbalance in support and restraint may become externally amplified [5, 7, 10–12, 14, 16, 17, 21, 22, 30, 31, 34, 37, 40–43].

7.2 | Midface

The midface occupies a central support-bearing position within the facial system and is therefore especially important to overall contour balance [2, 4–6, 8–13, 15, 17, 19, 20, 26–28, 31, 34, 35, 37, 42, 43]. Loss of deep support and reduced anterior projection weaken the foundational geometry on which midfacial contour depends [1, 2, 4–6, 8–13, 15, 17, 18, 31, 34–37, 42, 43]. Anchoring and suspensory relationships influence how this weakening is contained or expressed across adjacent regions, particularly at the lid-cheek junction and nasolabial interface [2–5, 7–17, 19, 20, 32, 34, 35, 37, 38, 40–43]. Because the midface serves as a major zone of projection and load sharing, altered force transmission may affect neighboring regions rather than remain confined locally [27, 32–34, 42, 43]. These changes become visible as flattening, reduced anterior fullness, and disruption of contour continuity [1–13, 15, 17, 19, 31, 34, 35, 37, 42, 43]. Midfacial aging thus shows how weakening of a central

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TABLE 4 | Regional clinical manifestations across the four dimensions of the Hammock hypothesis.

Region	Support base and foundational projection	Anchoring and suspensory restraint	Load transmission and dynamic redistribution	Surface deformation and visible expression	Typical manifestations
Periorbital region	Reduced deep support; orbital contour change; weakened lid-cheek foundation	Tear trough tethering; fixed-mobile demarcation; ligament-defined boundaries	High expressivity; frequent periorbital motion; repeated loading of thin tissues	Thin soft-tissue envelope; amplified contour contrast; highly sensitive surface readout	Tear trough prominence; lid-cheek transition change; exaggerated contour contrast
Midface	Loss of deep support; reduced anterior projection; weakened central support base	Transition support at adjacent interfaces; containment or spread of contour change across neighboring regions	Major zone of regional load sharing; altered force transmission to adjacent regions	Flattening; reduced anterior fullness; contour discontinuity	Midface flattening; reduced projection; secondary influence on the lower eyelid and nasolabial complex
Nasolabial region	Reduced adjacent projection; increased structural contrast across the interface	Fixed-mobile boundary effect; tethered support; concentrated imbalance at the transition zone	Repetitive movement across a constrained interface; reinforcement of preexisting imbalance	Localized indentation; surface contrast; fold accentuation	Nasolabial fold prominence; transition deformity; visible contour contrast
Lower face	Mandibular change; reduced deep structural support; weakened lower facial frame	Retained restraint at prejowl and jawline boundaries; focal contour breakdown	Force redistribution through the SMAS, superficial tissues, and facial movement	Boundary-based deformation; loss of contour continuity; juxtaposed fullness and hollowing	Jowl formation; prejowl hollowing; jawline blunting
Perioral and marionette regions	Reduced local projection; diminished structural resistance in the perioral complex	Altered restraint around the oral commissure and lower perioral zone	Recurrent loading from speech, expression, and oral function; motion-driven stress accumulation	Indentation; downturning; contour disharmony	Marionette prominence; oral commissure downturning; perioral indentation
Temple and lateral face	Loss of lateral volume and structural support; weakened facial frame	Reduced lateral support coherence; altered integration with adjacent units	Altered lateral load balance; modified mechanical context for neighboring regions	Temporal hollowing; altered lateral contour; altered apparent facial balance	Temporal hollowing; lateral contour loss; imbalance in overall facial framing

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support base may propagate through the broader facial support system and become visible beyond the most obviously affected site [4, 5, 8–17, 19, 20, 27, 28, 31–34, 37, 38, 40–43].

7.3 | Nasolabial Region

The nasolabial region is better understood as a transition deformity rather than as an isolated line [2–13, 15, 17, 19, 27, 31, 34, 35, 37]. Reduced projection in adjacent midfacial structures may increase structural contrast across the nasolabial interface [1–6, 8–13, 15, 17, 19, 31, 34–37, 43]. Fixed-mobile boundaries and tethered support relationships then help determine where this imbalance becomes visually concentrated [2–4, 7–14, 16, 17, 19, 32, 34, 37, 38, 41, 42]. Repetitive facial movement may further reinforce the effect by repeatedly loading tissues across a mechanically constrained transition zone [20, 27, 28]. The result becomes clinically apparent as increasing fold prominence, localized indentation, and contour contrast [1–6, 8–13, 15, 17, 19, 21, 22, 27, 29, 31, 34, 35, 37]. The nasolabial region, therefore, exemplifies how broader support disequilibrium may become most apparent at a structurally tethered interface rather than only at the site of greatest visible indentation [2–5, 7–17, 19, 32, 34, 35, 37, 38, 42].

7.4 | Lower Face

The lower face provides a clear example of how visible aging may arise from combined disturbance across multiple support dimensions rather than from tissue descent alone [2, 3, 6, 7, 9–17, 19, 20, 26–28, 31, 32, 38]. Mandibular change and loss of deeper structural support may weaken contour definition and reduce the foundational stability of the lower facial frame [1, 5, 6, 10–12, 15, 18, 27]. Retained points of restraint then help determine where contour breakdown becomes concentrated, particularly around the prejowl and jawline [2, 3, 5, 7, 10–13, 15–17, 19, 32, 38]. Redistribution of force through the SMAS, superficial tissues, and facial movement may further influence how soft-tissue fullness, displacement, and contour interruption appear across the region [7, 10–12, 17–20, 28, 38]. These processes become clinically evident as jowl formation, prejowl hollowing, and jawline blunting [1–3, 5–7, 9–13, 15–17, 19, 20, 27, 28, 31, 32, 38]. The lower face thus illustrates progressive loss of contour continuity in a region where support and restraint are unevenly distributed [7, 10–12, 16, 17, 19, 20, 31, 38].

7.5 | Perioral and Marionette Regions

The perioral and marionette regions are particularly influenced by recurrent movement, but their aging pattern also depends on reduced local support and containment [2–12, 15, 18, 20, 26, 27, 31, 36]. Age-related loss of projection and structural resistance may diminish stability within the perioral complex [1, 2, 4, 5, 9–12, 15, 18, 35]. Regional restraint then influences how adjacent tissues remain contained or become distorted around the oral commissure and lower perioral zone [3, 4, 7, 10–12, 15, 18, 27]. Frequent movement associated with speech, expression, and oral function imposes repeated stress on tissues that may already have reduced support and diminished resilience [6, 10–12, 15, 18, 21, 22, 27, 29]. These changes become visible as indentation, downturning, marionette prominence, and contour disharmony [1, 4–7, 9–12, 15, 18, 21, 22, 29]. This region, therefore, illustrates how repeated loading may convert latent structural vulnerability into persistent visible change [11, 12, 15, 18, 21, 22, 29].

7.6 | Temple and Lateral Face

The temple and lateral face contribute importantly to overall facial framing, lateral tension balance, and regional contour continuity [10–12, 15, 18–20, 26, 27, 33]. Loss of lateral volume and structural support may weaken the peripheral support frame that helps maintain whole-face balance [1, 2, 4, 8–13, 15, 31, 33]. Lateral support relationships influence how these regions remain integrated with adjacent facial units and how loss of coherence alters facial proportions [2–4, 7–13, 19, 20, 27, 34, 42]. Because these regions contribute to lateral contour integration, loss of temporal support may affect not only local volume perception but also the visual relationship among adjacent regions. Clinically, these changes may appear as temporal hollowing, altered lateral contour, and a change in apparent facial balance [1, 2, 4, 8–13, 15, 37, 43]. This region therefore emphasizes that facial support is not purely central or local, but distributed across peripheral as well as central facial relationships [3, 7, 10–12, 16, 17, 19, 20, 27, 33, 38].

8 | Strengths, Limitations, and Future Directions of the Hammock Hypothesis

The Hammock Hypothesis is proposed not as a replacement for existing anatomical models of facial aging, but as an interpretive framework through which they may be related within a mechanically coupled support system. Its principal strengths, limitations, and future directions are considered below.

8.1 | Conceptual Strengths of the Hypothesis

A major strength of the Hammock Hypothesis lies in its integrative capacity. It provides a common mechanical language through which skeletal support, fat compartments, retaining structures, SMAS-fascial continuity, skin behavior, and dynamic influences can be interpreted within a single conceptual system [2–22, 24, 26–32, 34, 38, 40–43]. In doing so, it helps unify observations that are often clinically related but anatomically discussed across separate literatures.

The framework is also valuable in its ability to relate whole-face support relationships to regional clinical expression [26, 27, 31, 33]. In many patients, visible deformities such as tear trough prominence, nasolabial fold accentuation, prejowl contour loss, or marionette change do not arise solely from isolated local failure, but reflect interactions among deep support, tethering, motion, and surface compliance [1–22, 29–31, 34–38, 40–43]. By conceptualizing these findings as regional expressions of broader support disequilibrium, the hypothesis helps explain why clinically similar surface findings may arise from different underlying structural patterns.

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The hypothesis may also serve as a useful clinical reasoning framework [26, 33]. The model encourages the clinician to ask not only what is visible, but why it has become visible at that location and in that form [10–13, 15, 26, 27, 33, 34, 37, 43]. This may improve diagnostic interpretation by shifting attention from isolated defects to support relationships, transition zones, vector imbalance, and regional coupling [6, 7, 10–20, 27, 32, 34, 37, 38, 40–43]. It may also help explain why direct correction at the point of maximal visibility may sometimes yield incomplete, unstable, or less natural results [7–13, 15, 17, 19, 26, 27, 31–34, 37, 38, 40–43]. By organizing facial aging into four interrelated support dimensions, the hypothesis also suggests testable predictions. In particular, it suggests that visible deformity may relate more closely to combined changes in support, restraint, motion, and surface expression than to any single anatomical variable considered in isolation [16, 17, 19–34, 37, 39, 42, 43].

8.2 | Limitations and Potential Criticisms

Despite these strengths, the Hammock Hypothesis has important limitations. Most fundamentally, it remains a conceptual analogy rather than a directly proven mechanistic model. The hammock metaphor is useful because it clarifies interdependence, load transfer, and visible deformation, but it also carries a risk of oversimplification if interpreted too literally. Facial aging is biologically more complex than any suspended structure, and the analogy should therefore not be understood as a one-to-one biomechanical equivalent [23–25, 39].

In addition, the four dimensions of the model, although analytically distinguishable, are not fully separable in vivo [14, 16, 17, 19–25, 27–29, 34, 38, 39, 42, 43]. Support base and foundational projection, anchoring and suspensory restraint, load transmission and dynamic redistribution, and surface deformation and visible expression overlap both anatomically and functionally, and in actual patients, these elements may be difficult to isolate, quantify, or rank in a reproducible way. At present, the framework is therefore better regarded as interpretive rather than quantitative [23–25, 39].

A related limitation is the potential for circular interpretation if visible surface findings are used both to infer support disequilibrium and to confirm the model. Future validation should therefore correlate clinical phenotypes with independent anatomical, imaging, or biomechanical measurements [14, 16, 17, 19–24, 27–34, 37–39, 42, 43]. This distinction is important if the hypothesis is to move beyond descriptive interpretation toward testable clinical and mechanistic utility.

The hypothesis may also be viewed as overlapping with existing integrative thinking in facial aging and rejuvenation [15, 26, 27, 31, 32]. This criticism is reasonable. The present model does not suggest that previous authors have ignored interdependence altogether; rather, its contribution is to formalize that interdependence more explicitly as a multilayer support-system concept and to provide a coherent mechanical vocabulary linking anatomical change to clinical phenotype. A further limitation is the absence of validation against standardized diagnostic or treatment algorithms [26, 32, 33]. The model may improve conceptual clarity without necessarily improving outcomes unless its predictions can be operationalized and tested.

The current framework also focuses primarily on structural and biomechanical relationships and does not fully incorporate extrinsic, biological, hormonal, molecular, or population-related modifiers of facial aging. Photoaging, endocrine transition, cellular senescence, inflammaging, and variation in facial morphology, soft-tissue distribution, dermal thickness, and skin phototype may influence the rate and regional pattern of support disequilibrium. These factors should not be regarded as separate from the Hammock framework, because they may modify foundational support, anchoring restraint, load transmission, and surface deformation in different ways. Future iterations of the model should therefore examine whether photoexposure history, sex and endocrine status, ethnicity or phototype, and molecular markers of tissue aging affect the relative contribution of each dimension to visible facial aging phenotypes [45–48].

8.3 | Future Directions

Future work should determine whether the Hammock Hypothesis can be translated into measurable, clinically meaningful constructs [23–25, 39]. Multimodal imaging may help correlate regional phenotype with combined changes in skeletal projection, deep and superficial tissue support, retaining structures, and skin behavior [5–17, 19–24, 27–32, 34, 37, 38, 40–43]. Three-dimensional photography, ultrasound, magnetic resonance imaging, and other layer-resolved imaging approaches may clarify whether visible contour change is better explained by integrated support imbalance than by isolated tissue variables [10–17, 19–22, 24, 27–30, 32–34, 37, 38, 40–43].

Future validation should define measurable parameters for each proposed dimension. For the support-base dimension, potential parameters include CT or cone-beam CT assessment of skeletal projection, three-dimensional surface projection, regional contour depth, and MRI-based deep fat volume. For anchoring and restraint, ultrasound, MRI, elastography, and dynamic imaging may help evaluate ligament-adjacent tissue behavior, fixed-mobile transition zones, tissue stiffness, and displacement under rest and animation. For load transmission, dynamic three-dimensional imaging, standardized video analysis, optical-flow methods, vector-displacement mapping, and expression-related strain analysis may be useful. For surface expression, three-dimensional wrinkle and fold-depth analysis, skin thickness measurement, cutometry, elastography, standardized photographic scales, and patient-reported outcome measures may help assess visible surface change and its relationship to deeper support imbalance.

Dynamic analysis warrants particular attention because the model explicitly incorporates load transmission and repeated motion [11, 18, 27, 28, 30]. Future studies could examine how facial expression, asymmetrical muscle use, and dynamic strain interact with regional support weakness over time, with longitudinal assessment of animated as well as static facial change likely to be especially informative [11, 18, 27, 30, 37].

The hypothesis may also be tested through phenotype-based clinical studies in which patients with similar visible deformities are

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stratified according to presumed dominant patterns of support imbalance and then treated according to different structural strategies [8, 9, 12, 13, 15, 26, 27, 31–33, 43]. Such an approach may help determine whether outcomes differ when intervention is aligned with the predicted pattern of support failure [12, 13, 15, 26, 27, 31–33, 43]. Future studies should also evaluate whether image-guided ligament-adjacent or sub-SMAS reinforcement can measurably alter local tissue density, SMAS tension, vector displacement, surface contour, and patient-reported outcomes, thereby testing whether targeted restoration of anchoring efficiency improves facial support behavior in vivo [7, 16, 17, 19, 20, 27, 32, 44].

In parallel, computational and biomechanical modeling may help refine the framework further. Finite-element and related simulation-based approaches could explore how changes in projection, restraint, tissue compliance, and repeated loading alter surface contour patterns, thereby providing a more formal biomechanical basis for the hypothesis [14, 16, 17, 19–22, 24, 27–31, 34, 42]. Ultimately, for the framework to become more clinically usable, it will require clearer diagnostic criteria, more reproducible terminology, and more practical ways to distinguish dominant support patterns across patients and facial regions [23–29, 31–33, 39].

9 | Conclusion

Facial aging is a multilayered and regionally heterogeneous process in which visible change emerges from coordinated alteration across bone, fat compartments, retaining structures, fascial continuity, muscle activity, and skin. The Hammock Hypothesis addresses this interpretive gap by framing facial aging as progressive disequilibrium within a mechanically coupled multilayer support system.

Its principal contribution lies not in proposing a new isolated anatomical component, but in providing an integrative structure–function framework through which visible aging can be interpreted in relation to alterations in support base and foundational projection, anchoring and suspensory restraint, load transmission and dynamic redistribution, and surface deformation and visible expression. The hypothesis should be regarded as a heuristic and hypothesis-generating framework rather than a fully validated mechanistic model. Its value will depend on whether its central propositions can be translated into measurable variables, correlated with regional aging patterns, and shown to improve diagnostic interpretation or treatment planning.

At present, terms such as support disequilibrium and anchoring efficiency should be regarded as conceptual descriptors rather than validated diagnostic endpoints. Future studies using three-dimensional imaging, ultrasound, MRI, dynamic motion analysis, elastography, finite-element modeling, and phenotype-based clinical assessment will be necessary to determine whether the proposed framework can be translated into measurable variables and clinically useful predictions.

Author Contributions

Chuan-Yuan Lin: conceptualization, writing – original draft, writing – review and editing, visualization, supervision. **Jui-Yu Lin:** conceptualization, writing – original draft, writing – review and editing, visualization, supervision. **Konstantin Frank:** writing – original draft, writing – review and editing. **Sachithevi Sivananthan:** writing – original draft, writing – review and editing. **Vanravi Vachatimanont:** writing – original draft, writing – review and editing. **Carmelo Crisafulli:** writing–original draft, writing – review and editing. **Fowzieh Rasras:** writing – original draft, writing – review and editing. **Razan Kadry:** writing – original draft, writing – review and editing. **Hala Alsadik:** writing – original draft, writing – review and editing. **Lehavit Ackerman:** writing – original draft, writing – review and editing. **Phuong Nguyen Ngoc Dong:** writing – original draft, writing – review and editing. **Lukas Prantl:** conceptualization, writing – original draft, writing – review and editing, supervision. All authors reviewed and approved the final manuscript for submission.

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Ethics Statement

Not applicable. This narrative review did not involve human participants, clinical intervention, or identifiable patient data.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing is not applicable to this article as no data sets were generated or analyzed during the current study.

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