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Mechanical Tension and Cutaneous Scarring: From Mechanotransduction to Fibrotic Persistence and Clinical Tension Management

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Abstract

Mechanical tension influences more than wound-edge approximation. It can also shape the biological course of scar formation. An important question is why the fibrotic effects of mechanical loading during early healing may persist beyond the initiating mechanical stimulus. External mechanical loading is sensed through integrin-focal adhesion kinase (FAK) signaling and the cytoskeleton, with RhoA/Rho-associated coiled-coil kinase (ROCK) and YAP/TAZ helping convert physical forces into profibrotic transcriptional responses. These signals also interact with TGF-β-related pathways and promote fibroblast activation, contraction, and extracellular matrix deposition. As matrix accumulates and tissue stiffness increases, the scar environment can itself provide sustained mechanical input and further reinforce mechanotransduction. In this way, an initiating external load may be converted into a self-reinforcing local fibrotic environment. Evidence supports this matrix-mediated reinforcement as one mechanism of fibrotic persistence. Fibroblast mechanical memory may provide an additional mechanism by maintaining features of an activated phenotype after prior mechanical exposure, although direct evidence in cutaneous scar tissue remains limited. These temporal effects have important clinical implications. Tension reduction should not focus only on closure tension at the time of surgery. Its effectiveness may also depend on when mechanical control begins, how long it is maintained, and whether the intensity of intervention matches the mechanical risk of the wound. This temporal mechanobiological framework supports a shift from single-step wound closure toward timely, sustained, and risk-matched control of the scar mechanical environment.

Categories: Plastic Surgery, Trauma, General Surgery
Keywords: cutaneous scarring, fibrotic persistence, matrix stiffness, mechanical tension, mechanotransduction

Introduction And Background

Cutaneous scarring is a common consequence of trauma and surgery. Some wounds develop widened scars, hypertrophic scars, keloids, or prolonged fibrosis that can affect appearance, function, and symptoms. Pathological scarring reflects an active fibrotic process involving persistent inflammation, fibroblast activation, and abnormal extracellular matrix remodeling [1-3]. Among the factors that shape this process, the mechanical environment is especially important because it can be directly modified in clinical practice.

Mechanical tension was traditionally viewed mainly as a surgical problem related to wound-edge approximation and closure stability. This view is now too limited. Mechanical loading also acts as a biological signal that influences cells and the extracellular matrix during scar formation. Several observations support this concept. Fetal skin can heal with minimal scarring in an environment characterized by low inflammation and low mechanical tension [4]. In adult skin, scars are often more prominent in anatomical regions exposed to greater or repeated mechanical loading [5,6]. Experimental studies have also shown a relationship between wound-closing tension and subsequent scar width [7]. These findings place mechanical tension upstream of several processes that shape the final scar phenotype. However, they also raise a more important temporal question. Mechanical loading during early healing can be substantial, while scar formation and maturation continue for months. The key issue is therefore not simply whether tension promotes scarring, but why the fibrotic consequences of mechanical loading can persist beyond the period in which the initiating load is strongest.

A temporal mechanobiological model provides one way to explain this persistence. External mechanical loading can first activate mechanotransduction through focal adhesions and the cytoskeleton. These signals alter mechanosensitive transcription and promote fibroblast activation, contraction, and extracellular matrix production [8,9]. The newly deposited and remodeled matrix can then become stiffer and transmit mechanical signals more efficiently. This process creates a local environment in which matrix stiffness and cellular contractility reinforce each other. In this way, an initially external mechanical stimulus may be converted into an increasingly self-reinforcing fibrotic microenvironment. Persistent cellular states after earlier mechanical exposure may further contribute to this transition from mechanical initiation to longer-lasting fibrosis.

Previous reviews have broadly addressed surgical wound tension, its biomechanical determinants, methods of assessment, and perioperative management [10]. These perspectives have established the importance of mechanical control in wound care. A scar-focused temporal framework can extend this view by linking early mechanical loading with later fibrotic persistence and with clinical decisions about when tension control should begin and how long it should be maintained. In this narrative review, we organize cutaneous scarring around three connected stages: mechanical initiation, matrix-mediated reinforcement, and fibrotic persistence. We use this framework to interpret the mechanical basis of scar formation, the mechanotransduction processes that connect force with fibrosis, and the clinical strategies used to modify wound tension. The central view is that tension management should not be treated as a single closure maneuver. Instead, scar prevention should be understood as control of the mechanical environment across time, with the timing, duration, and intensity of intervention matched to the mechanical risk of the wound.

A targeted narrative literature search was conducted in the Web of Science Core Collection for publications from 2015 through 2025. The search combined terms related to skin and cutaneous tissue with terms describing mechanical loading and mechanobiology, including "tension", "mechanical stress", "mechanical load", "mechanical force", "stretch", "strain", "mechanotransduction", and "mechanobiology", together with terms related to scarring and wound repair, including "scar", "wound healing", "wound repair", "hypertrophic scar", and "keloid". Records unrelated to biological tissue mechanics, including studies primarily concerning surface tension and nonbiological systems, were excluded by the search strategy. Literature was selected for this narrative review according to its relevance to cutaneous scar mechanobiology, mechanotransduction, fibrotic persistence, or clinical tension management. Seminal studies and additional relevant publications outside the structured search window were also incorporated through targeted searching and reference tracing when they directly informed the mechanistic or clinical framework discussed in this review.

Review

Mechanical initiation: how external loading shapes scar risk

Sources of Mechanical Loading in Healing Skin

Healing skin is exposed to several forms of mechanical loading. Resting skin tension is already present before injury, and this baseline tension differs across anatomical sites. After injury, tissue recoil pulls the wound edges apart. Wound-edge traction then appears when the surgeon approximates the skin and closes the defect. During early repair, wound contraction further changes the local force field. These forces do not remain fixed after closure. They change as edema increases or resolves, as collagen is deposited, and as the new matrix gains stiffness.

Postoperative movement adds another layer of mechanical load. Joint motion repeatedly stretches wounds located near mobile areas. Posture can also change the direction and magnitude of traction across the incision. A wound on the shoulder, chest, abdomen, or over a joint may experience repeated pulling even when the initial closure appears stable. Incision orientation also determines whether the wound lies along or across a dominant tension direction. Therefore, the wound mechanical environment is dynamic rather than static. Mechanical exposure continues after wound approximation and varies substantially in magnitude, direction, and duration.

Spatial and Anatomical Heterogeneity of Scar Mechanical Risk

Scar mechanical risk differs across the body because skin tension is spatially and directionally heterogeneous. The same incision length and closure method may produce different scar outcomes in different anatomical regions. This difference reflects local tissue thickness, elasticity, mobility, curvature, and resting tension. It also reflects the direction in which the incision is placed relative to local skin tension.

Langer lines, Kraissl lines, and relaxed skin tension lines provide practical ways to describe the directional behavior of skin. Langer lines indicate cleavage patterns of the skin and are often used to guide incision planning [11]. Kraissl lines are related to natural creases and muscle movement, which makes them useful in regions affected by facial expression or body motion [12]. Relaxed skin tension lines describe directions in which resting skin tension is relatively low and are widely used in dermatologic and plastic surgery [13]. Together, these concepts illustrate the direction-dependent mechanical behavior of skin.

This direction dependence has direct clinical meaning. Incisions placed along a lower-tension direction usually have less wound-edge traction and more stable approximation. Incisions that cross a dominant tension direction can experience stronger separation forces. Mobile regions create an additional risk because the wound is repeatedly loaded during daily activity. Wounds over joints, the anterior chest, the shoulder, and other high-motion areas often face both static tension and dynamic postoperative traction. These local conditions help explain why some wounds heal with narrow, flat scars, while others develop scar widening, elevation, or contracture. Scar mechanical risk is spatially heterogeneous.

Experimental and Clinical Evidence Linking Load to Scar Outcome

Evidence from molecular, animal, and clinical studies supports mechanical loading as a determinant of scar outcome. At the molecular level, mechanical loading can alter fibroblast behavior and scar-related gene expression. A recent study showed that mechanical stimulation changed HOX gene expression in fibroblasts, linking mechanical cues with differences among normal skin, hypertrophic scar, and keloid fibroblasts [14]. This finding is important because it shows that force does not only deform tissue. It can also change the biological program of scar-forming cells.

At the phenotypic level, wound tension is associated with measurable scar changes. Animal studies have shown a relationship between wound-closing tension and scar width [7]. This type of evidence connects mechanical loading with visible scar morphology. High tension can separate wound edges, widen scars, compress small vessels, reduce perfusion, and delay repair. These local changes can maintain inflammation and keep fibroblasts and myofibroblasts active. As collagen deposition increases and extracellular matrix remodeling becomes delayed, the scar may become wider, thicker, firmer, or more raised. A high-tension wound is therefore not only a wound that is difficult to close. It is a biological environment that promotes scar formation.

Interventional studies provide stronger support because they show that reducing mechanical load can improve scar outcomes. In a randomized controlled trial, postoperative paper tape reduced hypertrophic scar formation in surgical incisions that crossed Langer lines [15]. Mechanical stress shielding has also been shown to improve cutaneous scar formation in large-animal and early clinical studies [16]. These findings support the practical value of mechanical offloading. They also show that scar outcome can be modified by changing the wound's mechanical environment.

Mechanical loading may also have effects that extend beyond the period of direct force exposure. Experimental work has shown that mechanical force can prolong acute inflammation through T-cell-dependent pathways during scar formation [17]. This finding suggests that early or sustained loading can push the wound into a longer scar-promoting state. It also helps explain why short-term mechanical conditions during early healing may influence later scar phenotype.

Together, these findings support mechanical loading as an active and modifiable determinant of scar phenotype rather than merely a correlate of difficult wound closure. If mechanical loading acts as an upstream variable, the next question is how cells sense force and convert it into fibrotic biological signals.

Mechanotransduction: converting mechanical load into fibrotic activity

Mechanical load can shape scar formation because fibroblasts can read the physical state of the wound. These cells sense matrix tension and stiffness. They then convert these physical cues into intracellular signals. This process links external force with fibroblast activation, inflammatory signaling, and matrix production. In this way, mechanotransduction explains how wound tension becomes a fibrotic program.

Integrin-focal adhesion kinase (FAK): Mechanical Sensing at the Cell-Matrix Interface

Integrin-focal adhesion complexes provide one major interface through which fibroblasts sense extracellular mechanical conditions. The extracellular matrix is not only a scaffold around the cell. It also gives fibroblasts mechanical information. When matrix tension or stiffness increases, integrins cluster on the cell membrane. This clustering promotes focal adhesion assembly. Focal adhesions then connect the extracellular matrix with the actin cytoskeleton.

Focal adhesion kinase (FAK) is a key signaling molecule within focal adhesions. Its activation helps convert mechanical input into intracellular responses. Wong et al. demonstrated that FAK links mechanical force to cutaneous fibrosis through inflammatory signaling, and FAK inhibition reduced mechanically induced fibrosis in skin [8]. This places FAK at an important position between matrix mechanics and scar biology. Through this axis, extracellular force can promote fibroblast migration, survival, proliferation, inflammatory activation, and extracellular matrix remodeling. FAK therefore links the physical properties of the wound matrix with intracellular fibrotic signaling.

RhoA/Rho-Associated Coiled-Coil Kinase (ROCK): Converting External Load Into Intracellular Tension

Fibroblasts actively reorganize cytoskeletal tension in response to external mechanical loading. After integrins and focal adhesions sense the matrix, fibroblasts adjust their internal contractile system. This response depends strongly on the RhoA/Rho-associated coiled-coil kinase (ROCK) pathway.

FAK and integrin-related signaling can activate RhoA. Activated RhoA then stimulates Rho-associated protein kinase, or ROCK. ROCK promotes actomyosin contraction and stress fiber formation. These changes increase intracellular tension and strengthen the connection between focal adhesions and the actin cytoskeleton. In fibroblasts, cyclic mechanical strain activates RhoA and induces ROCK-dependent actin assembly. RhoA/ROCK-dependent actin contractility also supports mechanically induced extracellular matrix gene expression [18].

This step is central to the force-to-fibrosis chain. Extracellular loading becomes intracellular cytoskeletal tension. Stronger cytoskeletal tension then promotes focal adhesion maturation and prepares the cell for downstream nuclear signaling. As a result, fibroblasts do not only receive mechanical force. They actively amplify it inside the cell.

YAP/TAZ: Converting Mechanical State Into Sustained Transcription

Mechanical information reaches the nucleus through mechanosensitive transcriptional regulators. YAP and TAZ are central regulators in this process. When matrix stiffness increases, cytoskeletal tension also increases. This mechanical state promotes YAP/TAZ nuclear localization. Once YAP and TAZ enter the nucleus, they regulate genes related to cell proliferation, survival, matrix remodeling, and fibrosis.

Dupont et al. identified YAP/TAZ as key mediators of cellular responses to mechanical cues [9]. This mechanism is directly relevant to high-tension wounds. In these wounds, fibroblasts are exposed to external traction and increasing matrix stiffness at the same time. These conditions favor YAP/TAZ nuclear localization. Nuclear YAP/TAZ activity can then promote fibroblast activation and extracellular matrix-related gene expression.

YAP/TAZ therefore provide a mechanism by which a physical mechanical condition can be translated into a sustained transcriptional state. This transcriptional state supports matrix production and fibrotic remodeling.

TGF-β/Smad: Coupling Contraction With Matrix Reinforcement

TGF-β/Smad signaling is a major pro-fibrotic pathway in wound healing and scar formation. TGF-β promotes fibroblast activation and myofibroblast differentiation. Smad2 and Smad3 transmit TGF-β signals into the nucleus. These signals increase the expression of α-SMA, collagen, fibronectin, and other matrix-related molecules. Desmoulière et al. showed that TGF-β1 induces α-smooth muscle actin expression in fibroblasts and supports myofibroblast differentiation during wound healing and fibrocontractive disease [19].

Mechanical tension strengthens this pathway. A tense or stiff wound matrix can make fibroblasts more responsive to TGF-β signaling. At the same time, activated myofibroblasts generate contractile force. This force can further change the matrix around the cell. Wipff et al. showed that myofibroblast contraction can activate latent TGF-β1 from the extracellular matrix, and stressed tissues show stronger Smad2/3 signaling than relaxed tissues [20]. Mechanical stretch can also sustain the myofibroblast phenotype through latent TGF-β1 activation [21].

This creates a feedback relationship rather than a simple one-way pathway. Myofibroblast contraction promotes latent TGF-β activation. TGF-β/Smad signaling promotes further myofibroblast differentiation and matrix production. Collagen and fibronectin deposition then increase matrix stiffness. The stiffer matrix strengthens mechanosignaling and supports continued fibrotic activity.

MAPK and Signaling Crosstalk

MAPK signaling adds another layer to tension-related fibrotic activity. ERK and JNK can regulate fibroblast proliferation, inflammatory activity, extracellular matrix production, and connective tissue growth factor-related responses [22]. These effects are relevant because scar formation depends on both mechanical signals and biochemical signals.

Mechanical cues do not act through a single isolated pathway. They interact with growth factor signaling, inflammatory pathways, and matrix-regulating networks. Chiquet et al. showed that cyclic tensile strain and growth factors can regulate extracellular matrix genes through different contributions from Rho/ROCK and MAPK signaling [23]. This finding supports a network view of mechanotransduction.

In this network, focal adhesion signaling, cytoskeletal tension, YAP/TAZ activity, TGF-β/Smad signaling, and MAPK signaling influence each other. This crosstalk allows a physical force to become a coordinated cellular response. The final output is not pathway activation alone. The final output is fibroblast survival, proliferation, inflammatory signaling, myofibroblast differentiation, and extracellular matrix production.

Integrated Mechanotransduction Cascade

The mechanotransduction cascade can be understood as a continuous force-to-fibrosis process. Extracellular loading first changes the mechanical state of the wound matrix. Fibroblasts sense this state through integrin-focal adhesion complexes. FAK then connects matrix mechanics with intracellular inflammatory and fibrotic signaling. RhoA/ROCK converts this sensed force into cytoskeletal tension. YAP/TAZ then translate the mechanical state into sustained transcription. TGF-β/Smad signaling couples myofibroblast

contraction with matrix reinforcement. MAPK signaling connects this process with growth factor and inflammatory networks.

This cascade produces a clear fibrotic output. Fibroblasts become activated. Myofibroblasts generate contractile force. These cells produce collagen, fibronectin, and other extracellular matrix components. Matrix accumulation increases tissue stiffness. Increased stiffness then provides stronger mechanical cues to fibroblasts. This forms the central scar-specific loop: tension promotes fibrosis, fibrosis increases stiffness, and stiffness strengthens further mechanotransduction.

This model connects molecular mechanisms with clinical scar prevention. By reducing mechanical input, clinical tension management may interrupt this self-reinforcing loop and shift wound healing toward a lower-fibrosis environment. Mechanotransduction explains how force initiates fibrosis, but it does not by itself explain why fibrotic activity may persist after the initiating mechanical load has changed. The proposed transition from mechanical initiation to fibrotic persistence is summarized in Figure *1*.

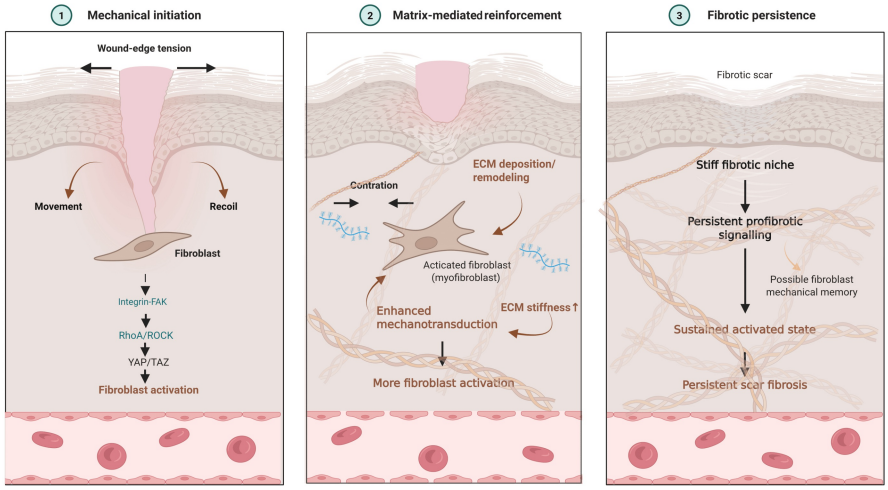


FIGURE 1: From mechanical initiation to fibrotic persistence in cutaneous scarring

Early wound-edge tension, movement, and tissue recoil initiate fibroblast mechanotransduction. Fibroblast activation, contraction, and extracellular matrix deposition progressively increase matrix stiffness, which reinforces mechanosensitive signaling. A stiff fibrotic niche, persistent profibrotic signaling, and possible fibroblast mechanical memory may subsequently contribute to maintenance of an activated state and persistent scar fibrosis.

Figure created by author using BioRender.

FAK - focal adhesion kinase; ROCK - Rho-associated coiled-coil kinase; ECM - extracellular matrix

From mechanical initiation to fibrotic persistence

Mechanical tension can initiate fibroblast activation during wound healing, but the biological effects of this stimulus may extend beyond the period of increased external loading. This creates an important question in scar mechanobiology: why can a temporary mechanical stimulus contribute to a scar state that persists for much longer? One explanation is that the wound gradually changes its own mechanical environment. Activated fibroblasts deposit extracellular matrix (ECM), generate contractile force, and remodel the surrounding tissue. These changes increase matrix stiffness and create new mechanical signals within the scar itself. The source of mechanical stimulation can therefore shift over time. Early signals mainly arise from external tension, movement, and tissue recoil, while later signals can increasingly arise from the stiffened fibrotic matrix. Cellular responses to previous mechanical environments may also persist after the original stimulus has changed. Together, these processes provide a temporal link between mechanical initiation and fibrotic persistence.

Matrix-Mediated Mechanical Reinforcement

Fibroblast activation changes both the composition and the mechanical properties of the healing wound. Activated fibroblasts produce collagen, fibronectin, and other ECM components. Myofibroblasts also generate contractile force through their actomyosin cytoskeleton. These processes remodel the matrix and progressively increase local tissue stiffness.

This change in stiffness is biologically important because fibroblasts can sense the mechanical properties of their surrounding matrix. A stiffer ECM promotes integrin clustering and focal adhesion maturation. It can also enhance focal adhesion kinase (FAK) signaling and increase RhoA/ROCK-dependent cytoskeletal tension. These changes favor YAP/TAZ nuclear localization and support continued fibroblast activation and matrix production [5,8,9].

The relationship between matrix production and mechanotransduction is therefore bidirectional. Fibroblast activation produces a stiffer matrix, and the stiffer matrix then provides stronger mechanical input to fibroblasts. TGF-β signaling can further strengthen this process. Myofibroblast contraction can increase the activation of matrix-associated latent TGF-β, while TGF-β promotes α-SMA expression, matrix production, and a more contractile fibroblast phenotype [24]. The scar microenvironment can therefore generate part of the mechanical signal that sustains its own progression.

The matrix gradually changes from a downstream product of fibrosis into an upstream mechanical regulator of further fibrosis. This transition is central to understanding how scar formation can become self-reinforcing. Early in healing, mechanical loading is mainly imposed by factors such as wound-edge traction, skin recoil, movement, and incision orientation. As fibrosis develops, the scar matrix itself becomes an additional source of mechanical stimulation. External loading and matrix-derived mechanical signals can then act together to maintain fibroblast activation.

When the Consequence Outlasts the Mechanical Exposure

Experimental cutaneous scar models show that the biological consequences of increased mechanical loading can persist beyond the period of augmented loading. In a classic murine model, Aarabi et al. applied increased mechanical stress for only one week during the proliferative phase of wound healing. This short period of loading was sufficient to produce hypertrophic scars that persisted for more than six months. The scars also showed marked increases in volume and cellularity [25].

A more recent cutaneous tension model showed a similar temporal pattern. Li et al. used suture anchoring to apply directional tension to healing mouse wounds. The suture anchors frequently failed at approximately two weeks and could no longer provide the intended augmented tension. However, the resulting scar phenotype persisted well beyond this period. The scars remained evident for several months, and mature scars at six months were still widened and hypercellular, with persistent fibrotic features [26].

These findings show a temporal mismatch between augmented mechanical exposure and the resulting scar phenotype. They do not imply that all mechanical forces disappear after experimental loading ends. Healing skin continues to experience baseline tension, tissue recoil, contraction, movement, and matrix-derived forces. The important point is that the biological consequences can outlast the period of experimentally increased loading.

This temporal mismatch can be explained in part by changes produced during the loading period itself. Mechanical stimulation activates fibroblasts and promotes contraction and ECM deposition. These changes increase matrix stiffness and alter the mechanical environment of the wound. The developing scar can therefore continue to generate pro-fibrotic mechanical signals even after the initial increase in external loading has ended.

This relationship gives early mechanical exposure particular importance. A relatively brief period of increased loading can alter the later course of scar remodeling. Early tension control may therefore influence more than immediate wound-edge stability. It may also reduce the chance that an early mechanical stimulus becomes converted into a longer-lasting fibrotic state.

Fibroblast Mechanical Memory

Matrix-mediated reinforcement provides a tissue-level explanation for persistent fibrosis. At the cellular level, fibroblast mechanical memory represents a second mechanistically plausible contributor to persistence. Evidence for this phenomenon is strongest in in vitro fibroblast systems and experimental fibrosis models, whereas direct demonstration in human cutaneous scars remains limited. Mechanical memory refers to the persistence of mechanically induced cellular phenotypes after the original mechanical environment has changed.

Studies in fibroblast and fibrosis models have shown that prolonged culture on stiff substrates can produce changes that do not immediately disappear when cells are transferred to a softer environment [27].

Fibroblasts can retain features associated with mechanical activation, including altered cytoskeletal organization, contractile behavior, and pro-fibrotic gene expression. The duration and strength of the previous mechanical exposure can influence how long these changes persist.

YAP/TAZ signaling has been linked to this type of mechanically induced persistence because these transcriptional regulators connect cytoskeletal tension with longer-lasting gene expression programs. TGF-β signaling can also help maintain an activated fibroblast state. Other studies have proposed roles for microRNAs, chromatin remodeling, and epigenetic regulation [28]. These mechanisms may allow a previous mechanical environment to influence later cell behavior even after the immediate mechanical conditions have changed.

Mechanical memory should not be treated as a replacement for matrix-mediated reinforcement. The two processes can act at different levels. A stiff scar matrix can continue to provide external mechanical input, while fibroblasts may also retain part of their previous mechanically induced phenotype. These mechanisms could reinforce each other and prolong fibroblast activation.

Mechanical memory has been demonstrated in fibroblast and fibrosis models, whereas its specific contribution to persistent human hypertrophic scars remains incompletely established. It is therefore most useful to view mechanical memory as a possible amplifier of fibrotic persistence rather than as the single established cause of scar persistence.

An Integrated Temporal Model of Scar Mechanobiology

These findings support a temporal model in which scar mechanobiology progresses through three connected phases.

Phase I - mechanical initiation: External tension, tissue recoil, postoperative movement, and other mechanical forces provide the initial loading environment. Fibroblasts sense these forces through integrins, focal adhesions, the cytoskeleton, and related mechanotransduction pathways. FAK, RhoA/ROCK, YAP/TAZ, TGF-β-related signaling, and other pathways then promote fibroblast activation and myofibroblast differentiation.

Phase II - matrix-mediated reinforcement: Activated fibroblasts increase contraction and ECM deposition. Collagen and fibronectin accumulate, and the matrix becomes progressively stiffer. This stiffer environment strengthens integrin-mediated mechanosensing, cytoskeletal tension, and YAP/TAZ activity. At this stage, the wound is no longer responding only to external mechanical loading. The developing scar matrix begins to generate and amplify its own mechanical signals.

Phase III - fibrotic persistence: A stiff fibrotic niche can maintain mechanotransduction and pro-fibrotic signaling over time. Persistent matrix stiffness, myofibroblast contraction, and TGF-β-related signaling can support an activated cellular state. Fibroblast mechanical memory may provide an additional layer of persistence in some settings. Together, these mechanisms can maintain fibrosis even when the period of increased external mechanical loading has passed.

This process can be summarized as a temporal sequence from external mechanical loading to mechanotransduction, fibroblast and myofibroblast activation, matrix deposition and stiffening, reinforced mechanosensing, sustained pro-fibrotic signaling, and ultimately persistent scar fibrosis. The key transition occurs when the mechanical environment is no longer determined mainly by forces outside the scar. As fibrosis develops, the matrix itself becomes part of the mechanism that maintains mechanical signaling. The scar therefore changes from a tissue responding to mechanical load into a tissue that can partly reproduce the mechanical conditions that favor further fibrosis.

This temporal model also provides a direct bridge to clinical tension management. If mechanical exposure can be biologically reinforced across time, effective scar prevention may depend not only on how much tension is reduced at closure, but also on when mechanical control is initiated and how long it is maintained.

Clinical tension reduction as control of the scar mechanical environment

Mechanical tension is clinically important because it changes the mechanical input received by healing tissue. Therefore, tension management should not be viewed only as a method for bringing wound edges together. Its broader purpose is to control the mechanical environment in which the scar develops. Different closure and offloading strategies achieve this goal at different tissue depths, over different distances, and for different periods of time. For this reason, techniques can be compared by their mechanical purpose rather than by their names. This perspective connects surgical practice directly with the mechanobiology described above.

Mechanical Objectives of Tension Management

Clinical tension management can be understood through four basic mechanical objectives: load sharing, load redistribution, load transfer, and offloading.

Load sharing allows several tissue layers to carry the closing force together. This reduces the amount of force concentrated at the superficial wound edge. Load redistribution spreads focal stress over a broader tissue area. This approach reduces local peaks in mechanical load and creates a more even force distribution around the incision. Load transfer moves part of the closing force from the superficial wound edge to deeper or more distant tissue structures. This becomes more important as the mechanical demand of the wound increases. Offloading reduces repeated mechanical input after closure and helps maintain the mechanical environment created during surgery.

These objectives are closely related, but they address different mechanical problems. A single technique may serve more than one purpose. The main question is therefore not which named suture is used, but which mechanical problem needs to be solved. Techniques should be classified by the mechanical problem they solve. These strategies are summarized according to their mechanical purpose, clinical context, and temporal role in Table *1*.

Mechanical strategy	Representative approaches	Primary mechanical purpose	Typical mechanical context	Temporal role
Layered load sharing	Buried dermal and subcutaneous closure	Distributes closing force across multiple tissue layers and reduces direct traction at the superficial wound edge	Low to moderate mechanical demand with adequate tissue mobility	Mainly intraoperative; establishes the initial mechanical foundation
Local tension redistribution	Heart-shaped sutures, LBD sutures, running barbed or fishbone-like techniques	Spreads focal traction across a broader local dermal and subcutaneous area	Moderate local tension or focal stress concentration	Mainly intraoperative and early postoperative
Deep or distant load transfer	Super-tension-relieving sutures, distant tension-relieving sutures, multilayer gradient redistribution techniques	Transfers part of the closing force from the wound edge to deeper or more distant tissue	High residual tension, large defects, scar revision, or limited local tissue reserve	Intraoperative redistribution with sustained support during early healing
Postoperative mechanical offloading	Paper tape, external tension-reduction devices, movement or posture modification	Reduces repeated traction and deformation after wound closure	Wounds exposed to persistent or dynamic postoperative loading	Extends mechanical control beyond surgery

TABLE 1: Mechanical classification of clinical tension-management strategies

Layered Load Sharing

Layered closure is the baseline strategy for mechanical load sharing. Buried dermal and subcutaneous sutures allow deeper tissue planes to participate in wound closure. This reduces the proportion of the closing force carried by the epidermal and superficial dermal edges. The main mechanical role of layered closure is therefore to prevent the superficial wound edge from bearing the full closing load [29,30].

This strategy is well suited to wounds with low or moderate mechanical demand. In these wounds, distributing force across several tissue planes can provide stable approximation while limiting direct traction on the skin edge. The principle is more important than the exact suture pattern. Deeper tissue support creates a mechanical foundation, while superficial sutures mainly refine edge alignment.

As mechanical demand increases, the required scale of redistribution also changes. Higher mechanical demand may require redistribution across a broader or deeper tissue volume. This transition leads from simple layered load sharing to techniques designed for more active redistribution of local force.

Local Tension Redistribution

Local tension-redistribution techniques aim to spread focal traction across a broader area of the dermis and superficial subcutaneous tissue. They modify the path or distribution of the suture so that force is not concentrated at a small number of points near the wound edge.

The heart-shaped suture, also described as wedge-shaped excision with a modified buried vertical mattress suture, illustrates this principle. Its extended intradermal pathway recruits a broader area of dermis into the suture loop, thereby providing tension relief and wound-edge eversion [30,31].

The looped, broad, and deep buried (LBD) suturing technique applies a similar principle over a broader local tissue volume. Its wider and deeper pathway allows the dermis and subcutaneous tissue to share more of the closing load and has been used in scar excision wounds [32].

Running barbed and fishbone-like sutures distribute force through multiple fixation points along the incision. Their mechanical value lies in reducing focal loading and creating more continuous local load sharing rather than relying on a few isolated points of traction [33,34].

These techniques differ in configuration, but their shared purpose is clear. They spread focal traction over a broader local dermal and subcutaneous area. The named technique is therefore secondary to the underlying mechanical function.

Deep and Distant Load Redistribution

Some wounds generate mechanical demands that extend beyond the capacity of local dermal redistribution. In these settings, deeper or more distant tissue can be recruited to carry part of the load. The scale of tension management therefore expands in both depth and distance.

Super-tension-relieving techniques use deeper tissue support to reduce the load transmitted directly to the wound edge. Zhang et al.'s super-tension-relieving approach is one example. It combines deep load sharing with sustained redistribution and can use slowly absorbable barbed sutures to maintain support during early healing [35,36].

Distant tension-relieving sutures extend the same principle beyond the immediate wound margin. By transferring part of the force farther from the scar edge, these techniques can reduce local mechanical concentration. This approach has been used in scar revision and can support long-term control of scar width [37].

The Zunyi suture is one representative approach within this mechanical category. Its design emphasizes multilayer load bearing and gradient redistribution. Deep anchoring and staggered placement allow force to be distributed across different tissue levels and distances. In reported anterolateral thigh flap donor-site closure, this strategy was used to transfer load away from the superficial wound edge and into a broader surrounding tissue volume [38].

These examples support a broader clinical principle. As mechanical demand increases, the scale of load redistribution may need to expand in depth and distance. The choice of technique should therefore follow the mechanical requirements of the wound rather than the name or complexity of the procedure.

Postoperative Mechanical Offloading

Closure ends the operation, but not mechanical exposure. Healing tissue continues to experience forces from skin recoil, body movement, posture, joint motion, and muscle activity. Postoperative offloading therefore extends mechanical control beyond the operating room and addresses the duration of mechanical input.

Paper tape provides a simple example. It can reduce multidirectional traction across a healing incision and has been associated with a lower risk of hypertrophic scar formation in wounds exposed to skin tension [15]. External tension-reduction devices provide a more sustained form of mechanical support. These systems can shield the incision from repeated traction and help preserve the reduction in wound-edge stress achieved during surgery [16,39].

Activity and posture modification also form part of postoperative mechanical management. Their relevance is greatest in wounds over joints, highly mobile regions, and areas exposed to repeated muscle movement. The goal is not complete immobilization. The goal is to reduce avoidable or repeated traction during periods when the scar-forming tissue remains mechanically responsive.

Mechanical offloading should also be separated conceptually from general scar therapy. Silicone gel, silicone sheets, and pressure therapy may be useful components of scar management, but they should not automatically be grouped with interventions whose primary purpose is direct reduction of tensile loading. Keeping these categories separate clarifies how each treatment modifies the scar environment.

Clinical tension management therefore operates across different depths, distances, and durations of load redistribution. This mechanical framework provides the basis for selecting the intensity of intervention

according to scar mechanical risk, which is developed in the following section.

Timing, duration, and risk-matched mechanical management

The clinical implication of mechanically reinforced fibrosis is that tension management should not be treated as a single intraoperative maneuver. Mechanical exposure changes over time, beginning with resting skin tension, tissue recoil, incision geometry, and wound-edge traction, and continuing through postoperative movement, contraction, and matrix remodeling. The relevant clinical questions are therefore not only how much tension is reduced at closure, but also when control begins, how long it is maintained, and which wounds require stronger intervention. A time- and risk-matched strategy provides a practical framework for translating mechanobiology into perioperative scar management.

Why Timing Matters

Mechanical control is most rational when it begins before excessive mechanical signaling becomes established. Incision planning, orientation relative to local tension lines, appropriate tissue mobilization, and redistribution of load during closure all act upstream of fibroblast activation by reducing avoidable mechanical input at the wound edge. Their role therefore extends beyond facilitating approximation.

Excessive traction, repeated deformation, or localized stress concentration may enhance mechanotransduction through focal adhesions, cytoskeletal tension, YAP/TAZ activity, and pro-fibrotic signaling. Early mechanical control is therefore biologically consistent with limiting the initiation of this response. In practice, scar prevention begins with the mechanical design of the wound: selecting a favorable incision trajectory when possible, mobilizing tissue sufficiently to avoid concentrated edge stress, and choosing a closure strategy that distributes rather than merely opposes tension. The objective is not simply primary closure, but prevention of excessive mechanical initiation.

Why Duration Matters

A mechanically stable wound at the end of surgery does not necessarily mark the end of scar-related mechanical risk. After closure, movement, tissue recoil, contraction, and progressive changes in extracellular matrix stiffness continue to alter local loading. Meanwhile, matrix deposition and myofibroblast contraction can transform the wound from a tissue exposed mainly to external loading into a mechanically reinforced local environment.

This distinction makes both the magnitude and duration of mechanical control clinically relevant. Magnitude describes how much load is reduced or redistributed at a given time, whereas duration describes how long that reduction is maintained while the wound remains mechanically responsive. A substantial intraoperative reduction in tension addresses the initial load but does not necessarily address repeated traction during subsequent healing. Sustained postoperative offloading can therefore help preserve the mechanical environment established at closure.

The duration of mechanical control may therefore be as clinically relevant as the magnitude of initial tension reduction. This provides a mechanistic rationale for postoperative taping, external offloading, movement modification, and related measures that reduce repeated deformation. These interventions are better understood as extensions of the same mechanical treatment strategy across time rather than as isolated postoperative adjuncts.

Conceptual Mechanical Risk Stratification

Mechanical management should also match the expected burden on the wound. A practical framework is to stratify wounds into low, moderate, and high mechanical risk according to incision orientation, regional mobility, defect size, tissue availability, residual closure tension, and previous scar behavior.

Low-risk wounds include small defects with favorable orientation, low mobility, adequate tissue laxity, and minimal residual tension. Low-risk wounds may be appropriately managed with layered closure and basic postoperative support.

Moderate-risk wounds are characterized by less favorable orientation, moderate mobility, greater tissue demand, or persistent but manageable residual tension. These wounds favor stronger deep load sharing, broader local redistribution of force, and planned postoperative offloading rather than superficial approximation alone.

High-risk wounds include scar revisions, large defects, flap donor sites, highly mobile regions, wounds with strong residual tension, and sites with repeated previous widening or hypertrophic scarring. Here, deeper or more distant load transfer, wider stress redistribution, sustained offloading, and structured scar surveillance become increasingly important.

This is a conceptual decision framework rather than a validated clinical scoring system or treatment algorithm. Its purpose is to translate current mechanobiological principles into a clinically interpretable relationship between mechanical burden and intervention intensity. Importantly, it shifts decision-making away from selecting a particular named technique and toward identifying the mechanical function that the wound requires. The optimal timing, duration, and intensity of mechanical tension control have not yet been defined by comparative clinical trials and should therefore be individualized according to wound characteristics, anatomical site, mobility, residual tension, and evolving scar behavior. This conceptual relationship between mechanical burden and intervention intensity is summarized in Figure 2.

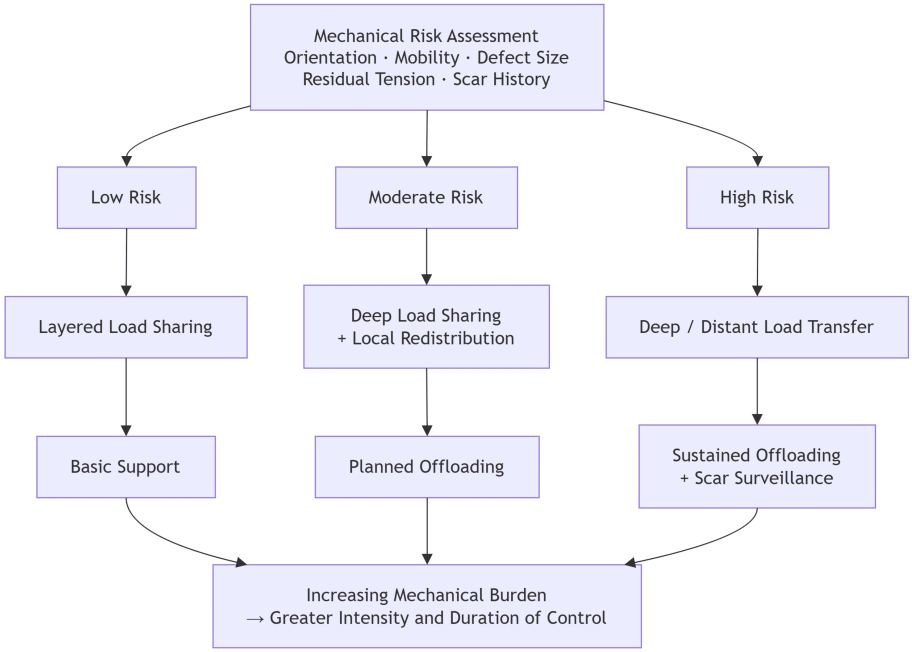


FIGURE 2: Conceptual risk-based framework for mechanical management of cutaneous scars

Mechanical risk is estimated from incision orientation, mobility, defect size, residual tension, and scar history. Increasing mechanical burden is matched with progressively broader, deeper, and more sustained mechanical control.

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Scar Surveillance and Adaptive Management

Mechanical management should remain adaptive after closure because scar behavior provides feedback on whether the initial strategy has been sufficient. Follow-up can integrate scar width, erythema, thickness, pliability, pigmentation, pain, pruritus, patient-reported symptoms, and structured instruments such as the Vancouver Scar Scale or Patient and Observer Scar Assessment Scale [40,41]. The central purpose is to identify an early trajectory toward unfavorable remodeling rather than simply accumulate scores.

Progressive widening, increasing thickness, persistent erythema, symptomatic hypertrophy, or recurrent traction-related change may indicate persistent mechanical loading or evolving fibrotic activity. These findings can prompt closer surveillance, reinforcement of offloading, adjustment of activity or support measures, and timely escalation to additional scar-directed interventions when appropriate.

The resulting pathway is continuous: risk assessment → intervention → surveillance → adjustment. In this model, mechanical management is not completed when the final suture is placed. It becomes a time-sensitive, risk-matched, and adaptive strategy for controlling the mechanical environment throughout scar formation.

Evidence boundaries and future directions

Evidence Supporting Mechanical Initiation and Reinforcement

The mechanobiological framework of cutaneous scarring rests on a well-supported core. Experimental and clinical evidence consistently indicates that mechanical loading can alter scar phenotype and that

fibroblasts respond dynamically to changes in extracellular stiffness, stretch, and cytoskeletal tension. At the cellular level, integrin-focal adhesion signaling provides a major interface for sensing extracellular mechanical conditions, while FAK, RhoA/ROCK, YAP/TAZ, and TGF-β-related pathways translate these physical cues into changes in fibroblast activation, contractility, and extracellular matrix production [5,8,9].

Importantly, the extracellular matrix is not simply a downstream product of this response. Activated fibroblasts increase collagen deposition, matrix remodeling, and tissue contraction, thereby progressively altering matrix stiffness and local force transmission. A stiffer matrix can in turn enhance focal adhesion formation, cytoskeletal tension, and mechanosensitive transcription. These observations provide a strong mechanistic basis for two linked stages of scar progression: mechanical initiation, in which externally imposed loading activates fibrotic signaling, and matrix-mediated reinforcement, in which the remodeled tissue increasingly contributes to its own mechanical signaling environment.

Defining the Transition to Fibrotic Persistence

The next major question concerns time. Most mechanotransduction studies establish how fibroblasts respond while mechanical stimulation is present, whereas clinical scars evolve over weeks to months after the original wound has been closed. The critical transition is therefore not simply whether loading activates fibroblasts, but when that activation becomes sufficiently reinforced to persist after the initiating mechanical condition has changed.

This transition can be approached along several complementary dimensions. First, the magnitude and duration of loading required to shift fibroblasts from a reversible mechanically activated state toward a persistent fibrotic phenotype need to be defined. Second, withdrawal experiments are needed to determine which responses rapidly return toward baseline after mechanical unloading and which remain sustained. Nuclear YAP/TAZ localization, focal adhesion signaling, α-SMA expression, TGF-β activity, matrix production, contractility, and matrix stiffness provide particularly relevant readouts.

A related question is whether fibroblasts involved in hypertrophic scars and keloids acquire a form of mechanical memory. Direct experiments in cutaneous scar-derived fibroblasts using hard-to-soft substrate transfer or stretch-and-release designs would clarify whether prior mechanical exposure can maintain an activated phenotype after the original stimulus has been removed. Establishing this phenomenon directly in human scar tissue would connect the concept of cellular mechanical memory to clinically persistent fibrosis.

The same temporal question applies clinically. The duration of postoperative offloading should ultimately be related not only to wound closure, but also to anatomical site, recovery of wound strength, residual mechanical load, scar phenotype, and evidence of ongoing mechanosensitive activity. This would shift mechanical management from a fixed postoperative schedule toward biologically informed duration of intervention.

Priority Experimental Framework

A particularly informative experimental design would follow a loading → withdrawal → longitudinal follow-up sequence. Cells or tissue models could be exposed to defined magnitudes and durations of mechanical loading, followed by complete or graded withdrawal of the stimulus. Serial assessment of YAP/TAZ activity, FAK-RhoA/ROCK signaling, α-SMA, TGF-β-related activity, ECM composition, contractility, and matrix stiffness would reveal whether the mechanically induced state resolves, decays slowly, or becomes self-maintaining.

Such experiments would directly connect mechanical dose, exposure duration, signal persistence, and matrix remodeling within one temporal framework. They could also identify the stage at which mechanical offloading is most capable of interrupting reinforcement before a persistent fibrotic state is established.

The key unanswered question is therefore not whether mechanical tension influences scarring, but when a mechanically induced fibrotic response becomes self-reinforcing and how that transition can be prevented.

Conclusions

Mechanical loading is not merely a technical issue of wound closure but an upstream biological input that can shape scar formation. Through mechanotransduction, external force is converted into fibroblast activation, cytoskeletal tension, and extracellular matrix remodeling. As matrix deposition, contraction, and tissue stiffening progress, the mechanical environment itself can become increasingly capable of sustaining fibrotic signaling. This provides a coherent framework for understanding cutaneous scarring as a progression from mechanical initiation, through matrix-mediated reinforcement, toward a potentially persistent fibrotic state. The central implication is that the biological effect of mechanical loading depends not only on whether force is present, but also on how strongly, where, and for how long that force is applied.

Accordingly, clinical tension management should move beyond the goal of immediate wound-edge

approximation toward a broader purpose: controlling the magnitude, distribution, and duration of mechanical input during the period in which scar-forming tissue remains mechanically responsive. Incision planning, layered load sharing, local or remote load transfer, and postoperative offloading can therefore be viewed as complementary components of a continuous mechanical-management strategy rather than isolated techniques. Viewing cutaneous scar progression from mechanical initiation to fibrotic persistence provides a mechanistic rationale for a timely, sustained, and risk-matched approach to control of the wound mechanical environment. At present, this approach is best regarded as an emerging, mechanism-informed clinical framework whose optimal timing, duration, and intensity require prospective clinical validation.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Xinyue Lai, Zairong Wei

Acquisition, analysis, or interpretation of data: Xinyue Lai

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Critical review of the manuscript for important intellectual content: Xinyue Lai, Zairong Wei

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References

1. Son D, Harijan A: Overview of surgical scar prevention and management . J Korean Med Sci. 2014, 29:751-7. 10.3346/jkms.2014.29.6.751
2. Ma Y, Liu Z, Miao L, Jiang X, Ruan H, Xuan R, Xu S: Mechanisms underlying pathological scarring by fibroblasts during wound healing. Int Wound J. 2023, 20:2190-206. 10.1111/iwj.14097
3. Zhang M, Chen H, Qian H, Wang C: Characterization of the skin keloid microenvironment . Cell Commun Signal. 2023, 21:207. 10.1186/s12964-023-01214-0
4. Monavarian M, Kader S, Moeinzadeh S, Jabbari E: Regenerative scar-free skin wound healing. Tissue Eng Part B Rev. 2019, 25:294-311.
5. Harn HI, Ogawa R, Hsu CK, Hughes MW, Tang MJ, Chuong CM: The tension biology of wound healing . Exp Dermatol. 2019, 28:464-71. 10.1111/exd.13460
6. Barnes LA, Marshall CD, Leavitt T, et al.: Mechanical forces in cutaneous wound healing: emerging therapies to minimize scar formation. Adv Wound Care (New Rochelle). 2018, 7:47-56. 10.1089/wound.2016.0709
7. Burgess LP, Morin GV, Rand M, Vossoughi J, Hollinger JO: Wound healing: relationship of wound closing tension to scar width in rats. Arch Otolaryngol Head Neck Surg. 1990, 116:798-802. 10.1001/archotol.1990.01870070046008
8. Wong VW, Rustad KC, Akaishi S, et al.: Focal adhesion kinase links mechanical force to skin fibrosis via inflammatory signaling. Nat Med. 2011, 18:148-52. 10.1038/nm.2574
9. Dupont S, Morsut L, Aragona M, et al.: Role of YAP/TAZ in mechanotransduction . Nature. 2011, 474:179-83. 10.1038/nature10137
10. Gong F, Wan B, Qi P, Wei Z: Biomechanical mechanism and clinical management progress of surgical wound tension. Front Surg. 2025, 12:1674382. 10.3389/fsurg.2025.1674382
11. Langer K: On the anatomy and physiology of the skin. I. The cleavability of the cutis . Br J Plast Surg. 1978, 31:3-8. 10.1016/0007-1226(78)90003-6
12. Hebiguchi T, Kato T, Yoshino H, Mizuno M: Z-shaped skin incision applying Kraissl's lines for an axillary thoracotomy. Surg Today. 2000, 30:139-41. 10.1007/PL00010061
13. Wilhelmi BJ, Blackwell SJ, Phillips LG: Langer's lines: to use or not to use . Plast Reconstr Surg. 1999,

104:208-14.
14. Kang M, Ko UH, Oh EJ, Kim HM, Chung HY, Shin JH: Tension-sensitive HOX gene expression in fibroblasts for differential scar formation. J Transl Med. 2025, 23:168. 10.1186/s12967-025-06191-1
15. Atkinson JA, McKenna KT, Barnett AG, McGrath DJ, Rudd M: A randomized, controlled trial to determine the efficacy of paper tape in preventing hypertrophic scar formation in surgical incisions that traverse Langer's skin tension lines. Plast Reconstr Surg. 2005, 116:1648-56. 10.1097/01.prs.0000187147.73963.a5
16. Gurtner GC, Dauskardt RH, Wong VW, et al.: Improving cutaneous scar formation by controlling the mechanical environment: large animal and phase I studies. Ann Surg. 2011, 254:217-25. 10.1097/SLA.0b013e318220b159
17. Wong VW, Paterno J, Sorkin M, et al.: Mechanical force prolongs acute inflammation via T-cell-dependent pathways during scar formation. FASEB J. 2011, 25:4498-510. 10.1096/fj.10-178087
18. Sarasa-Renedo A, Tunç-Civelek V, Chiquet M: Role of RhoA/ROCK-dependent actin contractility in the induction of tenascin-C by cyclic tensile strain. Exp Cell Res. 2006, 312:1361-70. 10.1016/j.yexcr.2005.12.025
19. Desmoulière A, Geinoz A, Gabbiani F, Gabbiani G: Transforming growth factor-beta 1 induces alpha-smooth muscle actin expression in granulation tissue myofibroblasts and in quiescent and growing cultured fibroblasts. J Cell Biol. 1993, 122:103-11. 10.1083/jcb.122.1.103
20. Wipff PJ, Rifkin DB, Meister JJ, Hinz B: Myofibroblast contraction activates latent TGF-beta1 from the extracellular matrix. J Cell Biol. 2007, 179:1311-23. 10.1083/jcb.200704042
21. Walker M, Godin M, Pelling AE: Mechanical stretch sustains myofibroblast phenotype and function in microtissues through latent TGF-β1 activation. Integr Biol (Camb). 2020, 12:199-210. 10.1093/intbio/zyaa015
22. Hu X, Wang H, Liu J, et al.: The role of ERK and JNK signaling in connective tissue growth factor induced extracellular matrix protein production and scar formation. Arch Dermatol Res. 2013, 305:433-45. 10.1007/s00403-013-1334-9
23. Chiquet M, Sarasa-Renedo A, Tunç-Civelek V: Induction of tenascin-C by cyclic tensile strain versus growth factors: distinct contributions by Rho/ROCK and MAPK signaling pathways. Biochim Biophys Acta. 2004, 1693:193-204. 10.1016/j.bbamcr.2004.08.001
24. Lin X, Lai Y: Scarring skin: mechanisms and therapies . Int J Mol Sci. 2024, 25:1458. 10.3390/ijms25031458
25. Aarabi S, Bhatt KA, Shi Y, et al.: Mechanical load initiates hypertrophic scar formation through decreased cellular apoptosis. FASEB J. 2007, 21:3250-61. 10.1096/fj.07-8218com
26. Li Y, Liu A, Wang J, et al.: Suture-anchored cutaneous tension induces persistent hypertrophic scarring in a novel murine model. Burns Trauma. 2024, 12:tkae051. 10.1093/burnst/tkae051
27. Balestrini JL, Chaudhry S, Sarrazy V, Koehler A, Hinz B: The mechanical memory of lung myofibroblasts . Integr Biol (Camb). 2012, 4:410-21. 10.1039/c2ib00149g
28. Kirk T, Ahmed A, Rognoni E: Fibroblast memory in development, homeostasis and disease . Cells. 2021, 10:2840. 10.3390/cells10112840
29. Buchanan PJ, Kung TA, Cederna PS: Evidence-based medicine: wound closure. Plast Reconstr Surg. 2016, 138:257S-70S. 10.1097/PRS.0000000000002775
30. Zhang W, Xie J, Zeng A: The origin and development of interrupted subcuticular suture: an important technique for achieving optimum wound closure. Dermatol Surg. 2022, 48:619-24. 10.1097/DSS.0000000000003437
31. Shu MG, Yang DY, He L: Surgical Skin Aesthetic Suturing Tutorial: Heart-Shaped Subcutaneous Suture Technique (Book in Chinese). Xi'an Jiaotong University Press, Xi'an; 2024.
32. Chai LL, Tang XW, Li ZX, Luo HJ, Jiang SQ, Yang DY: Clinical application of the looped, broad and deep buried suturing technique (LBD suture technique) in scar resection (Article in Chinese). Chin J Aest Med. 2019, 28:16-9. 10.15909/j.cnki.cn61-1347/r.003188
33. Chen LB, Wu XL, Chen ZA, Gao Z: A tension-reduced suture technique in scar plastic surgery (Article in Chinese). J Tissue Eng Reconstruct Surg. 2019, 15:63-4. 10.3969/j.issn.1673-0364.2019.02.001
34. Wang YF, Yang YT, Chen ZA, Wu XL: The running barbed tension-offloading suture: a technique update on tension wound closure. Asian J Surg. 2023, 46:3773-6. 10.1016/j.asjsur.2023.05.114
35. Chen J, Zhang YX: Clinical effect of Zhang's super tension-relieving suture for high-tension wound closure (Article in Chinese). Zhonghua Shao Shang Za Zhi. 2020, 36:339-45. 10.3760/cma.j.cn501120-20200314-00163
36. Min P, Zhang S, Sinaki DG, et al.: Using Zhang's supertension-relieving suture technique with slowly-absorbable barbed sutures in the management of pathological scars: a multicenter retrospective study. Burns Trauma. 2023, 11:tkad026. 10.1093/burnst/tkad026
37. Ma FX, Zhang YH, Dong YC, Ren P, Zhou HW, Huang R, Li J: Clinical efficacy of W-plasty resection combined with remote tension-reducing suture in upper limb scar revision (Article in Chinese). Zhonghua Shao Shang Yu Chuang Mian Xiu Fu Za Zhi. 2026, 42:350-8. 10.3760/cma.j.cn501225-20241226-00507
38. Chang S, Mo X, Wei Z, et al.: Application of Zunyi's suture method for donor site of anterolateral thigh flap (Article in Chinese). Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi. 2021, 35:477-82. 10.7507/1002-1892.202008101
39. Burm JS, Lee J, Kang SY, Park J: Scar revision in areas subjected to excessive tension using intraoperative and postoperative long-term tension reduction techniques. Arch Aesthetic Plast Surg. 2023, 29:207-12. 10.14730/aaps.2023.00850
40. Sullivan T, Smith J, Kermode J, McIver E, Courtemanche DJ: Rating the burn scar . J Burn Care Rehabil. 1990, 11:256-60. 10.1097/00004630-199005000-00014
41. Draaijers LJ, Tempelman FR, Botman YA, Tuinebreijer WE, Middelkoop E, Kreis RW, van Zuijlen PP: The patient and observer scar assessment scale: a reliable and feasible tool for scar evaluation. Plast Reconstr Surg. 2004, 113:1960-5. 10.1097/01.prs.0000122207.28773.56