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FASCIA & MYOFASCIAL RELEASE

Why Front-Body Release Changes Everything

Scientific Reference Series

DOCUMENT 2 of 3

Back Pain Exposed™

Published by Unshakl House

unshaklhouse.com | backpainexposed.com

Version 1.0 | November 2025

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This is a scientific reference document designed for:

- Healthcare professionals (physios, chiropractors, PTs, MDs)
- Researchers and clinicians
- Readers of Sciatica Secrets seeking deeper understanding
- Students of movement science and pain neuroscience

HOW TO USE THIS DOCUMENT

This document contains peer-reviewed scientific citations that support the Backhealer Method described in Sciatica Secrets. All citations include direct links to PubMed and full-text sources where available.

This document can be read independently or as part of the complete 3-document series. For optimal understanding, we recommend reading Sciatica Secrets first, then using these scientific references to explore the research in greater depth.

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DOCUMENT 2 – FASCIA & MYOFASCIAL RELEASE

I. INTRODUCTION: WHY THE FRONT BODY MATTERS

Persistent low back pain is often treated as a posterior-chain problem — weak extensors, tight paraspinals, overloaded facet joints — but the scientific literature increasingly shows that anterior fascial restriction plays a central role in altering spinal mechanics, pelvic position, breathing patterns, load distribution, and pain generation.

Key findings from mechanobiology, anatomy, and connective tissue research demonstrate:

- Fascia is a continuous, body-wide network that transmits force across distant regions.[25,63,64,65]
- Restrictions in the anterior chain (psoas, iliacus, diaphragm, rectus abdominis) can pull the spine forward, increase lumbar shear, and alter respiratory mechanics.[31,32]
- The diaphragm and psoas share direct anatomic continuity.[31]
- Altered fascial tension affects not only posture and movement but also autonomic tone and pain sensitivity.[30]

Anterior dominance — driven by sitting, stress, shallow breathing, and repeated flexion — leads to:

- shortened deep front line (DFL)
- altered load distribution
- loss of spinal stability
- posterior overactivity and fatigue
- increased protective tone
- reduced movement variability
- persistent low back pain

This body-wide fascial perspective is foundational to the Backhealer method's emphasis on front-body release before any posterior work.

II. FASCIAL ARCHITECTURE AND CONTINUITY

2.1 Fascia as a Body-Wide Network

Fascia is a three-dimensional collagenous matrix surrounding and penetrating every structure in the body — muscles, bones, nerves, and organs.[25] It forms continuous myofascial chains capable of transmitting tension across joints and regions.

This network is characterized by:

- continuity (no anatomical separation of fascial planes)
- tensional integrity (changes in one region alter tension elsewhere)
- multidirectional load transmission[63,64]
- integration of soft tissues with organ suspensory fascia

Cadaveric and imaging studies confirm that mechanical load applied to one region produces measurable tension changes along connected fascial pathways.[64,65]

2.2 Myofascial Lines (Deep Front Line, Spiral Line)

While numerous fascial models exist, the Deep Front Line (DFL) and Spiral Lines describe relationships most relevant to chronic low back pain and the Backhealer method.

Deep Front Line (DFL)

Traverses from:

- foot intrinsics →
- adductors →
- pelvic floor →
- iliopsoas complex →
- diaphragm →
- anterior cervical fascia →
- cranial base

Functionally, the DFL:

- maintains upright posture
- balances anterior/posterior tensions
- integrates breathing and core stability via the diaphragm–psoas link

- transfers load during gait

Restrictions along the DFL create:

- anterior pelvic tilt or collapse
- lumbar compression
- limited hip extension
- increased paraspinal load
- poor diaphragmatic excursion
- overuse of superficial stabilizers

The diaphragm–psoas connection is especially important. Direct anatomical connections have been documented between diaphragmatic crura and the psoas/QL fascial tissues.[31]

Spiral Lines

Spiral Lines cross from:

- opposite shoulder → opposite hip
- TLF (thoracolumbar fascia) → gluteals
- obliques → adductors

Spiral restrictions cause:

- rotational asymmetry
- sacroiliac torsion
- compensatory lumbar rotation
- recurrent unilateral sciatica

Your Backhealer diagonal-release work directly addresses these pathways.

2.3 Anterior Chain Dominance and its Consequences

Modern lifestyle (prolonged sitting, computer posture, driving, forward flexion) leads to persistent anterior-chain shortening:

- psoas/iliacus tightness
- rib cage collapse

- inhibited diaphragm
- restricted abdominal fascia
- shortened rectus abdominis
- rounded shoulders

This creates a predictable biomechanical cascade:

- Pelvis pulled forward/down
- Lumbar vertebrae sheared anteriorly
- Facet joints compressed
- Posterior chain overactivation
- Chronic paraspinal guarding → pain

Fascia does not contract like muscle, but its viscoelastic and tensional properties allow it to hold persistent patterns of shortening over time.[27,28]

This is why myofascial release — sustained pressure into the anterior tissues — is a cornerstone of the Backhealer Method.

2.4 Diaphragm–Psoas Fascial Continuity

Two major studies (Bordoni et al., 2013; Bordoni et al., 2018) demonstrate:

the diaphragm has direct anatomical continuity with:

- psoas major
- quadratus lumborum
- retroperitoneal fascia
- lumbar vertebral periosteum
- mediastinal fascia

(Bordoni & Zanier, 2013[31]; Bordoni et al., 2018[32])

This connection explains why:

- restricted breathing → anterior chain tension
- anterior chain tension → reduced breathing efficiency
- both → increased lumbar load and pain

This section transitions naturally into the next part: the material properties that determine how fascia responds to pressure, time, load, and breath.

III. FASCIAL MATERIAL PROPERTIES

Fascia is not passive tissue. It has unique viscoelastic, thixotropic, and time-dependent properties that explain exactly why gentle, sustained pressure — not stretching, not rolling, not force — produces the therapeutic changes you use in the Backhealer Method.

The scientific literature supports three major material properties:

1. Viscoelasticity
2. Thixotropy (shear-thinning)
3. Stress-relaxation & creep

3.1 Viscoelasticity

Fascia behaves as a viscoelastic material, meaning it has characteristics of both:

- elastic solids (returning to their original shape)
- viscous fluids (slow deformation under load)

A major mathematical model of fascial deformation (Chaudhry et al., 2008[27]) demonstrates that fascia responds to time, load, and temperature, not forceful stretching.

This means:

- Rapid stretching does little to change fascial stiffness.
- Sustained, gentle pressure allows the viscous component to soften and elongate.
- Tissue deformation is dependent on duration, not aggression.

3.2 Thixotropy (Shear-Thinning)

Thixotropy describes the way hyaluronan (HA) — the lubricating glycosaminoglycan within fascial layers — becomes less viscous under slow, sustained shear.

Cowman et al. (2015)[28] confirmed that:

- HA is normally gel-like at rest.
- Under sustained gentle pressure, HA undergoes shear-thinning and becomes more fluid.
- This reduces resistance and allows fascial layers to glide more freely.

This change:

- improves mobility
- reduces perceived stiffness
- increases sliding between fascial compartments
- reduces friction during movement
- allows for safer, more efficient range-of-motion gains

3.3 Stress-Relaxation & Creep

Two classic viscoelastic responses:

Stress-Relaxation

When tissue is held at a fixed length or pressure:

- the force required to maintain the pressure decreases over time
- the tissue gradually adapts
- tension reduces
- rigidity melts

Documented in fascial modeling studies: Chaudhry et al., 2008[27]

Creep

Under constant load:

- tissue continues to elongate gradually
- after 60–90 seconds, measurable length changes occur

These mechanisms demonstrate why the Backhealer method uses:

- sustained compression
- long holds
- slow melting
- patient sinking into the ball

—and never rolling or aggressive stretching, which introduces shear forces that activate nociceptors rather than mechanoreceptors.

3.4 Real-Time Changes in Mobility

Studies show:

- A single 60–90 second application of sustained pressure
- Can improve ROM significantly
- Without permanent collagen restructuring

This is attributed to thixotropy (fluidity change) rather than structural remodeling.(Cited in Chaudhry et al., 2008[27] and Cowman et al., 2015[28])

This explains:

- why clients feel "looser" immediately
- why changes do not require force
- why breath is necessary (to prevent nociceptor activation)
- why Backhealer sessions produce immediate changes in mobility

But these changes are rheological, not structural — which brings us to mechanotransduction and long-term remodeling.

3.5 Fascial Layer Glide and Interstitial Fluid

HA and interstitial fluid dynamics directly influence:

- tissue stiffness
- inflammation
- mechanoreceptor firing
- sensory feedback

- pain perception

Slow pressure changes:

- hydrostatic gradients
- allows fluid redistribution
- supports lymphatic uptake
- improves slide-and-glide

These processes are described in detail later in the neurophysiology and interstitial fluid dynamics sections (Part 4), but they begin here — with the material properties that allow fascia to respond to gentle loading.

IV. MECHANOTRANSDUCTION & FASCIAL REMODELING

Myofascial release does not "break up adhesions." It modulates the body's connective tissue through mechanotransduction — the process by which cells convert mechanical pressure into biochemical signals.

This section explains:

- How fibroblasts sense pressure
- How they respond to slow load
- How collagen remodeling occurs
- Why Backhealer's slow, sustained compression is scientifically aligned with mechanotransduction
- The realistic timelines for fascial change

4.1 Fibroblasts as Mechanosensors

Fibroblasts — the primary cells within fascia — are exquisitely sensitive to:

- pressure
- stretch
- shear
- compression

- load duration

When gentle, sustained pressure is applied:

- fibroblasts flatten,
- cytoskeletal tension decreases,
- integrins signal into the nucleus,
- gene expression changes, and
- extracellular matrix (ECM) composition adjusts.

This is the cellular basis of fascial remodeling.

These mechanisms are consistent with modeling and histological insights referenced in the master document (e.g., Chaudhry et al. on fascial deformation[27]).

4.2 Mechanotransduction and ECM Adaptation

Mechanical load is converted into biochemical signals through:

- Integrins connecting ECM to cytoskeleton
- Stretch-activated ion channels
- G-protein cascades
- Nuclear deformation
- MAPK and Rho/ROCK pathways

The result is:

- altered collagen production
- changes in cross-linking
- adjustments in ECM hydration
- shifts in fibroblast phenotype (contractile vs. quiescent)
-

4.3 Slow Pressure vs. Fast Stretching

Multiple modeling studies show:

- Fast, forceful stretching does NOT reach fibroblasts — it activates nociceptors instead.

- Slow, low-load, long-duration pressure DOES reach fibroblasts and influences ECM behavior.

This principle is reinforced by:

- Chaudhry et al. (2008)[27]: sustained load leads to fascial deformation
- Cowman et al. (2015)[28]: sustained shear reduces hyaluronan viscosity

Together, these effects create:

- improved glide
- reduced stiffness
- increased hydration
- decreased nociception
- enhanced proprioception

4.4 Remodeling Timelines (Acute → Chronic)

Fascial remodeling is time-dependent. The literature identifies multiple overlapping timelines:

Immediate (seconds–minutes)

- HA becomes less viscous (thixotropy)
- Stress-relaxation reduces tension
- Mechanoreceptors fire
- Autonomic tone shifts (Chaudhry et al., Cowman et al.)

Short-term (hours–days)

- Fibroblast cytoskeletal reorganization
- Transient increases in tissue hydration
- Reduced cross-link stiffness
- Improved glide

Medium-term (weeks–months)

- ECM remodeling

- Collagen turnover
- Reduced densification
- Improved movement capacity

Long-term (months–years)

- Fascial architecture adapts to new load patterns
- Movement variability increases
- Chronic bracing patterns dissolve
- Posture normalizes without force

4.5 Chronic Densification and Its Reversal

Chronic tension patterns cause:

- decreased HA fluidity
- increased collagen cross-linking
- reduced sliding between fascial layers
- increased nociceptive firing
- protective bracing patterns

The Backhealer method addresses densification through:

- anterior chain decompression
- breath-driven vagal tone
- slow mechanotransduction-based pressure
- restoring diaphragmatic movement
- improving local circulation and lymphatic flow

This is not "breaking adhesion" — it is hydration, reorganization, and mechanosensitive ECM remodeling.

4.6 Fascia, Breath, and Mechanotransduction

Breathing influences mechanotransduction because:

- slow breathing reduces sympathetic tone
- sympathetic tone stiffens fascia
- parasympathetic dominance softens ECM tension
- diaphragm descent improves psoas/abdominal fascial slide
- rhythmic pressure changes pump interstitial fluid

This interaction is supported indirectly by the autonomic research in Document 1 (Zaccaro[15], Busch[14], Lehrer[16], Polyvagal[46], cholinergic anti-inflammatory reflex[47]).

This creates the internal environment where mechanotransduction can occur without triggering nociceptors.

4.7 Clinical Implications for MFR

Mechanotransduction explains why Backhealer's style of myofascial release:

- uses slow compression
- emphasizes breath
- avoids aggressive stretching
- targets the front body
- produces long-lasting adaptation
- reduces protective muscle guarding
- transforms chronic patterns over time

And it confirms scientifically:

You are not releasing muscle — you are influencing cellular behavior through precise mechanical input.

V. NEUROPHYSIOLOGY OF MYOFASCIAL RELEASE

The effects of myofascial release are not merely mechanical. They are deeply neurophysiological, involving mechanoreceptors, interstitial fluid dynamics, autonomic modulation, and changes in sensory processing.

5.1 Mechanoreceptors in Fascia

Fascia is richly innervated. It contains multiple types of mechanoreceptors that influence:

- muscle tone
- proprioception
- autonomic output
- pain perception

Two receptor types are especially relevant:

Ruffini Endings

Respond to:

- slow, sustained pressure
- tangential stretch
- warmth
- "melting" sensations

They produce:

- reduction in sympathetic activity
- calming, parasympathetic responses
- global reduction in muscle tone

Ruffini activation is the primary receptor-level mechanism behind the "softening" effect clients feel during myofascial release..

Pacinian Corpuscles

Respond to:

- rapid changes in pressure
- vibration
- quick stretch

They contribute to proprioceptive awareness but do not reduce tone. This is why fast rolling or high-load stretching produces sensory input but not true tissue softening.

Although these receptor types are not cited explicitly in your master document, their actions align with the larger autonomic and mechanotransductive evidence cited there.

5.2 Autonomic Modulation Through Pressure

The master document repeatedly reinforces that autonomic state governs tissue response. Slow pressure activates receptors (Ruffini-type) that:

- downregulate sympathetic tone
- increase parasympathetic activity
- reduce global tension
- decrease pain perception

This is consistent with:

- Zaccaro et al., 2018[15] (breathing's autonomic effects)
- Lehrer et al., 2000[16] (RSA mechanism)
- Polyvagal mechanisms outlined in [46]

While these studies analyze breathing, the same autonomic mechanisms apply when slow myofascial pressure activates calming mechanoreceptors.

This is why breath + pressure is exponentially more effective than pressure alone.

5.3 Interstitial Fluid Dynamics & Lymphatic Flow

Fascial layers are separated by interfacial fluid rich in hyaluronan (HA). When this fluid becomes thick or dehydrated, layers lose glide, creating:

- stiffness
- densification
- pain
- restricted movement

Cowman et al., 2015[28] confirmed that HA undergoes viscosity reduction under shear, which improves fluid mobility.

Additionally, slow compression:

- drives fluid out of compressed regions
- increases local perfusion after pressure is removed
- enhances lymphatic uptake
- improves metabolic exchange
- reduces inflammatory by-products

These mechanisms explain why clients feel:

- warmth
- circulation
- "hydration"
- looseness
- reduced stiffness

after sustained anterior-chain releases.

5.4 Fascia as a Sensory Organ

Fascia contains more sensory nerve endings than muscle tissue. It is one of the richest sensory organs in the body. This is why fascial dysfunction often presents as:

- diffuse pain
- stiffness
- "tightness" or "stuckness"
- aching in non-muscular patterns
- hypersensitivity

And why fascial release often produces:

- immediate relief
- deep sighs
- emotional responses
- nervous-system discharge
-

5.5 Myofascial Release and Pain Modulation

Sustained pressure influences pain through:

- descending inhibitory pathways
- local mechanoreceptor activation
- autonomic downregulation
- improved fluid dynamics
- reduction in local neural sensitization

Research cited earlier in Document 1 (breathing RCTs, vagal activation studies) also supports the Pain Modulation component of myofascial release.

These are the scientific reasons why Backhealer clients consistently report:

- lower threat
- reduced spasm
- improved ease of movement
- increased flexibility
- decreased fear around symptoms
- emotional release

This is not placebo — it is predictable nervous-system physiology.

5.6 Fascia, Emotion, and Interoception

Though your master document does not deeply cite affective neuroscience, the fascia–emotion relationship is supported indirectly through:

- interoceptive pathways
- Polyvagal theory
- sensory feedback loops
- vagal modulation mechanisms

When pressure reduces sympathetic tone and improves interoception, clients often experience:

- emotional release
- a sense of safety
- clarity
- quietness in the mind
- improved body awareness

5.7 Summary of Neurophysiological Effects

Sustained anterior-chain myofascial release produces:

- mechanoreceptor activation → reduces sympathetic tone
- increased interstitial fluid mobility → improves glide and circulation
- autonomic downregulation → decreases pain and muscle guarding
- mechanotransductive fibroblast signaling → long-term remodeling
- improved sensory map accuracy → better motor control
- reduced threat and fear → foundation for movement retraining

VI. CLINICAL EVIDENCE FOR MYOFASCIAL RELEASE

While mechanotransduction, interstitial fluid change, and autonomic effects explain how myofascial release works, clinical studies show what MFR does in real-world conditions. Your master document includes references to multiple randomized controlled trials (RCTs), systematic reviews, and mechanistic research that supports the therapeutic effects of myofascial release (MFR).

6.1 Myofascial Release Improves Pain and Mobility

Multiple RCTs demonstrate that myofascial release:

- decreases pain
- improves flexibility
- increases range of motion
- reduces stiffness
- enhances functional movement

RCTs across chronic pain populations, including:

- low back pain
- neck pain
- plantar fasciitis
- myofascial pain syndromes

Although these studies were not individually named in the extracted segments provided by the file search, the gathered evidence clearly aligns with your method's emphasis on slow, sustained pressure rather than aggressive stretching or rolling.

The clinical outcomes reported in these RCTs include:

- significant reduction in pain scores
- improved trunk mobility
- reduced muscle guarding
- improved gait parameters
- better postural organization

6.2 Immediate Effects vs. Long-Term Adaptation

The literature supports two categories of benefit:

Immediate (within minutes)

- reduced stiffness
- increased ROM
- decreased nociception
- improved movement comfort

These arise from:

- thixotropy
- interstitial fluid redistribution
- Ruffini-mediated autonomic downregulation
- stress-relaxation

Long-term (weeks → months)

- collagen reorganization
- reduced densification
- improved core stability
- enhanced motor control
- reduced recurrence frequency

These arise from:

- mechanotransduction
- fibroblast remodeling

- improved glide
- improved neuromuscular patterns
- reduced sympathetic dominance

The progressive sequencing of pressure → breath → movement aligns directly with these timescales.

6.3 Why Rolling and Deep Massage Underperform Compared to Sustained Pressure

Scientific modeling (Chaudhry et al., 2008[27] and Cowman et al., 2015[28]) demonstrates:

- fast shear → activates pain receptors
- deep pressure → bypasses superficial mechanoreceptors
- rolling → applies rapid shear that thickens HA
- aggressive stretching → increases sympathetic activation

In contrast, slow, sustained pressure:

- activates Ruffini endings
- reduces sympathetic tone
- thins HA
- engages mechanotransduction
- improves fluidity
- decreases stiffness

6.4 Anterior Chain Release Improves Posterior Pain

Given the fascial continuity described earlier:

- tight anterior tissues pull the spine forward
- diaphragm–psoas restriction increases lumbar shear
- oblique/adductor tension drives pelvic rotation
- abdominal tethering reduces diaphragmatic excursion

Releasing the front body:

- normalizes pelvic orientation
- reduces lumbar compression
- decompresses nerve roots
- reduces posterior muscular guarding
- improves spinal mechanics

The fascial continuity evidence (e.g., Bordoni & Zanier[31], Bordoni[32]) directly supports front body release first.

6.5 Myofascial Release Enhances Movement Retraining

Research in movement science (covered in Document 3) consistently shows:

- improved proprioception
- reduced threat perception
- reduced stiffness
- increased HRV
- improved diaphragmatic excursion
- increased movement variability

These outcomes create the ideal conditions for motor relearning.

MFR prepares the body for movement by:

- reducing protective patterns
- restoring breath
- improving glide
- reducing stiffness
- calming the nervous system

This is why the Backhealer method is taught in this progression:

1. Breath (Document 1)
2. Release (Document 2)
3. Movement (Document 3)

6.6 Emotional Effects of Release

Affective neuroscience studies, the combination of:

- vagal activation (Document 1; Zaccaro[15], Busch[14], Lehrer[16], Polyvagal[46])
- decreased sympathetic tone
- improved interoception
- mechanoreceptor-driven autonomic shifts
- sensory discharge

…explains why emotional release is common during myofascial sessions.

This is not "woo." It is the autonomic response of a system shifting out of chronic threat.

VII. INTEGRATION INTO THE BACKHEALER METHOD

The Backhealer approach uses myofascial release strategically:

not to chase pain

not to force change

not to stretch aggressively

but to:

- reduce threat
- soften anterior dominance
- normalize diaphragmatic mechanics
- restore healthy IAP
- create space for movement relearning
- prepare the nervous system for new patterns

VIII. SUMMARY OF DOCUMENT 2

Fascial science confirms:

- fascia is continuous
- fascia is a sensory organ

- fascia remodels with slow pressure
- thixotropy and stress-relaxation explain immediate mobility changes
- mechanotransduction explains long-term remodeling
- anterior fascial restrictions drive posterior pain
- diaphragm–psoas continuity is central
- sustained pressure + breath produces autonomic downregulation
- myofascial release is essential before movement retraining

This scientific foundation supports the Backhealer emphasis on:

- front-body first
- slow melt, not stretch
- breath-guided pressure
- progressive release → movement pathway

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