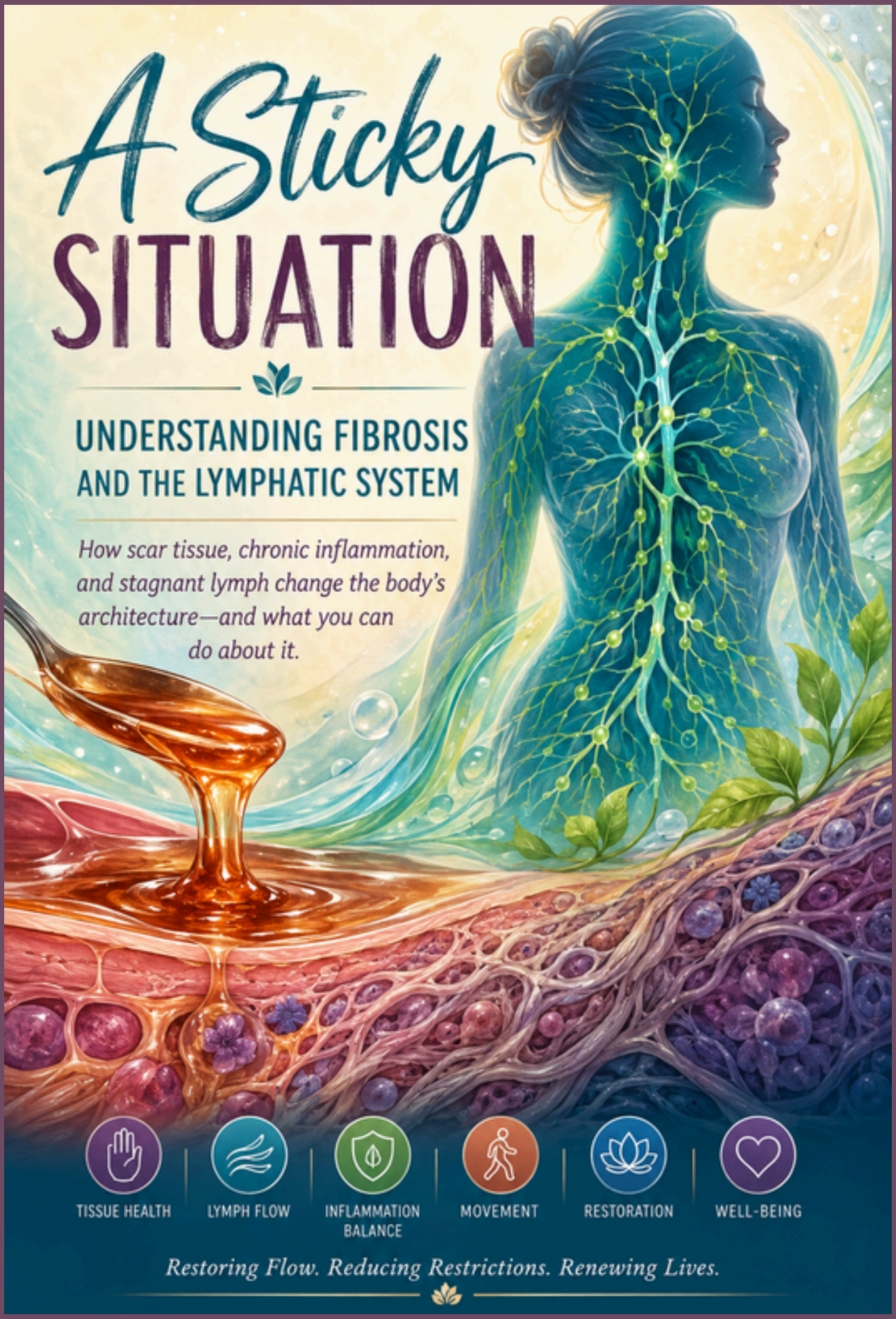

Laine Miller, PTA, LMT, CLT

Lymphatic Restoration Therapy

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Introduction

When most people hear the word fibrosis, they think of scar tissue.

Perhaps it's the firm area beneath a surgical incision, the stiffness that develops after an injury, or the hardened tissue that sometimes accompanies chronic swelling. While these are all examples of fibrosis, they tell only part of the story.

Fibrosis is not simply something that appears after an injury—it is the result of a complex series of biological events that begin long before collagen is ever deposited.

It develops within a remarkable living environment where cells communicate, immune responses are coordinated, fluids move continuously, and tissues are constantly being built down and rebuilt.

At the center of this environment lies the extracellular matrix (ECM)—a dynamic network that provides far more than structural support. The extracellular matrix is the medium through which nutrients are delivered, waste products are removed, immune cells travel, and healing begins.

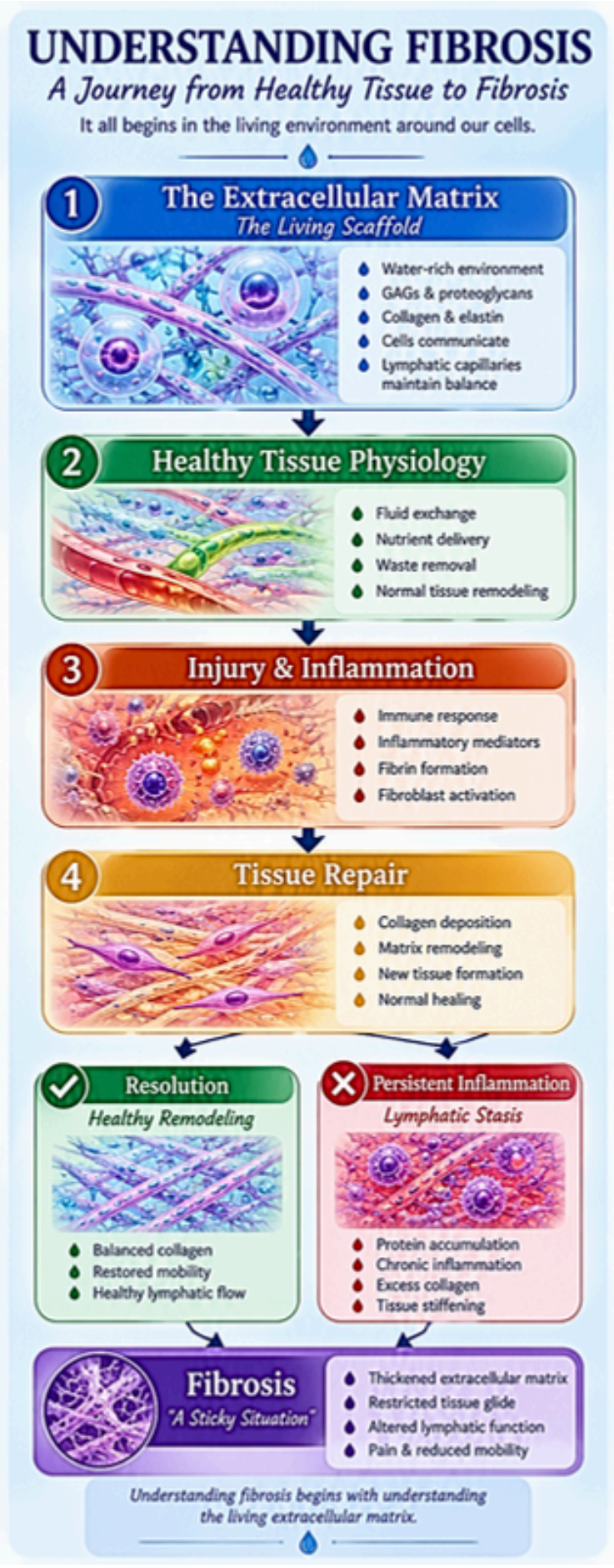
It is also where the lymphatic system performs one of its most important roles: maintaining the delicate balance of fluid and proteins that keeps our tissues healthy.

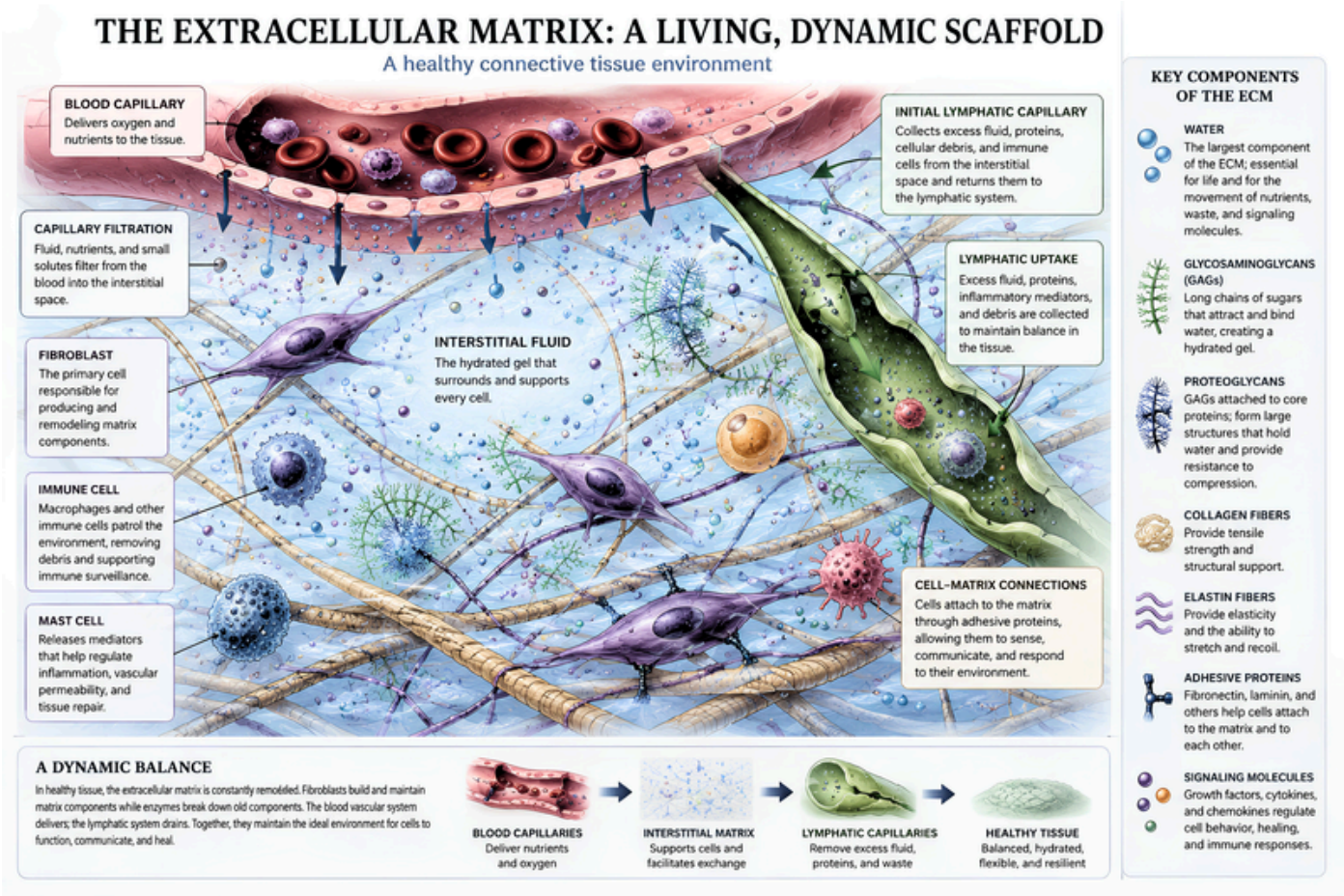
When this balance is disrupted by injury, inflammation, surgery, radiation, chronic disease, or persistent lymphatic stasis, the body responds by attempting to repair itself. Most of the time this process is remarkably successful. But when healing becomes prolonged or dysregulated, the same mechanisms designed to protect us can begin to reshape the extracellular matrix itself. The result may be tissue thickening, adhesions, restricted mobility, and fibrosis.

Understanding fibrosis, therefore, requires us to think beyond scar tissue alone. It requires us to understand the environment in which it forms.

This article begins at the microscopic level, exploring the living scaffold that surrounds every cell in the body. From there, we will follow the normal healing process, examine how fibrosis develops, and discuss its relationship with inflammation, lymphatic function, pain, and tissue remodeling. Along the way, we will explore common therapies, separate scientific evidence from popular myths, and consider how supporting healthy tissue physiology may influence long-term outcomes.

Before we can understand how fibrosis develops, we must first understand the living environment in which healing takes place.
Welcome to the extracellular matrix.





The Living Scaffold: Understanding the Extracellular Matrix

Before we can understand fibrosis, we must first understand the environment in which it develops.
Every cell in the body lives within an intricate, three-dimensional network known as the extracellular matrix (ECM). Far from being empty space or simply "connective tissue," the ECM is a dynamic, living scaffold that provides structural support, enables communication between cells, regulates the movement of fluids and proteins, and orchestrates the body's response to injury and healing.

Imagine building a house. The walls, roof, and windows are important, but without a solid framework to support them, the entire structure would collapse. The extracellular matrix serves as that framework for our tissues—but unlike the wooden frame of a house, it is alive. Every moment of every day, it is being built, remodeled, repaired, and maintained.
The ECM surrounds nearly every cell in the body, creating the environment in which those cells live and function. It provides both strength and flexibility while allowing nutrients, oxygen, waste products, immune cells, hormones, and signaling molecules to move throughout the tissue. It is also where healing begins after injury.

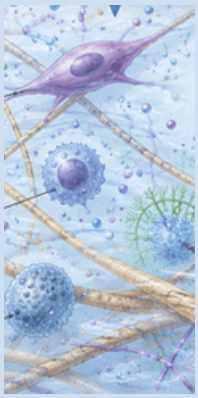
More Than Empty Space

For many years, the space between cells was thought to be little more than filler. We now know that nothing could be further from the truth.

Each component has a unique role, yet they work together as one integrated system to support tissue health.

The extracellular matrix is an active biological environment composed of:

- **Water**
- **Structural proteins, including collagen and elastin**
- **Glycosaminoglycans (GAGs)**
- **Proteoglycans**
- **Adhesive proteins such as fibronectin and laminin**
- **Fibroblasts and other resident cells**
- **Immune cells**
- **Blood capillaries**
- **Initial lymphatic capillaries**
- **A vast network of signaling molecules and growth factors**



A Living, Hydrated Environment

Water is the single largest component of the extracellular matrix and is essential for life. Rather than flowing freely through empty spaces, water is held within a delicate gel created by glycosaminoglycans (GAGs) and proteoglycans.

This hydrated gel acts like a biological sponge. It cushions tissues, resists compression, distributes mechanical forces, and creates an environment where nutrients and signaling molecules can move efficiently between the blood vessels and surrounding cells.

Because of this remarkable design, the extracellular matrix is not simply a structural scaffold—it is also the body's transportation and communication network.

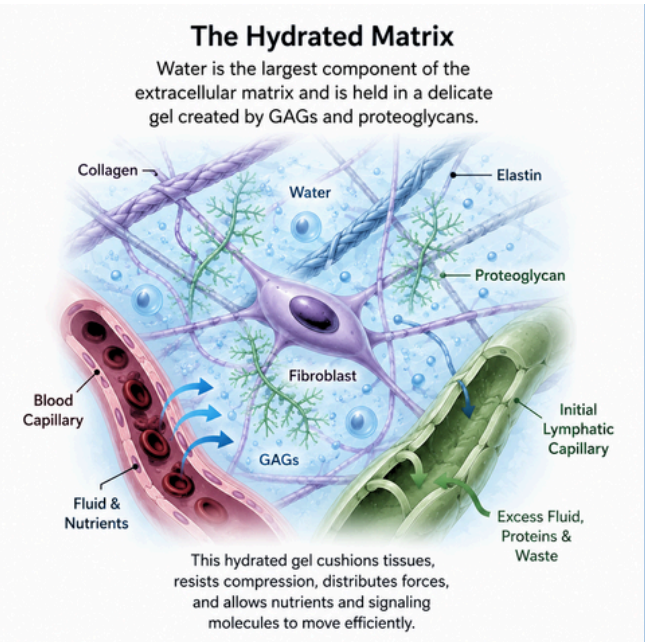


Figure 1. The hydrated extracellular matrix. Water is retained within a gel formed by glycosaminoglycans (GAGs) and proteoglycans, creating a resilient environment that cushions tissues, resists compression, and supports the movement of nutrients, signaling molecules, and excess fluid between the blood and lymphatic systems.

The Builders and the Building Materials

The extracellular matrix is continuously renewed.

Fibroblasts are the principal cells responsible for maintaining this environment.

They produce collagen, elastin, glycosaminoglycans, proteoglycans, and many of the proteins that give tissues their strength and flexibility. They also respond rapidly to injury by increasing production of these materials during the healing process.

Under healthy conditions, old matrix components are continuously broken down and replaced in a carefully regulated balance. This process, known as matrix remodeling, allows tissues to adapt to everyday stresses while maintaining normal function.

Where the Lymphatic System Fits In

Embedded within this living scaffold is another remarkable system—the initial lymphatic capillaries.

As blood capillaries deliver oxygen and nutrients to surrounding tissues, a portion of plasma filters into the extracellular matrix, becoming interstitial fluid. Most of this fluid returns directly to the bloodstream through the venous end of the capillary. The remaining fluid, along with proteins, cellular debris, inflammatory molecules, and immune cells, is collected by the initial lymphatic capillaries.

These delicate vessels help maintain the composition of the extracellular matrix by preventing excessive accumulation of fluid and proteins. In doing so, they play an essential role in preserving tissue health, regulating immune surveillance, and supporting normal healing.

The blood vascular system and lymphatic system do not function independently. Together they maintain the dynamic equilibrium of the extracellular matrix, constantly balancing fluid movement, nutrient delivery, waste removal, and immune function.

Setting the Stage for Fibrosis

A healthy extracellular matrix is remarkably adaptable. It can stretch, compress, remodel, and repair itself in response to the demands of daily life.

When injury occurs, however, this carefully balanced environment changes dramatically. Immune cells arrive, inflammatory mediators are released, fibroblasts become activated, and new collagen begins to form.

These changes are essential for healing—but if the response becomes excessive or prolonged, the same processes that protect us can begin to alter the very architecture of the extracellular matrix.

This is where the story of fibrosis begins.

When the Matrix Dries: How Dehydration May Contribute to Lymphatic Stasis and Fibrosis

The extracellular matrix functions much like a living sponge. Its hydrated gel allows cells to communicate, nutrients to diffuse, waste products to be removed, and tissues to glide smoothly during movement. This environment depends on an appropriate balance of water, proteins, and structural molecules to maintain its normal function.

When the body becomes dehydrated, the effects extend beyond thirst. Water availability within the extracellular matrix may decrease, altering the physical properties of this hydrated gel. As the matrix becomes less hydrated, its ability to cushion tissues and facilitate the movement of fluids and dissolved molecules may be reduced.

This does not mean that dehydration alone causes fibrosis. Fibrosis is a complex biological process driven primarily by injury, inflammation, and abnormal wound healing. However, dehydration may create conditions that are less favorable for efficient tissue repair and normal fluid dynamics.

The Builders and the Building Materials

The lymphatic system depends on fluid movement through the extracellular matrix. If that environment becomes less mobile—whether from dehydration, prolonged immobility, inflammation, edema, or developing fibrosis—the transport of fluid, proteins, and inflammatory mediators may become less efficient.

This can contribute to lymphatic stasis, where protein-rich fluid remains within the tissues longer than intended.

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Persistent lymphatic stasis may further alter the extracellular environment.

Protein accumulation can sustain inflammatory signaling, stimulate fibroblast activity, and encourage the deposition of collagen and other extracellular matrix components.

Over time, this may contribute to tissue thickening and fibrosis, particularly when combined with ongoing inflammation, surgical trauma, radiation injury, chronic venous or lymphatic insufficiency, or repetitive mechanical stress

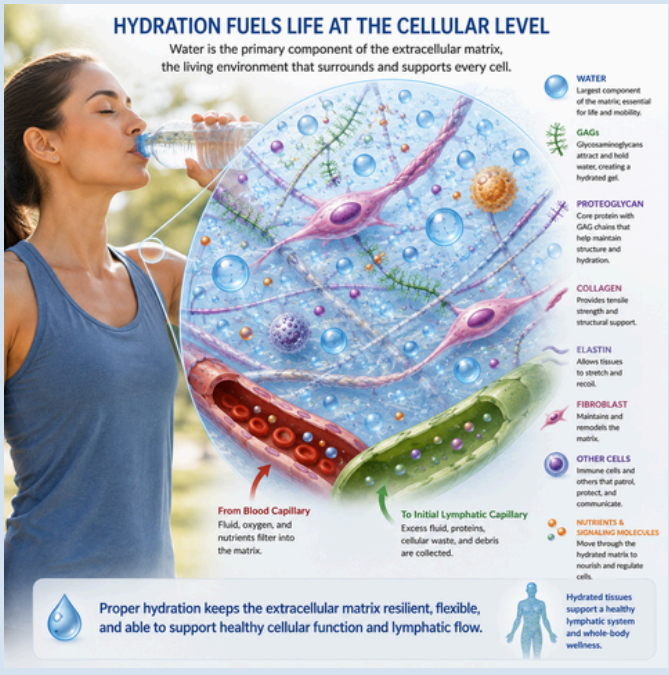
In many ways, hydration helps preserve the environment in which healthy tissue remodeling occurs.

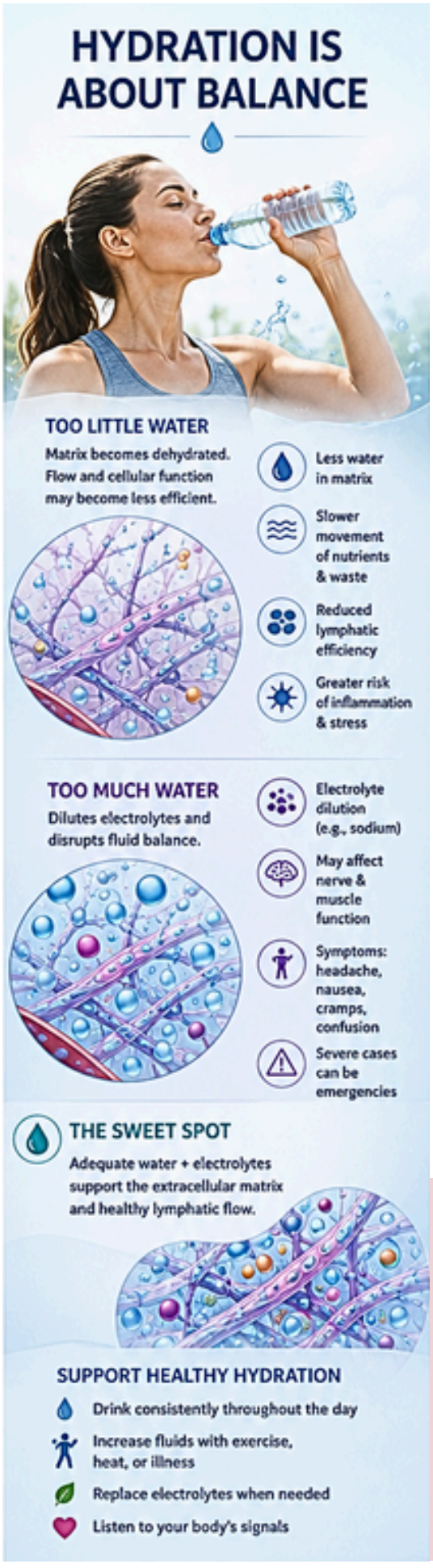
While drinking more water cannot reverse established fibrosis, maintaining adequate hydration supports the normal physiology of the extracellular matrix and complements other strategies that promote healthy tissue function, including movement, muscle activity, appropriate compression, and lymphatic care.

Rather than viewing hydration as a simple wellness recommendation, it is more accurate to see it as one of the foundational elements that helps maintain the living scaffold in which every cell and every lymphatic vessel must function.

A less hydrated matrix may contribute to:

- **Reduced diffusion of nutrients and oxygen to surrounding cells**
- **Slower movement of proteins, signaling molecules, and metabolic waste through the extracellular space**
- **Increased mechanical stiffness within the tissue environment**
- **Reduced tissue glide between fascial layers**
- **Less efficient transport of excess interstitial fluid toward the initial lymphatic capillaries**





Hydration Is About Balance, Not Excess

Hydration is often one of the first recommendations given to support lymphatic health, yet its importance goes far beyond simply drinking enough water.

Water is the largest component of the extracellular matrix (ECM)—the living environment that surrounds every cell in the body. Rather than flowing freely between cells, water is held within a hydrated gel formed by glycosaminoglycans (GAGs) and proteoglycans.

This gel provides cushioning, distributes mechanical forces, supports cellular communication, and creates the environment through which nutrients, oxygen, immune cells, and signaling molecules move.

The lymphatic system begins within this hydrated matrix. Initial lymphatic capillaries collect excess fluid, proteins, cellular debris, and inflammatory molecules from the extracellular space, helping to maintain a healthy tissue environment. When hydration is adequate, this matrix remains resilient, allowing normal fluid exchange and supporting healthy tissue function.

Hydration, however, is about balance—not volume.

Too little water may reduce the hydration of the extracellular matrix, making the movement of fluid, nutrients, and proteins through the tissues less efficient. Over time, this less favorable environment may contribute to tissue stiffness, reduced tissue glide, and lymphatic stasis, particularly when combined with inflammation, injury, or immobility.

On the other hand, drinking excessive amounts of water does not "flush" the lymphatic system or accelerate detoxification. In fact, consuming large amounts of water without adequate electrolytes can dilute sodium levels in the bloodstream, disrupting the body's carefully regulated fluid balance.

Healthy hydration depends on both water and electrolytes, including sodium, potassium, magnesium, and chloride. Together, they regulate how water moves into and out of cells and help maintain the optimal environment in which the extracellular matrix, blood vessels, and lymphatic system function.

The goal is not to drink as much water as possible—it is to maintain the healthy balance that allows every cell, every tissue, and every lymphatic vessel to perform its job effectively.

Key Takeaways

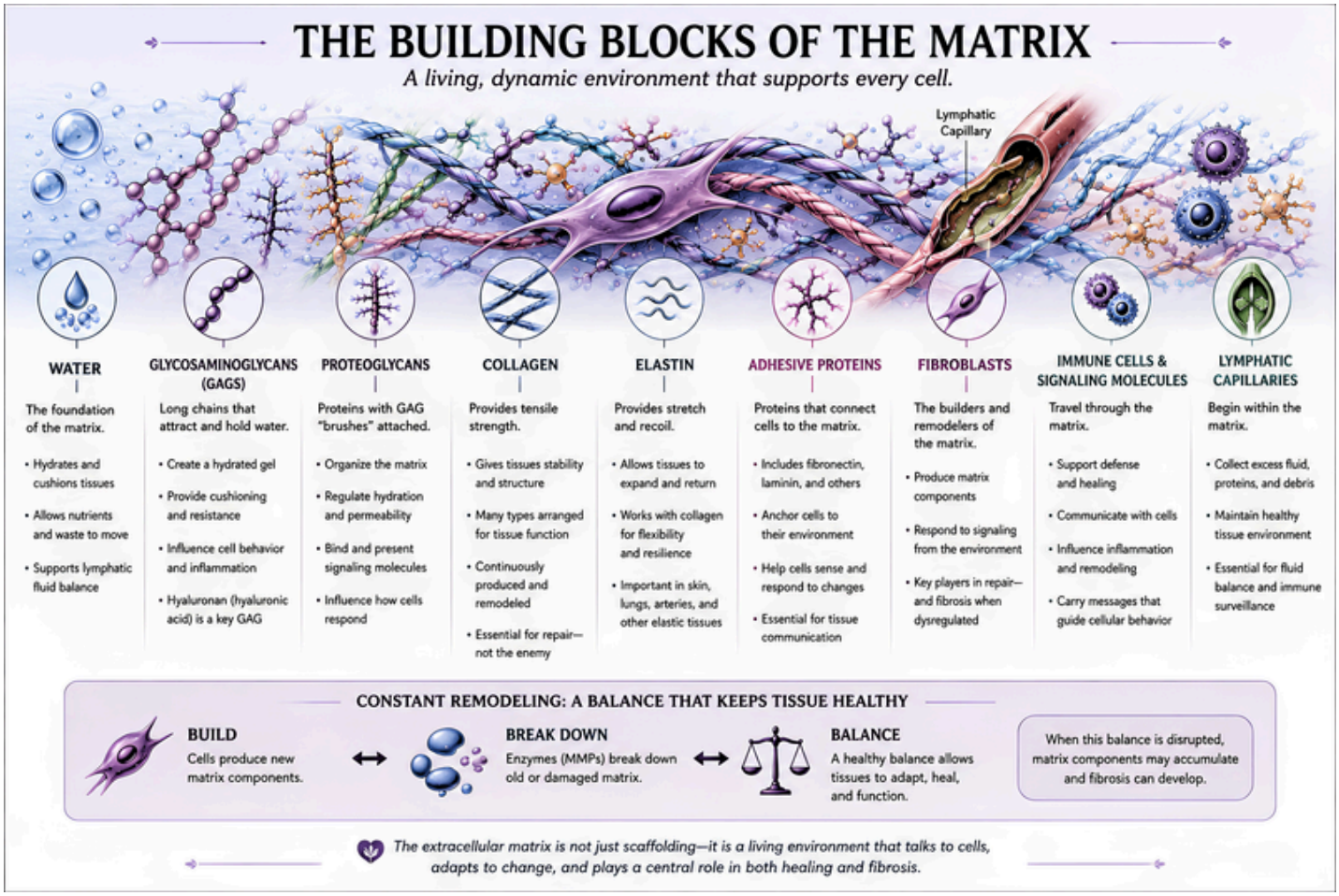
💧 **Water is the largest component of the extracellular matrix.**

🧬 **The extracellular matrix is a living, hydrated environment—not empty space.**

🌿 **The lymphatic system relies on this hydrated environment to collect excess fluid and proteins.**

⚖️ **Both dehydration and overhydration can disrupt normal tissue physiology.**

🥤 **Healthy hydration depends on the balance of water, electrolytes, movement, and overall health, not simply the number of glasses you drink each day.**



The Building Blocks of the Matrix

The extracellular matrix is not made from a single material. It is an intricate combination of water, carbohydrates, proteins, fibers, cells, and signaling molecules organized into a constantly changing three-dimensional environment. Together, these components give our tissues their shape, strength, flexibility, hydration, and ability to respond to the world around them.

Water forms the fluid foundation of this environment, while glycosaminoglycans (GAGs) and proteoglycans help attract and retain that water, creating the hydrated gel in which cells and fibers are suspended. Woven throughout this gel are structural proteins such as collagen and elastin.

Collagen provides tensile strength and stability, while elastin allows tissues to stretch and recoil. Adhesive proteins, including fibronectin and laminin, help connect cells to this surrounding network and allow them to sense changes within it.

But the matrix is not simply a collection of building materials. It contains living cells that continuously monitor, maintain, and modify their surroundings. Fibroblasts are among its primary builders and remodelers, producing many of the proteins and other components that make up the matrix. Immune cells and signaling molecules move through this environment as well, communicating information about injury, infection, inflammation, and repair.

The blood and lymphatic systems are intimately connected to this environment. Blood capillaries provide fluid, oxygen, nutrients, and other substances needed by the tissues, while initial lymphatic capillaries begin within the extracellular matrix, collecting excess interstitial fluid, proteins, cellular debris, and immune-related material. This continuous exchange helps maintain the composition and fluid balance of the tissue environment.

Perhaps most importantly, the extracellular matrix is never finished. Its components are continually being produced, broken down, reorganized, and replaced. Healthy tissue depends upon a balance between these processes. The matrix must be strong enough to provide support, flexible enough to permit movement, hydrated enough to facilitate exchange, and adaptable enough to respond when tissue is injured.

This becomes especially important when we begin talking about fibrosis. Fibrosis is not the appearance of foreign material within the tissue. Many of the substances found in fibrotic tissue—including collagen, proteoglycans, and other matrix proteins—are normal and necessary components of a healthy extracellular matrix. The problem develops when the signals controlling their production, breakdown, and organization become persistently altered.

To understand how that happens, we first need to meet the individual players that make up this remarkable living scaffold.

Glycosaminoglycans: Holding Water in the Matrix

Among the most important components of the extracellular matrix are glycosaminoglycans, commonly called GAGs.

GAGs are long chains of complex carbohydrates with a remarkable ability to attract and retain water. Their electrical charge draws water and positively charged ions into the extracellular environment, helping create the hydrated, gel-like consistency of healthy connective tissue.

This water-rich gel does much more than fill space. It helps tissues resist compression, provides cushioning around cells, and creates an environment through which nutrients, metabolites, signaling molecules, and other substances can move.

One particularly important GAG is hyaluronan, also known as hyaluronic acid. Hyaluronan has an extraordinary capacity to interact with water and plays important roles in tissue hydration, lubrication, inflammation, cell migration, wound healing, and matrix organization.

This makes GAGs especially interesting when we begin discussing edema, inflammation, lymphatic stasis, and fibrosis. The composition and behavior of the extracellular matrix can change significantly during disease or injury, and GAGs participate in that changing environment.

Proteoglycans: Organizing the Hydrated Gel

GAGs frequently attach to proteins, creating larger structures called proteoglycans.

One way to visualize a proteoglycan is as a tiny bottle brush. The central protein forms the handle while numerous GAG chains extend outward like bristles. Those chains attract water, allowing proteoglycans to occupy a surprisingly large volume within the extracellular space.

Proteoglycans help organize the matrix and influence its hydration, permeability, and mechanical properties. They also interact with growth factors and signaling molecules, helping regulate how cells respond to injury and changes within their environment.

The extracellular matrix is therefore not simply scaffolding around cells. It is part of the communication system that tells cells what is happening around them.

Collagen: Strength Without Rigidity

Collagen is perhaps the best-known component of connective tissue—and it will become particularly important as we explore fibrosis.

Collagen fibers provide tensile strength. They allow tissues to withstand pulling and mechanical forces without tearing. Different types of collagen are organized differently depending upon the needs of the tissue. Skin, tendons, fascia, blood vessels, cartilage, and organs all rely on collagen for structural integrity.

But collagen itself is not pathological. Healthy tissue needs collagen.

Every day, collagen is being produced, modified, organized, broken down, and replaced as part of normal tissue maintenance.

Problems arise when this carefully regulated process becomes disrupted and collagen production and deposition begin to exceed appropriate breakdown and remodeling.

That distinction becomes extremely important when discussing fibrosis.

Fibrosis is not simply the presence of collagen—it is abnormal extracellular matrix remodeling.

Elastin: Allowing Tissue to Give and Return

If collagen provides strength, elastin provides recoil.

Elastin fibers allow tissues to stretch and then return toward their original shape. This property is particularly important in tissues that repeatedly expand and contract, including the skin, lungs, arteries, and other elastic connective tissues.

Collagen and elastin therefore work together. One provides resistance to excessive stretching while the other provides flexibility.

Healthy connective tissue requires both stability and movement.

Adhesive Proteins: Connecting Cells to Their Environment

Proteins such as fibronectin and laminin help connect cells to the extracellular matrix.

These proteins act somewhat like biological anchors, allowing cells to attach to their surroundings. But these connections are not merely structural.

Through specialized receptors on the cell membrane, cells can sense mechanical tension, stiffness, chemical signals, and changes within the extracellular matrix.

Those signals can influence how a cell behaves—including whether it moves, divides, produces matrix components, or participates in tissue repair.

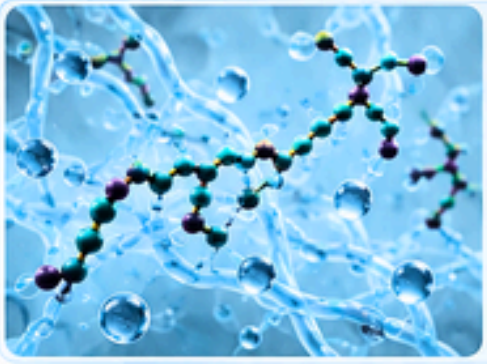
This ability of cells to sense and respond to mechanical forces is part of a process called mechanotransduction. The matrix talks to the cells.

And the cells respond by changing the matrix.

This continuous conversation is one of the reasons the extracellular matrix should be viewed as a living environment rather than passive connective tissue.

THE BUILDING BLOCKS OF THE *Matrix*

The **extracellular matrix** is a dynamic network of compounds that support, protect, and communicate with our cells.

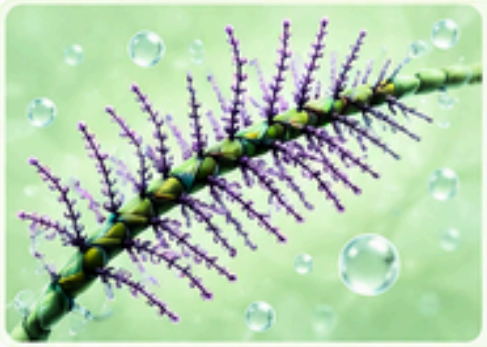


1. GLYCOSAMINOGLYCANS (GAGS)

Holding Water in the Matrix

- Long chains of complex carbohydrates that attract and hold water.
- Create a hydrated, gel-like consistency that cushions, protects, and nourishes tissue.
- Help tissues resist compression and allow nutrients, metabolites, and signals to move.

KEY EXAMPLE:
Hyaluronan
A GAG with an extraordinary capacity to hydrate, lubricate, and support healing.



2. PROTEOGLYCANS

Organizing the Hydrated Gel

- GAGs attach to proteins, forming larger structures called proteoglycans.
- These structures organize the matrix, influence hydration, permeability, and mechanical properties.
- Interact with growth factors and signaling molecules, helping regulate cell behavior and tissue repair.

VISUALIZE IT:
Like a tiny bottle brush—GAG chains extend outward from a protein core, attracting water and filling space.

3. COLLAGEN

Strength Without Rigidity

- The most abundant protein in connective tissue and a key component in fibrosis.
- Provides tensile strength—resisting pulling and mechanical forces.
- Different types are found in skin, tendons, fascia, blood vessels, cartilage, and organs.

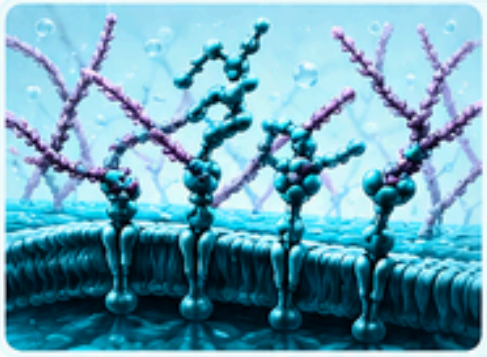
REMEMBER:
Collagen itself is not the problem. Problems arise when production or deposition becomes excessive or disorganized.

4. ELASTIN

Allowing Tissue to Give and Return

- Elastin fibers allow tissues to stretch and then return to their original shape.
- Essential in tissues that repeatedly expand and contract—skin, lungs, arteries, and more.
- Works with collagen: strength + elasticity = healthy, functional tissue.

FUNCTION:
Provides recoil and flexibility, preventing tissues from being too rigid or too loose.



5. ADHESIVE PROTEINS

Connecting Cells to Their Environment

- Proteins like fibronectin and laminin act as biological anchors.
- Connect cells to the extracellular matrix and to surrounding cells.
- Through receptors, cells sense mechanical tension, chemical signals, and change their behavior.
- This two-way communication—mechanotransduction—keeps the matrix dynamic and responsive.

WHY IT MATTERS:
Cells and the matrix are in a constant conversation that influences healing, movement, and overall tissue health.

TOGETHER, THESE COMPOUNDS CREATE A LIVING, ADAPTIVE ENVIRONMENT
that supports structure, movement, communication, and healing.

Lymphatic Restoration Therapy®
AI-generated simulation for educational purposes only.

The Matrix Is Constantly Being Remodeled

Even healthy tissue is never truly static.

Old or damaged matrix components are continuously broken down while new components are produced and organized. Specialized enzymes, including matrix metalloproteinases (MMPs), participate in breaking down extracellular matrix proteins. Their activity is carefully regulated, in part by molecules known as tissue inhibitors of metalloproteinases (TIMPs).

At the same time, fibroblasts and other cells produce new matrix components.

The result is an ongoing balance:

Production ↔ Breakdown ↔ Remodeling

This balance allows tissues to adapt to movement, mechanical stress, injury, aging, and changing physiological demands.

When remodeling remains balanced, tissue retains its strength, flexibility, hydration, and mobility.

When that balance becomes persistently disrupted, however, extracellular matrix components may begin to accumulate.

And one cell becomes particularly important in determining what happens next.

Meet the Fibroblast

Scattered throughout the extracellular matrix are specialized cells called fibroblasts.

Fibroblasts are among the primary caretakers and builders of connective tissue. They produce many of the components we have just discussed—including collagen, elastin, GAGs, proteoglycans, and other matrix proteins.

Under normal circumstances, fibroblasts quietly maintain and remodel the extracellular environment.
But when tissue is injured, their behavior changes.

Chemical signals released during inflammation tell fibroblasts that the tissue needs repair. They become more active, increase matrix production, and help rebuild the damaged area.

This response is essential for survival.
Without fibroblasts and collagen production, wounds would not close and damaged tissues could not regain their structural integrity.

The problem is not that fibroblasts produce collagen.
The problem begins when the signals telling them to keep building do not turn off.

And that distinction brings us one step closer to understanding fibrosis.

If Fibrosis Contains Collagen, Should I Stop Taking Collagen Supplements?

This is an excellent question—and it highlights an important distinction between the collagen we consume and the collagen our tissues produce.

The short answer is: having fibrosis does not automatically mean you need to stop dietary collagen.

Fibrosis is not caused by collagen from food or supplements simply traveling through the bloodstream and "settling" into an area of tissue.

Dietary Collagen and Tissue Collagen Are Not the Same Thing

When collagen is eaten—whether from food, bone broth, gelatin, or a collagen supplement—it enters the digestive system just like other dietary proteins. Digestive enzymes break it down primarily into amino acids and small collagen-derived peptides. These are absorbed through the intestine and enter the circulation.
The body can then use those amino acids as raw materials for many different purposes, including the synthesis of proteins throughout the body.

There is no direct pathway that looks like:

Collagen supplement → bloodstream → fibrotic tissue → more fibrosis

Fibrotic collagen is produced within the affected tissue.

So Where Does the Collagen in Fibrosis Come From?

Remember the fibroblast we just introduced?
Under normal circumstances, fibroblasts continuously maintain the extracellular matrix.

When tissue is injured, inflammatory and mechanical signals activate fibroblasts and other cells involved in repair. Some fibroblasts acquire a more contractile, matrix-producing phenotype often described as myofibroblasts.

These cells increase production of collagen and other extracellular matrix components to stabilize and repair damaged tissue.

That is normal healing.

The problem develops when the signals encouraging matrix production remain active longer than they should.

Persistent inflammation, repeated tissue injury, altered mechanical signaling, impaired matrix breakdown, and in conditions such as lymphedema—chronic changes in the tissue environment can contribute to continued extracellular matrix deposition and remodeling.

Over time:
Persistent signaling → fibroblast/myofibroblast activity → increased matrix production + altered breakdown → collagen accumulation and organization → tissue stiffening → fibrosis

So fibrosis is much more accurately described as a problem of dysregulated tissue remodeling than simply a problem of "too much collagen."

What About Lipedema and Lymphedema?

This distinction becomes especially important for people living with lipedema or lymphedema.

Fibrotic changes in these conditions occur within a complex tissue environment involving chronic inflammation, extracellular matrix remodeling, adipose/connective-tissue changes, altered fluid and protein handling, and—in lymphedema particularly—impaired lymphatic transport.

Simply removing collagen from the diet does not switch off those cellular processes.

Likewise, taking a collagen supplement should not be interpreted as directly "feeding" fibrosis.

Does That Mean Everyone Should Take Collagen

No. That's a different question.

Evidence for collagen supplementation varies depending upon the reason it is being used, and supplements aren't necessary for everyone. A person's overall protein intake, nutritional needs, medical conditions, allergies, medications, kidney function, and reason for supplementation should all be considered.

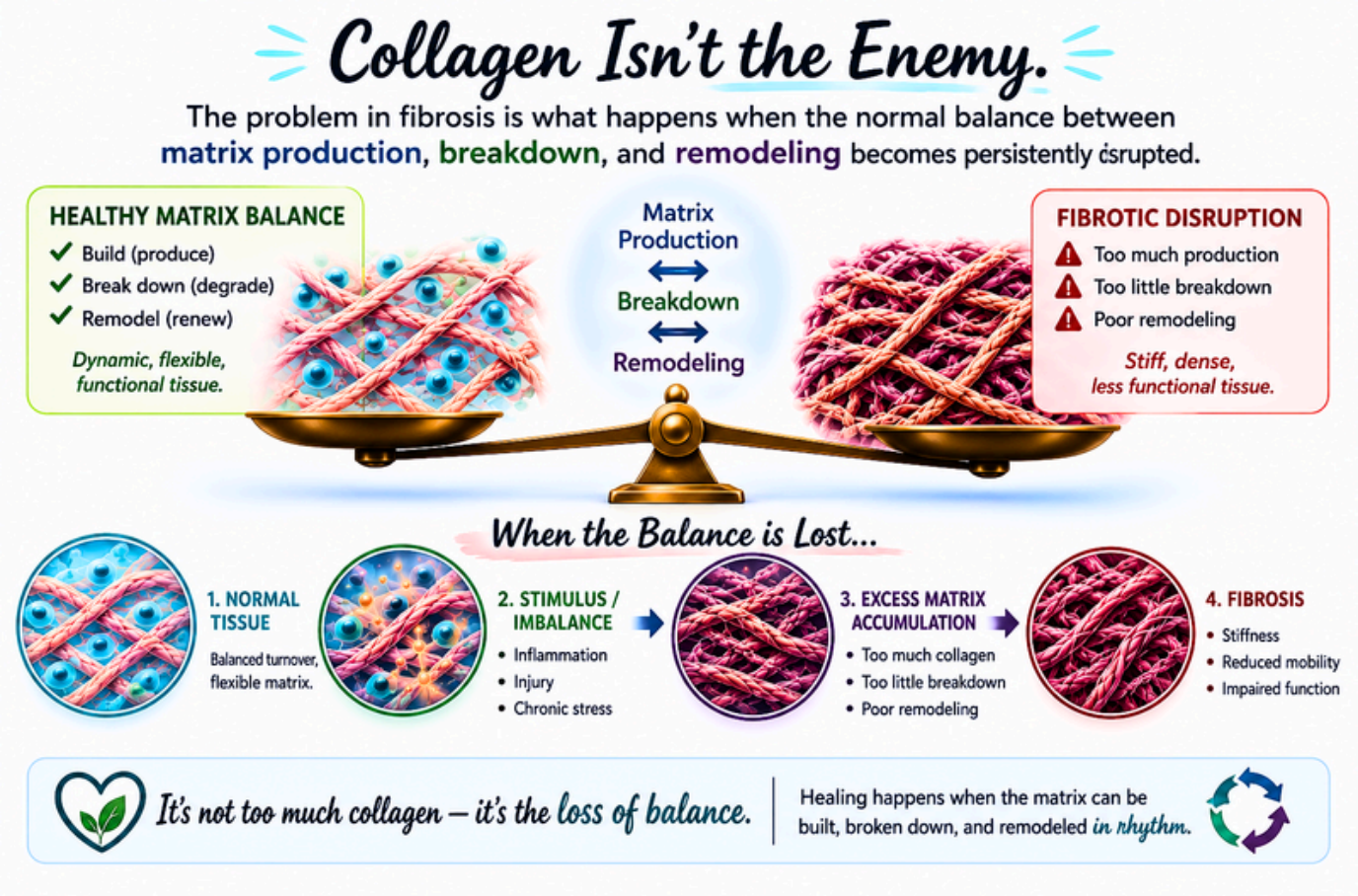
But "I have fibrosis, therefore I shouldn't consume collagen" is based on a misunderstanding of how fibrosis develops.

The Important Distinction

Dietary collagen provides nutrients. Fibrotic collagen is manufactured locally by cells responding to biological signals.

And that brings us back to one of the central themes of this article:

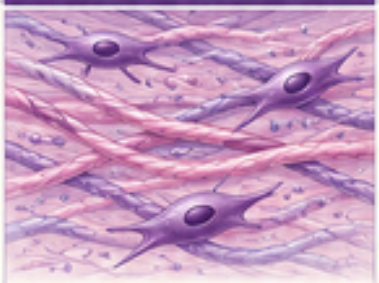
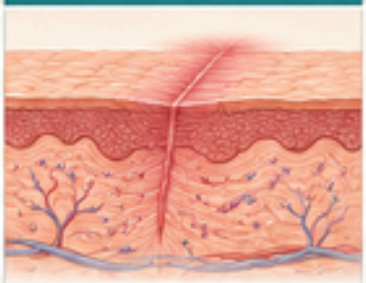
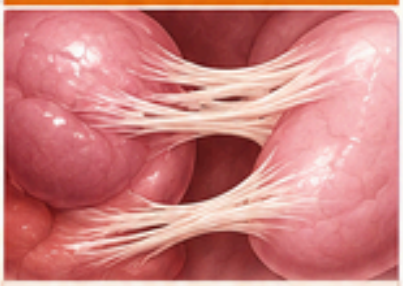
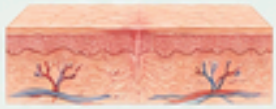
Collagen itself isn't the enemy. The problem in fibrosis is what happens when the normal balance between matrix production, breakdown, and remodeling becomes persistently disrupted.



3 MAIN TYPES OF FIBROSIS

Tissue Fibrosis • Scar • Adhesion

All three involve changes in collagen and the extracellular matrix, but they occur in different ways, for different reasons, and have different effects on the body.

	TISSUE FIBROSIS	SCAR	ADHESION
			
	Diffuse buildup of extracellular matrix within a tissue, leading to thickening and stiffness.	The body's organized response to a specific injury to repair damaged tissue.	Fibrous bands that abnormally connect tissues or organs that should move freely.
WHAT HAPPENS?	Matrix accumulates and tissue stiffens. 	Injured tissue is repaired. 	Separate tissues become connected. 
TYPICAL TRIGGER	Chronic/repeated injury or inflammation. 	Defined injury or incision. 	Injury/inflammation between adjacent surfaces. 
COLLAGEN'S ROLE	Persistent/altered deposition and remodeling. 	Restores structural integrity. 	Forms a fibrous bridge. 
NORMAL OR ABNORMAL?	Usually pathological when progressive. 	Usually a normal healing response. 	Generally an unwanted consequence of healing. 
MAIN EFFECT	Thickening and stiffness. 	Repairs damaged tissue. 	Restricts normal tissue glide and movement. 

THE BIG PICTURE

One person can have all three processes occurring in the same region. Understanding the differences helps us choose the safest and most effective approaches for care and recovery.

Fibrosis Is Not Just Scar Tissue

Understanding Tissue Fibrosis, Scars, and Adhesions:

The word fibrosis is often used interchangeably with scar tissue.

In everyday conversation, that may seem reasonable, because all three involve changes in collagen and the extracellular matrix. Biologically, however, tissue fibrosis, scars, and adhesions are different manifestations of tissue repair and remodeling.

All three involve many of the same players we have already met: fibroblasts, collagen, extracellular matrix proteins, inflammatory mediators, immune cells, and mechanical signaling.

What differs is where the process occurs, why it occurs, how the collagen is organized, and whether the normal healing response successfully resolves.

1. Tissue Fibrosis — When Remodeling Becomes Persistent

Tissue fibrosis is the excessive or abnormal accumulation and remodeling of extracellular matrix within a tissue.
It is not necessarily associated with a visible wound or incision.

Instead, fibrosis can develop gradually in response to persistent inflammation, chronic injury, radiation, altered mechanical forces, impaired lymphatic function, or disease processes that repeatedly stimulate tissue repair.

Fibroblasts and myofibroblasts remain activated and continue producing collagen and other matrix components. At the same time, normal matrix breakdown and remodeling may become altered.

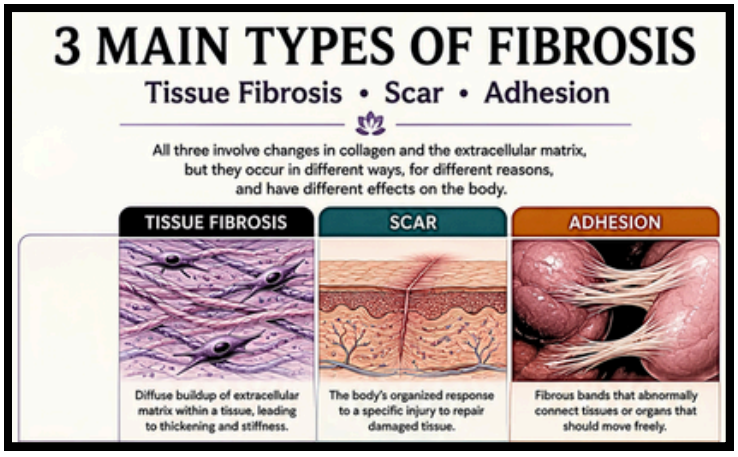
The result is a matrix that becomes denser, stiffer, and less adaptable.

In superficial tissues, this may be experienced as thickening, firmness, decreased tissue mobility, or reduced tissue glide.

In organs, fibrosis can progressively alter normal architecture and interfere with function.

This is particularly relevant in lymphedema, where chronic lymphatic dysfunction is associated with inflammation, adipose deposition, extracellular matrix remodeling, and progressive fibrosis.

Fibrotic changes can also occur in lipedema, although the underlying biology and tissue changes are not identical to those seen in lymphedema.



2. Scar Tissue — The Body's Repair Patch

A scar is the body's organized response to a specific tissue injury.

When tissue is cut, torn, burned, or surgically disrupted, the body must rapidly restore structural integrity. Fibroblasts produce collagen and other matrix materials to bridge and reinforce the injured area.

Early scar tissue is quite different from a mature scar. Initially, collagen fibers are relatively disorganized and the tissue may be vascular, raised, firm, or sensitive. Over the following months, the matrix continues to remodel.

Collagen fibers reorganize along lines of mechanical stress, cellularity and vascularity generally decrease, and the scar usually becomes softer and more mobile.

A scar therefore represents normal fibrosis as part of wound repair.

The goal isn't to eliminate all scar tissue. Without scar formation, many wounds could not regain sufficient structural strength.

Problems occur when scar formation becomes excessive or abnormal, as may occur with hypertrophic scars or keloids, or when deeper scar tissue restricts movement and normal tissue mechanics.

3. Adhesions — When Tissues Heal Together

An adhesion is different.

Instead of collagen primarily reinforcing an injured tissue, fibrous tissue forms an abnormal connection between surfaces or structures that normally should remain separate or glide independently.

Following surgery, trauma, infection, or inflammation, fibrin may temporarily connect adjacent surfaces as part of the early healing response. Normally, much of this temporary matrix is broken down as healing progresses.

If it persists and becomes organized, fibroblasts can migrate into the area and deposit collagen, transforming that temporary connection into a more permanent fibrous adhesion.

This is particularly important within the abdomen and pelvis, where organs normally need to move and glide relative to one another. Adhesions can also develop around tendons, fascial planes, nerves, joints, and surgical sites.

The problem isn't simply that collagen exists there.
The problem is that two structures that were designed to move independently have become mechanically connected.

Three Different Outcomes of Matrix Remodeling But They Can Overlap

This is where the story becomes more complicated—and clinically more interesting.

A surgical incision creates a scar, but the tissues beneath that incision may also develop fibrosis. Surgery within the abdomen may produce adhesions between neighboring structures. Radiation may produce progressive tissue fibrosis around an area that also contains surgical scars and adhesions.

One person can therefore have all three processes occurring in the same region.
And that distinction matters when we begin talking about treatment.

A mature surgical scar, diffuse lymphedema-related fibrosis, and an internal abdominal adhesion should not automatically be approached as though they are the same tissue problem. Their biology, location, mechanical behavior, risks, and potential response to treatment can be very different.

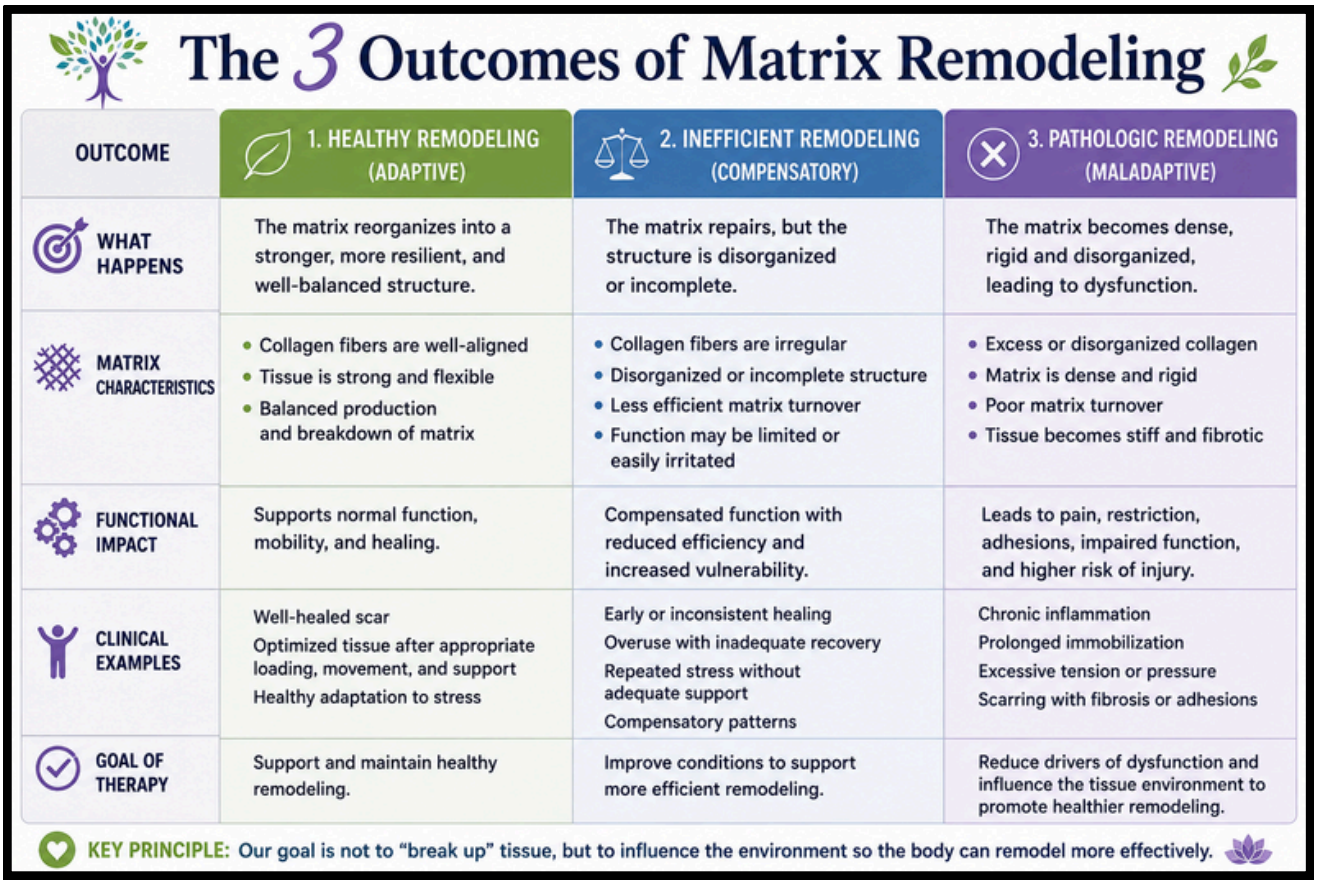
That gives us a strong transition into the next question:

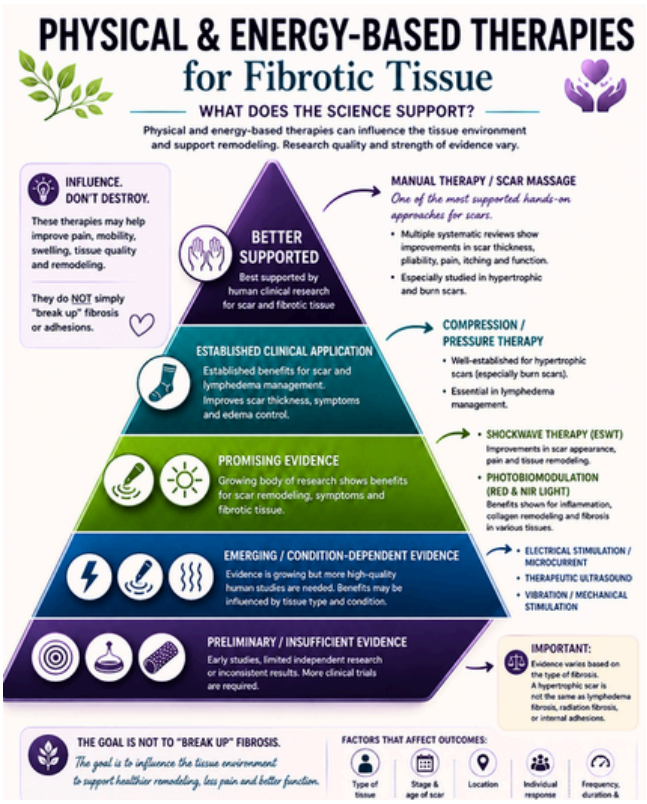
Can Fibrosis, Scars, and Adhesions Actually Be "Broken Up"?

That's where I think we should challenge the common language around aggressive massage, fascia blasting, gua sha, cupping, scar mobilization, and manual therapy—and explain instead what we may realistically be changing in the surrounding matrix, tissue mobility, fluid environment, and nervous system response.

Physical & Energy-Based Modalities: What Does the Science Say? Treating Fibrotic Tissue: Choosing the Right Approach

There are many therapeutic modalities that are promoted as methods to "break up" fibrotic tissue, scar tissue, or adhesions. Manual therapy, compression, vibration, ultrasound, electrical stimulation, photobiomodulation, shockwave therapy, negative-pressure techniques, Deep Oscillation, textured materials, and other approaches have all been used with the goal of influencing restricted or fibrotic tissue.





Physical and Energy-Based Modalities in Fibrosis Management

Once we understand fibrosis as a process of altered extracellular matrix remodeling, the way we think about treatment begins to change.

For decades, therapists and patients have used phrases such as “breaking up scar tissue,” “breaking down fibrosis,” or “releasing adhesions.” These descriptions are easy to visualize, but they may oversimplify what is actually occurring within living tissue.

Fibrotic tissue is not an inert substance that simply needs to be mechanically crushed or broken apart.

It is a biologically active environment containing collagen, elastin, proteoglycans, glycosaminoglycans, fibroblasts, immune cells, blood vessels, lymphatic vessels, nerves, interstitial fluid, and signaling molecules.

Treatment therefore becomes less about destroying fibrosis and more about determining whether we can influence the tissue environment in ways that support better function and, where biologically possible, healthier remodeling.

There Is No Single “Fibrosis Treatment"

A wide variety of physical and energy-based modalities are used in the management of scars and fibrotic or restricted tissues,

The scientific support for these interventions varies considerably.

Some have decades of clinical use and human research. Others have promising mechanistic or preliminary clinical evidence but require substantially more investigation.

Most importantly, evidence supporting a treatment for one type of tissue cannot automatically be applied to another.

A hypertrophic burn scar is not equivalent to lymphedema-associated fibrosis, radiation fibrosis, postsurgical fibrosis, lipedema-associated fibrotic change, or an internal adhesion.

The first step in treatment should therefore always be identifying what we are actually trying to change.

Manual Therapy: Remodel, Don't Break

Manual therapy is one of the most familiar approaches to scar and restricted-tissue management.

Scar massage and other forms of manual treatment have been studied for outcomes including pain, itching, scar thickness, pliability, mobility, and function.

The evidence is encouraging for some scar populations, although treatment protocols and study quality vary considerably.

The effects of manual therapy are unlikely to be explained simply by physically tearing collagen fibers apart.

Physical & Energy-Based Modalities: What Does the Science Say? Treating Fibrotic Tissue: Choosing the Right Approach

Fibroblasts do much more than produce collagen. These cells can sense the physical characteristics of the extracellular matrix around them.

Stretch, compression, shear forces, vibration, and changes in matrix stiffness can be detected through cellular connections with the surrounding matrix.

These mechanical forces are translated into biochemical signals through a process known as mechanotransduction.

Those signals can influence cellular behavior, including migration, proliferation, inflammatory communication, collagen production, matrix-degrading activity, and the organization of newly formed extracellular matrix.

The most supported modality continues to be manual therapy. Physical modalies such as current, vibration and deep tissue oscillation should be applied by skilled clinicians trained in that specific modality and most importantly should remain withing their scope of practice. These treatments are adjuncts to manual therapy and compression which remain at the top of the list.

This gives us a different way to think about manual therapy. When we stretch, compress, glide, or gently mobilize tissue, our hands may be doing something considerably more sophisticated than physically "breaking up" collagen.

We are changing the mechanical environment surrounding the cells. Pressure, direction, duration, repetition, and tissue movement all provide mechanical information. The response may include changes in tissue mobility, fluid dynamics, sensory input, and cellular signaling.

Whether those signals ultimately contribute to meaningful long-term remodeling depends upon many factors, including the condition and age of the tissue, inflammation, vascular and lymphatic function, mechanical loading, treatment dose, and the individual's healing response.

Perhaps the most useful way to view manual therapy, then, is not as a battle between our hands and resistant tissue, but as a conversation with a living biological system.

Our hands provide mechanical input; cells interpret that information and determine the biological response. This is why more pressure is not necessarily more effective.

The goal may not be to overpower fibrotic tissue, but to provide an appropriate mechanical stimulus that encourages movement, adaptation, and—where the biology permits—healthier extracellular matrix remodeling.

Manual Therapy Approaches Used for Fibrotic and Scarred Tissue

Manual therapy is not a single technique. Different forms of touch create different combinations of compression, tension, shear, stretch, oscillation, and tissue displacement.

Research suggests that several manual approaches may improve the mobility, pliability, symptoms, or function of scarred and restricted tissues, although the strength of evidence varies considerably.

Manual therapy should be performed by a licensed professional working within their scope of practice and depending on their level of training.

Working on healthy tissue that is healing normally may be different than working with someone dealing with radiation or burn fibrosis or trauma-related fibrosis. This also holds true for someone with lipedema or lymphedema.

Scar Massage and Scar Mobilization

Scar massage has some of the most direct manual-therapy research for abnormal scar tissue, particularly hypertrophic burn scars.

Techniques studied include:

- Gentle scar mobilization
- Circular massage
- Longitudinal and transverse mobilization
- Friction massage
- Effleurage
- Petrissage
- Oscillatory massage

Systematic reviews have reported improvements in outcomes including scar pliability, thickness or height, pain, itching, and function, although protocols vary and the overall quality of evidence is not uniformly strong.

Interestingly, different techniques may have different effects: friction and oscillatory approaches have been studied for scar function, while effleurage and petrissage have been associated with changes in scar appearance and pain.

Myofascial Release

Myofascial release generally uses low-load, sustained mechanical input rather than aggressive friction. The intention is to improve mobility between fascial and connective-tissue layers and reduce restrictions affecting movement.

A randomized trial involving women following breast-cancer surgery and radiotherapy found that a four-week myofascial-release program improved pain, shoulder range of motion, and function.

This population is particularly relevant because surgery and radiation can produce connective-tissue retraction and fibrotic changes.

However, the study measured clinical function rather than demonstrating that fibrosis itself had been histologically removed or reversed.

Soft-Tissue Mobilization

Soft-tissue mobilization is a broader category that may include:

- Skin rolling
- Tissue gliding
- Cross-fiber mobilization
- Fascial mobilization
- Sustained pressure
- Gentle tissue stretching
- Mobilization between tissue planes

These techniques may help improve tissue glide, mobility, range of motion, and symptoms, particularly when restriction develops around a healed scar.

Again, it is more accurate to describe these techniques as mobilizing and mechanically loading the tissue environment than as physically tearing fibrosis apart.

Manual Lymphatic Drainage

Manual lymphatic drainage occupies a somewhat different category.

MLD is not primarily intended to mechanically remodel collagen. Its potential contribution is through the fluid and lymphatic environment surrounding fibrotic tissue.

In lymphedema, chronic lymphatic dysfunction can contribute to inflammation and progressive extracellular-matrix remodeling.

MLD may assist with edema and symptoms in some patients, although systematic reviews have produced mixed findings regarding the additional benefit of MLD when it is added to comprehensive decongestive treatment.

For fibrotic tissue associated with lymphatic dysfunction, this distinction is important:

Manual lymphatic drainage may address the environment contributing to fibrosis without directly “breaking up” collagen.

The key is integrating it prior to fibrosis settling in. In combination with effective compression and movement therapies, MLD is highly effective in assisting wit fibrosis for lymphedema, post surgery and lipedema

Instrument-Assisted Soft-Tissue Mobilization: When More Is Not Better

Tools can also provide mechanical input to the extracellular matrix. Instrument-assisted soft-tissue mobilization (IASTM), including techniques performed with rigid scraping or mobilization tools, is frequently promoted for scar tissue, fascial restrictions, and adhesions.

However, this is where an important principle must be remembered:

More force does not necessarily mean more remodeling. Some research has reported improvements in range of motion, pain, and patient-reported function following IASTM.

However, the evidence remains mixed, and studies generally do not demonstrate that these instruments physically "break apart" fibrosis or remove adhesions. Improvements in mobility or symptoms should not automatically be interpreted as evidence that fibrotic collagen has been mechanically destroyed.

Redness, bruising, swelling, and soreness are also not measures of treatment success. These responses demonstrate that the tissue has received a mechanical stimulus—and, when excessive, may indicate tissue and vascular injury.

The goal should not be to create visible evidence that something was done. The goal is improved tissue mobility, decreased symptoms, better function, and an appropriate recovery response.

Special Considerations When Lymphatic Function Is Compromised

This becomes particularly important when treating fibrotic tissue in an area where lymphatic transport is reduced or impaired.

Mechanical treatment that produces significant bruising or tissue injury initiates an inflammatory response. Blood vessels become more permeable, additional fluid and inflammatory mediators enter the extracellular space, and cellular debris must ultimately be cleared from the tissue environment. A normally functioning lymphatic system participates in that clearance.

When lymphatic transport is already compromised, the tissue may have less capacity to efficiently manage an additional inflammatory and fluid burden. Persistent edema, accumulated proteins, inflammatory signaling, and altered immune activity are themselves associated with progressive extracellular matrix remodeling in chronic lymphedema.

This creates an important clinical consideration: a treatment intended to improve fibrotic tissue should not unnecessarily create another inflammatory challenge for a tissue environment already struggling with fluid and inflammatory clearance.

This does not mean that instrument-assisted techniques cannot be used around fibrotic or lymphatically compromised tissue. It means that the dose, pressure, tissue condition, stage of healing, and response following treatment matter tremendously.

Gentler mechanical input may sometimes be more appropriate than aggressive scraping or bruising techniques. Treatment should be followed by reassessment—not simply of how the tissue feels immediately afterward, but of its response over the following hours and days.

Does mobility improve?

Does the tissue remain softer?

Does pain decrease?

Does swelling increase?

Does the area become warmer, more tender, or more congested?

How quickly does the tissue recover?

These responses provide considerably more useful information than redness or bruising alone.

When fibrosis and lymphatic dysfunction coexist, the objective is not to overpower the tissue. It is to provide enough mechanical stimulus to encourage mobility and adaptation while respecting the tissue's ability to manage inflammation, fluid, and recovery.

IASTM can help improve pain, mobility, and range of motion, but fibrotic, scarred, or lymphatically compromised tissue often needs a gentler approach. Too much pressure can create bruising, swelling, pain, and added inflammation in an already challenged tissue environment. The goal is not more injury, but the right mechanical stimulus to support mobility and healthier remodeling. More force does not necessarily mean better remodeling.

From Clinical Instruments to Everyday Tissue Tools

IASTM is only one example of using an external tool to deliver mechanical input to the extracellular matrix. Similar principles apply to Gua Sha, scraping tools, suction cups, textured rollers, massage balls, vibration tools, and textured compression materials.

Each creates a different mechanical stimulus—compression, shear, traction, negative pressure, vibration, or repeated changes in pressure across the tissue.

The important consideration is dose. A tool that improves mobility or comfort when applied gently may produce a very different biological response when used aggressively enough to cause bruising, petechiae, significant redness, swelling, or prolonged soreness.

This distinction becomes especially relevant when working with scarred, fibrotic, edematous, or lymphatically compromised tissue, where additional inflammation and fluid accumulation may be poorly tolerated.

Rather than asking, "Which tool breaks up fibrosis best?", we can ask a much more useful question:
What type of mechanical information does this tool provide—and is that stimulus appropriate for this particular tissue?

This is where clinical reasoning and proper training come into play. Someone trained in working with lymphatic conditions will best be able to assess the outcomes, complications and reactions and modify th treatment accordingly

That gives us several very different categories to explore:

- Gua Sha & scraping tools — primarily shear and compression; useful to discuss the difference between gentle mobilization and intentionally producing petechiae.
- Cupping & negative pressure — lifts tissue rather than compressing it, potentially influencing tissue glide and superficial fluid dynamics; aggressive suction and bruising deserve separate consideration.
- Textured balls, rollers & handheld tools — provide localized, variable mechanical pressure and may allow patients to self-dose treatment.
- Textured compression & foam — provides prolonged, low-level variations in pressure rather than a brief aggressive stimulus.
- Vibrational tools — add repetitive mechanical oscillation, bringing us back to mechanotransduction and the question of whether repeated low-level stimulation can influence the tissue environment.

The tool is only the delivery system. The tissue response determines whether the dose was appropriate.

A stainless-steel instrument, cup, textured ball, therapist's hands, or compression garment can all provide mechanical information.

What differs is the direction, intensity, duration, frequency, and distribution of that stimulus.

For fibrotic tissue—particularly when lymphatic dysfunction or chronic inflammation is present—the objective should be enough stimulus to encourage mobility and adaptation without unnecessarily adding tissue injury and inflammatory burden.

Positive Pressure, Negative Pressure & Texture

Not All Mechanical Input Is the Same

Once we begin viewing manual therapy through the lens of mechanotransduction, many of the tools used for scars and fibrotic tissue start to make more sense.

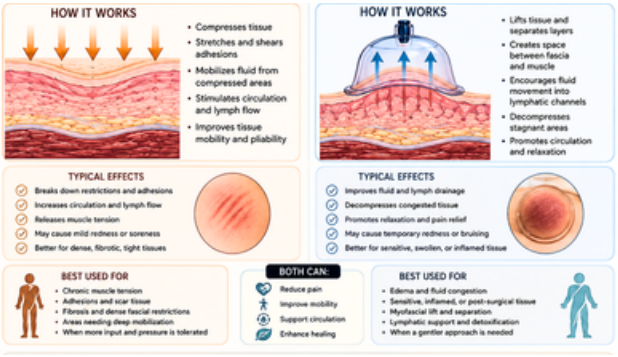
Hands, stainless-steel instruments, Gua Sha tools, suction cups, textured balls, rollers, vibration devices, foam, and textured compression garments may look very different, but they share one important characteristic:

They apply mechanical information to a living extracellular matrix.

What differs is the type of mechanical stimulus being delivered.

- Some tools push into the tissue.
- Others lift tissue away from underlying structures.
- Some create shear between tissue layers.
- Some provide repetitive vibration.
- Others provide gentle variations in pressure over many hours.

Understanding these differences may be more useful than simply asking which tool is best for fibrosis.



Positive Pressure: Compression, Shear & Tissue Displacement

Massage, IASTM, Gua Sha, rollers, massage balls, and many handheld tools primarily apply some combination of positive pressure, compression, shear, and tissue displacement.

When appropriately dosed, these forces may improve tissue mobility, temporarily alter tissue stiffness, influence sensory input, and provide mechanical signals to cells within the extracellular matrix.

The amount of force matters.

Gentle tissue mobilization and aggressive scraping are not biologically identical interventions.

When treatment produces significant petechiae, bruising, swelling, or prolonged soreness, small blood vessels and surrounding tissues have been stressed or injured sufficiently to initiate an inflammatory response.

That response may be intentionally sought in some traditional or therapeutic applications of scraping techniques. However, when working with fibrotic, edematous, irradiated, postsurgical, or lymphatically compromised tissue, deliberately creating additional inflammation deserves careful consideration.

The therapeutic question becomes:
How much mechanical stimulus does this tissue need—and how much can it recover from?

Gua Sha and Scraping Techniques

Gua Sha and similar scraping techniques apply pressure and shear across the surface of the tissue.

Gentler applications may function similarly to other forms of instrument-assisted mobilization by creating movement between superficial tissue layers and providing sensory and mechanical input.

More aggressive traditional Gua Sha intentionally produces petechiae or visible discoloration.

That distinction is important.

Visible redness or bruising demonstrates that a biological response occurred, but it does not prove that fibrosis or an adhesion was physically removed.

For tissues already affected by edema, inflammation, radiation injury, or impaired lymphatic transport, aggressive scraping may introduce an additional inflammatory and fluid burden.

Visible tissue reaction should not be confused with successful tissue remodeling.

Petechiae are tiny, pinpoint red, purple, or brown spots that appear when very small blood vessels called capillaries break and leak a small amount of blood beneath the skin.

In the context of Gua Sha or aggressive tissue work, pressure and shear can mechanically stress superficial capillaries and produce petechiae.

They are different from ordinary temporary redness because the blood has actually escaped from the vessels into the surrounding tissue.

Importantly, petechiae are not toxins being released, and producing more petechiae does not mean a treatment is more effective.

In tissue with impaired lymphatic function, intentionally creating substantial petechiae may also add cellular debris and an inflammatory burden that the tissue must subsequently manage.

Negative Pressure: Cupping and Suction

Cupping introduces a very different mechanical force. Instead of pushing tissue inward, suction creates negative pressure, lifting superficial tissues toward the cup.

This may create separation between superficial tissue layers, alter local blood flow, influence sensory input, and change the mechanical forces experienced by fascia and the extracellular matrix.

Gentle moving cups and strong stationary cups also represent very different treatment doses.

A cup that lightly lifts and glides tissue produces a different stimulus from one that remains stationary long enough to create significant ecchymosis.

Bruising following cupping does not represent toxins being pulled from the body. It reflects blood escaping from small vessels into surrounding tissue.

That material must subsequently be processed and cleared. For individuals with compromised lymphatic transport, this becomes especially relevant.

Negative pressure may be useful as a mechanical tool, but more suction is not necessarily more therapeutic.

Textured Tools: Creating Variable Mechanical Input

Textured balls, rollers, massage tools, and specialized foam introduce another interesting form of mechanical stimulation.

Rather than applying uniform pressure across a large surface, texture creates multiple small areas of changing pressure.

As the tool moves across the tissue, those pressure points continually change.

This may provide:

- Variable compression
- Localized tissue displacement
- Sensory stimulation
- Changes in tissue glide
- Repetitive mechanical input to the extracellular matrix

One potential advantage of textured tools is the ability to provide relatively low-intensity mechanical stimulation across a broader region without requiring aggressive pressure.

They may also allow individuals to participate in carefully instructed self-management. Again, the objective is not necessarily to crush fibrotic tissue beneath the texture.

The texture provides mechanical variation.

Textured Compression: Small Signals Over Long Periods

Textured compression applies a related principle over a much longer period.

Specialized foam, pads, or garments create variations in pressure against the skin rather than providing completely uniform compression. When movement occurs underneath the garment, these pressure differences continually change.

In lymphedema management, textured materials may be incorporated into compression strategies for areas of tissue firmness or fibrosis. The mechanical stimulus is very different from aggressive manual treatment.

Instead of delivering a large amount of force for several minutes, textured compression may provide low-level mechanical input over hours while simultaneously supporting edema management.

This introduces an important concept:

Intensity is only one part of treatment dosage.

A gentle stimulus delivered repeatedly may produce a very different biological response from an intense stimulus delivered briefly. Small Signals Over Long Periods

Vibration: Repetition Without Heavy Pressure

Vibrational tools introduce another dimension—frequency.

Instead of relying primarily on sustained pressure, vibration produces rapid repetitive mechanical oscillations.

Localized vibration balls and handheld devices can transmit mechanical energy into superficial tissues without necessarily requiring substantial therapist pressure.

This makes vibration particularly interesting from the perspective of mechanotransduction.

Cells are capable of responding to repetitive mechanical signals, but direct clinical evidence demonstrating that vibration reverses established fibrosis remains limited.

The potential value may lie in how vibration influences the broader tissue environment—including mobility, sensory input, circulation, muscle activity, and mechanical signaling.

Once again:

Biological plausibility is not the same as proven antifibrotic efficacy.

Different Tools. Different Signals.

We can therefore think of these modalities according to the mechanical information they provide:

Manual therapy
Compression + stretch + shear + tissue displacement

IASTM / Gua Sha
Focused pressure + shear

Cupping
Negative pressure + tissue lifting

Textured tools
Variable localized compression + movement

Textured compression
Low-level variable pressure + prolonged duration

Vibration
Rapid repetitive mechanical oscillation

None of these forces is automatically better than another.

The appropriate stimulus depends upon the tissue, condition, treatment goal, stage of healing, lymphatic function, pain response, and individual tolerance.

The Tissue Response Is the Guide

Perhaps one of the simplest principles for using mechanical modalities is this:

The tool delivers the stimulus. The tissue tells us whether the dose was appropriate.

After treatment, we should reassess.

- Did tissue mobility improve?
- Did range of motion change?
- Did pain decrease?
- Did swelling increase or decrease?
- Did the tissue become softer?
- Was function easier?
- How did the tissue feel later that day?
- What happened the following morning?
- Did the improvement persist?

These questions provide far more useful information than whether the treatment created redness, soreness, or bruising.

For fibrotic tissue—particularly when lymphatic dysfunction is present—the goal should be to provide enough mechanical information to encourage adaptation without unnecessarily increasing tissue injury, inflammation, or fluid burden.

The future of fibrosis treatment may therefore be less about finding the most aggressive tool and more about understanding how to combine different forms of mechanical input at the right intensity, duration, frequency, and stage of healing.

Let the Tissue Response Guide the Treatment

No matter which modality is chosen, treatment should not end when the tool is put down or the therapist's hands leave the tissue. One of the most important parts of treatment is what happens next.

Every intervention provides a stimulus. The tissue then responds to that stimulus.

That response gives us valuable information about whether the type, intensity, duration, and frequency of treatment were appropriate.

Redness, warmth, tenderness, temporary softening, increased movement, decreased pain, bruising, swelling, and soreness can all occur following physical treatment—but they do not mean the same thing.

Understanding those responses helps us decide whether to continue, modify, reduce, or discontinue an intervention.

Green, Yellow, and Red Responses

🟢 Green Response — Appropriate Stimulus

- Improved mobility
- Improved tissue glide
- Decreased pain or tightness
- Mild temporary redness
- Reduced swelling
- Improved function
- Improvement maintained after treatment
- Tissue returns comfortably to baseline or better
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Interpretation: The tissue appears to be tolerating the treatment dose.

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🟡 Yellow Response — Reassess the Dose

- Moderate soreness
- Temporary increase in swelling
- Increased sensitivity
- Redness lasting longer than expected
- Improvement that disappears quickly
- Fatigue or discomfort following treatment
- Tissue requiring prolonged recovery

Interpretation: The intervention may still be useful, but intensity, duration, frequency, treatment area, or technique may need modification.

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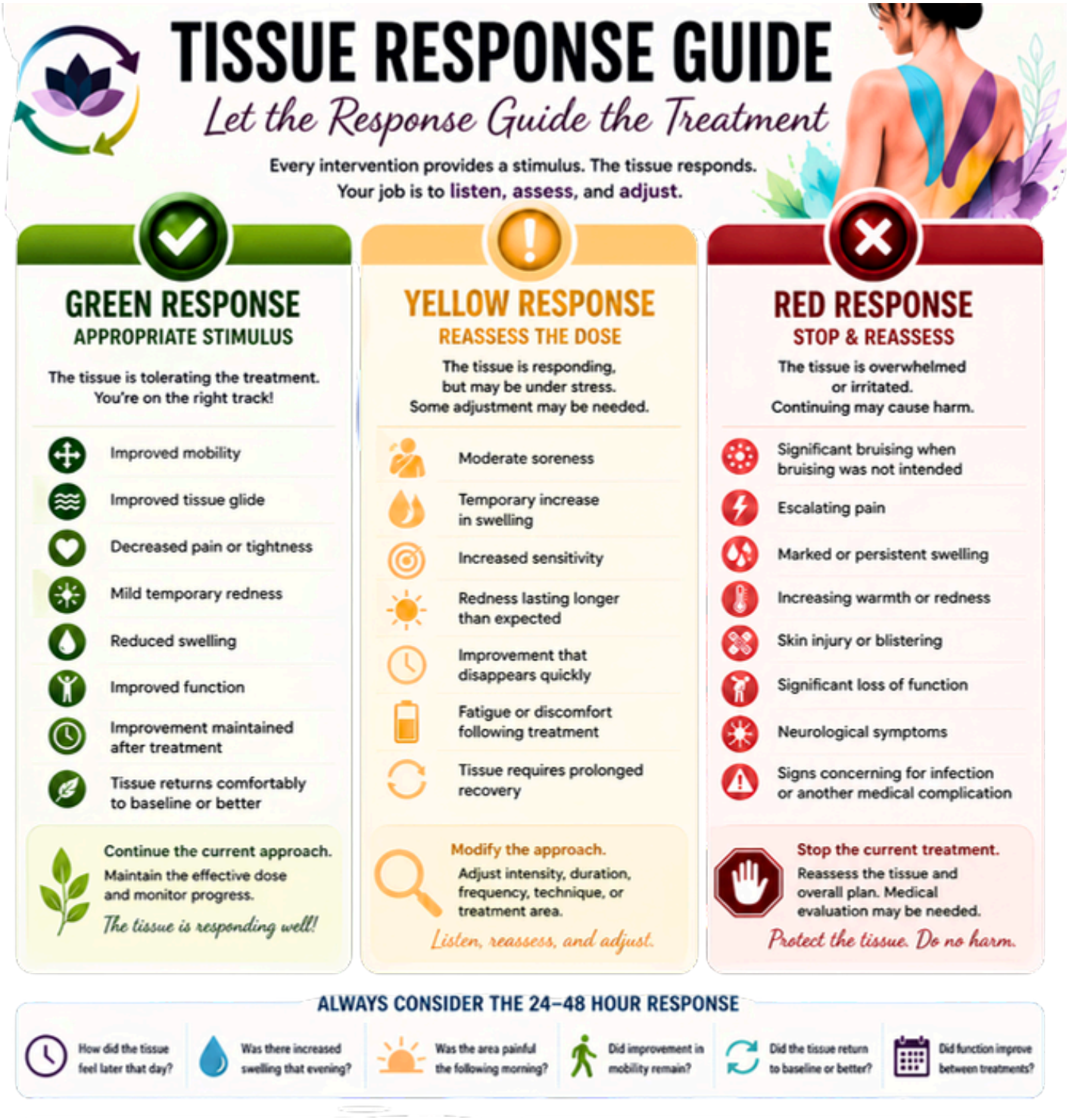
🔴 Red Response — Stop and Reassess

- Significant bruising when bruising was not intended
- Escalating pain
- Marked or persistent swelling
- Increasing warmth or redness
- Skin injury or blistering
- Significant loss of function
- Neurological symptoms
- Signs concerning for infection or another medical complication

Interpretation: Continuing the same treatment without reassessment may create more harm than benefit. Medical evaluation may be appropriate depending upon the symptoms



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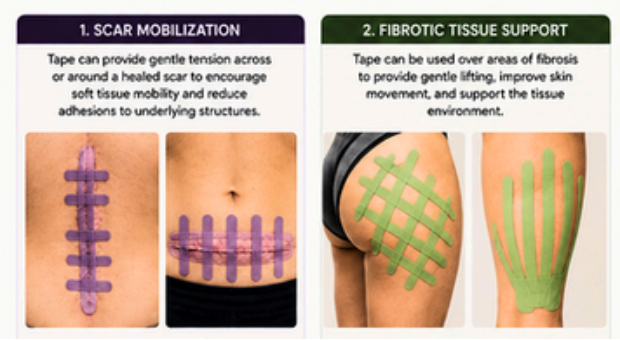
Therapeutic Taping for Scars and Fibrotic Tissue

1. Scar Mobilization
Tape can provide prolonged, low-level mechanical input to a healed scar. Depending on the application, it may create gentle tension across or around the scar and encourage mobility between superficial tissue layers.

2. **Fibrotic Tissue & Lymphatic Support**
When fibrosis occurs alongside edema or lymphatic dysfunction, lymphatic-style taping may be used as an adjunct to help manage swelling and the surrounding tissue environment. The important distinction is that tape does not directly dissolve fibrosis.

3. Mechanical Signaling
Tape provides something our hands cannot: very low-level mechanical stimulation over many hours or even days. Movement underneath appropriately applied elastic tape continually changes tension across the skin and superficial tissues. This makes taping interesting from a mechanotransduction perspective.

4. **Scar Remodeling**
For scars, taping may help influence tension and mechanical forces across healing tissue. However, we should distinguish elastic kinesiology tape from other scar-management products such as silicone gel sheets and paper tapes, which have their own evidence



Taping occupies an interesting place in the management of scars, edema, and fibrotic tissue. Unlike manual therapy, IASTM, cupping, or vibration—which generally provide mechanical stimulation during a defined treatment session—tape may remain on the body for many hours or several days.

This creates a very different mechanical dose.
Rather than applying substantial force for a short period of time, elastic therapeutic tape can provide relatively gentle, prolonged mechanical input while the person moves through normal daily activities.

Every breath, step, reach, or change in position alters the relationship between the tape, skin, and underlying superficial tissues.

From the perspective of mechanotransduction, this is particularly interesting. The tissue is not receiving one large mechanical event. Instead, it receives thousands of small changes in tension and movement throughout the day.

Taping Does Not "Break Up" Fibrosis

As with many physical modalities, taping is sometimes described as a way to break up scar tissue or fibrosis.

That description is probably too simplistic.
Tape does not mechanically dissolve collagen. Its potential value lies in altering the mechanical environment surrounding the tissue.

Depending upon the application, taping may be used to:

- Provide gentle mechanical input across a healed scar
- Encourage movement between superficial tissue layers
- Reduce tension across or around a scar
- Support movement without rigidly restricting it
- Provide sensory input
- Assist with edema management
- Complement compression and lymphatic treatment
- Provide prolonged low-level stimulation to areas of tissue firmness or restriction

The application should therefore be selected according to the treatment goal, rather than simply placing tape over every area that feels fibrotic.

Taping a Healed Scar

A healed scar may be taped in several ways depending upon its mobility, orientation, sensitivity, and surrounding tissue restrictions.

Short strips placed perpendicular to a scar can create gentle mechanical forces across the scar during movement.

Other applications may run parallel to the scar or create small multidirectional forces around areas where tissue mobility is restricted.

The goal is not to pull aggressively on the scar.

Instead, the tape can provide subtle, repeated mechanical input as the skin moves beneath it.

For a scar that feels tethered or restricted, the therapist may also consider the mobility of the surrounding tissue rather than focusing exclusively on the visible scar line.

A scar is part of a three-dimensional tissue environment. The restriction may extend above, below, beside, or beneath what we can see at the surface.

Taping Fibrotic Tissue

Fibrotic tissue may require a different strategy.

Rather than applying concentrated tension directly across one line, a broader application may distribute mechanical input across an area of firmness or restriction.

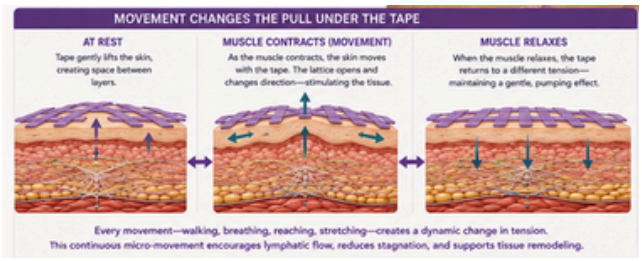
Lattice, basket-weave, fan, or multiple-strip applications can create small variations in tension across the skin.

As the individual moves, these variations continually change.

The important concept is not that the tape is physically pulling fibrosis apart.

It is providing variable, prolonged mechanical stimulation to the tissue environment.

This may be particularly useful when the tissue does not tolerate aggressive manual pressure.



Taping When Edema and Fibrosis Coexist

Fibrosis frequently occurs alongside edema, particularly in lymphedema and some postsurgical conditions.
In this situation, the treatment goal may involve both:
Managing the fluid environment
and
Supporting tissue mobility.

Lymphatic-style taping commonly uses narrow fan-shaped strips applied with relatively low tension. Movement beneath the elastic tape creates subtle changes in skin tension that may be incorporated into a broader edema-management strategy.

However, taping should not be presented as a substitute for established lymphedema management.

When clinically indicated, it may complement:

- Compression
- Movement and exercise
- Manual lymphatic drainage
- Skin care
- Positioning
- Patient self-management

This reinforces an important principle:

Nothing works in isolation. Everything works in combination.

Before using tape more broadly, apply a small piece to the inside of the forearm for 24 hours, then remove it and check the skin. Reactions vary by person and by tape type, and some brands offer tape designed for sensitive skin.

Choosing the Application: The Goal Determines the Tape

There is no single "fibrosis taping technique."

The appropriate application depends upon what the therapist is trying to accomplish.

Before applying tape, ask:

What am I trying to change?

If the primary problem is a restricted scar:

Consider an application that provides gentle multidirectional mechanical input around or across the scar.

If the primary problem is diffuse tissue firmness:
A broader lattice, fan, or multi-strip application may distribute mechanical stimulation across a larger area.

If edema is present:
A lymphatic-style application may be incorporated into the overall fluid-management strategy.

If movement creates pulling or discomfort:
Tape may be positioned to modify tension or provide sensory feedback during functional movement.

If the tissue is highly sensitive:
Begin conservatively.
Sometimes the most effective application is the one the tissue can comfortably tolerate for an extended period.

Low Force Can Still Be Powerful

One of the most important lessons from our discussion of fibrosis is that more force does not necessarily create more remodeling.

Taping illustrates this beautifully.
A relatively small mechanical stimulus delivered repeatedly for many hours may provide a very different biological signal from aggressive treatment delivered for several minutes.

.

Force × Duration × Frequency × Movement

We can think of the mechanical dose as a combination of:

This helps explain why a low-force intervention should not automatically be considered a weak intervention.
Its strength may lie in duration and repetition

Movement Makes the Application Dynamic

Tape applied to someone who never moves provides a different stimulus from tape worn during walking, breathing, reaching, exercise, or normal daily activity.
Movement continually changes tension between the tape and skin.

This creates a dynamic mechanical environment.
For this reason, taping often makes the most sense when it supports rather than restricts functional movement.
Manual therapy may help create mobility.

Tape may provide continued mechanical input.

Movement allows the body to use that mobility.
Together, they create a much more complete therapeutic strategy than any one intervention alone.

Skin Integrity Comes First

Taping is only useful if the skin tolerates it.
Scarred, irradiated, edematous, aging, and lymphatically compromised skin may be particularly vulnerable to irritation.

Tape should generally be applied only over closed, sufficiently healed skin, and caution is warranted with:

- Fragile or thin skin
- Radiation-damaged tissue
- Active dermatitis or rash
- Open wounds
- Active infection or cellulitis
- Significant skin sensitivity
- Known adhesive reactions
- Areas with impaired sensation
- Unexplained redness or inflammation

A small test application may be appropriate when skin tolerance is uncertain.

The ends of elastic tape generally should not be placed under substantial tension, since excessive tension can increase the risk of skin irritation.

Tape Is Not "Set It and Forget It"

Just as we reassess tissue after manual therapy, IASTM, cupping, or vibration, we should reassess the response to tape.

🟢 Positive Response
- The application is comfortable.
- Mobility improves.
- Pulling or tightness decreases.
- Swelling is stable or reduced.
- The skin remains healthy.
- Function becomes easier.
- Continue and monitor.

🟡 Caution Response
- Mild itching develops.
- The edges become uncomfortable.
- The skin becomes increasingly sensitive.
- Swelling changes unexpectedly.
- The tape feels restrictive rather than supportive.
- Remove or modify the application and reassess.

🔴 Stop Response
Remove the tape if there is:
- Significant itching or burning
- Blistering
- Skin tearing
- Persistent or increasing redness
- Significant swelling
- Increasing pain
- Rash or allergic reaction
-

Do not leave an irritating application in place simply because it was intended to remain for several days. The tissue response always takes priority over the planned treatment duration.

Taping can be left in place up to 5 days if well tolerated. For forst application I generally recommend 24 to 48 hours then remove and watch the skin for a day.

Patient/client education is important.

Elastic Tape Is Not the Same as Scar Tape

Another important distinction is that kinesiology-style elastic tape, paper tape, and silicone scar products are not interchangeable.

Elastic tape is primarily used to provide movement-related mechanical and sensory input.
Paper tapes have been studied for controlling mechanical tension across healing scars.

Silicone gels and silicone sheets have a different mechanism and a more established role in the prevention and management of hypertrophic scars and keloids.

Each should therefore be discussed according to its own evidence and intended purpose rather than simply grouping everything under "taping."

Assess. Apply. Move. Reassess.

Perhaps the simplest framework for therapeutic taping is:

ASSESS
What is limiting the tissue?
↓
APPLY
Choose the tape pattern and mechanical dose according to the goal.
↓
MOVE
Allow normal movement to create changing mechanical input.
↓
REASSESS
Evaluate the skin, swelling, mobility, pain, and function

Taping should not be judged by how elaborate the application looks.

A beautifully patterned application that irritates the skin or produces no functional benefit is not necessarily successful.

The most useful application is the one that provides an appropriate stimulus, is tolerated by the tissue, and contributes to a measurable improvement in the treatment goal..

Low force. Longer duration. Movement. Reassessment.

Together, these characteristics make taping a unique addition to the larger toolbox for scar, edema, and fibrotic-tissue management.

This Achilles taping example demonstrates how the lymphatic system and fibrosis can also contribute to injury. Although 2% of the population has lymphedema, 100% of people have a lymphatic system. Compromise within that system can lead to excess fluid and fibrosis. This application uses two techniques: a fibrosis star at the Achilles-calcaneal junction and lymphatic taping to help move excess fluid along the drainage pathway.

Beyond the Modality: Putting the Pieces Together

We have explored many different ways of influencing fibrotic tissue—from the therapist's hands to compression, textured materials, positive and negative pressure, vibration, energy-based modalities, and therapeutic taping.

Each provides a different stimulus. Each may influence a different part of the tissue environment. And each has its own evidence, limitations, precautions, and appropriate dose.

But fibrosis is not created by one process, and it is unlikely to be successfully managed by one intervention.

Fibrosis develops within a complex biological environment involving the extracellular matrix, fibroblasts, collagen production and breakdown, inflammation, fluid balance, mechanical forces, movement, vascular function, lymphatic transport, and cellular signaling.

If multiple systems contribute to the development and persistence of fibrosis, then effective management should consider those systems together.

This brings us back to one of the most important principles of this publication:

Nothing works in isolation. Everything works in combination.

Manual therapy may improve tissue mobility.
Manual lymphatic drainage and compression may help address the fluid environment when lymphatic dysfunction is present.

Movement provides repeated mechanical loading.
Exercise helps the body use newly available mobility.
Taping may extend gentle mechanical input beyond the treatment session.

Other physical or energy-based modalities may provide additional mechanical, electrical, acoustic, or photobiological signals when appropriately selected.
Hydration, nutrition, skin integrity, sleep, activity, and management of underlying medical conditions all influence the environment in which those interventions must work.

The question therefore becomes less about finding the best fibrosis treatment and more about constructing the right combination of interventions for the tissue in front of us.

Movement: Where Treatment Becomes Function

There is one component that deserves special attention before we leave the treatment discussion:
movement.

A therapist may temporarily improve tissue mobility, but the body must then learn to use that mobility.
Every step, breath, reach, stretch, and muscle contraction changes mechanical forces within the extracellular matrix.

Movement creates tension, compression, shear, and fluid displacement while providing continuous mechanical information to cells.
In this sense, exercise is not simply something performed after treatment.

Movement is part of the treatment.

Appropriately dosed movement may help maintain range of motion, support circulation and lymphatic transport, restore normal loading patterns, improve strength, and provide repeated mechanical signals that encourage tissues to adapt to functional demands.
This is why a treatment that produces temporary softening but no improvement in movement or function may be incomplete.

The ultimate goal is not simply softer tissue.
It is better tissue function.

Managing Fibrosis Is a Process

Fibrotic tissue rarely changes overnight or after one treatment.

Evaluate progress over time:

- pliability
- swelling
- range of motion
- pain
- ease of movement
- activity tolerance
- how long improvements last between treatments.

Adapt management as the tissue changes. What suits early healing may not suit later stages, and irradiated or lymphatically compromised tissue may require gentler techniques.

Assess. Treat. Reassess. Adapt.

Returning to Where We Started

At the beginning of this publication, we entered the microscopic world of the extracellular matrix.

We discovered that it is not empty space between our cells. It is a hydrated, responsive, continuously remodeling environment where cells communicate, mechanical forces are sensed, nutrients and signaling molecules move, and lymphatic capillaries begin collecting excess fluid and proteins.

We then followed what happens when that environment changes.

- **Inflammation alters signaling.**
- **Fibroblasts respond.**
- **Collagen is produced.**
- **Healing begins.**

And when the signals driving repair persist, normal remodeling can shift toward fibrosis.

Understanding that progression changes the way we think about treatment.

Perhaps fibrotic tissue is not simply something that needs to be broken apart. Perhaps it is an environment that needs to be

- **Understood.**
- **Supported.**
- **Mobilized.**
- **Loaded appropriately.**
- **Decongested when necessary.**
- **Allowed to recover.**
- **And continually reassessed**.

Our growing understanding of mechanotransduction, lymphatic physiology, inflammation, and extracellular matrix biology is giving us a much more sophisticated picture of tissue healing than we had even a few decades ago.

There is still much we do not know.

But one principle remains remarkably consistent:
The body responds to its environment.

Our role is to create the best environment we can for healthy adaptation, remodeling, movement, and function.

And perhaps that is the most important lesson of all. We don't simply treat fibrosis. We treat the environment in which fibrosis developed.

Nothing works in isolation. Everything works in combination.

A Final Thought

If there is one message I hope you take away from this publication, it is that fibrosis is not simply something hard beneath the skin that needs to be broken apart. It is the result of a remarkable biological process.

We began our journey inside the extracellular matrix—the hydrated, living environment surrounding our cells.

We learned how fibroblasts build and maintain that matrix, how collagen provides strength, how inflammation initiates repair, and how the lymphatic system continually helps maintain the balance of fluid, proteins, immune activity, and cellular waste within our tissues.

What have we discovered?

We also discovered how easily a process designed to protect us can become persistent.

When inflammation continues, lymphatic transport becomes impaired, injury is repeated, or normal remodeling does not resolve as intended, the extracellular matrix can gradually change.

Collagen accumulates and reorganizes. Tissue becomes denser. Mobility decreases. Fluid movement may become more difficult.

And fibrosis can become part of a cycle that is increasingly difficult for the body to resolve on its own.

There Is No Single Tool

Perhaps this is why there is no single answer to fibrosis. Throughout this publication we have explored many approaches:

- **Hydration.**
- **Nutrition.**
- **Manual therapy.**
- **Lymphatic support.**
- **Compression.**
- **Movement.**
- **Taping.**
- **Positive and negative pressure.**
- **Vibration.**
- **Electrical and energy-based modalities.**
- **Skin care.**
- **Self-management.**

Each may influence a different part of the tissue environment.

Some have stronger scientific evidence than others. Some have promising research that continues to evolve. Others are supported more by clinical experience than by large controlled trials.

And none should be expected to do everything.
The question should never simply be:

“What breaks up fibrosis?”
A better question is:
“What does this tissue need to move toward healthier function?”

- **Sometimes it needs movement.**
- **Sometimes it needs fluid management.**
- **Sometimes it needs mechanical input.**
- **Sometimes it needs compression.**
- **Sometimes it needs less inflammation**.

And sometimes it simply needs time and the opportunity to heal.

Listen to the Tissue

One of the most important lessons in these pages is that treatment should be a conversation with the tissue. Assess. Treat. Reassess. Adapt.

The strongest treatment is not necessarily the best treatment.

- **Bruising does not prove that fibrosis has been removed.**
- **Pain does not prove that a treatment is effective.**
- **Redness does not equal remodeling.**
- **And immediate softness does not necessarily represent permanent structural change.**

Instead, look for the outcomes that matter:

- Better movement.
- Less pain.
- Improved tissue mobility.
- Better management of swelling.
- Greater tolerance to activity.
- Healthier skin.
- Improved function.

And changes that persist over time.
Those are the responses that help guide the next step.

The Science Will Continue to Evolve

Our understanding of the extracellular matrix, mechanotransduction, fibroblast behavior, inflammation, and lymphatic physiology continues to grow.

So will our understanding of fibrosis.
Some of the therapies discussed in this publication will accumulate stronger evidence. Others may eventually prove less useful than we once believed.

New technologies and treatment strategies will emerge. That is how science works.

We continue to ask questions.
We challenge assumptions.
We observe.
We measure.
We learn.

And then we adjust what we do.

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Deep Oscillation — promising adjunct, but direct antifibrotic evidence remains limited

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Negative Pressure / Cupping — distinguish therapeutic suction from wound-vacuum research

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Lymphatic Restoration Therapy

Everything works in combination
Nothing works in isolation

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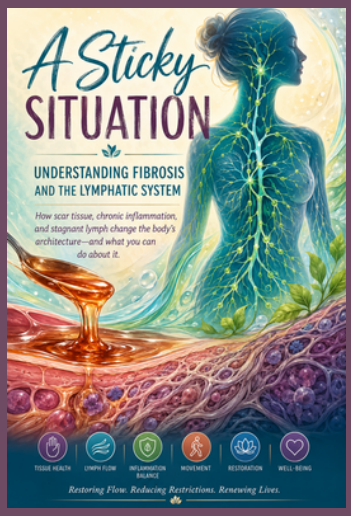

Fibrosis is often described simply as “scar tissue.” But the story is far more complex.

Fibrosis begins within the living environment surrounding our cells—the extracellular matrix—where inflammation, fluid balance, fibroblast activity, collagen production, mechanical forces, and the lymphatic system all intersect. Understanding this environment changes the way we think about why tissue becomes thickened, restricted, painful, or difficult to move—and how we approach its care.

In A Sticky Situation: Understanding Fibrosis and the Lymphatic System, Laine Miller takes readers from the microscopic world of the extracellular matrix through the development of inflammation, scars, adhesions, lymphatic stasis, and fibrosis. Complex physiology is translated into approachable language and visual education designed for therapists, patients, caregivers, and anyone wanting to better understand what is happening beneath the surface.

The publication also explores the growing science behind the many approaches used to manage fibrotic and scarred tissue—including manual therapy, lymphatic support, compression, textured materials, taping, movement, vibration, negative pressure, photobiomodulation, electrical and acoustic modalities, and more. Rather than asking simply, “How do we break up fibrosis?”, readers are encouraged to consider a more meaningful question:

How can we create a tissue environment that supports healthier remodeling and better function?

Grounded in research, clinical reasoning, and an appreciation for the interconnected nature of the human body, this publication offers a different way to look at fibrosis—not as an isolated problem to attack, but as a biological process to understand.

Science. Connection. Restoration.

Nothing works in isolation. Everything works in combination.

Laine Miller, PTA, LMT, CLT, C-MLD
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