NORTHWEST STEM CELL

Cellular Healing in Chronic Pain

Adipose-Derived Cellular Therapy + Hyperbaric Oxygen

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Topics

- Why adipose based cellular therapy makes sense biologically
- Why hyperbaric oxygen therapy fits chronic pain and tissue dysfunction
- Why stem cells and HBOT are effective in chronic pain patients

Chronic Pain Remains Poorly Addressed

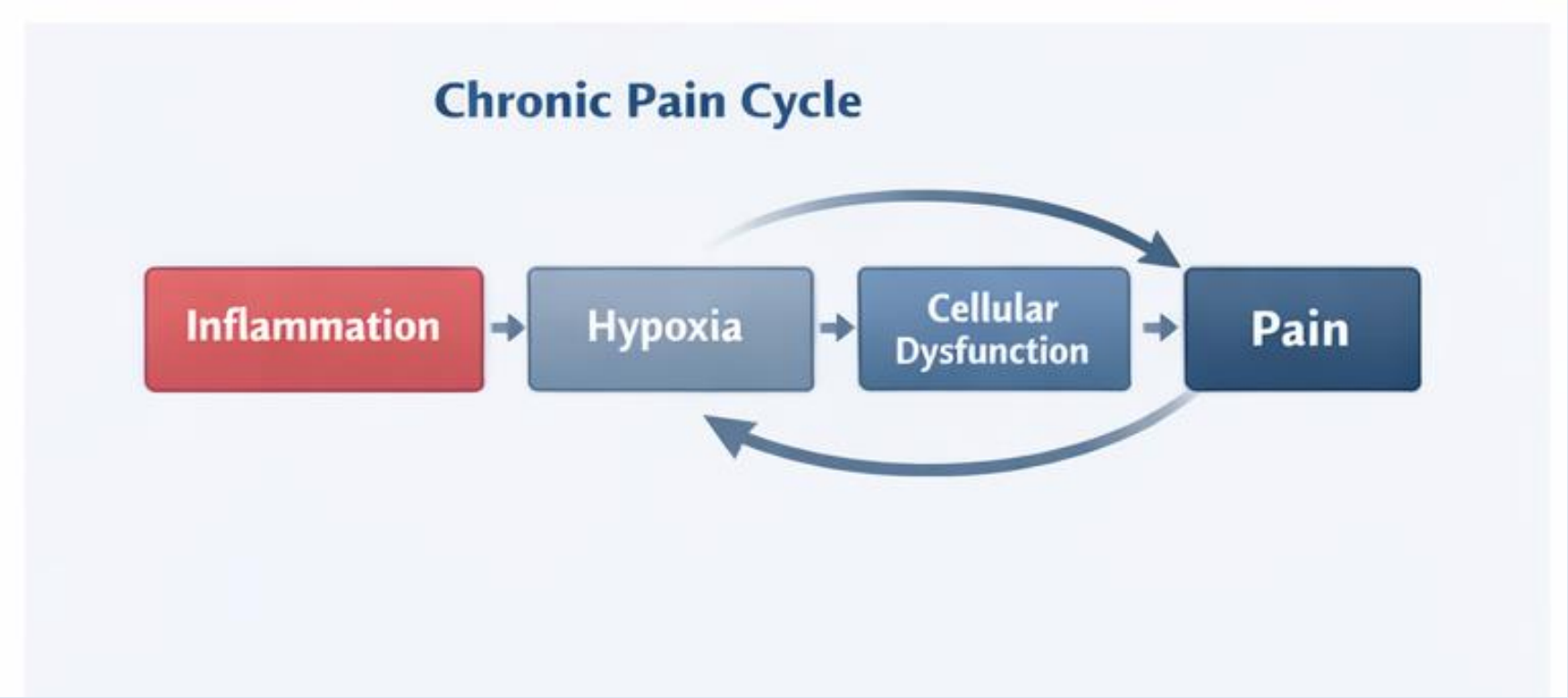
- High prevalence, high cost
- Symptom-focused treatments dominate
- Limited restoration of tissue biology

Chronic Pain = Failed Healing State

- Persistent inflammation
- Tissue hypoxia
- Impaired vascularity
- Dysregulated repair signaling

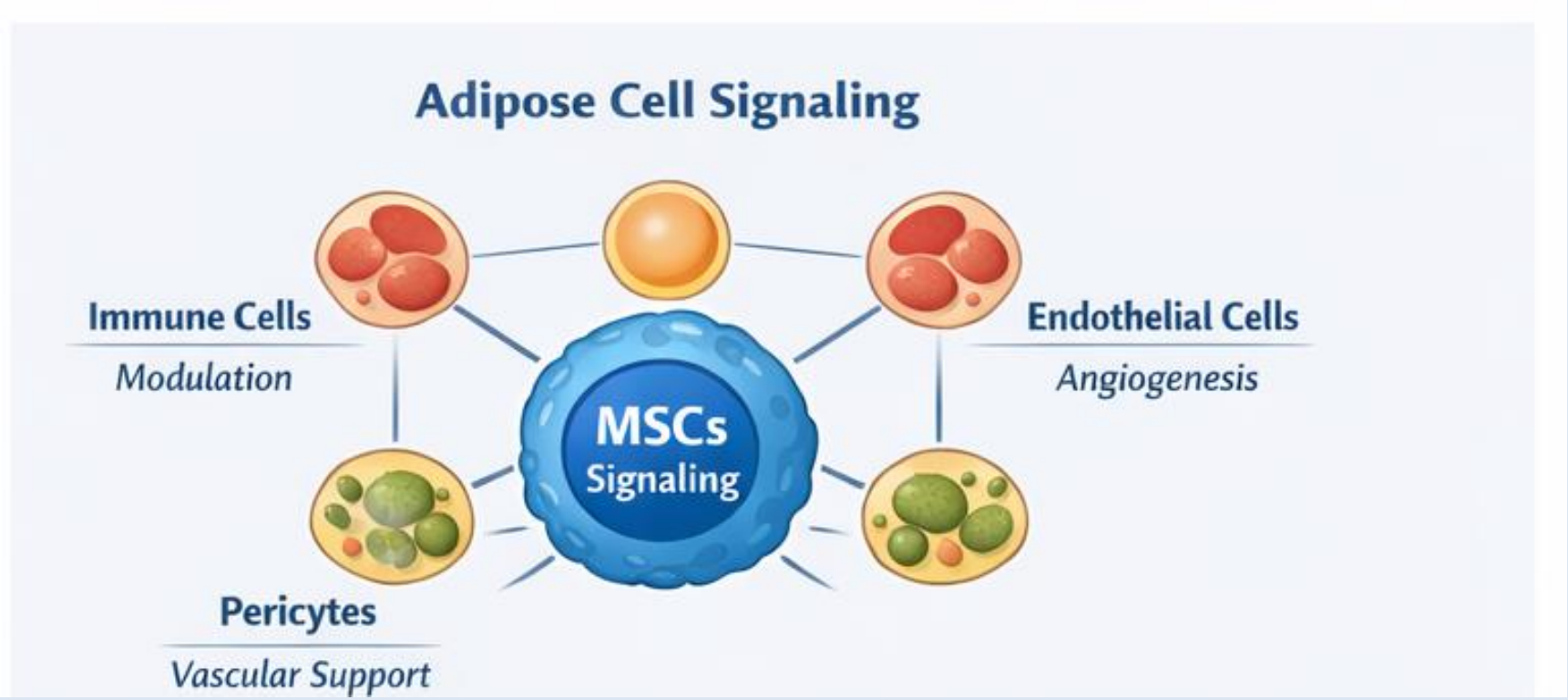
Pathophysiology Model

- Inflammation → Hypoxia → Cellular Dysfunction → Pain Loop



Why Adipose Tissue?

- High cellular yield (significantly greater than bone marrow)
- Rich stromal vascular fraction
- Strong paracrine signaling capacity
- Autologous and accessible



Cellular Composition

- Mesenchymal stem cells
- Pericytes
- Endothelial progenitors
- Immune-regulatory cells

Paracrine Signaling

- Cytokines + growth factors
- Dominant mechanism
- Coordinates repair

Immune Modulation

- ↓ TNF-α, IL-6
- ↑ anti-inflammatory signaling
- M1 → M2 shift

Tissue Repair

- VEGF angiogenesis
- Fibroblast activation
- Matrix remodeling

Clinical Relevance

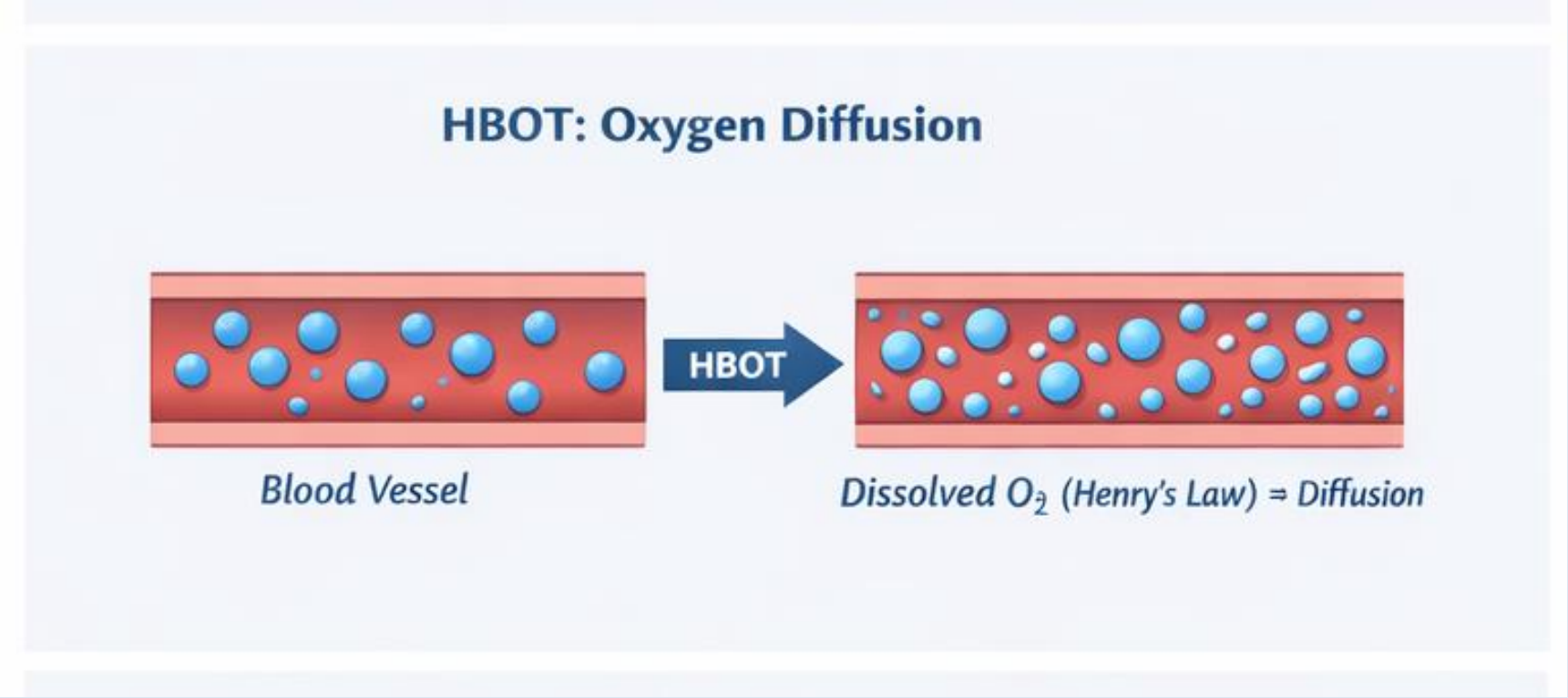
- Osteoarthritis
- Tendinopathy
- Disc pain
- Post-surgical dysfunction

HBOT Overview

- 100% oxygen under pressure
- Increased plasma oxygen
- Reaches hypoxic tissue

The Oxygen Problem

- Reduced perfusion
- Fibrosis
- Chronic inflammation



Oxygenation

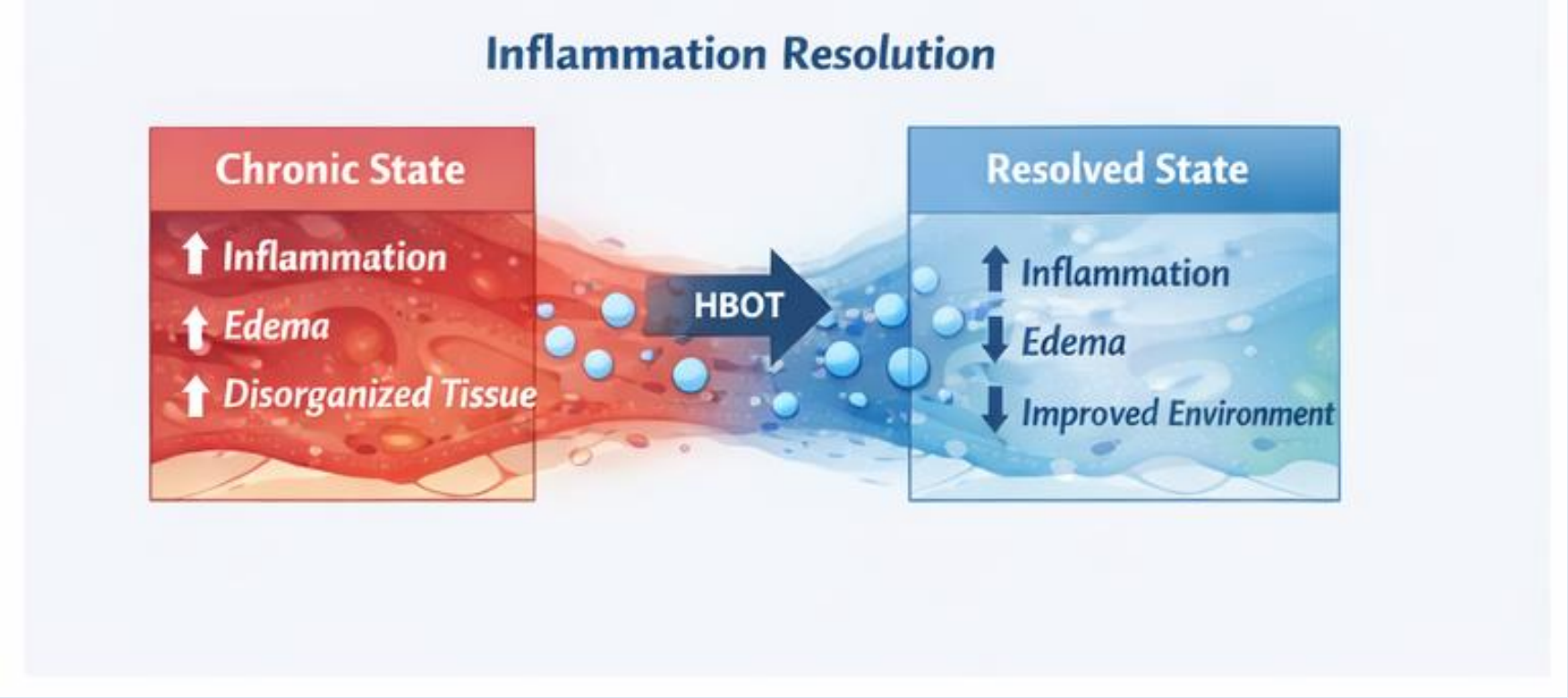
- 10–15x plasma oxygen
- Diffusion into tissue

Angiogenesis

- VEGF stimulation
- Capillary formation
- Improved perfusion

Inflammation Reduction

- Cytokine downregulation
- Edema reduction

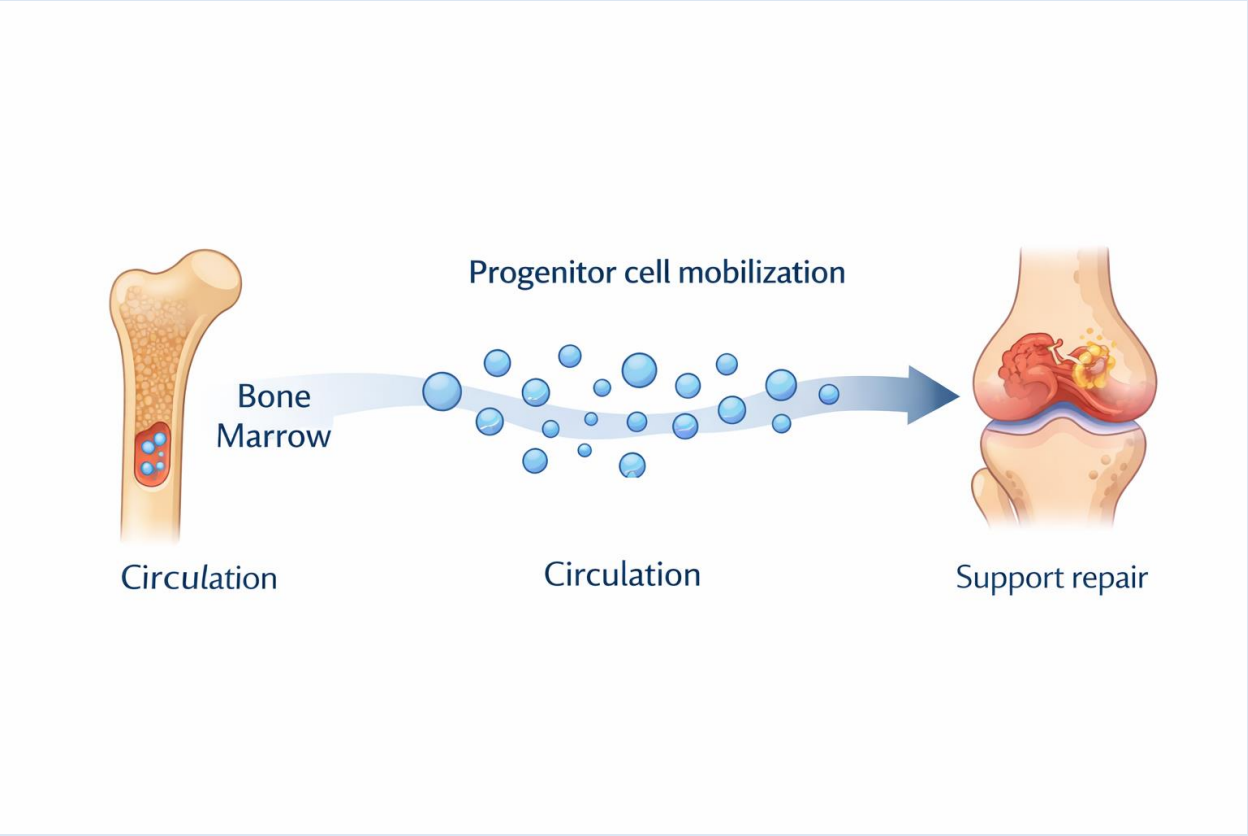


Mitochondrial Function

- ↑ oxidative phosphorylation
- ↑ ATP production
- Restoration of cellular energy capacity

Stem Cell Mobilization

- ↑ circulating progenitor cells
- NO-mediated mobilization
- Bone marrow → circulation

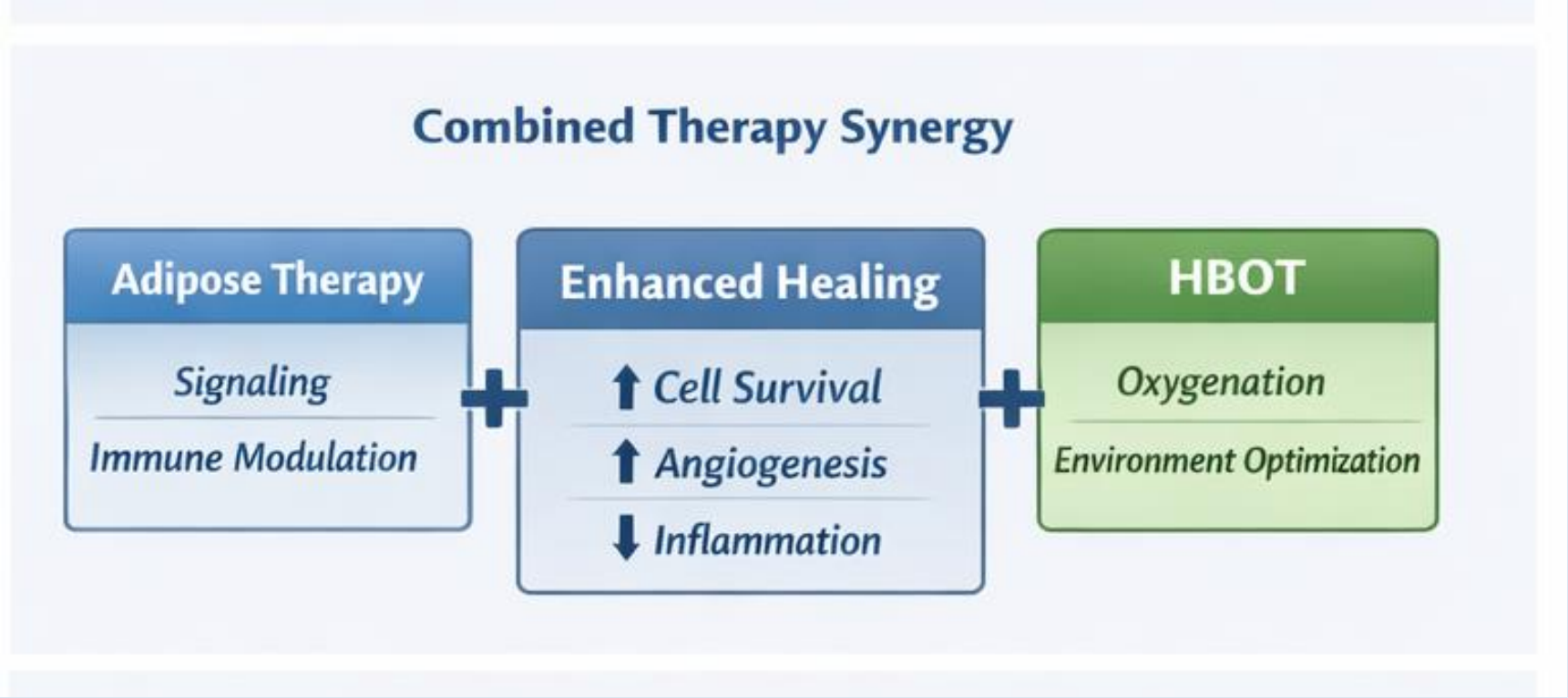


Limitations

- Stem Cells: limited survival in hypoxia
- HBOT: no signaling

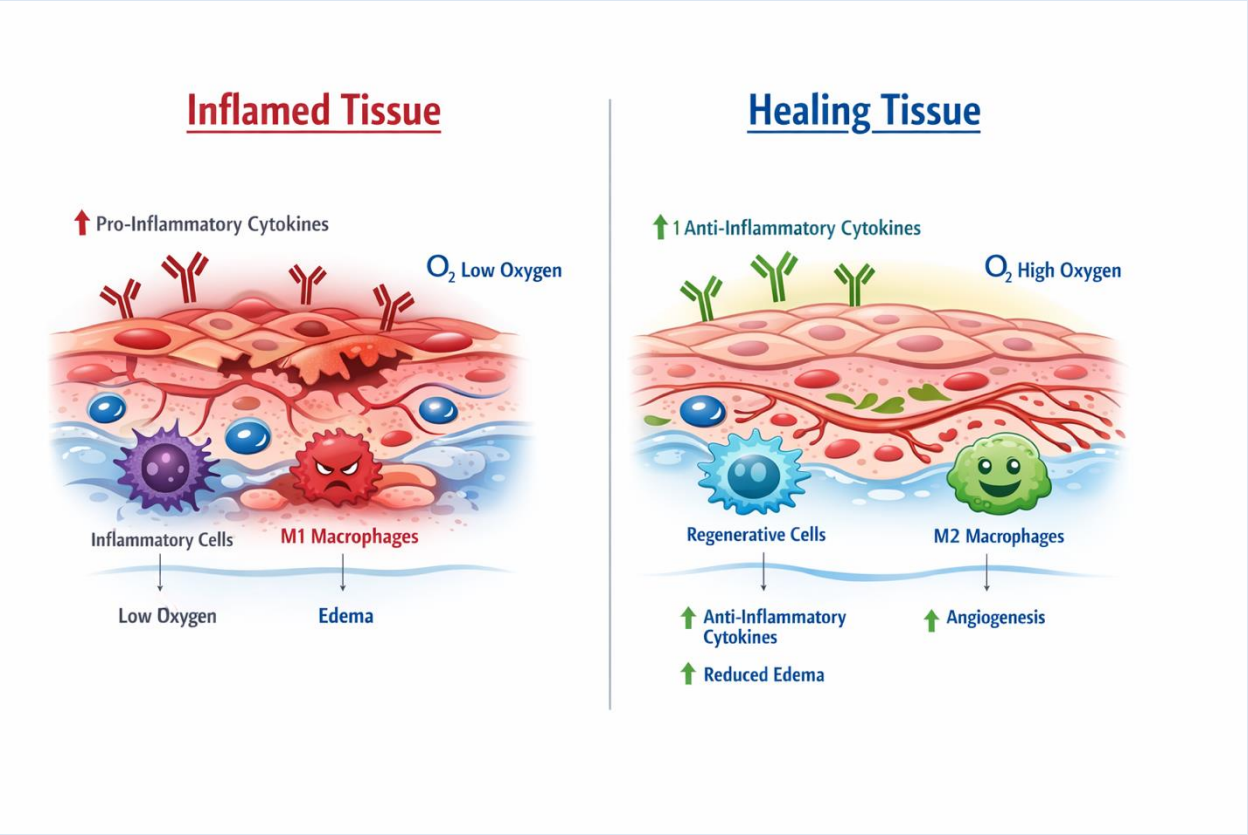
Synergy Model

- Adipose: signaling + repair
- HBOT: oxygenation + environment
- Combined: enhanced healing



Biological Synergy

- Improved cell survival
- Amplified signaling
- Enhanced angiogenesis
- Sustained anti-inflammatory effect



Clinical Translation

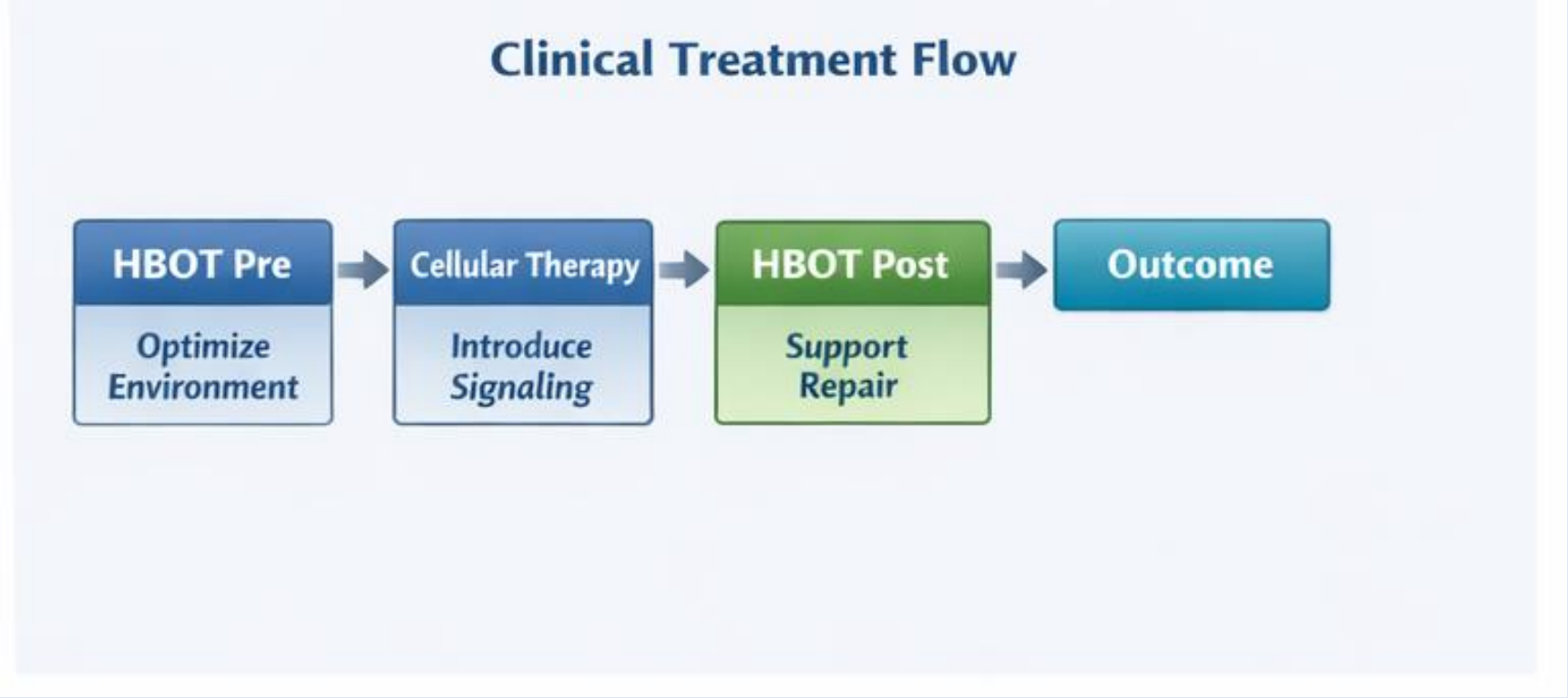
- Decreased pain
- Increased function
- Improved durability

Patient Profile

- Chronic joint/spine pain
- Failed conservative care
- Limited surgical options

Treatment Framework

- HBOT Pre-condition
- Cell therapy
- Post HBOT



Protocol Example

- 3 HBOT pre
- Cell therapy
- 7+ HBOT post

Case: Knee OA

- Baseline: chronic pain with functional limitation
- Intervention: adipose-derived therapy + HBOT protocol
- Outcome: ↓ pain, ↑ mobility, improved functional capacity

Case: Lumbar Pain

- **Lumbar Pain (Rheumatoid Arthritis–Associated)**
- Baseline: chronic lumbar pain with functional limitation
- Intervention: adipose-derived therapy + HBOT protocol
- Outcome: ↓ pain, ↑ function, improved activity tolerance

Evidence (Adipose)

- Caplan AI
- Pittenger et al.
- Paracrine signaling literature

Evidence (HBOT)

- Thom SR
- Angiogenesis + inflammation data

Key Takeaways

- Chronic pain often reflects a **dysregulated or incomplete healing state**
- Adipose therapy helps restore **biological signaling**
- HBOT helps optimize the **tissue environment**
- The combination enhances the potential for **durable tissue repair**

Final Thought

- You cannot restore function without first restoring the underlying biology.

Q&A