Shockwave Therapy Procedure

Course Outline

- History of Shockwave Therapy
- What is it? How does it differ from Ultrasound?
- Understanding the Difference Between Radial and Focused
- How mechanical acoustic waves are created by each type of technology
- Pneumatic vs Electromagnetic (Radial-RSWT)
- Electrohydraulic vs Piezoelectric vs Parabolic Cylinder Coils (FSWT)
- How does it really work? Learn the Science and Physics.
- Cleaning and Maintenance
- Hands on Technique Training plus with some real patients!
- How to do Cost Effective Marketing
- How to Create and Sell Cash Niche Packages
- Learn how to Utilize Gold & Silver Care Plans with Class IV Laser Synergy
- Learn our Turnkey Marketing system

Goals for Today

- ✓ Achieve Clinical Certainty with ESWT
- ✓ Learn How to Increase Revenue with Shockwave
- ✓ Discover Proper Marketing & Practice Building with ESWT

TABLE 5 Sample extracorporeal shockwave therapy (ESWT) milestones for trainee education with associated target competencies

Procedural skills: Extracorporeal shockwave therapy					
Level 0	**Level 1**	**Level 2**	**Level 3**	**Level 4 (Graduation target)**	**Level 5 (Aspirational)**
Foundational understanding of ESWT principles: -Physics -Knobology -Ergonomics -Safety protocols	Clinical knowledge of ESWT application: -Indications for use (radial vs. focused) -Risks -Benefits -Side effects -Contraindications	Demonstration or verbal description of appropriate protocols in a simulated setting: Preprocedural: -Setup -Positioning & ergonomics -Informed consent Procedural: -Device placement & settings -Titration goal -Anatomic landmarks, etc. Postprocedural: -Patient counseling -Device cleaning Performance of informed consent and time-out in a clinical setting Performance of beginner level ESWT procedures with **supervision** + signficant hands-on assistance	Performance of beginner/intermediate level ESWT procedures with supervision + little to no hands-on assistance (may require verbal assistance) Ability to titrate dose based on clinical response Generates appropriate documentation	Performance of advanced level ESWT procedures with supervision + little to no assistance Experience across an expanding spectrum of diagnoses and patient-specific factors, requiring individualization of care Ability to suggest protocol modifications based on clinical response Teaching of peers Publication of peer-reviewed work related to ESWT with significant faculty mentorship	Performance of **wide** range of advanced level ESWT procedures adeptly and efficiently Independent modification of treatment protocols to achieve targeted and measurable patient outcomes with integration of current literature and evidence-based treatment plans Teaching of peers and faculty Independent publication of peer-reviewed work related to ESWT
☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐					
Selection of a box between levels indicates that some, but not all, milestones in the higher level have been achieved.					
Comments:					

History & Medical Advancements

- 1980- First used in Urology as Lithotripsy to break up kidney stones to allow passing them without surgery
- ESWT is now FDA cleared for treatment of Plantar Fasciitis and widely used in Urology for ED.
- ESWT is now becoming a mainstream therapy for treatment of tendinopathies, myofascial pain syndromes, and calcific tendonitis.
- Urology for Erectile Disfunction, Pelvic Floor and Kidney Stones, and also Wounds and Skin Conditions

What is a Shock wave?

- ESWT stands for Extracorporeal Shock Wave Therapy. It is a non-invasive medical treatment that uses shock waves to stimulate healing and alleviate pain in certain medical conditions. The term "extracorporeal" means that the shock waves are generated outside the body and then directed towards the affected area.
- It is simply another form of energy that can be used to create physiologic changes by mechanically activating tissue. Shockwaves are Acoustic sound waves used to compress and manipulate tissue.

Shockwave Sounds Scary but Its Not!

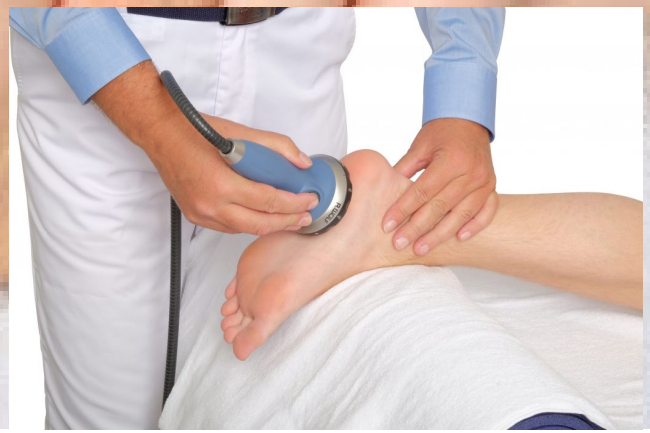
Acoustic Wave Therapy Just Isn't as Exciting!

- Tell Patients they Won't End up Like Marv!

People ask us all the time…What do we call it?

Its your choice but we recommend calling it what it is so Shockwave from our Experience.

Shockwave is like a defibrillator for soft tissue!

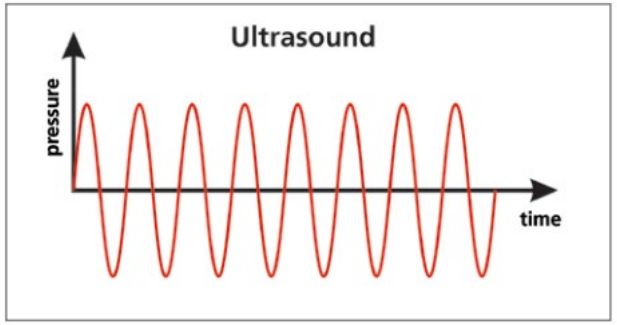


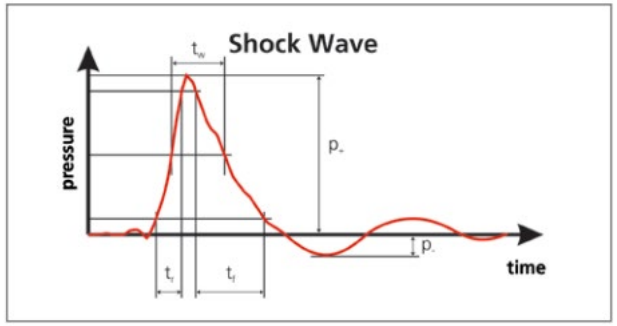
Acoustic wave (AKA “Shockwave” ©)

Powerful combination with a laser – pressure wave therapy is a modern method used to treat shoulder injuries, chronic Achilles tendonitis, plantar fasciitis, elbow tendinopathies and chronic stages of the myofascial syndrome (pain of the muscles).

How Does it Differ from Ultrasound?

- Ultrasound uses sound to vibrate tissue with a periodic oscillation to increase tissue temperature 4 degrees Celsius for rehab purposes such as extensibility of the tissues.
- Shockwaves are sound (acoustic)compression waves that mechanically remodel scar tissue which creates new tissue healing through a micro trauma. This process is called Me





Shock wave vs Radial Pulse

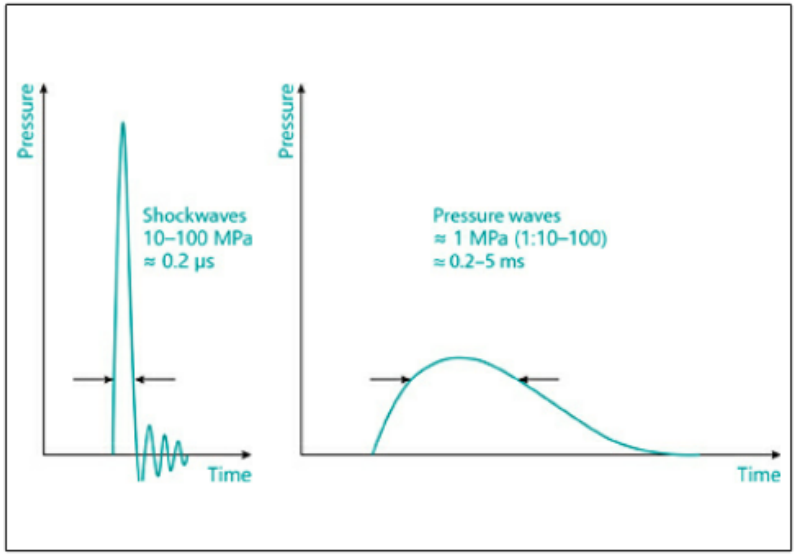


Fig. 3. Characteristic divergence between a shock-wave (SW; left), and a radial pressure pulse (right). Radial waves do implode to a negative pressure value (Figure 4), and is not depicted in this illustration. The negative pressure of radial waves occurs slower when compared to SWs. Image adapted from [84, 85].

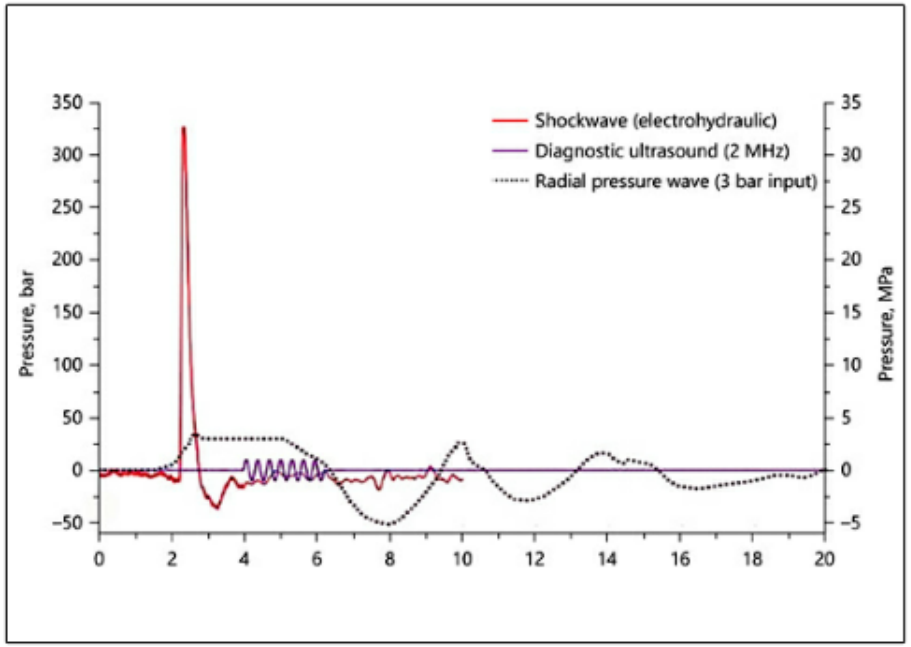


Fig. 4. Comparative characteristic divergence between an SW (Solid Red), ultrasound wave (Solid Blue), and a radial pulse wave (Dotted Black). Wave pattern, peak pressure, speed of wave rise time, and implosion. Image adapted from [85].

Radial Pressure Wave Technology

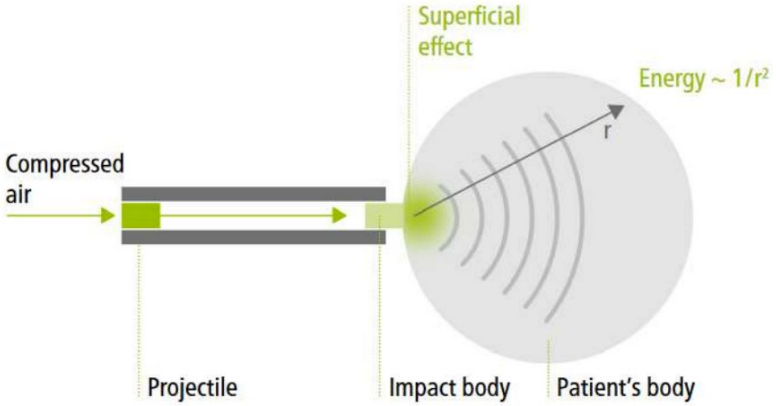
Creates the Effects of the Graston Technique

R-SW – Radial Pressure Wave

- Frequency 1 – 21 Hz
- Energy 1 – 5 bar
- Pulse transmitter multiple

Better Used for Chronic Conditions

Radial Pressure wave → **SLOW pulse** → **Low energy** → **SUPERFICIAL**

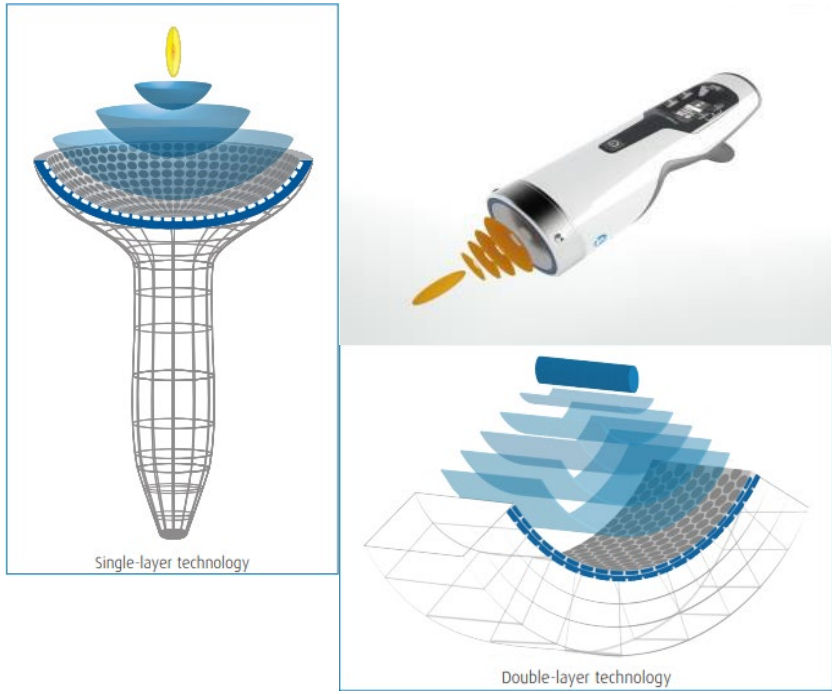


Focused Shock Wave Technology

(piezo electric crystals, electrohydraulic or electromagnetic cylinder coil)

F-SW – Focused Shock Wave

- Frequency 1 – 8 Hz
- Energy 0.01 – 0.55 mJ/mm²
- Energy 0.01 – 1.24 mJ/mm²
- Penetration depth 15 mm – 50 mm



Used for Acute & Chronic Conditions

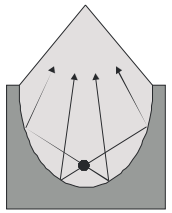
Focused shock wave → **FAST pulse** → **High energy** → **DEEP**

Shotgun Artillery Approach vs. Sniper

- If you prefer simple…
- Radial Pressure Waves are like the Shotgun Artillery approach which activates all tissues in its path.
- Focused Shockwaves are like a precise sniper, it only treats the damaged tissue in an efficient diagnostic way.

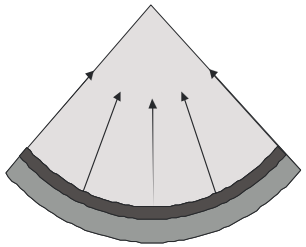
Focused Shock Wave Technology
How can focused shock waves be generated ?

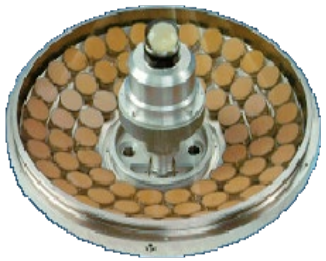
1980
Electrohydraulic
ellipsoid



Softwave TRT
Orthogold100
StemWave
Sanuwave

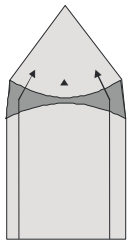
1985
Piezoelectric
sphere



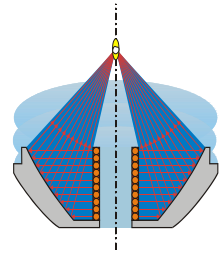


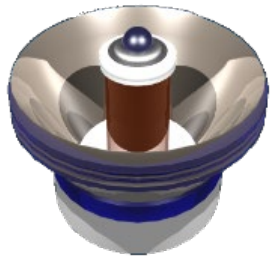
Piezowave2 Touch

1986
Electromagnetic
flat coil/lens



1989
Electromagnetic
cylinder
coil/paraboloid





Storz Duolith SD1 &
Chattanooga Intelect Focus

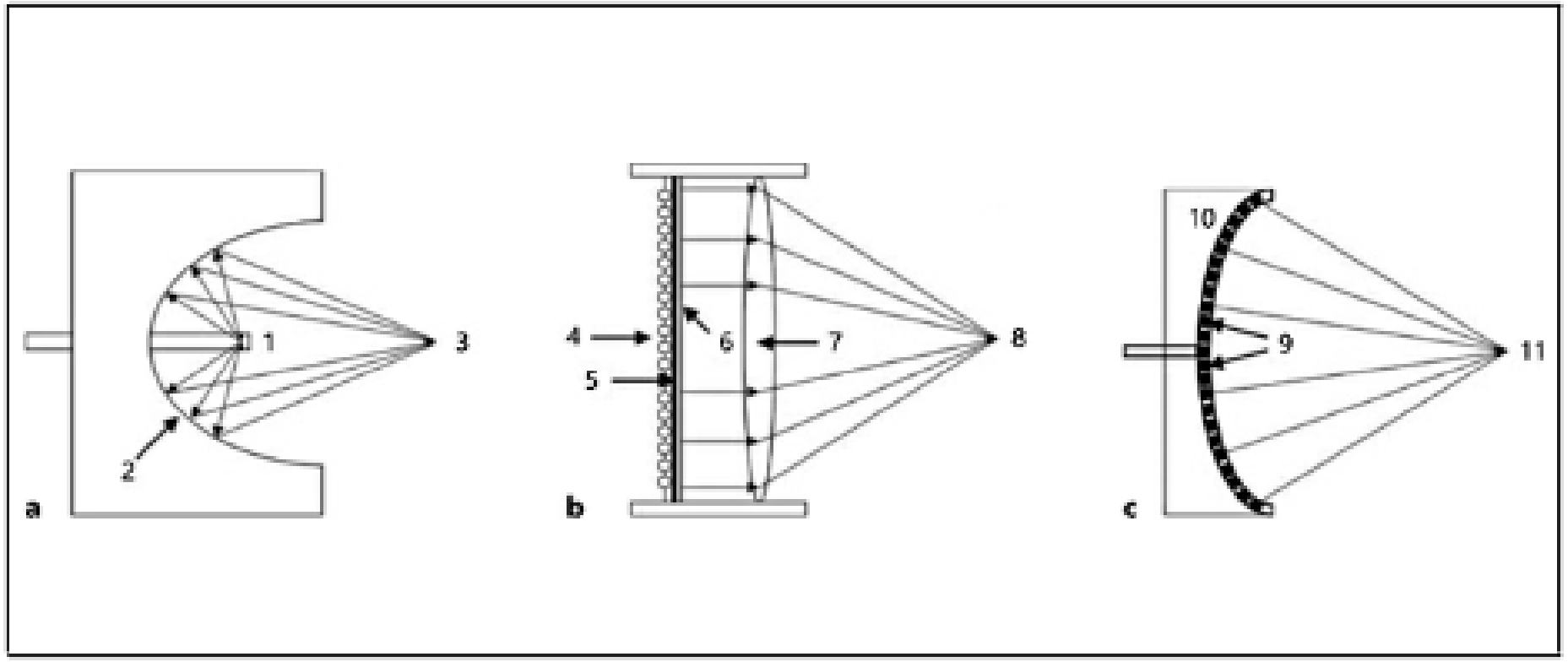
Radial Shock Wave Technology
How can radial pressure waves be generated ?

- Pneumatic & Electromagnetic
- Pneumatic- air compressor driven with a pipe and bullet, transmitter on the handpieces determine depth of penetration and surface area of treatment (Storz/Chatt/KDT Autowave)
- Electromagnetic- magnets are forced together into the transmit mer EnPuls P

Cleaning & Maintenance

- Radial Pneumatic (air compressor driven)
- Revision kit replacement every 1 million shocks, cleaning of the pipe daily or weekly, cleaning transmitter tips with alcohol wipes
- **Radial (Electromagnetic Zimmer)- canned air in cooling fans, alcohol to clean transmitters, replace transmitters when they mushroom down between 300k-1million shocks**
- Focused Shockwave
- **Piezoelectric Crystals (Piezowave2 Touch)- clean the gels pads and therapy sources daily, replace the therapy sources eventually between 20-30 million shocks**
- Electrohydraulic (Softwave/Stemwave)- Bleeding of the air line, replacement of the therapy source $2,500 every 100-300K shocks
- Electromagnetic Cylinder Coil (Paraboloid Storz/Chattanooga)- cleaning of the gel and water, maintenance and replacement of

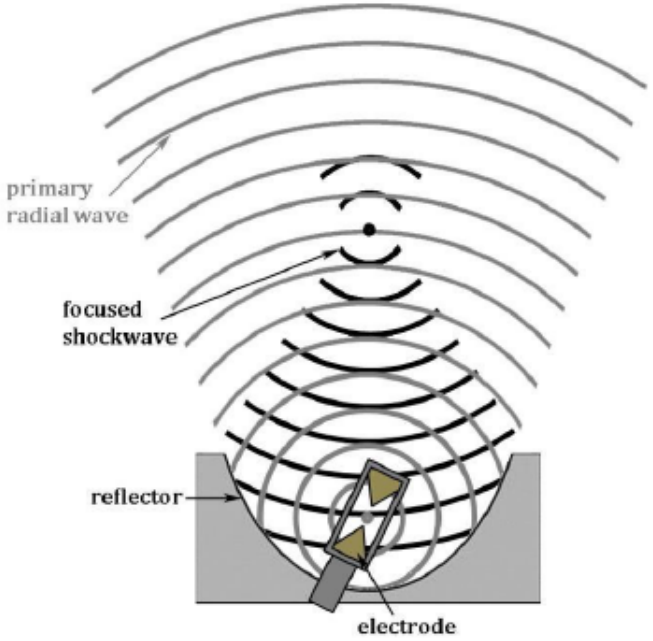
Shock wave Generation Technology



Electrohydraulic

Electrohydraulic Generation Principle

The oldest principle used in medicine is that in which the shock waves are generated by a spark plug, the electrohydraulic principle. The shock waves propagate in a medium (water) and are also focused in one place by a parabolic mirror.



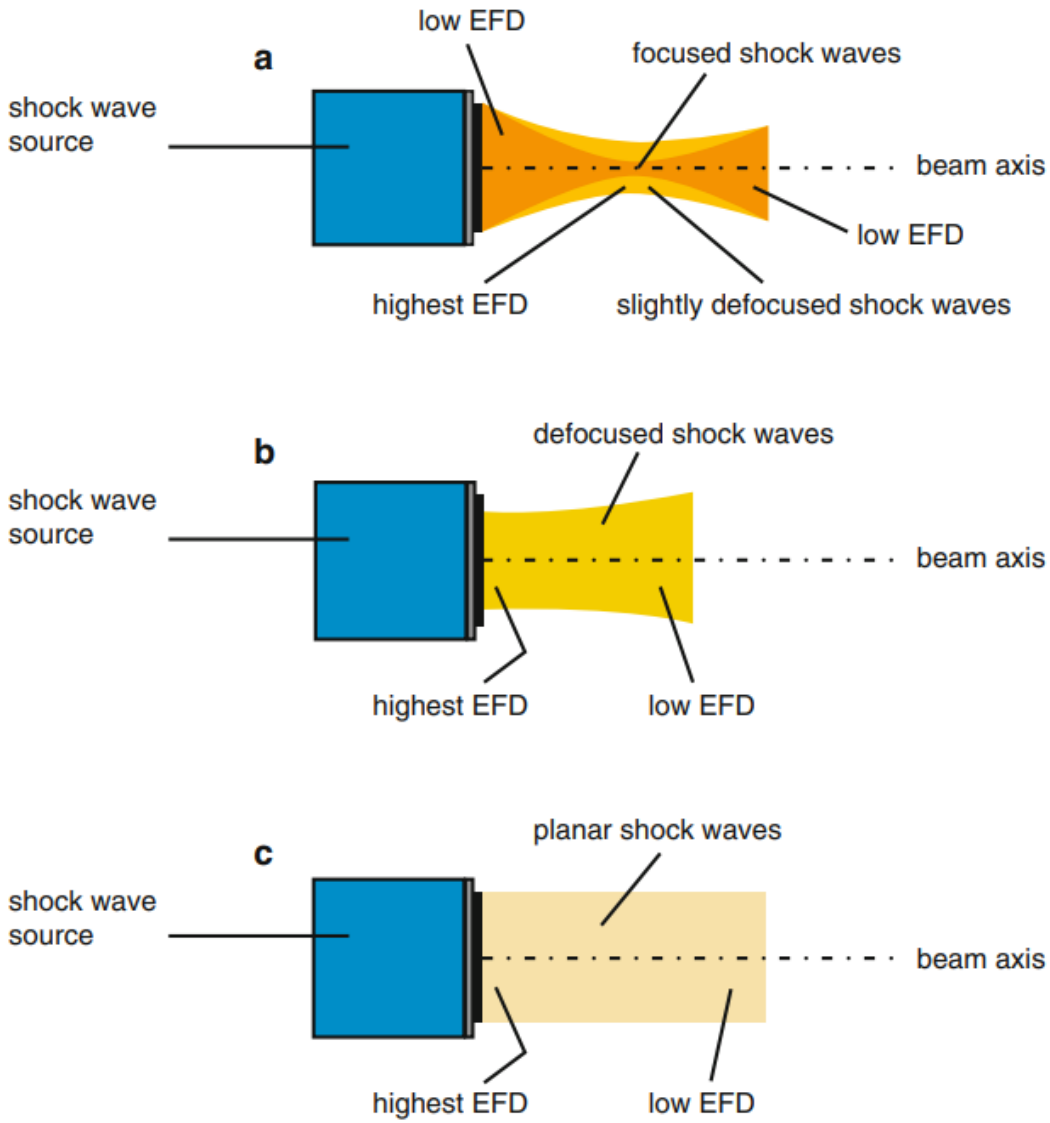


Fig. 6.1 Sketch of (**a**) a focused and a slightly defocused pressure wave beam, (**b**) a defocused beam, and (**c**) a plane beam

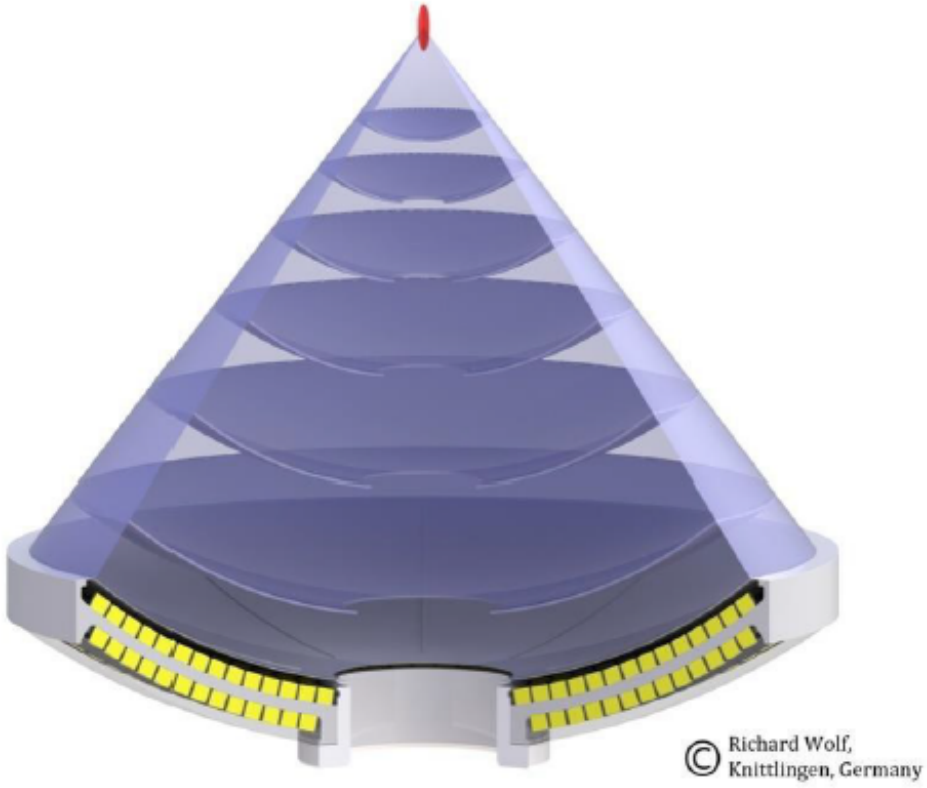
Focused shockwaves have the highest EFD at their focal depth

De focused have a more diffuse pressure distribution and get weaker as they travel

Piezoelectric

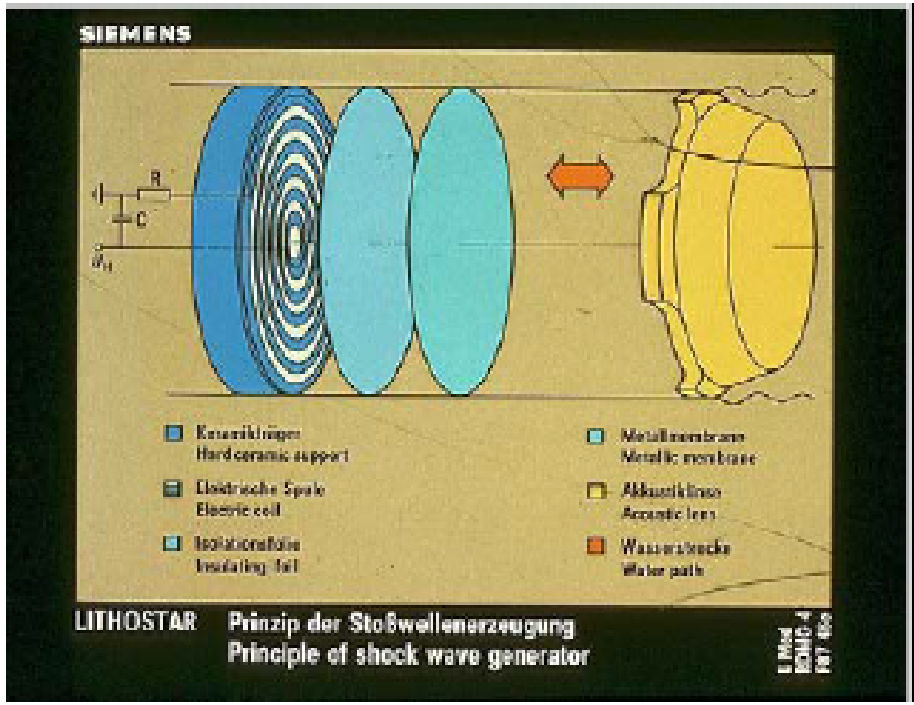
Piezoelectric Generation Principle

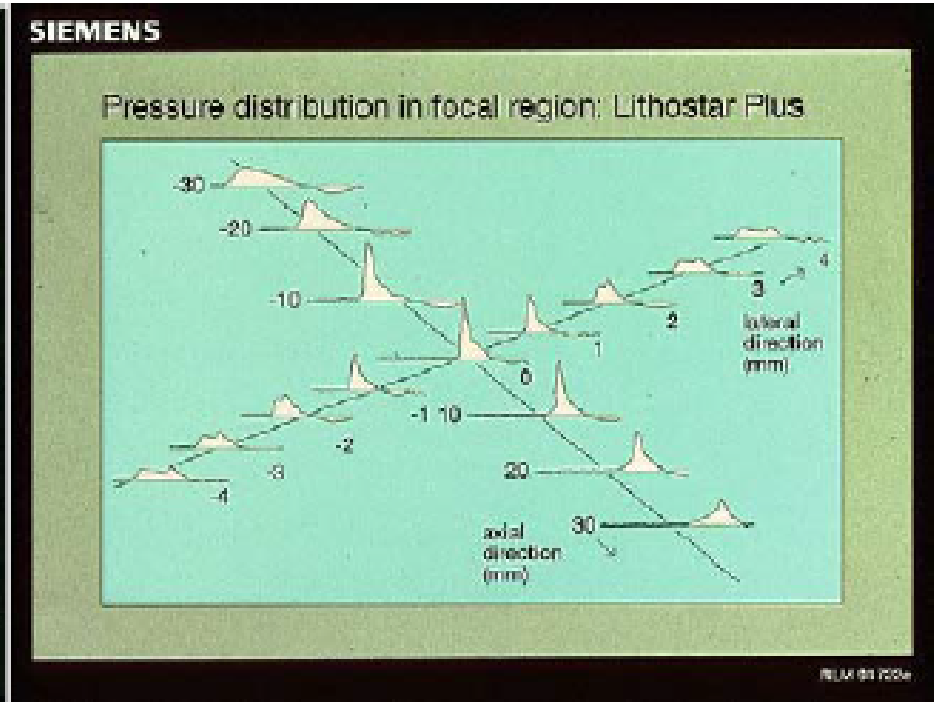
The piezoelectric principle is based on a focusing of pressure waves, which is produced by the electrical activation of piezoelectric crystals. The piezoelectric crystals are mounted in a shell in such a way that the pressure waves of each crystal meet in a focus.



Scheme of piezoelectric shock wave generation

Electromagnetic





A) The light blue membrane is deflected by the coil, which increases the pressure in the vessel between membrane and lens. The pressure is focused by the lens. B) One sees that only in the center (focus) the waves are divided and summed up in such a way that they get the classical shock wave form, outside the center there are more or less energetic pressure waves.

Shock Wave Characteristics

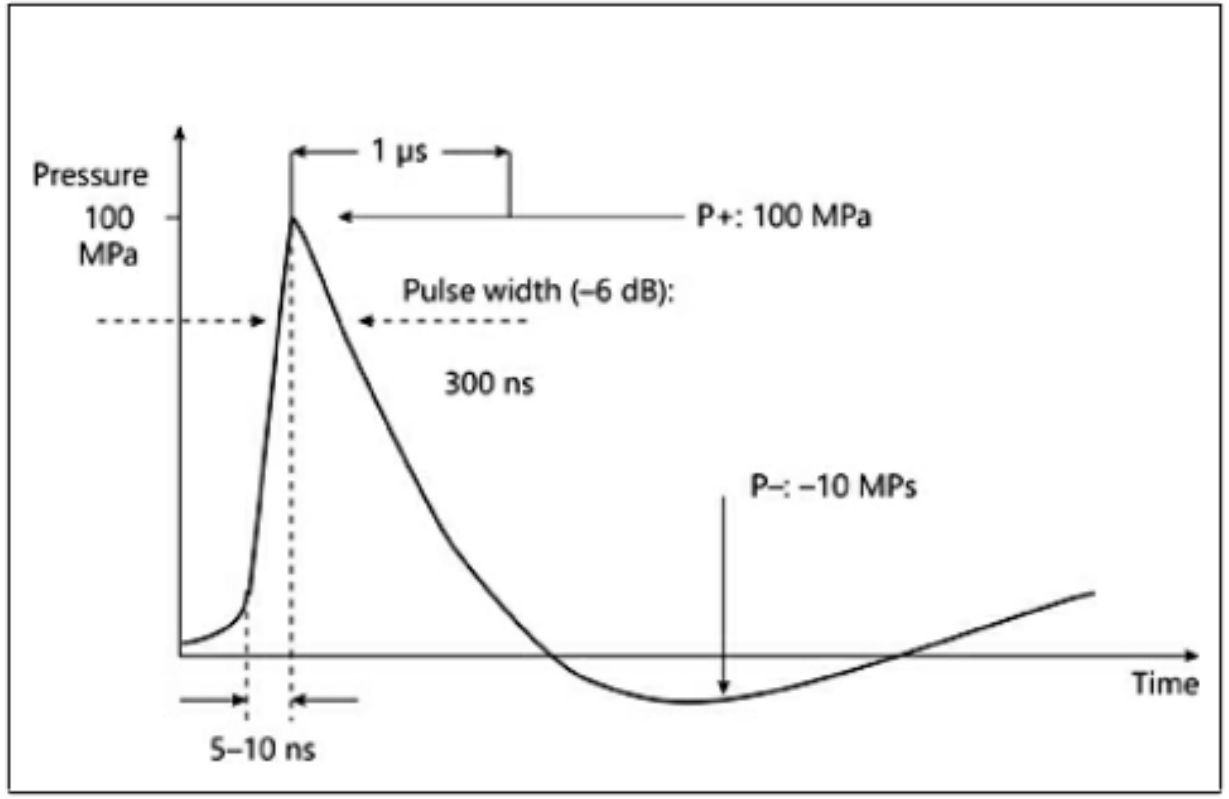
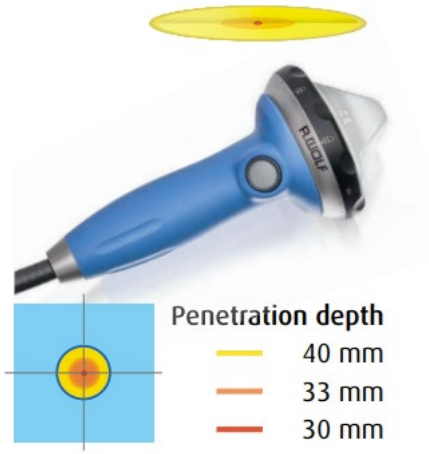


Fig. 1. Characteristics of a shockwave: phase 1: high pressure wave rise-time from basic ambient value, to a pressure value of approximately 100 MPa within <10 nanoseconds (ns). Phase 2: wave implosion to a negative pressure value of approximately –10 MPa within microseconds. Image adapted from [8].

Pertinent factors to consider when comparing SWT technology are ; pressure distribution , focal zone area , energy flux density , and the total energy concentration at the second wave (focal refraction) zone [28 , 29 , 34] .

Unfocused Shockwaves

- Electrohydraulic Parabolic Reflector design that creates a larger focal zone of treatment
- All focused shockwave devices have a focal zone where the energy is most concentrated to the center and the outside treatment zone which is referred to as the un-focal zone.
- Expensive device manufacturers use this for marketing to upsell buyers and convince the e the only machine th lts.



Yellow ring is the outside unfocused treatment zone

Pressure Values Explained

- 1 newton = 1 kilogram × 1 meter per second squared
- 1 joule = 1 newton × 1 meter
- 1 pascal = 1 newton / 1 square meter
- 1KPa=1 Bar
- To convert 100 megapascals (MPa) to atmospheres (atm), we can use the conversion factor:
- 1 MPa = 9.86923267 atm (approximately)
- Now, to find the equivalent value in atmospheres:
- 100 MPa × 9.86923267 atm/MPa ≈ 986.923267 atm
- So, 100 megapascals (MPa) is approximately equal to 986.92 atmospheres (atm).

Energy Flux Density (EFD)

- Measured in Millijoules Per square millimeter (mJ/mm2)
- Pinpoint 0.018 to 0.346 Max
- FBL Linear 0.007 to 0.139 Max
- Power: Intensity .1-18.0 Bars
- Frequency: 1-12 hz

Therapy source F7G3

Convenient therapy source with a small focal center

Focal characteristics and areas of application:

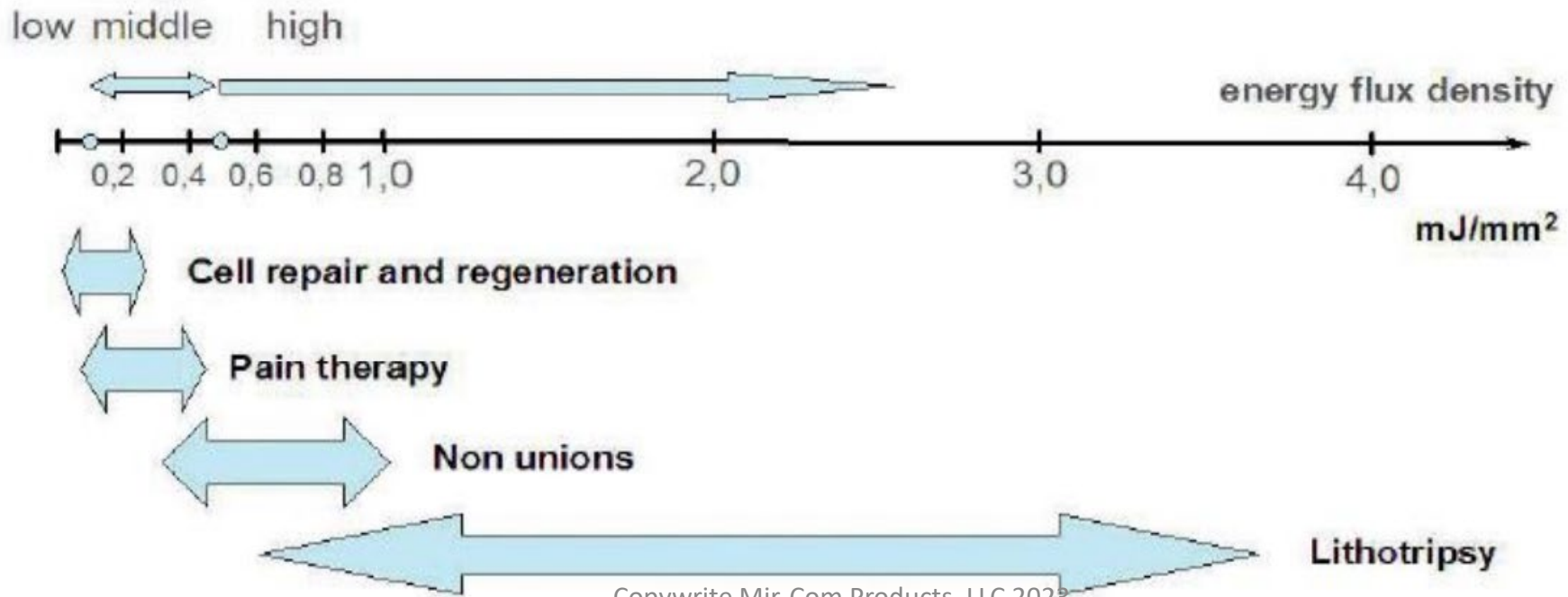
- small and precise focal zone to diagnose and locate pain points
- acute and chronic pain in muscles, tendons, and fascia due to muscle hardening and trigger points (e.g., in the upper and lower extremities, cervical, thoracic and lumbar spine, shoulder girdle, hip area)
- 30 mm penetration depth at the focal center
- Max. EFD: 0.146 mJ/mm²

LOW: Up to 0.08mJ/mm^2

MEDIUM :Up to 0.28mJ/mm^2

HIGH : > 0.28mJ/mm^2

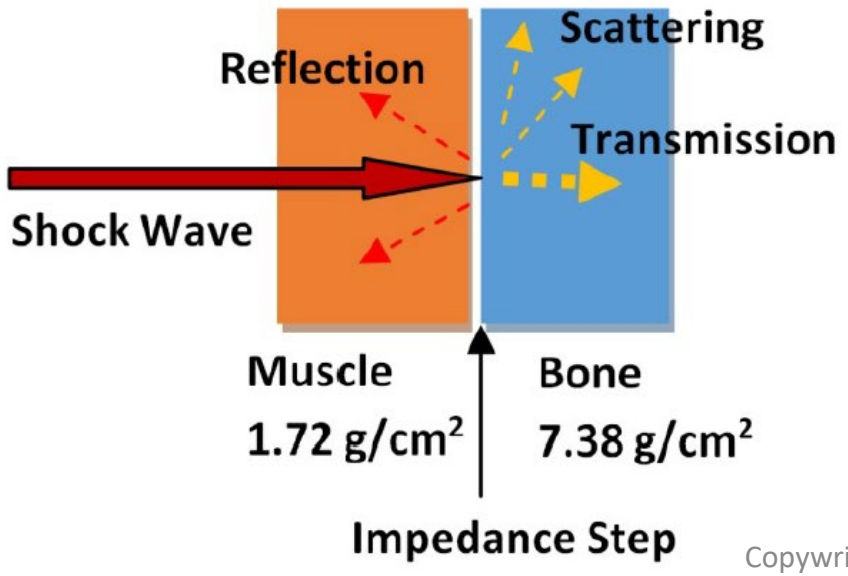
Rompe et al,1998



How do they move through the body?

Material	Density (kg / m^3)	Speed of Sound (m / s)	Acoustic Impedance (kg / m^2s)
Air	1.2	330	16
Water	1000	1437	1.44×10^6
Fat	970	1480	1.44×10^6
Muscle	1060	1570	1.66×10^6
Bones:			
Cortical	1700	3600	6.12×10^6
Cancellous	1000	1450	1.4×10^6

Table 1: The table shows how differently the properties of the media affect the shock waves. Impedance varies with water content of the tissue.



Acoustic impedance (Z) is defined as the product of the density (ρ) of the medium and the speed of sound (c) in that medium. Mathematically, it can be expressed as:

$Z = \rho \times c$

Where:

Z is the acoustic impedance (measured in Rayls, named after the British scientist Lord Rayleigh).

ρ is the density of the medium (in kilograms per cubic meter, kg/m^3).

c is the speed of sound in the medium (in meters per second, m/s).

Cavitation Bubbles

This is why patients experience the deep dull sensation

- The cavitation process begins when a sound wave passes through a liquid, causing the liquid pressure to fluctuate rapidly. If the pressure in certain regions of the liquid falls below the vapor pressure of dissolved gases or vapor nuclei present in the liquid, small gas bubbles start to form. These bubbles are called cavitation bubbles.

- PV=NRT
- P is the pressure of the gas (in pascals, Pa), V is the volume of the gas (in cubic meters, m^3), n is the amount of gas present (in moles, mol), R is the ideal gas constant (in joules per mole-kelvin, J/(mol·K)), and T is the absolute temperature of the gas (in kelvin, K).

Cavitation Bubbles

- As the sound wave continues to pass through the liquid, the pressure changes rapidly, leading to the growth of cavitation bubbles. During this growth phase, the bubbles can reach a significant size, but they remain stable as long as

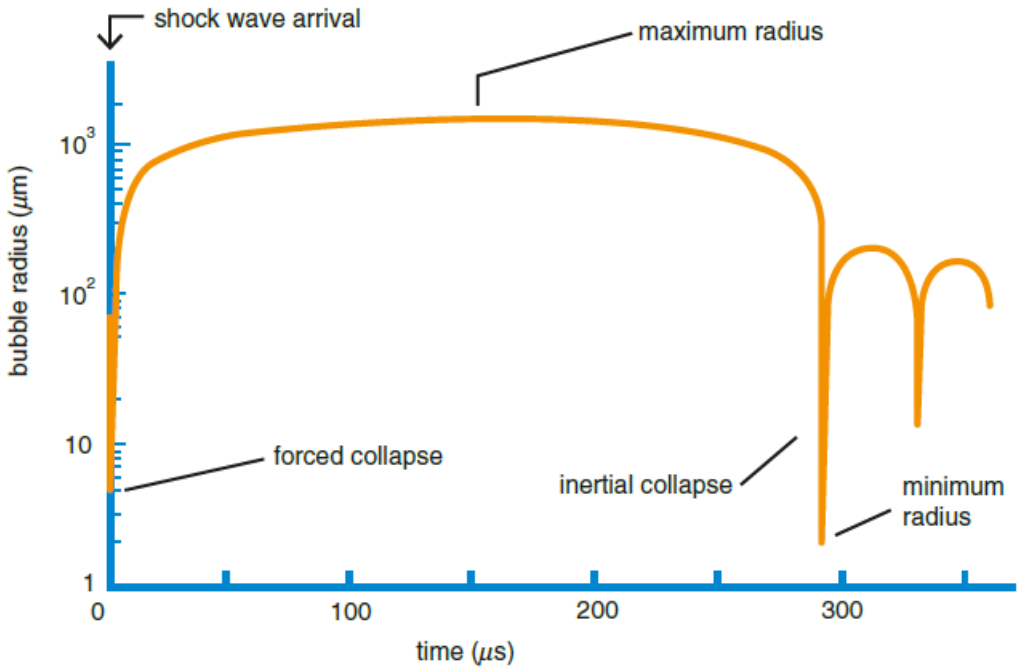


Fig. 4.8 Graph of a numerical simulation showing the variation of the radius of an air bubble in water (initial radius $R_0 = 0.07$ mm) exposed to a lithotripter shock wave ($p^+ = 100$ MPa). An abrupt forced bubble collapse is followed by an expansion and a second collapse. The second collapse occurred approximately 290 μs after arrival of the shock wave at $t = 0$. The bubble rebounds several times (not shown completely) until reaching equilibrium. (Courtesy of M. de Icaza-Herrera)

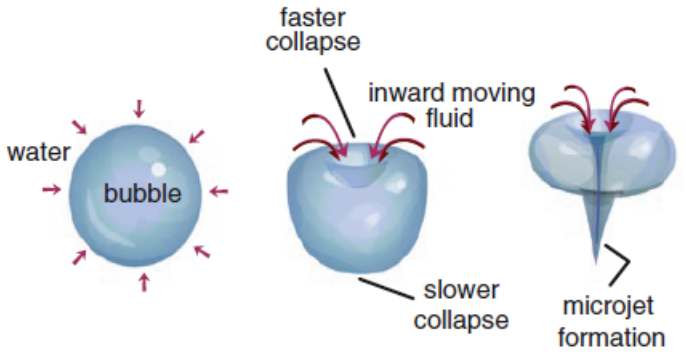


Fig. 4.9 Schematic of a collapsing microbubble in water and microjet emission after shock wave passage. Because an interface (not shown) is close to the bottom of the bubble, the bubble involutes from the top and develops a funnel-shaped protrusion and a fluid microjet in the direction of the boundary. Adapted from Wess (2004)

However, when the sound wave intensity decreases or changes direction, the pressure in the liquid surrounding the bubbles increases, causing them to collapse or implode. The collapse of the cavitation bubbles is a highly energetic event, generating localized shock waves and intense heat. The temperature at the center of the collapsing bubbles can reach **several thousand degrees Celsius** for a very short time.

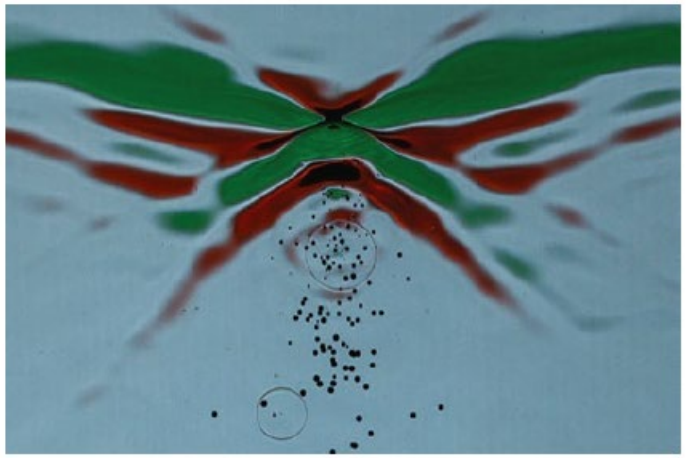
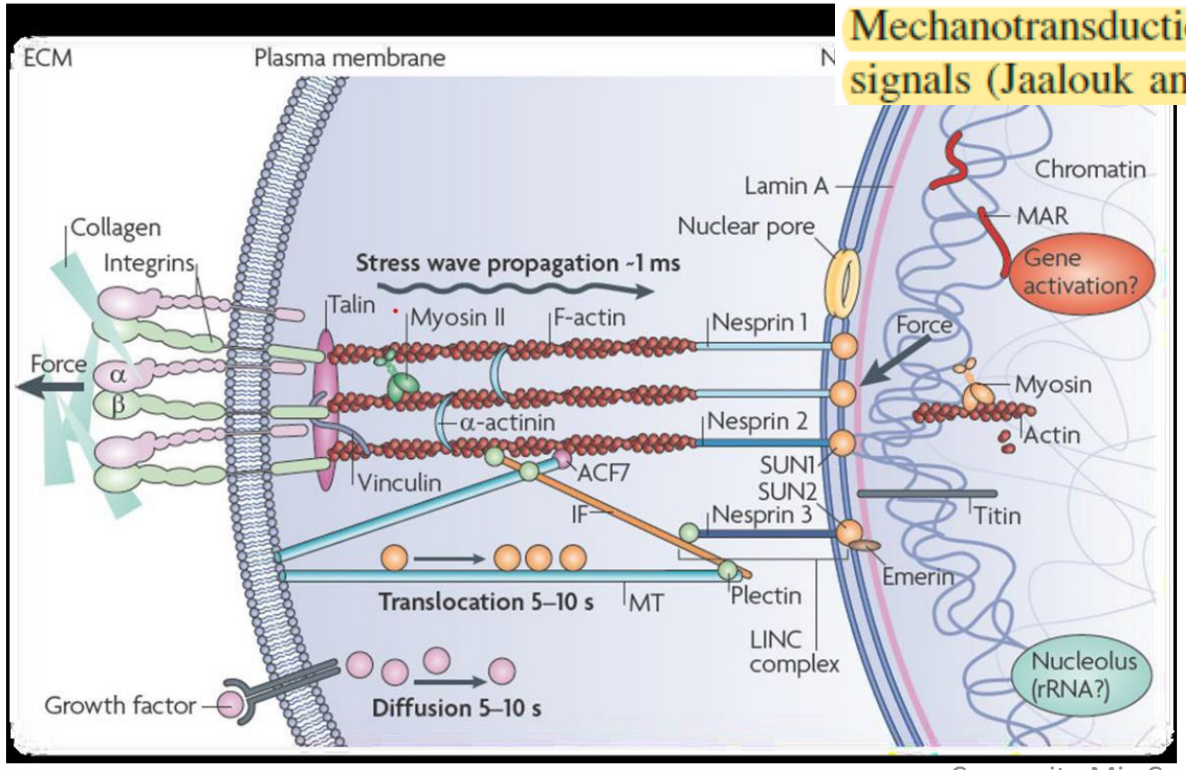


Fig. 4.10 Details of a focused shock wave field in water, showing generation of cavitation bubbles (*black dots*) in the negative pressure area. Bubbles grow and collapse, radiating spherical shock waves. Technique: color-schlieren optics displaying positive pressure gradients in red und negative pressure gradients in green (*eBook*) (Photograph: O. Wess and J. Mayer, Storz Medical AG, Tägerwilen, Switzerland)

Mechano-transduction

Effects of ESWT are related to biological reactions to mechanical stimulations (Suhr and Bloch 2012; Bloch and Suhr 2014; d'Agostino et al. 2015). Both direct and indirect pressure wave related phenomena generate biological responses. Biological tissue has the capability to sense different types of stress and transmit the information into the cellular system. The process, referred to as *mechanotransduction* has been observed in tendons, skeletal muscle, cartilage, endothelium, and connective tissue. Mechanotransduction involves the translation of physical stimuli into biochemical signals (Jaalouk and Lammerding 2009). A mechanoreceptor allows bone cells to



Mechanotransduction

The cytoskeleton structure of mammalian cells are able to detect and translate forces in minute nano-Newton ranges,

and play a pertinent role in homeostasis (i.e., stem cell differentiation) and in diseaseneoplastic processes) [125–128]. Protein folding, bone shaping, muscle contraction and regeneration, hearing, touch, lung regulation, and circulatory pressure are all in principle regulated by transducers that sense, respond, or react to this mechanosensory feedback complex.

Mechanotransduction Effects

- Increase vessel wall permeability resulting in diffusion of cytokines.
- Triggers Mitotic activity
- Collagen synthesis
- TGF-B1
- Nitric Oxide
- Vascular endothelial growth factor (VGEF)
- Bone morphogenic protein (BMP)
- Osteogenic protein
- Turn on mechanosensitive ion channels

Research Suggests Stem Cell Activation & Connective Tissue

Extracorporeal shock waves enhance normal fibroblast proliferation in vitro and activate mRNA expression for TGF-β1 and for collagen types I and III

Background and Purpose: Extracorporeal shock waves (ESWs) are used to good effect in the treatment of soft tissue injuries, but the underlying mechanisms are still unknown. We therefore determined the effects of ESWs on normal fibroblasts in vitro, in order to assess treatment-induced cell response.

Methods: A normal human fibroblast cell line (NHDF-12519) was treated with ESWs generated by a piezoelectric device (Piezoson 100; Richard Wolfe) using different protocols of impulses (300, 1,000, or 2,000 shots) and energy (0.11 or 0.22 mJ/mm2). Untreated controls and treated cells were cultivated for 12 days following a single shock-wave treatment. Viability, growth rate, and expression of mRNA for TGFβ-1 and collagen types I and III were evaluated at days 3, 6, 9, and 12.

Results:1 hour after shock-wave treatment, cell viability showed a decrease related mainly to impulse numbers applied. Fibroblasts treated with energy of 0.22 mJ/mm2 subsequently showed an increase in proliferation from day 6 to day 9 that was higher than in untreated controls, without interference with the normal cell kinetic profile. mRNA expression was also higher in treated fibroblasts than in untreated controls for TGFβ-1 on day 6 and day 9, for collagen type I on day 6, and for collagen type III on day 9.

Interpretation: These in vitro data confirm that the main factors involved in the repair process of connective tissues are activated by ESWs. The study gives the rationale for, and may provide schedules for, ESW treatment of tendinopathies.

Fibrosis

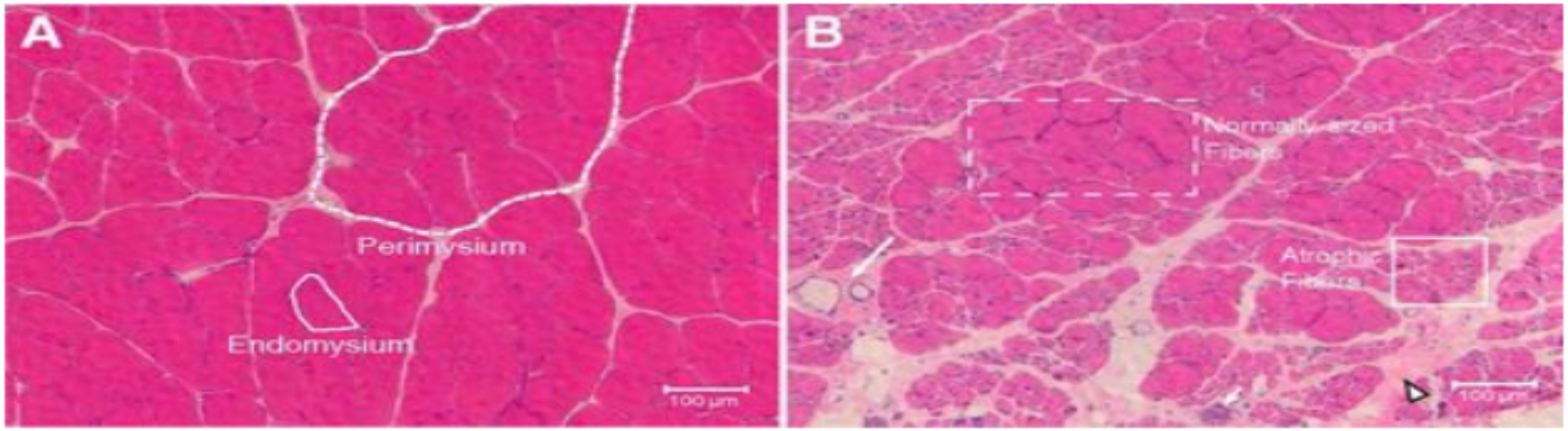
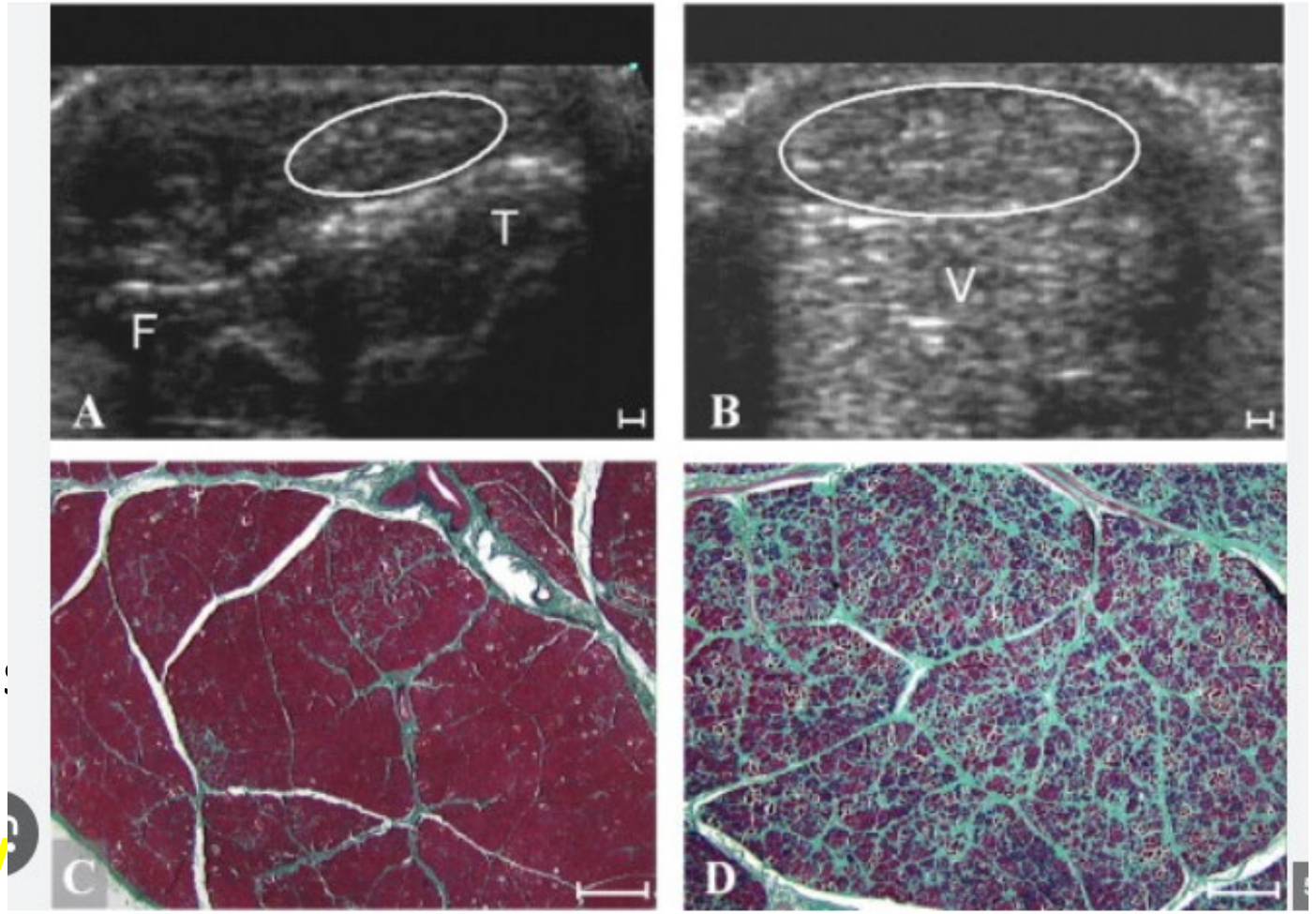


Fig. 1.

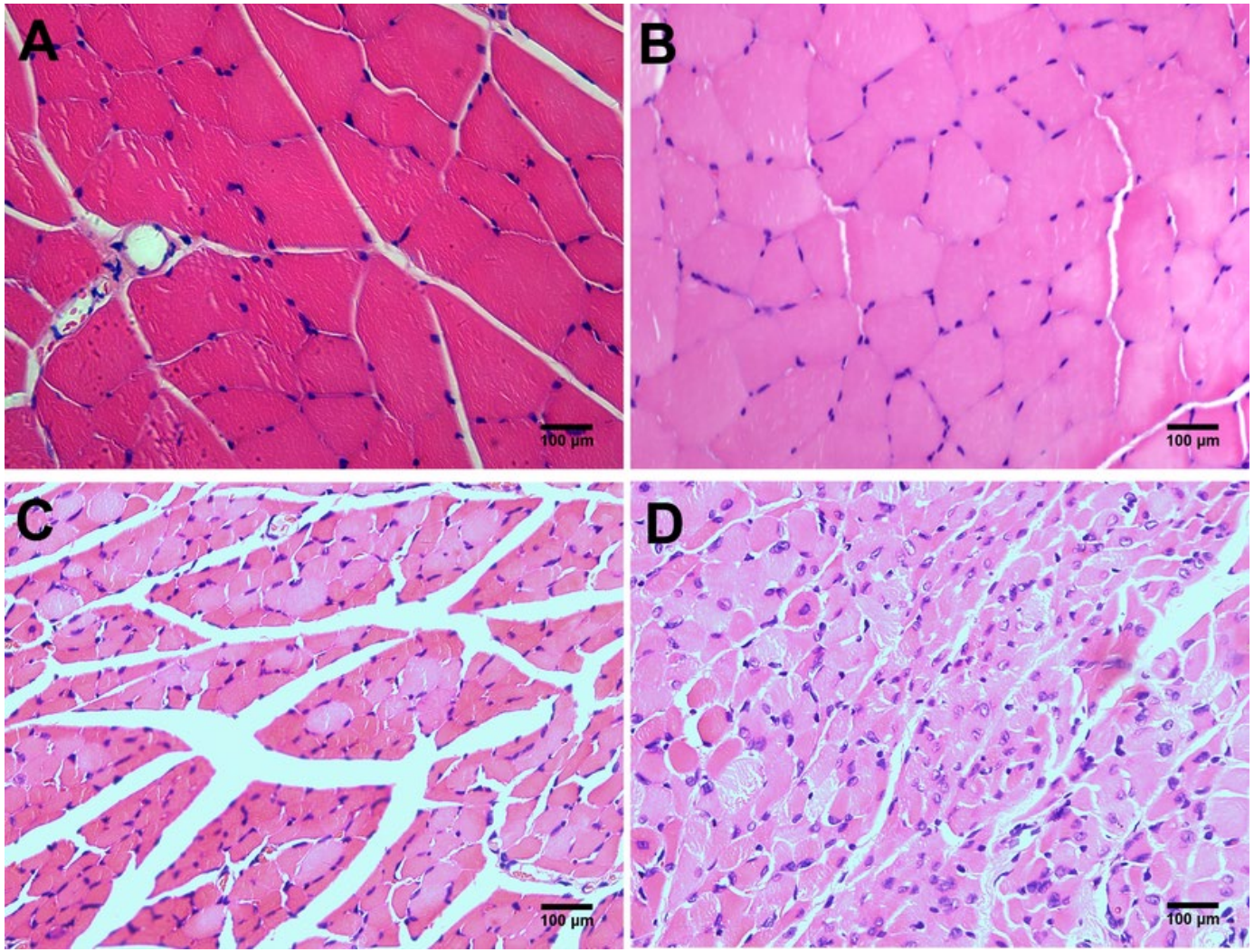
A: micrograph of a cross section of healthy skeletal (rat tibialis anterior) muscle demonstrating normal morphology that consists of tightly packed polygonal fibers with a small amount (~5%) of extracellular material. Traditional location of endomysial connective tissue is outlined with a solid line; perimysial tissue is outlined with a dashed line. However, as can be seen elsewhere in the micrograph, this distinction can be arbitrary. *B*: micrograph of a cross section of skeletal muscle demonstrating fibrotic morphology in which extracellular material is increased to ~20% of the cross section, fibers are loosely packed, extracellular space is hypercellular, and fiber sizes are highly variable. This muscle was injected twice with botulinum toxin type A (Botox, Allergan; 6 U/kg in 100 µl) at a 3-mo interval and tested after 6 mo.

Fibrosis

- Occurs due to poor healing
- Over production of the extra cellular matrix
- Poor tissue function
- Diminished circulation
- Many of the target pathologies treated with ESWT involve fibrosis
- Fibrotic tissue has different **acoustic impedance as shown by its** appearance on ultrasound.

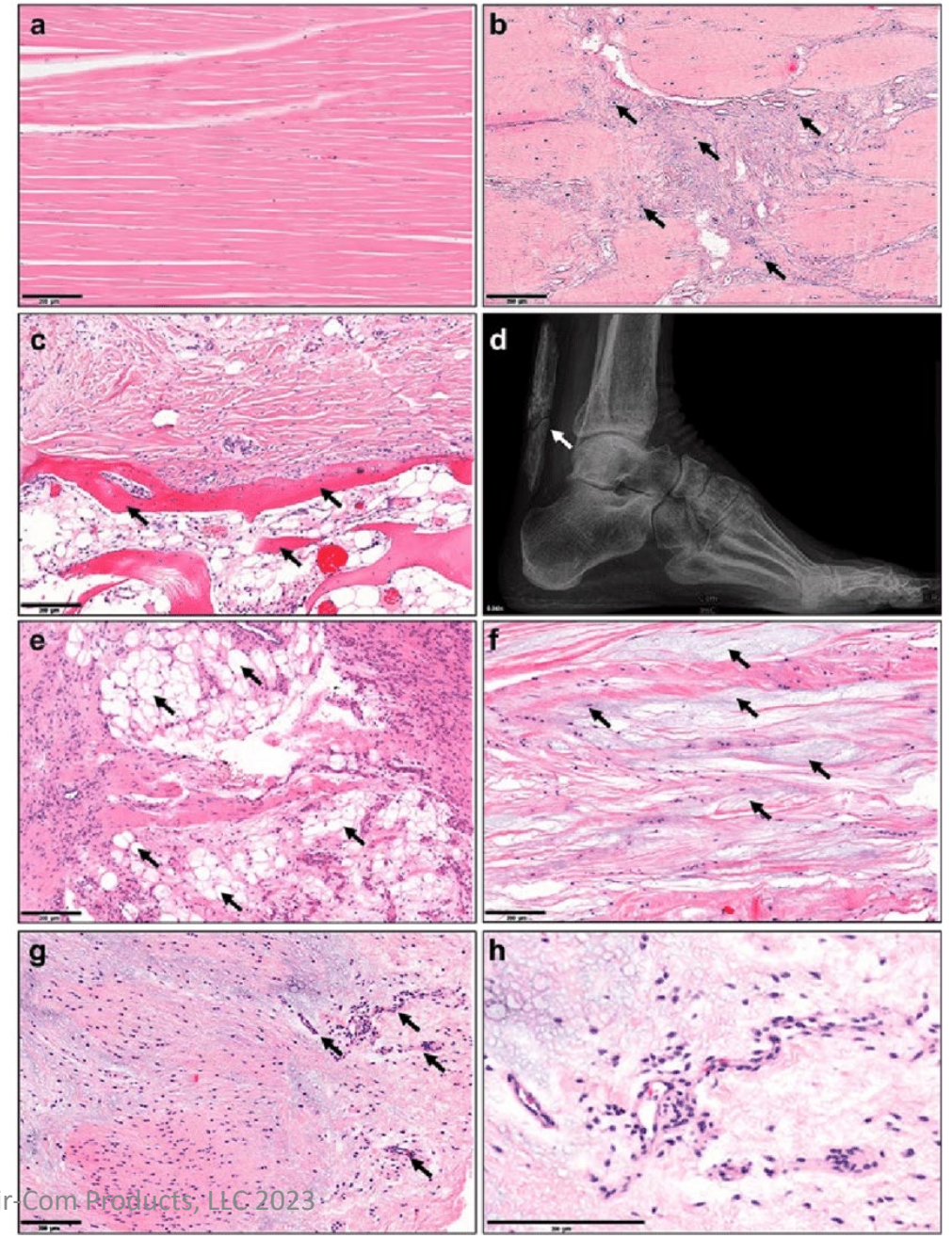


Muscle



Tendinopathy

Tendinopathy-related histopathological characteristics in human tendon tissue. Hematoxylin-eosin staining of (a) normal tendon tissue and abnormalities such as the (b) presence of chondrocytes, (c) ossification, (d) calcification of the Achilles tendon (X-ray image), (e) accumulation of adipocytes, (f) myxoid (or mucoid) degeneration and (g) hypervascularization. (h) Image g) 10 x zoomed in. Scale bars: 200 µm. Arrows indicate the corresponding feature per image. Images derived from the histopathological archive of Prof. Christoph Brochhausen, Institute of Pathology, University Regensburg.



TGF-B

- The primary effect of transforming growth factor-beta (TGF-β) is its role in regulating cellular processes involved in tissue development, growth, differentiation, and repair. TGF-β is a multifunctional cytokine that can influence a wide range of biological activities in various cell types. Its primary effects include:
- Cell Growth Inhibition: TGF-β can act as a negative regulator of cell growth by inhibiting cell proliferation. It can induce cell cycle arrest at different phases, preventing cells from dividing excessively. This effect is essential for maintaining tissue homeostasis and preventing uncontrolled cell growth, such as in cancer.
- Induction of Cell Differentiation: TGF-β plays a crucial role in promoting the differentiation of precursor cells into specialized cell types. It can direct the differentiation of stem cells into specific lineages during embryonic development and tissue repair.
- Regulation of Immune Responses: TGF-β is involved in immune system regulation. It can suppress the function of certain immune cells, modulate the production of inflammatory cytokines, and promote regulatory T cell development, which helps maintain immune tolerance and control inflammation.
- Extracellular Matrix Production and Remodeling: TGF-β stimulates the synthesis of extracellular matrix components, such as collagen and proteoglycans, and promotes tissue remodeling. This is essential for tissue repair, wound healing, and maintaining tissue integrity.
- Epithelial-Mesenchymal Transition (EMT): TGF-β can induce EMT, a process where epithelial cells lose their characteristics and acquire mesenchymal properties. EMT is critical in embryogenesis, tissue repair, and cancer metastasis.
- Angiogenesis: TGF-β can stimulate the formation of new blood vessels (angiogenesis) by influencing endothelial cell behavior. This is crucial for tissue growth and repair, as well as in pathological conditions such as cancer growth.
- Inflammation and Fibrosis: TGF-β plays a role in promoting tissue repair and fibrosis in response to injury or damage. However, excessive or prolonged TGF-β activity can lead to pathological fibrosis in certain organs.

Nitric Oxide

- Nitric oxide (NO) is a small gas molecule that serves as a signaling molecule in various physiological processes throughout the body. It plays a critical role in regulating the function of connective tissue and has several effects on these tissues:
- Vasodilation: NO is a potent vasodilator, meaning it relaxes and widens blood vessels. In connective tissues, including blood vessel walls, NO promotes vasodilation, leading to increased blood flow. This effect is crucial in regulating blood pressure and maintaining adequate oxygen and nutrient supply to tissues.
- Immune Regulation: NO has immunomodulatory effects and can influence the function of immune cells in connective tissues. It can act as a defense mechanism against infections by promoting the destruction of invading pathogens and modulating the immune response.
- Regulation of Extracellular Matrix: NO can affect the synthesis and degradation of the extracellular matrix components, such as collagen and proteoglycans, in connective tissues. It may influence the balance between matrix production and breakdown, contributing to tissue remodeling and repair processes.
- Anti-inflammatory Effects: NO can act as an anti-inflammatory molecule, inhibiting the production of pro-inflammatory cytokines and chemokines in connective tissues. It helps to regulate the inflammatory response and prevent excessive tissue damage during inflammation.
- Wound Healing: NO plays a role in wound healing and tissue repair. It promotes the migration and proliferation of fibroblasts, which are responsible for producing new collagen and extracellular matrix components in the healing process.
- Bone Remodeling: In bone tissue, NO can affect osteoblasts and osteoclasts, the cells responsible for bone formation and resorption, respectively. It can influence bone remodeling and maintenance of bone density.
- Cartilage Homeostasis: NO has been implicated in the regulation of chondrocyte function in cartilage. It may influence the synthesis and degradation of cartilage components, affecting cartilage homeostasis and function.

Prostaglandin E2

- Inflammation: PGE2 is a pro-inflammatory mediator, meaning it promotes inflammation. In response to tissue injury or infection, PGE2 is produced and released by various cells, including immune cells and fibroblasts. PGE2 induces vasodilation, increasing blood flow to the affected area, and enhances the permeability of blood vessels, allowing immune cells and inflammatory mediators to enter the tissue, leading to redness, heat, and swelling.
- Immune Response: PGE2 modulates the immune response by influencing the function of immune cells. It can stimulate the production of cytokines and chemokines, which attract and activate immune cells to the site of inflammation. PGE2 can also influence the behavior of immune cells, such as macrophages and T cells, promoting inflammation and tissue repair.
- Angiogenesis: PGE2 can stimulate angiogenesis, the formation of new blood vessels. In connective tissues, angiogenesis is

Prostaglandin E2

- promotes the release of vascular endothelial growth factor (VEGF), which plays a central role in angiogenesis.
- Modulation of Extracellular Matrix: PGE2 can influence the synthesis and remodeling of the extracellular matrix components in connective tissues. It can stimulate the production of collagen and other matrix molecules by fibroblasts, helping to support tissue repair and wound healing.
- Pain and Sensitization: PGE2 sensitizes nerve endings, contributing to the perception of pain. In inflamed tissues, PGE2 lowers the threshold for pain perception, making the affected area more sensitive to pain stimuli.
- Cartilage Degradation: In some inflammatory conditions, high levels of PGE2 can contribute to cartilage degradation. PGE2 can stimulate the production of matrix metalloproteinases (MMPs), enzymes that break down the extracellular matrix, including cartilage components, leading to joint damage in conditions such as osteoarthritis.

COX-2

- Prostaglandin Production: COX-2 is responsible for catalyzing the conversion of arachidonic acid to prostaglandins, including prostaglandin E2 (PGE2). Prostaglandins, especially PGE2, are potent inflammatory mediators that play a central role in promoting inflammation, vasodilation, increased vascular permeability, and the recruitment of immune cells to the site of injury or inflammation.
- Amplifying Inflammatory Response: COX-2-derived prostaglandins, particularly PGE2, can amplify the inflammatory response in connective tissues. They stimulate the synthesis and release of various pro-inflammatory cytokines and chemokines, further recruiting and activating immune cells, such as neutrophils and macrophages, which contribute to tissue damage.

COX-2

- Angiogenesis: COX-2 is involved in promoting angiogenesis, the formation of new blood vessels, in response to tissue injury or repair. Angiogenesis is essential for providing oxygen and nutrients to the injured area and supporting tissue regeneration.
- Extracellular Matrix Remodeling: COX-2 can influence the remodeling of the extracellular matrix in connective tissues. It can promote the production of matrix metalloproteinases (MMPs), which are enzymes that break down the extracellular matrix components, including collagen and proteoglycans. This process is important for tissue repair and remodeling but can become detrimental if excessive, contributing to tissue damage and diseases like osteoarthritis.
- Pain Sensitization: COX-2-derived prostaglandins, particularly PGE2, can sensitize nerve endings, leading to an increased perception of pain in inflamed tissues.

Substance P 17

- Neurogenic Inflammation: Substance P plays a role in neurogenic inflammation, which is a type of inflammation initiated by the activation of sensory nerve fibers (nociceptors). When tissues are injured or inflamed, sensory nerve fibers release Substance P, which contributes to vasodilation, increased vascular permeability, and the release of other inflammatory mediators, such as histamine. These changes can promote the recruitment of immune cells to the inflamed area and influence the function of nearby connective tissue.
- Pain Sensation: Substance P is a key neuropeptide involved in transmitting pain signals from the periphery to the central nervous system. By binding to its receptors (neurokinin 1 receptors) on sensory nerve fibers, Substance P enhances the transmission of pain signals, leading to the perception of pain. In the context of connective tissue, this can be relevant for conditions like arthritis, where inflamed tissues may produce Substance P, contributing to pain sensation.
- Modulation of Immune Responses: Substance P can influence immune cell function in connective tissues. It can stimulate the release of cytokines and chemokines by immune cells, leading to an amplification of the inflammatory response. Additionally, Substance P can attract immune cells to the site of inflammation, which can affect tissue repair and remodeling.

Substance P 17

- Wound Healing: Substance P has been implicated in wound healing processes, including the regulation of cell migration, proliferation, and extracellular matrix synthesis. It can influence fibroblasts, which are key cells involved in connective tissue repair and wound healing.
- Effects on Mast Cells: Substance P can activate mast cells, which are immune cells that play a role in the immune response and inflammation. Activation of mast cells can result in the release of various inflammatory mediators that can impact connective tissue function.

- VEGF (vessel endothelial growth factor)

- Angiogenesis: VEGF stimulates the formation of new blood vessels (angiogenesis) from pre-existing ones. During development, wound healing, and tissue repair, angiogenesis is essential for providing oxygen and nutrients to growing tissues. It also plays a critical role in tissue regeneration and recovery after injury.
- Vascular Permeability: VEGF increases the permeability of blood vessel walls, allowing various substances, such as immune cells and growth factors, to move in and out of blood vessels more easily. This increased vascular permeability is vital in inflammatory processes and immune responses, as it facilitates the migration of immune cells to sites of injury or infection.
- Vasodilation: VEGF can induce the relaxation and dilation of blood vessels, leading to increased blood flow to specific tissues. This function is essential in maintaining proper blood supply to various organs and tissues and regulating blood pressure.
- Cell Survival and Proliferation: VEGF promotes the survival and proliferation of endothelial cells, which form the inner lining of blood vessels. This effect is crucial during angiogenesis, as it supports the growth and expansion of new blood vessels.
- **Tissue Repair and Regeneration**: VEGF plays a significant role in tissue repair and regeneration after injury or damage. By promoting angiogenesis and stimulating cell proliferation, VEGF aids in the restoration of damaged tissues and enhances the healing process.

Big Picture

- Shockwaves when applied to the human body will rapidly change the pressure of the tissue.
- The rapidly changing pressure will stimulate mechanoreceptors over a large area resulting in a predictable biochemical and physiological response.
- Rapid pressure changes will also disrupt extra cellular matrix.
- The net effect is known as mechanotransduction.
- Mechanotransduction is the desired effect of ESWT.
- Mechanotransduction will lead to tissue remodeling and regeneration.
- You don’t want too much power.

Focused Contraindications and Why?

Contraindications for MyACT

In principle, MyACT is rarely contraindicated. The most recent information on contraindications is given in the current MyACT Operating Manuals. The Operating Manuals for therapy sources manufactured by Richard Wolf list the following contraindications:

- Infections
- Tumor tissue
- Blood clotting disorders (it may necessary to check the patient's coagulation status)
- Taking blood thinning medication
- Pregnancy
- Lung tissue in the focal area
- MyACT should not be used to treat the head
- Air-containing organs in the focal area such as the gastrointestinal tract, etc.

MyACT systems are only approved for use by trained medical specialists and may only be operated by qualified persons who have been trained in the application of MyACT for medical purposes. Before commencing treatment, the therapist must decide, based on the patient's general condition, whether a planned application should be carried out or not. For further information, please consult the recent specialist literature.

Radial Contraindications and Why?

General Contraindications:

1. Pregnant Women (abdomen, pelvis, and waist areas)
2. Post operation (wound, exudation)
3. Osteoporosis
4. Growing children (growth plates)
5. Coagulation defects
6. Bleeding tendency
7. Patient with pacemaker (safe to treat anywhere except over the chest or thoracic region)
8. Neoplasms
9. Cutaneous Infections
10. Lung area
11. Spine (Cervical, lumbar, and thoracic vertebrae)
12. Cortisone therapy up to 6 weeks before first treatment
13. Mental Disorder

Possible Side Effects:

- Reddening
- Swelling
- Pain
- Hematoma
- Petechiae, Red Spots
- Skin Lesions After Previous Cortisone Therapy

Treatment

Axiom: " the more you understand the exact pathology of your patient, the higher the chances of a positive outcome. "

Main indications for ESWT

- Primary categories:
  - Tendon
  - Ligament
  - Joint pain
  - Trigger Points
  - Muscle -clinical focus
  - Articular - focus on sub chondral bone
  - Bone fracture
  - Bone vascular
  - Skin wound healing
  - ED
  - Peyronie’s disease
  - Cardiovascular

Shockwave Techniques

- ***Flare Up vs. Wanding***

- Acute pain has been defined as pain with duration of less than 2-3 months while chronic pain has a duration of longer than 6 months.
- ***For Acute Pain*** , your clinical goal is not to flare up and inflame the tissue but to ***activate it gently or wanding technique*** in order to provide healthy fluid flows, oxygen and to reduce pain and edema through its mechanical mechanisms by low intensity sound compression manipulations***.*** We want to mechanically flush fluids with higher frequencies.
- ***For Chronic Pain ,*** you want to ***search and destroy or flare up***. This means adjust the frequency and power higher with communication from the patient until you find uncomfortable spots. Once you find that spot turn the power bar up until the patient is a 7/10 on the uncomfortable scale then hold it there until it starts to ease up or 1000 shocks maximum. You must be very attentive and keep communicating with the patient to ensure that they are able to tolerate the treatment without too much anxiety or pain. If its not easing up go off of that spot and come back to it later.
- The goal with acute pain is to reduce inflammation and chronic pain we want to flare it up to create inflammation and break up scar tissue which will make the body respond to that area and heal it.

Shockwave Techniques

- Clinical Focusing Technique
- Anatomical Sweep
- Radial Shockwave- apply pressure and work the tissue origin to insertion. Apply pressure and treat with small circles over the tendon. 500 shocks to the tendon/joint then 1500 shocks for the myofascial connective tissue.
- Think Graston Technique applications but more consistent results that you can delegate

Patient Protocols

- Frequency of Treatment- non-periodic or 1 to 2 times per week
- Condition Specific: Bone on Bone Knees, Arthritis, Back Pain, DJD should be 8 visits then a supportive care plan with 2x month for 3 months then 1x month. Patient can decide when to stop care. These conditions are best suited at 2x a week.
- Extremities should be 1x week for 6 weeks
- Athletes or patients with good muscle tone can be treated 2x a week which will cut their time of care down to 3 weeks vs 6.
- The minimum time between two visits is 48 hours.

Pre-Post Patient Instructions

This is provided for you in the KDT.TV Premium Resource Portal

MyACT Piezowave2 Therapy Pre/Post Treatment Instructions

Prior to Treatment:

1. Dress in a wardrobe that allows the therapist to access your skin in the treatment area.
2. 24 Hours prior to your treatment stop taking pain killers and any anti-inflammatories.
3. Drink a few extra glasses of water, keep hydrated.

Post Treatment:

1. Do not take any type of pain killer or anti-inflammatory medication for 48 hours.
2. Do not ice the treated area.

3. Drink a few extra glasses of water, keep hydrated.
4. Limit physical activities, if possible, try to rest as much as possible so your body can start the healing process.
5. Heating the area is fine if you have chronic pain.

Hands on Training with the Devices

Session 1

Treatment of Articular Cartilage

IVYSPRING INTERNATIONAL PUBLISHER

International Journal of Medical Sciences

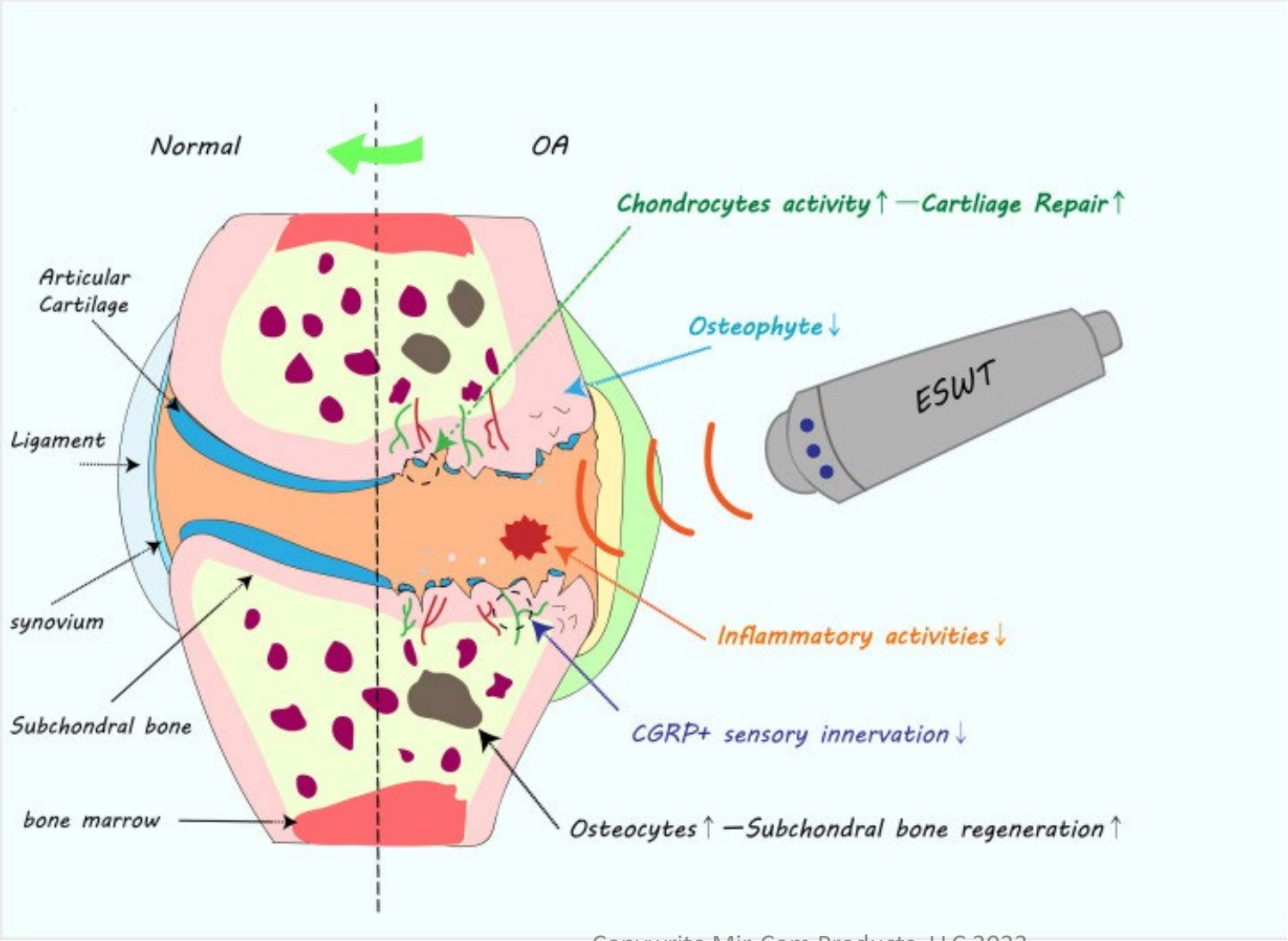
2019; 16(1): 156-166. doi: 10.7150/ijms.26659

Research Paper

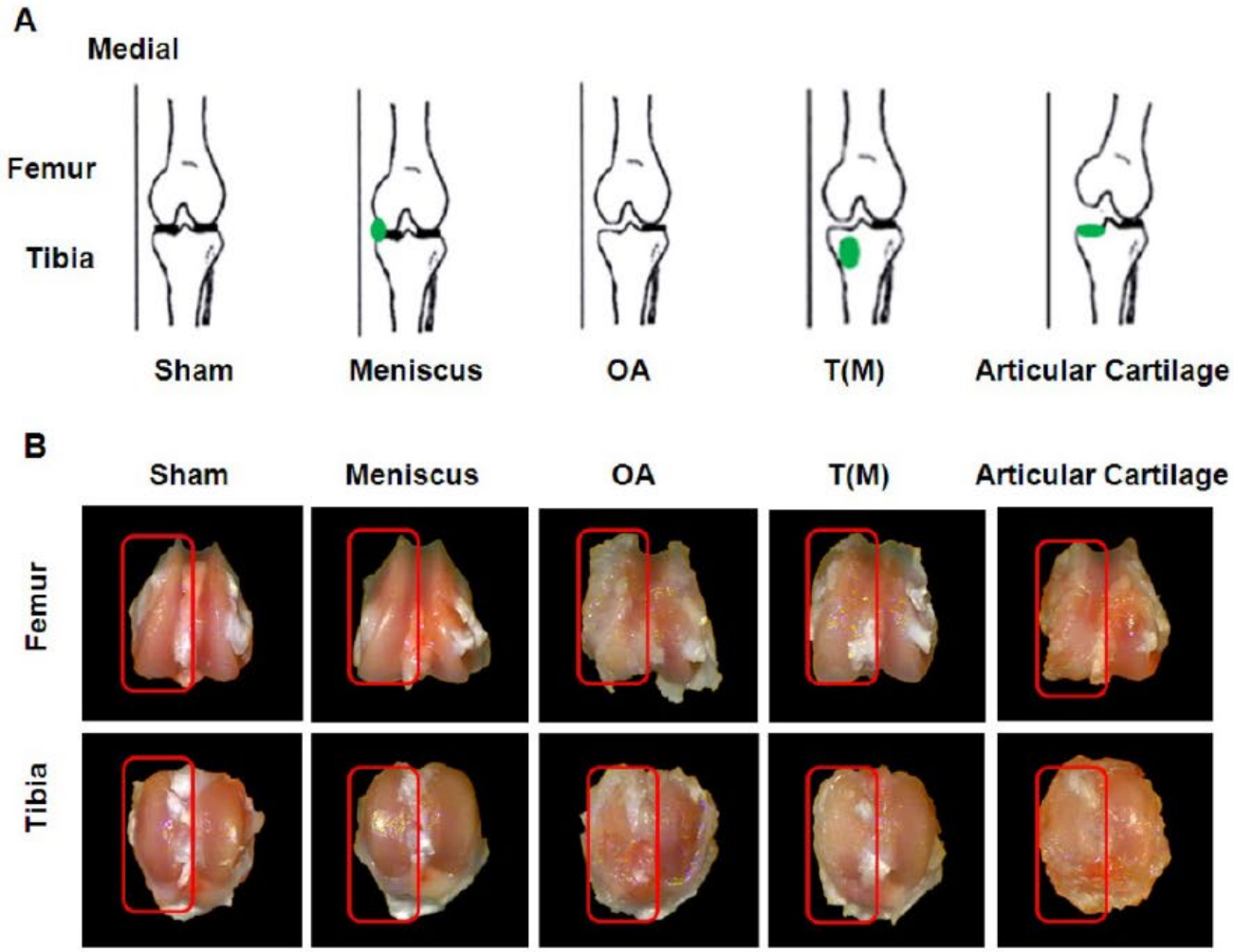
Shockwave Targeting on Subchondral Bone Is More Suitable than Articular Cartilage for Knee Osteoarthritis

Wen-Yi Chou[1,2], Jai-Hong Cheng[2,3]✉, Ching-Jen Wang[1,2]✉, Shan-Ling Hsu[1,2], Jen-Hung Chen[1], Chien-Yiu Huang[1,2]

Knee Arthritis



rats in each group (Fig. 1A). The group I was designated as Sham. They received sham arthrotomy of left knee without an anterior cruciate ligament transacted (ACLT) and medial meniscectomy (MMx). Group II was designated as Meniscus. They received sham arthrotomy of left knee without ACLT and MMx of the left knee, and then the shockwave applied to the medial edge of the meniscus. Group III was designated as OA. The animals received ACLT and MMx of left knee. Group IV was designated as T(M). Rats received ACLT and MMx of left knee and the shockwave applied to the proximal medial tibia plateaus. Group V was designated as Articular cartilage. The animals received ACLT and MMx of left knee and the shockwave applied to the articular cartilage surface of the proximal medial tibia plateaus. At 12-weeks post-surgery, the animals were scarified and the knees were collected from experiments.



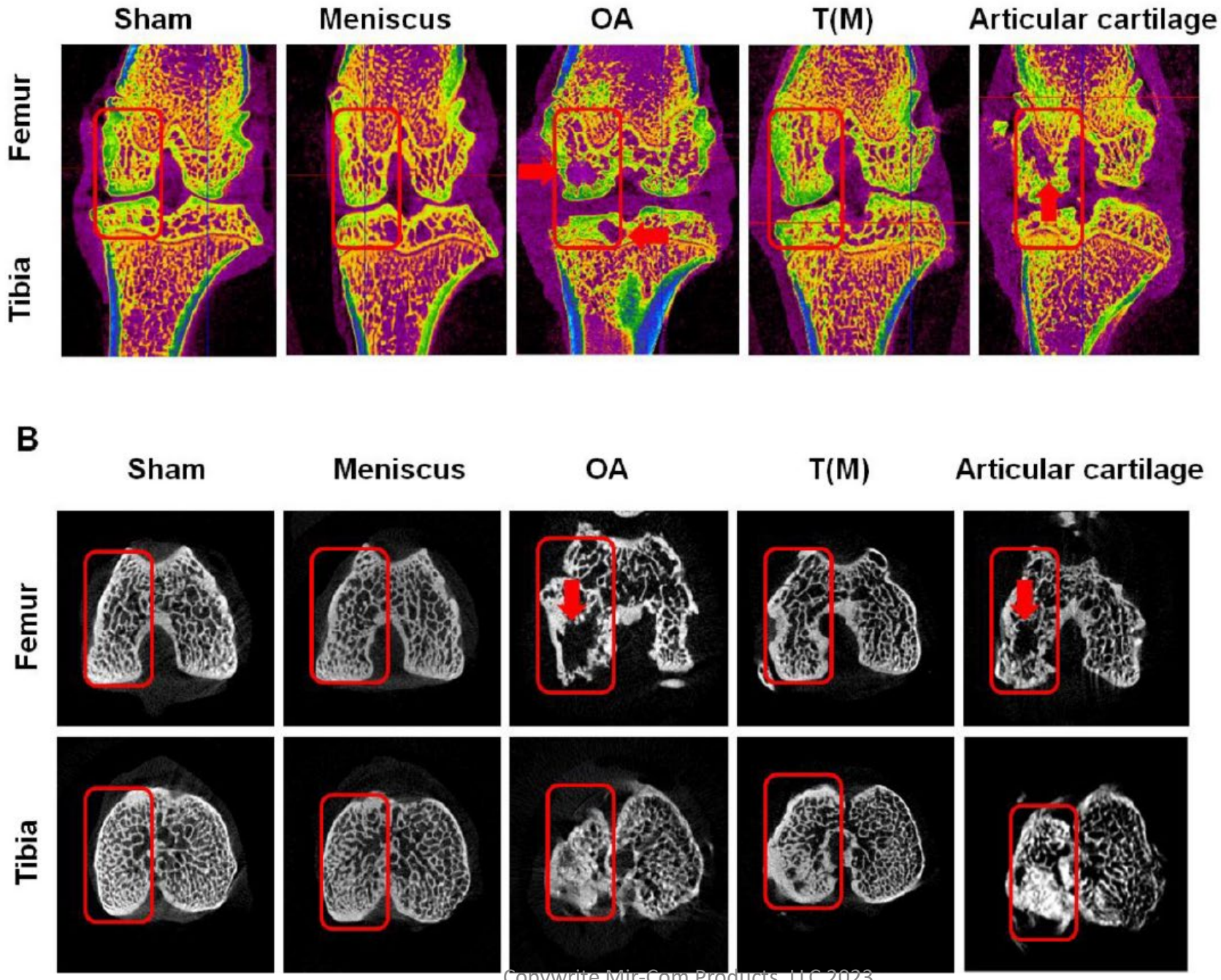
Sham= Sham Surgery with no procedure, no ESWT

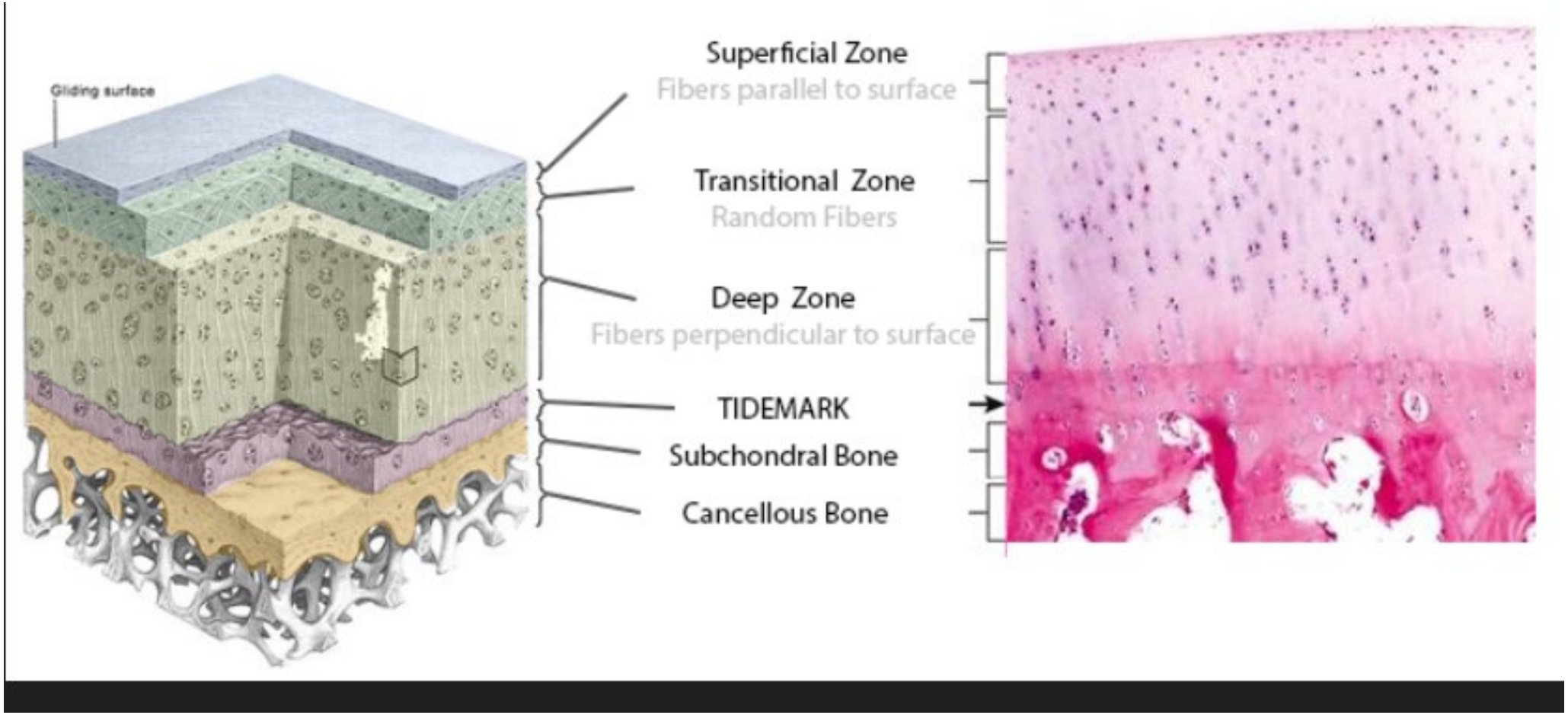
Meniscus= Sham surgery, ESWT applied to meniscus

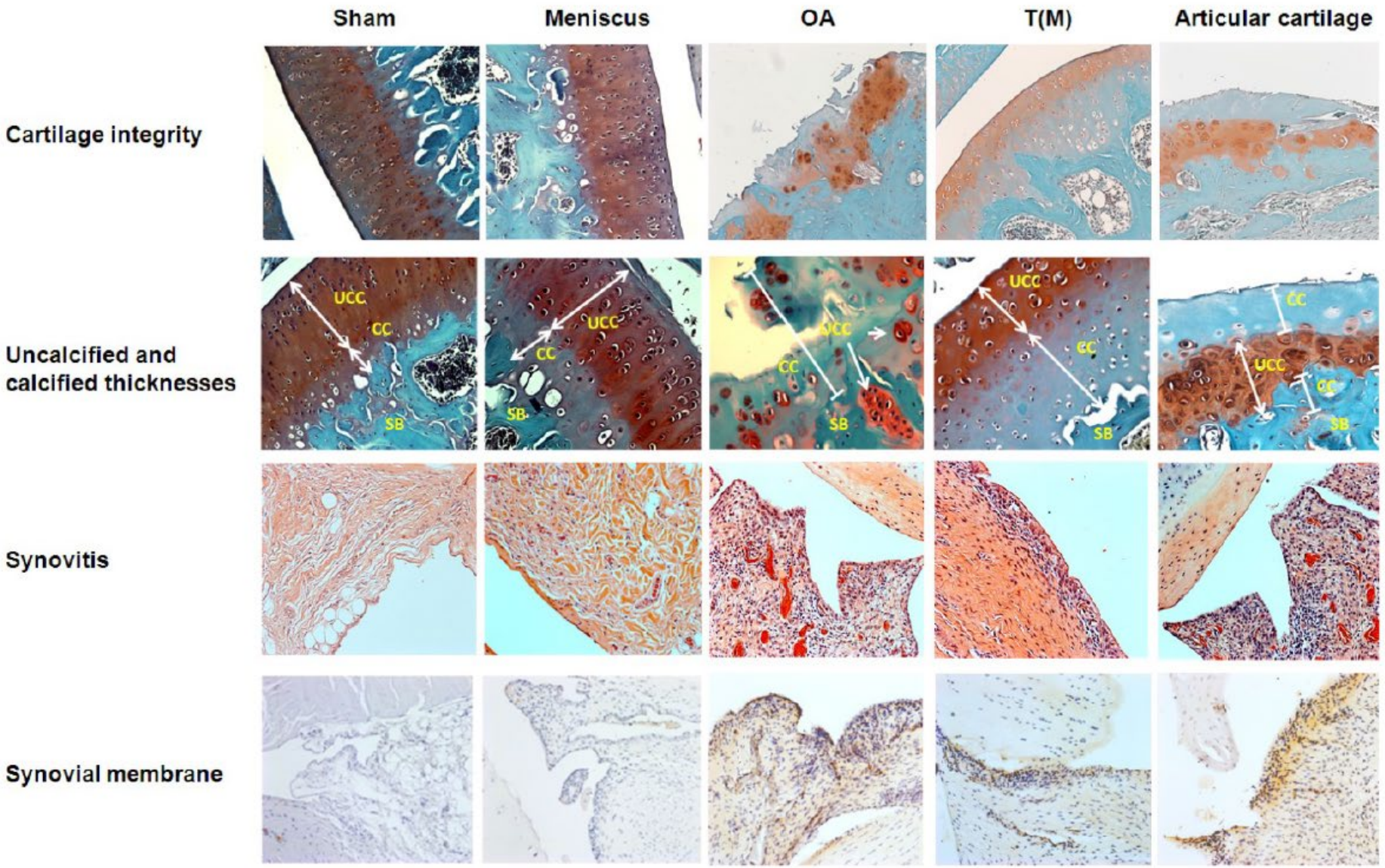
OA= Cut ACL and removed Meniscus and ACL no ESWT

TM= removal of ACL and Meniscus ESWT applied to medial tibia (subchondral)

TM= removal of ACL and meniscus ESWT applied to medial articular cartridge



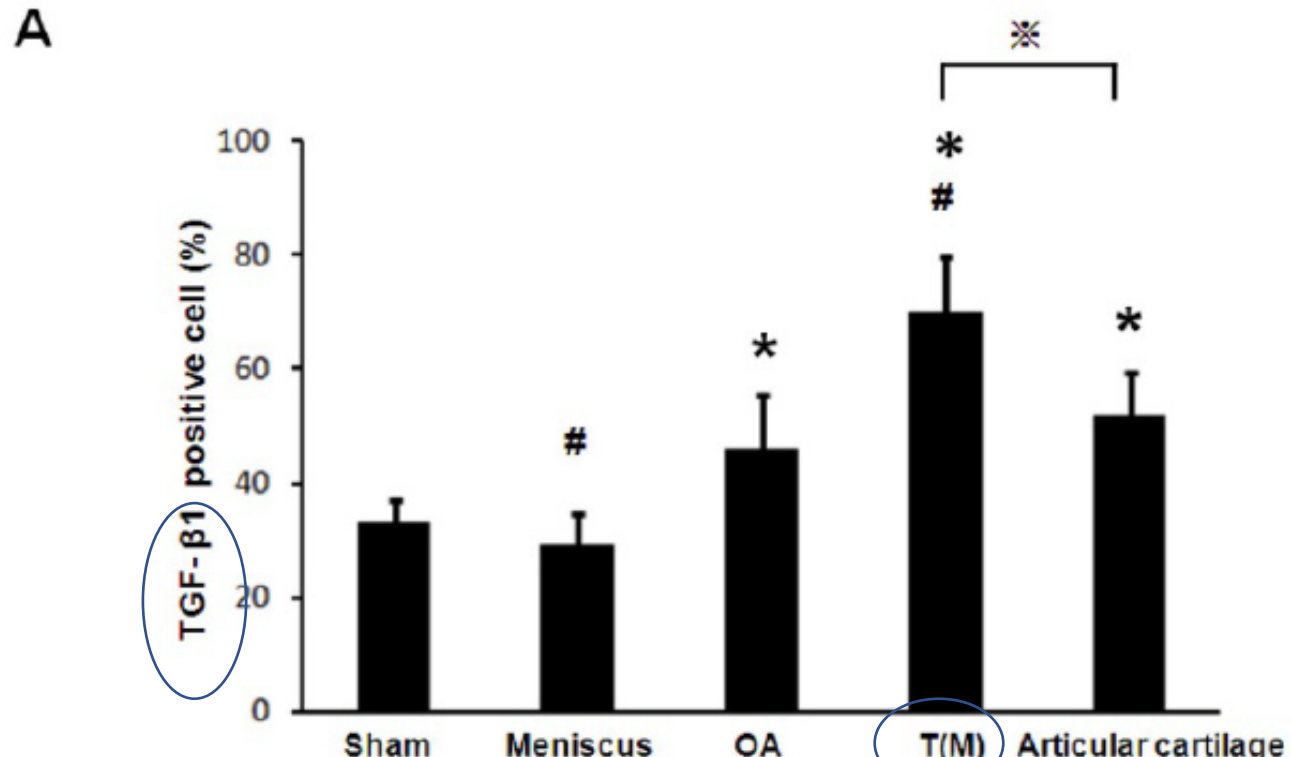




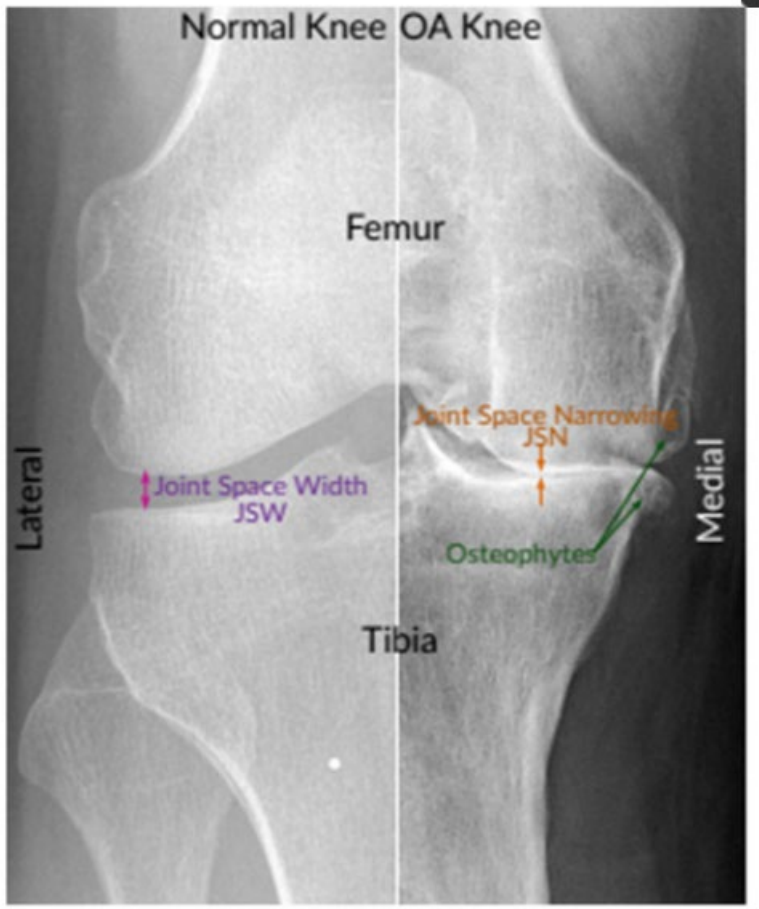
Induction of Cell Differentiation: TGF-β plays a crucial role in promoting the differentiation of precursor cells into specialized cell types. It can direct the differentiation of stem cells into specific lineages during embryonic development and tissue repair.

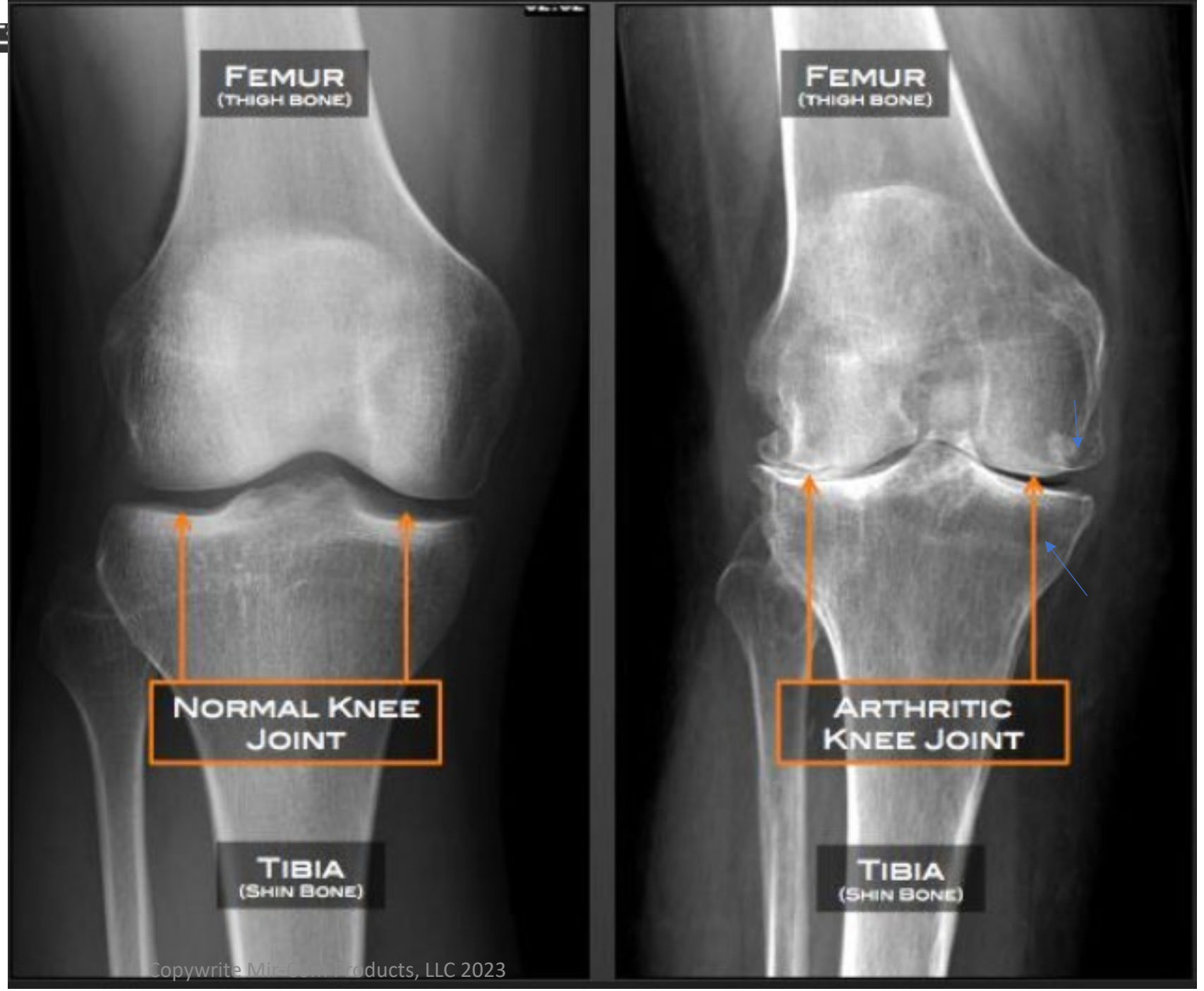
Regulation of Immune Responses: TGF-β is involved in immune system regulation. It can suppress the function of certain immune cells, modulate the production of inflammatory cytokines, and promote regulatory T cell development, which helps maintain immune tolerance and control inflammation.

Extracellular Matrix Production and Remodeling: TGF-β stimulates the synthesis of extracellular matrix components, such as collagen and proteoglycans, and promotes tissue remodeling. This is essential for tissue repair, wound healing, and maintaining tissue integrity.



	Sham	Meniscus	OA	T(M)	Articular cartilage
Mean (%)	33	29	46	70	52
Stand deviation	3.95	5.49	9.38	9.92	7.19





Front. Cell Dev. Biol., 21 January 2021
Sec. Molecular and Cellular Pathology
Volume 8 - 2020 | https://doi.org/10.3389/fcell.2020.607764

This article is part of the Research Topic
Novel Therapies for Combating Bone Diseases through Advances in Bone Remodeling
View all 38 Articles >

Subchondral Bone Remodeling: A Therapeutic Target for Osteoarthritis

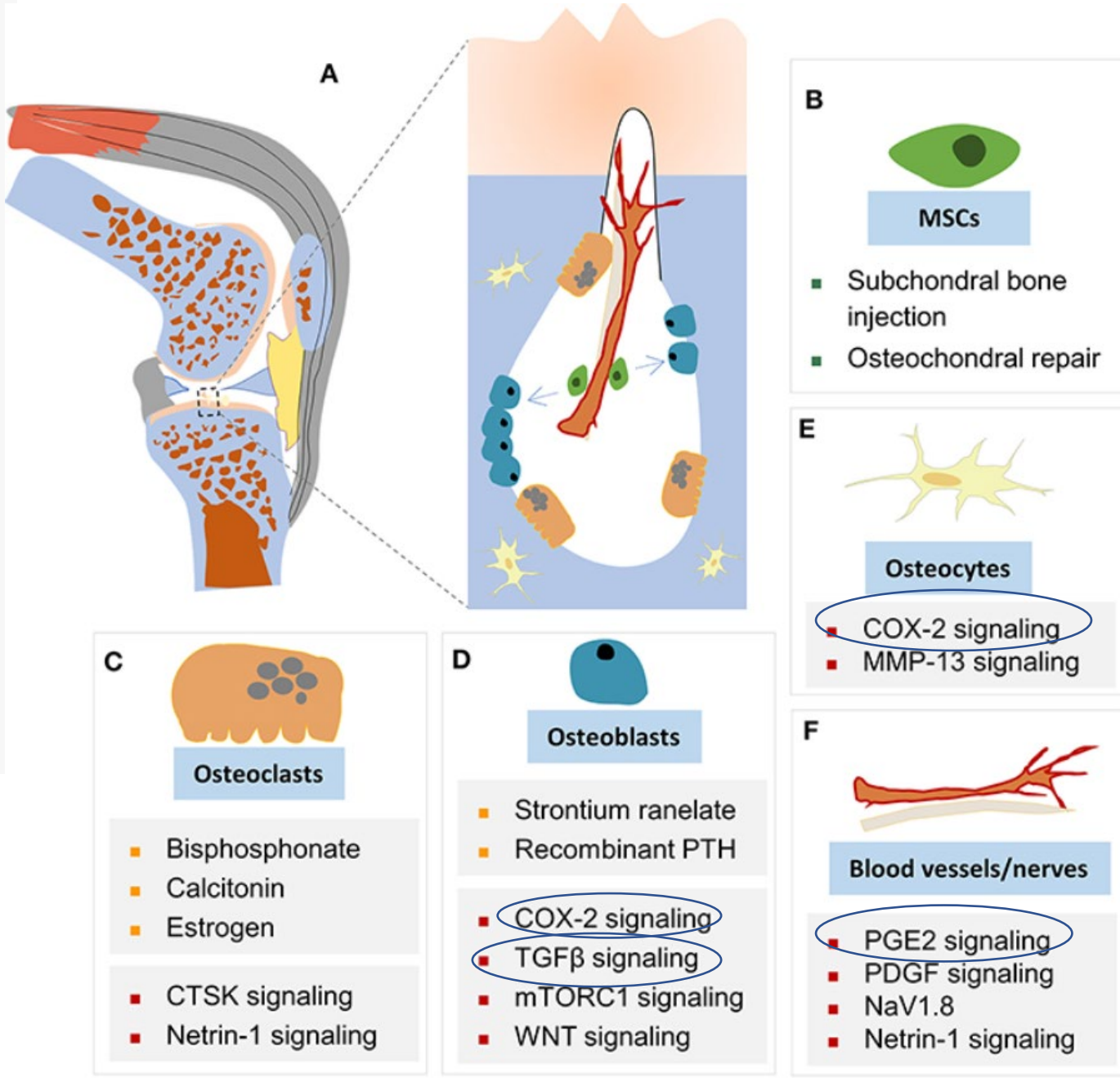
 Xiaobo Zhu[1,2†] Yau Tsz Chan[1,3†] Patrick S. H. Yung[1,2] Rocky S. Tuan[1,3*]
 Yangzi Jiang[1,3*]

[1] Institute for Tissue Engineering and Regenerative Medicine, The Chinese University of Hong Kong, Hong Kong, China
[2] Department of Orthopaedics & Traumatology, Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong, China
[3] School of Biomedical Sciences, The Chinese University of Hong Kong, Hong Kong, China

There is emerging awareness that subchondral bone remodeling plays an important role in the development of osteoarthritis (OA). This review presents recent investigations on the cellular and molecular mechanism of subchondral bone remodeling, and summarizes the current interventions and potential therapeutic targets related to OA subchondral bone remodeling. The first part of this review covers key cells and molecular mediators involved in subchondral bone remodeling (osteoclasts, osteoblasts, osteocytes, bone extracellular matrix, vascularization, nerve innervation, and related signaling pathways). The second part of this review describes candidate

Currently cell biologists are trying to figure out how to target:
COX-2
PGE2
TGFB

For the treatment of osteoarthritis of the knee



ELSEVIER

The Spine Journal 21 (2021) 160–177

THE SPINE JOURNAL

Basic Science

Low energy extracorporeal shock wave therapy combined with low tension traction can better reshape the microenvironment in degenerated intervertebral disc regeneration and repair

Yan-Jun Che, PhD, MD[a,b,#], Jun-Jun Hou, MD[c,d,#], Jiang-Bo Guo, MD[a], Ting Liang, PhD[a], Wen Zhang, PhD[a], Yan Lu, PhD, MD[d], Hui-Lin Yang, PhD, MD[a], Yue Feng Hao, PhD, MD[e], Zong-Ping Luo, PhD[a,*]

[a] *Orthopaedic Institute, Department of Orthopaedics, The First Affiliated Hospital of SooChow University, 708 Renmin Rd, Suzhou, Jiangsu 215007, People's Republic of China*

[b] *Department of Orthopedics, Heping Hospital Affiliated to Changzhi Medical College, Changzhi, Shanxi, People's Republic of China*

[c] *Department of Geriatrics, Xinghu Hospital, SuZhou industrial park, Suzhou, Jiangsu, People's Republic of China*

[d] *Department of Endocrinology, The First Affiliated Hospital of SooChow University, Suzhou, Jiangsu, People's Republic of China*

[e] *Orthopedics and Sports medicine center, The Affiliated Suzhou Hospital of Nanjing Medical University, Suzhou 215000, Jiangsu, People's Republic of China*

Received 3 June 2020; revised 7 August 2020; accepted 7 August 2020

Abstract

BACKGROUND: Low-tension traction is more effective than high-tension traction in restoring the height and rehydration of a degenerated disc and to some extent the bony endplate. This might better reshape the microenvironment for disc regeneration and repair. However, the repair of the combination of endplate sclerosis, osteophyte formation, and even collapse leading to partial or nearly complete occlusion of the nutrient channel is greatly limited.

Control

Control with devices removed

Traction

ESWT

Traction + ESWT

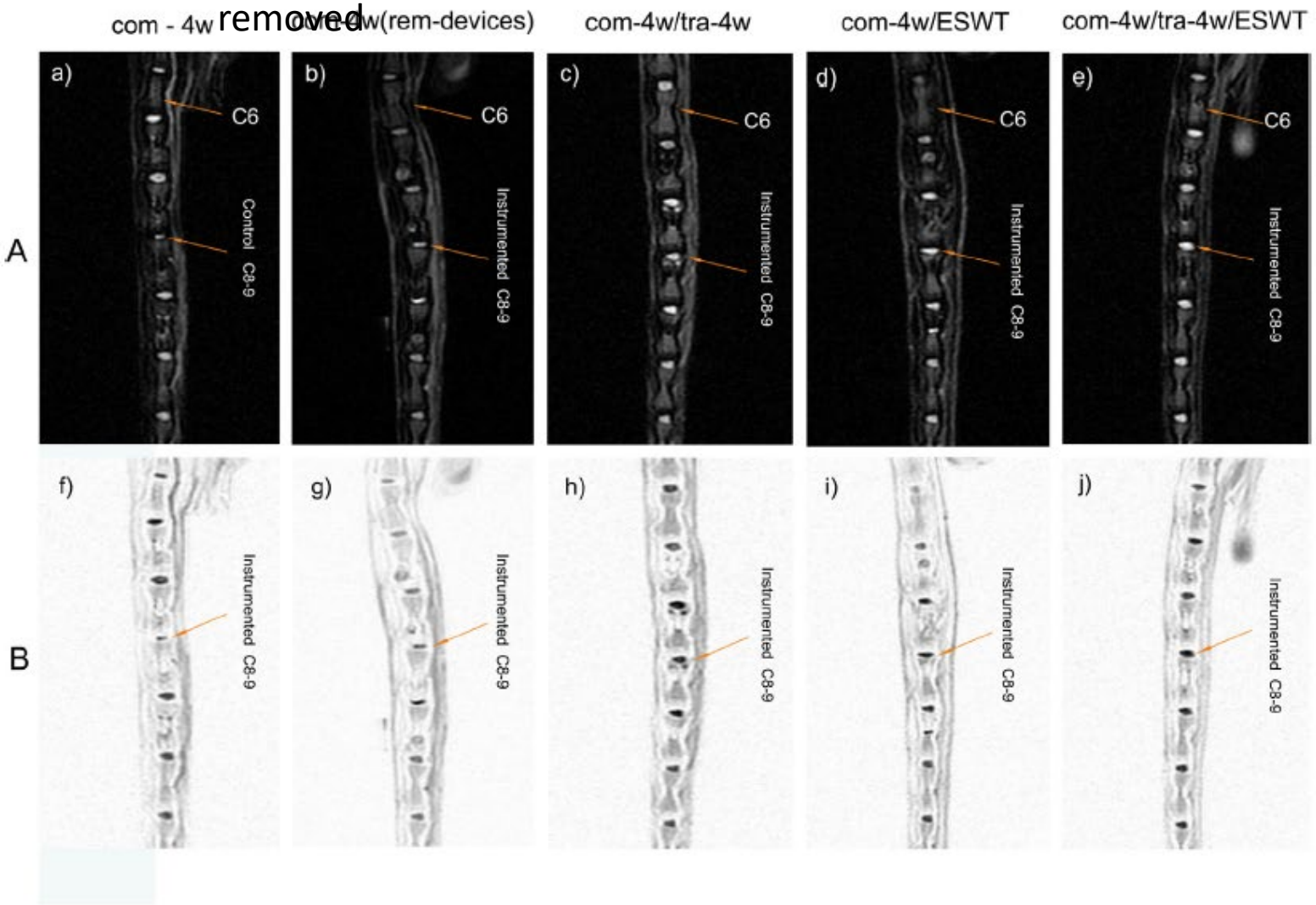


Fig. 2. Disc height and T2 signal strength. (Top) (A–E) is a representative T2 image of sagittal magnetic resonance imaging (MRI) scan of intervertebral discs in each group; (Bottom) (F–J) is the inverse image corresponding to Top (A–E).

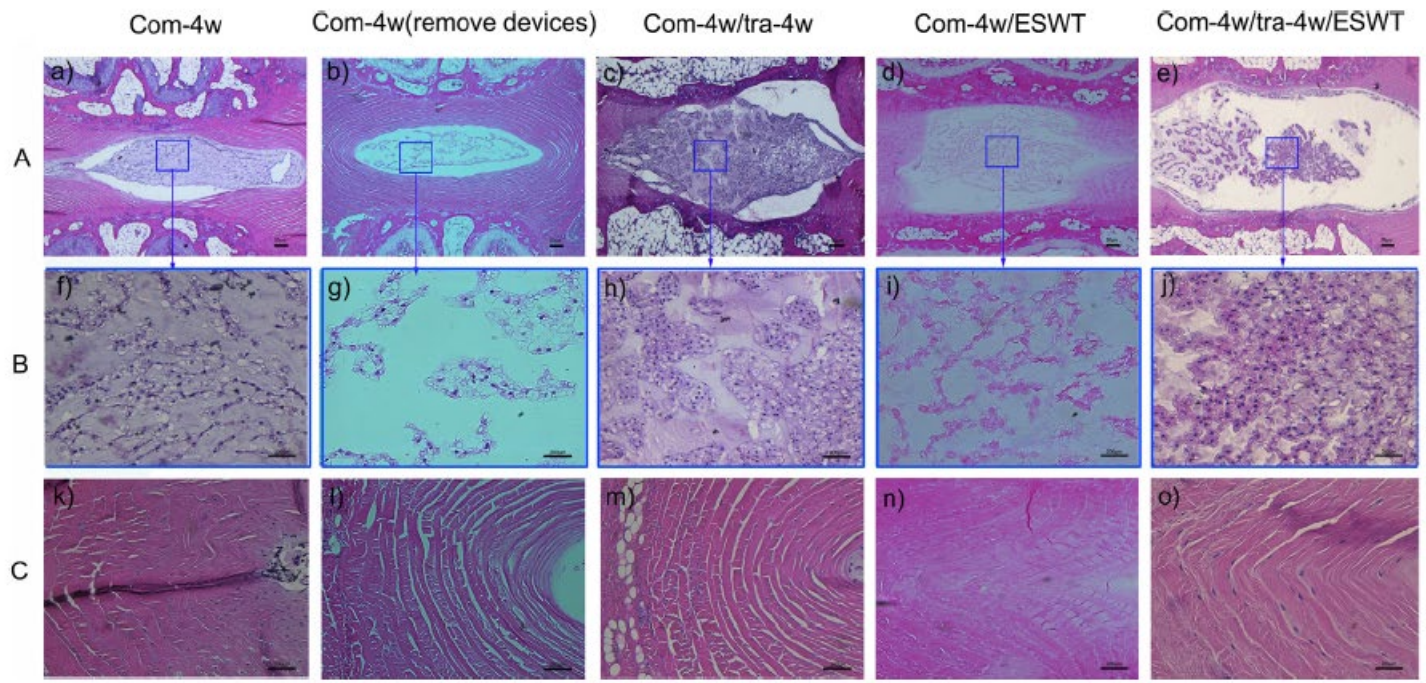


Fig. 4. Histological analysis (H&E). (Top) A to E: intervertebral disc (IVD) at magnification 50×. F to J, IVD at a higher magnification (200×), and K to O, IVD at magnification 200×; (Middle) F to J: in the middle row are enlarged images of the blue solid line box of the top row (A–E); (Bottom) K to O on the bottom row are enlarged images of the left anulus fibrosus in top row (A–E).

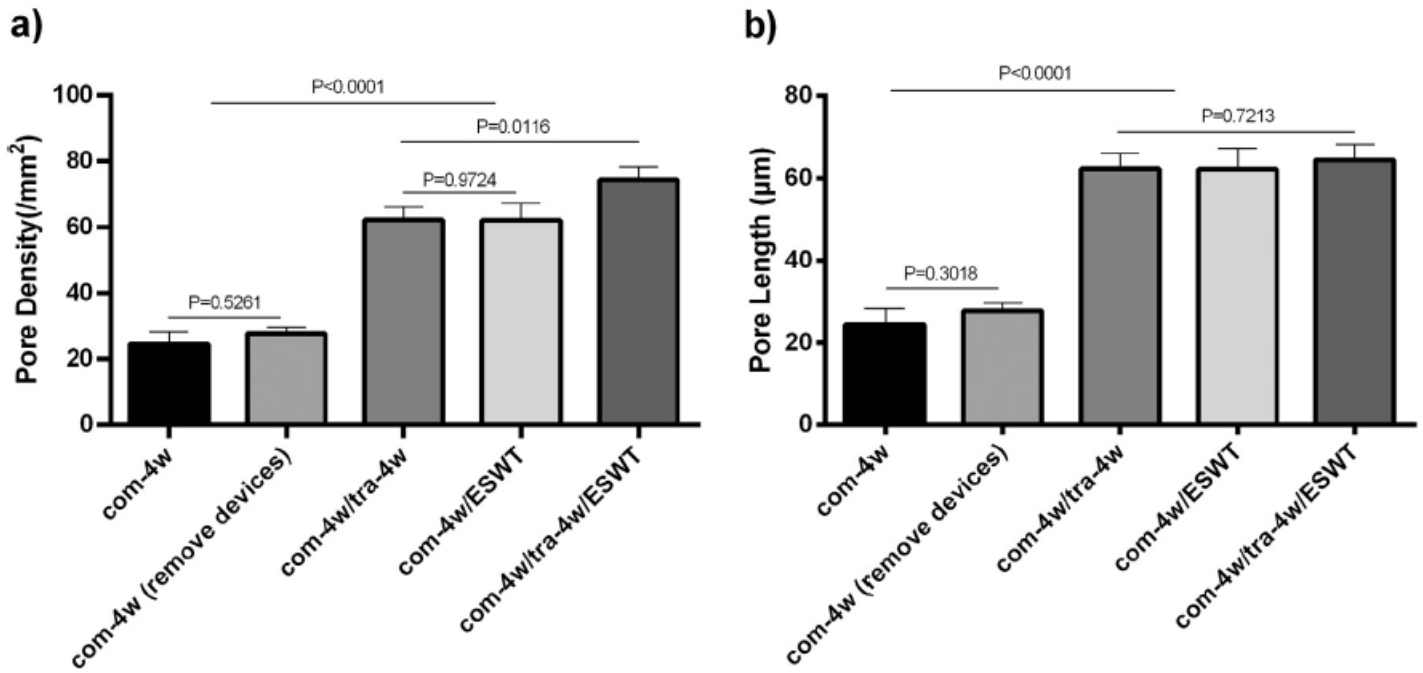


Fig. 10. (Left to Right). Statistical analysis of pore density and diameter of bony endplate. (Left) pore density analysis of bony endplate; (Right) pore diameter analysis of bony endplate. $p<.05$ indicates statistical significance.

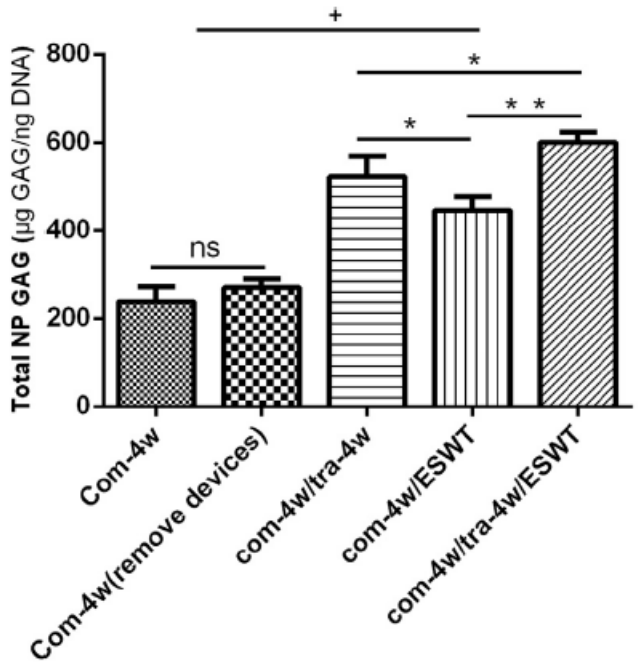


Fig. 6. Glycosaminoglycan (GAG) assay. (*) indicated significant difference between groups ($p<.05$); (**) indicated significant differences between groups ($p<.001$); (+) indicated significant difference between groups ($p<.0001$); (ns) indicated no significant difference between groups ($p>.05$).

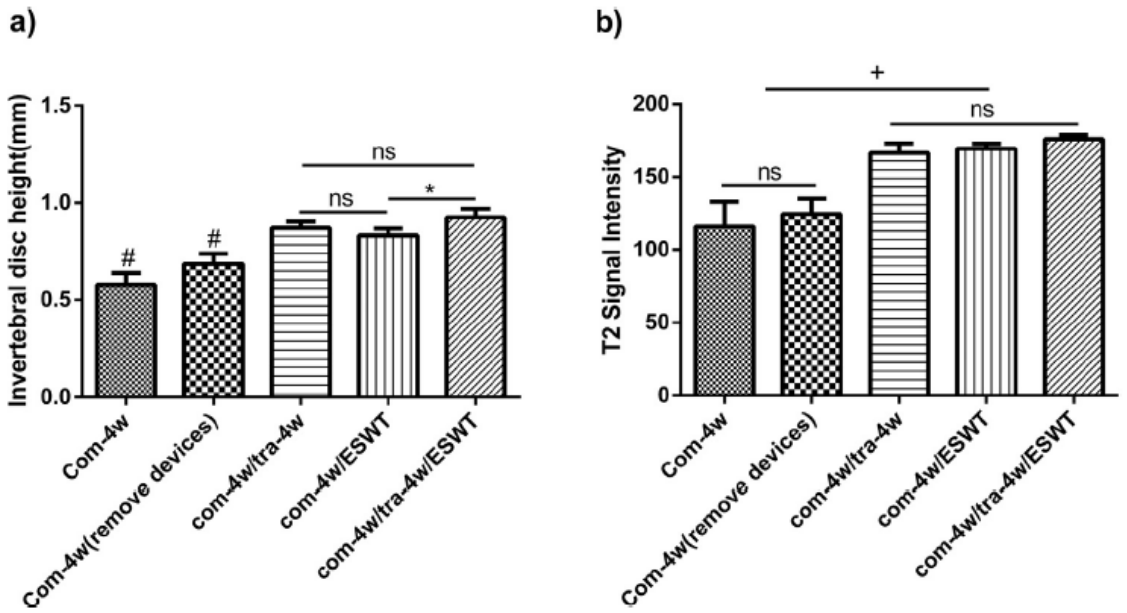


Fig. 3. (Left to Right). Statistical analysis of disc height and T2 signal strength. (Left) Disc height: (#) indicated that there were no significant differences between groups, but there were significant differences with remaining groups ($p<.05$); (*) indicated significant differences between groups ($p<.05$); (ns) indicated no significant difference between groups ($p>.05$); (Right) T2 signal strength: (+) indicated significant difference with other groups ($p<.0001$); (ns) indicated no significant difference between groups ($p>.05$).

Main Take away from the Disc Study

Low tension traction in combination with ESWT promoted regeneration of the intervertebral discs by inhibition of extracellular matrix degradation and maintenance of nucleus pulposus cell activity

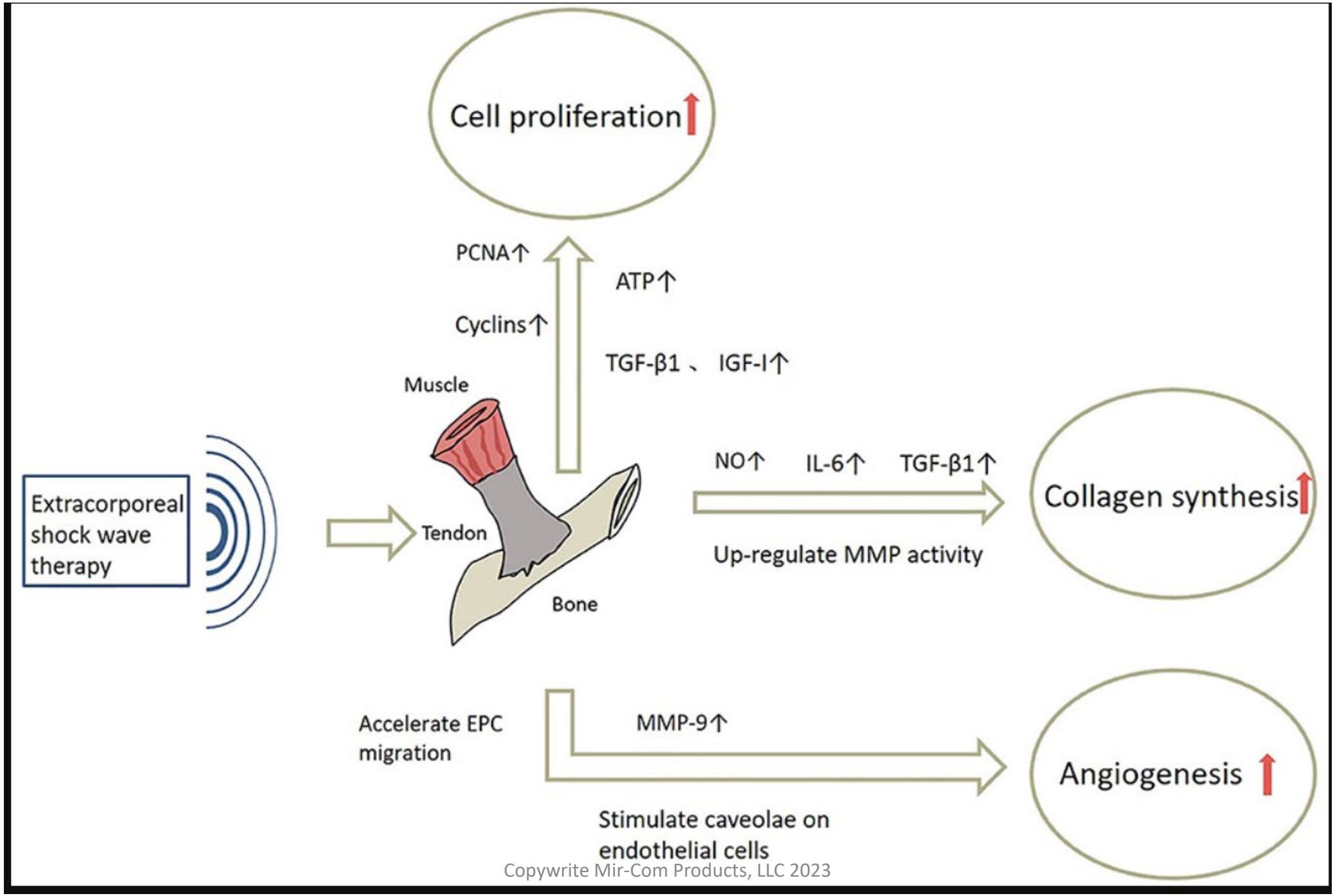
ESWT combined with low tension traction can better reconstruct the bony endplate and facilitate opening of the nutrient channels

Low energy ESWT combined with low tension traction reduced AF ring tension and NP nuclear stress, remodeling the disc biomechanical microenvironment

REVIEW article

Front. Vet. Sci., 22 July 2022
Sec. Comparative and Clinical Medicine
Volume 9 - 2022 | https://doi.org/10.3389/fvets.2022.851894

Biological response of extracorporeal shock wave therapy to tendinopathy *in vivo* (review)



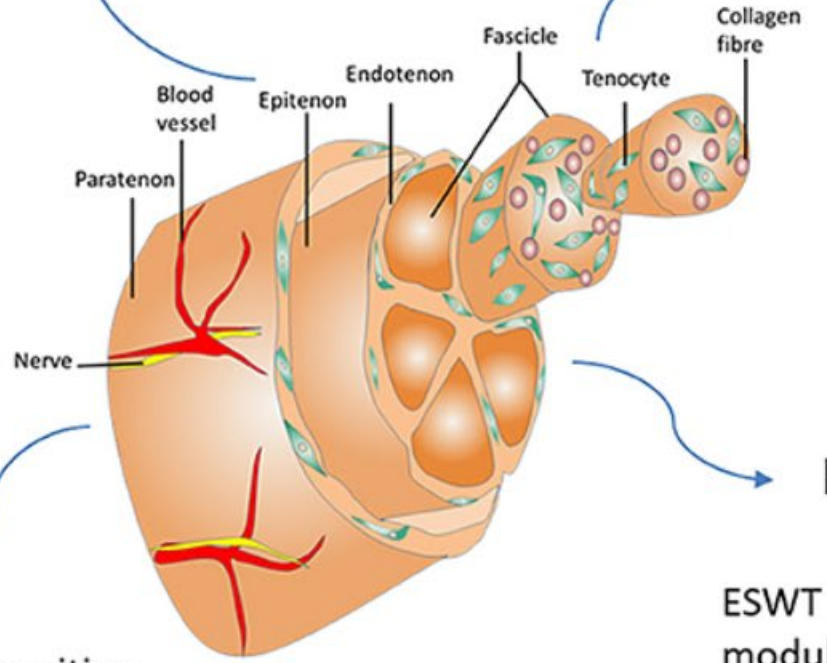
Extracorporeal shock wave therapy

Neuropeptides

ESWT inhibits inflammation by reducing CGRP and SP.

TLR3

ESWT can regulate inflammation by stimulating TLR3 to mediate the complex interaction between IL-6 and IL-10.



NO

ESWT induces moderate NO production to modulate biological responses in vivo shows a positive anti-inflammatory effect.

Macrophage

ESWT can ultimately achieve positive anti-inflammatory effects by modulating the activity of macrophages.

As a safe and widely used therapeutic tool, ESWT plays different roles in each of the three phases of tendon healing, eliciting different biological responses: during the inflammatory phase, ESWT increases the number of new blood vessels in the normal tendon-bone junction by regulating the release of growth factors and some other active substances, thus promoting an increase in nutrients and further accelerating tendon healing; while during the proliferative phase, ESWT stimulates tendon cell proliferation and collagen synthesis by generating mechanical stimulation; and in the remodeling phase, ESWT fosters ECM remodeling by regulating the tendon inflammatory response, which ultimately helps to achieve wound healing and tissue regeneration (2, 8, 106).

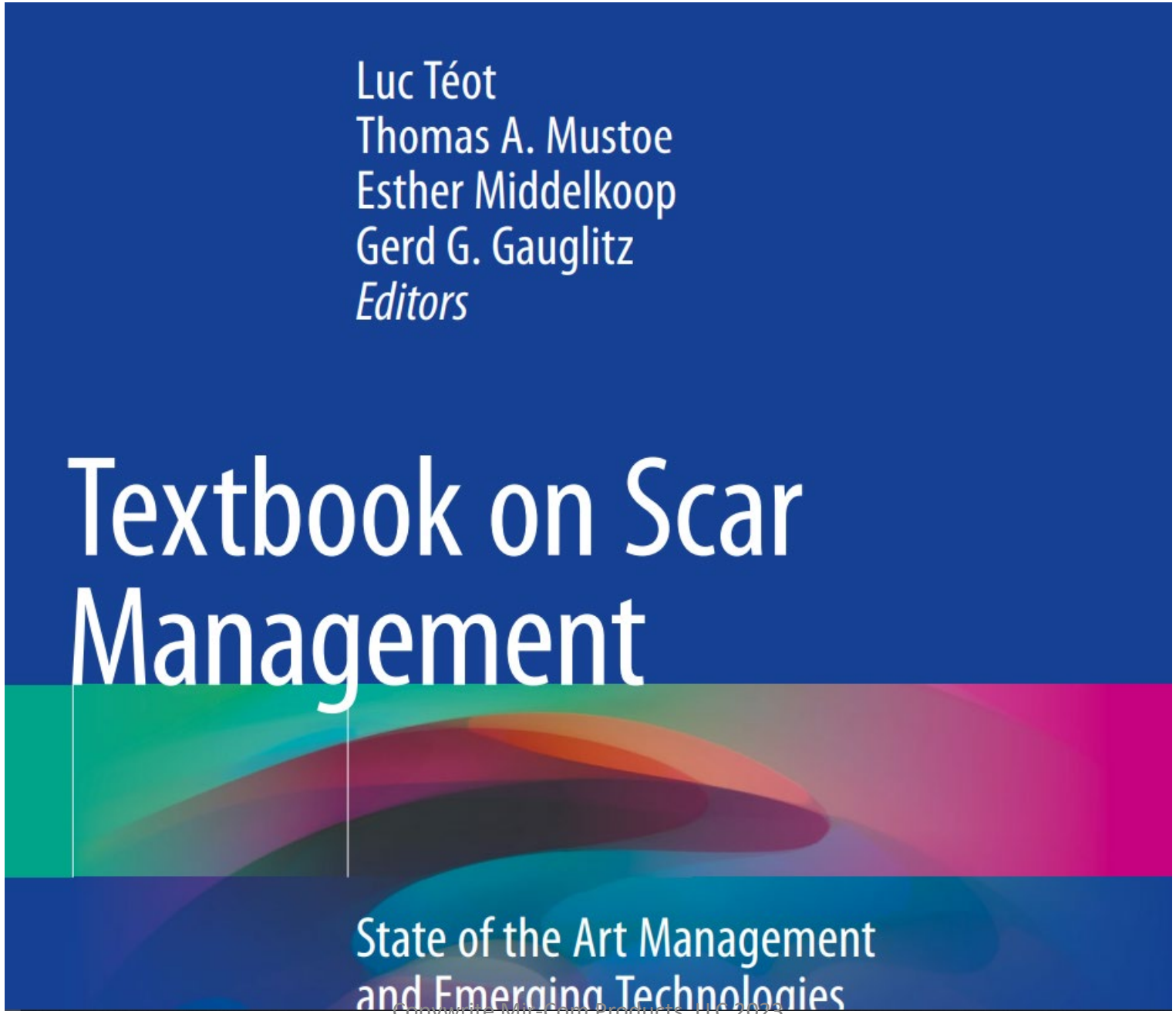


Table 55.1 Suggested SWT settings for electro-hydraulic devices when treating wounds or scars

From: Shock Wave Therapy for Wound Healing and Scar Treatment

	Energy flux density	Number of pulses	Pulse frequency	Treatment interval	Number of treatments
SWT for wound healing	0.03–0.20 mJ/mm^2	500–1000	4–6 Hz	1× per week	1–3
SWT for scar treatment	0.15–0.33 mJ/mm^2	800–1500	4–6 Hz	1× per week	8–12

dependency of these mechanotransduction events [9]. High-energy SWT can suppress cell growth, while low-energy shock waves might enhance cell proliferation [19]. SWT applied with an EFD of 0.01–0.03 mJ/mm^2 can modulate the inflammatory pathway in which macrophages are involved [12], and with an EFD of 0.08 mJ/mm^2 SWT can regulate inflammation via the TLR3 pathway. The EFD for soft tissue indications is typically in the range of 0.08–0.25 mJ/mm^2 [4], while scars and fibrosis are treated with an EFD ranging between 0.15 and 0.33 mJ/mm^2. SWT settings of 0.22 mJ/mm^2 and 1000 pulses seem to be ideal for fibroblast viability and growth [20]. Fibroblast viability was also influenced by the number of pulses. The higher the number, the more risk for cell destruction [11]. Each cell type seems to be responsive to SWT but probably with different optimal device settings and ranges of mechanical stimulation, thus developing different biochemical effects [8]. A study by Lee et al. showed that the EFD plays an important role in the targeting of specific mechano-signaling pathways, with 0.12 mJ/mm^2 being the optimal dose for activating the mTOR-FAK pathway [21] and 0.10 mJ/mm^2 showed the best results for inhibiting TGF-β1/Smad pathway [22] (◘ Table 55.1).