SANO HomeQure and Blood Circulation

Proven biological mechanisms — a reference document for physicians and therapists

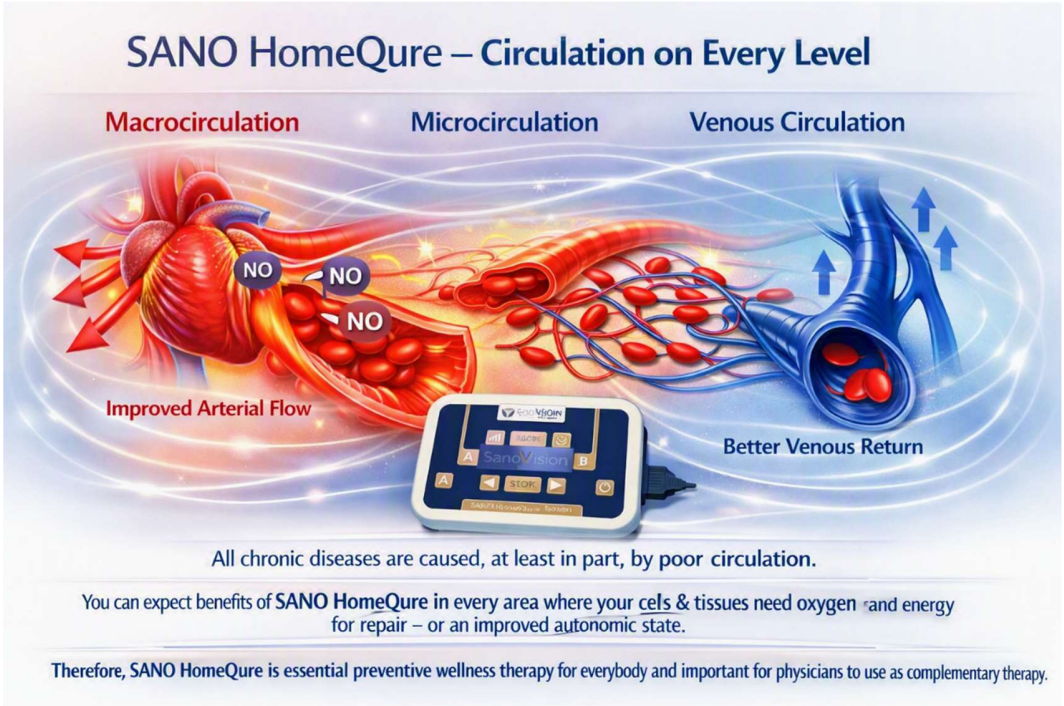
Introduction and Purpose of This Document

SANO HomeQure produces measurable biological effects across several systems: improved blood circulation, support for cellular energy production, a shift toward parasympathetic dominance in the autonomic nervous system, and reduced inflammation. Of these, improved circulation is arguably the most foundational, because oxygen and nutrient delivery is the rate-limiting step for almost every repair and regulatory process in the body.

This document focuses on that single effect and describes it mechanistically: which specific, published biological pathways are involved, and where in the vascular tree each one acts. We distinguish between two levels of the circulation that behave differently and are regulated by different mechanisms:

- **Macro circulation —** the heart, the large conduit arteries, and the veins: the vessels that move blood in bulk, in litres per minute, over distance.
- **Micro circulation —** arterioles, capillaries and venules, vessels under roughly 100 micrometres in diameter — and specifically the estimated 10-40 billion capillaries where the actual exchange of oxygen, nutrients and metabolic waste with the body's cells takes place.

A note on the evidence base: the mechanisms below are drawn from the published, peer-reviewed literature on pulsed electromagnetic fields (PEMF) and low-frequency magnetic field exposure generally — the device class HomeQure belongs to, and some specific HomeQure-branded clinical trials specifically. HomeQure's frequency programs and intensity range (from below 1 microtesla up to various millitesla in practitioner mode) fall within, or exceed, the ranges used in the cited studies, which is why these mechanisms are directly applicable to the device. Specific studies are referenced throughout, and a full source list is provided at the end, so the evidence can be evaluated directly.



Biological mechanisms how SANO HomeQure improves blood circulation (for physicians)

Overview: Which Mechanism Acts Where

The table below maps each mechanism discussed in this document to where it primarily acts in the vascular tree. Several — nitric oxide signalling in particular — act on both levels simultaneously.

Mechanism	Primary site	What it does
Nitric oxide (NO) mediated vasodilation	Both — conduit arteries AND resistance arterioles	Widens vessels via Ca2+/calmodulin -> eNOS -> NO signalling. Vessel radius has a fourth-power effect on flow (Hagen-Poiseuille), so this is the single highest-leverage mechanism for bulk flow.
Autonomic / parasympathetic modulation	Both	Shifts systemic vascular tone and heart rate variability; reduces sympathetic vasoconstrictive drive throughout the vascular tree.
Reduced RBC aggregation (zeta potential)	Micro (capillaries, venules) — also lowers bulk viscosity relevant to macro flow	Restores red cell surface charge so cells travel singly rather than as rouleaux, easing passage through low-shear small vessels.
Improved RBC deformability	Micro — specifically capillary passage	Lets individual red cells fold to fit through capillaries narrower than their own resting diameter (see detailed section below).
Reduced platelet hyperaggregability, lower fibrinogen, improved fibrinolysis	Micro (prevents microthrombi/sludging) and macro (fibrinogen affects whole-blood viscosity)	Reduces pathological clotting tendency and viscosity without disabling injury-triggered hemostasis (see detailed section below).
Reduced edema / improved lymphatic drainage	Micro	Relieves interstitial pressure that otherwise compresses capillaries from outside.
Reduced inflammation	Micro primarily (leukocyte adhesion, capillary plugging), with systemic macro effects	Inflammatory mediators independently drive vasoconstriction and endothelial/microvascular dysfunction.
Venous smooth-muscle and autonomic support	Macro (large veins) and micro (venules)	Supports the non-oxygenation side of circulation — see the venous section below.

Macrocirculation: Large Arteries and Veins

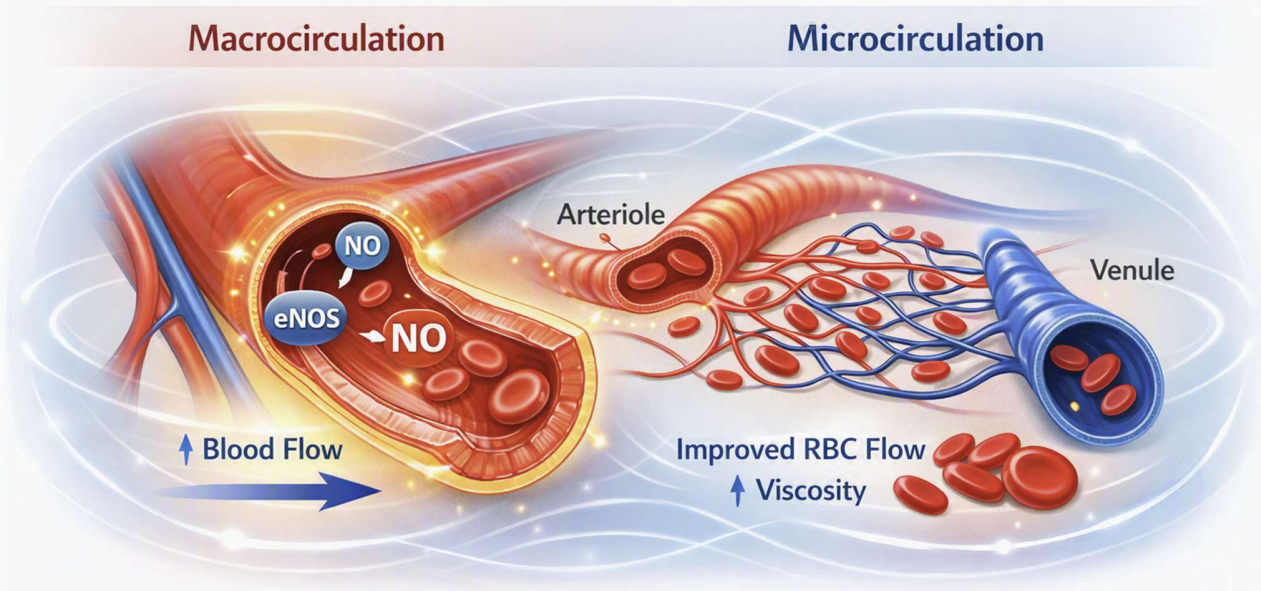
At the macrocirculatory level, the two effects with the clearest published support are nitric-oxide-mediated dilation of conduit arteries, and autonomic modulation of systemic vascular tone.

Nitric oxide and conduit-artery dilation. Human trials using low-frequency magnetic fields (for example 50 Hz) have shown measurable increases in brachial artery flow-mediated dilation — a standard clinical test of large-artery endothelial function — mediated through the calcium/calmodulin -> endothelial nitric oxide synthase (eNOS) -> NO pathway. Separately, randomized trials of PEMF therapy have shown reductions in systolic and diastolic blood pressure alongside improved flow-mediated dilation. Notably, at least one trial found this vascular improvement occurred without a corresponding rise in circulating NO levels, indicating the benefit is driven by local endothelial signalling rather than requiring a systemic increase in circulating NO — the effect is real even where blood NO levels alone would suggest otherwise.

Autonomic modulation. PEMF exposure shifts autonomic balance toward parasympathetic dominance, reducing the sympathetic vasoconstrictive tone that otherwise narrows arteries and veins throughout the body. This is a systemic, whole-vascular-tree effect rather than a local one, and it is consistent with the subjective relaxation response many patients report during and after treatment.

Microcirculation: Arterioles, Capillaries and Venules

This is where oxygen, nutrients and metabolic waste are actually exchanged with tissue, and it is regulated by a different set of factors than the macrocirculation — arterioles have smooth muscle and respond to the same NO/autonomic signalling described above, but capillaries themselves have no smooth muscle at all and cannot dilate. At the capillary level, flow is governed almost entirely by blood rheology (how easily the blood itself flows) and by the mechanical properties of the cells passing through. That makes red blood cell behaviour, platelet behaviour, and interstitial/lymphatic conditions the dominant variables — the two mechanisms below are the ones most specific to this level of the circulation.



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Red Blood Cell Deformability — Detailed Mechanism

A capillary can be narrower than the red blood cell trying to pass through it: capillaries run roughly 5-8 micrometres in diameter, while a resting red blood cell is about 7.5 micrometres across. The only way the cell gets through is by deforming — folding into a parachute or slipper shape on entry, then relaxing back to its normal biconcave disc shape afterward. This is not passive; it depends on the mechanical properties of the cell membrane and the cytoskeleton just beneath it, both of which are actively maintained by the cell's own metabolism. Three converging pathways are relevant to how a pulsed magnetic field could support this:

- **Reduced oxidative stress on the membrane.** The red cell membrane stays flexible only if lipid peroxidation and oxidation of membrane proteins — particularly Band 3, the anion-exchange protein that anchors the cytoskeleton — are kept low. Elevated reactive oxygen species (ROS) are a well-documented cause of increased membrane rigidity, Band 3 clustering and cytoskeletal disorganisation, all of which reduce deformability. PEMF exposure has repeatedly been shown, across multiple cell types, to modulate ROS levels and antioxidant enzyme activity; to the extent this extends to circulating red cells, it would help preserve rather than degrade membrane flexibility.
- **Support for the calcium/ion-pump balance that governs cell shape.** Deformability is acutely sensitive to intracellular calcium: when calcium rises, it opens the Gardos channel, the cell loses potassium and shrinks, and it stiffens. Keeping intracellular calcium low depends on a calcium-ATPase pump in the membrane working properly, which depends on a steady ATP supply. This is worth being precise about for a physician audience: mature red blood cells have no nucleus and no mitochondria — unlike almost every other cell type PEMF research usually discusses, red cells cannot generate ATP via oxidative phosphorylation and rely entirely on glycolysis. So the relevant benefit here is not improved mitochondrial function in the red cell itself — it has none — but improved substrate delivery: better local oxygen and glucose delivery via improved arteriolar and capillary flow (the macro/microcirculation mechanisms above) supports the glycolytic ATP production that keeps the calcium pump, and therefore deformability, working normally. This is a feedback loop — better perfusion helps red cells stay flexible, which in turn helps perfusion.
- **A rheological effect layered on top.** Some of what gets measured as "improved deformability" in flow studies is actually a downstream consequence of reduced aggregation (see zeta potential, above): a red cell squeezing through a capillary as part of a rouleau experiences very different mechanical stress than the same cell travelling alone. Disaggregating rouleaux improves functional passage through the microcirculation even without necessarily changing the intrinsic stiffness of any single cell's membrane.

Evidence quality note: *the calcium- and oxidative-stress-based mechanisms above are well established in red blood cell biology generally. Direct, isolated-PEMF-only studies measuring red cell deformability before and after magnetic field exposure are fewer in number than the nitric-oxide/vasodilation literature. This is a genuine, biologically coherent mechanism, but the evidence base behind it is thinner than for some of the other items in this document.*

> ***In simple terms:***
> *Red blood cells are actually a bit bigger than the tiniest capillaries they need to pass through, so they have to squeeze and fold to fit — almost like a car flexing to slip through a tight gap — then spring back to their normal shape afterward. How easily they can do this depends on how flexible and healthy the cell's outer membrane is. HomeQure supports that flexibility in a few ways: it helps protect the membrane from wear and tear, it helps keep the cell's internal chemistry balanced so the cell doesn't stiffen up, and it helps stop red blood cells from sticking together in clumps — so each one can move through the smallest vessels on its own, more easily.*

Platelet Adhesiveness, Fibrinogen and Fibrinolysis — Detailed Mechanism, and Why It Does Not Work Against Wound Healing

What the evidence shows. Extremely-low-frequency electromagnetic fields in the range of roughly 1.9 to 65 millitesla have been shown to inhibit platelet aggregation, with proposed mechanisms including modulation of intracellular calcium, reactive oxygen species, and enzyme activity within the platelet — the same broad category of calcium/redox-sensitive signalling implicated in the nitric oxide pathway above. Separate work using alternating electromagnetic fields in the 500-5,000 Hz range has demonstrated measurable hypocoagulation, and even breakdown of already-formed thrombi, at higher intensities. In one integrative-care protocol that included PEMF among other interventions, circulating fibrinogen fell by 28%; fibrinogen is a clotting factor, but it is also, independently, one of the main plasma proteins that promotes red-cell aggregation and drives whole-blood viscosity, so a reduction supports flow through two separate routes at once. (That fibrinogen figure comes from a combined-therapy protocol rather than PEMF in isolation, and should be read as suggestive rather than definitive for HomeQure specifically.)

> ***In simple terms:***
> *Studies have found that magnetic field therapy like this makes platelets — the blood cells responsible for clotting — less likely to clump together, likely by affecting calcium and other chemical activity inside those cells. Similar research has shown it can even help break down existing small clots at higher strengths. One study also found that this kind of therapy lowered fibrinogen — a clotting protein in the blood — by 28%. That matters for circulation too, because fibrinogen doesn't just help blood clot, it also makes red blood cells stick together and thickens the blood overall. So less fibrinogen means smoother, easier-flowing blood, on top of less clotting risk.*

Why this does not translate into a wound-healing risk. This is a fair and common question from patients and colleagues, and it deserves a careful answer rather than a dismissal, because on the surface "reduces platelet stickiness" and "helps heal wounds" sound contradictory. The resolution is that these effects operate on two different physiological processes on two different timescales.

- **Acute hemostasis** — the process that stops bleeding at the moment of injury — is triggered locally when platelets encounter subendothelial collagen and von Willebrand factor exposed by actual vessel wall damage. This is an immediate, injury-triggered adhesion cascade at the specific site of damage, not a reflection of a person's baseline circulating platelet "stickiness." Reducing systemic platelet hyperaggregability does not remove the collagen/vWF trigger that initiates a hemostatic plug at a fresh wound.
- **Wound healing** — the days-to-weeks process of tissue repair that follows hemostasis — depends on the opposite of clotting: it depends on blood flow. The proliferative and remodelling phases require sustained oxygen delivery, nutrient supply and immune cell trafficking to the wound bed. Excess or persistent microclotting in the surrounding tissue is, if anything, a recognised contributor to poor healing — this is well documented in chronic venous ulcers and diabetic foot ulcers, where microvascular thrombosis and sludging are part of the underlying pathology. Improving flow and reducing pathological micro-aggregation during this phase supports healing rather than working against it.

This is also borne out empirically, not just theoretically. A randomized, double-blind, placebo-controlled trial in patients with chronic diabetic foot ulcers found that active PEMF (60-minute sessions, 12 Hz, approximately 1.2 mT, 14 sessions over 3 weeks) produced an 18% reduction in wound size versus 10% in the sham-treated group, alongside documented improvement in microcirculation. A separate randomized trial combining electromagnetic field therapy with exercise showed significant improvement in venous leg ulcer healing at 12 weeks. If reduced platelet adhesiveness were functionally impairing hemostasis-dependent healing, these trials would show that — they show the opposite.
A note on clinical caution. *None of this is a claim that PEMF is without any relevance to hemostasis. Patients on anticoagulant or antiplatelet medication, or with known bleeding disorders, should continue to be managed with the same clinical judgement applied to any adjunct therapy with published effects on hemostatic parameters. The point of this section is narrower: the mechanism does not work against wound healing in the way the surface-level concern suggests, and the controlled-trial evidence for wound healing supports that.*

> ***In simple terms:***
> *HomeQure helps make blood a little less "sticky," so blood cells clump together less and blood flows more smoothly. That might sound like it could interfere with healing, since clotting is what closes a wound — but it doesn't. Stopping the bleeding right when you're injured is a separate, local reaction your body triggers automatically at the wound itself; HomeQure doesn't switch that off. What actually determines how well a wound heals over the following days and weeks is blood flow — getting enough oxygen and nutrients to the area. So by improving circulation, HomeQure supports healing rather than working against it. This has also been tested directly: in clinical trials, people using this type of therapy healed wounds faster than those who didn't.*

Reduced Edema, Lymphatic Support, and Reduced Inflammation

Two further microcirculatory contributors round out the picture.

- **Reduced interstitial fluid buildup**, supported by **improved lymphatic drainage**, relieves the external pressure that edema places on capillaries, which otherwise restricts flow independently of anything happening inside the vessel.

- **HomeQure's anti-inflammatory effects**: Because inflammatory mediators independently promote vasoconstriction, endothelial dysfunction and leukocyte adhesion to capillary walls (which can physically obstruct capillary flow), SANO HomeQure's documented anti-inflammatory effects act as a further, separate support for microcirculatory flow rather than a duplicate of the mechanisms above.

SANO HomeQure and Venous Circulation Specifically

Circulation is not only about oxygenated blood moving out to the tissues. Roughly 60-75% of total blood volume is on the venous side at any given moment, and venous congestion is a component of a wide range of presentations — from simple dependent edema, to chronic venous insufficiency, to the general sluggish circulation seen with prolonged immobility.

Improved arterial delivery does nothing for this side of the circuit on its own; venous return is a separate hydraulic problem, driven physiologically by residual cardiac pressure, the skeletal-muscle pump, the respiratory pump, and venous valves — not by the same mechanisms that deliver oxygenated blood outward.

The mechanisms above that are most relevant to the venous side are:

- reduced blood viscosity and reduced red-cell aggregation, which lower the resistance venous blood has to overcome on its return trip;
- mild relaxation of venous smooth-muscle tone;
- autonomic support for venomotor tone; and
- improved lymphatic drainage, which relieves the interstitial pressure that can otherwise impede venous filling from the tissue side.

HomeQure's contribution to venous circulation works through blood rheology and smooth-muscle/autonomic tone rather than through the oxygenation pathway — which is precisely why it is able to support both sides of the circulation through a shared but non-overlapping set of mechanisms.

> ***In simple terms:***
> *SANO HomeQure doesn't only support the vital micro circulation — the flow through your smallest vessels that delivers oxygen and nutrients to your cells and carries away waste. It also improves macro circulation, meaning better, faster blood flow through the large arteries.*
> *And importantly, it also supports venous circulation — the return flow of blood back to the heart through your veins. This matters because at any given moment, roughly 60 to 65% of your total blood volume is sitting on the venous side. If that return flow is sluggish, it shows up as things like heavy or swollen legs. HomeQure helps here too, by making blood flow more easily and giving gentle support to the veins pushing blood back upward.*
> *So HomeQure supports circulation on every level: getting blood out to the tissues, moving it efficiently through the big vessels, and helping it make its way back to the heart.*

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What Improved Circulation Delivers Downstream

Better macro- and microcirculatory flow is not the end point; it is the mechanism that enables several further, measurable benefits:

- **Oxygenation:** more red blood cells reaching more capillaries, and deforming effectively enough to pass through them, means more oxygen delivered per unit time to tissue that needs it.
- **Detoxification:** the same capillary exchange that delivers oxygen and nutrients also carries away CO2 and metabolic waste products; improved microcirculatory flow speeds this clearance.
- **Cellular energy production:** for nucleated tissue cells — muscle, nerve, connective tissue, essentially every cell type other than the mature red blood cell — improved oxygen delivery is a direct input to mitochondrial oxidative phosphorylation, supporting the ATP production those cells need for repair and normal function.

Because these effects are systemic rather than tied to a single diagnosis, the practical implication is broad:

> **used as a complementary therapy, SANO HomeQure is likely to support — not replace — whatever primary treatment or medication a patient is already receiving, in any condition where the underlying limitation includes a cellular oxygen/energy deficit or a restricted circulatory or autonomic state.**

That includes wound and fracture healing, post-surgical recovery, chronic inflammatory conditions, and stress-related or autonomic-dysregulation presentations, among others. The common denominator across HomeQure's indications is not a specific diagnosis but a specific physiological need: better delivery of oxygen and energy substrate to cells that are trying to repair or regulate themselves.

Summary

SANO HomeQure supports circulation through multiple, parallel, independently documented mechanisms rather than a single pathway:

- nitric-oxide-mediated dilation of both conduit arteries and resistance arterioles;
- autonomic (parasympathetic) modulation of vascular tone;
- reduced red blood cell aggregation via zeta-potential effects;
- improved red blood cell deformability, most plausibly through reduced oxidative membrane stress and better-supported calcium/ATP handling;
- reduced platelet hyperaggregability and fibrinogen, without evidence of impairing the acute, injury-triggered hemostatic response that wound healing actually depends on;
- reduced edema and improved lymphatic drainage; and reduced inflammation.

Some of these mechanisms act primarily on the macrocirculation, some primarily on the microcirculation, and several — nitric oxide signalling in particular — act on both. Together they improve oxygen and nutrient delivery to tissue, support clearance of metabolic waste, and help create the circulatory and autonomic conditions that other therapies and medications generally depend on to work well. That is the basis for using SANO HomeQure as a complementary therapy across a broad range of indications: not because it treats any one diagnosis directly, but because almost every repair and regulatory process in the body is, at some level, rate-limited by how well oxygen and energy reach the cells doing the work.

Source Notes

Key references used in preparing this document (for follow-up reading, not formatted as a full bibliography):

- Observation of Red Blood Cell Membrane Charge Using Magnetic Beads Under Pulsed Magnetic Field (IEEE) — pulsed magnetic field improves RBC aggregation/mobility via zeta potential.
- Electrokinetic properties of healthy erythrocyte membranes under static magnetic field exposure (Frontiers in Chemistry, PMC10620691) — increased negative surface charge after magnetic exposure.
- A 50 Hz magnetic field affects hemodynamics, ECG and vascular endothelial function in healthy adults (PMC8341886) — flow-mediated dilation via the NO pathway.
- The impact of pulsed electromagnetic field therapy on blood pressure and circulating nitric oxide levels (randomized trial, metabolic syndrome) — vascular improvement without a rise in circulating NO.
- Red blood cell oxidative stress impairs oxygen delivery and induces red blood cell aging (Frontiers in Physiology) — ROS, Band 3 clustering, and membrane rigidity.
- Calcium dynamically alters erythrocyte mechanical response to shear (ScienceDirect) — Gardos channel, calcium and deformability.
- Electromagnetic fields (1.9-65 mT, ELF range) inhibit platelet aggregation via calcium/ROS/enzyme modulation; electromagnetic oscillation at 500-5,000 Hz produces hypocoagulation and thrombus breakdown.
- The effect of magnetic fields on platelets, blood coagulation and fibrinolysis in guinea pigs (PubMed 6676735) — foundational animal study.
- Pulsed electromagnetic field therapy promotes healing and microcirculation of chronic diabetic foot ulcers (randomized, double-blind, placebo-controlled pilot study) — 18% vs 10% wound-size reduction at 3 weeks.
- Effect of combined electromagnetic field and exercise on wound healing in venous leg ulcers (randomized controlled trial, PMC10303563).
- Signalling pathways underlying pulsed electromagnetic fields in bone repair (Frontiers in Bioengineering and Biotechnology; Cellular Physiology and Biochemistry, Karger) — Ca2+/calmodulin/VGCC mechanism, applicable across cell types.
- Microcirculation: Physiology, Pathophysiology, and Clinical Application (PMC7114900); CV Physiology microcirculation reference — macro/micro vessel definitions and diameters.
- Estimates of ~10-40 billion capillaries and ~900 m2 total capillary surface area in the human body (multiple physiology references).