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Vascular collagen-based biomaterials for vascular Tissue engineering

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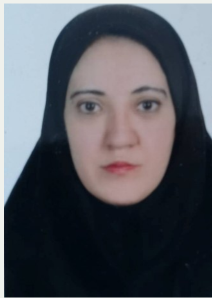

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Background: Cardiovascular diseases demand functional vascular substitutes, but autologous grafts are often unavailable. Collagen, the primary extracellular matrix component of native blood vessels, is a key biomaterial for vascular tissue engineering (VTE).

Aim: This review systematically examines collagen-based biomaterials in VTE, covering biological rationale, fabrication strategies, preclinical outcomes, and translational challenges.

Results: Native vascular ECM (collagens I, III, IV) provides mechanical integrity and cell-matrix interactions via integrins and discoidin domain receptors. Discussed scaffold architectures include collagen hydrogels (support capillary networks but lack strength), electrospun collagen–synthetic blends (achieve clinically relevant burst pressures), crosslinked matrices (enhanced durability), and decellularized vascular scaffolds (preserve native ECM). Advanced biofabrication—3D bioprinting and interpenetrating network hydrogels—enables perfusable, cell-responsive conduits. Functionalization with heparin, growth factors, or copper ions confers antithrombotic and pro-angiogenic properties. Preclinical large-animal studies report >90% patency with endothelialization. However, clinical translation is limited by residual thrombogenicity, intimal hyperplasia, sterilization compatibility, manufacturing scalability, and regulatory complexity.

Conclusion: Future directions include smart, environment-responsive collagen materials that recruit endogenous cells and degrade synchronously with new tissue formation. With continued interdisciplinary efforts, collagen-based vascular grafts hold realistic potential to transform cardiovascular surgery.

Keywords: Collagen; vascular tissue engineering; electrospinning; 3D bioprinting; decellularized matrix; vascular graft; preclinical models; clinical translation

Introduction

Cardiovascular diseases remain the leading cause of death worldwide, creating an urgent clinical need for functional vascular substitutes. Although autologous vessels are the gold standard for bypass grafting, their limited availability due to prior harvest, disease, or anatomical mismatch drives demand for off-the-shelf alternatives. Tissue engineering offers a transformative solution by fabricating living conduits that integrate, grow, and remodel with host tissue, thereby avoiding the chronic failure modes of synthetic

grafts such as infection, stenosis, and thrombosis (1). Tissue engineering offers a transformative solution by fabricating living conduits that integrate, grow, and remodel with host tissue, thereby avoiding the chronic failure modes of synthetic grafts such as infection, stenosis, and thrombosis. These engineered vessels aim to replicate not only the mechanical properties of native arteries but also the biological functions—including vasoactivity, endothelial barrier function, and matrix turnover. By using cells, scaffolds, and bioactive cues, tissue-engineered vascular grafts can potentially adapt to the hemodynamic environment, support endothelialization, and promote long-term patency without the need for lifelong anticoagulation (2). Central to this approach is the extracellular matrix (ECM), a native scaffold that provides structural support and biochemical cues to vascular cells. The ECM in blood vessels is a dynamic, three-dimensional network composed primarily of collagen, elastin, proteoglycans, and glycoproteins. Beyond its mechanical function, the ECM regulates cell adhesion, migration, proliferation, and differentiation through specific receptor interactions and by sequestering growth factors. Therefore, recapitulating key features of the native ECM is a major goal in VTE (3). Its universal presence across species, inherent biocompatibility, low immunogenicity (especially for collagen type I), and versatility in forming diverse architectures—including hydrogels, nanofibrous scaffolds, sponges, and films—make it an invaluable building block. Collagen can be extracted from animal sources such as bovine, porcine, or produced recombinantly, and its mechanical properties and degradation rate can be tuned by crosslinking, blending with other polymers, or combining with reinforcing materials. (2). This review describes collagen-based biomaterials in VTE, covering fundamental biological rationales, advanced fabrication strategies, preclinical validation, and translational challenges, while also highlighting emerging opportunities to engineer clinically translatable, next-generation vascular substitutes.

Collagen in the Vascular Wall: Biological assessments

Collagen is used in vascular tissue engineering because it naturally interacts with vascular cells. In blood vessels, collagen (mainly types I and III) does more than provide strength—it also stores growth factors that control healing and inflammation. Collagen binds directly to vascular smooth muscle cells (VSMCs) and endothelial cells (ECs) via integrin receptors, triggering signals that regulate cell behavior (4). For example, collagen keeps VSMCs in a healthy, contractile state (preventing harmful overgrowth) and helps ECs form a smooth, blood-clot-resistant lining. Therefore, testing collagen-based scaffolds should go beyond basic cell compatibility. Researchers must check whether the scaffold maintains VSMC function, supports EC barrier integrity, resists platelet sticking, and avoids chronic inflammation (5). Advanced lab models (like vascular chips with flow) are used to study how collagen structure affects these outcomes. Ultimately, understanding how collagen guides cell behavior is key to designing vascular grafts that work not just mechanically but also biologically for long-term success (6).

Structure and Function of the Vascular Extracellular Matrix

The vascular extracellular matrix is a dynamic, hierarchically organized network of collagens, elastin, proteoglycans, and glycoproteins. Collagen types I and III dominate the tunica media and adventitia, providing the tensile strength required to withstand pulsatile hemodynamic loads, whereas type IV collagen forms a specialized two-dimensional network in the subendothelial basement membrane to which endothelial cells directly attach(7). In healthy vessels, type I collagen fibrils in the media are oriented circumferentially around smooth muscle cells, while type IV collagen in the subendothelium

supports endothelial cell adhesion, spreading, and quiescence. Atherosclerosis and vascular injury disrupt this architecture, exposing interstitial collagens to flowing blood and consequently triggering platelet adhesion, coagulation, and smooth muscle cell phenotypic switching from a contractile to a synthetic, proliferative state. Therefore, any biomaterial intended for vascular reconstruction must recapitulate not only the bulk composition but also the topographic and mechanical cues of the native extracellular matrix to avoid these pathological cascades.

.Collagen Receptors and Cell–Matrix Interactions

Cellular recognition of collagen is mediated primarily by integrins α1β1 and α2β1, which bind the GFOGER sequence within the collagen triple helix, as well as by discoidin domain receptors DDR1 and DDR2, which function as receptor tyrosine kinases(8). Endothelial cells express α2β1 integrin, enabling firm adhesion to collagen type I-coated surfaces; however, unmodified collagen surfaces can paradoxically promote platelet adhesion unless antithrombotic moieties such as heparin are co-immobilized. Smooth muscle cells utilize both integrins and discoidin domain receptors to sense collagen matrix stiffness(9): compliant matrices favor a quiescent, contractile phenotype, whereas stiff matrices promote a dedifferentiated, migratory phenotype associated with intimal hyperplasia. This mechanosensing loop underscores the critical importance of matching scaffold mechanical properties to the target tissue, a design parameter that will recur throughout subsequent sections (10).

Collagen Sources and Molecular Configuration

Collagen for biomedical use is extracted predominantly from bovine or porcine skin and tendon, yielding type I atelocollagen rendered soluble and non-immunogenic by pepsin-mediated removal of telopeptides. Batch-to-batch variability in molecular weight distribution, cross-link density, and purity has historically impeded standardization (11). To address these limitations, recombinant human collagen (types I and III) expressed in yeast, plant, or mammalian systems has been developed, offering superior lot-to-lot consistency, elimination of zoonotic pathogen transmission, and the ability to introduce specific biofunctional domains through genetic modification (12). Studies have shown that electrospun bilayer vascular grafts incorporating recombinant human collagen peptides with polycaprolactone support human umbilical vein endothelial cell adhesion and rat smooth muscle cell infiltration in a manner comparable to or exceeding that of animal-derived collagen (13). Marine collagen, particularly from fish skin, has recently gained attention as a sustainable alternative free from mammalian disease concerns and acceptable in regions where bovine or porcine products are restricted. A single-step crosslinking method allows collagen to be used as a shear-recovering material for 3D extrusion printing. When this material is combined with vascular endothelial growth factor, it helps mesenchymal stem cells develop into angiogenic cells. Together, these tissue engineering strategies give us more options for precisely controlling how a scaffold behaves with living tissue and the immune system (14).

Collagen-Based Scaffolds for Vascular Tissue Engineering

Collagen is highly versatile and can be turned into hydrogels, fibrous meshes, decellularized matrices, or porous sponges, making it a key material for engineering blood vessels(15). This section reviews the main scaffold types: hydrogels that naturally form and support capillary-like networks but are mechanically weak; electrospun scaffolds that mimic natural

tissue fibers and are often mixed with synthetic polymers to handle blood pressure; crosslinking methods (using chemicals like EDC/NHS or genipin, or enzymes and sugars) that improve strength and resistance to breakdown; and decellularized vascular matrices that preserve natural collagen structure but need modification to prevent blood clotting(16, 17). Each approach has its own strengths and weaknesses, which determine whether it is best for lab research or actual medical use. Table 1.

Table1. Summary of Collagen-Based Vascular Scaffolds, Fabrication Methods, and Performance Outcomes

Scaffold Type	Key Features	Fabrication / Strategy	Performance / Outcome	Ref
Collagen hydrogels	Formed by pH/ temperature-induced fibrillogenesis; high water content; native-like fibrillar architecture	Monomer solution neutralization and warming	HUVECs embedded in collagen I hydrogel spontaneously form capillary-like networks under serum-free conditions; elastic modulus too low (tens to hundreds of Pascals) for arterial pressure	(18)
Electrospun collagen scaffolds	Nano- to micrometer fibers; blends with PCL, PLA, or polyurethane for strength	Triple-step electrospinning; EDC/ NHS crosslinking	Three-layer PLA/collagen/PLA-PCL/ PCL-gelatin graft: axial stress 9.56 MPa, radial stress 9.31 MPa; PCL/collagen/ heparin graft supports anticoagulation; 25% collagen I in PCL optimizes cell viability	(19)
PLA-collagen	Physical (DHT, UV), chemical (EDC/NHS, genipin), enzymatic (sortase A, Factor XIII),	EDC/NHS crosslinking of PLA-collagen; genipin	EDC/NHS increases max stress genipin enhances endothelial regeneration with low cytotoxicity	(20)
Decellularized vascular scaffolds	Preserved native ECM architecture (collagen + elastin); mechanical anisotropy	Decellularization of allogeneic/ xenogeneic vessels; freeze-drying + ethanol treatment for thrombogenicity reduction	Pure collagen or elastin scaffolds support de novo ECM synthesis (new collagen in elastin scaffold, new elastin in collagen scaffold); ethanol treatment reduces platelet adhesion and vWF adsorption by ~80% without exogenous chemicals	(21)

Advanced Biofabrication and Smart Material Design

The transition from passive scaffold design to active, bioinstructive constructs has been accelerated by emerging biofabrication technologies and rational modification of collagen at the molecular level.

3D Bioprinting of Collagen-Based Vascular Constructs

3D bioprinting allows researchers to create blood vessel structures that are shaped like real tissues and are highly accurate and reproducible. In a recent study, Lee et al(22), used a technique called FRESH to print collagen and rebuild parts of the human heart, including blood vessels. More recently, a new bioink made from lung tissue ECM and gelatin (GelMA) was used with a method called SLAM to print tube-like structures. This combination worked better than gelatin alone at helping endothelial cells attach, grow, and form vessel-like networks. Another advance uses multiple materials: printing vascular cells inside a collagen bath, which helps form capillary networks and increases VE-cadherin, a protein important for blood vessel walls(23). This approach creates open channels and interconnected networks that mimic natural small blood vessels. Even newer is 4D bioprinting, where collagen nanofibers with embedded nanoparticles shrink in a controlled way, causing the material to compact and reshape itself like developing tissue. Together, these methods blur the line between non-living materials and living tissues(24).

Functionalization with Growth Factors and Bioactive Cues

Collagen scaffolds can do more than just mimic tissue structure—they can also actively release bioactive molecules to boost their performance. For example, collagen matrices with heparin can release growth factor bFGF slowly, which helps blood vessels form on grafts(25). Heparin also prevents clotting and stores the growth factor. Another protein, pleiotrophin, when released from a collagen hydrogel, improves how well vascular grafts integrate with the body(26). Copper ions attached to a catechol coating on the material can produce nitric oxide, which helps keep blood vessels healthy—similar to what natural endothelium does(27). Attaching VEGF to collagen scaffolds (even those from fish) helps turn stem cells into blood vessel cells, offering a sustainable approach for engineering vascular tissues(28). These strategies turn collagen from a simple structural material into an active player in tissue regeneration.

Collagen-Based Interpenetrating Network (IPN) Hydrogels

Interpenetrating networks that combine collagen with a synthetic polymer represent a powerful strategy to decouple biological activity from mechanical properties(29). McCoy et al. developed a poly(ethylene glycol) (PEG)–collagen type I IPN in which endothelial cells and fibroblasts were co-encapsulated. By independently tuning the initial stiffness (via MMP-sensitive peptide cross-linker concentration) and the degradation rate (via cross-linker identity), they found that stiff and slowly degrading IPN hydrogels supported the most extensive vascular network assembly, while soft and rapidly degrading hydrogels showed reduced cell-mediated stiffening (30). Critically, the collagen fibrillar network was rapidly remodeled by the encapsulated cells; when collagen was added as dry-spun fibers or was not fully crosslinked, vasculogenic assembly was impaired, highlighting that a contiguous, three-dimensional fibrillar network is essential for supporting microvascular morphogenesis(31). These findings provide quantitative design rules for hydrogel-based vascularization strategies.

Cellular Responses and Remodeling in Collagen Scaffolds

The ultimate success of a collagen-based vascular graft depends on its dynamic interaction with host cells, which orchestrates the remodeling of the scaffold into a living tissue (4).Fig1.

Endothelial Cell Behavior on Collagen Substrates

Endothelial cells serve as the gatekeepers of vascular homeostasis. When seeded on collagen type I hydrogels, endothelial cells attach, spread, and form confluent monolayers more rapidly than on tissue culture plastic(32). However, a subtle understanding has emerged: whereas endothelial cells remain strictly confined to the surface of native collagen gels, they can invade collagen matrices that have been modified by covalent crosslinking or blended with other extracellular matrix components(33). Powell et al(34). demonstrated that endothelial cells, but not smooth muscle cells, are restricted to the surface of collagen gels—a behavior attributable to the synthesis of a smooth muscle-specific collagenase that enables smooth muscle cell invasion. This differential invasive capacity has profound implications for scaffold design: endothelialization of the luminal surface is essential for thromboresistance, but transmural endothelial migration must be prevented to avoid aberrant angiogenesis. Recent studies have refined these observations. When an appropriate three-dimensional matrix environment is provided, even smooth muscle cells can display blood vessel formation behavior, indicating that the collagen gel configuration directly dictates cellular morphogenesis. The integration of insoluble elastin into collagen scaffolds reduces stiffness, improves viscoelasticity, and induces a contractile phenotype in smooth muscle cells, thereby suppressing the synthetic phenotype associated with intimal hyperplasia. These cell–matrix interactions underscore the importance of designing scaffolds that differentially instruct endothelial and smooth muscle cell behavior (35).

Vascular Remodeling and Tissue Integration In Vivo

Studies in large animals have shown how collagen -based artificial blood vessels perform inside the body. In one study, a three-layer polyester tube coated with collagen was implanted as a neck artery bypass in sheep. After 161 days, 92% of the grafts were still open and working. The tissue had healed well, with a new inner layer forming. In another study, a double-layer graft was placed in the leg artery of dogs. After 8 weeks, it remained open without bulging or narrowing, and the inner surface was lined with cells that looked like natural endothelium. These results suggest that collagen-based grafts can become functional blood vessels under realistic conditions.However, the healing process is not always perfect (36, 37). Unmodified collagen scaffolds can be broken down by enzymes called MMPs, which come from immune cells. This can cause the graft to lose strength too soon. How fast the scaffold breaks down varies—from as little as 10 days for collagen sponges to 2 months for decellularized vessels. Macrophages tend to resorb collagen, which is first replaced by granulation tissue and then by scar-like fibrous tissue. So, the balance between scaffold breakdown and new tissue formation is key to long-term success (38).

Biodegradation and Immunological Considerations

The immune response to collagen implants depends on the source of collagen, how it is crosslinked, and any leftover antigens. Collagen from cows and pigs causes little immune reaction in humans because it is very similar to human collagen. However, the telopeptide parts (the main antigens) must be removed with enzymes to keep immunogenicity low. Uncrosslinked pig collagen coated on vascular grafts keeps its triple helix structure and low immunogenicity, similar to cow collagen, and supports new tissue growth without too much inflammation.(39)Chemical crosslinking improves strength but can leave toxic residues or create new antigens that cause chronic inflammation. Genipin -crosslinked collagen is less toxic than glutaraldehyde-crosslinked one, but the immune response still depends on the dose. Photooxidation or using pentagalloyl glucose for crosslinking on decellularized small blood vessels from animals improves their strength and biocompatibility while reducing inflammation, breakdown, and calcification in the body(40, 41). These findings show we must balance the need for mechanical strength with the need for immune compatibility.

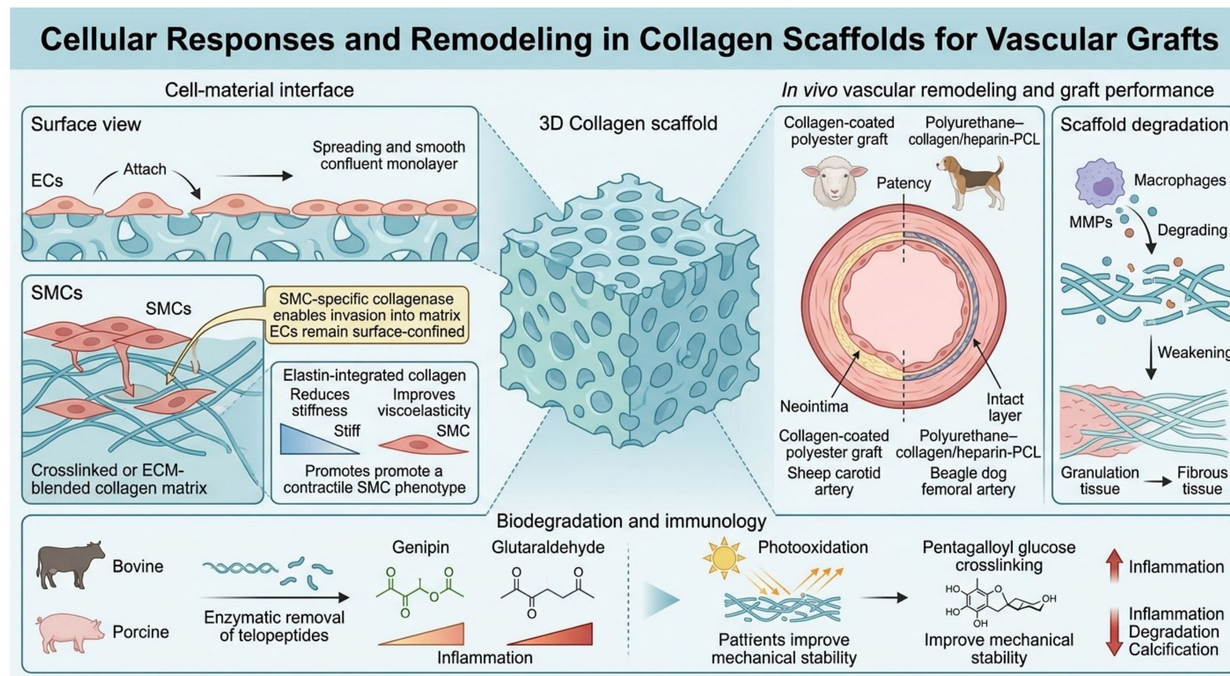

Fig. 1. Cellular responses and remodeling in collagen-based vascular grafts. The figure summarizes key interactions between cells and collagen scaffolds, along with in vivo remodeling outcomes and immunological considerations. The cell-material interface distinguishes between endothelial cells (ECs), which remain confined to the surface of unmodified collagen gels, and smooth muscle cells (SMCs), which invade the matrix via SMC-specific collagenase. Crosslinked or ECM-blended collagen matrices alter this behavior. The in vivo vascular remodeling panel illustrates outcomes such as neointima formation, graft integration (e.g., collagen-coated polyester grafts in sheep, polyurethane–collagen/heparin-PCL grafts in canine femoral arteries), and the transition from granulation tissue to fibrous tissue. The biodegradation and immunology section compares crosslinkers (genipin vs. glutaraldehyde), collagen sources (bovine, porcine) with enzymatic telopeptide removal, and methods such as photooxidation and pentagalloyl glucose crosslinking that modulate inflammation, degradation, and calcification.

Conclusion

Collagen-based biomaterials occupy a central position in the landscape of vascular tissue engineering, owing to their biological authenticity, versatility, and capacity for rational design. The field has progressed from simple collagen coatings to intricate, multicomponent constructs fabricated by electrospinning, 3D bioprinting, and interpenetrating network strategies, all supported by a robust mechanistic understanding of how collagen microstructure and crosslinking dictate cellular behavior. Preclinical studies in sheep and canine models have demonstrated that composite collagen–synthetic grafts can achieve patency rates exceeding 90% at mid-term follow-up, with histological evidence of endothelialization and neointima formation. Nonetheless, the translation of these technologies to routine clinical practice is impeded by unresolved challenges in thromboresistance, long-term remodeling, scalable manufacturing, and regulatory standardization. Future efforts must focus on the development of smart collagen materials that actively respond to the host environment—releasing anti-thrombotic agents, recruiting endogenous progenitor cells, and degrading at a rate precisely matched to new tissue formation. The integration of patient-specific iPSC-derived cells, genome editing for enhanced graft performance, and high-content screening in organ-on-a-chip platforms repre-

sents a plausible roadmap toward the next generation of living vascular grafts. With sustained investment and interdisciplinary collaboration, collagen-based biomaterials hold the realistic promise of transforming the surgical management of cardiovascular disease.

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