

Review

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Review

The Extracellular Matrix in Liver Regeneration: Biological and Therapeutic Insights

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Abstract

The liver possesses a remarkable regenerative capacity following injury, a process fundamentally orchestrated by the dynamic extracellular matrix (ECM). Far beyond a passive scaffold, the liver matrisome functions as an integrative mechano-biochemical circuit. It comprises a core structural network and regulatory non-core components that together establish a dynamic niche. This niche stores and releases mitogenic cues, transmits mechanical forces, and coordinates multicellular crosstalk. Through receptors like integrins and mechanosensitive channels, ECM-derived signals converge on key pathways, including Hippo-YAP/TAZ and Wnt/ β -catenin, to drive hepatocyte proliferation and tissue restructuring. The balance between matrix stabilization and remodeling dictates the outcome, guiding physiological regeneration versus fibrotic progression. Consequently, the ECM emerges as a central therapeutic target and a blueprint for engineering strategies aimed at restoring liver function. Strategies to recalibrate its composition, mechanics, and remodeling, from pharmacological inhibitors to bioengineered decellularized ECM scaffolds, hold transformative potential for steering liver repair and combating chronic disease.

Keywords: ECM remodeling; liver regeneration; matrix–cell signaling

1. Introduction

The liver plays essential roles in systemic metabolism, detoxification and immune homeostasis. In response to acute injury or partial tissue loss, it displays a remarkable capacity for regeneration [1]. Central to this process is the proliferative expansion of hepatocytes, the primary parenchymal cells that restore liver mass and function [2]. Following partial hepatectomy or acute toxic insult, quiescent hepatocytes rapidly re-enter the cell cycle to replace the lost tissue. At the same time, non-parenchymal cells (NPCs), including sinusoidal endothelial cells (SECs), Kupffer cells (KCs), liver stellate cells (HSCs) and biliary epithelial cells (BECs), coordinate angiogenic, inflammatory and remodeling programs that collectively sustain hepatocyte proliferation and tissue restructuring [3–5]. This highly orchestrated response is governed by temporally regulated cytokine and growth factor networks, together with precise paracrine communication among liver cell populations [6].

Among the diverse regulatory cues guiding liver repair, the extracellular matrix (ECM) plays an indispensable role [5]. Far from being a static scaffold, the liver ECM constitutes a dynamic structural and biochemical environment that actively directs hepatocyte behavior during regeneration [7,8].

Following liver injury, ECM remodeling critically influences the balance between tissue restoration and pathological repair by influencing cell proliferation, migration and tissue organization [7,9,10]. Appropriate ECM remodeling facilitates hepatocyte regeneration and architectural recovery, whereas excessive or disordered matrix accumulation disrupts these processes and promotes fibrotic progression [9,11]. Thus, the ECM serves not only as a structural framework but also as a central regulator of liver regeneration, ultimately determining whether injury leads to effective repair or chronic fibrosis.

Given that the ECM supports physiological regeneration while also driving fibrotic pathology when dysregulated, a deeper understanding of its dynamic regulation is crucial for the development of novel therapeutic strategies. This review considers the ECM across three interrelated dimensions: its composition, its structural features, and the surrounding multicellular microenvironment, and outlines how these elements collectively govern hepatocyte proliferation, regeneration, and tissue restoration following injury.

2. Dynamic Roles of the Core and Non-Core Liver Matrisomal Components in Balancing Liver Regeneration and Fibrosis

The liver ECM orchestrates liver regeneration through a hierarchical partnership between its core and non-core matrisome components. The core matrisome establishes the structural and biochemical foundation, whereas the non-core matrisome refines this framework through enzymatic remodeling, affiliated protein activity and spatiotemporal regulation of signaling molecules. Together, these elements function as a biomechanical and biochemical integrator that converts injury cues into regenerative programs. The balance this system maintains, defined by the interplay between stability and plasticity, ultimately shapes the outcome of liver repair and positions the ECM as a central regulator of regeneration versus fibrosis (Figure 1).

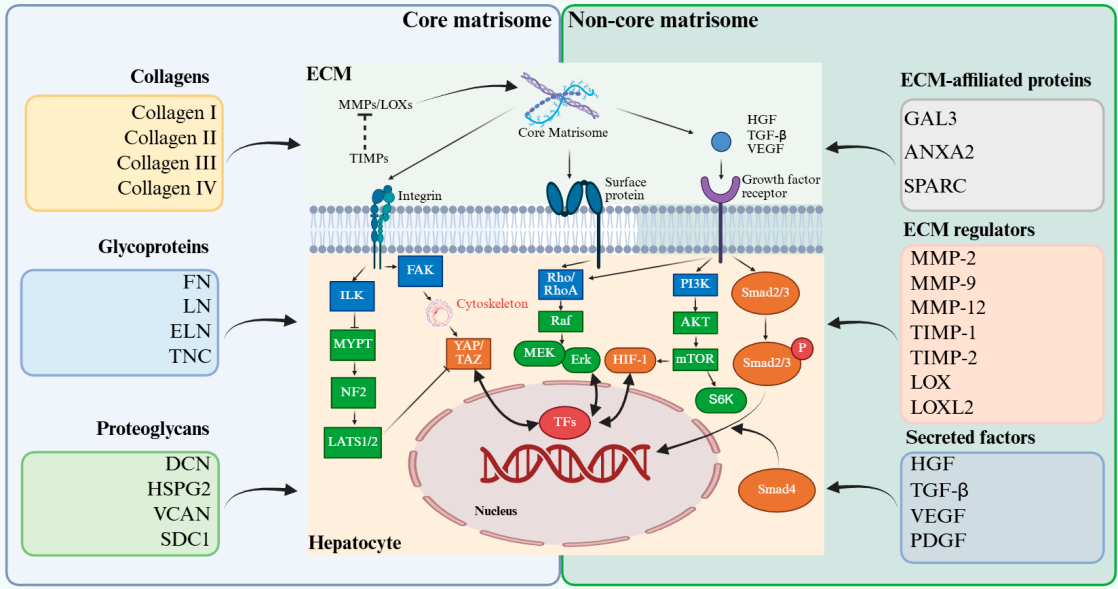


Figure 1. The liver matrisome as an integrative regulator of liver regeneration.

3.1. Core Matrisome

The liver core matrisome, which consists of collagens, glycoproteins and proteoglycans, forms the fundamental structural and biochemical foundation for liver regeneration [8,12,13]. During the early regenerative phase, activated HSCs and periportal hepatocytes rapidly synthesize fibrillar collagens, such as types I and III, to assemble a provisional scaffold that preserves parenchymal architecture and transmits mechanosensory cues through integrin focal adhesion and cytoskeletal signaling pathways [13–15]. This transient collagenous framework not only provides tensile support

but also acts as a reservoir for growth factors including hepatocyte growth factor (HGF) and epidermal growth factor (EGF), enabling spatially controlled release that promotes synchronized hepatocyte proliferation [16–18]. Concurrently, ECM glycoproteins, most prominently fibronectin, laminins and tenascin C, coordinate cell adhesion, migration and survival [19,20]. Fibronectin engages integrin $\alpha 5 \beta 1$ to activate cytoprotective signaling, whereas laminins and elastin help restore lobular polarity and mechanical elasticity, forming fibrillar conduits that guide the movement of hepatocytes and progenitor cells [21]. Proteoglycans such as decorin (DCN), perlecan and versican (VCNA) further refine the regenerative niche by temporally sequestering and releasing mitogenic factors and by buffering profibrotic mediators, including transforming growth factor- β (TGF- β) [22,23].

Collectively, these core ECM components form a dynamic and adaptive matrix that integrates biochemical gradients with mechanical cues to sustain tissue plasticity and functional recovery [18,24]. The balance between structural stability and matrix remodeling is a defining feature of successful regeneration, and it distinguishes physiological repair from maladaptive fibrotic responses [25]. The precision with which the core matrisome governs this transition highlights its role not as a passive scaffold but as an active conductor in the orchestration of liver regeneration, coordinating the restoration of liver structure and function.

3.2. Non-Core Matrisome

Beyond the core matrisome, the non-core matrisome encompasses a broad spectrum of ECM regulators, ECM-affiliated proteins and matrix-bound secreted factors that collectively fine-tune the regenerative niche [26]. Matrix metalloproteinases (MMPs) and their tissue inhibitors (TIMPs) form a tightly regulated proteolytic system that controls the rate of ECM turnover, releasing sequestered mitogens while preventing excessive degradation and structural collapse [27,28]. Complementing this process, lysyl oxidase family enzymes (LOX and LOXL isoforms) catalyze collagen cross-linking to enhance mechanical stability while preserving the pliability required for cell migration and tissue reorganization [29–31]. ECM-affiliated proteins such as galectin-3 (GAL3), annexin A2 (ANXA2) and secreted protein acidic and rich in cysteine (SPARC) function as molecular modulators of cell-matrix adhesion, immune-cell recruitment and angiogenic remodeling, thereby linking biochemical cues to tissue morphogenesis [32–34]. Simultaneously, the ECM serves as a dynamic reservoir for secreted growth factors such as HGF, TGF- β , vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF), with their bioavailability regulated by proteolytic activity and mechanical inputs [26,35]. The controlled release of these ligands in space and time provides the precision necessary for synchronized hepatocyte proliferation and vascular repair [36].

Collectively, these non-core matrisome components transform the ECM into an information-rich signaling interface that integrates proteolytic remodeling, mechanical feedback, and paracrine crosstalk [37]. Through these mechanisms, they critically influence the balance between adaptive regeneration and pathological scar formation. Thus, deciphering how non-core matrisome elements dynamically recalibrate matrix composition and mechanics may unlock novel strategies to reprogram fibrotic ECM toward a pro-regenerative state.

3. Liver ECM Orchestrates Regeneration Through a Mechano-Biochemical Circuit

The ECM that emerges after liver injury is not a passive scaffold but a dynamic and instructive niche that integrates mechanical and biochemical cues to guide tissue regeneration [24,38,39]. Activation of LOX family enzymes (LOX and LOXL1–4) promotes covalent cross-linking of type I and type III collagens, strengthening the newly formed matrix and modulating its mechanical properties [40,41]. This stabilization preserves the cohesion of the regenerating parenchyma while avoiding excessive stiffening that would favor fibrotic progression [42,43]. In parallel, MMPs such as MMP-2 and MMP-9, along with related proteases, remodel the ECM by cleaving overly stabilized

fibrils and releasing matrix-bound morphogens, including HGF, VEGF and epidermal growth factor (EGF) [12,42]. The dynamic balance between LOX-mediated stabilization and MMP-driven remodeling defines an optimal microenvironment that harmonizes stiffness, porosity and ligand availability to support hepatocyte adhesion, polarization and coordinated proliferation [41,44].

Hepatocytes and NPCs sense these physical cues through specialized mechanosensors. Integrins (e.g., $\alpha 5 \beta 1$, $\alpha v \beta 3$) clustered in focal adhesions recruit focal adhesion kinase (FAK) and Proto-oncogene tyrosine-protein kinase Src, activating the RAS-RAF-MEK-ERK and PI3K-AKT-mTOR cascades to promote anabolic growth and survival [40,45]. Concurrently, integrin engagement stimulates RhoA-ROCK-mediated actomyosin contractility, amplifying FAK signaling in a positive feedback loop that refines focal adhesion maturation and strength [46]. Mechanical shear and tensile strain are also sensed by ion channels such as Piezo1 and TRPV4; subsequent Ca^{2+} influx activates calcineurin-NFAT and CaMKII/PKC-MAPK pathways, reinforcing proliferation-associated transcription and promoting Yes-associated protein/transcriptional coactivator with PDZ-binding motif (YAP/TAZ) dephosphorylation [47,48]. Central to this network is the Hippo-YAP/TAZ axis: increased cytoskeletal tension or matrix rigidity suppresses MST1/2-LATS1/2 kinase activity, enabling YAP and TAZ to translocate into the nucleus; there, they partner with TEA domain transcription factor family (TEAD) transcription factors to drive expression of pro-proliferative genes such as Cyclin D1, connective tissue growth factor (CTGF), Cysteine-rich angiogenic inducer 61 (Cyr61), and amphiregulin (AREG) to induce pro-proliferative genes such as Cyclin D1, CTGF, Cyr61, and AREG [46,49]. This mechanical circuitry further converges with canonical Wnt/ β -catenin signaling, destruction complex (Axin/GSK3 β) and stabilizing β -catenin, which then cooperates with T cell factor (TCF), and is further potentiated by YAP/TAZ activation, to promote transcriptional synergy that drives hepatocytes through the G1/S transition and restores liver mass [40,44].

Mechanical homeostasis is equally crucial during the resolution phase. Persistent LOX activity or excessive cross-linking locks the ECM into a rigid, fibrotic state, restricting hepatocyte migration and sustaining HSC activation [43]. Conversely, broad MMP inhibition compromises matrix clearance and growth factor release [12]. Disruption of integrin-FAK signaling or enforced Hippo pathway activation impairs YAP/TAZ-dependent transcription and delays tissue recovery [34]. Restoring optimal matrix compliance, achieved through balanced LOX/MMP activity and tuned actomyosin tension, reactivates FAK-ERK/AKT, YAP/TAZ-TEAD, and β -catenin-TCF programs in a coordinated manner [34,43]. Together, these insights support a unified framework in which the liver ECM functions as an integrated mechano-biochemical circuit, synchronizing matrix stiffness, calcium flux and transcriptional crosstalk to orchestrate regeneration [50]. The liver exemplifies, perhaps more clearly than any other organ, that successful repair depends not only on biochemistry but also on biomechanics [51,52].

Taken together, the liver's regenerative capability is fundamentally guided by a mechano-biochemical network centered on the dynamic ECM (Figure 2). This system establishes a tunable mechanical microenvironment through balanced LOX- and MMP-mediated remodeling, creating an optimal physical niche for hepatocyte proliferation. Mechanical cues are transduced via integrin-mediated pathways and mechanosensitive ion channels, converging on key transcriptional regulators including YAP/TAZ, β -catenin, and ERK-dependent signaling to coordinate proliferative and morphogenetic programs. This regulatory network enables transient physical stimuli to elicit sustained pro-regenerative responses, yet the same mechanisms can promote fibrosis and pathogenesis under conditions of sustained mechanical imbalance. Future therapeutic strategies aimed at restoring regenerative capacity will need to target this intricate mechanochemical code that bridges physical cues with biological outcomes in liver repair.

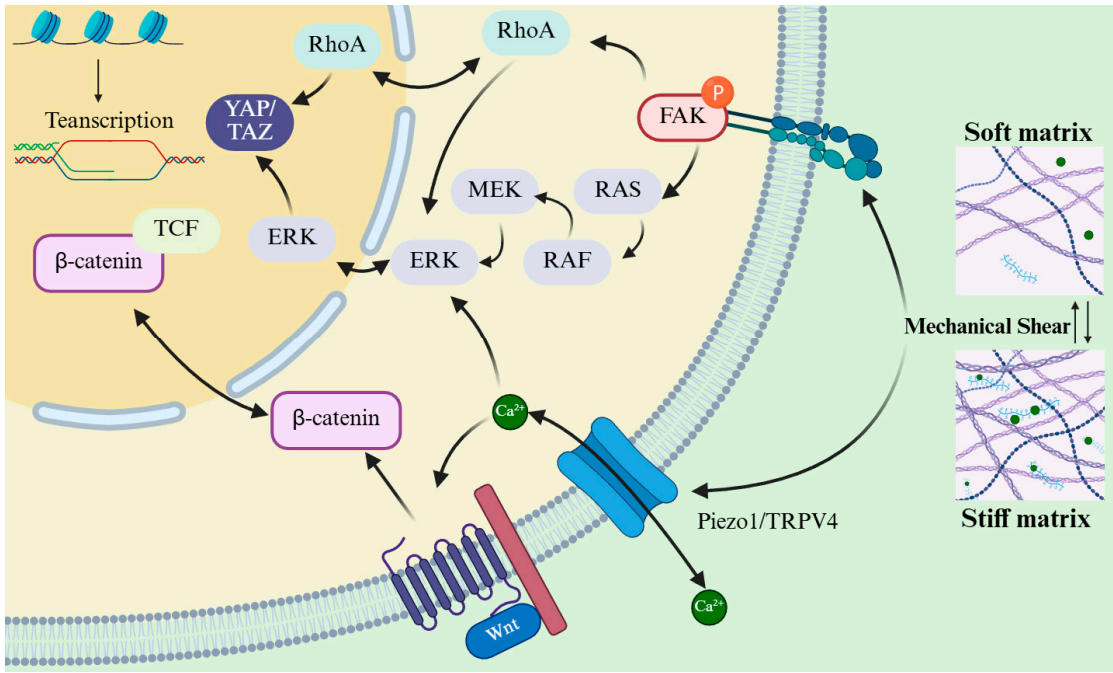


Figure 2. Mechanical signaling networks in liver regeneration.

4. Liver ECM Coordinates Liver Regeneration Through Multicellular Crosstalk

Following acute liver injury or partial hepatectomy, the liver ECM rapidly and coordinately transitions from a structural scaffold into a dynamic signaling hub. This reprogrammed microenvironment integrates biochemical, mechanical, and immunological cues to direct multicellular regeneration [51,52]. Its transient remodeling is sustained through intricate crosstalk among hepatocytes, NPCs, and BECs, each contributing distinct ECM-modifying and -sensing capabilities [53,54] (Figure 3).

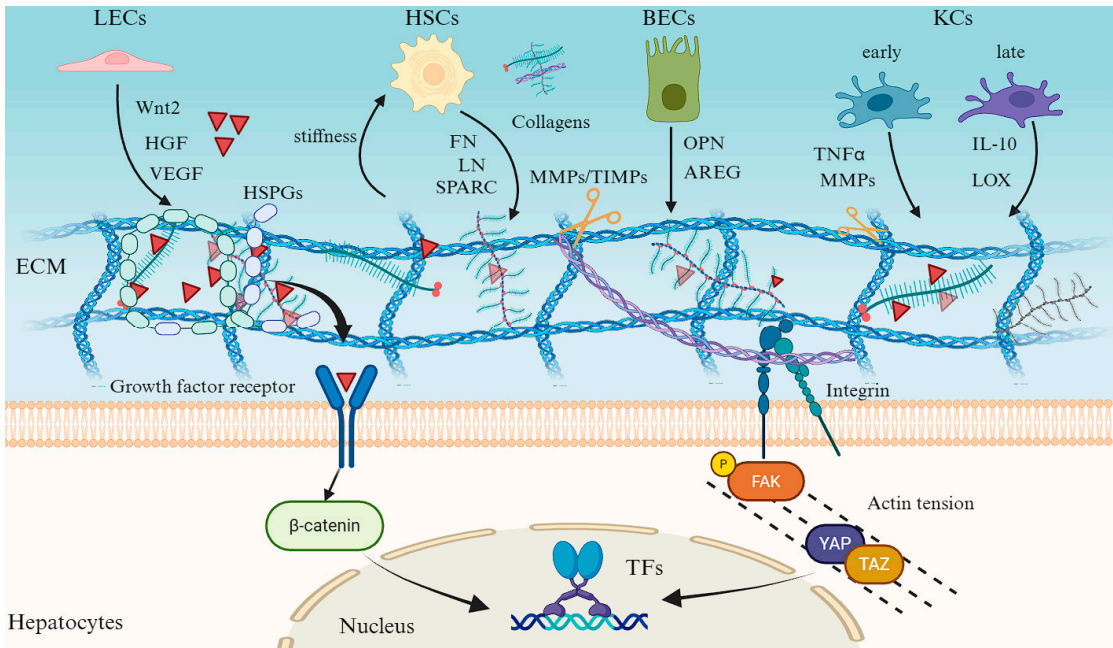


Figure 3. Multicellular crosstalk mediated by the liver ECM during regeneration.

Liver SECs act as early orchestrators of the regenerative niche. Through the release of angiocrine factors, including Wnt2, HGF, and VEGF, they stimulate hepatocyte proliferation and remodel the

surrounding matrix through uPA- and MMP-dependent pathways [55–58]. These secreted factors are sequestered within the ECM by heparan sulfate proteoglycans and are subsequently released in response to local mechanical and proteolytic signals, creating spatially resolved mitogenic gradients that align with sinusoidal flow [59,60].

Concurrently, HSCs sense alterations in ECM stiffness and composition through integrin-FAK and YAP/TAZ-mediated mechanotransduction [61,62]. This prompts their transition into a transiently activated, pro-regenerative phenotype. In this state, HSCs deposit fibronectin, laminin, and matricellular proteins such as SPARC and osteopontin (OPN), which provide both adhesive ligands and mechanical elasticity to facilitate hepatocyte spreading, ductular cell migration, and vascular stabilization [63–66]. These HSCs-derived matrices also act as molecular scaffolds that coordinate growth-factor presentation and regulate endothelial permeability, thereby linking mechanical feedback to metabolic and transcriptional reprogramming in regenerating hepatocytes [67,68].

BECs, traditionally regarded as passive ductal components, are now recognized as pivotal contributors to ECM remodeling and regenerative patterning. Upon injury, BECs upregulate the secretion of matricrine mediators such as AREG, OPN, and CTGF, which bind ECM glycosaminoglycans and enhance hepatocyte proliferation at the ductal-parenchymal interface [69–72]. Simultaneously, BECs release MMPs and TIMPs, enabling finely tuned ECM turnover that facilitates ductular-reaction expansion and the migration of progenitor-like cells into periportal zones [73–75]. Crosstalk between BECs and KCs further amplifies this process: KCs-derived Wnt ligands sustain BECs-to-hepatocytes differentiation, whereas BECs-derived cytokines such as IL-6 and OPN recruit KCs and shape their activation state toward a reparative phenotype [6,76,77].

KCs and infiltrating monocyte-derived macrophages serve as temporal conductors of ECM homeostasis. During the early inflammatory phase, these macrophages release TNF- α , MMP-9, and MMP-12 to degrade necrotic ECM and release matrix-bound mitogens such as HGF and VEGF [78–80]. As regeneration proceeds, macrophages shift to an IL-10–dominant, M2-like state, promoting ECM reassembly through induction of collagen cross-linking enzymes (LOX family) and secretion of anti-fibrotic mediators [81–83]. This biphasic pattern of ECM degradation and reconstitution ensures that hepatocytes and BECs are exposed to temporally ordered mechanical and biochemical cues—initially permissive for proliferation, subsequently restrictive to facilitate termination and remodeling [84–86].

Ultimately, these intercellular interactions convert the ECM into a self-regulating mechano-biochemical network. Through integrin signaling, cytoskeletal tension and mechanosensitive transcriptional programs such as YAP/TAZ and β -catenin, hepatocytes and NPCs continuously interpret and reshape their matrix context. The resulting regenerative circuit, which integrates endothelial angiocrine output, HSC mechanotransduction, macrophage-driven remodeling and cholangiocyte plasticity, precisely restores liver architecture and function. In essence, liver regeneration exemplifies a paradigm in which ECM dynamics act as both the language and the logic of multicellular coordination, translating injury-induced physical and molecular perturbations into an ordered program of tissue reconstruction.

5. ECM as a Therapeutic Target and Engineering Blueprint Orchestrating Liver Regeneration

Recent advances in liver regenerative medicine increasingly recognize the ECM not merely as a structural scaffold, but as a therapeutic target and engineering template capable of directing cellular behavior and tissue repair [87,88]. Modulating ECM composition, cross-linking, and mechanical properties has emerged as a powerful strategy to promote liver regeneration after injury or resection (Table 1).

Pharmacologic targeting of fibrogenic signaling constitutes a major class of ECM-directed therapies. Within this framework, selected interventions targeting the TGF- β axis exert direct pro-regenerative effects in addition to antifibrotic activity. As a master regulator of hepatic stellate cell

activation and SMAD2/3-driven matrix deposition, TGF- β signaling is antagonized by recombinant decorin, which sequesters extracellular ligands, and by LY2157299, a selective TGF- β receptor I inhibitor, both of which are associated with enhanced hepatocyte proliferation and accelerated liver mass recovery following partial hepatectomy or toxic injury [89–91]. Other modulators of this pathway, including SB525334, phosphocreatine, 3-HBI and astaxanthin, primarily attenuate fibrogenic and oxidative injury, thereby relieving microenvironmental constraints that permit, rather than directly induce, endogenous regeneration [92–95]. Therapeutic targeting of ECM biomechanics further modulates regenerative competence. Collagen cross-linking mediated by LOX and LOXL enzymes drives matrix stiffening and limits tissue compliance, thereby constraining hepatocyte renewal [29]. Selective inhibition of LOXL2 using AB0023 or GS341 directly couples matrix softening with hepatocyte proliferation, whereas broader LOX inhibition predominantly suppresses pathological remodeling and injury, exerting indirect pro-regenerative effects[31,96–98]. Dual LOXL2/LOXL3 inhibition with PXS-5153A similarly improves matrix compliance, establishing a biomechanical milieu permissive for regeneration [99]. Dynamic regulation of ECM turnover constitutes an additional axis of regenerative control. Augmentation of matrix degradation through MMP-13 gene delivery or MMP-9 suppression promotes hepatocyte cell-cycle re-entry and liver regeneration, in part by releasing matrix-sequestered mitogenic cues and restoring ECM plasticity [100–103]. Finally, canonical regenerative signaling pathways are tightly interwoven with ECM remodeling. Normalization of aberrant Wnt activity using DKK1 or sFRP5 restores hepatocyte proliferative capacity, while disruption of β -catenin-CBP-dependent transcription with ICG-001 robustly enhances hepatocyte proliferation across experimental systems [104–106]. PRI-724 demonstrates early clinical activity in HCV-associated fibrosis, consistent with enhanced regenerative competence despite limited direct proliferation readouts [107,108]. Recombinant BMP-7 represents a bona fide pro-regenerative cue following partial hepatectomy, acting through stromal reprogramming and coordinated ECM remodeling to support hepatocyte renewal [109].

In parallel, bioengineering strategies increasingly seek to reconstruct the instructive roles of liver ECM. Among these, decellularized ECM (dECM) hydrogels and scaffolds are the most established platforms, retaining organ-specific biochemical complexity and supporting hepatocyte adhesion, vascularization and ductal morphogenesis in preclinical models [26,110,111]. Advanced dECM configurations, such as cryogelated scaffolds and nanoparticle-functionalized ECM hydrogels, restore liver architecture while modulating local immune responses [112]. Injectable immunoregulatory hydrogels promote hepatoprotective macrophage phenotypes in ischemia–reperfusion models, whereas THBS1-enriched dECM bioinks enhance liver organoid maturation by recapitulating developmental cues [113,114]. Other formulations, including self-assembling dECM adhesives, bioengineered lobular constructs, and hydrogels optimized for hepatocyte transplantation or culture, further expand the regenerative toolkit [115–118]. As dECM platforms evolve in biological specificity and design flexibility, they are increasingly viewed not as passive scaffolds, but as programmable matrices capable of directing liver repair.

Table 1. Summary of ECM-targeted strategies for liver regeneration.

Target	Intervention	Models	Effect	References
TGF- β	Recombinant decorin (binds to TGF- β)	PHx or CCl ₄ mouse models	++	[89]
	LY2157299	PHx or CCl ₄ mouse models	+	[90,91]
LOXL2	AB0023	TAA or DDC mouse models	+++	[31]
	GS341	CCl ₄ mouse models	++	[96]
MMP-13 MMP-9	MMP13-encoding plasmids (pBGI-MMP13)	TAA mouse models	++	[101]
	MMP-9 antisense oligonucleotides	PHx rat models	++	[103]

Wnt3a/4/5a	DKK1	BDL mouse models, primary mouse HSCs	++	[104]
Wnt5a	sFRP5	CCl ₄ mouse models	+	[105]
Wnt pathway	ICG-001	CCl ₄ mouse models, LX-2 cells, primary human fibroblasts	++	[106]
	PRI-724	Human HCV	+	[108]
BMP-7	Recombinant human BMP-7 (rhBMP-7)	PHx mouse models	+	
	Injectable hydrogel	Rat models of liver ischemia/reperfusion injury	++	[113]
dECM	L-ECM hydrogel	TAA rat models	++	[115]
	dECM-Cryogel implantable scaffold	Liver failure rat models	+	[116]
	Self-assembling dECM hydrogel adhesive	Rabbit models of hemorrhage liver injury	++	[117]
	dECM-based bioengineered lobules	Mouse models of acute liver failure	++	[118]
	THBS1-dECM	PHx mouse models, primary mouse hepatocytes	+++	[114]
	L-ECM hydrogel	Rat primary hepatocytes	++	[112]

* the observed effect was categorized into three levels based on the percentage of response: + (0–25%), ++ (26–50%), +++ (51–75%).

Collectively, these ECM-centered molecular and engineering innovations converge on a common principle: effective liver regeneration depends on the restoration of a compliant, growth-factor-responsive, and dynamically remodeled ECM. By simultaneously recalibrating biochemical signaling and mechanical feedback, ECM-targeted therapeutics and biomimetic scaffolds hold transformative potential for clinical translation—offering regenerative platforms that not only repair but actively reprogram the injured liver toward physiological homeostasis.

6. Discussion

Liver regeneration exemplifies a remarkable capacity for tissue restoration in adults, with the ECM now recognized as a central regulatory platform rather than a passive scaffold. Acting as an integrative mechano-biochemical circuit, the ECM converts structural, cellular and molecular inputs into spatially and temporally organized programs that determine regenerative success or failure, balancing functional repair against fibrotic progression. Looking ahead, a major frontier lies in decoding the ECM as a multicomponent signaling system. Future research can move beyond cataloging individual matrix constituents and instead elucidate how biochemical cues, mechanical forces and intercellular communication are coherently integrated within the matrix microenvironment. Systems-level insights enabled by advanced omics, bioinformatics and engineered in vitro models will be critical for predicting and therapeutically steering regenerative outcomes with precision.

Translationally, the ECM is rapidly emerging as both a therapeutic target and an engineering blueprint. Strategies that recalibrate matrix stiffness, restore growth factor bioavailability or interfere with pathological cross-linking show promise in redirecting fibrotic microenvironments toward regeneration. At the same time, biofabricated scaffolds such as dECM hydrogels, zoned bioprinted constructs and EV-functionalized matrices increasingly recapitulate the native matrix niche, providing new avenues for organ repair and replacement. The convergence of matrix biology with mechanobiology, biomaterials science and computational modeling is opening transformative opportunities for the treatment of liver disease. By leveraging the ECM not only as a target but also as an instructional tool, regenerative

hepatology can shift from a reactive discipline to a proactive one, designing microenvironments that do not merely respond to injury but actively orchestrate tissue reconstruction.

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Abbreviations

The following abbreviations are used in this manuscript:

ANXA2	Annexin A2
AREG	Amphiregulin
BDL	Bile duct ligation
BECs	Biliary epithelial cells
BMP-7	Bone morphogenetic protein 7
BAPN	β-aminopropionitrile
CTGF	Connective tissue growth factor
CYR61	Cysteine-rich angiogenic inducer 61
dECM	Decellularized extracellular matrix
DCN	Decorin
DDC	3,5-diethoxycarbonyl-1,4-dihydrocollidine model
ECM	Extracellular matrix
EGF	Epidermal growth factor
EV	Extracellular vesicle
FAK	Focal adhesion kinase
GAL3	Galectin-3
GSK3β	Glycogen synthase kinase 3 beta
HA	Hyaluronic acid
HCV	Hepatitis C virus
HGF	Hepatocyte growth factor
HSC	Hepatic stellate cell
KC	Kupffer cell
LOX/LOXL	Lysyl oxidase / lysyl oxidase-like enzymes
LSEC	Liver sinusoidal endothelial cells
MMPs	Matrix metalloproteinases
NASH	Non-alcoholic steatohepatitis
NFAT	Nuclear factor of activated T cells
OPN	Osteopontin
PDGF	Platelet-derived growth factor
PHx	Partial hepatectomy
RAF	Rapidly accelerated fibrosarcoma kinase
RAS	Rat sarcoma family GTPase
SPARC	Secreted protein acidic and rich in cysteine
SPP1	Secreted phosphoprotein 1
TAA	Thioacetamide

TCF	T cell factor
TEAD	TEA domain transcription factor family
TIMPs	Tissue inhibitors of metalloproteinases
TRPV4	Transient receptor potential vanilloid 4
VCAN	Versican
VEGF	Vascular endothelial growth factor

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