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Nanofiber Scaffolds for Spinal Cord Injury Repair: Mimicking the Brain's Extracellular Matrix — A Narrative Review

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Abstract

Spinal cord injury (SCI) results in severe and often irreversible neurological deficits owing to the disruption of neuronal circuits and the limited regenerative capacity of central nervous system axons. Conventional therapies provide only partial relief, underscoring the need for advanced biomaterial-based interventions. Nanofiber scaffolds that emulate the architecture and biochemical functionality of the native extracellular matrix (ECM) have emerged as powerful platforms to promote axonal regeneration and neural repair. This review synthesizes recent progress in the design of nanofiber scaffolds for SCI, emphasizing strategies that replicate key ECM features—including structural anisotropy, mechanical compliance, and presentation of bioactive ligands. We outline the composition and signaling roles of the healthy brain ECM, describe its pathological remodeling after injury, and relate these changes to the design criteria for functional scaffolds. Current fabrication approaches, particularly electrospinning of natural, synthetic, and hybrid polymers, are discussed in the context of fiber alignment, porosity, and surface functionalization. Mechanistic insights into how these constructs guide axonal extension, modulate glial and immune responses, and support neuronal survival are critically evaluated. Preclinical evidence demonstrates significant anatomical and functional recovery in rodent models, although challenges remain in integration, controlled degradation, immunogenicity, and scalable manufacturing. Finally, emerging directions—including stimulus-responsive and combinatorial scaffold systems—are highlighted as avenues toward clinically translatable solutions for restoring function after SCI.

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Literature Search

This narrative review synthesizes peer-reviewed literature on nanofiber scaffolds for spinal cord injury (SCI) with emphasis on extracellular matrix (ECM) biomimicry, architectural guidance, biochemical functionalization, mechanisms of action, and translation. Sources were identified in PubMed and Web of Science using combinations of "spinal cord injury," "nanofiber," "electrospinning," "extracellular matrix," "axonal regeneration," "CSPG," and "laminin," prioritizing studies from the past 10–12 years while including seminal earlier work. Reference lists of key articles were hand-searched. In vivo preclinical studies were emphasized; in vitro data were included when mechanistically clarifying. This is not a systematic review; risk of bias was considered qualitatively (blinding, randomization, sample-size reporting, and outcome measures).

Introduction

Spinal cord injury (SCI) is a catastrophic neurological condition that results in severe motor, sensory, and

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autonomic dysfunction, profoundly affecting patients' independence and quality of life. The ensuing paralysis, sensory loss, and neuropathic pain impose lifelong burdens on individuals and families while posing major socioeconomic and healthcare challenges worldwide (1). Despite decades of intensive research, therapeutic options capable of restoring meaningful neurological function remain elusive. Current clinical management focuses on acute stabilization, prevention of secondary damage, and rehabilitation, with limited capacity for neural tissue regeneration or circuit reconstruction (2).

A principal obstacle to recovery lies in the central nervous system's (CNS) intrinsic inability to regenerate damaged axons after injury. Following SCI, a cascade of primary and secondary pathological events unfolds, including ischemia, neuronal death, demyelination, and chronic inflammation. These processes culminate in the formation of a dense glial scar composed of reactive astrocytes, microglia, and infiltrating immune cells (3). The scar serves both protective and inhibitory roles—it restores physical integrity but erects a formidable biochemical and mechanical barrier to axonal regrowth (4, 5). Among the molecular components implicated, the extracellular matrix (ECM) is pivotal. In the healthy CNS, the ECM orchestrates neuronal adhesion, migration, and synaptic signaling (6). After SCI, it undergoes profound remodeling with deposition of chondroitin sulfate proteoglycans (CSPGs) and other inhibitory molecules within the glial scar, transforming a growth-permissive substrate into an inhibitory terrain that impedes axonal extension (7).

In this context, biomaterial-based regenerative strategies have emerged as a promising frontier. Nanofiber scaffolds uniquely emulate the architecture and biochemical functionality of the native ECM (8, 9). Engineered at the nanoscale, they provide aligned topographical cues to direct axonal elongation, deliver therapeutic agents, and modulate inflammation to create a permissive niche (10). By tuning composition, fiber orientation, porosity, and surface chemistry, these scaffolds can bridge lesion gaps and facilitate re-establishment of neural connectivity (11). This review outlines the structural and functional organization of the CNS ECM and its disruption after injury; examines how these biological principles inform nanofiber design and fabrication; evaluates mechanisms of action and preclinical evidence; and discusses translational challenges and emerging strategies—including smart and combinatorial scaffolds—that could advance these materials toward clinical application.

1. The Brain's Extracellular Matrix: A Blueprint for Regeneration

1.1 Composition and Architecture of the Healthy CNS ECM

The CNS ECM is a dynamic, highly organized network that permeates neural tissue and regulates development, synaptic plasticity, and repair. Its macromolecular constituents—proteoglycans with sulfated and non-sulfated glycosaminoglycans (GAGs), fibrous proteins, and specialized glycoproteins—create a hydrated, viscoelastic milieu that stores and presents signaling cues. Hyaluronic acid maintains tissue hydration and facilitates cell motility; proteoglycans such as aggrecan and versican bind growth factors and modulate pericellular mechanics; and fibrous proteins including collagen (types IV and VI), laminin, and fibronectin provide structural integrity and integrin-mediated signaling. Tenascins and thrombospondins tune synaptogenesis and neuron–glia communication. At the tissue level, the ECM forms perineuronal nets and perivascular matrices that shape diffusion, stabilize synapses, and contribute to blood–brain barrier function. Its nanoscale porosity, anisotropy, and viscoelasticity are closely matched to neuronal function.

1.2 Functional Roles in Development and Plasticity

Beyond structure, the ECM governs neuronal migration, axonal pathfinding, and synaptic organization during development. In the mature CNS it remains plastic, remodeling to accommodate learning-related changes while perineuronal nets constrain excessive plasticity to stabilize circuits. ECM-bound trophic factors support neuronal survival and homeostasis, and ECM–integrin interactions orchestrate cytoskeletal dynamics in growth cones. Through crosstalk with astrocytes and microglia, the ECM also calibrates neuroimmune tone and glial phenotypes.

1.3 ECM Remodeling and Glial Scarring After SCI

SCI disrupts this matrix and precipitates a secondary cascade characterized by inflammation, demyelination, and glial scar formation. Reactive astrocytes and oligodendrocyte-lineage cells deposit CSPGs such as NG2 and aggrecan, which engage neuronal receptors and trigger growth-cone collapse (12, 13). The scar restores tissue continuity but creates a dense, biochemically inhibitory and mechanically mismatched barrier. Concurrent microglial activation and cytokine release reinforce a chronic inflammatory state, shifting the niche from growth-permissive to growth-inhibitory(4) .

1.4 Design Implications for Biomimicry

A rational scaffold should reinstate permissive ECM features while mitigating post-injury inhibition. Three principles follow: reproduce anisotropic topography that channels axons; supply adhesive and trophic cues that stabilize and extend neurites; and neutralize inhibitory signaling associated with CSPGs and chronic

inflammation. These principles underpin nanofiber scaffold design for SCI repair.

2. Nanofiber Scaffolds: Design Principles and Fabrication

2.1 Material Choice and Hybridization

Material selection dictates mechanics, degradation, and functionalization capacity. Natural polymers (collagen, fibrin, hyaluronic acid, alginate) offer intrinsic bioactivity and cell affinity but often require reinforcement or cross-linking for stability. Synthetic polyesters (PLGA, PCL) and hydrophilic frameworks (PEG) provide tunable strength and predictable degradation yet lack inherent adhesivity, necessitating biochemical modification (14). Hybrid systems leverage the bioactivity of natural components within a mechanically reliable synthetic backbone—for example, collagen-functionalized PLGA or fibrin-modified PCL—to couple cell-instructive chemistry with precise engineering control (15-17).

2.2 Fabricating ECM-Like Architecture

Electrospinning is the most versatile route to ECM-mimetic fibers across tens of nanometers to micrometers, enabling control over fiber diameter, alignment, and porosity via polymer concentration, voltage, collector speed, and ambient conditions. Rotating or gap collectors produce aligned arrays that emulate the anisotropy of white-matter tracts and provide potent contact guidance (18). Self-assembling peptide systems offer exquisitely bioactive nanofibrils but limited load-bearing capacity, whereas emerging 3D bioprinting approaches enable macro-scale patterning and multimaterial constructs while still chasing true nanoscale fidelity. Regardless of method, matching viscoelasticity to spinal cord tissue minimizes interfacial stress and favors integration.

2.3 Programming Biochemical Interactions

Structural fidelity alone is insufficient. Covalent grafting or physical incorporation of laminin-derived peptides, RGD motifs, and ECM proteins enhances integrin engagement and growth-cone traction. Encapsulation or surface loading of neurotrophic factors (BDNF, GDNF, NGF) enables sustained local delivery that counters the short half-life of free proteins (19). Immunomodulatory design—using chondroitinase ABC to degrade CSPGs, small-molecule anti-inflammatories, or cytokine-tuning coatings—attenuates astrocytic hypertrophy and chronic microglial activation, shifting the lesion milieu toward regeneration.

Table 1. Materials and design features for nanofiber scaffolds in SCI

Material class	Typical polymers / examples	Key advantages	Key limitations	Usual fiber diameter (nm–μm)	Approx. modulus window (kPa)	Degradation profile	Common functionalizations
Natural	Collagen (I/IV), fibrin, hyaluronic acid, silk	Intrinsic bioactivity; cell adhesion; enzymatic remodeling	Batch variability; weaker mechanics; faster/less predictable degradation	100–800	0.1–10	Weeks (often rapid without crosslinking)	Laminin fragments, RGD, growth factors
Synthetic polyesters	PLGA, PCL, PLLA	Tunable mechanics & degradation; scalable manufacturing	Hydrophobic; no innate adhesivity; acidic by-products (PLGA)	200–1500	10–300	Months (PCL slower; PLGA tunable)	RGD/IKVAV, heparin, PEGylation, factor loading
Hydrophilic networks	PEG and derivatives	Protein-resistant; controllable chemistry; good for release	Lacks bioactivity; often needs blending/coating	200–800 (as blends/coaxial)	1–50	Variable (via linker hydrolysis)	Peptides, proteoglycan mimetics, enzymes
Hybrid composites	Collagen–PLGA, fibrin–PCL, HA–PCL	Bioactivity + mechanical control; improved integration	More complex fabrication/QC	150–1000	1–150	Tunable (by blend ratios/crosslinking)	Dual ligands; multi-cargo (e.g., BDNF + ChABC)

3. Mechanisms of Action in Axonal Regeneration

3.1 Contact Guidance and Circuit Re-establishment

Aligned nanofibers present anisotropic tracks that growth cones interpret as preferred paths, converting random sprouting into directed extension across lesion gaps. This contact guidance recapitulates the geometry of native fascicles, improving the likelihood of target re-innervation and functional reconnection (20).

3.2 Neurotrophic Support and Adhesive Signaling

Sustained release of neurotrophins from within the fiber matrix supports neuron survival, promotes axon

elongation, and stabilizes nascent synapses. Adhesion motifs engage integrins to trigger focal-adhesion signaling and cytoskeletal remodeling, increasing traction forces and forward advance (21).

3.3 Immune and Glial Modulation

Scaffold chemistry and release profiles can temper astrocytic scarring and microglial activation while preserving beneficial functions such as debris clearance. By reducing CSPG burden and inflammatory cytokines, the scaffold lowers biochemical barriers and extends the temporal window for axonal growth (22).

3.4 Endogenous Recruitment and Local Therapy

Gradients of chemotactic factors encourage endogenous neural stem and progenitor cell migration into the construct, where a supportive niche promotes differentiation and integration. As localized drug-delivery depots, nanofibers enable multi-agent, time-staggered therapy—such as concurrent CSPG degradation and neurotrophin support—while minimizing systemic exposure (23).

4. Preclinical Progress and Translational Barriers

4.1 Evidence for Efficacy in Animal Models

Across rodent and large-animal SCI models, electrospun PLGA, PCL, and collagen scaffolds—often functionalized with neurotrophic factors or integrin-binding peptides—consistently increase axonal crossing, reduce glial scarring, and improve locomotor and sensory outcomes. Histology demonstrates dense axonal ingrowth, vascular infiltration, and host–implant continuity, supporting the premise that architectural anisotropy coupled with biochemical programming yields additive benefits (20, 24, 25).

4.2 Key Challenges on the Path to the Clinic

Translation hinges on solving interdependent problems. Mechanical and biological integration must be seamless to prevent micromotion, fibrotic encapsulation, or channel collapse. Degradation kinetics require narrow tuning—premature loss sacrifices guidance; prolonged persistence risks chronic irritation—while by-products should be non-acidic and easily cleared. Neuroimmune compatibility remains a long-term concern in the CNS, demanding materials and surfaces that avoid sustained microglial activation. Manufacturability and scale pose practical hurdles: human-scale, anatomically conformal constructs with tight lot-to-lot reproducibility and sterility must be produced under quality systems (26). Finally, clinical heterogeneity—variation in lesion size, location, chronicity, and comorbidities—argues for modular, adaptable designs and precise, minimally traumatic surgical delivery.

Discussion

The accumulated evidence indicates that nanofiber scaffolds can reproduce essential ECM features—anisotropy, compliant mechanics, and ligand presentation—to convert an inhibitory niche into one that supports axonal extension and circuit repair (27, 28). Yet three cross-cutting issues temper interpretation of preclinical gains and define the agenda for translation. First, heterogeneity in species, injury paradigms, lesion chronicity, scaffold geometry, fiber metrics, and biochemical cargo complicates cross-study comparisons and effect-size estimates(29, 30). Harmonized reporting is needed, including explicit orientation metrics, diameter distributions, viscoelastic windows, drug-loading efficiency, and in vivo release kinetics (31). Second, scaffold performance is tightly coupled to mechanics and degradation, which remain under-reported; matching viscoelastic properties to spinal tissue and aligning degradation with axonal growth rates should be treated as primary design criteria (32, 33). Third, immune and glial modulation must be precisely titrated: attenuating astrocytic hypertrophy and microglial activation improves axon crossing, but excessive suppression may blunt protective roles (34). Immuno-informed designs that bias toward pro-repair phenotypes while preserving phagocytic function are preferable to broad suppression (35).

From these themes, several practical rules emerge: use aligned architectures with documented orientation metrics; tune modulus and loss tangent to approximate spinal parenchyma and disclose full stress–relaxation profiles; employ modular biochemical programming—adhesion motifs plus one primary trophic cue, and when indicated a targeted anti-inhibitory agent—delivered with validated local kinetics; predefine a degradation window and verify by imaging and gravimetry in vivo; and incorporate quantitative histology beyond axon counts to link mechanism to outcome (36, 37). Translationally, manufacturability and clinical usability will determine viability as much as biology (38, 39). GMP-amenable workflows, surgeon-friendly geometries, and non-invasive imaging surrogates for longitudinal monitoring will accelerate credible clinical evaluation. Head-to-head comparisons within a single model and chronic-phase implantation studies should be prioritized, and combinations with neuromodulation or targeted rehabilitation may reveal synergistic circuit re-engagement (40).

Conclusion

Nanofiber scaffolds that recapitulate the structural and biochemical complexity of the brain's ECM represent one of the most promising frontiers in SCI repair. By recreating anisotropic architecture and

molecular signaling, these constructs provide directional guidance for regenerating axons, support neuronal survival, and enable localized, sustained delivery of therapeutic agents. Preclinical studies across multiple animal models consistently demonstrate enhanced axonal bridging, reduced glial scarring, and measurable improvements in function, underscoring translational potential. Clinical realization, however, remains contingent on solving critical engineering and biological challenges: seamless host integration; precise control of mechanical compliance and degradation kinetics; mitigation of long-term immune responses; and scalable, reproducible manufacturing under clinical quality constraints. Emerging directions—including smart, stimulus-responsive scaffolds; combination therapies with stem cells, targeted gene delivery, or electrical stimulation; and non-invasive imaging/biosensing for real-time monitoring—point to increasingly adaptive and multifunctional systems. With sustained interdisciplinary collaboration and clinically mindful engineering, these biomimetic platforms are poised to evolve from experimental tools into transformative therapies capable of restoring meaningful function and independence for individuals living with SCI.

Abbreviations

SCI, spinal cord injury; CNS, central nervous system; ECM, extracellular matrix; CSPG, chondroitin sulfate proteoglycan; HA, hyaluronic acid; PLGA, poly(lactic-co-glycolic acid); PCL, polycaprolactone; PEG, poly(ethylene glycol); BDNF, brain-derived neurotrophic factor; GDNF, glial cell line–derived neurotrophic factor; NGF, nerve growth factor; ChABC, chondroitinase ABC; GMP, good manufacturing practice; MRI, magnetic resonance imaging; BBB score, Basso–Beattie–Bresnahan locomotor rating.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data and Materials Availability

No datasets were generated or analyzed for this review. All data cited are available in the referenced publications.

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