Symposium

Glial Regulation of Motor Circuit Formation, Function, and Regeneration

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From simple planaria to humans, proper assembly and maintenance of motor circuits is key to organismal survival. Each year, ∼250,000–500,000 individuals suffer a spinal cord injury with little hope of successful recovery. Moreover, motor circuits are a primary target of prevalent neurodegenerative conditions like ALS and Parkinson's disease. Thus, understanding the mechanisms that build, maintain, and rebuild motor circuits is of substantial clinical interest. Recent data indicate that glia, a heterogeneous group of non-neuronal cells within the nervous system, directly communicate with neurons to tune motor circuit dynamics. We now know glia are often active drivers of neural development and pathology, rather than passive bystanders. This mini-review will highlight recent advances in our understanding of how diverse glial cell types promote proper construction, function, and deconstruction of the key circuits that drive motor behavior.

Introduction

The execution of coordinated movement depends on precise transmission of information throughout distributed motor circuits spanning upper motor neurons in the motor cortex, lower motor neurons in the spinal cord, peripheral nerves, and neuromuscular junction (NMJ). Beyond locomotor command, motor innervation of peripheral organs such as the diaphragm also regulates controlled breathing; thus, motor circuit function is essential for life. While motor circuits are built from a complex array of neurons, motor neurons execute the final commands that drive muscle contraction. Motor neurons occupy the unique position of having a "foot" in both worlds: soma and dendrites in the central nervous system (CNS), axon, and thus outputs, in the peripheral nervous system (PNS). For this reason, they contact a greater diversity of glial (non-neuronal) cells than maybe any other type of neuron, including oligodendrocytes, microglia, astrocytes, myelinating Schwann cells, and terminal Schwann cells (Fig. 1). In this short review, we discuss established and emerging roles of glia in the establishment, maintenance, and regeneration of motor circuits across evolutionary scales.

Glia Use Multiple "Senses" to Build Functional Motor Circuits

Across nervous systems, neuronal circuit assembly is a multistep sequence beginning with neurogenesis and initial cell migration, elaboration and pathfinding of neurites and axons, structural/functional maturation of synapses, and ultimately activity-dependent refinement of developing circuitry. Neurons are born prior to most glial cell types (see more on microglia below) and form the initial environment into which glia develop (Lewis and Eisen, 2003; Jessen and Mirsky, 2005; Deneen et al., 2006; Mosti et al., 2025); thus, glia are built to sense, respond to, and feedback on neuronal circuitry. Indeed, the activity-dependent nature of many neuron–glia relationships in developing motor circuits reflects this temporal delay in gliogenesis, and neuronal activity during motor learning drives significant changes in glial transcription even in adult circuits (Sun et al., 2025). While glial cells play roles at most stages of motor circuit development, in this chapter, we discuss the role of diverse glial types in two activity-dependent phases of circuit development—circuit refinement versus circuit stabilization. Finally, we discuss the emergence of mechanotransduction as a new glial "sense" that modulates motor circuit maturation.

Glia in motor circuit refinement

A key feature of developing circuits is excess. Neurons, neurites, and synapses are first generated in excess, followed by periods of activity-dependent pruning of unnecessary circuit components. As pruning can be considered an umbrella term for multiple distinct cellular behaviors (Eyo and Molofsky, 2023), here we define pruning as the active or passive elimination of axonal fragments, including whole synapses, by glial cells. In the periphery, NMJ-associated terminal Schwann cells take up excess membranes following axonal shedding to facilitate pruning of supernumerary motor inputs onto a given muscle fiber (Bishop et al., 2004) and potentially, by pinching off axonal compartments to refine NMJ connections through trogocytosis (Schafer et al., 2025). At the invertebrate NMJ, glia similarly phagocytose synaptic boutons in an activity-dependent manner using the evolutionarily conserved MEGF10/Draper/Ced-6 pathway

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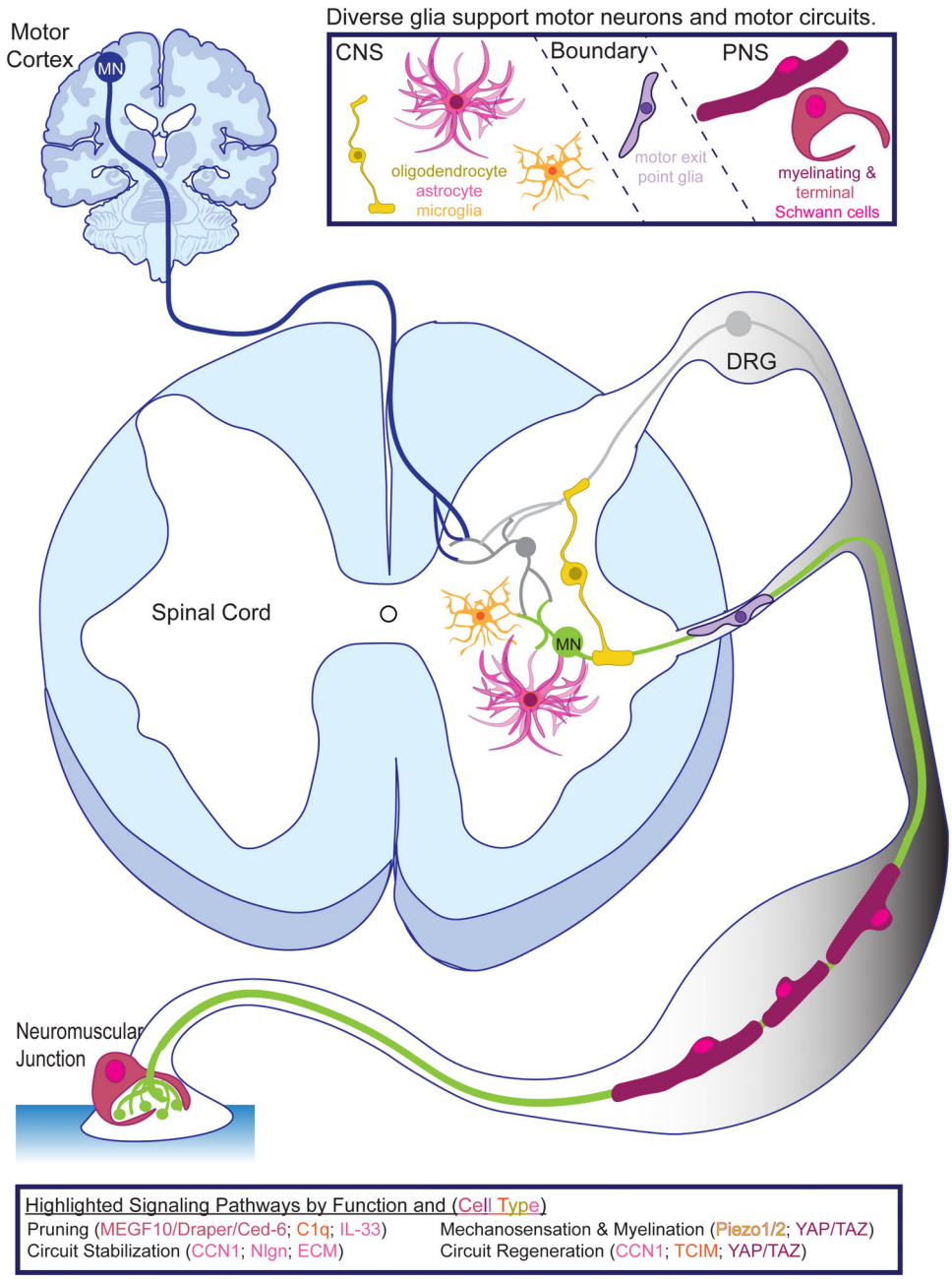


Figure 1. Motor circuits are engaged by multiple central and peripheral nervous system glial cell types. Motor-driven behavior requires precise communication between multiple distinct circuits spanning upper motor neurons (MNs, dark blue) in the motor cortex, lower MNs in the spinal cord (green), peripheral nerves, and ultimately, neuromuscular junctions (NMJs). Within the brain and spinal cord, these circuits are contacted and supported by diverse CNS glial cell types, including astrocytes, microglia, and oligodendrocyte lineage cells. As lower MN axons exit into the periphery, they engage a new set of glial cells, including motor exit point glia at the CNS-PNS boundary. Once intermingled with sensory (DRG) neurons within peripheral nerves, motor axons are supported by myelinating Schwann cells and finally terminal Schwann cells at the NMJ. Pathways highlighted in one or more phases of motor circuit assembly/reassembly are listed below and colored by target cell type(s).

(Fuentes-Medel et al., 2009). As professional phagocytes, microglia—the tissue resident immune cells of the CNS—are key mediators of circuit refinement in the brain and spinal cord. Microglia are the first glial cells to populate the developing CNS. Microglia enter both brain and spinal cord prior to and concurrent with neurogenesis (reviewed in Kolos and Korzhevskii, 2020) and are thus well-poised to regulate the initial phases of neural circuit development. Indeed, microglia can even induce programmed cell death of excess neurons and then clear neuronal corpses in the developing brain (Marín-Teva et al., 2004), and microglia-derived signals regulate mammalian motor neuron survival in vitro (Sedel et al., 2004). Moreover, microglia play prominent roles in the refinement of both somatosensory circuits (Xu et al., 2024) and motor circuits (Vainchtein et al., 2018; Vukojicic et al., 2019) in the dorsal and ventral horns of the spinal cord, respectively. In the CNS, the proprioceptive-motor neuron reflex arc is one of the earliest sensory-motor circuits to develop and is responsible for coordinating muscle stretch and contraction for effortless movement. During embryogenesis, motor dendrites are innervated by type 1a proprioceptive afferents both within (segmental) and across segments (intersegmental) of the spinal cord. In a recent preprint, Nagaraja et al. characterized a

critical period during the first 2 weeks of mouse postnatal development during which proprioceptive inputs from neighboring segments are pruned away to produce a robust, segmental reflex arc (Nagaraja et al., 2025). Consistent with a previous report (Vukojicic et al., 2019), the authors demonstrate that this pruning relies on activity-dependent upregulation of the classical complement protein C1q in microglia, and deletion of C1q blocks the developmental transition from an intersegmental to a monosynaptic, segmental reflex arc (Nagaraja et al., 2025). Conversely, excessive microglial pruning within the circuit drives sensorimotor deficits and was previously linked to spinal muscular atrophy, a devastating neurodevelopmental disorder characterized by motor neuron loss and death (Mercuri et al., 2022). Thus, microglial pruning within motor circuits must be carefully titrated to produce circuits with high specificity that remain stable.

Alongside microglia, perisynaptic astrocytes and myelinating oligodendrocytes are two prominent macroglial populations that interact with CNS motor circuits. In sensory circuits, oligodendrocyte precursor cells and astrocytes both prune synapses in an activity-dependent manner (Chung et al., 2013; Auguste et al., 2022; Xiao et al., 2022). Whether these macroglia prune motor circuits is not yet known; however, recent data indicate that astrocytes use immune signaling to nonautonomously direct microglial pruning in spinal cord development. Specifically, astrocytes release the cytokine Interleukin-33 (IL-33) to trigger IL1RL1 receptor-mediated signaling in microglia to promote pruning of both excitatory (vGlut2+) and inhibitory (VGAT+) inputs onto α-motor neurons. Accordingly, astrocyte-specific deletion of *Il33* increases inputs onto α-motor neurons at P30 in mice and disrupts sensory-motor reflex responses (Vainchtein et al., 2018). Interestingly, microglia can also prune astrocyte processes (Gu et al., 2025) and developing oligodendrocytes (Hughes and Appel, 2020); thus, deeper exploration of the molecular mechanisms that balance the activity of these professional and nonprofessional phagocytes in CNS development is warranted.

Glia in motor circuit stabilization

While activity-dependent circuit plasticity facilitates learning in higher cortical circuits (see below, Myelin as a dynamic regulator of motor computation), spinal motor circuits are largely wired for homeostasis. Following initial waves of circuit refinement, motor circuit stabilization coincides with the maturation of the CNS macroglia and Schwann cells in the periphery, and disruption of glial cell development destabilizes motor circuit function (Chernoff, 1981; Suter et al., 1992; Duff et al., 2026). Across distinct CNS circuits, recent data demonstrate that maturation of perisynaptic astrocytes represses developmental plasticity. This was first demonstrated in 1989 by the classic Müller and Best study whereby implantation of immature astrocytes into the mature cat visual cortex was sufficient to reopen ocular dominance plasticity (Müller and Best, 1989). Ocular dominance plasticity refers to the experience-dependent refinement of thalamocortical connections to balance connections downstream of the left versus right eye during post-natal development (Min, 2025). A role for astrocytes in closure of the ocular dominance critical period has been validated in mouse by multiple independent groups and is dependent on astrocyte-regulated maturation of inhibitory circuits (Ribot et al., 2021; Gegenhuber et al., 2026), and astrocyte-derived CCN1 (cellular communication network factor 1) was recently shown to sustain circuit stability in the adult visual cortex (Sancho et al., 2026). While understudied in spinal circuits, astrocytic repression of motor plasticity serves to stabilize locomotor function in the developing *Drosophila* nerve cord. A 2021 study by Ackerman et al. identified a critical period of activity-dependent remodeling of fly motor dendrites that peaks in late embryogenesis and is closed by the end of the first larval stage. Loss of motor plasticity coincided with astrocyte maturation, and genetic depletion of astrocytes was sufficient to extend motor plasticity and disrupt mature locomotor behavior. Similar to findings in visual cortex, astrocyte regulation of inhibition, extracellular matrix composition, and synaptic signaling via neuroligins were each found to contribute to critical period closure (Ackerman et al., 2021). Thus, astrocytes can promote circuit remodeling by modulation of microglial pruning (see above) and stabilize motor circuits once remodeling is complete. What triggers this developmental switch in astrocytes from promoting to repressing plasticity remains to be defined.

A key stabilizing force in the developing nervous system is myelin. Myelin—a multilamellar, lipid-rich sheath generated by neural stem cell-derived oligodendrocytes in the CNS (Simons et al., 2024) and neural crest-derived Schwann cells in the periphery (Salzer et al., 2024)—is essential for rapid action potential propagation in jawed vertebrates. Myelin is an integral and active component of neural circuits that helps guide the flow of information throughout the nervous system. Mutations in myelin-associated proteins such as myelin basic protein (e.g., shiverer mice) therefore result in severe dysmyelination of the CNS, produce pronounced behavioral deficits including seizure and limb weakness, and are ultimately lethal at juvenile stages (Chernoff, 1981). Similarly, mutants lacking peripheral myelin exhibit progressive hindlimb weakness and early lethality (de Waegh and Brady, 1990; Suh et al., 1997; Park et al., 2022). While Schwann cell development and myelination are tightly regulated by concentrations of axonal surface cues including neuregulin I type III (Taveggia et al., 2005), oligodendrocyte myelination is heavily influenced by neuronal activity—a concept called myelin plasticity or activity-dependent myelination (Simons et al., 2024). Accordingly, oligodendrocyte myelination exhibits greater variability within and across CNS regions (Domínguez-Iturza et al., 2026) and even across a single axon (Tomassy et al., 2014). Within the spinal cord, this has been best-studied in zebrafish due to their amenability to longitudinal live imaging. Here, reports across multiple groups demonstrate that synaptic vesicle fusion acts as a feedforward loop to stabilize nascent myelin sheaths (Hines et al., 2015; Almeida et al., 2021). Blocking synaptic release through neuron-specific expression of tetanus toxin reduces myelination of some, but not all, axonal types (Hines et al., 2015; Mensch et al., 2015; Koudelka et al., 2016), congruent with recent reports of functional OPC heterogeneity in the zebrafish spinal cord (Marisca et al., 2020). Interestingly, though mechanistically similar (Simons et al., 2024), activity-dependent myelination and adaptive (experience-dependent) myelination show distinct temporal differences. As the spinal cord is densely myelinated, activity-dependent myelination of spinal cord circuits is largely restricted to development. Changes in motor experience minimally alter spinal cord OPC dynamics and myelination in adulthood but drive robust changes to premotor and motor cortex to facilitate motor learning (Gibson et al., 2014; Bacmeister et al., 2022; discussed more below).

Mechanosensation Drives CNS and PNS Myelination

With the discovery of activity-dependent myelination in the mid-2010s, myelin plasticity became and has remained a key focus of the glial community for the last decade. Recent studies suggest that oligodendrocytes and Schwann cells use more than one "sense" to decode how much myelin a given axonal segment

requires. Piezos were among the first characterized mechanically active channels for which Dr. Ardem Patapoutian received the Nobel Prize in Physiology or Medicine in 2021. With this discovery, researchers across many fields began to test the role of Piezos in different cellular contexts, including the glial community (Segel et al., 2019; Acheta et al., 2022; Brandt and Smith, 2023; Coombs et al., 2026; Dyckow-Schubart et al., 2026). While many more mechanically active channels have since been discovered, a recent report from Acheta et al. determined that Schwann cells are enriched for PIEZO1 and PIEZO2. The authors demonstrated that Schwann cell-specific knock-out of *Piezo1* in mouse increases myelin length and thickness. Using pharmacological approaches, the authors determined that Piezo1 likely acts through TAZ, a mechanotransducer that relays changes in niche stiffness to drive Schwann cell proliferation and myelination. Interestingly, cell-specific knock-out of *Piezo2* causes hypomyelination, yet loss of both *Piezo1* and *Piezo2* phenocopies the Piezo1-dependent hypermyelination phenotype without influencing TAZ (Acheta et al., 2022). These data suggest that the coincident activities of Piezo1 and Piezo2 coordinate rates of Schwann cell myelination.

Like Schwann cells, oligodendrocyte myelination requires significant changes to the underlying cytoskeleton (Zuchero et al., 2015; Simons et al., 2024) that are likely to trigger mechanically active channels. Indeed, oligodendrocytes also express both *Piezo1* and *Piezo2* (Zhang et al., 2014; Marques et al., 2016), and recent reports implicate both Piezos in CNS myelination (Young et al., 2025; Coombs et al., 2026). Using a multimodel approach, a recent preprint (Coombs et al., 2026) demonstrated that rodent OPCs exhibit mechanically sensitive currents and that *Piezo1* knock-out OPCs cultured with inert nanofibers exhibit reduced myelin sheath length without reducing sheath number; OPC proliferation and capacity to differentiate were unchanged. Leveraging oligodendrocyte-specific CRISPR/Cas13d knockdown approaches in zebrafish, the authors went on to test how disruption of *piezo1*, *piezo2*, or the two receptors combined would impact oligodendrocyte myelination during spinal cord development in vivo. The authors determined that whereas knockdown of *piezo1* reduces the total number of myelin sheaths an individual oligodendrocyte makes without changing summed myelin sheath length (e.g., myelin output), disruption of *piezo2* reduces sheath formation and myelin output over time. Accordingly, knockdown of both receptors exacerbated changes to myelin sheath number, length, and total output and led to greater myelin instability (Coombs et al., 2026). Thus, Piezos function in both Schwann cells and oligodendrocytes to regulate myelin output (Acheta et al., 2022; Coombs et al., 2026). Finally, while Coombs et al. did not identify any changes in OPC proliferation in vitro or in vivo in the spinal cord following knockdown of *piezo1* or *piezo2* (Coombs et al., 2026), other studies outside of the motor circuit identified roles for Piezos in OPC-sensing of niche stiffness and in turn, proliferation (Segel et al., 2019). Given that spinal circuits exhibit greater developmental restrictions on multiple forms of cellular plasticity, a careful examination of the role of Piezos in OPCs across distinct circuits may uncover context-specific roles of mechanosensation in oligodendrocyte biology.

Glia Maintain Motor Circuit Performance

While neurons establish the structural framework of motor networks, glial cells are increasingly recognized as essential regulators of circuit performance. Rather than functioning solely as support cells, glia continuously maintain the physiological environment required for neuronal communication, preserve axonal integrity, regulate synaptic activity, and dynamically shape the timing of information transfer. Together, these complementary functions ensure that motor circuits remain stable enough to support reliable movement while retaining the plasticity necessary for motor learning and behavioral adaptation.

Glial regulation of motor circuit homeostasis

Motor circuit function depends on maintaining tightly regulated extracellular conditions that allow neurons to generate and propagate action potentials with high temporal precision. Astrocytes play a central role in this process by buffering extracellular potassium, rapidly clearing glutamate from synapses, providing metabolic support to active neurons, and coupling neuronal activity to local blood flow (Allen and Eroglu, 2017; Barros et al., 2024). These homeostatic functions prevent excessive neuronal excitation while ensuring that motor networks remain responsive during sustained activity. Beyond maintaining ionic balance, astrocytes actively participate in circuit plasticity. Changes in astrocytic calcium signaling accompany motor learning and influence synaptic transmission within the motor cortex (Padmashri and Dunaevsky, 2025), indicating that astrocytes contribute directly to activity-dependent refinement of motor circuits rather than simply maintaining neuronal metabolism. Consistent with this broader role, studies in *Drosophila* demonstrated that astrocytes actively terminate a developmental critical period for motor circuit plasticity through neuroligin–neurexin signaling with motor neurons, stabilizing dendritic architecture and ensuring proper locomotor behavior (Ackerman et al., 2021; see above, Glia in motor circuit stabilization). Together, these findings suggest that astrocytes regulate motor circuits across lifespan, from establishing appropriate developmental windows of plasticity to supporting adaptive remodeling in the mature nervous system.

Recent single-cell transcriptomic analyses reveal substantial regional specialization among astrocytes, suggesting that distinct astrocyte populations are adapted to support the physiological demands of different neural circuits, including motor circuits, although the functional significance of this heterogeneity is only beginning to be understood (Batiuk et al., 2020; Bayraktar et al., 2020).

Microglia similarly contribute to the maintenance of healthy motor circuits through continuous surveillance of the neural environment. Far from serving exclusively as immune cells, microglia dynamically interact with neurons to regulate synaptic remodeling, remove dysfunctional cellular components, and preserve network stability throughout adulthood (Wake et al., 2009). In vivo imaging studies demonstrate that neuronal activity rapidly alters microglial process dynamics, while disruption of microglial signaling impairs synaptic plasticity and motor learning (Badimon et al., 2020; Silva et al., 2021). Parkhurst et al. demonstrated that genetic ablation of microglia in the mouse brain impaired performance across several learning paradigms and markedly reduced motor learning-dependent synaptogenesis (Parkhurst et al., 2013). They further identified microglia-derived brain-derived neurotrophic factor (BDNF) as an important mediator of learning-associated synapse formation. Together, astrocytes and microglia maintain the cellular environment required for stable neuronal communication, allowing motor circuits to remain both robust and adaptable during changing behavioral demands.

Homeostasis must also be preserved along the entire length of motor axons. In the peripheral nervous system, Schwann cells maintain nodal organization, provide metabolic support, and

preserve axonal integrity throughout adulthood (reviewed in Jessen and Mirsky, 2022). At the CNS-PNS transition zone, boundary cells such as motor exit point (MEP) glia contribute to this continuous glial network by maintaining proximal motor roots and supporting uninterrupted communication between central and peripheral motor pathways (Fontenas and Kucenas, 2018). Collectively, these glial populations ensure that motor commands generated within the CNS are transmitted efficiently to peripheral targets.

Myelin as a dynamic regulator of motor computation

Although myelin has traditionally been viewed as a structural specialization that increases conduction velocity, accumulating evidence indicates that it actively regulates neural circuit function (Kato et al., 2020). Coordinated movement depends on the synchronous activation of spatially distributed neuronal populations, making the precise timing of action potential arrival as important as transmission speed itself. Myelin influences these temporal relationships by regulating conduction velocity, spike timing, and synchronization across motor pathways, thereby shaping how information is processed within motor circuits (reviewed in Gagnon et al., 2026).

Seminal work from the Richardson laboratory established myelin plasticity as an important component of motor learning. McKenzie et al. demonstrated that the generation of new oligodendrocytes is required for acquiring a novel motor skill, while Xiao et al. showed that motor learning rapidly promotes oligodendrocyte differentiation and subsequent myelin remodeling, highlighting both rapid and longer-term adaptations of the oligodendrocyte lineage during learning (McKenzie et al., 2014; Xiao et al., 2016). This concept has been strengthened by studies demonstrating that myelin remains remarkably plastic throughout adulthood. Motor learning stimulates the differentiation of new oligodendrocytes while promoting remodeling of existing myelin sheaths within the motor cortex (Bacmeister et al., 2020, 2022). Rather than uniformly increasing conduction velocity, adaptive myelination appears to fine-tune conduction delays across distributed networks, improving synchrony between converging neuronal pathways and optimizing motor performance. These findings establish myelin as an additional substrate for neural plasticity that complements activity-dependent changes in synaptic connectivity during motor learning.

Direct evidence that myelin shapes motor circuit computation has recently emerged from longitudinal imaging, electrophysiology, and computational modeling. In a recent preprint, Gagnon and colleagues demonstrated that experimental demyelination disrupts cortical network dynamics despite relatively preserved neuronal architecture. Loss of myelin alters neuronal synchrony, changes cell type-specific firing patterns, and impairs the smooth execution of skilled reaching. Computational analyses further identified action potential propagation failures within inhibitory axons as a principal mechanism underlying these circuit abnormalities. Although remyelination restored many large-scale features of cortical activity, persistent deficits in movement smoothness suggested that precise myelin organization is required for complete functional recovery. These findings suggest that myelin is not merely a passive insulator but an active regulator of motor computation whose organization directly influences network dynamics and behavior (Gagnon et al., 2025).

Together, these studies establish glia as integral components of healthy motor circuits. Astrocytes maintain the metabolic and ionic environment necessary for neuronal signaling, microglia preserve synaptic organization and network stability, Schwann cells and MEP glia support reliable transmission along peripheral motor pathways, and oligodendrocytes dynamically regulate the timing of information flow through adaptive myelination. These complementary functions maintain the physiological and computational properties required for coordinated movement. However, these homeostatic roles are not fixed. Following injury, the same glial populations undergo extensive molecular and functional reprograming, transitioning from maintaining established motor circuits to actively coordinating tissue repair and circuit reconstruction. This plasticity forms the basis of the regenerative responses discussed in the following section.

Glia Orchestrate Motor Circuit Repair Following Spinal Cord and Peripheral Nerve Injury

Injury to the nervous system disrupts motor circuits through the loss of neurons, axons, synapses, and myelin. As discussed in the previous section, glia continuously maintain the physiological environment, connectivity, and timing required for normal motor function. Following injury, however, these same cells undergo extensive molecular and functional reprograming that transforms them from regulators of circuit homeostasis into active coordinators of tissue repair. Rather than acting independently, astrocytes, microglia, oligodendrocyte lineage cells, Schwann cells, and MEP glia engage highly-coordinated responses that regulate inflammation, remodel extracellular matrix, guide regenerating axons, and restore myelinated pathways. Consequently, successful functional recovery depends not only on neuronal regeneration but also on the ability of glial populations to collectively reconstruct the cellular environment that supports motor circuit function.

Remodeling the injury environment

One of the earliest consequences of spinal cord injury is the rapid activation of astrocytes and microglia, which together establish the cellular environment that determines subsequent repair. Astrocytes rapidly proliferate around the lesion and undergo extensive transcriptional remodeling. Historically, this reactive response was viewed primarily as detrimental because astrocytes produce chondroitin sulfate proteoglycans and other extracellular matrix molecules that inhibit axon growth (Silver and Miller, 2004). However, genetic ablation studies demonstrated that preventing astrocytic scar formation results in greater tissue degeneration, impairs blood-spinal cord barrier integrity, and reduces locomotor recovery, fundamentally changing the interpretation of reactive gliosis (Anderson et al., 2016). Rather than representing a uniformly inhibitory response, reactive astrocytes stabilize damaged tissue while simultaneously creating an environment that both protects surviving neural circuits and constrains inappropriate axon growth.

Recent transcriptomic analyses have further demonstrated that astrocyte reactivity encompasses multiple molecularly distinct states whose functions evolve throughout the repair process, rather than representing a single pathological phenotype (Escartin et al., 2021). Extending this concept, McCallum and colleagues identified a population of lesion-remote astrocytes that undergo molecular reprograming several millimeters from the injury site (McCallum et al., 2026). Surprisingly, these distant astrocytes regulate microglial activation, influence oligodendrocyte lineage responses, and promote white matter repair despite remaining outside the lesion core. Mechanistically, this reparative program is driven in part by astrocyte-derived CCN1, which activates syndecan 4-dependent signaling in microglia to promote a transcriptional state specialized for myelin debris clearance, lipid handling, and tissue remodeling. The resulting microenvironment facilitates oligodendrocyte

progenitor differentiation and remyelination, demonstrating that lesion-remote astrocytes actively orchestrate regenerative processes through intercellular communication rather than simply responding to local damage. These findings redefine spinal cord injury as a coordinated glial response that extends along intact motor pathways, rather than a process confined to the immediate lesion border.

Microglia are equally important in coordinating the early regenerative environment. Following injury, they rapidly clear cellular debris, secrete inflammatory mediators, and regulate communication with astrocytes and infiltrating immune cells. Although prolonged inflammation contributes to secondary degeneration, transient microglial activation is essential for successful repair. Bellver-Landete et al. demonstrated that depletion of microglia disrupts astrocytic scar organization, delays wound closure, and significantly impairs locomotor recovery after spinal cord injury (Bellver-Landete et al., 2019). Li et al. showed that neonatal microglia transiently adopt a pro-regenerative state that promotes scar-free wound healing and axon regrowth after spinal cord injury, in contrast to adult microglia, which fail to mount this reparative response (Li et al., 2020).

Recent single-cell transcriptomic studies have identified multiple injury-associated microglial states that coordinate communication with astrocytes, infiltrating macrophages, and other resident glia through dynamic ligand-receptor signaling networks during spinal cord repair (Brennan et al., 2022). Parallel single-cell analyses further revealed injury-induced heterogeneity within astrocyte and oligodendrocyte progenitor populations, demonstrating coordinated multicellular remodeling of the injured spinal cord (Floriddia et al., 2020; Wei et al., 2023).

Rebuilding motor pathways

While mammalian spinal cord regeneration remains limited, studies in zebrafish demonstrate that glial plasticity can actively support reconstruction of functional motor circuits. Following complete spinal cord transection, adult zebrafish regenerate descending axons and recover coordinated swimming within weeks. Live imaging and lineage-tracing studies revealed that spinal cord progenitors undergo localized epithelial-to-mesenchymal transition following injury, generating regenerative glial populations that remodel the lesion environment while preserving spinal cord architecture (Klatt Shaw et al., 2021). Rather than producing a chronic inhibitory scar, these glia establish a permissive substrate that supports axonal regrowth.

Subsequent work identified a transient population of *ctgfa*-expressing progenitor-derived glia that bridge the lesion gap and are required for successful axon regeneration and locomotor recovery (Zhou et al., 2023). More recently, single-cell transcriptomic analyses resolved multiple sequential regenerative glial states that coordinate extracellular matrix remodeling, immune regulation, oligodendrogenesis, and neuronal regeneration throughout spinal cord repair (Saraswathy et al., 2024). Complementing these findings, Klatt Shaw and colleagues demonstrated that successful regeneration also depends on a transient population of highly phagocytic microglia and macrophages that rapidly clear lipid-rich debris following injury. They identified the transcriptional regulator *tcim* as a key driver of this pro-regenerative immune state, showing that elevated phagocytic activity promotes efficient debris clearance, limits chronic inflammation, and is required for functional spinal cord repair (Klatt Shaw et al., 2026). Remarkably, expression of human TCIM was sufficient to enhance phagocytic capacity in mammalian macrophages, suggesting that regenerative immune programs are evolutionarily conserved despite the limited regenerative capacity of the mammalian spinal cord. Collectively, these studies demonstrate that successful regeneration depends not on a single regenerative glial population but on coordinated transitions among multiple glial and immune cell states that evolve throughout the repair process.

Reconstructing peripheral motor pathways

Peripheral motor nerves possess a much greater regenerative capacity than the mammalian spinal cord, owing largely to the remarkable plasticity of Schwann cells. Following injury, Schwann cells dedifferentiate and rapidly convert into specialized repair Schwann cells through a transcriptional program centered on c-Jun activation (Arthur-Farraj et al., 2012). These repair Schwann cells downregulate myelin-associated genes while upregulating trophic factors, extracellular matrix molecules, cytokines, and guidance cues that promote axon regeneration, recruit macrophages, and facilitate myelin debris clearance (reviewed in Jessen and Arthur-Farraj, 2019). Building upon this regenerative framework, a recent preprint by Acheta and colleagues suggests that Schwann cell plasticity may also be amenable to therapeutic modulation. Using low-intensity ultrasound (LIU), the authors report selective activation of the Hippo pathway effector TAZ in Schwann cells, leading to increased Schwann cell proliferation and nerve growth factor (NGF) expression without activating the closely related mechanotransducer YAP (Acheta et al., 2025). These preliminary findings highlight the intriguing possibility that noninvasive mechanical stimulation could enhance intrinsic Schwann cell repair programs and represent a novel therapeutic strategy for promoting peripheral nerve regeneration. Recent single-cell studies further reveal substantial heterogeneity among repair Schwann cells, identifying distinct transcriptional programs that regulate inflammation, regeneration, and remyelination (Zhao et al., 2024). These findings emphasize the inherent plasticity of Schwann cells, showing that differentiated cells can reversibly activate a repair program that promotes axon regeneration, remodels the injury environment, and facilitates functional recovery.

Growing evidence suggests that reconstruction of motor pathways requires coordinated interactions among multiple glial populations rather than Schwann cells alone. In the PNS, perineurial glia actively contribute to nerve repair by rapidly bridging the injury gap, coordinating with Schwann cells and immune cells, and providing a structural scaffold that guides regenerating axons back toward their distal targets (Lewis and Kucenas, 2014).

At the CNS-PNS transition zone, MEP glia establish a specialized myelinating interface linking oligodendrocytes within the spinal cord to Schwann cells along peripheral motor nerves (Fontenas and Kucenas, 2021). This specialized glial territory highlights the close functional integration of central and peripheral myelinating systems and raises the possibility that these boundaries become more permissive during regeneration. Consistent with this idea, loss of peripheral myelinating glia in zebrafish triggers the recruitment of spinal cord-derived OPCs onto peripheral motor nerves (Fontenas and Kucenas, 2018, 2021). Ongoing work from the Fontenas laboratory further suggests that these ectopic OPCs differentiate into myelinating oligodendrocytes capable of generating compact, functional myelin sheaths around peripheral motor axons (Fontenas laboratory, unpublished observations). Together, these findings indicate that the nervous system can recruit alternative myelinating glia when Schwann cells are absent, revealing an unexpected capacity for cross-compartment glial compensation during motor nerve repair. Collectively, these

studies suggest that successful reconstruction of functional motor pathways requires not only axonal regeneration and remyelination but also restoration of the specialized glial architecture that integrates the CNS and PNS.

Conclusion and Future Perspectives

Together, recent work demonstrates that the same glial populations responsible for maintaining healthy motor circuits are redeployed following injury to coordinate tissue repair and functional recovery. Rather than representing entirely new cellular identities engaging novel molecular pathways, regenerative glial states emerge through context-dependent reprograming of homeostatic mechanisms that regulate inflammation, extracellular matrix remodeling, axon guidance, and remyelination. Across the CNS and PNS, successful reconstruction of motor pathways depends on coordinated communication among multiple glial populations rather than the activity of any single cell type. An important question is whether repair also reengages developmental glial programs that initially guide motor circuit formation, and whether failure to reactivate these programs contributes to the limited regenerative capacity of the adult mammalian nervous system. Defining how developmental and homeostatic glial programs are repurposed following injury, and why they are more effectively mobilized in regenerative species such as zebrafish than in mammals, may reveal strategies to promote motor circuit repair and functional recovery. Do glia recognize particular motor neuron subtypes, axons, synapses, or muscles? We still know relatively little about the molecular mechanisms allowing glia to exert highly localized effects within a circuit. Single-cell and spatial approaches have uncovered numerous glial states, but transcriptional diversity does not necessarily establish functional relevance. An important next step will be to connect these molecularly defined glial states to their specific functions within motor circuits.

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