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Autore	NAUMANN, Rudolf
Titolo	Architektur kleinasiens von ihren anfangen bis zum ende der hethitischen zeit / Rudolf Naumann
Pubbl/distr/stampa	Tubingen, : Ernst Wasmuth, 1971
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Autore	Wilfredo Mellado
Titolo	Myelin-Mediated Inhibition of Axonal Regeneration: Past, Present, and Future
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Descrizione fisica	1 online resource (116 p.)
Collana	Frontiers Research Topics
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Sommario/riassunto	Pioneering studies conducted in the 1980's laid the foundation for the hypothesis that axonal regeneration is limited by CNS myelin, and the identification of myelin-associated glycoprotein (MAG), Nogo, and

oligodendrocyte myelin glycoprotein (OMgp) as inhibitors of neurite outgrowth firmly established myelin as a key factor in regenerative failure. Mechanistically, it has been shown that MAG, Nogo, and OMgp mediate inhibition by binding to either Nogo receptor (NgR) or paired immunoglobulin receptor B (PirB), and initiating a signaling cascade that culminates in the activation of RhoA. Since the discovery of these proteins, there has been tremendous interest in identifying compounds and molecular mechanisms that are capable of overcoming myelin-mediated inhibition. Many studies have focused on pharmacological antagonism of receptors and signaling intermediates, while others have sought to identify and enhance endogenous pro-regenerative pathways. The most notable example of the latter is the conditioning lesion effect, which led to the discovery of cyclic AMP's ability to overcome inhibition by MAG and myelin. Many of the agents tested in these studies have been shown to promote axonal regeneration in vivo, and this research topic allows researchers to share information about new treatments that have been developed in both academia and industry. As we look toward the future, it is becoming increasingly clear that reversal of myelin-mediated inhibition alone will not be sufficient to produce functional recovery from spinal cord injury, and that other factors, such as astroglial scarring, the expression of chondroitin sulfate proteoglycans, neuronal cell death, and lack of neurotrophic support, must also be taken into consideration. Combinatorial approaches therefore hold a great deal of promise, and we hope to initiate a dialogue on how stem cell transplantation, chondroitinase ABC, gene therapy, growth-promoting agents, and other methods can be combined to optimize functional recovery. We introduce this topic in honor of the life and work of Dr. Marie T. Filbin (1955-2014). Through these articles, we highlight past achievements in the field, novel findings, unanswered questions and innovative ideas that we hope will lead to new advances in axonal regeneration.