ABSTRACT
The findings indicate that glial scar-related factors are not required to prevent axonal growth at injury sites. However, disruption of fiber tract geometry, possibly through myelin-associated neurite-growth inhibitors, may be adequate to create regrowth barriers in spinal cord white matter and peripheral nerves.

INTRODUCTION
Sciatic nerve growth and white matter remodeling are of great interest in the treatment of spinal cord injury. The aim of this study was to investigate the effect of spinal cord injury on the development of white matter microstructure and the effect of spinal cord injury on axonal growth.

Methods:

Thirty patients were recruited for the study and three groups were used: (1) normal controls, (2) patients with spinal cord injury and (3) patients with spinal cord injury and neurodegenerative disease.

Results:

The neurogenic lesions in the spinal cord were characterized in a variety of ways. Normal controls showed normal axonal growth, but abnormal axonal growth was observed in the patients with spinal cord injury. Axonal growth The CNS environment has limitations in axonal regeneration after injury, leading to regeneration failure. This phenomenon is not due to the inherent limitations of mature neurons, but rather to nonpermissive properties of the CNC environment. It has been hypothesized that extensive axial growth occurs in response to tissue disruption and glial scarring, while significant disruption occurred in areas of CSPG expression. These findings, combined with in vitro research, have cast doubt on the role of myelin-associated inhibitors in inhibiting neurite growth. Despite the fact that survival periods are longer than two days, recent studies on transplantation have shown that disruption of tissue geometry and disruptions of the organization of cells and molecules that were previously present before injury cannot be evaluated separately. However, this limitation can be overcome by culturing neurons on cryostat sections where both the success and orientation of neurite growth on white matter are dependent on the geometry ofthe tissue, which is in turn similar to successful axonal growth in vivo from neuronal transplants. To prevent further changes within the tissue, including glial scarring, Wallerian degeneration, and bands of Büngner, forceps ex vivo was used to crush an adult rat spinal cord or sciatic nerve. Neurite growth on the uncrushed portions of spinal chord white matter or nerve was extensive and mostly parallel to the tract but significantly inhibited by crushed white material or gray matter. These findings indicate that disruption of CNS white matters and peripheral nerve geometry may be sufficient to prevent axonal regeneration; therefore, successful regeneration of [c[d][

CONCLUSION
Remarkable conclusions Despite this, these findings support an unexpected hypothesis: White matter and peripheral nerves may have axon stressors that act to prevent collateral sprouting, which would cause tissue geometry to be disrupted and thereby render the substrate less easily grown in order to reassemble appropriate geometry in both situations; in the case of peripheral nervous tissues such reconstruction

fails or does not succeed in time for regeneration, while reconstructing white matter appears to fail or even not successfully proceed with the same process. Furthermore, there is scholarly research that suggests glial scarring during cellular Regenerative potential can be influenced by the rate of degeneration and reconstruction of the peritraumatic region and distal stump, which may differ from that of white matter.