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EDITOR'S PAGE

Neuroprotection in spinal cord injury

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Dear *Spinal Cord* reader,

The review by Onose *et al*. in this issue deals with neuroprotection. The authors state that recent studies, especially in the genetic, immune, histochemical and bio (nano)-technological fields, have provided new insight into the cellular and molecular mechanisms occurring within Central Nervous System (CNS)—including Spinal cord injury (SCI). In their review, Onose *et al*. describe a newly emerging spectrum of therapies aiming to antagonize the 'secondary injury' pathways (i.e. to provide neuroprotection) and also to repair such classically irreparable structures. The authors also note that there have been identified many molecules, primarily expressed by heterogenous glial and neural subpopulations of cells, that are critical either directly or indirectly for tissue sparing, angiogenesis, neural plasticity and respectively, also, various substances/energy vectors with regenerative properties: MAG, OMgp, KDI, Nogo, NgR, the Rho signaling pathway, EphA4, GFAP, different subtypes of serotonergic and glutamatergic receptors, antigens, antibodies, immune modulators, adhesion molecules, scavengers, neurotrophic factors, enzymes, hormones, collagen scar inhibitors, remyelinating agents and neurogenetic/plasticity inducers, all aiming to preserve or re-establish the morphology and functional connections across the lesion site.

Accordingly, modern research and experimental SCI therapies focus on several intricate, rather overlapping therapeutic objectives and means including neuroprotective, neurotrophic, neuro-restorative, neuroreparative, neuroregenerative, neuro(re)constructive and neurogenetic interventions. The first three of these therapeutical directions are generically assimilated as 'neuroprotective' and are synthetically presented and commented in the paper, in an attempt to conceptually systematize them; thus, the goal of this review is, by emphasizing the state-of-the art in the domain, to optimise theoretical support in selecting the most effective pharmacological and physical interventions for preventing, as much as possible, paralysis and for maximizing recovery chances after SCI.

In the same field is the original work by Torres *et al*. The group probed the effect of metabolic inhibition after an acute traumatic Spinal cord injury (TSCI) using a standardized contusion model (NYU impactor) in order to ascertain if the metabolic inhibition is a 'secondary mechanism of injury' or a mechanism of protection. The results show that the partial and transitory inhibition of the aerobic metabolism after an acute TSCI could be a self-protection mechanism instead of being a 'secondary mechanism of injury'.

Cristante *et al*. evaluated in a prospective, non-randomized clinical series, the effect of autogenous undifferentiated stem cell infusion for the treatment of patients with chronic Spinal cord injury (SCI) on somatosensory evoked potentials (SSEPs).

Fouad *et al*. applied Schwann cells in Matrigel filled guidance channels, olfactory ensheathing glia, and chondroitinase ABC at the lesion site following a complete thoracic SCI in rats and studied the resulting bladder function.

Several more very interesting studies can be found in this issue. They increase our knowledge about obesity identification (Laughton *et al*.), tolerance in intrathecal baclofen therapy (Heetla *et al*.), walking ability at discharge in non-traumatic SCI individuals (Sturt *et al*.), deep venous thrombosis in acute Spinal cord injury (Mathur and Agarwal), Survivin in damaged nerve and Spinal cord (Amiri *et al*.) and the injury of an aberrant vertebral artery during a routine corpectomy (case report Eskander *et al*.).

Enjoy exploring them

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