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Session PSTR399 - Spinal Cord Injury: Cellular and Molecular Mechanisms II

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PSTR399.06 / D40 - Role of purinergic and connexin signaling in the awakening of a stem cell niche in the spinal cord

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Presenter at Poster
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Poster

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Abstract
The ependyma of the spinal cord is a latent stem cell niche that is reactivated by injury to contribute new cells to the glial scar. However, the mechanisms by which ependymal cells are reactivated by spinal cord injury (SCI) remain poorly understood. Purinergic signaling may have a role as extracellular ATP rises after SCI and EC have functional P2X7 receptors (P2X7r). In addition, we have shown that after SCI, gap junction blockade prevents injury-induced proliferation. We speculate that purinergic and connexin (Cx) signaling may be important to the reactivation of the ependymal stem cell niche.To explore the role of P2X7r we injected the selective analog BzATP nearby the central canal. We tested ependymal cell proliferation by EdU uptake and Cx26 expression by immunohistochemistry after SCI or in vivo injection of BzATP. Glial fibrillary acidic protein (GFAP) expression was monitored by using a GFAP-EGFP transgenic mouse. To address the impact of Cx26 we used a Cre-lox system in adult mice to selectively delete Cx26 from EC. We found that similar to injury, injection of BzATP induced the proliferation of ependymal cells and shifted ependymal cells to a GFAP phenotype. BzATP did not induce these changes in ependymal cells of P2X7r knock out mice. In vivo blockade of P2X7r with the potent and selective antagonist AZ10606120 reduced significantly the injury-induced proliferation of ependymal cells. Like injury, P2X7r activation led to the expression of Cx26. Remarkably, genetic deletion of Cx26 in EC prevented the effect of P2X7r activation on ependymal cell proliferation. Our results show that purinergic and Cx signaling are key pathways for the reactivation of the ependyma by injury. Cx26 seems to be downstream to P2X7r and appears as a relevant target to modulate the reaction of EC to injury to achieve better self-repair.