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PEG-PDLLA micelle treatment improves axonal function of the corpus callosum following traumatic brain injury.

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Abstract

The initial pathological changes of diffuse axonal injury following traumatic brain injury (TBI) include membrane disruption and loss of ionic homeostasis, which further lead to dysfunction of axonal conduction and axon disconnection. Resealing the axolemma is therefore a potential therapeutic strategy for the early treatment of TBI. Monomethoxy poly (ethylene glycol)-poly (D, L-lactic acid) di-block copolymer micelles (mPEG-PDLLA) have been shown to restore depressed compound action potentials (CAPs) of spinal axons and promote functional recovery after spinal cord injury. Here, we evaluate the effect of the micelles on repairing the injured cortical axons following TBI. Adult mice subjected to controlled cortical impact (CCI) were treated with intravenous injection of the micelles at 0 h or 4 h after injury. Evoked CAPs were recorded from the corpus callosum of coronal cortical slices at 2 days after injury. The CCI caused significant decreases in the amplitudes of two CAP peaks that were respectively generated by the faster myelinated axons and slower unmyelinated axons. Micelle treatment at both 0 h and 4 h after CCI resulted in significant increases in both CAP peak amplitudes. Injection of fluorescent dye-labeled micelles revealed high fluorescent staining in cortical gray and white matters underneath the impact site. Labeling membrane-perforated neurons by injecting a membrane impermeable dye Texas Red-labeled dextran into lateral ventricles at 2 h post-CCI revealed that immediate micelle injection after CCI did not reduce the number of dye-stained cortical neurons and dentate granule cells of the hippocampus, indicating its ineffectiveness in repairing plasma membrane of neuronal somata. We conclude that intravenous administration of mPEG-PDLLA micelles immediately or at 4 h after TBI allows brain penetration via the compromised blood brain-barrier, and thereby improves the function of both myelinated and unmyelinated axons of the corpus callosum.

KEYWORDS: CAP; TBI; axon; cerebral cortex; micelles

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