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Minocycline plus N-acteylcysteine induces remyelination, synergistically protects oligodendrocytes and modifies neuroinflammation in a rat model of mild traumatic brain injury.

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Abstract

Mild traumatic brain injury afflicts over 2 million people annually and little can be done for the underlying injury. The Food and Drug Administration-approved drugs Minocycline plus N-acetylcysteine (MINO plus NAC) synergistically improved cognition and memory in a rat mild controlled cortical impact (mCCI) model of traumatic brain injury.[3] The underlying cellular and molecular mechanisms of the drug combination are unknown. This study addressed the effect of the drug combination on white matter damage and neuroinflammation after mCCI. Brain tissue from mCCI rats given either sham-injury, saline, MINO alone, NAC alone, or MINO plus NAC was investigated via histology and qPCR at four time points (2, 4, 7, and 14 days post-injury) for markers of white matter damage and neuroinflammation. MINO plus NAC synergistically protected resident oligodendrocytes and decreased the number of oligodendrocyte precursor cells. Activation of microglia/macrophages (MP/MG) was synergistically increased in white matter two days post-injury after MINO plus NAC treatment. Patterns of M1 and M2 MP/MG were also altered after treatment. The modulation of neuroinflammation is a potential mechanism to promote remyelination and improve cognition and memory. These data also provide new and important insights into how drug treatments can induce repair after traumatic brain injury.

KEYWORDS: Brain trauma; immunohistochemistry; inflammation; microglia; oligodendrocytes; white matter

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