

Omega-3 fatty acids for brain and spinal cord injury: neuroprotection and beyond...

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Summary

Traumatic injury in the central nervous system triggers a host of tissue destructive processes and is for many individuals a life-changing event. Spinal cord injury and brain injury can have dramatic and irreversible consequences, and no neuroprotective or neuroregenerative treatment is currently available. It is appropriate to review the pathophysiology concepts and the proposed sequence of events which leads to tissue demise following neurotrauma, and consider the principles governing the present clinical translation. There is emerging evidence across a range of experimental models of traumatic injury of the brain and spinal cord, that omega-3 fatty acids have therapeutic and prophylactic potential. The endpoints used to characterize the efficacy of these compounds range from behavioural functional outcomes to histological evidence of tissue protection. The data suggest an intrinsic protection of neuronal and non-neuronal cells and a reduction of the damaging neuroinflammation that is triggered by injury. Results supporting this concept continue to be generated through use of transgenic mice with altered production of long chain omega-3 fatty acids, or through the use of various treatment regimes, from acute administration of fatty acids to dietary supplementation with fatty acids or with complex preparations enriched in fatty acids. The talk will review the accumulating evidence that supports the therapeutic potential of long-chain omega-3 fatty acids in central nervous system trauma and in peripheral nervous system injury and will highlight some of the questions that need to be answered concerning the molecular and cellular mechanisms that underlie the observed beneficial effects. Concerning neurodegeneration, one of the most important trends in the neurotherapeutics of the future is the temporal shift in the critical period of intervention aimed at aborting neurodegenerative processes. Neurodegenerative diseases such as Alzheimer's disease or Parkinson's disease, which have been historically considered distinct entities, although heterogeneous, are now viewed from a novel angle, influenced by network modelling and with a new understanding of common features such as the non-random propagation of abnormal protein aggregates. Interestingly, such pathological aggregation can be initiated by traumatic injury – thus, the challenge of neuroprotection bridges the acute and the chronic dimensions and requires entirely new approaches.

Reference

Michael-Titus AT and Priestley JV. Omega-3 fatty acids and traumatic neurological injury: from neuroprotection to neuroplasticity? Trends in Neurosciences. 2014 Jan;37(1):30-8.