Blocking a Receptor Protein Following Traumatic Brain Injury Improves Brain Function

Introduction:

- Traumatic brain injury (TBI) is a major cause of deaths worldwide.
- It leads to the progressive destruction of neurons or brain cells, causing a decline in brain function.
- Previous studies have found that in TBI, neuronal degeneration or breakdown depends on the presence of proteins called growth factors.
- The p75 neurotrophin receptor (p75NTR), which regulates multiple cellular functions, is activated after brain injury in the adult brain, where it is normally inhibited.
- The expression of this neurotrophin could lead to cell death.
- Researchers tried to determine whether preventing the expression of p75NTR or blocking the ligands for its receptor would reduce the extent of secondary neuronal cell death in the brain.

Methods:

- Adult male mice were subjected to controlled cortical impact, causing brain injury.
- The injured mice were divided into two groups and were intranasally administered either:
  - silencer RNA that can prevent the expression (or production) of p75NTR sequence, or
  - antibodies to block the functioning of pro-nerve growth factor (pro-NGF) and pro-brain-derived neurotrophic factor (pro-BDNF), factors that control the growth of nerves.

Results:

Histopathology finding as well as sensorimotor tests found that preventing p75NTR induction following injury limited the extent of neuronal damage and improved neurologic function as did the blocking of pro-NGF or pro-BDNF ligands.

Conclusion:

- Preventing the induction of p75NTR or blocking the proneurotrophin ligands provides neuroprotection and preserves brain function after TBI, improving treatment outcomes.
- These findings could lead to development of new therapeutic approaches to reduce neuronal injuries and degeneration following TBI.
Title of the paper: Proneurotrophins Induce Apoptotic Neuronal Death After Controlled Cortical Impact Injury in Adult Mice

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