

Natureza do trabalho: Resumo

TÍTULO

INTRATHECAL INJECTION OF CYTOKINES IN THE INDUCTION OF PAIN – REVIEW AND STANDARDIZATION OF THE TECHNIQUE IN MICE

MILLENE ARAUJO ROMERO, PAULA DE ALMEIDA SOUZA SANTOS DA COSTA, MATHEUS BAPTISTA PASSOS, ANA CLAUDIA PICCINELLI, CÂNDIDA KASSUYA

UNIVERSIDADE FEDERAL DE GRANDE DOURADOS, UFGD, DOURADOS, MS, BRASIL

RESUMO

Introduction: Cytokines are essential polypeptides for inflammatory response, which are produced at the site of injury. Their overproduction generates hemodynamic instability and may play an important role in pain. Recent evidence have suggested that TNF mediates central mechanisms of neuropathic pain through glial cells via activation of the p38-MAPK system. IL-1 β is also released during inflammatory and neuropathic pain conditions activating immune response and signal transduction both in the periphery and the central nervous system (CNS). Intrathecal injection of inflammatory cytokines induce hyperalgesia, allodynia and increases chronic pain and it is a viable and widely used animal model.

Objectives: In order to standardize the technique, the present work has investigated the hyperalgesic effects of TNF and IL-1 β when administered intrathecally in mice. **Material and Methods:** Male Swiss mice (n=6) received TNF (500ng) or IL-1 β (1000pcg) or sterile saline as control intrathecally. Mechanical sensitivity was measured with an electronic Von Frey apparatus after 1, 2, 3 and 4 hours of the injections.

Results: When administered intrathecally, TNF and IL-1 β significantly decreased mechanical sensitivity after 2 and 3 hours of the injection when compared to control group in mice. Maximum inhibition was 53 \pm 11% for TNF and 72 \pm 3% for IL-1 β after 3 hours of the injection. **Discussion and Conclusion:** All results of the present work show that intrathecal injection of cytokines cause hyperalgesia in mice after 2 and 3 hours of the injection. Performing tests of mechanical sensitivity with von Frey establishes a relationship between cytokines and pain. It is possible that increased sensitivity of mice to IL-1 β is due to greater range of inflammatory mediators activated by it. This study is crucial to understand the function of these cytokines in the CNS, and the relation of its changes with various diseases.