INTERLEUKIN-18 IN BRAIN TRAUMA OF THE NEONATE

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Traumatic brain injury (TBI) is a common cause of morbidity and death in the young population. Many mechanisms of brain damage had been proposed in TBI, among them – inflammatory cell involvement (1). The study of immune cell participation in TBI can be translated into novel strategies for repair after TBI and other acute central nervous system (CNS), therefore it has attracted a significant interest in the literature. A cross-talk between activated immune cells and neural cells could contribute to subsequent recruitment of inflammatory mediators into the brain, and thus to brain damage (1). In this volume of SSM, Goranova et al. (2) describe the involvement of a cytokine, interleukin-18 (IL-18), in perinatal TBI. As expected, a significant activation of microglia occurred after the experimental injury the researchers performed. Notably, both the protein and the mRNA levels of IL-18 and another cytokine, IL-1, were significantly increased after the injury. IL-1 is a known mediator of brain damage after TBI (3), however, the involvement of IL-18 has remained unresolved to date. Notably, both IL-1 and IL-18 are processed by the inflammasome – a large intracellular multiprotein complex that also activates caspase-1 in apoptosis (4). Indeed, the authors find an increased number of apoptotic cells in the vicinity of IL-1 and/or IL-18-positive cells. The latter turn out to be microglia (2), but a more careful analysis is necessary to precisely track down all cellular phenotypes that might express IL-1 and/or IL-18. Nevertheless, the data so far suggest a role for IL-18 in TBI as IL-18-deficient mice appear to be protected, at least in part, from this type of brain damage (2). IL-18 thus is novel molecular target for neuroprotection in this clinically significant field of neurosciences.

REFERENCES