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## Poster Presentation

### Trauma and Inflammation

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#### Abstract

One of the important reasons for death among the worldwide's population is trauma which is injury or damage to a biological organism caused by physical harm from an external source. After trauma, the immune system and the early inflammatory immune response activation lead to massive injury. Trauma causes activation of nearly all components of the immune system; it also activates the neuroendocrine system. Some endogenous immune triggers seem to have the potential to serve as novel biomarkers in predicting post-traumatic complications. Some mediators release in local tissue by accumulation of toxic by products of metabolic respiration and spillover of these mediators into the peripheral bloodstream causes extensive tissue injury. Hormones like ACTH, corticosteroids and catecholamines as well as cytokines, chemokines and alarmins perform important roles in the starting and persistence of the pro-inflammatory response after severe injury. Inflammation starts after trauma, immediately as a consequence of an efficient host defense system that is capable of sensing exogenous and pathogen-derived danger signals. Host defense system also sense endogenous, multifunctional alarm signals, and both of these two signals can initiate an inflammatory response. A number of receptors are involved in the host inflammatory response, including Toll-like receptors and new family of nucleotide oligomerization domain-like receptors capable of sensing the presence of danger signals in the cytoplasm. Toll-like receptors recognize host-derived, endogenous 'damage signals' like heat shock proteins and 'alarmins' such as the nuclear protein high-mobility group box protein 1, which are presented as a result of tissue trauma. It can be concluded that when trauma occurs, some pathways trigger, that results to inflammation. The neuroendocrine system, the immune system, hormones, some important mediators and receptors contributes to inflammatory responses.

**Keywords:** Inflammation, Trauma, Neuroendocrine.

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