Immunopathological Response to Severe Trauma and Burn: 
A Pilot Study in Progress

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Injury causes changes in coagulation and inflammation that appear to be interconnected. Microparticles and damage-associated molecular patterns (DAMPs) are expressed after injury due to cell damage and/or necrosis. These factors can act as initiators of inflammation and coagulation via interaction with specific receptors on the cell surface and instigate a cascade of intracellular events within immune cells, such as monocytes causing cytokine release or platelets altering coagulation. Experimental data suggests that burn and trauma are accompanied by elevated plasma levels of microparticles, DAMPs and cytokines, but there is limited data integrating mediators of inflammation (DAMPs, cytokines etc) and coagulopathy (microparticles, D-dimers etc). The aim of this pilot study is to correlate changes in the amounts of microparticles, DAMPs and cytokines with common parameters of coagulation and coagulopathy in patients with severe trauma (ISS>15) and burn. Blood samples were taken during the initial in-hospital resuscitation of trauma (n=10) and burn (n=10) patients before admission to the intensive care unit. DAMPs and D-dimer were measured by enzyme-linked immunosorbent assay, whereas cytokines were assayed by Bioplex assay. Microparticles were characterized using flow cytometry. Significant differences were observed between normal, trauma and burn patients in HSP-72, IL-22, TNF-α, D-dimer, IL-10 and IL-33. In general, trauma patients tended to have the greatest elevations in these parameters. Analysis of correlations between these factors and clinical data are ongoing. We predict that a potentially unique and unknown correlation between mediators of inflammation and coagulopathy after major injury will emerge from these studies.