Introduction
This study set out to quantify the ability of CytoSorb® hemoadsorbent polymer beads (CytoSorbents Corporation, USA) to adsorb a broad selection of inflammatory pathogen-associated molecular pattern molecules (PAMPS), damage-associated molecular pattern molecules (DAMPs) and cytokines from whole blood in a single compartment, in vitro recirculation system. PAMPS, such as bacterial exotoxins, cause either direct damage or trigger an immune response in the host to fight infection leading to the production of high levels of cytokines and the release of DAMPS into the bloodstream, which can trigger a maladaptive systemic inflammatory response syndrome (SIRS) that can further contribute to organ injury. The benefits of cytokine reduction using extracorporeal blood filtration with hemadsorbant porous polymer beads has been demonstrated in septic animals, yet the adsorption of other toxins and inflammatory factors may also contribute to the observed benefits.

Methods
Purified proteins S100A8, complement C5a, procalcitonin, HMGB-1, MIP1-α, IL-6, IFN-γ, TNF-α, Staph enterotoxin TSST-1 and aflatoxin B1 were added to 265 ml 3.8% citrated whole bovine blood at expected clinical concentrations and recirculated through a 20 mL CytoSorb® polymer-filled device or control (no bead) device at a flow rate of 140 ml/min for five hours. Fetal bovine serum was used for the Staph aureus α-toxin (α-hemolysin; 1.5µg/mL) recirculation due to the presence of anti-hemolysin antibodies in blood obtained from adult donors. Plasma was analyzed by ELISA.

Results
Hemoperfusion of whole blood through porous polymer bead devices for five hours removed substantial quantities of a broad spectrum of DAMPS, PAMPS and cytokines (Table 1). Levels of the inflammatory proteins were reduced by <20% during the five hour hemoperfusion through a control device (data not shown).

Conclusions
This study demonstrates that CytoSorb® hemoadsorbent polymer beads are capable of reducing a broad range of toxic DAMPS and PAMPS from blood providing a means, in addition to cytokine reduction, of reducing the uncontrolled inflammatory cascade that contributes to a maladaptive SIRS response, organ injury, multiple organ dysfunction syndrome (MODS) and death in patients with a broad range of life-threatening inflammatory conditions such as sepsis, trauma, lung injury, pancreatitis, acute respiratory distress syndrome and many others. Further study to elucidate the potential clinical impact is warranted.