Conclusions

- Aloe-emodin inhibited HMGB1 release in activated macrophage cells, by inhibiting LPS induced HMGB1 translocation from nucleus to cytoplasm.

- Aloe-emodin decreased the release of LPS-induced pro-inflammatory cytokines (TNF-α, IL-1β) in activated macrophages.

- Aloe-emodin decreased the acute inflammation induced stress markers NO, iNOS, HO-1 which may be a mechanism of HMGB1 regulation.

- Aloe-emodin protected mice from endotoxemic lethality.

- Aloe-emodin decreased the LPS induced proinflammatory cytokines (TNF-α, IL-1β) in mice at early hours of onset of disease.

- Aloe-emodin decreased the organ dysfunction (liver, kidney, lung) as seen by serum markers and histopathology study.

- Aloe-emodin decreased neutrophil infiltration and proinflammatory cytokine (TNF-α) in lungs.

- Psychosine showed different effect on LPS-induced inflammatory mediators depending on nature of cell.

- Effects on primary peritoneal macrophages resemble the in-vivo pathobiology of psychosine which hints a possibility of major role of HMGB1 in psychosine induced Krabbe disease.