Scavenging of inflammatory molecules improves sepsis in mice

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Sepsis is a life-threatening complication of infection in which the molecules that the body releases to fight an infection trigger widespread inflammatory responses, resulting in damage to multiple organ systems.

In this issue of *JCI Insight*, Huan Yang of the Feinstein Institute for Medical Research, Ulf Andersson of the Karolinska Institutet and colleagues, report on a method to scavenge inflammatory molecules that mediate sepsis in mice.

Treatment of mice with the protein haptoglobin resulted in removal of free hemoglobin as well as the protein HMGB1, which had not been previously known to interact with haptoglobin, but is a known mediator of inflammation.

Yang and colleagues demonstrated that immune cells known as macrophages removed haptoglobin/hemoglobin complexes as well as haptoglobin/HMGB1 complexes, thereby preventing these complexes from causing tissue damage.

These findings suggest that haptoglobin-based therapies could potentially be used to treat HMGB1-mediated inflammatory diseases such as sepsis.

More information: Huan Yang et al, Identification of CD163 as an antiinflammatory receptor for HMGB1-haptoglobin complexes, *JCI Insight* (2016). DOI: 10.1172/jci.insight.85375