

# Bio-hybrid device acts as 'thermostat' to control systemic inflammation in sepsis

May 14 2012

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A small, external bioreactor holding human cells pumped out an anti-inflammatory protein to prevent organ damage and other complications in a rat with acute inflammation caused by bacterial products in a model of sepsis, according to a report from researchers at the University of Pittsburgh School of Medicine and the McGowan Institute for Regenerative Medicine. The findings were published today in the inaugural issue of *Disruptive Science and Technology*.

Inflammation is a necessary [biological response](#) that brings cells and proteins to the site of tissue injury to contend with foreign agents, such as bacteria and the products they produce, and to begin the healing process, explained senior author Yoram Vodovotz, Ph.D., professor, Department of Surgery, and director, Center for Inflammation and Regenerative Modeling at the McGowan Institute. But sometimes, the inflammatory response escalates to create damage on its own, triggering more inflammation in a self-sustaining and dangerous cycle.

"In sepsis, for example, the inflammatory response evolves almost too quickly, but the available treatment strategies aim to prevent inflammation entirely," he said. "A better approach would be to turn down the response when it's too strong, yet still have appropriate inflammation signaling to promote tissue repair."

During inflammation, the body makes a protein called tumor necrosis factor-alpha, or TNF- $\alpha$ . It also makes its counterpart, soluble TNF- $\alpha$  receptor, or sTNFR, which binds to and reduces the level of TNF- $\alpha$ . In

some situations, such as sepsis, not enough sTNFR is made to limit the [inflammatory response](#).

Dr. Vodovotz and his team loaded a small bioreactor with human liver cells engineered to make sTNFR continuously. Through an intravenous line, blood from an anesthetized rat experiencing acute but transient inflammation was pumped through the bioreactor, exposing it to the engineered cells.

When the [bioreactor](#) was loaded with sTNFR-producing cells, sTNFR levels rose beyond what the animal could produce on its own, while TNF- $\alpha$  levels dropped, as did other markers of inflammation. The animal's blood pressure also improved, and markers of [organ damage](#) were reduced.

"This bio-hybrid device acts as a kind of inflammation thermostat," Dr. Vodovotz said. "By loading it with cells that produce different amounts of sTNFR, or other inflammatory blockers, we may soon be able to tailor our interventions to carefully balance inflammation and immune responses based on the patient's medical situation."

His team now is exploring the effectiveness of cells engineered to produce sTNFR based on the individual production of TNF- $\alpha$ , rather than continuously, in order to create a disease-specific response for each patient. Such a personalized medicine therapy platform could be extended based on emerging knowledge regarding the biology of inflammation.

The Vodovotz group also is creating computer models of inflammation, which could be used to engineer the next generation of this device. The portability of the device could be particularly useful on the battlefield, where early intervention to control systemic inflammation after injury might improve the chances of survival.

Provided by University of Pittsburgh Schools of the Health Sciences

Citation: Bio-hybrid device acts as 'thermostat' to control systemic inflammation in sepsis (2012, May 14) retrieved 27 April 2024 from <https://medicalxpress.com/news/2012-05-bio-hybrid-device-thermostat-inflammation-sepsis.html>

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