Alterations in fatty acid synthesis linked to sepsis inflammation

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Sepsis is a leading cause of death for patients in intensive care units. The excessive systemic inflammation in individuals with sepsis damages organs and can lead to death. Therapeutic options for sepsis are limited and the factors that promote this excessive response to infection are poorly understood.

A new study in the *Journal of Clinical Investigation* identifies a metabolic pathway that underlies sepsis inflammation. Augustine Choi and colleagues at Weill Cornell Medical College found that a mitochondrial uncoupling protein, UCP2, is elevated in patients with sepsis.

In mouse model of sepsis, lack of this protein improved survival. The authors determined that expression of UCP2 induces fatty acid synthesis, which in turn activates inflammatory pathways.

The results of this study suggest that UCP2 should be further explored as a potential therapeutic target for inflammatory diseases.

More information: UCP2-induced fatty acid synthase promotes NLRP3 inflammasome activation during sepsis, *J Clin Invest*,. DOI: 10.1172/JCI78253

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