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UQCRC2 Absence Reduced Mitochondrial Damage of Small Intestinal Epithelial Cells in Sepsis by Nrf2/HO-1 Signaling Pathway

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ABSTRACT This study was to explore the feasibility of UQCRC2 on mitochondrial damage of small intestinal epithelial cells (SIECs) in a model of sepsis to evaluate its mechanism. UQCRC2 mRNA and protein expression in patients or mice models of sepsis were up-regulated. UQCRC2 up-regulation accelerated mitochondrial damage, increased inflammation and oxidative stress of SIECs in vitro model of sepsis through the inhibition of Nrf2/HO-1 signalling pathway. UQCRC2 absence inhibited mitochondrial damage, and reduced inflammation and oxidative stress of SIECs in vitro model of sepsis. In the mice model of sepsis, sh-UQCRC2 also reduced mitochondrial damage, inflammation and oxidative stress in colon tissue. Taken together, the researchers conclude that UQCRC2 suppressed the Nrf2/HO-1 signalling pathway to promote mitochondrial damage of SIECs in sepsis, and provide molecular insight into the mechanisms by which the UQCRC2 absence regulates mitochondrial damage of SIECs in model of sepsis.