1-SS-42 Student Sessions

Effects of estrogen on septic inflammatory responses in skeletal muscle

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Sepsis is a potentially fatal or life shortening syndrome due to infection induced systemic inflammatory responses. Numerous experimental and clinical studies indicate sex differences in sepsis. The outcome and survival rates from sepsis are better in women than in men. Morbidity due to sepsis is complicated by myopathy, and patients face long-term disability due to muscle atrophy and paralysis called intensive care unit acquired weakness (ICU-AW). Here, we examined the effects of estrogen on the septic inflammatory responses in skeletal muscle. 17β-estradiol (E2) attenuated muscle weakness induced by polymicrobial sepsis in ovariectomized mice. Furthermore, E2 attenuated atrophy, and inflammatory cytokine productions induced by lipopolysaccharide (LPS), an endotoxin in C2C12 myotubes. On the other hand, E2 did not change proteolysis pathways such as LPS induced atrogin-1/MAFbx up-regulation and autophagosome formation in C2C12 myotubes. These findings indicate that E2 protects skeletal muscle from septic damage by reducing inflammatory cytokines. According to this study, estrogen should be one of the factors of sex difference in sepsis. Compounds with estrogen-like action may be potential seeds for drugs for ICU-AW treatment.