Results: The NAFLD cellular model shows significant lipid accumulation and lipid peroxidation compared to control hepatocytes. Both DCA and UDCA significantly decreased lipid accumulation without altering cell viability. Besides, DCA showed a greater ability in decreasing lipid peroxidation level.

Conclusions: Our findings demonstrate that both BAs improved lipid dysmetabolism and oxidative stress condition in the steatotic hepatocytes. UDCA seems to have the best protective and beneficial potential as it is able to both alleviate lipid accumulation in the steatotic liver cells, but also play antioxidant effect.

55ASM-0061 ST | Ablation of Aquaporin-9 ameliorates the systemic inflammation of LPS-induced endotoxic shock in mouse

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Background: Septic shock is the most severe complication of sepsis, characterized by a systemic inflammatory response following bacterial infection, leading to multiple organ failure and dramatically high mortality (42% at 28 days after diagnosis). Aquaporin-9 (AQP9), a membrane channel protein expressed mainly in hepatocytes and leukocytes, has been recently associated with inflammatory and infectious responses, thus triggering strong interest in AQP9 as a potential target for reducing septic shock-dependent mortality. Following up on previous *in vitro* work demonstrating AQP9 involvement in LPS-induced maturation of murine bone marrow dendritic cells and proinflammatory cytokines release, here we evaluated whether AQP9 contributes to murine systemic inflammation during endotoxic shock.

Materials and Methods: Wild type (*Aqp9*$^{+/+}$; WT) and *Aqp9* gene knockout (*Aqp9*$^{-/-}$; KO) male mice aged 9-12 weeks were submitted to endotoxic shock by i.*p*. injection of LPS (40 mg/kg) and the related survival times were followed during 72 hours. Electronic paramagnetic resonance and confocal microscopy were employed to analyse the nitric oxide (NO) and superoxide anion (O_2^-) production, and the expression of inducible NO-synthase (iNOS) and cyclooxygenase-2 (COX-2), respectively, in the liver, kidneys, aorta, heart and lungs of the mouse specimens.

Results: LPS-treated KO mice survived significantly longer than corresponding WT mice, and 25% of the KO mice fully recovered from the endotoxin treatment. The LPS-injected KO mice showed lower inflammatory NO and O_2^- productions and reduced iNOS and COX-2 levels through impaired NF-κB p65 expression/activation in liver, kidney, aorta and heart compared to the LPS-treated WT mice. Consistent with these results, treatment of a rodent hepatoma cell line (FaO) with the AQP9 blocker HTS13268 prevented the LPS-induced increase of inflammatory NO and O_2^-.

Conclusions: Overall, these findings suggest a role for AQP9 in the early acute phase of LPS-induced endotoxic shock involving the NF-κB signaling pathway. Modulation of AQP9 expression/activity may reveal useful in developing novel endotoxemia therapeutics.

55ASM-0109 ST | Effects of physical exercise in biochemical parameters and dorsolateral prostate lesions: Data from a rat model of prostate cancer

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Background: Prostate cancer (PCa) is among the most prevalent cancers worldwide. Physical exercise is widely recognized due to its beneficial effects. This study aimed to evaluate the effects of physical exercise on biochemical parameters and in dorsolateral prostate lesions in a rat model of PCa.

Materials and Methods: Ninety-five male Wistar Unilever rats were randomly divided into eight groups sacrificed at 35 (groups I) or 61 weeks of age (groups II): control sedentary groups (Cont+Sed I ($n = 10$); Cont+Sed II ($n = 10$)); induced sedentary group (PCa+Sed I ($n = 10$); PCa+Sed II ($n = 15$)); control exercised groups (Cont+EX I ($n = 10$); Cont+EX II