ABSTRACT

Acute kidney injury (AKI) complicates more than one third of intensive care unit admissions and is burdened by high morbidity and mortality of affected patients, with incidence steadily rising. Sepsis is the leading cause of AKI in critically ill. Despite growing insights into the pathogenesis of sepsis- induced AKI, we are so far not able to define successful AKI prevention and treatment. We aimed at assessing molecular mechanisms of sepsis- induced AKI using clinically relevant large animal model of sepsis and implementing new techniques of molecular biology- genomics and proteomics.

Although acidosis is a common acid base disorder in critically ill, its role remains controversial. It is unknown whether acidosis is a marker of disease severity or is directly implicated in pathogenesis of acute organ dysfunction states. Its protective role is discussed with growing evidence of acidosis induced cellular energetics downregulation and reduced oxygen demand in stress conditions. We aimed to evaluate physiological effects of different types of acidosis on healthy organism on systemic and regional level, including a complex research of its effects on kidney to search for new AKI preventive and treatment modalities, which permissive acidosis could represent.

Key words

Acute kidney injury – sepsis – acidosis - mitochondria