Hyperglycemia and insulin resistance are tightly interconnected pathophysiological conditions that increase the risk for development of cardiovascular diseases. Our current knowledge indicates that one of important reasons leading to the development of insulin resistance could lies in a dysfunction of adipose tissue with subsequent ectopic lipids accumulation in muscles and liver. Adipose tissue represents the main body storage site of energy. Furthermore, adipose tissue is also an active endocrine organ producing numerous biologically active molecules, e.g. cytokines, proinflammatory or to a lesser degree anti-inflammatory factors or components of the renin-angiotensin-aldosterone system. Some of the components of the local renin-angiotensin-aldosterone system produced by the adipose tissue can directly induce local insulin resistance within the adipose tissue.

Hyperglycemia and insulin resistance are also common in critically ill patients and exaggeration of insulin resistance in these patients is accompanied by increased morbidity and mortality. Our previous study has demonstrated that adipose tissue plays an important role in the development of hyperglycemia and insulin resistance in critically ill patients by production of numerous proinflammatory cytokines. First part of this dissertation thesis is therefore focused on changes of local renin angiotensin system in critically ill patients, because this system is tightly connected to the insulin resistance. In our study we found, that cardiac surgery increases angiotensinogen mRNA expression in epicardial but not in subcutaneous adipose tissue suggesting the possible involvement of epicardial adipose tissue in the process of development of local and possibly systemic insulin resistance in these patients.