PNEUMONIA COURSE ON THE BACKGROUND OF TYPE 2 DIABETES ASSOCIATES WITH DISORDERS OF ENERGY METABOLISM AND LEPTIN REGULATION

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Metabolic disorders worsen the course of acute pneumonia, which requires elucidation of molecular mechanisms that regulate energy metabolism in comorbid pathology. In rats with experimental insulin resistance and type 2 diabetes, we induced acute lung injury by injecting bacterial lipopolysaccharide (LPS). We found that increasing severity of pneumonia and mortality was depended on the mitochondrial dysfunction caused by diabetes. The changes in the expression of PGC-1α, IGF-1, SREBP-2, NLRP3 and DND1 proteins, leptin receptors, microRNA-1, -34a, and -320 were found in lungs and myocardium under these conditions. Under the influence of moderate periodic hypoxia, we showed the link between the induction of hypoxia-dependent proteins and microRNA and the level of glycemia. For the first time, the role of mitochondrial leptin receptors in changes in energy metabolism during LPS-induced lung inflammation and metabolic disorders was established. Thus, metabolic regulatory proteins, leptin receptors and microRNAs may be links of close interaction of regulatory mechanisms that are responsible for the severity of lung inflammation in comorbid development with the type 2 diabetes.