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Metabolomic Findings in Sepsis as a Damage of Host-Microbial Metabolism Integration

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Abstract

Metabolomics globally evaluates the totality of the endogenous metabolites in patient's body, at the same time reflecting gene function, enzyme activity and degree of organ dysfunction in sepsis. The authors performed the analysis of the main chemical classes of low molecular weight compounds (amino acids, polyols, fatty acids, hydroxy acids, amines, nucleotides and their derivatives) that quantitatively distinguish patients with sepsis from healthy ones. The following keywords were used to find papers published in the Scopus and Web of Science databases from 2008 to 2015: (marker OR biomarker) AND (sepsis OR critical ill OR pneumonia OR hypoxia). Key words for the search were the following: metabolomics, metabolic profiling, sepsis, metabolism, biomarkers, critically ill patients, multiple organ failure. Several metabolomic findings in sepsis are still waiting for an explanation. When assessing metabolomic analysis results in patients with sepsis we should take into account the intervention of microbial metabolism. Among the low molecular weight compounds detected in septic patient blood, a special attention should be paid to the molecules which could be attributed to "common metabolites" of man and bacteria. The genomic region overlap and the production of enzymes which are similar in function and final products could be a possible reason for this phenomenon. For example, microbial biodegradation products of aromatic compounds are increased many times in blood of patients with sepsis. On the one hand, it shows a high metabolic activity of the bacteria. On the other hand, these molecules are intermediates in the metabolism of aromatic amino acids such as tyrosine and phenylalanine in human body. It is important that there are many clinical studies, which confirmed the diagnostic and prognostic significance of series of aromatic metabolites, including those with intrinsic biological activity. We can't exclude the presence of signaling pathways, cell receptors, transmembrane transporters and others which are common for a human and bacteria and their direct participation in mechanisms of organ dysfunction and hypotension in sepsis. Thus, today, we should not limit ourselves studying eukaryotic cells while searching for new molecular mechanisms of sepsis-associated organ failure and septic shock. We should take into account and simulate in the experiments the