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Analysis of key genes and pathways involved in acute lung injury in a mouse model

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ABSTRACT. A mouse model of acute lung injury (ALI) was chosen in this study to explore the key genes and pathways involved in the process of ALI with microarray technology. Gene expression microarray data were downloaded from the Gene Expression Omnibus database. Mice from the experimental group were further divided into 6 subgroups, which received octadecenoate treatments for 1, 1.5, 3, 4, 18, and 24 h. Differentially co-expressed genes were screened to uncover the pathogenesis of ALI. Almost all of the differentially co-expressed genes were identified at two times: 1.5 and 3 h. Functional analysis revealed that several inflammation-related pathways were significantly enriched. Ubiquitin-mediated proteolysis, hematopoietic cell lineage, and leukocyte transendothelial migration were enriched at 1.5 h. The B cell receptor signaling pathway, T cell receptor signaling pathway, natural killer cell-mediated cytotoxicity, Fc epsilon RI signaling pathway, and ubiquitin-mediated proteolysis were significantly enriched at 3 h. It could be inferred that ALI initiated at 1.5 h and lasted through 3 h. However, co-expression patterns were not found from 4 h onward. In conclusion, several key genes and pathways implicated

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in the development of ALI were found in this study using the mouse model, among which ubiquitin-mediated proteolysis appears to play an important role in the process.

Key words: Acute lung injury; KEGG pathway; Mouse model; Inflammation

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