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A comparative study between infectious and systemic inflammation

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Abstract

Activation of innate immune system may occur as a result of either external (mostly infection-mediated inflammation) or internal factors (systemic inflammation). Distinct stimuli act on the immune cells to induce diverse pathways leading to characteristic gene expressions in these cases. Bacterial inflammation, caused primarily by its lipopolysaccharides (LPS), conceives an array of diseases including intestinal bowel disease (IBD), ulcerative colitis and sepsis. In contrast, release of pro-inflammatory cytokines such as IL-6 or TNF-α leads to chronic inflammatory diseases, for example, rheumatoid arthritis (RA), juvenile idiopathic arthritis, Castleman's disease, etc. It is important to understand the signatures of infectious and systemic gene expression for better designing of treatment regime against inflammatory diseases. To understand the distinctive pattern of gene expression between infectious inflammation and systemic inflammation, THP-1 macrophages were treated individually with LPS (100 ng/mL), IL-6 (50 ng/mL) or TNF-α (10 ng/mL) and global transcriptomic analysis was performed using Agilent's human 8x15K array. The common set of differentially expressed genes in IL-6 and TNF-α-treated cohorts were compared with LPS-treated cohorts. Our analysis revealed that 2743 and 150 genes contributed to LPS-mediated inflammation and systemic inflammation with respect to untreated samples, respectively (fold change ≥ 1.5). 868 commonly expressed genes contributed to systemic inflammation with respect to LPS-mediated inflammation. Among these commonly expressed genes, only 68 genes were observed to contribute to both types of inflammation, suggesting their importance in activation of diverse pathways in LPS-mediated and systemic inflammation. A detailed functional annotation of these genes revealed that EGR1, JUN, NF-kB, REL, STAT-1 and BCL-3 are important transcription factors (TFs) for distinctive signatures between these two types of inflammation. In addition, these TFs were found to be involved in innate immune response. Further investigation into the gene expression dataset from rheumatoid arthritis patients (treated with anti-TNF-α antibody) revealed 24 genes which are present within these 68 genes having an inverse mode of expression. This observation suggests the importance of these 24 genes for designing novel therapeutic targets.

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