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Predicting Signaling Cascades of Inflammasome-Induced Inflammatory Cell Death Following SARS-Cov-2 Infection through A Bioinformatic Approach

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ABSTRACT

Introduction: Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is characterized by respiratory failure, pneumonia, coagulation, and multiorgan failure due to deregulation of cytokine release. Inflammasome activation has been reported with tissue injury and increased disease severity. Here, we conducted a bioinformatic analysis to predict possible cellular and molecular mechanisms of inflammasome activation-induced cell injury/death in COVID-19.

Methods and Materials: All genes- associated with SARS-CoV-2 and inflammasome were extracted from GeneWeaver. Afterward, common genes were selected for further enrichment analysis. Common genes were uploaded into the ToppGene database to predict significant molecular functions, biological processes, Cellular components, signaling pathways, and microRNAs for both SARS-CoV2 and inflammasome. Cytoscape was also used to reconstruct the drug-genetic network for shared genes.

Results: Our results demonstrated that 62 genes were related to inflammasome, and 2379 genes were associated with SARS-CoV-2. Among these gene sets, nine genes such as tumor necrosis factor, cathepsin B, baculoviral IAP repeat containing 3, interleukin 6, absent in melanoma 2, leptin receptor, NLR family pyrin domain containing 1, nucleotide-binding oligomerization domain containing 1, and signal transducer and activator of transcription one were shared between inflammasome and COVID-19. NOD-like receptor signaling, nucleotide-binding domain leucine-rich repeat-containing receptor NLR signaling, TNF-related weak inducer of apoptosis TWEAK signaling, SARS-CoV2 innate immunity evasion and cell-specific immune, and toll-like receptor signaling were the most significant involved pathways in pyroptosis following COVID-19. The external side of the plasma membrane, membrane raft, ISGF3 complex, AIM2 inflammasome complex, and endolysosome lumen were the main cellular components that may disrupt following inflammasome activation in COVID-19.

Conclusion and Discussion: Notably, our findings predicted some microRNAs and revealed central signaling cascades following inflammasome activity, which may benefit future therapeutic targets in COVID-19.

Keywords: Inflammasomes, Inflammation, SARS-CoV-2

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