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ABSTRACT

The COVID-19 contagion commenced by the "Severe Acute Respiratory Syndrome Coronavirus 2" has become a prime health concern. It has already meted out insuperable damage to human lives and the global economy. Despite the pioneering development of vaccines, the fluctuation in strain pattern of the novel coronavirus has led to immune evasion and posed a challenge to the efficacy of vaccines primarily industrialized against the prototype strain. Inclusive efforts are already underway at a war footing to find the best drugs or drug combinations to address future attempts in sinking the disease. On this account, drug repurposing strategies introduced ahead of time were employed to identify the potential drugs intended to contain the disastrous virus outbreak. This study aims to define a network-based approach to filter a set of approved and experimental drugs from the DrugBank database redirected as competing treatments for the COVID-19 disease therapy. Our method described the interaction networks of seventeen (17) repurposable drugs defining topological and statistical features with shared biological processes of the host cells. The drug-gene interaction data specifically outlined from the Drug Gene Interaction Database was followed by the network construction through GeneMania and Cytoscape. The networks then underwent enrichment analysis through the EnrichNet tool to observe the functional linkage between the gene-sets and pathways, including the user-defined dataset and the reference dataset. Finally, the drugs arrayed under the significance measure of pathways with overlapping genes identified thirty-nine (39) drug-pathway interactions of statistical significance and insignificant similarity scores for two drugs (Human-Interferon Beta and Elbasvir) against cellular pathways. For comparative review and target assessment of all the drug interaction modules, we first labeled the disease-associated genes and pathways from previously extracted results of text mining resources and interactome studies on coronaviruses to compare them with our findings. Our results illustrated drug-target interactions for fifteen (15) genes contributed between the disease comorbidities and eight (8) pathways essential to pathogenesis. This strategy concludes the importance of repurposing target-based drugs against clinically significant genes and pathways for the COVID-19 disease therapy.

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