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Commentary

Brief Note on Acute Respiratory and Down Syndrome

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DESCRIPTION

The recent outbreak of the novel Acute Respiratory Syndrome SARS-Coronavirus 2 (SARS-CoV-2) has caused more than 86 million COVID-19 cases and nearly 2 million deaths, with statistics expected to rise in the coming months in COVID-19 is particularly severe in the elderly (>over 60 years of age), especially in chronic medical conditions (i.e., hypertension, diabetes, cardiovascular disease, chronic lung disease or cancer). However, the rapid spread of SARS-Cov-2 infection has prevented studies on other vulnerable populations, such as people with Intellectual Disabilities (ID). Down Syndrome (DS) is the most common genetic form of ID. People with DS suffer from comorbidities such as obesity, type I diabetes or Congenital Heart Disease (CHD), which are associated with poor COVID-19 prognosis in the general population. Hypotonia, developmental delay, obstructive sleep apnea, craniofacial disorders, immunodeficiency and heart problems, as well as gastro esophageal reflux also increase the risk of common and respiratory infections and may develop more serious complications such as pneumonia, hospitalization, intubation and more deaths due to secondary bacterial infections during other viral respiratory infections such as influenza 2 and Respiratory Syncytial Virus (RSV) in DS. Another risk factor is a significant weakening of the immune response in DS4. Defects in trisomic individuals include functional abnormalities in a variety of immune cells (i.e., T- and B-cells, monocytes and neutrophils) and are important in vaccine-developing, suboptimal antibody responses. Several studies have found reductions in T and B cells and increased serum inflammatory cytokine levels in individuals with DS, indicating suboptimal immunization. In the general population, patients with severe COVID-19 generally show elevated cytokine levels (IL-6, IL-10, IL-2, IL-7 and TNF-α), lymphopenia (in $CD4^+$ and $CD8^+$ T-cells) and decreased IFN-Γ expression in $CD4^+$ T-cells, They are associated with severe COVID-19 and the so-called "cytokine cyclone", which is a high immune response to external stimuli, which predicts a worsening COVID-19 diagnosis and correlates with the severity of pathogenic coronavirus infections. DS is characterized by 'inflammatory priming' of immune cells, which corresponds to the level of cytokines in the blood in the absence of viral infections. Furthermore, the Interferon Response (IFN), DS and interferon alpha and beta receptor subunits 1 and 2 (IFNAR1 and IFNAR2), which are crucial for initiating and enhancing cytokine release, are hypersensitive to heterodynamic cleavage cells. Finally, recent evidence suggests that an increase in Trans membrane protease serine 2 (TMPRSS2) expressions may lead to an increase in infection/infection with lower viral titers. Corona virus cell entry is based on the binding of virus spike (S) proteins to cellular receptors and the priming of S proteins by host cell proteins. The TMPRSS2 gene in Chr21q22 encodes a proteolytically activating serine protein S protein to enter the Angiotensin-Converting Enzyme 2 (ACE2) as an entry receptor for the SARS-CoV-2 virus Host cell. In fact, the TMPRSS2 inhibitor approved for clinical use has been proposed as a blocked entry treatment option.

In theory, all of these factors could lead to further damage in the DS, which is activated by triplet genes encoded by human chromosome 21 (HSA21). However, although genetic expression studies show the highest percentage of differential genes expressing HSA21, the transcriptome extends the mitigation gene, with many non-HSA21 genes largely uncontrolled or uncontrolled. To understand the potential differential effect of COVID19 in individuals with DS compared to the general population, we mapped the pathways affected by SARS-CoV-2 and the transcriptomic changes induced by trisomy on proteins. We argue that other genes that are consistently regulated in DS other than TMPRSS2 can define the genetic risk factors of DS-COVID19 comorbidity. We analyzed 69 DS transcriptomic and proteomic studies and found that genetic mapping on HSA21 was not consistently regulated in different tissues and states. We have also argued that the levels of proteins that interact or regulate with the HSA21 gene (HSA21 interactors) may be regulated in trisomy 21 cases. We analyzed the overlap between the molecular pathways in COVID-19 and their interaction with host proteins. Viral proteins were created from the SARS-CoV-2 and DS-SARS-CoV-2 networks.

To gain insight into the interaction between DS genes and SARS-cov2 infection and pathogenesis, they identified genes

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associated with the molecular pathways involved in COVID-19 and host proteins that interact with viral proteins from SARS-CoV-2. We created the DS-SARS-CoV-2 network by analyzing the overlaps of these genes that consistently differentiate DS (using public transcriptomic datasets) with HSA21 genes, HSA21 interactors, and other genes. We identified COVID-19 protection and risk factors in HSA21 genes and interactors and/or DS uncontrolled genes that may affect the susceptibility of individuals with DS during the infection phase and during the transition to acute respiratory distress syndrome. Our analysis suggests that DS individuals are more susceptible to infection due to triplets of TMPRSS2 during the infection phase, which primes the viral S protein to enter the host cells. However, since anti-viral interferon I signaling is also regulated in the DS, it enhances the initial anti-viral response, inhibiting viral genome release, viral replication, and viral assembly. In the second pro-inflammatory immune pathogenic stage of the infection, the prognosis for DS patients may deteriorate further due to the regulation of inflammatory genes that favor the normal cytokine cyst of COVID-19. We have also identified strong regulation of the NLRP3 gene, which is crucial for the maintenance of homeostasis against pathogenic infections, possibly leading to bacterial infection complications.