

Scientists at DBT-NBRC get some preliminary clues for developing anti-Covid-19 therapies

Severe acute respiratory syndrome coronavirus (SARS-CoV2) is a highly pathogenic respiratory virus that causes morbidity and mortality in humans. SARS-CoV-2 enters host cells by binding to angiotensin converting enzyme–related carboxy-peptidase (ACE2) receptor. ‘Cytokine storm’ characterized by elevated levels of pro-inflammatory cytokines is a profound hallmark of severe COVID-19 infections, and accounts for acute respiratory distress syndrome in patients. The lungs are the major pathological target of SARS-CoV2 infection. The dysregulated inflammation characteristics of lung cancer have marked similarities with the clinical manifestation of SARS-COV2 infection. Given the intriguing commonalities between SARS-CoV2 infection and lung cancer in regard to pathological derangements and heightened inflammatory response, we analyzed The Cancer Genome Atlas (TCGA) data sets of lung cancer for correlations between ACE2 and genes associated with inflammatory responses. Our analysis has identified the involvement of (i) ACE2-BDKRB1-inflammatory network and (ii) FOLR1-Nitric oxide cross-talk in contributing to dysregulated inflammation in lung cancer. This comparative analysis has highlighted that functional integration of diverse intracellular signaling networks in lung cancer can be extrapolated to explain the aberrant inflammatory response in COVID-19 infection. With the pandemic COVID-19 spreading unabated, the current challenges are to identify therapeutic strategies and efficiently evaluate their activity in clinical setting. On seeing COVID19 through the lens of lung cancer, the finding suggest that therapeutics with safety and tolerability profiles targeting BDKRB1 and FOLR1 in lung cancers could be repurposed for calming the cytokine storm in COVID-19 infection.

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