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EC PULMONOLOGY AND RESPIRATORY MEDICINE

Editorial

Lung Cell Damage in Corona Virus Infection: Study Showing Altered "Protein Phosphorylation" as a Key Factor in COVID-19 Infection

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Received: December 15, 2020; **Published:** December 29, 2020

In a multi-group collaborative study involving the National Emerging Infectious Disease Laboratories (NEIDL), the Centre for Regenerative Medicine (CReM), and the Centre for Network Systems Biology (CNSB), scientists at the Boston University School of Medicine (BUSM) have apparently decoded a response of human lung cells to the coronavirus. This breakthrough could ultimately help in identifying clinically approved medications for other medical conditions which could be redirected for treatment of COVID-19 infections.

This study [1] assessed the response of bioengineered human alveolar cells using highly precise mass spectrometry technology.

Based on this analysis, BUSM researchers[1] have identified host proteins and pathways in pulmonary alveolar cells whose levels change upon infection by the SARS-CoV-2. This could provide good insights into the pathological process and help develop new therapeutic outcomes to block COVID-19.

This study found an important type of protein modification called "phosphorylation" which became altered in these infected lung cells. Normally, phosphorylation of proteins plays an important role in regulating protein function inside cells, and both protein abundance and protein phosphorylation are tightly-controlled processes in the case of normal lung cells.

In this study it was shown that SARS-CoV-2 throws the lung cells into chaos, causing abnormal changes in protein amounts and frequency of protein phosphorylation inside infected lung cells. These abnormal changes help the virus to multiply and eventually destroy the lung alveolar cells. The destruction of infected alveolar cells is responsible for the widespread lung injury.

According to the researchers, as soon as the SARS-CoV-2 enters the lung cells, it rapidly utilizes the cell's core resources, which are normally required for the cell's growth and normal functions. The virus utilizes these resources to multiply, while avoiding being attacked by the body's immune system. Consequently, new virus particles form which subsequently leave the highly damaged lung cells, thereby causing them to self-destruct. These new viruses in turn infect other cells, and the pathological process is repeated again.

The researchers examined lung alveolar cells from one-to-24 hours following infection with SARS-CoV-2 to understand what changes occur in lung cells immediately (at one, three and six hours after infection by SARS-CoV-2) and what changes occur later (at 24 hours after infection). These changes were then compared with normal, healthy cells.

The results showed that in comparison to normal healthy lung cells, SARS-CoV-2 infected alveolar cells showed a profound increase of proteins and phosphorylation as early as one hour following COVID-19 infection resulting in the destruction of the host lung cells.

The researchers after analysing their data found that at least 18 pre-existing clinically approved drugs developed originally for other medical diseases, can be potentially used for treating COVID-19 infection. These medications have shown a fairly good effect in blocking the proliferation of the SARS-CoV-2 in alveolar lung cells.

Hence, the study results could help create more potent therapeutic regimes to successfully fight the COVID-19 infection, and flatten the curve of the pandemic.

Bibliography

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Volume 10 Issue 1 January 2021