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COMMENTARY

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An Overview-Oxidative Stress Induced Respiratory Disease Associated with Covid-19

Robhin Condliffey*
Department of chemistry, Imperial College London, United Kingdom

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Commentary

In most of the cases respiratory disease are caused by novel corona virus SARS-CoV-2 which leads to Severe COVID-19 infection that triggers imbalanced and uncontrolled cytokine response also called as cytokine storm and vascular thrombosis. These factors may further lead to the development of acute respiratory distress syndrome (ARDS) which is a major cause of death of the COVID-19 patients. Due to excessive activation of immune and endothelial cells, and platelets the pathological changes in organs and tissues are triggered by an imbalanced host reaction to the cause infection. The oxidative stress accompanying cell activation may completely affect COVID-19 pathogenesis. COVID-19 caused by the SARS-CoV-2 may be a complex disease during which interaction of the virus with target cells, action of the immune system and therefore the body's systemic response to these events are closely intertwined. Many respiratory viral infections, including COVID-19, cause death of the infected cells, activation of innate immune reaction, and secretion of inflammatory cytokines. All these processes are related with development of oxidative stress, which makes a crucial contribution to pathogenesis of the viral infections.

Respiratory Viruses Caused by Oxidative Stress

Several data has been confirmed that there is a development of oxidative stress in many viral infections including respiratory diseases. An excessive amount of reactive oxygen species (ROS) is produced in various tissues along with influenza infection, which includes alveolar epithelium and endothelium. Oxidative stress is typical for infection of human respiratory rhinoviruses and lot of other viruses. It includes high level of oxidized biomolecules such as DNA, lipids, and proteins for patients infected with the influenza virus. Additionally, in the lung tissue samples from the deceased influenza patients the elevated ROS production up regulates NO (nitric oxide) synthase-2 (NOS2) expression, and high level of nitrated proteins indicating developed oxidative and nitrosative stress were observed.

On the whole, all patients suffering with viral infections will be affected by chronic oxidative stress impacting the disease pathogenesis including impaired immune functions, apoptosis and inflammatory response, along with organ and tissue dysfunction.

These also found to have various beneficial activities to give relief from the diseases. The extractions such as aqueous and alcoholic of cinnamon significantly inhibit fatty acid oxidation and lipid peroxidation. Also, different flavonoids having free radical scavenging activities as well as antioxidant properties were seen. There are several common traits between oxidative stress and therefore the risk of severe COVID-19 infection. Within the lung tissues affected by virus replication, Pneumonia caused by SARS-CoV-2 induces over activation of immune response. This pathological process is always accompanied by oxidative stress.

By infecting type II Pneumocytes, SARS-CoV-2 is capable of causing severe pneumonia. These cells contain an outside number of mitochondria synthesizing acetyl-CoA to be used for production of fatty acids and phospholipids, which constitute pulmonary surfactants on the surface of epithelial cells. A patient with moderate and severe COVID-19 often tends to develop respiratory distress compensated by oxygen therapy that causes oxidative stress.

Endothelium Inflammatory Activation in Covid-19

Endothelium itself may be a target of SARS-CoV-2 corona virus and through the COVID-19 infection pro-inflammatory Cytokines were released. Here the

Contact Robhin Condliffey robin.condliffey@hs.uk Department of chemistry, Imperial College London, United Kingdom

activated endothelial cells release pro-inflammatory cytokines and chemokines that recruit immune cells into the area of inflammation.

By the enhancement of endothelial permeability for macromolecules which induce lung edema the endothelial activation by pro-inflammatory cytokines is accompanied. Insulating properties of the micro vascular endothelium are determined mainly by the intercellular VE-cadherin based adherens junctions.

Disruption of the glycocalyx not only leads to enhanced endothelial permeability but also increases the availability of adhesion molecules, thereby stimulating leukocyte and platelet attachment.

In the TNF-dependent up regulated expression of the adhesion molecules and enhanced endothelial permeability the increased ROS production plays a crucial role. The main ROS target in apoptotic endothelial cell induced by high TNF concentrations is Mitochondria. Vascular endothelial growth factor (VEGF) plays an important role during disruption of the pulmonary endothelium function.

Endothelial VEGF expression is up regulated upon inflammation under hypoxic conditions. During COVID-19, angiogenesis seems to develop from the endothelium damage.

The use of antioxidants seems reasonable at the stage requiring inhibition of inflammatory reactions during COVID-19. Antioxidants may influence thrombogenesis, which may be a common and dangerous complication during COVID-19. Cytokine storm may end in the ROS-dependent apoptosis in endothelial cells. Lowering oxidative stress by antioxidants may end in the decreased viral load.