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Biochemistry of Vitamin C: COVID-19 Treatment

Jennifer Fernandiz*

Editorial office, Biochemistry: An Indian Journal, India

***Corresponding author:** Jennifer Fernandiz, Editorial office, Biochemistry: An Indian Journal, India, E-Mail: chemicalinformatics@chemjournals.org

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Abstract

Vitamin C's (L-ascorbic acid) role as an antioxidant and cofactor in a variety of metabolic activities has a long history and is well-known today. Many issues about its method of action and the benefits it provides to human health remain unanswered. This is true not only for the specified doses but also for the delivery route. Furthermore, there are many unanswered uncertainties about vitamin C's therapeutic efficacy in a variety of human (infectious) disorders, as well as its immune system activity and antiviral potential. Vitamin C's ability to function as both an antioxidant and a prooxidant stresses its oxidation-reduction (redox) potential in real-life situations. The effect of intravenous vitamin C supplementation in patients with SARS-CoV-2 warrants specific consideration today. To better comprehend the current new difficulties associated with vitamin C, this review will highlight known facts regarding vitamin C and its modes of action.

Keywords: SARS-CoV 2; Therapeutic; Viral cells; Clinical illnesses; Cytokines; Vitamin-C

Introduction

With the introduction of a new virus from the beta coronavirus family known as SARS-CoV-2, the antiviral capabilities of vitamin C have been called into question once again. Coronaviruses of the beta genus (CoVs) are single-stranded RNA viruses that are enclosed and positive-sense [1]. They primarily infect bats, but they can also infect other species such as rodents and humans. The Middle East Respiratory Syndrome coronavirus (MERS-CoV) and the Severe Acute Respiratory Syndrome coronaviruses SARS-CoV-1 and SARS-CoV-2 have both produced human outbreaks. Coronavirus Disease 2019 is the result of an infection caused by the virus SARSCoV-2 (COVID-19). SARS-CoV-2 infects lung endothelial cells through binding to the Angiotensin-Converting Enzyme 2 (ACE2) receptor *via* Spike Glycoprotein (S) (ECS). ACE2 receptors can be found in a variety of tissues, including hematopoietic cells, kidneys, and intestines. The presence of SARS-CoV-2-infected ECs in various organs of deceased patients supports this theory. Dysfunction, lysis, and death are all symptoms of infected ECs. Furthermore, through producing leukocyte adhesion molecules, ECs increase inflammation in injured tissues, inducing leukocyte buildup and extravasation. The activation of leukocytes causes a cytokine storm. Activated neutrophils and macrophages produce an excess of reactive oxygen and nitrogen species after being attracted to the lungs [2].

On the one hand, ROS and RNS regulate immune response by activating transcription factors that control the expression of inflammatory cytokines and chemokines through oxidant-induced activation. ROS, on the other hand, can behave as oxidants, destroying viral cells as well as lung (heart) cells, further causing EC dysfunction and, ultimately, lung tissue destruction. The loss of microvascular barrier function in the lungs happens in cases of extensive endothelial cell injury, resulting in increased vascular permeability. Furthermore, SARS-CoV-2 binding to the ACE2 receptor prevents ACE2 from degrading angiotensin II, causing the angiotensin-vasopressor system to malfunction. Increased vascular permeability is also a result of decreased ACE2 activity. EC contractility is also aided by immune cells, inflammatory cytokines, and vasoactive substances. Fluid seeps and fills alveolar sacks as a result of these events. Finally, the cytokines IL-1 and TNF cause fluid retention in the lungs (pulmonary edema). High amounts of cytokines, which are constantly produced as a reaction to viral infection, amplify all of these symptoms-EC failure, inflammation, vasodilation, and the formation of blood clots. The COVID-19 can show in a variety of ways, from asymptomatic to severe pneumonia. Sepsis occurs when the body's response to infection causes damage to its tissues and organs. Pneumonia is the most prevalent infectious cause of sepsis. Acute Respiratory Distress Syndrome (ARDS) and septic shock are caused by alveolar dysfunction and severe lung injury, which can progress to multiple organ failures and death. As a result of fluid buildup in the lungs, ARDS prevents needed oxygen

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from entering the lungs, resulting in hypoxic respiratory failure. As a result, ARDS and septic shock, as well as concurrent clinical illnesses such as hypertension, cardiovascular and cerebrovascular disorders, and diabetes, are the leading causes of ICU admission and mortality in COVID-19 patients.

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Conclusion

The most commonly utilized supplement is vitamin C. Historically, it was similar to aspirin. In both good and bad health. Regardless of the technique, amount, or duration of supplementation, the COVID-19 epidemic continues today. We could say it's reborn as a Phoenix, or perhaps it's more accurate to say it's a Phoenix all the time. A redox Phoenix, utilized long before we knew what it was and long before we understood how it worked *in vivo.*

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