Editorial

Corona-Cov-2 (COVID-19) and ginseng: Comparison of possible use in COVID-19 and influenza

ABSTRACT

In the 1918 influenza pandemic, more than 95% of mortalities were ascribed to bacterial pneumonia. After the primary influenza infection, the innate immune system is attenuated, and the susceptibility to bacteria is increased. Subsequent bacterial pneumonia exacerbates morbidity and increases the mortality rate. Similarly, COVID-19 infection attenuates innate immunity and results in pneumonia. In addition, the current pneumococcal conjugate vaccine may have limited defense against secondary pneumococcal infection after influenza infection. Therefore, until a fully protective vaccine is available, a method of increasing immunity may be helpful. Ginseng has been shown to increase the defense against influenza in clinical trials and animal experiments, as well as the defense against pneumococcal pneumonia in animal experiments. Based on these findings, ginseng is suspected to be helpful for providing immunity against COVID-19.

Copyright 2021, The Korean Society of Ginseng, Published by Elsevier. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).
and mortality [7,9]. Host receptors for bacterial attachment and infection are exposed, and host receptor expression is increased. Viruses and bacteria express factors that overturn, suppress, or eliminate the host immune response. As a result, pathogens can overgrow, increasing the inflammatory response and leading to immune-mediated host damage. Bacteria are generally secondary invaders during influenza infection, but they express virulence factors that promote viral pathogenesis, increasing the amount of virus and decreasing its likelihood to be eliminated. Thus, the disease worsens, and the mortality rate increases [7,9] (Fig. 1A).

The population older than 65 years is increasing globally. Since aged persons are highly susceptible to respiratory diseases due to weakened immune system, the mortality rate of this population could be higher than ever before. The mortality rate in those older than 70 years from pneumonia showed no change from 1990 to 2017, whereas in those younger than 5 years decreased steadily due to introduction of vaccines (https://ourworldindata.org/pneumonia). The currently used pneumococcal 13-valent conjugate vaccine may have limited defense against secondary pneumococcal infection after primary influenza infection, and this limited protection has been associated with impaired induction of anti-pneumococcal humoral response [11]. In addition, antibodies produced after COVID-19 infection contain a short-acting protective antibody that is effective for only weeks or months [12]. Guidelines of the United States Food & Drug Administration on the approval process for COVID-19 vaccines requires products to show at least 50% prevention or decrease of disease severity (https://www.fda.gov/media/139638).

COVID-19 causes a variety of diseases from respiratory diseases to enteritis. The COVID-19 spike protein binds to the receptor ACE2 (angiotensin-converting enzyme 2) [3] in human epithelial cells in the airway and lung tissue. However, ACE2 in human can be induced by not only influenza infection, but also IFN [13]. ACE2 is present in a wide variety of cells comprising not only lung, but also brain, heart, placenta, pancreas, and enterocytes, suggesting that COVID-19 can affect many organs [13]. The COVID-19 spike protein is identical to eight amino acids of the sodium channel protein in human epithelial cells, suggesting that the virus may interfere with function of this channel. This competition can disrupt or dysregulate sodium channel activation and can explain why COVID-19 patients sometimes experience surplus fluid in the lungs [14]. Therefore, until a vaccine that can fully defend against these infectious respiratory diseases is supplied, a method of increasing immunity may be helpful.

The incidence rate of common cold symptom complex (CCSC) including flu during a 2-year period in Japan demonstrated a 1.38% incidence in patients taking Korean Red Ginseng (KRG) compared to the 4.89% in patients not taking KRG, indicating preventive effects of KRG on the CCSC [15]. Consistently, in mice experiments, KRG increased the defense against influenza virus H1N1 [16] and H5N1 [17,18]. KRG protected tissue after H1N1 influenza virus infection via stimulation of anti-viral cytokine IFN-γ secretion [19]. Moreover, KRG provided protection against bacterial pneumonia-septicemia caused by pneumococcus in animal experiments by decreasing inflammatory cytokine secretion, resulting in a decrease in mortality rate [20]. KRG could enhance vaccine efficacy via increase of phagocytosis, inhibition of reactive oxygen species production, and reduction of apoptosis signaling and inflammation [21].

Taken together, these finding indicate that the pathogenesis of COVID-19 is similar to that of influenza. Lack of efficient pneumococcal vaccine during respiratory viral and bacterial co-infections suggests the need for ginseng products, which have been shown to alleviate respiratory viral and bacterial co-infections, for non-specific immune stimulation and protection as auxiliary agents.

Acknowledgements

This work was supported by a National Research Foundation of Korea grant (NRF-2018R1A2A1A05078102). The funding body played no role in the study design, data collection and analysis, decision to publish, or preparation of the manuscript.

References


