



Can sunlight affect COVID-19 outcomes? What is the available evidence?

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Dear Editor,

Coronavirus disease 2019 (COVID-19) has rapidly spread into a global pandemic that challenges the economic, medical, and public health stability of nations worldwide.¹ Severe acute respiratory syndrome coronavirus (SARS-CoV)-2, first identified in China in December 2019 as the "novel coronavirus," causes COVID-19 by utilizing angiotensin-converting enzyme 2 receptors and transmembrane protease, serine 2 (TMPRSS2, located on host cell surfaces) for entry and infection. Thus, TMPRSS2 is required for SARS-CoV-2 infectivity.¹

Interestingly, total prostate-specific antigen levels vary by season. The prolonged daylight hours of spring and summer are associated with reduced androgen sensitivity, which lowers TMPRSS2 expression and subsequently reduces SARS-CoV-2 infectivity. Conversely, the shortened daylight hours of autumn and winter are associated with increased SARS-CoV-2 infectivity.²

Dopico et al.³ demonstrated that the cellular composition of blood also varies by season: soluble interleukin (IL)-6 receptor and C-reactive protein concentrations increase during winter and estradiol receptor gene expression increases during summer. This is pertinent because SARS-CoV-2 infections cause cytokine storms and systemic inflammatory responses that are mediated by the release of large amounts of pro-inflammatory cytokines and chemokines (e.g., IL-1b, IL-6, and interferon- α) from immune effector cells.¹

Sunlight exposure mediates the conversion of previtamin D3 (precholecalciferol) into vitamin D3 (cholecalciferol) by liver enzymes. Vitamin D receptors are highly expressed by monocytes and by B and T lymphocytes, which modulate immune cell function. Thus, vitamin D deficiency is primarily associated with increased susceptibility to, and severity of, many infectious diseases. More specifically, it is associated with low concentrations of the pro-inflammatory cytokine IL-6, which plays a significant role in COVID-19–induced acute respiratory distress syndrome. In addition, low 25-hydroxyvitamin D concentrations may increase COVID-19 mortality.⁴ Taken together, studies indicate that sunlight improves immunity by increasing vitamin D production and thus may be useful for the treatment of COVID-19.

The environmental survival of viruses such as SARS-CoV-1 depends on factors, including sunlight, humidity, and temperature. On the other hand, 90% of SARS-CoV-2 is inactivated every 6.8 minutes in simulated saliva and every 14.3 minutes in culture media exposed to simulated sunlight (representative of summer at 40° N latitude). Indeed, during summer, 11 to 34 minutes of midday sunlight can inactivate $\geq 90\%$ of SARS-CoV 2 viruses in most locations.⁵

In conclusion, sunlight modulates immune system function by inducing vitamin D production.

elSSN: 2383-4625

Received: 15 March 2021 Revised: 13 April 2021 Accepted: 20 April 2021

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How to cite this article:

Khazdair MR, Goren A. Can sunlight affect COVID-19 outcomes? What is the available evidence?. Clin Exp Emerg Med 2021;8(3):249-250. https://doi. org/10.15441/ceem.21.045

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It also reduces androgen sensitivity and lowers TMPRSS2 expression. Considering that SARS-CoV-2 are dependent on TMPRSS2 for infectivity, sunlight may be useful in the fight against SARS-CoV-2.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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