The outbreak of SARS-CoV2 has been declared a global pandemic by the WHO. With no standard of care for the treatment of COVID-19, there is an urgent need to identify therapies that may be effective in treatment. Here we provide a systematic review of the available literature and an impetus for further research on the use of Gal-3 inhibitors in the treatment of COVID-19.

Additionally, a key domain in the spike protein of β-coronaviridae, a genus which includes SARS-CoV2, is nearly identical in morphology to human Gal-3. These spike proteins are critical for the virus’ entry into host cells. Recent evidence has implicated the development of cytokine release syndrome (CRS) as the major cause of fatality in COVID-19 patients, with elevated levels of IL-6 and TNF-α observed in patients. Inhibitors of the small molecule Gal-3 have been shown to reduce the levels of IL-6, IL-1β, and TNF-α. We propose a dual mechanism by which Gal-3 inhibition may be beneficial in the treatment of COVID-19, both suppressing the host inflammatory response and impeding viral attachment to host cells.

Further research into the role of extracellular sialic acids in SARS-CoV2 attachment is necessary to fully understand the role of Gal-3 inhibition in antiviral therapy.