

## Different Potential Therapies Discovered for COVID-19

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### DESCRIPTION

The COVID-19 outbreak, which was responsible for the world's fear in late 2019, was brought on by the SARS-CoV-2 coronavirus, a single-stranded RNA virus with spike-like glycoprotein projections. Wuhan, People's Republic of China, was the site of the virus's initial detection in December 2019 [1]. The coronavirus has been dubbed COVID-19 by the World Health Organization (WHO). Although the coronavirus's official source has not been identified, bats and snakes are thought to be potential hosts.

Convalescent serum or plasma as a frontline therapy became less common as improved antibiotics, antivirals, and vaccinations were developed. There are numerous antibodies against the pathogens in the sera of the infection-recovered patients. In this technique of treatment, the patient's serum is administered to a patient who has recently contracted the same virus, allowing the recipient's particular antibodies to neutralise the pathogen [2, 3]. The genetically modified receptor known as the Chimeric Antigen Receptor (CAR) is frequently utilized to treat different malignancies. Cells that exhibit virus antigens can be precisely targeted by engineered NK cells that produce CAR molecules. Since NK cells are crucial in the immune system's antiviral response to SARS-CoV-2, CAR-NK modified cells have been suggested as a fresh method of treating COVID-19. A target antigen that can be used to create CAR-NK cells that are resistant to SARS-CoV-2 is ACE-2. Phase I/II clinical research study NCT04324996 was started to examine the effectiveness of COVID-19 pneumonia treatment using commercially available NKG2D-ACE2 CAR-NK cells [4]. Type 1 DCs (DC-1) function as antigen-presenting cells and as part of the innate immune system to exercise their antiviral immune response.

Although the use of modified DCs in cancer therapy is currently quite popular, it is also conceivable to use them to treat infectious diseases. By expressing NKG2D, DCs can stimulate NK cells. However, it is believed that the main mechanism contributing to the development of respiratory inflammation and lung tissue destruction in ARDS is the hypersecretion of IL-6. The

proinflammatory effects of patients with severe COVID-19 could be reduced by applying DC-blocking medications and employing modified DCs [5,6] Type 1 Macrophages (M1), which promote inflammation, and type 2 Macrophages (M2), which inhibit inflammation, are the two subtypes of macrophages. Usually, *in vitro* conditions could be used to produce these cells from monocytes. Similar to this, in the *in vivo* setting, circulating monocytes penetrate organs and undergo macrophage differentiation. By secreting proinflammatory cytokines including IL-6 and IL-1, M1 macrophages help cause significant inflammation during the COVID-19 pandemic. Macrophages can be altered in two different ways to control the hyperinflammatory state. The first strategy might involve modifying M1 macrophages to secrete less proinflammatory cytokines, and the second strategy might involve using M2 macrophages to reduce lung inflammation. Further research on macrophage therapy may be undertaken in light of the use of macrophages in the treatment of COVID-19.

### CONCLUSION

Given the rising numbers of verified cases and fatalities, COVID-19 transmission is still uncontrollable as of this writing. Due of COVID-19's new characteristics, the pharmaceutical and scientific research and development companies are under unprecedented pressure to expedite the development of treatments and vaccines. Although the area is continuously changing, the treatment approaches covered in the manuscript reflect the state of the art at the time of writing. Combination therapy has been endorsed for the pandemic's important COVID-19 patient care. In the future, additional specialised randomised, controlled clinical trials will be required to ascertain the most efficient evidence-based COVID-19 management and treatment strategies.

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