

All of these therapeutic approaches have revealed both in vitro and in vivo anti-CoV potential. Although in vitro research carried out with these therapeutics showed efficacy, most need appropriate support from randomized animal or human trials. Therefore, they might be of limited applicability and require trials against SARS-CoV-2 to gain practical usefulness. The binding of SARS-CoV-2 with ACE2 leads to the exacerbation of pneumonia as a consequence of the imbalance in the renin-angiotensin system (RAS). The virus-induced pulmonary inflammatory responses may be reduced by the administration of ACE inhibitors (ACEI) and angiotensin type-1 receptor (AT1R) (207).

Several investigations have suggested the use of small-molecule inhibitors for the potential control of SARS-CoV infections. Drugs of the FDA-approved compound library were screened to identify four small-molecule inhibitors of MERS-CoV (chlorpromazine, chloroquine, lopinavir, and lopemir) that inhibited viral replication. These compounds also hinder SARS-CoV and human CoVs (208). Therapeutic strategies involving the use of specific antibodies or compounds that neutralize cytokines and their receptors will help to restrain the inflammatory responses. Such drugs acting specifically in the respiratory tract will help to