

PEDIATRICS

**Clinical and epidemiological characteristics of children with COVID-19**

*Pediatrics* <https://doi.org/10.1542/peds.2020-0702> (2020)

Compared with adults, children infected with COVID-19 experience mild disease; however, younger children are vulnerable to the disease and may experience some of the more severe outcomes.

COVID-19 is a severe respiratory disease caused by SARS-CoV-2. Understanding whether children are affected differently from adults is important for clinical and containment strategies.

Dong et al. analyzed data from 2,143 reported cases of children with COVID-19 reported to the Chinese Center for Disease Control and Prevention. They found that children of all ages are susceptible, without significant sex differences, but that infants in particular had severe outcomes. There was also evidence of human-to-human transmission of the disease. HS

<https://doi.org/10.1038/s41591-020-0846-z>

VIRAL ENTRY

**Functionally assessing coronavirus entry**

*Nature Microbiology* <https://doi.org/10.1038/s41564-020-0688-y> (2020)

Screening of the receptor-binding domain of lineage B coronaviruses identifies their ability to infect cells of different species, their receptor usage and other boundaries to entry.

Coronaviruses can cross the species barrier into humans, and one of these lineage

B coronaviruses, SARS-CoV-2, is currently causing the global pandemic of COVID-19. Further understanding of the process through which these viruses infect human cells would aid in preparing for future potential outbreaks.

Letko et al. have developed a rapid, cost-effective screening platform for coronavirus entry into human cells that requires only the synthesis of the receptor-binding domain and have screened all published lineage B coronaviruses. They identify some lineage B coronaviruses that are able to enter human cells but find that some are challenged by the human protease. They confirm that SARS-CoV-2 uses the human ACE2 receptor to enter cells. HS

<https://doi.org/10.1038/s41591-020-0847-y>

EPIDEMIOLOGY

**Spread of SARS-CoV-2**

*Science* <https://doi.org/10.1126/science.aba9757> (2020)

*Science* <https://doi.org/10.1126/science.abb3221> (2020)

The outbreak of SARS-CoV-2, which causes the respiratory disease termed COVID-19, started in Wuhan, China, and is now a pandemic. An understanding of the prevalence and contagiousness of the disease, and of whether the strategies used to contain it to date have been successful, is important for understanding future containment strategies.

One strategy for containment of SARS-CoV-2 is restriction of travel. After 23 January, 2020, long-range travel restrictions in China were imposed by shutting down airports. Chinazzi et al. have used a metapopulation-level disease-transmission model to predict the effects of the travel limitations on the spread of the epidemic.

They have found that these measures only modestly affected the epidemic's trajectory.

Li et al. have used analysis of reported infections within China along with mobility data to infer the epidemiology of SARS-CoV-2. Their analyses indicate that before the travel restrictions, 86% of infections were undocumented. Importantly, undocumented infections were the infection source for 79% of documented cases, thus further indicating that containment will be challenging. HS

<https://doi.org/10.1038/s41591-020-0850-3>

VIROLOGY

**Virological assessment of SARS-CoV-2**

*Nature* <https://doi.org/10.1038/s41586-020-2196-x> (2010)



Credit: Iryna Veklich / Moment / Getty

Virological assessment of SARS-CoV-2 identifies evidence for active viral replication in the throat.

COVID-19 is an acute respiratory-tract infection caused by a coronavirus related to that which caused severe acute respiratory syndrome (SARS). Both use a virus receptor predominantly expressed in the lung, and this aspect is thought to have limited the contagion of SARS.

A research group in Germany has analyzed viral RNA production, isolated active virus and followed the seroconversion of nine people with COVID-19 who were known to have contracted the infection from an index case. The individuals presented diverse symptoms including those of a mild upper-respiratory-tract infection. Infectious virus was isolated from throat- and lung derived-samples but not from stool, blood or urine samples; viral clearance did not occur directly after seroconversion. The data suggest active viral replication in the throat as well as the lungs, thus providing direct implications for infection containment. HS

<https://doi.org/10.1038/s41591-020-0848-x>

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CLINICAL TRIALS

**Lopinavir-ritonavir in severe COVID-19**

*N. Engl. J. Med.* <https://doi.org/10.1056/NEJMoa2001282> (2020)

A randomized, controlled, open-label trial of lopinavir-ritonavir in patients with severe COVID-19 shows no benefit for the primary endpoint beyond standard care but shows benefit for some secondary endpoints.

Currently, there are no effective antiviral treatments against the novel coronavirus SARS-CoV-2, the cause of the respiratory illness termed COVID-19. During the outbreak of severe acute respiratory syndrome (SARS) in 2003, in vitro testing identified lopinavir, an HIV aspartate protease inhibitor, to have activity against this virus.

Cao et al. carried out a randomized, controlled, open-label trial for lopinavir-ritonavir (ritonavir helps to stabilize lopinavir) in 199 hospitalized patients with severe COVID-19, of whom 99 were assigned to the treatment group, and 100 received the standard of care. The authors found no benefit of lopinavir-ritonavir time to clinical improvement beyond the standard of care, although lopinavir-ritonavir was found to have benefit for some secondary endpoints, the safety of the treatment was confirmed, and future trials will verify the results. HS

<https://doi.org/10.1038/s41591-020-0849-9>