DHS SCIENCE AND TECHNOLOGY

# Master Question List for COVID-19 (caused by SARS-CoV-2)

## Weekly Report

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For comments or questions related to the contents of this document, please contact the DHS S&T Hazard Awareness & Characterization Technology Center at HACTechnologyCenter@hq.dhs.gov.

Homeland Security

Science and Technology

## FOREWORD

The Department of Homeland Security (DHS) is paying close attention to the evolving Coronavirus Infectious Disease (COVID-19) situation in order to protect our nation. DHS is working very closely with the Centers for Disease Control and Prevention (CDC), other federal agencies, and public health officials to implement public health control measures related to travelers and materials crossing our borders from the affected regions.

Based on the response to a similar product generated in 2014 in response to the Ebolavirus outbreak in West Africa, the DHS Science and Technology Directorate (DHS S&T) developed the following "master question list" that quickly summarizes what is known, what additional information is needed, and who may be working to address such fundamental questions as, "What is the infectious dose?" and "How long does the virus persist in the environment?" The Master Question List (MQL) is intended to quickly present the current state of available information to government decision makers in the operational response to COVID-19 and allow structured and scientifically guided discussions across the federal government without burdening them with the need to review scientific reports, and to prevent duplication of efforts by highlighting and coordinating research.

The information contained in the following table has been assembled and evaluated by experts from publicly available sources to include reports and articles found in scientific and technical journals, selected sources on the internet, and various media reports. It is intended to serve as a "quick reference" tool and should not be regarded as comprehensive source of information, nor as necessarily representing the official policies, either expressed or implied, of the DHS or the U.S. Government. DHS does not endorse any products or commercial services mentioned in this document. All sources of the information provided are cited so that individual users of this document may independently evaluate the source of that information and its suitability for any particular use. This document is a "living document" that will be updated as needed when new information becomes available.

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## Infectious Dose – How much agent will make a healthy individual ill?

### What do we know?

**The human infectious dose of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is unknown by all exposure routes. Studies from other animal models are used as surrogates for humans. Based on primate models, the inhalation median infectious dose ($ID_{50}$) in humans is likely less than 10,000 PFU, and possibly less than 1,000 PFU.**

*Non-human primates*

- A total dose of approximately 700,000 plaque-forming units (PFU) of the novel coronavirus SARS-CoV-2 infected cynomolgus macaques via combination intranasal and intratracheal exposure ($10^6$ $TCID_{50}$ total dose).[479] Macaques did not exhibit clinical symptoms, but shed virus from the nose and throat.[479]
- Rhesus and cynomolgus macaques showed mild to moderate clinical infections at doses of $4.75x10^6$ PFU (SARS-CoV-2 delivered through several routes), while common marmosets developed mild infections when exposed to $1.0x10^6$ PFU intranasally.[351]
- Rhesus macaques are effectively infected with SARS-CoV-2 via the ocular conjunctival and intratracheal route at a dose of approximately 700,000 PFU ($10^6$ $TCID_{50}$).[141] Rhesus macaques infected with 2,600,000 $TCID_{50}$ of SARS-CoV-2 by the intranasal, intratracheal, oral and ocular routes combined recapitulate moderate human disease.[406]
- African green monkeys replicate aspects of human disease, including severe pathological symptoms (exposed to 500,000 PFU via intranasal and intratracheal routes),[612] mild clinical symptoms (aerosol exposures between 5,000 and 16,000 PFU),[225] and acute respiratory distress syndrome (ARDS), with small particle aerosol exposure doses as low as 2,000 PFU.[55]
- Aerosol exposure of three primate species (African green monkeys, cynomolgus macaques, and rhesus macaques) via a Collison nebulizer resulted in mild clinical disease in all animals with doses between 28,700 and 48,600 PFU.[268]

*Rodents*

- Golden Syrian hamsters exposed to 80,000 $TCID_{50}$ (~56,000 PFU) via the intranasal route developed clinical symptoms reminiscent of mild human infections (all hamsters infected).[520] In a separate study, immunosuppressed Golden Syrian hamsters showed severe clinical symptoms (including death) after exposure to 100-10,000 PFU via intranasal challenge.[62]
- Golden Syrian hamsters infected with 100,000 PFU intranasally exhibited mild clinical symptoms and developed neutralizing antibodies,[96] and were also capable of infecting individuals in separate cages. In another study, older hamsters had more severe symptoms and developed fewer neutralizing antibodies than younger hamsters.[425]
- Mice genetically modified to express the human ACE2 receptor (transgenic hACE2 mice) were inoculated intranasally with 100,000 $TCID_{50}$ (~70,000 PFU), and all mice developed pathological symptoms consistent with COVID-19.[37]
- Transgenic (hACE2) mice became infected after timed aerosol exposure (36 $TCID_{50}$/minute) to between 900 and 1080 $TCID_{50}$ (~630-756 PFU). All mice (4/4) exposed for 25-30 minutes became infected, while no mice (0/8) became infected after exposure for 0-20 minutes (up to 720 $TCID_{50}$, ~504 PFU).[38] Key methodological details (e.g., particle size, quantification of actual aerosol dose) are missing from the study's report.
- Transgenic (hACE2) mice exposed intranasally to 400,000 PFU of SARS-CoV-2 develop typical human symptoms.[540]

*Other animal models*

- Ferrets infected with 316,000 $TCID_{50}$[282] or 600,000 $TCID_{50}$[468] of SARS-CoV-2 by the intranasal route show similar symptoms to human disease.[282, 468] Uninfected ferrets in direct contact with infected ferrets test positive and show disease as early as 2 days post-contact.[282] In one study, direct contact was required to transfer infection between ferrets,[282] however, transmission without direct contact was found in another study.[468]
- In a ferret study, 1 in 6 individuals exposed to $10^2$ PFU via the intranasal route became infected, while 12 out of 12 individuals exposed to $>10^4$ PFU became infected.[490]
- Domestic cats exposed to 100,000 PFU of SARS-CoV-2 via the intranasal route developed severe pathological symptoms including lesions in the nose, throat, and lungs.[518] In a separate study, infected cats showed no clinical signs, but were able to shed virus and transmit to other cats.[57]

*Related Coronaviruses*

- The infectious dose for severe acute respiratory syndrome coronavirus 1 (SARS-CoV-1) in mice is estimated to be between 67-540 PFU (average 240 PFU, intranasal route).[136, 138]
- Genetically modified mice expressing DPP4 exposed intranasally to doses of Middle East respiratory syndrome coronavirus (MERS-CoV) between 100 and 500,000 PFU show signs of infection. Infection with higher doses result in severe syndromes.[15, 119, 321, 656]

### What do we need to know?

**Identifying the infectious dose for humans by the various routes through which we become infected is critical to the effective development of computational models to predict risk, develop diagnostics and countermeasures, and effective decontamination strategies. Animal studies are a plausible surrogate.**

- Human infectious dose by aerosol, surface contact (fomite), fecal-oral routes, and other potential routes of exposure
- Most appropriate animal model(s) to estimate the human infectious dose for SARS-CoV-2
- Does exposure dose determine disease severity?

## Transmissibility – How does it spread from one host to another? How easily is it spread?

### What do we know?

**SARS-CoV-2 is passed easily between humans, likely through close contact with relatively large droplets and possibly through smaller aerosolized particles.**

- As of 7/28/2020, pandemic COVID-19 has caused at least 16,495,309 infections and 654,327 deaths[261] in 188 countries and territories.[85, 508, 601] There are 4,294,770 confirmed COVID-19 cases across all 50 US states, with 148,056 deaths.[261]
- Initial high-quality estimates of human transmissibility ($R_0$) range from 2.2 to 3.1,[367, 434, 473, 620, 655] though recent estimates suggest that early transmission rates were higher.[495] Transmission rates can vary substantially among neighboring populations.[202, 582] The majority of new infections come from relatively few infectious individuals.[12, 161]
- SARS-CoV-2 is believed to spread through close contact and droplet transmission,[90] with fomite transmission[264] and close-contact aerosol transmission likely.[22, 63, 212, 399] On 7/9/2020, the WHO acknowledged that aerosol transmission is plausible, and could not be ruled out in all cases.[603]
- SARS-CoV-2 replicates in the upper respiratory tract,[237] and infectious virus is detectable in throat and lung tissue for at least 8 days.[607] Respiratory fluids from severely ill patients contained higher viral RNA loads than respiratory fluids from mildly ill patients,[659] but similar viral RNA loads have been found in asymptomatic and symptomatic individuals.[310]
- Contamination of patient rooms with aerosolized SARS-CoV-2 in the human respirable range (0.25-2.5 µm) indicates the potential for airborne transmission.[344] Viral RNA was detected up to 4 meters from ICU patient beds.[215] Infectious virus has been found in aerosol samples from COVID-19 patient hospital rooms, primarily in small particles (<1µm).[497]
- SARS-CoV-2 may be spread by conversation and exhalation.[8, 319, 498, 531] A preliminary study in China detailing a restaurant-associated outbreak supports transmission via aerosol.[329] Contact tracing in Japan has identified clusters associated with large gatherings in bars, restaurants, music festivals, and other social activities involving close contact.[642] Clusters of COVID-19 cases tend to come from indoor locations such as bars,[312] and offices.[435]
- Experimentally infected ferrets were able to transmit SARS-CoV-2 to other ferrets through the air (ferrets in an adjacent enclosure, separated by 10 cm).[469] Similar results have been documented in transgenic mice.[38]
- At least one case of vertical transmission has been confirmed from mother to infant prior to birth,[568] though most evidence suggests this is rare.[102, 107, 110, 506, 549, 632, 638]
- SARS-CoV-2 RNA has been found in semen from both clinically symptomatic and recovered cases,[320] but the potential for sexual transmission is unknown. Infectious SARS-CoV-2 has been cultured from patient feces[625] and urine.[538]
- Older children (>10 years old) appear to transmit SARS-CoV-2 as frequently as adults, while younger children (<10 years old) appear to transmit infection less often.[436] These estimates, however, were generated during school closures, and may underestimate the risk of infection from school-age children.

**Individuals can transmit SARS-CoV-2 to others before they have symptoms.**

- Individuals may be infectious for 1-3 days prior to symptom onset,[587] and culturable virus has been found up to 6 days prior to symptom onset.[30] Pre-symptomatic[56, 289, 530, 537, 629, 658] or asymptomatic[36, 243, 356] patients can transmit SARS-CoV-2, and asymptomatic individuals shed virus for as long as mildly symptomatic individuals.[347] At least 12% of all cases are estimated to be due to asymptomatic transmission.[153] It has been estimated that 23-56% of infections may be caused by pre-symptomatic transmission.[79, 230, 341] Individuals are most infectious before symptoms begin and within 5 days of symptom onset,[109] and pre-symptomatic individuals contribute to environmental contamination.[262]
- Attack rates of the virus are higher among household members than casual contacts.[67, 515] The attack rate ranges from 11%,[49] 16%,[325] and 38%[481] of household members, with rates increasing with age.[481] The attack rate for children is low in households with an adult COVID-19 case.[528, 643] Individuals transmit infection to household members before they exhibit symptoms at least as often as they do after symptoms develop.[265] Transmission rates are high in confined areas such as prisons.[493]

**Undetected cases play a major role in transmission, and most cases are not reported.[647]**

- Models suggest up to 86% of early COVID-19 cases in China were undetected, and these infections were the source for 79% of reported cases.[324] Models estimate that the true number of cases may be approximately 11 times greater than the reported number of cases in the UK,[634] and 5 to 10 times greater than the reported number of cases in the US.[266, 489, 521]

**Individuals who have recovered clinically, but test positive, appear unable to transmit COVID-19.**

- Individuals who have clinically recovered from COVID-19, but later show PCR positive tests, are likely not infectious.[277]

### What do we need to know?

**The relative contribution of different routes of transmission, such as close contact and droplet transmission versus aerosol transmission and contaminated objects and surfaces (fomites), is unknown and requires additional research.**

- Capability of SARS-CoV-2 to be transmitted by contact with fomites (phones, doorknobs, surfaces, clothing, etc.)
- Is sexual transmission possible?
- Is it possible to determine the route by which someone became infected by the clinical presentation or progression of disease?

## Host Range – How many species does it infect? Can it transfer from species to species?

### What do we know?

**SARS-CoV-2 is closely related to other coronaviruses circulating in bats in Southeast Asia. Previous coronaviruses have passed through an intermediate mammal host before infecting humans, but the identity of the SARS-CoV-2 intermediate host is unknown.**

- Early genomic analysis indicates similarity to SARS-CoV-1,[663] with a suggested bat origin.[120, 663]
- Positive samples from the South China Seafood Market strongly suggests a wildlife source,[92] though it is possible that the virus was circulating in humans before the disease was associated with the seafood market.[42, 121, 627, 639]
- Analysis of SARS-CoV-2 genomes suggests that a non-bat intermediate species is responsible for the beginning of the outbreak.[478] The identity of the intermediate host remains unknown.[328, 335, 337]
- Viruses similar to SARS-CoV-2 were present in pangolin samples collected several years ago,[303] and pangolins positive for coronaviruses related to SARS-CoV-2 exhibited clinical symptoms such as cough and shortness of breath.[327] Additionally, there is evidence of vertical transmission in pangolins, suggesting circulation in natural populations.[327]
- However, a survey of 334 pangolins did not identify coronavirus nucleic acid in 'upstream' market chain samples, suggesting that positive samples from pangolins may be the result of exposure to infected humans, wildlife or other animals within the wildlife trade network. These data suggest that pangolins are incidental hosts of coronaviruses.[313] Additional research is needed to identify whether pangolins are a natural host of SARS-COV-2-related coronaviruses.

**SARS-CoV-2 uses the same receptor for cell entry as the SARS-CoV-1 coronavirus that circulated in 2002/2003.**

- Experiments show that SARS-CoV-2 Spike (S) receptor-binding domain binds the human cell receptor (ACE2) stronger than SARS-CoV-1,[615] potentially explaining its high transmissibility. The same work suggests that differences between SARS-CoV-2 and SARS-CoV-1 Spike proteins may limit the therapeutic ability of SARS antibody treatments.[615]
- Modeling of SARS-CoV-2 Spike and ACE2 proteins suggests that SARS-CoV-2 can bind and infect human, bat, civet, monkey and swine cells.[572] Host range predictions based on structural modeling, however, are difficult,[187] and additional animal studies are needed to better define the host range.
- In vitro experiments suggest a broad host range for SARS-CoV-2, with more than 44 potential animal hosts, based on viral binding to species-specific ACE2 orthologs.[342] The host range is predicted to be limited primarily to mammals.
- Genetic and protein analysis of primates suggests that African and Asian primates are likely more susceptible to SARS-CoV-2, while South and Central American primates are likely less susceptible.[383] Identifying the SARS-CoV-2 host range is important for identifying animal reservoirs.
- Changes in proteolytic cleavage of the Spike protein can also affect cell entry and animal host range, in addition to receptor binding.[385]

**To date, ferrets, mink, hamsters, cats, deer mice, and primates have been shown to be susceptible to SARS-CoV-2 infection. It is unknown whether these animals can transmit infection to humans.**

- Animal model studies suggest that Golden Syrian hamsters, primates, and ferrets may be susceptible to infection.[96, 282] In the Netherlands, farmed mink developed breathing and gastrointestinal issues, which was diagnosed as SARS-CoV-2 infection.[1] It is thought that an infected mink has transmitted SARS-CoV-2 to a human.[298] Golden Syrian hamsters are able to infect other hamsters via direct contact and close quarters aerosol transmission.[520]
- Domestic cats are susceptible to infection with SARS-CoV-2 (100,000-520,000 PFU via the intranasal route[518] or a combination of routes[217]), and can transmit the virus to other cats via droplet or short-distance aerosol.[518] Dogs exposed to SARS-CoV-2 produced anti-SARS-CoV-2 antibodies[57] but exhibited no clinical symptoms.[518, 525]
- Deer mice can be experimentally infected with SARS-CoV-2 via intranasal exposure to $10^5$ $TCID_{50}$ of virus, and are able to transmit the virus to uninfected deer mice through direct contact.[208]
- Wild cats (tigers)[586] can be infected with SARS-CoV-2, although their ability to spread to humans is unknown.[368, 652] Two cases of SARS-CoV-2 infection have been confirmed in pet domestic cats.[84]
- Ducks, chickens, and pigs remained uninfected after experimental SARS-CoV-2 exposure (30,000 CFU for ducks and chickens, 100,000 PFU for pigs, all via intranasal route).[518] There is currently no evidence that SARS-CoV-2 infects livestock.[250]
- Pigs and chickens were not susceptible to SARS-CoV-2 infection when exposed to an intranasal dose of $10^5$ $TCID_{50}$ (~70,000 PFU).[503] Fruit bats and ferrets were susceptible to this same exposure.[503]
- Chicken, turkey, duck, quail, and geese were not susceptible to SARS-CoV-2 after experimental exposures.[536]

### What do we need to know?

**Several animal models have been developed to recreate human-like illness, though to date they have been infected with high dose exposures. Lower dose studies may better replicate human disease acquisition.**

- What is the intermediate host(s)?
- Can infected animals transmit to humans (e.g., pet cats to humans)?
- Can SARS-CoV-2 circulate in animal reservoir populations, potentially leading to future spillover events?

## Incubation Period – How long after infection do symptoms appear? Are people infectious during this time?

### What do we know?

**The majority of individuals develop symptoms within 14 days of exposure. For most people, it takes at least 2 days to develop symptoms, and on average symptoms develop 5 days after exposure. Incubating individuals can transmit disease for several days before symptom onset. Some individuals never develop symptoms but can still transmit disease.**

- The incubation period of COVID-19 is between 5[309] and 6[588] days.[633] Fewer than 2.5% of infected individuals show symptoms sooner than 2 days after exposure.[309]
- There is evidence that younger (<14) and older (>75) individuals have longer COVID-19 incubation periods, creating a U-shaped relationship between incubation period length and patient age.[290]
- Individuals can test positive for COVID-19 even if they lack clinical symptoms.[36, 95, 213, 548, 658]
- Individuals can be infectious while asymptomatic,[90, 483, 548, 658] and asymptomatic and pre-symptomatic individuals have similar amounts of virus in the nose and throat compared to symptomatic patients.[30, 281, 667]
- Peak infectiousness may be during the incubation period, one day before symptoms develop.[230] Infectious virus has been cultured in patients up to 6 days before the development of symptoms.[30]
- Infectious period is unknown, but possibly up to 10-14 days.[6, 324, 508]
- Asymptomatic individuals are estimated to be infectious for a median of 9.5 days.[241]
- On average, there are approximately 4[153] to 7.5[323] days between symptom onset in successive cases of a single transmission chain (i.e., the serial interval). Based on data from 339 transmission chains in China, the mean serial interval is between 4.6[633] and 5.29 days.[152]
- The serial interval of COVID-19 has declined substantially over time as a result of increased case isolation,[18] meaning individuals tend to transmit virus for less time.
- Children are estimated to shed virus for 15 days on average, with asymptomatic individuals shedding virus for less time (11 days) than symptomatic individuals (17 days).[353]
- Most hospitalized individuals are admitted within 8-14 days of symptom onset.[661]
- Asymptomatic and mildly ill patients who test positive for SARS-CoV-2 take less time to test negative than severely ill patients.[315]
- Patients infected by asymptomatic or young (<20 years old) individuals may take longer to develop symptoms than those infected by other groups of individuals.[588]
- Viral RNA loads in the upper respiratory tract tend to peak within a few days of symptom onset and become undetectable approximately two weeks after symptoms begin.[570] The duration of the infectious period is unknown,[570] though patients can test positive for SARS-CoV-2 viral RNA for extended periods of time, particularly in stool samples.

### What do we need to know?

**The incubation period is well-characterized. Patients may be infectious, however, for days before symptoms develop.**

- What is the average infectious period during which individuals can transmit the disease?
- How infectious are asymptomatic and pre-symptomatic individuals compared to mildly, moderately, or severely ill patients?

## Clinical Presentation – What are the signs and symptoms of an infected person?

### What do we know?

**Approximately 40% of cases are asymptomatic.[423] Most symptomatic cases are mild, but severe disease can be found in any age group.[4] Older individuals and those with underlying conditions are at higher risk of serious illness and death.**

- Between 16% and 58% of patients are asymptomatic throughout the course of their infection.[69, 310, 315, 395, 414, 542, 554]
- Most symptomatic COVID-19 cases are mild (81%, n=44,000 cases).[548, 604] Initial COVID-19 symptoms include fever (87.9% overall, but only 44-52% present with fever initially),[27, 213] cough (67.7%),[213] fatigue, shortness of breath, headache, and reduced lymphocyte count.[91, 98, 242] Chills, muscle pain, headache, sore throat, and loss of taste or smell[441, 631] are also possible COVID-19 symptoms.[91] GI symptoms are present in approximately 9% of patients,[480] but may be more common in severe cases.[274] Neurological symptoms such as agitation,[231] loss of coordination,[369] and stroke[564] may present with COVID-19,[439] may be more common in severe cases,[128] and neurological involvement (e.g., encephalitis) can be seen in brain tissue on autopsy.[569] Ocular issues[622] and skin lesions[188] may also be symptoms of COVID-19.[58] There are concerns that COVID-19 can lead to new-onset diabetes.[485]
- COVID-19 symptoms like fatigue and shortness of breath commonly persist for weeks[547] to months[77] after initial onset.
- Complications include acute respiratory distress syndrome (ARDS, 17-29% of hospitalized patients, leading to death in 4-15% of cases),[106, 242, 574] pneumonia,[430] cardiac injury (20%),[519] secondary infection, kidney damage,[28, 534] arrhythmia, sepsis, stroke (1.6% of hospitalized patients),[388] and shock.[213, 242, 574, 661] Most deaths are caused by respiratory failure or respiratory failure combined with heart damage.[484] Half of hospitalized COVID-19 patients show abnormal heart scans.[155]
- Clinically, COVID-19 appears to present as three different phenotypes, including ARDS.[477]
- Approximately 15% of hospitalized patients are classified as severe,[213, 548] and approximately 5% of patients are admitted to the ICU.[213, 548] Patient deterioration can be rapid.[207] The survival rate of patients requiring mechanical ventilation varies widely (e.g., 35%,[249] 70%,[32] 75.5%[470]). Higher SARS-CoV-2 viral RNA load on admission (measured by RT-PCR cycle threshold values) have been associated with greater risk of intubation and death.[361] Approximately 42% of ICU patients die from COVID-19, though the rate is variable across studies.[29]
- Recent evidence suggests that SARS-CoV-2 may attack blood vessels in the lung, leading to clotting complications and ARDS.[11, 565] Clotting may be associated with severely ill COVID-19 patients[284] and those with ARDS,[128] and affects multiple human organ systems.[461] COVID-19 patients should be monitored for possible thrombosis.[318] In autopsies of several COVID-19 patients, there was evidence of diffuse alveolar damage (DAD)[501] and increased blood clotting.[502]
- COVID-19 patients undergoing unrelated surgical procedures have high levels of postoperative complications and death.[410]

**The case fatality rate (CFR) varies substantially by patient age and underlying comorbidities.[605]**

- Cardiovascular disease, obesity,[13, 447] hypertension,[650] diabetes, and respiratory conditions all increase the CFR.[548, 661] Hypertension and obesity are common in the US[190] and contribute to mortality.[28, 432]
- Individuals >60 are at higher risk of death, and the CFR for individuals >85 is between 10% and 27%.[548, 661] In a small study, men exhibited more severe symptoms and died at higher rates than women.[263] The effect of comorbidities on the likelihood of severe symptoms is higher for men.[386]

**Additional studies on vulnerable subpopulations are required.**

- Black, Asian, and Minority Ethnic (BAME) populations acquire SARS-CoV-2 infection at higher rates than other groups[429] and are disproportionately represented in hospitalized populations.[190, 452] African American communities contribute disproportionately to the number of deaths in the US.[233, 392] Hospitalization rates in Native American, Hispanic, and Black populations are 4-5 times higher than those in non-Hispanic white populations.[87] In the US, Hispanic and Black patients tend to die at younger ages than white patients.[614]
- Pregnant women appear to develop severe symptoms at the same rate as the general population[105, 272, 645] or at a slightly elevated rate.[159] Severe symptoms in pregnant women may be associated with underlying conditions such as obesity.[345] There is some evidence that rates of stillbirth and preterm delivery have increased during the COVID-19 pandemic,[384] though these instances have not been conclusively linked to maternal COVID-19 infection.[280] More work is needed.

**Children are susceptible to COVID-19,[149] though generally show milder[100, 352] or no symptoms.**

- Between 21-28% of children may be asymptomatic.[352, 437, 454] Most symptomatic children present with mild or moderate symptoms,[206, 437] with few exhibiting severe or clinical illness.[619]
- Severe symptoms in children are possible[340] and more likely in those with complex medical histories[513] or underlying conditions such as obesity.[644] Infants are susceptible to illness,[150] and infant deaths have been recorded.[65, 352]
- The WHO[600] and US CDC[260] have issued case definitions for a rare condition in children (termed Pediatric Multi-System Inflammatory Syndrome)[200] linked to COVID-19 infection.[474]

### What do we need to know?

**The true case fatality rate is unknown, as the exact number of cases is uncertain. Testing priorities and case definitions vary by location.**

- How does the asymptomatic fraction vary across age groups?
- How does COVID-19 contribute to pregnancy complications?

## Protective Immunity – How long does the immune response provide protection from reinfection?

### What do we know?

**Infected patients show productive immune responses, but the duration of any protection is unknown. Initial evidence suggests that the neutralizing antibody response does not last more than a few months, though this varies by severity.**

- In a small comparison series (n=74), both asymptomatic and mildly symptomatic individuals showed reductions in IgG antibody levels 8 weeks after infection.[347] The half-life of one antibody (IgG) has been estimated at 36 days in COVID-19 patients.[248] The correlation to long-term immunity is unknown.
- In a larger study (n=175), most patients developed neutralizing antibodies within 10-15 days after disease onset. Elderly patients had significantly higher neutralizing antibody titers than younger patients.[617] In a separate study, elderly patients also showed higher viral loads than younger patients.[552] Approximately half of infected individuals on an aircraft carrier developed detectable neutralizing antibody responses to SARS-CoV-2.[441]
- In a study of 285 COVID-19 patients, 100% developed antiviral immunoglobulin-G within 19 days of symptom onset.[346] The neutralizing ability of these antibodies was not tested.[346] In a smaller in vitro study (n=23 patients), levels of antibodies (immunoglobulins M and G) were positively correlated with SARS-CoV-2 neutralizing ability.[552] In a smaller study of 44 patients, plasma from 91% demonstrated SARS-CoV-2 neutralizing ability, appearing ~8 days after symptom onset.[541]
- A small subset of COVID-19 patients in China (8%) did not develop a serological response to infection, though the potential for reinfection in these patients is unknown.[617] Similarly, between 16.7% (for IgG) and 51.7% (for IgM) of patients in a separate study did not exhibit any immune response, in terms of production of those two types of antibodies.[544]
- In a study of 221 COVID-19 patients, levels of two types of antibodies (IgM and IgG) were not associated with the severity of symptoms.[240] However, in a smaller study, patients with severe disease showed stronger antibody responses than those with non-severe symptoms.[552] Severely ill individuals develop higher levels of neutralizing antibodies[334] and greater T-cell response frequencies[509] than mildly symptomatic or asymptomatic individuals.
- The early recovery phase of COVID-19 patients is characterized by inflammatory immune response.[591]
- Two studies identified key components of the adaptive immune system ($CD4^+$ T cells) in the majority of recovered COVID-19 patients, and these cells reacted to SARS-CoV-2 Spike protein.[60, 210] These studies also identified Spike protein responses in $CD4^+$ T cells of ~30-40% of unexposed patients,[210] suggesting some cross-reactivity between other circulating human coronaviruses and SARS-CoV-2.[60, 210] Long-lasting T-cell responses have been seen in SARS-CoV-1 patients, and T-cell cross-reactivity reactivity between other coronaviruses and SARS-CoV-2 suggest additional immune protection.[311] The strength and duration of any T-cell derived protection is currently unknown.
- Children do not appear to be protected from SARS-CoV-2 infection or severe COVID-19 symptoms by historical exposure to seasonal coronaviruses.[511]

**Currently, there is no evidence that recovered patients can be reinfected with SARS-CoV-2.**

- Two studies suggest limited reinfection potential in macaques. In the first, two experimentally infected macaques were not capable of being reinfected 28 days after their primary infection resolved.[142] In the second, rhesus macaques exposed to different doses of SARS-CoV-2 via the intranasal and intratracheal routes ($10^4$ – $10^6$ PFU) developed pathological infection and were protected upon secondary challenge 35 days after initial exposure.[97]
- Ferrets infected with $10^2$-$10^4$ PFU were protected from acute lung injury following secondary challenge with SARS-CoV-2 28 days after initial exposure, but they did exhibit clinical symptoms such as lethargy and ruffled fur.[490] Cats exposed to SARS-CoV-2 after initial recovery did not shed virus, suggesting some protective effect of primary infection.[57]
- According to the WHO, there is no evidence of re-infection with SARS-CoV-2 after recovery.[308]
- Patients can test positive via PCR for up to 37 days after symptoms appear,[661] and after recovery and hospital discharge.[304]
- **The strength and duration of any immunity after initial COVID-19 infection is unknown.[19, 597]**
- In a small study (n=65), 95% of patients developed neutralizing antibodies within 8 days of symptom onset,[510] but neutralizing antibody titers declined substantially when assayed after 60 days.[510] Individuals with more severe infections developed higher neutralizing antibody levels that persisted longer than those with asymptomatic or mild infections.[510] Protective antibody immunity may depend on the severity of initial infection, and may not persist for more than a few months, which is consistent with observations in other human coronaviruses.
- In a 35-year study of 10 men, immunity to seasonal coronaviruses waned after one year.[156] Reinfection was observed between one and three years after initial infection.[156]
- Previous studies on coronavirus immunity suggest that neutralizing antibodies may wane after several years.[72, 621]

### What do we need to know?

**As the pandemic continues, long-term monitoring of immune activity and reinfection status is needed.**

- How long does the immune response last? Is there evidence of waning immunity?
- Can humans become reinfected, or are reports of reinfection vestiges from initial infection?
- How do different components of the immune response contribute to long-term protection?
- How does initial disease severity affect the type, magnitude, and timing of any protective immune response?

## Clinical Diagnosis – Are there tools to diagnose infected individuals? When during infection are they effective?

### What do we know?

**Diagnosis relies on identifying the genetic signature of the virus in patient nose, throat, or sputum samples, or by identifying SARS-CoV-2 antibodies in individuals exposed to the virus. Confirmed cases are still underreported.[227]**

- The US CDC has expanded testing criteria to include symptomatic patients at clinician discretion.[40]
- PCR protocols and primers have been widely shared internationally.[82, 123, 323, 517, 596, 602]
- A combination of pharyngeal (throat) RT-PCR and chest tomography is recommended,[466] particularly when results from one test are inconclusive.[294] A single throat swab detects 78.2% of infections, and duplicate tests identify 86.2% of infections.[466] PCR tests using saliva are at least as effective as those using nasopharyngeal swabs.[103, 624] Evaluation of seven RT-PCR diagnostic test kits in China showed high overall accuracy, but some variability among test kits.[573]
- Nasal and pharyngeal swabs may be less effective as diagnostic specimens than sputum and bronchoalveolar lavage fluid,[579] although evidence is mixed.[607] Combination RT-PCR and serology (antibody) testing may increase the ability to diagnose patients with mild symptoms, or identify patients at higher risk of severe disease.[657] Assays targeting antibodies against the nucleocapsid protein (N) instead of the Spike protein (S) of SARS-CoV-2 may improve detection.[66]
- The timing of diagnostic PCR tests impacts results. The false-negative rate for RT PCR tests is lowest between 7 and 9 days after exposure, and PCR tests are more likely to give false-negative results before symptoms begin (within 4 days of exposure) and more than 14 days after exposure.[301] The role of temporal changes in immunological response and variation of diagnostic test results based on symptom severity warrants additional study.[291]
- Diagnostic test results from at-home, mid-nasal swabs were comparable to clinician-conducted nasopharyngeal swabs, though false-negatives were observed in individuals with low viral titer.[373]
- Asymptomatic individuals have a higher likelihood of testing negative for a specific antibody (IgG) compared to symptomatic patients, potentially due to lower viral loads (as measured by RT-PCR).[590]
- The FDA issued an Emergency Use Authorization for an antigen-based diagnostic assay, limited to use in certified laboratories (clinical laboratory improvement amendments, CLIA).[166]
- The FDA released an Emergency Use Authorization enabling laboratories to develop and use tests in-house for patient diagnosis.[171] Tests from the US CDC are available to states.[82, 90] Rapid test kits have been produced by universities and industry.[47, 53, 131, 169, 567] Home tests are being developed, though they cannot be used for diagnosis and have not been approved by the FDA.[407-408, 433] The US CDC is developing serological tests to assess SARS-CoV-2 exposure prevalence.[270]
- The CRISPR-Cas12a system is being used to develop fluorescence-based COVID-19 diagnostic tests.[245]
- US deaths due to COVID-19 have been underreported by up to 35% (March – April).[611] In New York City, up to 5,293 (22%) of period-specific excess deaths are unexplained and could be related to the pandemic.[421]
- Immunological indicators[34, 160, 229, 244, 449, 539, 575] and fasting blood glucose levels[578] may help differentiate between severe and non-severe cases, and decision-support tools for diagnosing severe infections have been developed.[618]
- Individuals who test positive again after hospital discharge were more likely to have had short hospital stays, be younger than 18, and have had mild or moderate COVID-19 symptoms.[641]

**Validated serological (antibody) assays are being used to help determine who has been exposed to SARS-CoV-2. Serological evidence of exposure does not indicate immunity.**

- Repeated serological testing is necessary to identify asymptomatic[451] and other undetected patients.[496] Exclusively testing symptomatic healthcare workers is likely to exclude a large fraction of COVID-19 positive personnel.[533]
- Research has shown high variability in the ability of tests (ELISA[420] and lateral flow assays) by different manufacturers to accurately detect positive and negative cases (sensitivity and specificity, respectively).[307, 592] The FDA has excluded several dozen serological diagnostic assays based on failure to conform to updated regulatory requirements.[168] Researchers have designed a standardized ELISA procedure for SARS-CoV-2 serology samples.[285]
- Meta-analysis suggests that lateral flow assays (LFIA) are less accurate than ELISA or chemiluminescent methods (CLIA), but that the target of serological studies (e.g., IgG or IgM) does not affect accuracy.[331] Most reported serological studies suffer from bias related to selected patients, limiting their applicability to general populations.[331]
- The false positive rate of serological assays may account for a substantial portion of reported exposures,[45] particularly if the true proportion of positive patients is low.

### What do we need to know?

**In general, PCR tests appear to be sensitive and specific, though confirmation via chest CT is recommended. The sensitivity and specificity of serological testing methods is variable.**

- How many serological tests need to be done to obtain an accurate picture of underlying exposure?
- What fraction of exposed individuals fail to develop antibody responses that are the target of serological assays?
- What is the relationship between disease severity and the timing of positive serological assays?

## Medical Treatments – Are there effective treatments?

### What do we know?

**Treatment for COVID-19 is primarily supportive care,[213, 372] and no single standard of care exists. Drug trials are ongoing. Remdesivir shows promise for reducing symptom duration[44] and mortality[198] in humans.**

- Remdesivir can reduce the duration of symptoms in infected individuals, from 15 days to 11 days on average (compared to controls).[44] Remdesivir received an Emergency Use Authorization from FDA[413] and is recommended for use in the EU.[606]
- A press release reports that remdesivir reduced 14-day mortality in COVID-19 patients across racial and ethnic groups when compared to standard of care alone, though concerns exist regarding the appropriate control group.[198]
- A randomized clinical trial of remdesivir found no significant clinical benefits (n=237 patients), but the trial ended early.[584]

**Hydroxychloroquine is associated with risk of cardiac arrhythmias and provides limited to no clinical benefit at this time.**

- Hydroxychloroquine does not benefit mild-moderate COVID-19 cases,[81] was associated with adverse cardiac events in severely ill patients,[279] and showed no efficacy as treatment or pre-exposure prophylaxis in non-human primates.[365] Several large clinical trials have stopped administering hydroxychloroquine due to lack of efficacy.[224, 234, 409] Other existing studies have found no benefit of hydroxychloroquine (with or without azithromycin)[20, 99, 104, 193, 236, 287, 360, 362, 527, 545] as well as cardiac side effects[46, 115, 196, 258, 363, 387, 500] and elevated risk of mortality.[360, 475] Hydroxychloroquine does not protect individuals from infection either before[194] or after exposure.[59, 394] The FDA revoked its EUA for the drug on 6/15/20.[165]
- Initial results purporting benefits of hydroxychloroquine and azithromycin[192] have been called into question. One small clinical trial (n=62) suggests that hydroxychloroquine can reduce recovery time compared to control group,[108] but lacks key methodological details.[108] A small retrospective study (n=48) found benefits to hydroxychloroquine, though details on patient study population selection were limited.[637] A larger retrospective study (n=2,541) found that hydroxychloroquine reduced mortality.[31] However, concerns still exist over the patient selection protocol and the time-course of the study.[314]

**Dexamethasone may significantly reduce mortality in severely ill and ventilated patients.**

- Dexamethasone is associated with substantial reductions in mortality for patients receiving mechanical ventilation, and smaller benefits for those receiving supplemental oxygen.[235] Dexamethasone did not reduce mortality in patients who did not need oxygen or mechanical ventilation.[235]

**Other pharmaceutical interventions are being investigated.**

- Several studies of methylprednisolone suggest clinical benefits in severely ill patients (e.g., reduction in ventilator use, mortality), but have not been tested separately from other standard-of-care treatments.[124, 366, 486, 494, 499] Providing anti-inflammatory treatments in the first few days of hospital admission may be beneficial.[391] Other corticosteroids are also being studied and show some evidence of clinical improvement (ventilator-free days),[116] though the benefits of glucocorticoids may depend heavily on patient inflammation (beneficial if high, detrimental if low).[278]
- There is evidence for efficacy of several interferon-based treatments, including interferon beta-1b,[247] interferon beta-1a,[135] and interferon alpha-2b.[444] In these studies, interferons were generally administered with other treatments. A press release suggests that an inhaled interferon beta reduced the need for mechanical ventilation.[450]
- Observational studies have found benefits of tocilizumab[176, 226, 482, 529, 628] in severe COVID-19 patients, and Phase II trial results show limited reductions in mortality.[446] Tocilizumab efficacy may depend on C-reactive protein levels[185, 371] and may be more beneficial when administered early.[205, 403] Tocilizumab has been associated with reduced risk of severe illness[25, 286] and death,[403] but also an increased risk of secondary (non-COVID-19) infection.[214] Many studies of tocilizumab suffer from non-random patient assignments and the confounding influence of concomitant treatments, despite showing some clinical benefits.[201, 228, 400, 532, 553] Randomized clinical trials are needed. Other trials have found benefits of itolizumab[51] but no consistent benefits from sarilumab.[463]
- Limited, preliminary evidence supports the efficacy of favipiravir,[101] intravenous immunoglobulin,[75] baricitinib,[73] ivermectin,[458] leflunomide,[239] and pidotimod.[556] Lenzilumab, a monoclonal antibody, showed benefits to oxygenation levels in severely ill patients (n=12).[546] There is no clinical benefit from combination ritonavir/lopinavir.[74, 209, 330] The kinase inhibitor ruxolitinib may help to reduce symptom duration and mortality.[76] The anticoagulant heparin is being used to mitigate risks of pulmonary embolism.[160] Systemic anticoagulant use was associated with reduced mortality rates in severely ill patients.[431] Anakinra has showed some evidence of clinical benefit in small observational studies.[80]
- Trials are ongoing to evaluate the efficacy of a blood cleaning device used to reduce inflammatory neutrophils.[636]
- Passive antibody therapy (convalescent serum)[78] is being given to patients,[170] appears safe,[271] and several small trials (<50 patients) suggest benefits from convalescent patient plasma for infected patients.[162, 339, 359, 370, 492, 514, 516] Some trial data suggest benefits of plasma in terms of reduced hospitalization time,[10] though evidence is mixed.[147, 197, 322]

### What do we need to know?

**Additional information on treatment efficacy is required, particularly from large randomized clinical trials.**

- Do monoclonal antibodies exhibit any efficacy in human trials?
- Are there treatments that reduce the development of severe symptoms when administered early?
- Do androgen levels in males alter disease severity?[204, 398, 571]

## Vaccines – Are there effective vaccines?

### What do we know?

**Work is ongoing to develop and produce a SARS-CoV-2 vaccine (e.g., Operation Warp Speed).[41, 218, 221-223, 412] Early results are being released, but evidence should be considered preliminary until larger trials are completed.**

*Phase III Trials (testing for efficacy):*

- Moderna has begun Phase III trials of its COVID-19 vaccine, which will target 30,000 participants.[397]
- University of Oxford's ChAdOx1 candidate (now called AZD1222) has begun Phase II/III human trials.[427]
- Sinovac will begin Phase III trials of its CoronaVac candidate in healthcare professionals.[522]
- Sinopharm will begin Phase III trials of its inactivated SARS-CoV-2 vaccine candidate.[39]

*Phase II Trials (initial testing for efficacy, continued testing for safety):*

- CanSino's Ad5-nCoV adenovirus vaccine candidate has undergone Phase II human trials.[332] China has given approval to vaccinate members of its military with the product.[338] Phase II trial results showed positive immune responses in most patients, but also indicated that prior infection with circulating adenoviruses may inhibit vaccine efficacy.[664]
- Moderna has begun its Phase II trial of mRNA-1273 with 600 participants.[375]
- Sinovac reported no severe adverse events among 600 Phase II participants given their CoronaVac candidate (inactivated virus), and 90% of patients developed neutralizing antibodies 14 days after administration.[524]
- Sinopharm reported neutralizing antibody development in all 1,120 participants given its inactivated virus vaccine (two times, 14 days apart) with no severe adverse events.[333]
- Inovio has registered for a Phase II trial of their INO-4800 DNA vaccine candidate.[254]

*Phase I Trials (initial testing for safety)*:

- mRNA vaccines developed by several groups are currently being tested in Phase I trials, including CureVac (candidate is CVnCoV),[129] the Chinese Academy of Military Sciences (ARCoV),[140] BioNTech and Pfizer (BNT162 program),[448] Moderna (mRNA-1273),[396] and Arcturus (ARCT-021).[26] Data from a Phase I trial of Moderna's mRNA-1273 candidate suggest that the vaccine is well-tolerated by human subjects, and induces an antibody response against SARS-CoV-2.[257] Preliminary Phase I/II results for BioNTech's BNT162b1 mRNA candidate show mild side effects in low dose groups, and patients generated neutralizing antibodies at 21 days post vaccination.[405]
- Adenovirus-based vaccines from several groups are being tested in Phase I trials, including CanSino (Ad5-nCoV),[665] Johnson and Johnson (Ad.26-COV2-S),[267] the University of Oxford (ChAdOx1, now called AZD1222),[562] and Gamaleya Research Institute of Epidemiology and Microbiology (Gam-COVID-Vac Lyo).[599] Phase I trial results for the CanSino vaccine (Ad5-nCoV) showed few severe adverse reactions in humans within 28 days of follow-up and appreciable antibody and T-cell responses.[665] In Phase I/II trials, the ChAdOx-1 COVID-19 (AZD1222) vaccine showed a tolerable safety profile and most recipients developed positive T-cell and neutralizing antibody responses.[184]
- Several groups have developed heat-inactivated vaccine candidates, including the Chinese Academy of Medical Sciences,[507] the Beijing Institute of Biological Products,[453] the Wuhan Institute of Biological Products,[623] Immunitor LLC (V-Sars),[382] and Sinovac Biotech (CoronaVac).[523] Sinovac Biotech has reported that their inactivated virus vaccine (CoronaVac) shows protective effects in rhesus macaques, particularly at high vaccine doses.[189]
- Several groups are developing recombinant subunit vaccines, including Vaxine Pty (Covax-19),[566] Clover Biopharmaceuticals (SCB-2019),[381] Novavax (NVX-CoV2373),[377] and the Chinese Academy of Sciences (RBD-Dimer).[348] The University of Queensland has started Phase I trials of its UQ vaccine candidate, which uses Spike protein subunits.[455]
- Several groups are testing DNA vaccines in Phase I trials, including Inovio (INO-4800),[253] Genexine (GX-19)[195] and AnGes (AG0301-COVID19).[24] Results from Inovio's INO-4800 show no serious adverse side effects and high immunogenicity.[253]
- Imperial College London is beginning Phase I/II trials of their RNA vaccine candidate, LNP-nCoVsnRNA.[418]
- Shenzhen Geno-Immune Medical Institute is testing its aAPC[380] and lentiviral (LV-SMENP-DC)[378] vaccines.
- Symvivo Corporation (Canada) will begin a Phase I trial of its oral bacTRL-Spike vaccine candidate.[376]
- Aivita will begin a Phase Ib/II clinical trial of its DC-ATA candidate, comprised of dendritic cells and SARS-CoV-2 antigens.[379]
- Medicago will begin the Phase I trials of their vaccine, a plant-derived virus-like-particle candidate.[374]
- Phase I/II trials are beginning for vaccine candidates from Zydus Cadila (ZyCoV-D, DNA plasmid)[668] and Baharat (Covaxin, inactivated rabies virus used as carrier for SARS-CoV-2 proteins).[164]
- Kentucky BioProcessing will begin Phase I/II trials of their KBP-COVID-19 candidate based on a tobacco plant platform.[52]

*Non-target vaccines*

- The potential benefits of non-SARS-CoV-2 vaccines, such as *Bacillus* Calmette-Guerin (BCG), are under investigation.[163, 512]

### What do we need to know?

**Published results from randomized clinical trials (Phase I – III) are needed.**

- Safety and efficacy of vaccine candidates in humans, particularly from Phase III trials
- Length of any vaccine-derived immunity
- Evidence for vaccine-derived enhancement (immunopotentiation)

## Non-pharmaceutical Interventions – Are public health control measures effective at reducing spread?

### What do we know?

**Broad-scale control measures such as stay-at-home orders are effective at reducing transmission.**

- Social distancing and other policies are estimated to have reduced COVID-19 spread by 44% in Hong Kong[127] and reduced spread throughout China,[296, 300, 302, 349, 364, 577] Europe,[191, 276] and the US.[295] Restrictive lockdowns in China are estimated to have reduced disease transmission within only a few days[666] by reducing contacts.[648] In China, modeling suggests that a one-day delay in implementing control measures increased the time needed to curtail an outbreak by 2.4 days.[151] In the US, each day of delay in emergency declarations and school closures was associated with a 5-6% increase in mortality.[635]
- Modeling demonstrates that multifaceted restrictions and quarantines in China reduced the $R_0$ of SARS-CoV-2 from greater than 3 to less than 1 between January 23 and February 5.[428] Additionally, movement restrictions and other control measures helped limit the amount of time where community transmission was possible (i.e., $R_0 > 1$).[649]
- A US county-level model found that shelter in place orders (SIPOs) and restaurant and bar closures were associated with large reductions in exponential growth rate of cases.[125] School closures and cancellation of large gatherings had smaller effects.[125] Similarly, researchers found that a larger number of public health interventions in place was strongly associated with lower COVID-19 growth rates in the next week.[273] Individual behaviors such as wearing face coverings and practicing social distancing have been associated with reduced risk of COVID-19 infection.[441]
- Mobility[181, 306] and physical contact rates[259] decline after public health control measures are implemented. Mobility reductions in the US have been associated with significant reductions in COVID-19 case growth.[35] Mobile phone data has shown that social distancing and reductions in both non-essential visits to stores and overall movement distance led to lower transmission rates of SARS-CoV-2.[402]
- Modeling suggests that travel restrictions delay peak prevalence by only a few days but do not limit epidemic size.[17]
- Models indicate that a combination of school closures, work restrictions, and other measures are required to effectively limit transmission.[175, 292] School closures alone appear insufficient.[256, 302]
- Non-pharmaceutical interventions in China did not reduce transmission equally across all populations.[428]
- Two modeling studies identified large reductions in transmission due to country lockdowns[183] and other social distancing measures,[238] with substantial variation in the efficacy of particular policies in different countries.[183, 238]
- Contact tracing to identify infected individuals reduces the amount of time infectious individuals can transmit disease in a population and increases the time between successive cases.[49] Robust contact tracing and case finding may be needed to control COVID-19 in the US, but requires additional resources.[585] In South Korea, early implementation of rapid contact tracing, testing, and quarantine was able to reduce the transmission rate of COVID-19.[537] Contact tracing combined with high levels of testing may limit COVID-19 resurgence once initial social distancing policies are relaxed.[16, 177] Contact tracing is likely to be more effective in combination with measures such as expanded testing and physical distancing.[299]

**Research is needed to help plan for easing of restrictions. Testing is critical, and synchronized interventions may help.**

- Relaxing public health interventions is projected to increase cases and deaths.[132, 559] As of 7/28/2020, 28 US states are experiencing increases in the average daily rate of new confirmed cases, and 24 US states are experiencing increases in the average daily rate of new COVID-19 deaths (for the prior 14 days).[417]
- Modeling suggests that optimal control policies involve quickly quarantining infected individuals, and that periods of social distancing or lock-down may be effective in reducing overall exposure from asymptomatic or unconfirmed cases.[555] Testing is critical to balancing public health and economic costs.[555] Rolling interventions, whereby social distancing measures are put into place every few weeks, may keep healthcare demand below a critical point.[634] Undetected cases, can lead to elevated risk of re-emergence after restrictions are lifted, highlighting the need for robust testing strategies.[219]
- Synchronizing public health interventions and lockdowns across US state lines may reduce the total number of interventions necessary to eliminate transmission as COVID-19 cases continue to resurge.[487]
- Modeling indicates that COVID-19 is likely to become endemic in the US population, with regular, periodic outbreaks, and that additional social or physical distancing measures may be required for several years to keep cases below critical care capacity in absence of a vaccine or effective therapeutic.[283] Results depend on the duration of immunity after exposure.[283]
- Balancing control measures to maintain $R_0$ below 1 may be more efficient than allowing $R_0$ to increase above 1.[316]
- The WHO has released guidelines on public health strategy,[595] and Johns Hopkins released a report outlining how to re-open certain categories of activities (e.g., schools, restaurants, events) while reducing COVID-19 risk.[476]
- Surveys indicate that the majority of Americans were complying with non-pharmaceutical interventions.[130] In the US, mask use increased after recommendations from the White House Task force and CDC.[179]

### What do we need to know?

**As different US states have implemented differing control measures at various times, a comprehensive analysis of social distancing efficacy has not yet been conducted.**

- What are plausible options for relaxing social distancing and other intervention measures without resulting in a resurgence of COVID-19 cases?

## Environmental Stability – How long does the agent live in the environment?

### What do we know?

**SARS-CoV-2 can persist on surfaces for at least 3 days and on the surface of a surgical mask for up to 7 days depending on conditions. If aerosolized intentionally, SARS-CoV-2 is stable for at least several hours. The seasonality of COVID-19 transmission is unknown. SARS-CoV-2 on surfaces is inactivated rapidly with sunlight.**

*SARS-CoV-2 Data*

- In simulated saliva on stainless steel surfaces, SARS-CoV-2 exhibits negligible decay over 60 minutes in darkness, but loses 90% of infectivity every 6.8-12.8 minutes, depending on the intensity of simulated UVB radiation levels.[462]
- The Department of Homeland Security (DHS) developed a data-based model for SARS-CoV-2 decay on inert surfaces (stainless steel, ABS plastic and nitrile rubber) at varying temperature and relative humidity. This model estimates virus decay in the absence of exposure to direct sunlight.[146]
- SARS-CoV-2 can persist on plastic and metal surfaces between 3 days (21-23°C, 40% RH)[561] and 7 days (22°C, 65% RH). Infectious virus can be recovered from a surgical mask after 7 days (22°C, 65% RH).[114]
- At room temperature (22°C), SARS-CoV-2 remains detectable (via plaque assay) on paper currency for up to 24 hours, on clothing for up to 4 hours, and on skin for up to 96 hours.[220] Persistence is reduced with warmer temperatures (37°C), and enhanced at colder temperatures (4°C).[220]
- SARS-CoV-2 persists for less than 3 days within the pages of library books, and for less than 1 day on the exterior of book and DVD covers.[3]
- Both temperature and humidity contribute to SARS-CoV-2 survival on nonporous surfaces, with cooler, less humid environments facilitating survival (stainless steel, ABS plastic, and nitrile rubber; indoors only; simulated saliva matrix).[54]
- Experimental studies using SARS-CoV-2 aerosols (1.78-1.96 µm mass median aerodynamic diameter in artificial saliva matrix) found that simulated sunlight rapidly inactivates the virus, with 90% reductions in infectious concentration after 6 minutes in high-intensity sunlight (similar to mid-June) and 19 minutes in low-intensity sunlight (similar to early March or October).[505] In dark conditions, the half-life of aerosolized SARS-CoV-2 is approximately 86 minutes in simulated saliva matrix.[505] Humidity had no significant impact on aerosolized virus survival.[505]
- DHS developed a tool for estimating the decay of airborne SARS-CoV-2 in different environmental conditions.[145]
- SARS-CoV-2 has an aerosol half-life of 2.7 hours (without sunlight, particles <5 µm, tested at 21-23°C and 65% RH).[561]
- Research suggests SARS-CoV-2 retains infectivity as an aerosol for up to 16 hours in appropriate conditions (23°C, 53% RH, no sunlight).[173]
- SARS-CoV-2 is susceptible to heat treatment (70°C) but can persist for at least two weeks at refrigerated temperatures (4°C).[114, 460]
- SARS-CoV-2 genetic material (RNA) was detected in symptomatic and asymptomatic cruise ship passenger rooms up to 17 days after cabins were vacated. The infectiousness of this material is not known.[401]
- In a preliminary study, SARS-CoV-2 stability was enhanced when present with bovine serum albumin, which is commonly used to represent sources of protein found in human sputum.[438]
- No strong evidence exists showing a reduction in transmission with seasonal increase in temperature and humidity.[355] Modeling suggests that even accounting for potential reductions in transmission due to weather and behavioral changes, public health interventions will still need to be in effect to limit COVID-19 transmission.[389]
- A recent study determined that approximately 0.1-1% of initial SARS-CoV-2 inoculated on plastic, stainless steel, glass, ceramics, wood, latex gloves, cotton, paper, and surgical masks remained after 48 hours.[343] Approximately 0.1% of SARS-CoV-2 remains in fecal matter after 6 hours.[343] Approximately 0.1% of SARS-CoV-2 in human urine persists after 4-5 days.[343]
- RNA in clinical samples collected in viral transport medium is stable at 18-25°C or 2-8°C for up to 21 days without impacting real-time RT-PCR results.[526] RNA in clinical samples is also stable at 4°C for up for 4 weeks with regard to quantitative RT-PCR testing (given that the sample contains 5,000 copies/mL). Separately, storage of RNA in PBS at room-temperature (18-25°C) resulted in unstable sample concentrations.[443]
- SARS-CoV-2 was detectable on wooden chopsticks used by symptomatic and asymptomatic COVID-19 patients, though sample sizes were small and no efforts were made to isolate infectious virus.[354]

### What do we need to know?

**Additional testing on SARS-CoV-2, as opposed to surrogate viruses, is needed to support initial estimates of stability. Tests quantifying infectivity, rather than the presence of viral RNA, are needed.**

- Duration of SARS-CoV-2 infectivity via fomites and surfaces (contact hazard)
- Stability of SARS-CoV-2 on PPE (e.g., Tyvek)
- Stability of SARS-CoV-2 in food (to date, no known infections from contaminated food).[593]

## Decontamination – What are effective methods to kill the agent in the environment?

### What do we know?

**Soap and water, as well as common alcohol and chlorine-based cleaners, hand sanitizers, and disinfectants are effective at inactivating SARS-CoV-2 on hands and surfaces.**

*SARS-CoV-2*

- Alcohol-based hand rubs are effective at inactivating SARS-CoV-2.[297]
- Chlorine bleach (1%, 2%), 70% ethanol and 0.05% chlorhexidine are effective against live virus in lab tests.[113]
- Twice-daily cleaning with sodium dichloroisocyanurate decontaminated surfaces in COVID-19 patient hospital rooms.[422]
- EPA has released a list of SARS-CoV-2 disinfectants, but most solutions were not tested on SARS-CoV-2.[14] Several solutions have been tested against SARS-CoV-2 and found to be effective, including those based on para-chloro-meta-xylenol, salicylic acid, and quaternary ammonium compounds.[252] Two of these products, Lysol Disinfectant Spray (EPA Reg No. 777-99) and Lysol Disinfectant Max Cover Mist (EPA Reg No. 777-127) have specifically been approved for SARS-CoV-2 decontamination.[357]
- Oral antiseptic rinses used in pre-procedural rinses for dentistry containing povidone-iodine (PVP-I) are effective decontaminants of SARS-CoV-2, with 15-sec and 30-sec contact times completely inactivating SARS-CoV-2 at concentrations above 0.5% in lab tests.[50]
- Holder pasteurization of donor breast milk spiked with SARS-CoV-2 rendered the virus inactive, demonstrating that standard decontamination procedures are effective at reducing risk of COVID-19 risk in infants via donor breast milk.[558]
- Efforts are ongoing to create paint-on surfaces that can rapidly inactivate SARS-CoV-2.[43]
- Researchers have identified four methods capable of decontaminating N95 respirators while maintaining physical integrity (fit factor): UV radiation, heating to 70°C, and vaporized hydrogen peroxide (VHP).[178] Ethanol (70%) was associated with loss of physical integrity.[178]
- Hydrogen peroxide vapor (VHP) can repeatedly decontaminate N95 respirators.[471] Devices capable of decontaminating 80,000 masks per day have been granted Emergency Use Authorization from the FDA.[166]
- The FDA has issued an Emergency Use Authorization for a system capable of decontaminating ten N95 masks at a time using devices already present in many US hospitals.[61]
- Respirator decontamination methods such as VHP appear to maintain filtration efficiency after repeated decontamination cycles.[442] Several decontamination methods, including VHP, moist heat, and UVC, are capable of decontaminating N95 respirators for 10-20 cycles without loss of fit or filtration efficiency.[7]

*Other Coronaviruses*

- Chlorine-based[599] and ethanol-based[122] solutions are recommended.
- Heat treatment (56°C) is sufficient to kill coronaviruses,[457, 660] though effectiveness depends partly on protein in the sample.[457]
- 70% ethanol, 50% isopropanol, sodium hypochlorite (0.02% bleach), and UV radiation can inactivate several coronaviruses (MHV and CCV).[491]
- Ethanol-based biocides effectively disinfect coronaviruses dried on surfaces, including ethanol containing gels similar to hand sanitizer.[246, 608]
- Surface spray disinfectants such as Mikrobac, Dismozon, and Korsolex are effective at reducing infectivity of the closely related SARS-CoV-1 after 30 minutes of contact.[456]
- Coronaviruses may be resistant to heat inactivation for up to 7 days when stabilized in stool.[550-551]
- Coronaviruses are more stable in matrixes such as respiratory sputum.[154]

### What do we need to know?

**Additional decontamination studies, particularly with regard to PPE and other items in short supply, are needed.**

- What is the minimal contact time for disinfectants?
- Does contamination with human fluids/waste alter disinfectant efficacy profiles?
- How effective is air filtration at reducing transmission in healthcare, airplanes, and public spaces?
- Are landfills and wastewater treatment plants effective at inactivating SARS-CoV-2?
- Is heat or UV decontamination effective to clean N95 masks, respirators and other types of PPE for multi-use?

## PPE – What PPE is effective, and who should be using it?

### What do we know?

**The effectiveness of PPE for SARS-CoV-2 is currently unknown, and data from other related coronaviruses are used for guidance. Healthcare workers are at high risk of acquiring COVID-19, even with recommended PPE.**

- Healthcare worker illnesses[548] demonstrates human-to-human transmission despite isolation, PPE, and infection control.[504] Risk of transmission to healthcare workers is high.[465] Contacts with healthcare workers tend to transmit COVID-19 more often than other casual contacts.[581] Over 50% of US healthcare workers infected with COVID-19 report work in a healthcare setting as their single source of exposure.[68] Hospital-acquired infection rates fell after introduction of comprehensive infection control measures, including expanded testing and use of PPE for all patient contacts.[472] Universal masking policies also reduced the rate of new healthcare worker infections.[580]
- A modeling study suggests that healthcare workers are primarily at risk from droplet and inhalation exposure (compared to contact with fomites), with greater risk while in closer proximity to patients.[269]
- "Healthcare personnel entering the room [of SARS-CoV-2 patients] should use standard precautions, contact precautions, airborne precautions, and use eye protection (e.g., goggles or a face shield)."[88] WHO indicates healthcare workers should wear clean long-sleeve gowns as well as gloves.[598] PPE that covers all skin may reduce exposure to pathogens.[174, 589]
- Respirators (NIOSH-certified N95, EUFFP2 or equivalent) are recommended for those dealing with possible aerosols.[599] Additional protection, such as a Powered Air Purifying Respirator (PAPR) with a full hood, should be considered for high-risk procedures (i.e., intubation, ventilation).[64]
- KN95 respirators are, under certain conditions, approved for use under FDA Emergency Use Authorization.[167] On May 7, the FDA rescinded a number of KN95 models that no longer meet the EUA criteria and are no longer authorized.[172]
- A study suggests that P100 respirators with removable filter cartridges have similar filtration efficiency compared to N95 respirators and could plausibly be used if N95 respirators were in short supply.[440]
- Particular care should be taken with "duckbill" N95 respirators, which may fail fit tests after repeated doffing.[139] Dome-shaped N95 respirators also failed fit tests after extended use.[139]

**Non-medical Masks may be effective at slowing transmission, though data are sparse.**[2, 5]

- On 4/3/2020, the US CDC recommended wearing cloth face masks in public where social distancing measures are difficult to maintain.[89] The WHO recommends that the general population wear non-medical masks when in public settings and when physical distancing is difficult, and that vulnerable populations (e.g., elderly) wear medical masks when close contact is likely.[594] Infected individuals wearing facemasks in the home before the onset of symptoms was associated with a reduction in household transmission.[583]
- Modeling suggests that widespread use of facemasks is effective at reducing transmission.[411] Modeling shows that masks can be effective even when individual mask efficiency is low, though their benefits are maximized when most of the population wears masks.[180] Even minimally effective masks can help avert fatalities when transmission rates are low or decreasing.[157]
- A meta-analysis of SARS-CoV-1, MERS, and COVID-19 transmission events found evidence that wearing face masks and eye protection were each associated with lower risk of transmission.[117] N95 respirators were associated with a larger reduction in transmission risk compared to surgical face masks.[117] Physical distance (>1 or 2 meters) was also associated with lower transmission risk.[117] In a separate meta-analysis, N95 respirators were found to be beneficial for reducing the occurrence of respiratory illness in health care professionals including influenza, though surgical masks were similarly effective for influenza.[419] N95 respirators were associated with large reductions (up to 80%) in SARS-CoV-1 infections.[419]
- Surgical face masks, respirators and homemade face masks may prevent transmission of coronaviruses from infectious individuals (with or without symptoms) to other individuals.[134, 317, 560] Surgical masks were associated with a significant reduction in the amount of seasonal coronavirus (not SARS-CoV-2) expressed as aerosol particles (<5 µm).[317]
- The efficacy of homemade PPE, made with T-shirts, bandanas, or similar materials, is less than standard PPE, but may offer some protection if no other options are available.[118, 133, 467] Some non-standard materials (e.g., cotton, cotton hybrids) may be able to filter out >90% of simulant particles >0.3µm,[288] while other materials (e.g., T-shirt, vacuum cleaner bag, towels) appear to have lower filtration efficacy (~35-62%).[576] Of 42 homemade materials tested, the three with the greatest filtration efficiencies were layered cotton with raised visible fibers.[646]

### What do we need to know?

**Most PPE recommendations have not been made on SARS-CoV-2 data, and comparative efficacy of different PPE for different tasks (e.g., intubation) is unknown. Identification of efficacious PPE for healthcare workers is critical due to their high rates of infection.**

- Is COVID-19 transmitted by the aerosol/airborne route (in droplets and particles <5µm), and if so what is the impact?
- How effective are barriers such as N95 respirators or surgical masks for SARS-CoV-2?
- What is the appropriate PPE for first responders? Airport screeners?
- What are proper procedures for reducing spread and transmission rates in medical facilities?
- How effective are homemade masks at reducing SARS-CoV-2 transmission?

| Forensics – Natural vs intentional use? Tests to be used for attribution. |
|---|
| **What do we know?** |
| **All current evidence supports the natural emergence of SARS-CoV-2 via a bat and possible intermediate mammal species.**<br>• Genomic analysis places SARS-CoV-2 into the beta-coronavirus clade, with close relationship to bat coronaviruses. The SARS-CoV-2 virus is distinct from SARS-CoV-1 and MERS viruses.[148]<br>• Genomic analysis suggests that SARS-CoV-2 is a natural variant and is unlikely to be human-derived or otherwise created by "recombination" with other circulating strains of coronavirus.[21, 663]<br>• Comparing genomes of multiple coronaviruses using machine-learning has identified key genomic signatures shared among high case fatality rate coronaviruses (SARS-CoV-1, SARS-CoV-2, MERS) and animal counterparts.[216] These data further suggest that SARS-CoV-2 emergence is the result of natural emergence and that there is a potential for future zoonotic transmission of additional pathogenic strains to humans.[216]<br>• Deletion mutants were identified at low levels in human clinical samples, suggesting that the PRRA furin cleavage site alone is not fully responsible for human infection, but does confer a fitness advantage in the human host.[610] Additional whole-genome sequencing in humans would help to confirm this finding.<br>• Genomic data support at least two plausible origins of SARS-CoV-2: "(i) natural selection in a non-human animal host prior to zoonotic transfer, and (ii) natural selection in humans following zoonotic transfer."[21] Both scenarios are consistent with the observed genetic changes found in all known SARS-CoV-2 isolates.<br>• Some SARS-CoV-2 genomic evidence indicates a close relationship with pangolin coronaviruses,[609] and data suggest that pangolins may be a natural host for beta-coronaviruses.[335, 337] Genomic evidence suggests a plausible recombination event between a circulating coronavirus in pangolins and bats could be the source of SARS-CoV-2.[326, 626] Emerging studies are showing that bats are not the only reservoir of SARS-like coronaviruses.[653] Additional research is needed.<br>• There are multiple studies showing that the SARS-CoV-2 S protein receptor binding domain, the portion of the protein responsible for binding the human receptor ACE2, was acquired through recombination between coronaviruses from pangolins and bats.[21, 326, 336, 653] These studies suggest that pangolins may have played an intermediate role in the adaptation of SARS-CoV-2 to be able to bind to the human ACE2 receptor. Additional research is needed.<br>• A novel bat coronavirus (RmYN02) has been identified in China with an insertion in the viral furin cleavage site. While distinct from the insertion in SARS-CoV-2, this evidence shows that such insertions can occur naturally.[662]<br>• Additionally, "[…] SARS-CoV-2 is not derived from any previously used virus backbone," reducing the likelihood of laboratory origination,[21] and "[…] genomic evidence does not support the idea that SARS-CoV-2 is a laboratory construct, [though] it is currently impossible to prove or disprove the other theories of its origin."[21]<br>• Work with other coronaviruses has indicated that heparan sulfate dependence can be an indicator of prior cell passage, due to a mutation in the previous furin enzyme recognition motif.[137] |
| **What do we need to know?** |
| **Identifying the intermediate species between bats and humans would aid in reducing potential spillover from a natural source. Wide sampling of bats, other wild animals, and humans is needed to address the origin of SARS-CoV-2.**<br>• What tests for attribution exist for coronavirus emergence?<br>• What is the identity of the intermediate species?<br>• Are there closely related circulating coronaviruses in bats or other animals with the novel PRRA cleavage site found in SARS-CoV-2? |

## Genomics – How does the disease agent compare to previous strains?

### What do we know?

**Current evidence suggests that SARS-CoV-2 accumulates substitutions and mutations at a similar rate as other coronaviruses. Mutations and deletions in specific portions of the SARS-CoV-2 genome have not been linked to any changes in transmission or disease severity, though modeling work is attempting to identify possible changes.**

- There have been no documented cases of SARS-CoV-2 prior to December 2019. Preliminary genomic analyses, however, suggest that the first human cases of SARS-CoV-2 emerged between 10/19/2019 – 12/17/2019.[23, 42, 459]
- Analysis of more than 7,000 SARS-CoV-2 genome samples provides an estimated mutation rate of $6x10^{-4}$ nucleotides per genome per year.[563] The same analysis estimates the emergence of SARS-CoV-2 in humans between October and December 2019.[563] This aligns with the first known human cases in China in early December 2019, in Europe in late December 2019,[144] circulation in the US (Washington State) in February 2020,[613] and circulation in Mexico in March, 2020.[543] In both California[143] and New York City,[203] phylogenetic evidence supports multiple introductions of SARS-CoV-2 from both inside and outside the US.
- Despite evidence of variation in the genome[94] and areas under positive selection,[70] there are no known associations between particular mutations and changes in transmission or virulence.[71] Thus, there is currently no evidence of distinct SARS-CoV-2 phenotypes at this time.[358, 563] Research attempting to define clades or subgroups of SARS-CoV-2 based solely on genomic features has suffered from limited data[640] and sampling bias.[186] In 94 COVID-19 patients, there was no association between viral genotype and clinical severity.[654]
- Phylogenetic and clinical analysis suggests the D614G mutation in the Spike protein is associated with higher rates of SARS-CoV-2 transmission, but no change in clinical severity in infected patients.[293] However, it is difficult to determine whether this mutation is overrepresented due to founder effects, or whether it truly spreads more rapidly than other isolates. Preliminary experimental evidence suggests that this mutation increases infectivity in cell lines, but additional animal model work is needed to confirm the effect of this mutation on transmission.[651]
- Recent analysis of >16,000 genomes of SARS-CoV-2 suggests two major introductions in the US, one associated with the West coast and one with the Eastern portion of the US.[404]
- A genome-wide association study in humans identified two loci corresponding to higher risk of severe COVID-19 (3p.21.31 and 9q34.2), including one associated with blood type.[158] Individuals with type-O blood showed reduced risk of severe disease, while individuals with type-A blood showed an increased risk.[158]
- SARS-CoV-2 is acquiring nucleotide changes at a rate that suggests the virus is undergoing purifying selection (that the genome is stabilizing toward a common genome).[616] Low genetic diversity early in the epidemic suggests that SARS-CoV-2 was capable of jumping to human and other mammalian hosts,[616] and that additional jumps into humans from reservoir species may occur.
- Phylogenetics suggest that SARS-CoV-2 is of bat origin, but is closely related to coronaviruses found in pangolins.[335, 337]
- The SARS-CoV-2 Spike protein, which mediates entry into host cells and is the major determinant of host range, is very similar to the SARS-CoV-1 Spike protein.[350] The rest of the genome is more closely related to two separate bat[350] and coronaviruses found in pangolins.[337]
- An analysis of SARS-CoV-2 sequences from Singapore has identified a large nucleotide (382 bp) deletion in ORF-8.[535] In Arizona, researchers identified an 81-base pair deletion (removing 27 amino acids) in the ORF-7a protein, indicating that mutations can be detected by routine sentinel surveillance. The function of these deletions are unknown at this time.[232]
- A recent report of virus mutations within patients needs more research.[275] Additional analysis of data suggests the results may be due to experimental methods.[199, 630]
- Structural modeling suggests that observed changes in the genetic sequence of the SARS-CoV-2 Spike protein may enhance binding of the virus to human ACE2 receptors.[424] More specifically, changes to two residues (Q493 and N501) are linked with improving the stability of the virus-receptor binding complex.[424] Additionally, structural modeling identified several existing mutations that may enhance the stability of the receptor binding domain, potentially increasing binding efficacy.[426] Infectivity assays are needed to validate the genotypic changes and possible phenotypic results identified in these studies.
- A key difference between SARS-CoV-2 and other beta-coronaviruses is the presence of a polybasic furin cleavage site in the Spike protein (insertion of a PRRA amino acid sequence between S1 and S2).[126]
- The US CDC is launching a national genomics consortium to assess SARS-CoV-2 genomic changes over time.[83]

### What do we need to know?

**Research linking genetic changes to differences in phenotype (e.g., transmissibility, virulence, progression in patients) is needed.**

- Are there similar genomic differences in the progression of coronavirus strains from bat to intermediate species to human?
- Are there different strains or clades of circulating virus? If so, do they differ in virulence?
- What are the mutations in SARS-CoV-2 that allowed human infection and transmission?
- How quickly does the virus mutate and could the changes impact the effectiveness of vaccines or therapeutics?

## Forecasting – What forecasting models and methods exist?

### What do we know?

**There are many groups focused on forecasting cases, hospitalizations, or fatalities due to COVID-19. Each model has its own methods and goals, summarized in this section. An evaluation of model performance is beyond the scope of this document. Assumptions and limitations of each model are detailed at the linked reference.**

*US CDC forecasting*

**The US CDC is hosting an ongoing forecasting initiative, and provides ensemble forecasts based on the arithmetic mean of participating groups.[86]**

- Columbia University Model: Spatially-explicit SEIR model incorporating contact rate reductions due to social distancing. Estimates total cases and risk of healthcare overrun.[488]
- Imperial College London: Week-ahead forecasts of cases, deaths, and transmissibility ($R_0$) at the country-level. Transmissibility estimates used to forecast incidence based on Poisson renewal process.[48]
- Institute of Health Metrics and Evaluation (IHME): Mechanistic SEIR model combined with curve-fitting techniques to forecast cases, hospital resource use, and deaths at the state and country level.[251]
- Los Alamos National Laboratory: Forecasts of state-level cases and deaths based on statistical growth model fit to reported data. Implicitly accounts for effects of social distancing and other control measures.[305]
- Massachusetts Institute of Technology: Mechanistic SEIR model that forecasts cases, hospitalizations, and deaths. Also includes estimates of intervention measures, allows users to project based on different intervention scenarios (e.g., social distancing lasting for 3 vs. 4 weeks).[393]
- Northeastern University: Spatially explicit, agent-based epidemic model used to forecast fatalities, hospital resource use, and the cumulative attack rate (proportion of the population infected) for unmitigated and mitigated scenarios.[415]
- Notre Dame University: Agent-based model forecasting cases and deaths for Midwest states. Includes effectiveness of control measures like social distancing.[445]
- University of California, Los Angeles: Mechanistic SIR model with statistical optimization to find best-fitting parameter values. Estimates confirmed and active cases, fatalities, and transmission rates at the national and state levels.[557]
- University of Chicago: Age-structured SEIR model that accounts for asymptomatic individuals and the effectiveness of social distancing policies. Forecasts only for Illinois.[112]
- University of Geneva: Country-level forecasts of cases, deaths, and transmissibility ($R_0$). Uses statistical models fit to reported data, not mechanistic models.[182]
- University of Massachusetts, Amherst: Aggregation of state and national forecasts to create ensemble model.[464]
- University of Texas, Austin: Machine learning model aimed at identifying links between social distancing measures and changes in death rates. Forecasts fatalities at the state, metropolitan area, and national level. Cannot be used to make projections beyond initial infection wave.[390]
- Youyang Gu: Mechanistic SEIR model coupled with machine learning algorithms to minimize error between predicted and observed values. Forecasts deaths and infections at the state and national level, including 60 non-US countries. Includes effects of public health control efforts.[211]
- Auquan: SEIR model used to forecast deaths and illnesses at the country and state level.[33]
- CovidSim: SEIR model allowing users to simulate the effects of future intervention policies at the state and national level (US only).[111]

*Other forecasting efforts*:

- University of Georgia: Statistical models used to estimate the current number of symptomatic and incubating individuals, beyond what is reported (e.g., "nowcasts"). Available at the state and national level for the US.[93]
- Hospital IQ has a dashboard that forecasts hospital and ICU admissions for each county in the US. Relies in part on IHME forecasts.[255]
- COVID Act Now: State and county-level dashboard focused on re-opening strategies, showing trends in four metrics related to COVID-19 risk (change in cases, total testing capacity, fraction of positive tests, and availability of ICU beds). Fundamentally uses an SEIR model fit to observed data.[416]
- Researchers use a rolling window analysis incorporating uncertainty in the generation time distribution to estimate time-varying transmission rates in US states (the effective reproduction number, $R_{eff}$ or $R_t$).[9]

### What do we need to know?

**Forecasts differ in how they handle public health interventions such as shelter-in-place orders and tracking how methods change in the near future will be important for understanding limitations going forward.**

*Table 1. Definitions of commonly-used acronyms*

| Acronym/Term | Definition | Description |
|---|---|---|
| ACE2 | Angiotensin-converting enzyme 2 | Acts as a receptor for SARS-CoV and SARS-CoV-2, allowing entry into human cells |
| Airborne transmission | Aerosolization of infectious particles | Aerosolized particles can spread for long distances (e.g., between hospital rooms via HVAC systems). Particles generally <5 µm. |
| ARDS | Acute respiratory distress syndrome | Leakage of fluid into the lungs which inhibits respiration and leads to death |
| Attack rate | Proportion of "at-risk" individuals who develop infection | Defined in terms of "at-risk" population such as schools or households, defines the proportion of individuals in those populations who become infected after contact with an infectious individual |
| CCV | Canine coronavirus | Canine coronavirus |
| CFR | Case Fatality Rate | Number of deaths divided by confirmed patients |
| CoV | Coronavirus | Virus typified by crown-like structures when viewed under electron microscope |
| COVID-19 | Coronavirus disease 19 | Official name for the disease caused by the SARS-CoV-2 virus. |
| Droplet transmission | Sneezing, coughing | Transmission via droplets requires relatively close contact (e.g., within 6 feet) |
| ELISA | Enzyme-linked immunosorbent assay | Method for serological testing of antibodies |
| Fomite | Inanimate vector of disease | Surfaces such as hospital beds, doorknobs, healthcare worker gowns, faucets, etc. |
| HCW | Healthcare worker | Doctors, nurses, technicians dealing with patients or samples |
| Incubation period | Time between infection and symptom onset | Time between infection and onset of symptoms typically establishes guidelines for isolating patients before transmission is possible |
| Infectious period | Length of time an individual can transmit infection to others | Reducing the infectious period is a key method of reducing overall transmission; hospitalization, isolation, and quarantine are all effective methods |
| Intranasal | Agent deposited into external nares of subject | Simulates inhalation exposure by depositing liquid solution of pathogen/virus into the nose of a test animal, where it is then taken up by the respiratory system. |
| MERS | Middle-East Respiratory Syndrome | Coronavirus with over 2,000 cases in regional outbreak since 2012 |
| MHV | Mouse hepatitis virus | Coronavirus surrogate |
| Nosocomial | Healthcare- or hospital-associated infections | Characteristic of SARS and MERS outbreaks, lead to refinement of infection control procedures |
| PCR | Polymerase chain reaction | PCR (or real-time [RT] or quantitative [Q] PCR) is a method of increasing the amount of genetic material in a sample, which is then used for diagnostic testing to confirm the presence of SARS-CoV-2 |
| PFU | Plaque forming unit | Measurement of the number of infectious virus particles as determined by plaque forming assay. A measurement of sample infectivity. |
| PPE | Personal protective equipment | Gowns, masks, gloves, and any other measures used to prevent spread between individuals |
| $R_0$ | Basic reproduction number | A measure of transmissibility. Specifically, the average number of new infections caused by a typical infectious individual in a wholly susceptible population. |

| Acronym/Term | Definition | Description |
|---|---|---|
| SARS | Severe Acute Respiratory Syndrome | Coronavirus with over 8,000 cases in global 2002-2003 outbreak |
| SARS-CoV-2 | Severe acute respiratory syndrome coronavirus 2 | Official name for the virus previously known as 2019-nCoV. |
| SEIR | Susceptible (S), exposed (E), infected (I), and resistant (R) | A type of modeling that incorporates the flow of people between the following states: susceptible (S), exposed (E), infected (I), and resistant (R), and is being used for SARS-CoV-2 forecasting |
| Serial interval | Length of time between symptom onset of successive cases in a transmission chain | The serial interval can be used to estimate $R_0$, and is useful for estimating the rate of outbreak spread |
| SIR | Susceptible (S), infected (I), and resistant (R) | A type of modeling that incorporates the flow of people between the following states: susceptible (S), infected (I), and resistant (R), and is being used for SARS-CoV-2 forecasting |
| $TCID_{50}$ | 50% Tissue Culture Infectious Dose | The number of infectious units which will infect 50% of tissue culture monolayers. A measurement of sample infectivity. |
| Transgenic | Genetically modified | In this case, animal models modified to be more susceptible to MERS and/or SARS by adding proteins or receptors necessary for infection |

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