DHS SCIENCE AND TECHNOLOGY

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

Bi-annual Report

July 2023

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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SARS-COV-2 – Master Question List

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FOREWORD

The Department of Homeland Security's (DHS) Science and Technology Directorate (S&T) developed the following "Master Question List" (MQL) that quickly summarizes what is known and what additional information is needed to address such fundamental questions as, "What is the infectious dose?" and "How long does the virus persist in the environment?" This 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 the need to review scientific reports, and to prevent duplication of efforts by highlighting and coordinating research.

INTRODUCTION

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a member of the coronavirus family and is the causative agent of COVID-19. Members of the coronavirus family cause a variety of diseases from head or chest colds to more severe and rare diseases such as severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS). Like other respiratory viruses, coronaviruses spread quickly through droplets expelled when an individual breathes, coughs, sneezes, or speaks. As SARS-CoV-2 continues to spread through populations, genetic changes can accumulate over time and form distinct evolutionary lineages or variants with differing mutation rates, transmissibility, vaccine efficacy, and pathogenicity.
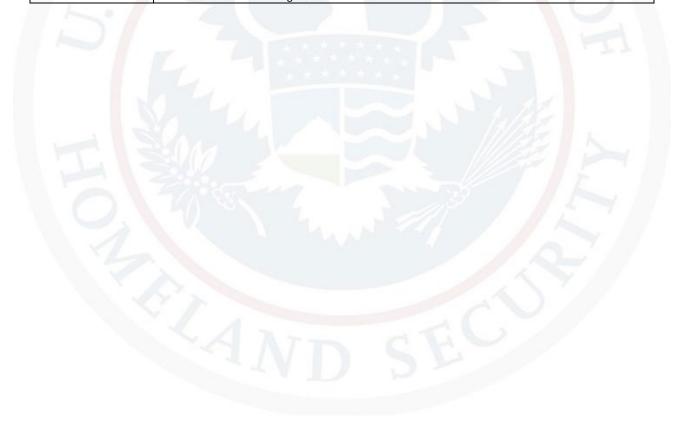
KEY UPDATES

- COVID-19 has caused at least 768,654,968 infections and 6,953,483 deaths globally. In the United States, 103,436,829 cases and 1,127,152 deaths have been confirmed.
- The Omicron variant accounts for 100% of new cases in the U.S., with subvariant XBB.1.16 being the most prevalent.
- Infection of golden Syrian hamsters with the Alpha variant, administered by inhalation at two different particle sizes, suggests particle size is a relevant factor influencing transmission risk.
- Receptor binding studies of Omicron with the ACE2 receptors of mammalian hosts indicated that this variant binds to the ACE2 receptor of 122 other mammals better than human ACE2, suggesting that the host range may increase.
- A large comparison across variants shows the mean incubation period for the wild-type strain at 4.61 days; Alpha at 4.96, Beta and Gamma at 5.18; Delta at 4.43; and Omicron at 3.61 days with the incubation period increasing with age and in females.
- Omicron variants result in less frequent and less severe COVID-19 lung damage compared to previous variants and a lower affinity for the lower respiratory tract.
- Prolonged symptoms (≥ 3 months post-infection) were more common during pre-Delta variant than with Delta or Omicron variants.
- The FDA's Vaccines and Related Biological Products Advisory Committee (VRBPAC) unanimously voted that a monovalent COVID vaccine with an XBB-lineage of the Omicron variant (XBB 1.5 is recommended) should be used for all vaccinations starting in fall 2023.

Major Findings by Topic	
Topic	**Overview of Current Knowledge**
INFECTIOUS DOSE	In a human challenge study (36 adults between 18-29 years), an intranasal dose of 10 median tissue culture infectious dose ($TCID_{50}$) (~7 plaque forming units [PFU]) of wild-type virus successfully infected 53% of healthy volunteers, with 89% developing mild/moderate symptoms. Decreased threshold for infectivity has been modeled in newer variants, suggesting SARS-CoV-2 infection can occur from 500 virus copies of the wild-type strain, 300 virus copies of Delta variant, and 100 virus copies of Omicron variant. There is no preferential animal model for SARS-CoV-2, as clinical signs, recovery, and transmission do not fully recapitulate human disease. Particle size exposure may be a relevant factor influencing transmission risk based on different activities influencing exhaled particle sizes.
TRANSMISSIBILITY	As of 7/24/2023, COVID-19 has caused at least 768,654,968 infections and 6,953,483 deaths globally. In the United States, 103,436,829 cases and 1,127,152 deaths have been confirmed. Cases and fatalities are likely underestimated. As of 7/11/2023, the Omicron variant accounts for 100% of new cases in the U.S., with subvariant XBB.1.16 being the most prevalent. SARS-CoV-2 is transmitted easily between humans, primarily through close contact (either direct or within 6 feet) and aerosol transmission. Within households, the two main risk factors include higher viral loads in the index patient and sharing a room with an infectious patient, which increases the odds of transmission by 40% and 199% respectively. COVID-19 vaccines reduce transmission rates by approximately 54% (range of 38-66%). There is substantial variation of transmission among individuals. Individuals are contagious 1-3 days prior to symptom onset. Pre- or asymptomatic patients can transmit SARS-CoV-2. Most transmission occurs prior to and within 5 days of symptom onset.
HOST RANGE	SARS-CoV-2 is closely related to other coronaviruses circulating in Southeast Asia bat populations. Previous coronaviruses have passed through an intermediate mammal host before infecting humans, but the presence or identity of the SARS-CoV-2 intermediate host remains unknown. Several animal species are susceptible to SARS-CoV-2 infection at varying degrees. These species include, but are not limited to, bat, hamster, mink, deer, rats, rabbits, voles, dogs, cats, and large wild cats. Farm animals are generally not susceptible to SARS-CoV-2 infection. Receptor binding studies of Omicron with the ACE2 receptors of mammalian hosts showed that this variant binds to the ACE2 receptor of 122 other mammals better than human ACE2, indicating that the host range of this variant may increase.
INCUBATION PERIOD	On average, symptoms develop 5 days after exposure with a range of 2-14 days. Incubating individuals can transmit disease for several days before symptom onset. Some individuals never develop symptoms but can still transmit disease. It is estimated that most individuals are no longer infectious beyond 10 days after symptom onset. Individuals can shed virus for several weeks, though it is not necessarily infectious. A large comparison across variants shows the mean incubation period for the wild-type strain at 4.61 days; Alpha at 4.96, Beta and Gamma at 5.18; Delta at 4.43; and Omicron at 3.61 days with the incubation period increasing with age and in females.
ACUTE CLINICAL PRESENTATION	Most symptomatic COVID-19 cases are mild (81%). Fever, cough, and shortness of breath are generally the most common symptoms, followed by malaise, and fatigue. Chills, muscle pain, joint pain, sore throat, gastrointestinal symptoms, neurological symptoms, and dermatological symptoms also occur. COVID-19 is more severe than seasonal influenza. Adults >60 years old and those with comorbidities are at elevated risk of hospitalization and death. Children are susceptible to SARS-CoV-2, though generally show milder or no symptoms. Minority populations are disproportionately affected by COVID-19. Omicron variants result in less frequent and less severe COVID-19 lung damage compared to previous variants and a lower affinity for the lower respiratory tract.
CHRONIC CLINICAL PRESENTATION	COVID-19 symptoms commonly persist for weeks to months after initial onset in up to 73% of those infected. Long-term symptoms such as fatigue, smell/taste disorders, and neurological impairment may affect the ability to return to work. In a cohort of COVID-19 patients, 39% reported symptoms 7-9 months after initial infection, with fatigue, loss of taste or smell, shortness of breath, and headache as the most common chronic symptoms.

Major Findings by Topic	
Topic	**Overview of Current Knowledge**
CHRONIC CLINICAL PRESENTATION	One year after intensive care unit (ICU) admission for COVID-19, lingering physical (74% of 246 ICU patients), mental (26%), and cognitive (16%) symptoms were common, with 58% of patients experiencing issues when returning fully to work. Prolonged symptoms (≥ 3 months post-infection) were more common during pre-Delta variant than with Delta or Omicron variants.
PROTECTIVE IMMUNITY	Recovered individuals appear protected against reinfection for 3-6 months. Reinfection is rare, though the true frequency is unknown and novel variants may increase reinfection frequency. Immune responses persist in most patients for >6 months. Evidence suggests BA.2.10.1 and BA.2.75 recombinant, XBB, is the most antibody-evasive variant identified to date.
CLINICAL DIAGNOSIS	Diagnosis of COVID-19 is based on symptoms consistent with COVID-19, polymerase chain reaction (PCR)-based testing, and/or the presence of SARS-CoV-2 antigen in individuals (detected by enzyme-linked immunosorbent assay [ELISA]). Screening solely by temperature or other symptoms is unreliable. The timing of diagnostic PCR tests impacts results. The false-negative rate for real-time (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. Asymptomatic individuals without COVID-19 symptoms can be diagnosed with SARS-CoV-2 infection by the same tests. In children, viral loads from saliva correlated better with clinical outcomes than viral loads from nasopharyngeal swabs, which may also be true for adults, as saliva tests consistently yield less false-negative results.
MEDICAL TREATMENTS	For hospitalized and critically ill patients, dexamethasone is strongly recommended; if dexamethasone is unavailable, the use of alternative corticosteroids (hydrocortisone, methylprednisolone, prednisone) is recommended. Paxlovid, molnupiravir, and remdesivir (all nucleoside analogs) remain highly effective against both BA.1 and BA.2 (Omicron subvariants). The World Health Organization (WHO) strongly recommends the use of nirmatrelvir-ritonavir in patients with non-severe illness at the highest risk of hospitalization.
VACCINES	Four safe and effective vaccines are currently being administered in the U.S., two with full FDA approval (Pfizer/BioNTech and Moderna) and have been approved as booster doses. In addition, both Pfizer and Moderna have developed "bivalent" boosters, which include components of the wild-type strain as well as the BA.4 and BA.5 lineages of the Omicron variant. The FDA's VRBPAC unanimously voted that a monovalent COVID vaccine with an XBB-lineage of the Omicron variant (XBB.1.5 is recommended) should be used for all vaccinations starting in fall 2023. A third dose of an mRNA vaccine was 76.5% effective at reducing hospitalizations during the U.S. Omicron wave. Patients who receive a booster vaccination are 66% less likely to develop a symptomatic infection from the SARS-CoV-2 Omicron variant compared to un-boosted individuals.
NON-PHARMACEUTICAL INTERVENTIONS	Broad-scale control measures such as stay-at-home orders and widespread face mask use effectively reduce transmission. Individual behaviors (e.g., face masks, social distancing) have been associated with reduced risk of COVID-19 infection. Particular focus should be placed on minimizing large gatherings where superspreading events are more likely.
ENVIRONMENTAL STABILITY	SARS-CoV-2 variants display differences in environmental stability. SARS-CoV-2 can survive on surfaces from hours to weeks and is stable in air for at least several hours, depending on the presence of UV light, temperature, and humidity. Transmission via contaminated surfaces is not considered to be common. Stability of SARS-CoV-2 RNA in clinical samples depends on temperature and transport medium. There is currently no evidence that SARS-CoV-2 is transmitted to people through food or food packaging.
DECONTAMINATION	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. Several methods exist for decontaminating N95 respirators and other personal protective equipment (PPE).

Major Findings by Topic	
Topic	**Overview of Current Knowledge**
DECONTAMINATION	Increased resistance to ethanol was demonstrated by Alpha, Beta, Delta, and Omicron variants; however, all strains were inactivated by 35% ethanol after 15 seconds.
PERSONAL PROTECTIVE EQUIPMENT	Face masks (medical and non-medical) are effective at reducing infections from SARS-CoV-2. Mask fit is a critical component of effectiveness, in addition to filter efficiency. Healthcare workers are at elevated risk of acquiring COVID-19, even with recommended PPE.
GENOMICS	Current evidence suggests that SARS-CoV-2 accumulates mutations at a rate similar to other coronaviruses. Immunosuppressed patients are a possible source of viral variants due to prolonged virus replication within a single host. XBB.1.16 is outcompeting all other variants and accounts for over 17% of sequenced strains in the U.S. Currently, XBB.1.5 and XBB.1.16 are listed as variants of interest (VOIs) and BA.2.75, CH.1.1, XBB, XBB.1.9.1, XBB.1.9.2, and XBB.2.3 are listed as variants under monitoring (VUM) by the WHO.
FORECASTING	Several platforms provide digital dashboards summarizing the current state of the pandemic in U.S. states and counties. The CDC no longer provides forecasts of COVID-19 cases, as too many observations were falling outside of forecast intervals (i.e., forecast accuracy was generally low). Innovative approaches may be needed to forecast COVID-19 re-emergence after elimination or during periods of low incidence. Several characteristics of SARS-CoV-2 mutations, such as their prevalence and relationship to immune escape, may enable forecasting mutations that are likely to show up in future variants months before their emergence.

Infectious Dose – How much agent will make a healthy individual ill?
What do we know?

The human infectious dose is estimated to be 10 $TCID_{50}$ (~7 PFU) of wild-type SARS-CoV-2 (originating strain), delivered intranasally in a human challenge study (36 adults between 18-29 years). Healthy volunteers (53%) were successfully infected with 89% developing mild-to-moderate symptoms.[1] Infectious virus was shed from contagious individuals for 10 days after inoculation, and could be detected as early as 24 hours post-infection.[1]

- While there is a likely dose-infection relationship, there is no significant link between dose and severity in humans.[2]
- There is no preferential animal model for SARS-CoV-2 as clinical signs, recovery, and transmission vary between species.[3]
- Transgenic models may represent extreme conditions with unnatural gene expression patterns and rapid lethality, as the random integration strategy used to insert additional angiotensin-converting enzyme 2 (ACE2) copies is largely stochastic.[4]

An estimate of the human infectious dose of SARS-CoV-2 from primate research is 36-179 viral particles (PFU) necessary to cause infection via the inhalation route.

Non-Human Primates (NHP)
- Several NHP species (rhesus macaques, African green monkeys, cynomolgus macaques) are able to replicate aspects of human SARS-CoV-2 infection[5] from mild[6] to severe illness,[7] including acute respiratory distress syndrome (ARDS).[8]
- In cynomolgus macaques, the median dose required to induce wild-type SARS-CoV-2 seropositivity was 52 $TCID_{50}$ (approximately 36.4 PFU) via the inhalation route.[9] The median dose needed to induce fever was 256 $TCID_{50}$ (approximately 179.2 PFU) via the inhalation route,[9] which also suggests that symptom severity may be dose-dependent in macaques,[9] although severity is not necessarily dependent upon viral load.[10]
- Larger doses of SARS-CoV-2 have been shown to infect NHPs via the inhalation route[5-8, 11] or its surrogates (intranasal, intratracheal, combination routes)[7, 9, 11-12] and the ocular route.[13] Intragastric exposure does not appear to result in NHP infection.[13]

Rodents and Other Animal Models
- Many rodent models are modified to express human airway cells,[15] and the infectious dose of SARS-CoV-2 in these models is estimated at 4 x 10^3 $TCID_{50}$.[16] Golden Syrian hamsters and ferrets can replicate COVID-19 symptoms observed in humans[17-20] and are able to transmit to animals in separate cages without direct contact.[19, 21]
- In hamsters, higher infectivity at low doses of the Alpha variant of SARS-CoV-2 compared to wild-type virus suggests a mechanism for increased variant transmissibility.[22]
- Infection of golden Syrian hamsters with the Alpha variant administered by inhalation at two different particle sizes (1.3 μm or 5.2 μm) determined a significantly lower ID_{50} of 0.3 $TCID_{50}$ (0.2 PFU) for small particle aerosol exposure versus 11.5 $TCID_{50}$ (8.05 PFU) for large particle exposure, suggesting exhaled particle size due to various activities is a relevant factor influencing transmission risk.[23]
- One in six ferrets exposed intranasally to 10^2 PFU became infected, while 12 out of 12 animals exposed to >10^4 PFU became infected.[24]
- A median lethal dose (LD_{50}) of 10^4 PFU was calculated for K18-hACE2 mice.[2]
- SARS-CoV-2 Omicron was shown to infect C57BL/6 mice (10^5 $TCID_{50}$/mouse intranasal exposure dose) and cause changes in the lungs. A mutation in the spike protein was critical for this adaptation.[219]

Modeling Estimates
- The infectious dose of a pathogen can be estimated by the amount of genetic material passed between infector and infectee (called "bottleneck" size);[25] using epidemiological data, sequencing data, and statistics, the average "bottleneck" size for SARS-CoV-2 has been estimated as ~1,200 viral particles, though exposure routes were not possible to identify.[26] Bottleneck size is estimated to be 1-8 among households[27] and ~1,000 in a well-traced outbreak of Delta variant in Guangzhou.[28]
- Modeling aerosol exposures from five case studies suggests the inhalation median infectious dose (ID_{50}) for SARS-CoV-2 is approximately 361-2,000 viral particles, which is approximately 250-1,400 PFU.[29]
- Viral load of Omicron was calculated to be 10-100x higher than Delta,[30] which raises concerns regarding increased SARS-CoV-2 transmission.
- Decreased threshold for infectivity has been modeled in newer variants. SARS-CoV-2 infection can occur from 500 virus copies of the wild-type strain, 300 virus copies of Delta variant, and 100 virus copies of Omicron variant.[30]

Related Coronaviruses
- Humans exposed intranasally to ~70 PFU of seasonal coronavirus 229E developed infections,[31] with a plausible intranasal ID_{50} of 10 $TCID_{50}$ (~7 PFU).[32-33] The inhalation ID_{50} of seasonal coronavirus 229E is unknown in humans.

What do we need to know?

- How does the infectious dose vary among the different routes of transmission for humans?

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, primarily through close contact and aerosol transmission.[34-37]

- As of 7/24/2023, COVID-19 has caused at least 768,654,968 infections and 6,953,483 deaths globally.[38-39] In the United States, 103,436,829 cases and 1,127,152 deaths have been confirmed.[38] Cases[40] and fatalities are likely underestimated.[41-44]
- SARS-CoV-2 can spread via aerosol or "airborne" transmission[45] beyond six feet in certain situations[46] (i.e., enclosed spaces with inadequate ventilation).[47] The risk of infection from fomites[48] is believed to be low,[49] potentially due to mucins in saliva and mucus.[50] Vertical transmission is rare.[51-52] Hospital transmission follows community incidence[53] and may involve superspreading events.[54]

Several variants (Delta, Gamma, Alpha, Beta, Kappa, Eta, Omicron) have higher transmission than wild-type SARS-CoV-2.[55]

- As of 7/11/2023, the Omicron variant accounts for 100% of new cases in the U.S. (XBB.1.16 is 17.5%, XBB.1.5 is 16.1%, XBB.2.3 is 13.4%, EG.5 is 13.0%, and XBB.1.16.1 is 10.4%).[56]
- Omicron variants do not have higher viral loads than the Delta variant;[57] however, they replicate faster in human bronchial tissue, but slower in deeper lung tissues, explaining its higher transmissibility but lower disease severity than prior variants.[58]
- Elevated infection risk from Omicron subvariants, including XBB, is partly due to their enhanced ability to evade the human immune response, even in vaccinated and previously infected individuals.[59-65]

COVID-19 vaccines reduce transmission rates.[66]

- Low vaccination rates facilitate COVID-19 transmission.[67] Vaccination provides protection by reducing viral load[68] and transmission.[69-71] Booster vaccinations significantly reduced infections from both Delta and Omicron variants.[72-73] Those fully vaccinated showed a vaccine effectiveness of 66.8% with a 95% effectiveness against death during a Delta outbreak.[74]
- Studies in household transmission showed that cases with children ≤ 11 years old had higher odds of transmission (45%) within the household during outbreaks with Omicron variants. Overall, there was reduced infectiousness in primary cases and reduced susceptibility in contacts when both were vaccinated.[75] Additional studies have suggested this same pattern of increased risk for children and adolescents, but analysis on vaccinated children is lacking.[76]

The amount of infectious virus emitted from an infectious individual is unclear but appears highly variable.

- In a small study (n=38), researchers found median SARS-CoV-2 aerosol emission rates of 70, 110, and 80 RNA genome copies per minute for breathing, talking, and singing, respectively, with substantial variation among individuals and higher emission rates closer to symptom onset.[77]
- In a study of 25 patients infected with Omicron, 40% exhaled detectable virus, with 11 patients exhaling between 4.4-5.8 x 10^7 genome copies per hour.[82] In a separate study, 85% of exhaled particles containing virus were in the fine range (<5μm), with talking and singing producing more particles (844 and 1,094 per minute, respectively) and a greater proportion of fine particles (93.1% and 83.2%, respectively) compared to breathing (65 particles per minute, 54.2% <5μm).[83]
- Higher viral loads in COVID-19 patients result in higher secondary attack rates,[84] with a rate of 12% with viral loads below 1^{06} genome copies/mL (nasopharyngeal swab), and 24% with viral loads more than 1^{0}10 viral genome copies/mL.[85]
- Within households, the two main risk factors include higher viral loads in the index patient and sharing a room with an infectious patient which increased the odds of transmission by 40% and 199% respectively.[86]

Asymptomatic or pre-symptomatic individuals can transmit SARS-CoV-2[87] and play a large role in new case growth.[88]

- Individuals are infectious 1-3 days prior to symptom onset.[89-90] Pre-[91-96] or asymptomatic[97-99] patients can transmit SARS-CoV-2.[100] Most transmission occurs prior to[101] and within 5 days of symptom onset,[102-103] as seen in case studies[104] and models.[105]
- In a case series of 622 index patients infected with the Omicron variant, half of the 455 secondary cases were due to transmission before symptom onset of the index patient (only symptomatic cases were examined in the study).[106]
- Asymptomatic cases transmit less often than symptomatic ones,[107-109] causing 66-83%[101, 110-111] fewer cases, due to lower viral loads.[112] The likelihood of symptoms in secondary cases increases with severity of symptoms in the primary case.[103]

Infection risk is particularly high indoors,[113] while outdoor transmission is rare.[114]

- SARS-CoV-2 may be spread by conversation and exhalation[115-118] in indoor areas such as restaurants[119-120] or offices.[121] Clusters are often associated with large indoor gatherings,[122-123] including bars,[124] restaurants,[125] and gyms.[126-127]

Superspreading events appear common in SARS-CoV-2 transmission and may be crucial for controlling spread.

- Most new infections originate from a few infectious individuals (overdispersion parameter k = 0.2-0.5).[128-133]
- Individuals vary greatly in their estimated infectivity. Importantly, this variation is not entirely due to the number of viral genome copies estimated by nasal or saliva samples,[59] which suggests that there may be host-specific indicators of infectiousness, and superspreading potential that could identify individuals at high risk of onward transmission.[59]

Infection in children is underestimated,[135-136] and children of any age can acquire and transmit infection.[137] There is some evidence that younger children (<10-15) are less susceptible[138-139] and less infectious[119] than older children and adults.[140-141]

- Children transmit SARS-CoV-2 less often than adults,[142] though they have both higher[143] and lower[112] viral loads compared to adults.[144-145] Transmission in schools is generally low,[146-150] follows community incidence,[151-153] and can be mitigated.[154]

What do we need to know?

- What are the transmission rates of Omicron in different environments among varying ages and vaccination status?
- What is the relationship between detectable SARS-CoV-2 RNA in breath samples and contagiousness?[155]

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 presence or identity of the SARS-CoV-2 intermediate host is unknown.[156-159] Bat coronaviruses with >95% identity to SARS-CoV-2 have been identified in horseshoe (*Rhinolophus*) bat species from Laos;[160] however, whether a direct jump from bats to humans occurred is unknown.[161]

- Horseshoe bat fecal samples collected during the pandemic from Great Britain were shown to be positive for related sarbecoviruses (SARS Betacoronavirus) but not SARS-CoV-2.[162]
- In the U.S., experimentally exposed big brown bats (*Eptesicus fuscus*) to SARS-CoV-2 via oropharyngeal and nasal route found no subsequent signs of infection, symptoms, or transmission.[170]
- Receptor binding studies of Omicron with the ACE2 receptors of mammalian hosts showed that this variant binds to the ACE2 receptor of 122 other mammals better than human ACE2, indicating that the host range of this variant may increase.[222]

Several animal species are susceptible to SARS-CoV-2 infection.[176]

- White-tailed deer are susceptible to SARS-CoV-2 and can transmit the virus to other deer.[164-166] Deer in four U.S. states were found to have SARS-CoV-2 antibodies,[167] and recent evidence epidemiologically links deer-to-human transmission.[168]
- SARS-CoV-2 cases in mink on U.S. farms show high mortality rates.[177-178] High reinfection levels in farmed mink likely contributed to the detection of virus with several additional mutations compared to the wild-type strain.[179-180] Infected mink have been linked to human infections.[159, 178, 181] Humans and mink are able to transmit infectious virus back and forth.[182] Aerosol concentrations of viral RNA on mink farms can be high, leading to occupational exposure risks.[183]
- Domestic cats are susceptible to infection with SARS-CoV-2,[192-193-194] and can transmit virus to other cats via droplet or short-distance aerosol.[192] Serial passage of SARS-CoV-2 in domestic cats attenuates transmissibility, suggesting they are not long-term reservoirs.[195] Stray cats in Spain were found to be seropositive at low frequencies (1.6% to 3.5%);[196] 7.1% of domestic cats in Argentina were found to be seropositive;[197] and evidence exists of household human-to-cat transmission,[198-199] and to veterinarians.[200] Shelter cats are not thought to be a reservoir for human infections.[201]
- Dogs exposed to SARS-CoV-2 produce anti-SARS-CoV-2 antibodies,[212-214] but exhibit no clinical symptoms.[192, 213]
- A literature review by the European Food Safety Authority noted that cats, ferrets, mink, hamsters, carnivores, great apes, and white-tailed deer are the highest at risk for SARS-CoV-2 infection and transmission.[175] White-tailed deer, Syrian hamsters, and mink are considered the only known wildlife reservoir currently.[169]
- Farm animals (ducks,[192] chickens,[207] pigs,[192] turkey,[208] geese,[208] and cattle[209]) are generally not susceptible to SARS-CoV-2. Sheep can be infected with SARS-CoV-2, but have limited transmission potential.[210] A serology survey of 612 sheep and goats, belonging to 24 different farms, found that 23 were ELISA positive (17 sheep, 6 goats).[211] The overall risk of human-to-animal transmission in commercial breeding/livestock operations appears to be low. Buffaloes, goats, sheep, horses, carrier pigeons, rabbits, hens, snakes, pigs, and cows all tested negative after prolonged exposure to positive keepers.[186]
- Retrospective genome-wide studies on mammalian SARS-CoV-2 strains indicate most transmission events involved minks, and rarely involved cats, dogs, or deer.[163]
- Other susceptible species include puma, snow leopard, fishing cat, binturong, coatimundi, lynx, tiger, lion, hippopotamus,[161] black-tailed marmoset, giant anteater, cougar, Indian leopard, spotted hyena, Asian small-clawed otter, manatee, and mule deer.[217-218]
- Rats in New York City were found to be serologically positive (16.5%) and Sprague Dawley rats were susceptible to Alpha, Delta, and Omicron variants.[220]
- Research studies have shown that invertebrates, birds, reptiles, and amphibians are not susceptible.[221]

There is no single animal model to study the full spectrum of COVID-19 phenotypes in humans, although several models allow the study of SARS-CoV-2 induced respiratory disease.[189]

- Several NHPs are susceptible to infection with SARS-CoV-2 including cynomolgus macaques,[12] African green monkeys,[7] rhesus macaques,[187] and pigtail macaques.[188]
- Golden Syrian hamsters,[159] ferrets,[19, 21] deer mice,[171] rabbits,[173] and raccoon dogs[190] are susceptible to infection and can be experimentally infected with SARS-CoV-2 via intranasal exposure.
- Bank voles (*Myodes glareolus*) seroconvert after exposure, but are asymptomatic and do not transmit infection to others.[174]

What do we need to know?

We need to know the best animal model for replicating human infection by various exposure routes.

- What is the intermediate host(s) (if any)?
- 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?

On average, symptoms develop 5 days after exposure with a range of 2-14 days. Incubating individuals can transmit disease for several days before symptom onset. Some individuals never develop symptoms but can still transmit disease.

- By general consensus, the incubation period of COVID-19 is between 5 and 6 days.[223-225] Few infected individuals show symptoms sooner than 2 days after exposure.[225] However, some models calculate an incubation period between 7 and 8 days, suggesting that 5-10% of individuals are still infectious after a 14-day quarantine.[226]
- 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,[227] while adolescent and young adult populations (15-24 years old) have been estimated at ~2 days.[228]
- Individuals can test positive for COVID-19 even if they lack clinical symptoms.[91, 97, 229-231] Individuals can be infectious while asymptomatic,[91, 230, 232-233] and asymptomatic and pre-symptomatic individuals have similar amounts of virus in the nose and throat compared to symptomatic patients.[90, 234-235]
- Peak infectiousness may be during the incubation period, one day before symptoms develop.[236] Infectious virus has been cultured in patients up to 6 days before the development of symptoms.[90]
- Of individuals quarantining after a COVID-19 contact at home, 19% of individuals undergoing a 7-day quarantine were at risk of developing and potentially transmitting COVID-19,[237] which indicates that quarantines of less than 14 days carries risk of disease and transmission, and that care should be taken after a shortened quarantine period (e.g., wearing a mask, distancing).[237]

Some SARS-CoV-2 variants have a shorter incubation period and serial interval.

- Initial estimates of wild-type SARS-CoV-2 serial interval (time between successive cases) was 5.8 days.[238] There is some evidence that the Delta variant spreads faster than prior virus lineages 2.9 vs 5.7 days, respectively.[239-240] The incubation period of the Delta variant was estimated at 6.64 days. Cases in females and cases with severe symptoms had relatively longer mean incubation periods than cases in males and cases with non-severe symptoms, respectively.[241]
- The incubation period for Omicron variants has been estimated at 2 to 3 days,[106, 242-244] shorter than previous variants.[245-247]
- In a small study (n=12 transmission pairs) the average time of the serial interval of the Omicron variant was 2.9 days, which is faster than for wild-type virus.[248] Larger studies have confirmed that the serial interval is decreasing. The average range for Delta is 2.3 to 5.8 days and for Omicron is 2.1 to 4.8 days.[242-243, 249-250]
- A large comparison across variants shows the mean incubation period for the wild-type strain at 4.61 days; Alpha at 4.96, Beta and Gamma at 5.18; at Delta 4.43; and Omicron 3.61 days with the incubation period increasing with age and in females.[251]

It is estimated that most individuals are no longer infectious beyond 10 days after symptom onset.[252]

- A systematic review of published studies on SARS-CoV-1, SARS-CoV-2, and MERS-CoV found none that reported isolation of infectious virus from COVID-19 patients beyond 9 days from symptom onset, despite high viral loads by genetic tests.[253]
- While the amount of virus needed to infect another individual is unknown, mild-moderate COVID-19 cases appear to be infectious for no longer than 10 days after symptom onset, while severely ill or immunocompromised patients may be infectious for 20-70 days[254] after symptom onset; individuals can also transmit infection before symptoms appear.[255]
- Asymptomatic individuals are estimated to be infectious for between 5.76[256] and 9.5 days.[257]
- Among 53 healthcare workers infected with the Omicron variant, rates of viral culture positivity – a surrogate for the probability of shedding infectious virus – were 83%, 52%, 13.5%, and 8% at 5, 7, 10, and 14 days after initially testing positive, respectively; in 19% of cases, patients shed infectious virus after their symptoms stopped.[258]
- In a small study of 11 Omicron cases, individuals were not infectious (estimated by tissue culture infectivity of collected samples) beyond 8 days after symptom onset.[259]
- In a small (n=14) sample of young, healthy, vaccinated patients, rapid COVID-19 antigen test results were plausible surrogates for the infectiousness of collected samples, with those testing negative shedding non-infectious samples.[260]
- The generation time (time between infection events in a chain of transmission) for SARS-CoV-2 is estimated as 4-5 days.[261]

Individuals can shed virus for several weeks, though it is not necessarily infectious.

- 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).[262]
- Asymptomatic and mild infections testing positive for SARS-CoV-2 take less time to test negative than severely ill patients.[263]
- Viral RNA loads in the upper respiratory tract tend to peak within a few days of symptom onset and become undetectable approximately 2 weeks after symptoms begin.[264] The duration of the infectious period is unknown,[264] though patients can test positive for SARS-CoV-2 viral RNA for extended periods of time, particularly in stool samples.[264]

What do we need to know?

We need to know the incubation duration and length of infectivity in different patient populations.

- How soon can asymptomatic patients transmit infection after exposure?
- Does the incubation period correlate with disease severity or exposure dose?

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

What do we know?

Most symptomatic cases are mild, but severe disease can appear in any age group. Older individuals and those with underlying conditions[265] are at higher risk of serious illness and death, as are men.[266]

- Most symptomatic COVID-19 cases are mild (81%).[267] COVID-19 causes a wide range of symptoms including fever or chills, cough, shortness of breath or difficulty breathing, fatigue, muscle or body aches, new loss of taste or smell, sore throat, congestion or runny nose, nausea or vomiting, and diarrhea.[268]
- Multisystem Inflammatory Syndrome in Adults (MIS-A) is a rare condition that may develop days to weeks after SARS-CoV-2 infection where inflammation occurs in the heart, gastrointestinal tract, skin or brain.[269]

The Omicron variant and subvariants are less likely to result in hospitalization or death than the Delta variant.[270]

- The Omicron variant has a significantly higher replication in human bronchi compared to wild-type and Delta variants, potentially increasing infectious particles released by breathing and speaking, explaining its rapid forward transmissibility.[58]
- In a retrospective cohort study, the Omicron variant resulted in a 59% lower likelihood of hospitalization or death than the Delta variant,[270] though elevated case counts resulted in greater absolute hospitalizations,[271] and estimating variant severity with population-level data is difficult.[272]
- Omicron variants result in less frequent and less severe COVID-19 lung damage compared to previous variants[274] and a lower affinity for the lower respiratory tract.[275]

COVID-19 is more severe than seasonal influenza,[280] evidenced by higher ICU admission[281] and mortality rates.[282]

In the U.S., 29-34% of hospitalized patients required ICU admission, and 12.6-13.6% died from COVID-19.[283-284]

- Higher SARS-CoV-2 RNA loads at admission have been linked to greater risk of death,[285-288] though this is not universal.[289]
- High viral loads (reverse transcription polymerase chain reaction [RT-PCR] cycle threshold value <28) are associated with symptom severity 6 months after illness onset.[290]
- COVID-19 also causes pneumonia,[291] cardiac injury,[292] kidney damage,[293-294] pancreatitis,[295] arrhythmia, sepsis, stroke,[296-297] respiratory complications,[298] and shock.[231, 299-301] SARS-CoV-2 weakens blood vessels in the lungs[302] and is associated with hyperactive platelets,[303] leading to ARDS.[304-305] Clotting affects multiple organs[306] and is present in 15-27% of cases.[307]
- Low oxygen saturation and shallow breathing upon hospital admission are associated with elevated mortality risk.[308]
- The risk of severe COVID-19 may be influenced by the environment, as in one study where elevated ozone (10x greater than acceptable concentrations) predisposed mice to severe illness by upregulating cellular entry proteins (e.g., *Tmprss2*).[309]

Adults >60 years old[310] and those with comorbidities[311-312] are at elevated risk of hospitalization[313] and death.[230, 301]

- Cardiovascular disease,[314] obesity,[315-317] hypertension,[318] diabetes,[319-320] cancer,[321] down syndrome,[322] and respiratory conditions increase the case fatality rate (CFR).[230, 301] Kidney disease,[323] dialysis,[324] and lack of physical activity[325] may increase disease severity.
- Estimates of the age-specific infection fatality rate were identified in a large meta-analysis analysis using data publicly disseminated prior to September 18, 2020: 0-34 years = 0.004%; 35-44 years = 0.068%; 45-54 years = 0.23%; 55-64 years = 0.75%; 65-74 years = 2.5%; 75-84 years = 8.5%; 85 and older = 28.3%.[326] Provisional death counts through April 15, 2023 indicate that rate of death from COVID-19 for 85 and older are 360 times higher than 18-29 year-olds.[327]

Minority populations are disproportionately affected by COVID-19[328] and appears to be linked to underlying conditions.[329]

- Minority ethnic populations[330] acquire SARS-CoV-2 infection at higher rates,[331-334] are hospitalized,[335-336] and die disproportionately.[337-338] Hispanic and Black COVID-19 patients tend to die at younger ages.[339]
- Pregnant women with COVID-19 have higher mortality rates compared to those without;[340] the proportion of pregnant patients with severe COVID-19 increased after the introduction of the Delta variant.[341]
- Individuals with physical or intellectual disabilities are at greater risk of poor COVID-19 outcomes, including mortality, ICU admission, and lengthy hospital stays.[342]

Children are susceptible to COVID-19,[343] though generally show milder[262, 344] or no symptoms.[345]

- Children appear primed to mount early, effective immune (particularly interferon) responses to SARS-CoV-2, among other features, which help to explain their lower rates of severe disease and death compared to adults.[346-347]
- Children (21% to 28% <19 years old) may be asymptomatic.[262, 348-349] Most symptomatic children show mild or moderate symptoms.[349-350] Severe disease in children[351] and infants[352-353] is more likely in those with complex medical histories.[354-355]
- Multisystem Inflammatory Syndrome in Children (MIS-C) is a rare inflammatory condition, occurring in 1 of approximately 3,000 to 4,000 children that had COVID-19 infection early in the pandemic. Symptoms generally appear 2-6 weeks after infection and can occur when children had no or few symptoms of COVID-19.[356]

What do we need to know?

We need to know the impact of new SARS-CoV-2 variants on presentation and disease severity.

- What therapeutics can be used as treatments?[357]
- What treatments reduce transmissibility of SARS-CoV-2 and improve outcomes for immunocompromised?[358-359]

Chronic Clinical Presentation – What are the long-term symptoms of COVID-19 infection?

What do we know?

Long COVID is a multisystemic illness, sometimes referred to as Post-Acute Sequelae of SARS-CoV-2 infection (PASC),[360] long-haul COVID, post-COVID-19 conditions, or chronic COVID where symptoms linger for weeks, months, or years after initial diagnosis of COVID-19.[361] The incidence is estimated at 10-30% for non-hospitalized cases, 50-70% for hospitalized cases, and 10-12% for vaccinated cases.[362]

- Risk factors potentially include obesity,[363] age,[364] female sex,[365] type 2 diabetes, Epstein-Barr Virus reactivation, presence of specific autoantibodies, connective tissue disorders, attention deficit hyperactivity disorder, chronic urticaria and allergic rhinitis, although no identified preexisting conditions have been identified in a third of Long COVID cases.[362]
- Prolonged symptoms (≥ 3 months post-infection) were more common during pre-Delta variant than with Delta or Omicron variants.[362]
- Hospital readmission rates are 9-29% of COVID-19 patients.[366-368]
- Long-term sequelae of SARS-CoV-2 infection appear to be linked to pre-existing conditions. Underlying autoimmune or internal complications observed following COVID-19 may be attributed to viral infection stimulating a broad immune response exacerbating underlying conditions, with symptoms ranging from vascular and cardiac issues,[369-370] central nervous system and demyelination issues,[371] and sex-specific reproductive complications.[372-374]
- The importance of initial symptom severity for subsequent development of PASC is unclear, with some studies showing high risk in mildly ill patients,[375] while others show higher risk in severely ill patients.[376]
- Long-term symptoms such as fatigue,[377] smell/taste disorders,[378-379] and neurological impairment[380] may affect the ability to return to work.[380]
- Approximately 8% of mildly ill individuals had disrupted work schedules 8 months after initial illness.[381]
- In a cohort of 410 COVID-19 patients, 39% reported symptoms 7-9 months after initial infection, with fatigue, loss of taste or smell, dyspnea (shortness of breath), and headache as the most common chronic symptoms.[382]
- In a smaller study (n=96), 77% of patients reported ongoing symptoms 12 months after initial infection, with the most common symptoms being reduced exercise capacity, fatigue, dyspnea, difficulties with concentration, and finding correct words during speech.[383]
- Over 203 symptoms were reported by Long COVID patients in a large (n=3,762) survey.[380]
- In the UK, individuals vaccinated with the Pfizer/BioNTech, AstraZeneca, or Moderna vaccines had a 50% lower chance of experiencing COVID-19 symptoms lasting more than one month compared to unvaccinated individuals.[384]
- T cell and antibody responses did not differ between individuals with acute or chronic COVID-19 nine months post-infection,[385] suggesting that differences in immune response are not the only cause of PASC.
- In a small cohort of pediatric patients with MIS-C (n=86), long-term outcomes at one year after initial diagnosis were positive, with no fatalities and two hospital readmissions (thought to be unrelated to MIS-C).[386]
- Vaccination reduces the odds of hospitalization and number of symptoms within the first week of illness, and reduced long-duration (≥28 days) symptoms following the second dose.[384, 387-388]

Researchers are identifying methods to diagnose patients with Long COVID early.

- Corneal scans in 40 patients who had recovered from acute COVID-19 showed greater corneal nerve fiber damage in those who reported neurological symptoms up to 4 weeks post-infection compared to those without neurological symptoms, suggesting that corneal microscopy could be a potential rapid objective test when evaluating Long COVID patients.[389]
- Researchers examined plasma and isolated peripheral blood mononuclear cells from 224 healthy and sick individuals (including 121 with PASC symptoms) and, using bioinformatics to analyze cytokines, were able to discriminate between severe disease and PASC.[390]
- PASC patients may be differentiated from severe COVID-19 patients by the type and persistence of monocytes and SARS-CoV-2 proteins (e.g., S1) in the body.[390]
- Different antibody levels correspond to different manifestations of PASC (e.g., neurological, respiratory, gastrointestinal), suggesting that patient risk may be assessed for both PASC risk and presentation.[391]
- One year after ICU admission for COVID-19, lingering physical (74% of 246 ICU patients), mental (26%), and cognitive (16%) symptoms were common, with 58% of patients experiencing issues returning fully to work.[392]

What do we need to know?

We need to know the rate of PASC and chronic symptoms in different patient populations.

- What is the frequency, mechanism,[393] and clinical implication of Long COVID?[394-396]
- How many symptoms are linked to Long COVID?
- Does vaccination of people currently experiencing Long COVID improve symptoms?[387]
- How prevalent are chronic symptoms in children or individuals over 60?[397]
- Do variants change the risk of Long COVID?[388]
- Does previous diagnosis of COVID-19 confer complications during pregnancy after viral clearance?
- What latent pathogens are potentially reactivated in Long COVID?[398]

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

What do we know?

Recovered and vaccinated individuals appear protected against reinfection for several months; however, the duration of protection is variable and depends on the individual and the variants circulating at the time.

- Neutralizing antibody responses are present within 8-19 days after symptom onset[399] and can persist for many months.[54, 400-401] Some unvaccinated individuals have no detectible neutralizing antibody activity after infection.[402] Individuals with more severe infections developed higher neutralizing antibody levels that persisted longer.[402-403] Others have found higher total immunoglobulin (Ig) levels, and symptom severity could be used to predict protective immunity.[404]
- Convalescent patients are expected to have long-lasting protection against SARS-CoV-2, especially after vaccination.[405-406]
- Immunocompromised individuals typically have a weaker response to immunization, leading to lower levels of protective immunity and a greater likelihood of reinfection, especially if new variants are circulating.[407]
- Vaccine-derived immunity is robust in pregnant and lactating women.[408] Antibodies are transferred to neonates by the placenta[409] and through breast milk,[410] if the woman gets vaccinated during pregnancy [411] or recovers from a natural infection[412] during pregnancy.
- In a 2023 review of protective immunity in 65 studies from 19 countries over 2.5 years, excluding hybrid immunity (vaccination plus recovery from natural infection), analysis of the data showed that immunity from previous infection was highly protective against reinfection from earlier variants such as Alpha and Delta (82%) and only moderately protective against reinfection with Omicron (45%). While reinfection can still occur, the protection from infection via acquired immunity reduces the risk of severe disease and death from any variant by 88% for over 10 months.[413]

While protection against reinfection wanes over a couple months, especially as new variants circulate, protection against serious disease or hospitalization remains high, with hybrid immunity providing the highest magnitude and duration of protection.[414] Even with mild to no symptoms, individuals are able to spread the virus during a reinfection.[415]

- In a large metadata review of 26 studies before and during the Omicron wave, prior infection led to a 25% reduction in reinfection and a 75% reduction in severe disease or hospitalization at 12 months. Hybrid immunity led to a 42% reduction in reinfection and a 97% reduction in serious disease or hospitalization at 12 months.[414]

The impact of emerging SARS-CoV-2 variants on protective immunity and reinfection risk is unclear.

- SARS-CoV-2 mutations can reduce responses to serum from vaccinated patients.[416]
- Neutralization of the Omicron variant has been observed in groups with 3-4 antigenic exposures such as 2 vaccinations plus booster[417-421] or convalescent plus 2 vaccinations.[417, 421-422] However, the significant antigenic shift of Omicron compared to earlier variants causes a substantial reduction in protective immunity, both in naturally infected and vaccinated people.[423]
- Neutralizing antibodies rapidly decline by Day 91, with differences between Omicron sublineages.[424] Preliminary evidence suggests BA.2.10.1 and BA.2.75 recombinant, XBB, is the most antibody-evasive variant identified.[425]
- Natural immunity obtained prior to Omicron provides 46% protection against the BA.1 and BA.2 Omicron variants; immunity generated by 3 vaccinations confers 52% protection, and a hybrid of natural- and vaccine-induced immunity provided 77% protection.[426] Individuals that receive two doses of vaccine showed 76.8% protection from severe illness for up to 6 months, but no protection against Omicron infections.[426]
- Vaccinated patients with prior infection before vaccination had a higher antibody and B cell response and faster increase in antibodies by Day 7 than those who did not have a prior infection, suggesting a benefit in hybrid humoral immunity.[427] Longer lasting immunity was identified following two doses of vaccine regardless of prior infection.[427]
- Vaccination followed by breakthrough infection with BA.1 produces neutralizing antibodies to earlier variants,[428-429] but can be evaded by variants BA.4/5,[273, 430-431] and breakthrough infections with BA.2 have shown protective neutralizing antibodies.[432]
- Long-term analysis of healthcare workers found that triple vaccinated, COVID-19 naïve individuals showed increased B cell and T cell recall and cross-neutralizing antibodies against previous variants following infection with Omicron, but immunity against Omicron itself was dampened. Previous infection with Alpha reduced this cross-neutralization and prior infection with wild-type halted the immune boosting effect after infection with Omicron, but still protected from severe disease.[433]
- Patient serum from individuals infected with non-Omicron variants exhibited poor neutralization ability against the Omicron variant, suggesting low protective immunity against Omicron from infection with prior variants.[434]

Reinfection with a homologous SARS-CoV-2 strain is possible,[435] though the true frequency is unclear.

- Infection with SARS-CoV-2 appears to provide at least an 80-83% reduction in the risk of reinfection for at least 5 months,[436-439] and reinfection was plausibly identified in 2.4-6.7% of patients.[440-441]
- Studies during circulation of wild-type and Alpha variants showed the risk of hospitalization and death were substantially lower for reinfected individuals compared to those with primary infections.[442]

What do we need to know?

We need to know the frequency and severity of reinfection, as well as the protective effects of immune components.

- How long does protective immunity last for children compared to adults?
- What is the probability of reinfection, particularly with SARS-CoV-2 variants?
- Is infection with the Omicron variant more likely to lead to protection against future variants?

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

What do we know?

Diagnosis of COVID-19 is based on symptoms consistent with COVID-19 and PCR-based testing of active cases, and/or the presence of SARS-CoV-2 antibodies in individuals. Screening solely by temperature or other symptoms is unreliable.

- As of 5/9/2023, the U.S. Food and Drug Administration (FDA) has granted Emergency Use Authorization (EUA) for 449 test and sample collection devices, including 302 molecular tests and sample collection devices, 83 antibody and other immune response tests and 63 antigen tests, and 1 diagnostic breath test. There are 78 authorized molecular tests and 1 antibody authorized for home-collected samples. Additionally, there is 1 prescription at-home molecular test, 2 prescription at-home antigen tests, 30 over-the-counter at-home antigen tests, and 4 over-the-counter molecular tests with EUA.[443]
- Multiplex PCR assays able to detect influenza A, influenza B, respiratory syncytial virus and SARS-CoV-2 have been developed for rapid differential diagnosis of respiratory viruses.[444]
- Serology tests allow health care professionals to identify individuals who have developed an adaptive immune response to SARS-CoV-2, but the tests should not be used to diagnose or exclude acute COVID-19 infection.[445]
- The FDA granted EUA to a non-invasive, non-diagnostic device based on machine learning algorithms that screens for biomarkers of SARS-CoV-2 infection in asymptomatic individuals older than 5 years.[446]
- 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.[447] Low viral loads can lead to false-negative RT-PCR tests.[448]
- The duration of PCR-detectable viral samples is longer in the lower respiratory tract than the upper respiratory tract; nasopharyngeal sampling is most effective (89%) between 0 and 4 days after symptom onset, but falls significantly (to 54%) by 10 to 14 days.[449] After 10 days, alternative testing methods (e.g., lower respiratory samples) may be necessary.[449]
- Trained dogs show high accuracy for SARS-CoV-2 detection (sensitivity = 0.88, specificity = 0.99), and could be used to identify individuals needing confirmation via rapid antigen or molecular testing.[450] The FDA has now approved the InspectIR COVID-19 breathalyzer that uses gas chromatography-mass spectrometry to detect SARS-CoV-2.[451]
- While nasopharyngeal swabs are the gold standard for COVID-19 diagnosis, pooled nasal and throat swabs also show high diagnostic accuracy, while saliva, nasal swabs, and throat swabs all showed lower accuracy.[452] However, homogenization of saliva samples prior to RNA extraction increases diagnostic accuracy, with results comparable to nasopharyngeal swabs.[453] The detection of Omicron-infected patients from saliva by PCR testing was comparable to nasopharyngeal swabs.[454]
- Researchers have demonstrated the utility of disposable, bio-functional strips for SARS-CoV-2 identification.[455]
- In children, viral loads from saliva correlated better with clinical outcomes than viral loads from nasopharyngeal swabs.[456]
- Rapid tests based on RT-PCR or standard laboratory nucleic acid amplification tests (NAATs) are preferred over rapid isothermal NAATs in symptomatic individuals to reduce the chance of false-positives.[457]
- New diagnostic methods involving augmented RT-PCR,[458] exhaled breath condensate[459-460] and the microbiome[461] are being developed.
- Symptom-based screening at airports was ineffective at detecting cases (9 identified out of 766,044 passengers screened),[462] and intensive screening on a U.S. military base during mandatory quarantine did not identify any COVID-19 cases.[463]
- Infrared temperature readings may be misleading when used at the entrance of buildings with low outdoor temperatures.[464]
- Foam swabs lead to more accurate diagnostic tests than polyester swabs for collecting patient samples, though polyester swabs are adequate to be used in case of a shortage in foam swabs.[465]
- Immunological indicators,[299, 416, 466-475] blood glucose levels,[416] oxygen levels,[476] and bilirubin levels[477] may help identify future severe cases,[478] and tools for diagnosing severe infections[479-481] and predicting mortality[482] exist.
- Self- or caregiver-taken diagnostic swabs could be as accurate as those taken by healthcare workers in some instances.[483]
- Wearable technology may be able to detect COVID-19 days before symptoms begin,[484-485] and several attempts to create mobile applications for disease notification are underway.[486-487]
- Aerosol detection devices are capable of identifying SARS-CoV-2 in the air (minimum of approximately 6,000 particles).[488] Improvements to aerosol sampling protocols (e.g., use of fetal calf serum during elution, using polytetrafluoroethylene filters instead of glass fiber) may lower the limit of detection to 10-50 genome copies/m^3.[489]
- Patients with long-term or chronic COVID-19 appear to have auto-antibodies not present in patients who have recovered, sparking interest in developing a diagnostic blood test to identify the proteins.[490]

Validated serological (antibody) assays are being used to help determine who has been exposed to SARS-CoV-2.[491]

- Meta-analysis suggests that lateral flow immunoassays (LFIA) are less accurate than ELISA or chemiluminescent methods, but that the target of serological studies (e.g., IgG or IgM) does not affect accuracy.[492]
- LFIA testing showed lower accuracy in pregnant women than other patient cohorts.[493]

What do we need to know?

We need to identify additional factors that affect the accuracy of serological or PCR-based diagnostic tests.

- What is the relationship between disease severity and the timing of positive serological assays?
- Are certain subpopulations (e.g., those with blood cancers)[494] more likely to show false-negative tests?
- How likely are children of different ages to test positive via RT-PCR? 52-90% of cases in children are missed by RT-PCR.[362]

Medical Treatments – Are there effective treatments?

What do we know?

Detailed, real-time COVID-19 treatment recommendations are provided by the U.S. Centers for Disease Control and Prevention (CDC),[495] the World Health Organization (WHO),[496-497] the U.S. National Institutes of Health,[498] Infectious Disease Society of America,[499] and British Medical Journal[500] in regularly updated guidance documents based on ongoing analysis of evidence from clinical trials.

- A list of all current FDA approved, or FDA EUA authorized products for medical treatment of COVID can be found here.[501]
- Most people present with mild symptoms and the most common medical treatments are over the counter medicines such as acetaminophen (Tylenol) or ibuprofen (Motrin, Advil) to treat the symptoms of fever and discomfort.[495, 502]
- For hospitalized, critically ill patients on mechanical ventilation or extracorporeal membrane oxygenation (with organ failure and ARDS), dexamethasone is strongly recommended; if unavailable, use of alternative corticosteroids (hydrocortisone, methylprednisolone, prednisone) is recommended.[400, 503-508] Methylprednisolone may increase duration of viral shedding.[509]
- The WHO strongly recommends against convalescent plasma use for non-severe COVID-19 patients, but can be used in severe patients in clinical research settings.[510] The treatment fails to show benefits in large, randomized trials.[400]
- For patients with non-severe illness at highest risk of hospitalization, the WHO[511] and the CDC[495] recommends use of Paxlovid (nirmatrelvir-ritonavir). More recently bamlanivimab and etesevimab monoclonal antibodies have received an FDA EUA for treatment of these patients.[512]
- The WHO recommends against use of fluvoxamine and colchicine in patients with non-severe illness. Fluvoxamine can be used under clinical trial guidelines.[513]
- Veklury (remdesivir) may be considered for patients at high risk of severe disease[514] although prior clinical trial results found no benefit from remdesivir plus standard of care.[515] The WHO notes the conditional recommendation for use of remdesivir in patients with severe COVID-19, and conditional recommendation against use of remdesivir in patients with critical COVID-19.[513]
- The WHO recommends FDA approved Olumiant (baricitnib)[516] as an alternative to interleukin-6 (IL-6) blockers, with corticosteroids for severe or critical COVID-19 patients.[517] Baricitinib combined with standard of care reduced all-cause mortality.[518] Additionally, they recommend that IL-6 blockers can be combined with the baricitnib and corticosteroids for severe or critical COVID-19.[513]
- The WHO strongly recommends against use of Xevudy (sotrovimab) in patients with non-severe disease and recommends against use of REGEN-COV (casirivimab-imdevimab), reversing previous recommendation based on reduced activity to newer variants.[513]
- The FDA issued an EUA for Paxlovid (nirmatrelvir co-administered with ritonavir) for the treatment of mild-to-moderate COVID-19 in adults and children (≥12 years old).[519] This treatment showed an 89% reduction in risk of hospitalization and death in high-risk individuals.[520] A clinical trial of Paxlovid found non-significant reduction (51%) for either vaccinated or unvaccinated patients, suggesting limited benefits for those at low hospitalization risk.[521]
- Actemra (tocilizumab) is FDA EUA approved for certain children and young adults with severe acute COVID-19.[522-523]
- Clinical trials for Lagevrio (molnupiravir) show a 30% reduction in hospitalization and death,[524-525] increase in viral clearance, but no effect on symptom duration.[526] Potential side effects may include cancer and birth defects. The FDA has approved an EUA in mild-to-moderate cases in adults ≥18 years old, when no other FDA approved drugs are available.[527-528]
- Preliminary results suggest high-dose anticoagulants may reduce mechanical ventilation rates for mild-to-moderate COVID-19.[529] The WHO recommends a standard dosing level,[530] as high anticoagulant doses were not more effective at improving outcomes for critically ill patients.[531-532]
- Prior use of statins,[533-534] renin-angiotensin-aldosterone system inhibitors,[535] anticoagulants,[536] non-steroidal anti-inflammatory drugs (NSAIDs),[537] and ACE inhibitors[538] do not appear to elevate COVID-19 risk, and potential benefits of aspirin use require assessment in a clinical trial.[539]

What do we need to know?

We need clear, randomized trials for treatment efficacy in patients with both severe and mild/moderate illness.
- What treatment, or combination of treatments, is most effective for different disease severities and patient demographics?
- What is the efficacy of transmission-blocking peptides[540] and nasal sprays in humans?[541]
- Are inflammation inhibitors effective at improving the outcome of COVID-19 associated hyperinflammation?[542]

Vaccines – Are there effective vaccines?
What do we know?

Four safe and effective vaccines are currently being administered in the U.S., two with full FDA approval (Pfizer/BioNTech and Moderna) and two under FDA EUA (Johnson and Johnson/Janssen and Novavax).[543] All four vaccines have been approved as booster doses, and all but Novavax can serve as a booster regardless of the initial vaccination type.[543] In addition, both Pfizer and Moderna have developed 'bivalent' boosters that include components of the wild-type strain, as well as the BA.4 and BA.5 lineages of the Omicron variant.[544]

- As of 5/11/2023, 230.64 million in the U.S. were fully vaccinated against the wild-type strain of SARS-CoV-2, and 56.4 million have received one of the updated bivalent boosters.[545]
- In June 2023, the FDA's VRBPAC met to review safety, efficacy, and immunogenicity of all vaccines in conjunction with circulating strains to provide guidance for the composition and schedule of vaccinations in 2023-2024. The committee unanimously voted that a monovalent COVID vaccine with an XBB-lineage of the Omicron variant (XBB 1.5 is recommended) should be used for all vaccinations starting in fall 2023.[546-547]
- In August 2022, the FDA authorized Omicron-specific "bivalent boosters," which include the original vaccine as well as BA.4 and BA.5 components of Omicron, by both Pfizer and Moderna.[544, 548] Initial studies showed increased neutralizing antibody responses against Omicron variants,[544, 549-552] and early real-world efficacy data indicates the bivalent booster (after 2 to 4 monovalent doses) provides reductions in hospitalization of up to 56% in adults compared to no vaccination.[553] Most hospitalizations have occurred in individuals older than 65 and those with underlying conditions, whether vaccinated or not.[554]
- While the efficacies of the initial monovalent vaccines from Pfizer and Moderna were high (91.3%[555] and 94.1%[556] respectively) and consistent across age,[557] race, ethnicity, and sex,[556] protection from the monovalent vaccines dropped to 20-25% once Omicron became the prominent circulating strain. Introducing bivalent vaccines or boosters raised the efficacy against symptomatic COVID to 59-73%,[558-559] thereby changing the vaccination strategy going forward.
- In April 2023, the FDA simplified the vaccination schedule for Pfizer and Moderna by amending the EUA and authorizing only their bivalent (wild-type virus plus Omicron strain) vaccines for all vaccinations, initial and booster, for all individuals 6 months and older. Both companies' initial vaccines are no longer authorized for use in the U.S.[560-561]
- The CDC prepared a summary table of all U.S. COVID vaccine manufacturers and dosing schedules per age group.[562]
- Vaccination has provided robust protection from infection,[563-565] evidenced by low rates of breakthrough infections.[566] Vaccinated individuals generally experience milder symptoms than unvaccinated individuals.[567-569]
- Vaccination is safe and efficacious in pregnant women, with no increase in risk of adverse pregnancy or fetal or neonatal outcomes. Vaccination during pregnancy provides protection through placental transfer, as well as through breastmilk.[411]
- **Pfizer/BioNTech** - mRNA vaccine named BNT162b2 (Comirnaty).[68] FDA approval[571] and WHO Emergency Use Listing (EUL).[572]
  - Depending on vaccination history, children 6 months-4 years old receive up to 3 doses bivalent vaccine, and individuals 5 years and older receive up to 1 dose bivalent vaccine (full detailed vaccination schedule provided in reference).[562]
- **Moderna** - mRNA vaccine named mRNA-1273 (Spikevax or Elasomeran).[573, 574-575] Approved by the FDA,[576] also approved in Canada[577] and European Union (EU),[578] and WHO EUL.[579]
  - Depending on vaccination history, children 6 months-5 years old receive up to 2 doses bivalent vaccine, and individuals 6 years and older receive up to 1 dose bivalent vaccine (full detailed vaccination schedule provided in reference).[562]
- **Johnson & Johnson/Janssen** - adenovirus vaccine[580] named Janssen COVID-19 vaccine. U.S. EUA[581] and approved in EU.[578]
  - In clinical trials, the vaccine was 77-85% effective at preventing severe and critical COVID-19, and 67% effective at preventing moderate to severe COVID-19 prior to the Omicron variant.[581]
  - This vaccine is limited to adults 18 and older without access to any other vaccine or who would otherwise receive no vaccine at all, due to the risk of thrombosis with thrombocytopenia syndrome.[581-582]
- **Novavax -** a protein subunit-based vaccine named NVX-CoV2373, Covovax, or Nuvaxovid depending on location.[583] There is FDA EUA for adults over 18 and adolescents 12-17 years old,[584-586] as well as conditional approval by the European Commission and the WHO.[587-588] This vaccine is the first protein-based COVID vaccine available in the U.S.
  - The vaccine is given as 2 shots, 21 days (U.S.) or 8 weeks (EU) apart. In the U.S., it is recommended as a 2-dose primary series for children 12 and older, with a booster for adults 18 and older, 6+ months after the primary series.[589]
  - Novavax's vaccine shows 90% efficacy against mild, moderate, and severe disease in adults 18 and older,[587] and 80% effective in adolescents 12-17 years old prior to the Omicron variant.[583-584]

What do we need to know?

We need to understand the long-term impact of SARS-CoV-2 variants on vaccine efficacy and the need for boosters.
- What are the correlates between neutralizing antibody levels and vaccine-induced efficacy in humans?
- How protective are vaccines in those taking immunosuppressants[590] or with autoimmune disorders?

Non-Pharmaceutical Intervention (NPI) – Are public health control measures effective at reducing spread?
What do we know?

Broad-scale control measures such as stay-at-home orders and widespread face mask use effectively reduced transmission early in the pandemic, though the enhanced transmissibility of the Omicron variant has limited effectiveness of some measures.

- Social distancing and other policies quickly reduced spread of wild-type SARS-CoV-2 throughout China,[591-596] Europe,[597-598] and the U.S.[599-600] Delaying control measures increases outbreak duration,[601] cases,[602] mortality,[603] and effective viral population size.[604]
- U.S. states[605] with mask mandates had lower case growth rates and higher likelihoods of controlling transmission.[606] Modeling shows slight increases in mask adherence can reduce simulated infections.[607] In the U.S., shelter-in-place orders[608] and restaurant and bar closures were associated with large reductions in exponential growth rate of cases.[609]
- Mobility reductions in the U.S. have been associated with reductions in COVID-19 case growth.[610-611] Social distancing and reductions in both non-essential visits to stores and overall movement distance led to lower transmission rates.[612]
- Reductions in transmission appear 6-9 days after the implementation of NPIs, and increased transmission is visible 14-20 days after NPIs are lifted.[613] Re-opening restaurants in the U.S. was associated with significantly higher mortality 61-100 days after relaxation of restrictions in a largely unvaccinated population.[614]
- Reducing capacity at crowded indoor locations,[616] increasing indoor air flow rates,[617] adding portable air cleaners,[618] and wearing masks may reduce indoor transmission.[619] Aerosol infection risk is not uniform in indoor environments, and can be greatly impacted by patterns of ventilation.[620]
- NPIs and societal aid focused on populations at highest risk of infection, such as those living or working in crowded environments, are more effective than NPIs that assume equal risk of infection and transmission across subpopulations.[621] Similarly, targeting NPIs at those in high-risk occupations may help reduce COVID-19 burden.[622]
- Layered control strategies in Shanghai were overwhelmed during a period of high importation rate of new Omicron variant cases in early 2021; only after a city-wide lockdown did transmission rates fall substantially.[623] Similarly, regular testing, isolation, case investigation, contact tracing, and quarantine at a U.S. university was insufficient to stop a large outbreak of the Omicron variant, despite substantial levels of vaccination among students.[624]

Individual behaviors (e.g., face masks, social distancing) have been associated with reduced risk of COVID-19 infection.[625-626]

- Face masks inhibited transmission by both reducing the number of exhaled particles from infectious individuals, as well as reducing the number of inhaled particles when worn by uninfected individuals.[627] The safety efficacy of masks depends largely on the type of mask, the way it is worn, and the overall fit.[628] A large analysis across 56 countries found that mask wearing reduced the mean transmission rate by 19%.[629] In a study of K-12 school districts across nine states, those with universal masking policies reported 3.6 times fewer secondary infections than those with optional masking policies.[630]
- A cross-sectional online survey found NPIs such as using sanitizer, quarantine, and isolation can decrease incidence and mortality of COVID-19.[631]

Particular focus should be placed on minimizing large gatherings where superspreading events are more likely.[632-633]

- Eliminating superspreading events[634] can result in slower case growth while easing broadly restrictive interventions.[635] Focusing interventions on high-risk activities or locations (e.g., gyms, bars, and restaurants) may help reduce transmission.[636]
- Reducing community prevalence, increasing ventilation,[637] and universal testing can reduce spread in schools.[638]
- As children are estimated to be less susceptible to SARS-CoV-2 infection, school closures are relatively ineffective NPIs,[639] though there is some evidence they were impactful at the national level.[615, 640] Modeling shows that masks, increased ventilation, and portable air purifiers are effective at reducing infection risk.[641]

Research is needed to plan the path to SARS-CoV-2 elimination via pharmaceutical and NPIs.

- Travel restrictions may be effective in certain conditions, such as when countries have low incidence themselves.[642] Travel restrictions, though, are only effective at reducing the importation of novel variants if effectively implemented in a short time window; quarantines for travelers may be more broadly effective at reducing variant importation risk.[643]
- Researchers have proposed an Omicron variant model to provide insights to coordinate NPIs and vaccination.[644]

Lifting NPIs before widespread vaccine uptake is predicted to increase COVID-19 cases and deaths.[645-646]

- Modeling suggests that NPIs will need to be in place for 6-12 months after the initiation of vaccination campaigns.[80, 647] NPIs and vaccines work synergistically to reduce disease burden, and both are needed when vaccine coverage rates are low.[648-649]
- Modeling shows that NPIs can reduce the likelihood of vaccine-resistant variant emergence, as the simulated emergence of vaccine-resistant strains was highest when vaccination levels were high (60%), but transmission was uncontrolled.[650]
- A modified susceptible-exposed-infectious-recovered (SEIR) modeling is proposed to assess the effectiveness of social distancing, ban on gatherings, and vaccination strategies; however, this study was specifically for Seoul, South Korea and additional modeling is necessary to assess efficacy of NPIs in other regions .[651]

What do we need to know?

We need to understand the magnitude of measures necessary to limit spread of new SARS-CoV-2 variants.

- How does NPI effectiveness change over time as a result of changes in adherence or behavior?

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

What do we know?

SARS-CoV-2 variants display differences in environmental stability.
- Variants Alpha, Beta, Delta, and Omicron subvariants BA.1 and BA.2 have higher environmental stability on human skin and plastic (13 and 141 hours, respectively) compared to the wild-type strain, with greatest stability recorded in Omicron.[59]

The stability of SARS-CoV-2 in biological fluids of animals was measured.
- SARS-CoV-2 is stable for up to 1 day in the saliva of cats, sheep, and white-tailed deer regardless of the environmental conditions. It can remain infectious for up to 6 days in feces and 15 days in fecal suspension of white-tailed deer, whereas the virus is rather unstable in cat and sheep feces. The longest survival of SARS-CoV-2 was in the urine of cats, sheep, and white-tailed deer.[652]
- Side-by-side comparison with different SARS-CoV-2 strains showed that the Alpha, Delta, and Omicron variants were less stable than the wild type-like strain in white-tailed deer fecal suspension.[652]

SARS-CoV-2 can survive on surfaces from hours to days and is stable in air for at least several hours, depending on UV light, temperature, and humidity.[653] Viable SARS-CoV-2 and/or RNA can be recovered from contaminated surfaces; however, transmission via contaminated surfaces is not considered to be common.
- Both temperature and humidity contribute to SARS-CoV-2 survival on non-porous surfaces, with cooler, less humid environments facilitating survival (stainless steel, ABS plastic, and nitrile rubber; indoors only; simulated saliva matrix).[654] Persistence is reduced with warmer temperatures (37°C), and enhanced at colder temperatures (4°C).[655]
- SARS-CoV-2 was shown to be stable for 3 days (21-23°C, 40% RH)[656] up to 7 days (25-27°C; 35% RH) on smooth surfaces, such as: plastic, stainless steel, glass, ceramics, wood, latex gloves, and surgical masks.[657] Infectious virus can be recovered from a surgical mask after 7 days (22°C, 65% RH)[658] and other PPE for at least 72 hours at 22°C.[659]
- In indoor environments, infectious virus persisted on cloth for up to 1 day; on steel and concrete for up to 3 days; and on nitrile, Tyvek, N95 respirators, Styrofoam, cardboard, rubber, and glass for up to 4 days.[660]
- It is important to realize that SARS-CoV-2 detection by various methods does not confirm the presence of live virus, with a recent study finding no live virus on banknotes.[655]

In the absence of sunlight, SARS-CoV-2 can persist on surfaces for weeks.
- In the absence of sunlight, infectious SARS-CoV-2 can remain on non-porous surfaces for at least 28 days (20°C; 50% RH); higher temperatures greatly reduce the environmental stability of SARS-CoV-2.[661] This value is longer than other stability estimates,[656, 658, 661] potentially due to a fluid matrix with more protein to simulate human respiratory fluid and a higher inoculation dose.[661] In simulated saliva on stainless steel surfaces, SARS-CoV-2 shows negligible decay over 60 minutes in darkness, but loses 90% of infectivity every 6.8-12.8 minutes, depending on simulated UVB radiation.[662]
- SARS-CoV-2 was shown to have an aerosol half-life of 2.7 hours (without sunlight, particles <5 µm, tested at 21-23°C and 65% RH),[656] retaining infectivity for up to 16 hours in appropriate conditions (23°C, 53% RH, no sunlight).[663]

SARS-CoV-2 survival in the air is highly dependent on the presence of UV light and temperature.
- Experimental studies using SARS-CoV-2 aerosols 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).[664] In dark conditions, the half-life of aerosolized SARS-CoV-2 is approximately 86 minutes in simulated saliva.[664] Humidity alone had no significant impact on aerosolized virus survival.[664]

There is currently no evidence that SARS-CoV-2 is transmitted to people through food or food packaging.[665-666]
- SARS-CoV-2 can persist for at least 2 weeks at refrigerated temperatures (4°C),[658, 667] and the spike G614 variant remains infectious at 4°C for over 30 days.[668] SARS-CoV-2 maintains infectivity for at least 30 days when inoculated on frozen foods and stored at -20°C.[668] Infectious SARS-CoV-2 has been found on frozen food packaging, but has not been linked to actual infections.[669] Several outbreaks have a hypothesized food origin.[670]
- Studies have shown that cold-chain transportation and salt were risk factors that could prolong SARS-CoV-2 viability due to the low freezing point of seawater, which slows the rate of freezing and thawing of the virus and effectively maintains the structural integrity. This suggests that when transporting seafood and other goods that may contain seawater, the outer packaging of the goods should be properly washed in advance to reduce the presence of salt ions, which may reduce the risk of virus transmission from food sources.[671-672]

SARS-CoV-2-contaminated wastewater may cause infections,[673-674] particularly in areas of poor waste management, and via landfill leachate, in which the virus may be transmitted to humans from pollution.[675]
- Surrogate studies of human and animal coronaviruses suggest wastewater is not conducive to long-term virus survival, as coronaviruses undergo spontaneous and progressive inactivation because of temperature and organic or microbial pollution.[676] SARS-CoV-2 RNA was found in untreated liquid and solid waste systems (infectious virus was not quantified), though typical treatment effectively eliminated viral RNA from effluent.[677-678]

What do we need to know?

We need to quantify the duration of viable SARS-CoV-2 on surfaces, not simply the presence of RNA.
- Are certain SARS-CoV-2 variants more or less stable on surfaces or in aerosols?

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.

- While a 4-5 log (99.99-99.999%) reduction in viral titer is often used as a metric of effective decontamination,[679-680] achieving this level of reduction is dependent on methodological issues like the initial viral stock concentration.
- A systematic review identified sunlight, UV light, ethanol, hydrogen peroxide, and hypochlorite as methods to reduce surface contamination.[653] However, the levels of decontamination necessary to affect transmission *per se* are still unknown.[653] Selected studies found positive results favoring photo-irradiation as an antiviral therapy, but the best dosimetry, safety, and efficiency of light as a tool for decontamination are variable and related to initial viral load, radiant exposure, surface characteristic, relative humidity, light equipment model, distance from irradiation, and wavelength.[681]
- Chlorine bleach (1%, 2%), 70% ethanol, and 0.05% chlorhexidine are effective against live virus in laboratory tests.[658] Disinfection using 1% sodium hypochlorite dispersed by cold fogging significantly reduces the amount of disinfectant used without compromising inactivation.[682]
- The U.S. Environmental Protection Agency has released a list of SARS-CoV-2 disinfectants that are effective against all strains and variants of SARS-CoV-2.[683]
- Twice-daily cleaning with sodium dichloroisocyanurate decontaminated surfaces in COVID-19 patient hospital rooms.[684] Regular disinfection of hospital rooms (with benzalkonium wipes) can reduce the presence of SARS-CoV-2 on surfaces.[685]
- Heat, soap, and ethanol were also able to decontaminate SARS-CoV-2 variants (Alpha and Beta) on various surfaces.[686] Increased resistance to ethanol was demonstrated by Alpha, Beta, Delta, and Omicron strains; however, all strains were completely inactivated by 35% ethanol after 15 seconds.[59]
- Steam heat can provide high levels of decontamination (>3 log reduction) for transit-related materials contaminated with SARS-CoV-2 using a commercially available steam generator with a manageable exposure time of 2-5 seconds.[687]
- Mass public transit systems treated with Natural Protective Shield 360°, a self-decontaminating coating, showed 100% reduction of SARS-CoV-2 for up to 20 days post-application.[688]
- Free chlorine used in wastewater treatment successfully and rapidly disinfects SARS-CoV-2.[678]
- Concentrations of 10-20 mg/mL ZnO-NP-45 (Zinc Oxide Nanoparticles) can inactivate Delta and Omicron SARS-CoV-2 variants by a factor of more than 10^6.[689]

Several methods exist for decontaminating N95 respirators[690] and other PPE.

- Researchers have identified three methods capable of decontaminating N95 respirators while maintaining physical integrity: UV radiation, heating to 70°C, and vaporized hydrogen peroxide (VHP).[691-692] Dry heat and UV decontamination can also be used under certain conditions.[693] Ethanol (70%) was associated with loss of physical integrity.[691]
- Germicidal UVC decontamination methods for N95s was successful when both sides were irradiated for a minimum of 120 seconds at 1.3 Joules/cm^2 (yielding a 3.5 log reduction) and can be scaled for large-scale decontamination efforts.[694]
- Several decontamination methods, including VHP, UVC,[695] moist heat,[696] and dry heat[697] are capable of decontaminating N95 respirators for 2-15 cycles without loss of fit or filtration efficiency. Overall wear time and number of uses are the primary contributors to functional degradation.[699]
- Shape and structure of specific N95 models impact the efficacy of UVC decontamination methods.[700]
- A thermal inactivation model for SARS-CoV-2 provides estimates of infectivity reduction based on time and temperature.[701]
- Medical mask material inoculated with SARS-CoV-2 was treated with light-activated methylene blue (MB) at 10 μg concentration, then exposed to white light at 50,000 lux, which inactivated SARS-CoV-2 in 5 minutes.[702] Pretreatment of mask material with MB successfully disinfected masks following virus inoculation and subsequent light exposure.[702]

Air filtration and purification systems may reduce concentration of airborne SARS-CoV-2.

- High efficiency particulate air (HEPA) filtration systems have consistently outperformed other methods of air purification.[704]
- Indoor air filters based on non-thermal plasma or reactive oxygen species may be effective at reducing circulating SARS-CoV-2 concentrations, estimated by reductions in surrogate virus, though additional testing on live SARS-CoV-2 virus is needed.[705] UV light combined with a fibrous filter was ~100% efficient at removing aerosolized 0.1-2.5 μm particles of SARS-CoV-2.[706] Various materials like ozone, hydrogen peroxide, alcohol, and titanium dioxide were described in these studies to disinfect places contaminated by SARS-CoV-2.[707]
- Air purification via photoelectrochemical oxidation utilizes reactive oxygen species to inactivate viruses, bacteria, and molds. Although untested with SARS-CoV-2, Molekule air purification technology is approximately 99% effective at inactivating M2 bacteriophage virus, which is smaller than SARS-CoV-2.[708]

What do we need to know?

We need additional SARS-CoV-2 decontamination studies, particularly regarding indoor aerosol transmission.

- Does contamination with human fluids/waste alter disinfectant efficacy profiles?
- How do different testing methods and standards affect decontamination efficacy estimates?
- Are specific decontamination methods needed for different SARS-CoV-2 variants?

Personal Protective Equipment (PPE) – What PPE is effective, and who should be using it?

What do we know?

Recommended PPE for healthcare workers caring for patients with COVID-19 includes respirator (N95 or higher), face shield or goggles, gown, and clean gloves, combined with regular hand hygiene.[709]

Face masks appear effective at reducing infections from SARS-CoV-2. Healthcare workers are at elevated risk of acquiring COVID-19, even with recommended PPE.

- Contacts with healthcare workers tend to transmit COVID-19 more often than other casual contacts.[710] Risk of COVID-19 infection was higher when healthcare workers were exposed to an infected coworker than when exposed to patients.[711]
- 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.[712]
- Healthcare worker self-infection during doffing of PPE positively correlated with length of shift and number of positive COVID-19 patients in the ward.[713]
- Respirators (National Institute for Occupational Safety and Health [NIOSH]-certified N95, EUFFP2 or equivalent) are recommended for those working with potential aerosols,[714] though procedure type is not the only factor influencing risk of aerosol generation in hospitals.[715] Additional protection (powered air purifying respirator [PAPR] with hood), should be considered for high-risk procedures.[716]
- In a meta-analysis, N95 respirators were found to be beneficial for reducing the occurrence of respiratory illness in health care professionals including influenza and SARS-CoV-2, though surgical masks were similarly effective for influenza.[717-718] N95 respirators were associated with up to 80% reductions in SARS-CoV-1 infections.[717-718]
- 85% of tested N95 respirators passed fit tests after at least five cycles of standard donning/doffing and dry heat decontamination,[719] though extended use has previously been associated with fit failures.[720]
- Mathematical modeling suggests that mask efficacy depends heavily on the aerosol concentration of SARS-CoV-2, with higher efficacy in situations with lower aerosol concentrations; pairing mask use with other interventions that reduce aerosol concentrations, such as increasing ventilation, can greatly reduce transmission risk.[721]

A recent meta-analysis compared protection from COVID-19 infection across N95, surgical, and non-medical masks worn with high compliance versus low compliance/no mask. Overall increased protection was shown for an uninfected individual wearing any type of mask with high compliance, with greatest protection from N95 masks, then surgical, then non-medical masks.[718]

- As of September 27, 2022, the CDC recommends that individuals with symptoms, a positive test, caring for someone who has COVID-19, or recent exposure should wear a mask, as well as individuals using indoor public transportation.[722-724]
- The CDC maintains a list of NIOSH-tested facemasks,[725] and also maintains a list of single-use and reusable masks that meet updated ASTM F3502-21 standards.[726]
- In a prospective cohort study in the U.S., the secondary attack rate of SARS-CoV-2 was higher when at least one person was not wearing a face mask compared to when both individuals were wearing a mask (25.6% vs. 12.5%, respectively).[727]
- Surgical face masks, respirators, and homemade face masks may prevent transmission of coronaviruses from infectious individuals to other individuals.[728-730] Surgical masks were associated with a reduction of approximately 48% in the amount of seasonal coronavirus expressed as fine aerosol particles (<5 μm) and 77% in coarse aerosols (>5 μm), though the majority of viral RNA was exhaled in fine aerosol particles.[731-730] Homemade masks reduce overall flow from breathing and coughing (63-86% reduction), but also generate leakage jets facing downward and backward from the wearer's face.[732]

Mask fit is another critical component of effectiveness,[733] in addition to filter efficiency.

- Fit modifications are recommended if warranted, such as knotting ear loops, tucking in sides of mask, and double-masking.[734] Wearing a plastic brace around the sides of the mask blocked ≥95% of aerosols produced by coughing compared to ≥56% in unmodified masks.[734]
- The CDC recommends masks without exhalation vents or valves,[735] as these can allow particles to pass through unfiltered.[736]
- A 4-mm hole in an N-95 mask demonstrated up to a 10% increase in 1-4 μm particles passing into the mask.[737]
- When properly fitted, surgical masks provide infection control at the source, whereas N95s and PAPRs provide respiratory protection.[737]

What do we need to know?

We need to continue assessing PPE effectiveness with specific regard to SARS-CoV-2 instead of surrogates.

- Can mask efficacy be predicted from material composition?
- What is the efficacy of combining multiple facemasks compared with single multilayered masks?[738]
- What is the risk of COVID-19 to those collecting nasopharyngeal swabs?[739]
- Should decontamination methods be optimized for individual makes/models of PPE?

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

What do we know?

While ancestral SARS-CoV-2 accumulates mutations at a similar rate as other coronaviruses, variants possess genomes that accrue at approximately two mutations per month. Molecular clock models indicate that variant emergence has been driven by a substitution rate increase of approximately 4-fold the background phylogenetic rate estimate.[740] A novel near-neutral balanced selection theory has been proposed to explain the genomic substitution rate of SARS-CoV-2 which leads to a molecular clock feature.[741]

- The estimated mutation rate for SARS-CoV-2 is 26.9 nucleotides per genome, per year.[742] Another group reviewed literature and found the rate of evolution to be from 10^{-3} to 10^{-4} substitutions per site per year but varied among variants.[743] Yet another group reviewed literature and found the mutation rate to be from 1×10^{-6} to 2×10^{-6} mutations per nucleotide per replication cycle, but was estimated to acquire about 2 evolutionary changes per month or 2×10^{-6} per site per day.[169]
- More than 6 million SARS-CoV-2 genomes were analyzed to identify mutations associated with virus transmissibility, which include mutations in spike, nucleocapsid, and nonstructural proteins.[744]
- The CDC currently considers Omicron (including subvariants) the only variant of concern (VOC) in the U.S.[746]

B.1.1.7 (20I/501Y.V1) (VOC202012/01) (Alpha) – The B.1.1.7 variant is associated with 50-75% higher transmission than wild-type virus.[747-748] Contains several spike protein mutations (HV 69-70 deletion, N501Y, N493K).[749]

- The E484K mutation has appeared independently in individuals with the B.1.1.7 variant in the UK[754] and U.S.[755]

B.1.617.2 (Delta) – Initially identified in India in January 2021, contains several mutations of concern (E484Q and L452R).[756]

- B.1.617.2 has 13 sublineages; of these, AY.1 and AY.2 possess a mutation of concern at K417N that is also present in Beta and Gamma variants.[761] This mutation affects class I antibody binding[762-763] and reduces affinity to ACE2, but stabilizes ACE2 binding in the presence of the E484K mutation.[763-764]
- In October 2021, the UK identified a Delta subvariant (B.1.617.2.4.2; AY.4.2) that was associated with an increase of cases with a secondary attack rate of 12.4%.[765]

B.1.351 (20H/501Y.V2) (Beta) – Identified in South Africa in December 2020[766] with mutations N501Y, E484K, and K417N.[767]

- This variant is resistant to neutralization from convalescent plasma and vaccine recipient sera.[768] Convalescent serum from patients with B.1.351 infection shows high neutralization ability against wild-type virus.[769] The B.1.351 variant is partially resistant to monoclonal antibody casirivimab and is fully resistant to bamlanivimab.[757]

P.1 (20J/501Y.V3) (Gamma) – First identified in Brazil;[770] contains K417N, E484K, and N501Y mutations.[770]

- The variant is estimated to be 1.7-2.4 times more transmissible than wild-type SARS-CoV-2.[771]
- Less resistant to neutralization than Beta, suggesting RBD mutations are not the only factor influencing variant immune escape.[772] The H655Y mutation is associated with reduced neutralization,[773] and has arisen in animal models.[774-775]

C.37 (Lambda variant) – *In vitro* studies suggest two single mutations (T76I, L452Q) make the Lambda variant more infectious than wild-type virus, while a deletion mutation (RSYLTPGD246-253N) increases antibody resistance.[776]

B.1.429 (Epsilon) ([CAL.20C (20C/S:452R)] [GH/452R.V1 (B.1.429+B.1.427)]) – L452R[777] mutation located on the spike protein was first reported in Denmark[778] and increased in prevalence in California.[779] The B.1.429 lineage is more transmissible and leads to more severe disease than wild-type SARS-CoV-2,[780] and is partially resistant to antibodies.[781-782]

B.1.621 (Mu) – Includes B.1.621.1. Mutations of note in spike: E484K, N501Y, D614G, P681H. Preliminary studies suggest the Mu variant is resistant to convalescent patient sera and sera from those vaccinated with the Pfizer/BioNTech vaccine.[783-784]

B.1.1.529 (Omicron) – Detected on November 26, 2021 in South Africa and Botswana, the Omicron variant includes 21 unique mutations in the spike gene with 14 shared with other prior variants.[785] The location of many of these mutations may play a role in ACE2 binding and antibody recognition.[785] The risk of severe outcomes is greatly reduced in patients infected with Omicron versus Delta strains.[786, 789] In March 2023, the WHO noted that over 98% of available sequences since February 2022 were Omicron, which prompted changing the variant tracking system to track Omicron variants independently.[787]

- BA.4 and BA.5 first detected at the end of April 2022; these two sublineages outcompeted BA.2 and BA.2.12.1.[790]
- BA.5 has shown a transmission advantage over prior Omicron variants, lower neutralizing antibody titers compared to BA.1, and reduced neutralization activity to sotrovimab and casirivimab/imdevimab.[793]
- Further analysis of subvariants of BA.4/5 suggest they continue to evolve to escape neutralizing antibodies.[794]
- A BA.2.10.1 and BA.2.75 recombinant, XBB, is highly immune evasive.[64-65, 425]
- XBB.1.5 has a mutation that increases receptor binding affinity and infectivity.[795]
- Currently, XBB.1.5 and XBB.1.16 are listed as variants of interest (VOIs) and BA.2.75, CH.1.1, XBB, XBB.1.9.1, XBB.1.9.2, and XBB.2.3 are listed as variants under monitoring (VUMs) by the WHO.[796]
- XBB.1.5 and XBB.1.9 predominated worldwide in February 2023. XBB.1.16 emerged from XBB.1.5.[797] XBB.1.16 has a reproductive number 1.13 fold higher than XBB.1.5.[797]
- The most prevalent strain in the U.S. as of 7/11/2023 is XBB.1.16.1. It is estimated that ~99% of sequenced strains in the U.S. since March 2023 have been Omicron variants.[56, 797]

What do we need to know?

We need to identify differences in transmissibility or severity caused by different SARS-CoV-2 mutations and variants.

- What are the mechanisms driving the resistance of variants to neutralization by the immune system?
- How do variants affect the likelihood of reinfection or coinfection?
- How prevalent are coinfections with multiple strains, and what is their clinical progression?[798]

Forecasting – What forecasting models and methods exist?

What do we know?

Several platforms provide digital dashboards summarizing the current status of the pandemic in U.S. states and counties.

- The CDC maintains a dashboard of state-level COVID-19 vaccination data for first and second doses.[545]
- COVID Act Now: State and county-level dashboard showing trends in four metrics related to COVID-19 risk.[799]
- Maps and dashboards depicting COVID-19 infection rates do not necessarily increase likelihood of adhering to NPIs; additional information is needed to influence perceptions of individual risk.[800]

The CDC provides ensemble forecasts of fatalities based on the average of many participating groups.[802] Ensemble forecasts generally show better predictive accuracy than individual forecast models.[803]

- The CDC no longer provides forecasts of COVID-19 cases, as too many observations were falling outside of forecast intervals (i.e., forecast accuracy was generally low).[804] The CDC National SARS-CoV-2 Genomic Surveillance system collects sequences to rapidly identify variants and make short term predictions on variant prevalence using Nowcast.[56]
- Columbia University Model: Spatially explicit SEIR model incorporating contact rate reductions due to social distancing.[805]
- Johns Hopkins University Infectious Disease Dynamics Lab: Modeling to rapidly assess impact of different interventions.[806]
- Northeastern University: Spatially explicit, agent-based epidemic model used to forecast fatalities, hospital resource use, and the cumulative attack rate for unmitigated and mitigated scenarios.[807-808]
- Several groups have evaluated wastewater to determine the relationships between SARS-CoV-2 in wastewater and COVID-19 clinical cases and hospitalizations.[809-812] Wastewater may be useful in forecasting local SARS-CoV-2 prevalence[647] and early identification of variant spread.[813]

Additional forecasting efforts were designed to assess the effects of interventions such as social distancing and vaccination.

- U.S. COVID-19 cases could be estimated under different scenarios of vaccine efficacy and need for NPIs such as face masks and physical distancing.[814]
- Prioritizing vaccines for elderly individuals with high COVID-19 mortality maximizes life-years saved by vaccination,[817] though prioritization of older or younger individuals for initial vaccine distribution may depend on the stage of the pandemic in a location. Vaccination focused on interrupting transmission (prioritizing younger individuals) may reduce mortality more than prioritizing vulnerable groups when vaccine campaign initiation is delayed.[818]
- CovidSim: SEIR model allow users to simulate effects of future intervention policies at state and national levels (U.S. only).[819]
- Covasim: Agent-based model for testing effects of intervention measures, also available as Python library.[820]
- Researchers use a rolling window analysis incorporating uncertainty in the generation time distribution to estimate time-varying transmission rates in U.S. states (the effective reproduction number, R_{eff} or R_t).[822]
- Accounting for superspreading in forecast models can increase model accuracy and precision,[823] while incorporating memory effects (e.g., the duration of individual infectiousness) can also increase forecast model fit to data.[824]
- New approaches may be needed to forecast COVID-19 re-emergence after elimination or during periods of low incidence.[825]
- Coronaviruses have generally been considered 'winter viruses', and seasonal variations have been noted.[826]
- A two-parameter Weibull-distribution applied to case data and swab-testing rates can be used to predict infection rates.[827]
- An agent-based and extended SEIR model was developed to forecast future waves in Japan that considered vaccination, virus mutation, government policies, and PCR test results.[828]
- A group used a Bayesian latent variable model to predict short term trends in cases and hospitalizations regionally in Wisconsin.[830] A two-component model was developed to forecast healthcare demands using a mathematical epidemiology model that fed into a clinical pathway model.[831]
- SARS-CoV-2 co-infection or interaction with another pathogen may be important for future risk modeling.[833]

There are several efforts aimed at forecasting important SARS-CoV-2 mutations in an effort to predict emerging variants and their characteristics.

- Characteristics of SARS-CoV-2 mutations (e.g., prevalence, receptor binding ability, relationship to immune escape) may enable forecasting which mutations are likely to show up in future variants months before their emergence.[835-836]
- Researchers have developed an early warning system for variant emergence based on ongoing analysis of SARS-CoV-2 mutations.[837] Improved prediction models correctly projected Omicron BA.4 and BA.5 to become predominant variants.[838]
- The CoVigator tool was developed to collect genomic data, process/denotate variants, and highlight findings as a way to track variant emergence.[839]

What do we need to know?

- How will different vaccine uptake rates, spillover, and movement between countries affect local resurgence?
- To what extent does waning immunity, changes in human behavior, and seasonal changes affect COVID-19 transmission rates with the Omicron variant?
- How accurate are/were the model predictions? Which factors in the model systems should be used during future waves/pandemics?

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 that 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.
CDC	Centers for Disease Control and Prevention	N/A
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.
DHS S&T	U.S. Department of Homeland Security's Science and Technology Directorate	N/A
DNA	Deoxyribonucleic Acid	Molecule that encodes genetic information.
Droplet transmission	Sneezing, coughing	Transmission via droplets requires relatively close contact (e.g., within 6 feet).
EC	European Commission	N/A
ELISA	Enzyme-Linked Immunosorbent Assay	Method for serological testing of antibodies.
EPA	U.S. Environmental Protection Agency	N/A
EU	European Union	N/A
EUA	Emergency Use Authorization	N/A
FDA	U.S. Food and Drug Administration	N/A
Fomite	Inanimate vector of disease	Surfaces such as hospital beds, doorknobs, healthcare worker gowns, and faucets.
HEPA	High Efficiency Particulate Air	An efficiency standard for air filters.
HVAC	Heating, Ventilation, and Air Conditioning	N/A
ICU	Intensive Care Unit	N/A
ID_{50}	Median Infectious Dose	The dose necessary to infect 50% of the target population. Generally, assumes typical, healthy, adult individuals.
Ig	Immunoglobulin	Proteins of the immune system that can act as antibodies.
IL-6	Interleukin-6	A cell signaling protein that can act ither as pro- or anti-inflammatory.
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.

Acronym/Term	Definition	Description
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.
LD_{50}	Median Lethal Dose	The dose necessary to be lethal in 50% of the target population. Generally, assumes typical, healthy, adult individuals.
MERS	Middle East Respiratory Syndrome	Coronavirus with over 2,000 cases in regional outbreak since 2012.
MIS	Multisystem Inflammatory Syndrome	A rare but serious condition that is associated with inflammation of several organ systems due to SARS-CoV-2 infection.
MQL	Master Question List	N/A
mRNA	Messenger RNA	Molecule that encodes protein sequences.
Mutation	Change in SARS-CoV-2 genome relative to wild-type or reference strain	Mutations are alterations (e.g., insertions, deletions, transpositions) in the RNA genome of SARS-CoV-2 that may or may not affect viral function. Mutations are often defined by the change in amino acid encoded by a sequence at a particular location. For instance, the N501Y mutation changes the 501st amino acid from an asparagine (N) to a tyrosine (Y). SARS-CoV-2 variants are usually comprised of multiple mutations, and mutations can arise in distinct SARS-CoV-2 variants.
NHP	Non-Human Primate	N/A
NIH	National Institutes of Health	N/A
NIOSH	National Institute for Occupational Safety and Health	N/A
Non-variant SARS-CoV-2	"wild-type" or comparator strain or variant in any comparison of novel or emerging variants	Used to indicate a comparison between a new or emerging variant (such as B.1.1.7) and an older strain or variant of SARS-CoV-2 (e.g., WA-1). There is no single "non-variant" SARS-CoV-2 virus. See also "wild-type."
NPI	Non-Pharmaceutical Intervention	Public health control measures designed to reduce transmission, such as social distancing, movement restrictions, and face mask requirements.
PAPR	Powered Air Purifying Respirator	Devices that are battery-powered and pass air through filters to provide clear air to the user.
PASC	Post-Acute Sequelae of SARS-CoV-2 Infection	Term used to encompass symptoms experienced for weeks to months after a primary infection with SARS-CoV-2.
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.
RAAS	Renin-Angiotensin-Aldosterone System	A hormonal system that influences blood pressure and volume.
RBD	Receptor Binding Domain	Protein domain used by virus to gain entry into host cells by recognizing specific host cell receptors (e.g., ACE2).

Acronym/Term	Definition	Description
RNA	Ribonucleic Acid	Genetic material for some viruses. Also, the intermediate between DNA and protein.
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, Exposed, Infected, and Resistant	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.
$TCID_{50}$	Median Tissue Culture Infectious Dose	The number of infectious units that 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.
UK	United Kingdom	N/A
Variant	SARS-CoV-2 lineage with shared collection of mutations	Variants are used to designate distinct SARS-CoV-2 lineages that share any number of mutations. For instance, the B.1.1.7 variant is characterized by an HV 69-70 deletion, N501Y mutation, and N493K mutation.
Vertical transmission	Transmission from mother to fetus	Generally understood as intrauterine transmission via blood or placenta; not the same as transmission during or after birth.
VHP	Vaporized Hydrogen Peroxide	An antimicrobial vapor used to decontaminate enclosed areas.
VOC	Variants of Concern	Variants of the virus that have evidence of impact on spread, severity, testing, treatment, and vaccination.
WHO	World Health Organization	N/A
Wild-type	Original SARS-CoV-2 strains	Generally considered to be early SARS-CoV-2 strains spreading from Wuhan, China to other locations, before the emergence of newer variants of interest or concern (e.g., Alpha, Delta). See also "non-variant."

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