SARS-CoV-2 (COVID-19) by the numbers

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Comments are welcome; this article is being updated on an ongoing basis at: https://bit.ly/2WOeN64

Size & Content
- Diameter: ~100 nm
- Volume: ~10^6 nm^3 = 10^-3 fl
- Mass: ~10^3 MDa = 1 fg

Genome
- Nucleotide identity to SARS-CoV-2
  - bat CoV: 96%
  - pangolin CoV: 91%
  - SARS-CoV-1: 80%
  - MERS: 55%
  - common cold CoV: 50%

  Spike trimer
  - Length: ~10 nm
  - Copies per virion: ~100
  - Affinity to ACE2
    - receptor K_d = ~100 nM
    - primed by TMPRSS2

  Nucleoprotein
  - 150 copies (measured for SARS-CoV-1)

  Envelope protein
  - 100 copies (measured for TGEV coronavirus)

  Polyprotein
  - 300 monomers

  Length: ~30kb; β-coronavirus with 10-14 ORFs (24-27 proteins)

Evolution rate: ~10^-3 mt^-1 yr^-1 (measured for SARS-CoV-1)
Mutation rate: ~10^-6 mt^-1 cycle^-1 (measured for MHV coronavirus)

Replication Timescales
- in tissue-culture
  - Virion entry into cell: ~10 min (measured for SARS-CoV-1)
  - Eclipse period: ~10 hrs (time to make intracellular virions)
  - Burst size: ~10^3 virions (measured for MHV coronavirus)

Host Cells
- (tentative list; number of cells per person)
  - Type I & II pneumocytes (~10^11 cells)
  - Alveolar macrophage (~10^10 cells)
  - Mucous cell in nasal cavity (~10^9 cells)
  - Host cell volume: ~10^3 μm^3 = 10^3 fl

Concentration
- maximal observed values following diagnosis
  - (Woelefe et al. 2020; Kim et al. 2020; Pan et al. 2020)
  - Nasopharynx: 10^6-10^8 RNAs/swab
  - Throat: 10^6-10^8 RNAs/swab
  - Stool: 10^6-10^10 RNAs/g
  - Sputum: 10^6-10^11 RNAs/mL

Anti-body Response - Seroconversion
- Antibodies appear in blood after: ~10-20 days
- Maintenance of antibody response: ~2-3 years (measured for SARS-CoV-1)

Virus Environmental Stability
- Relevance to personal safety unclear

  - Aerosols: ~1 hr (time to decay 1000-fold)
  - Surfaces: ~1-7 hr (time to decay 1000-fold)

  - (van Doremalen et al. 2020, 2020)

Antibodies appear in blood after: ~10-20 days
Maintenance of antibody response: ~2-3 years (measured for SARS-CoV-1)

“Characteristic” Infection Progression in a Single Patient

Basic reproductive number R_0: typically 2-4
- Varies further across space and time (Li et al. 2020, Park et al. 2020)
- (number of new cases directly generated from a single case)

Infection with virus
- Incubation period: ~5 days
- Diagnosis after ~5 days
- Symptomatic
- Exposed
- Infectious

- Latent period: ~3 days
- Interval of half-maximum infectiousness: ~4 days

Case Fatality Rate (CDC 2020)
- 0.8%-1.0% (uncorrected)
- Infected Fatality Rate: 0.3%-1.3%

- Recovery: mild cases: ~2 weeks
- Severe cases: ~6 weeks

Inter-individual variability is substantial and not well characterized. The estimates are parameter fits for population median in China and do not describe this variability (Li et al. 2020, He et al. 2020).

Note the difference in notation between the symbol R_0, which indicates “approximately” and connotes accuracy to within a factor 2, and the symbol –, which indicates “order of magnitude” or accuracy to within a factor of 10.
Abstract
The current SARS-CoV-2 pandemic is a harsh reminder of the fact that, whether in a single human host or a wave of infection across continents, viral dynamics is often a story about the numbers. In this snapshot, our aim is to provide a one-stop, curated graphical source for the key numbers that help us understand the virus driving our current global crisis. The discussion is framed around two broad themes: 1) the biology of the virus itself and 2) the characteristics of the infection of a single human host. Our one-page summary provides the key numbers pertaining to SARS-CoV-2, based mostly on peer-reviewed literature. The numbers reported in summary format are substantiated by the annotated references below. Readers are urged to remember that much uncertainty remains and knowledge of this pandemic and the virus driving it is rapidly evolving. In the paragraphs below we provide "back of the envelope" calculations that exemplify the insights that can be gained from knowing some key numbers and using quantitative logic. These calculations serve to improve our intuition through sanity checks, but do not replace detailed epidemiological analysis.

1. How long does it take a single infected person to yield one million infected people?
If everybody continued to behave as usual, how long would it take the pandemic to spread from one person to a million infected victims? The basic reproduction number, $R_0$, suggests each infection directly generates 2-3 more infections in the absence of countermeasures like social distancing. Once a person is infected, it takes a period of time known as the latent period before they are able to transmit the virus. The current best-estimate of the median latent time is $\approx 3$ days followed by $\approx 4$ days of close to maximal infectiousness ($Li$ et al. 2020, $He$ et al. 2020). The exact durations vary among people, and some are infectious for much longer. Using $R_0=3$, the number of cases will quadruple every $\approx 7$ days or double every $\approx 3$ days. 1000-fold growth (going from one case to 10$^3$) requires 10 doublings since $2^{10}=10^3$; 3 days $\times$ 10 doublings = 30 days, or about one month. So we expect $\approx 1000x$ growth in one month, million-fold (going from one case to 10$^6$) in three months. Even though this calculation is highly simplified, ignoring the effects of "super-spreaders", herd-immunity and incomplete testing, it emphasizes the fact that viruses can spread at a bewildering pace when no countermeasures are taken. This illustrates why it is crucial to limit the spread of the virus by social distancing measures. For fuller discussion of the meaning of $R_0$, the latent and infectious periods, as well as various caveats, see the "Definitions" section.

2. What is the effect of social distancing?
A highly simplified quantitative example helps clarify the need for social distancing. Suppose that you are infected and you encounter 50 people over the course of a day of working, commuting, socializing and running errands. To make the numbers round, let's further suppose that you have a 2% chance of transmitting the virus to each of these encounters, so that you are likely to infect 1 new person each day. If you are infectious for 4 days, then you will infect 4 others on average, which is on the high end of the $R_0$ value for SARS-CoV-2, and a billion fold ($10^9$) in three months. Even though this calculation is highly simplified, ignoring the effects of "super-spreaders", herd-immunity and incomplete testing, it emphasizes the fact that viruses can spread at a bewildering pace when no countermeasures are taken. This illustrates why it is crucial to limit the spread of the virus by social distancing measures. For fuller discussion of the meaning of $R_0$, the latent and infectious periods, as well as various caveats, see the "Definitions" section.

3. Why is the quarantine period two weeks?
The period of time from infection to symptoms is termed the incubation period. The median SARS-CoV-2 incubation period is estimated to be roughly 5 days (Lauer et al. 2020). Yet there is much person-to-person variation. Approximately 99% of those showing symptoms will show them before day 14, which explains the two week confinement period. Importantly, this analysis neglects infected people who never show symptoms. Since asymptomatic people are not usually tested, it is still not clear how many such cases there are or how long asymptomatic people remain infectious for.

4. How do N95 masks block SARS-CoV-2?
N95 masks are designed to remove more than 95% of all particles that are at least 0.3 microns ($\mu m$) in diameter ($NIOSH 42 CFR Part 84$). In fact, measurements of the particle filtration efficiency of N95 masks show that they are capable of filtering $\approx 99.8%$ of particles with a diameter of $<0.1 \mu m$ ($Regnasyamy$ et al. 2017). SARS-CoV-2 is an enveloped virus $\approx 0.1 \mu m$ in diameter, so N95 masks are capable of filtering most free virions, but they do more than that. How? Viruses are often transmitted through respiratory droplets produced by coughing and sneezing. Respiratory droplets are usually divided into two size bins, large droplets ($> 5 \mu m$ in diameter) that fall rapidly to the ground and are thus transmitted only over short distances, and small droplets ($\approx 5 \mu m$ in diameter). Small droplets can evaporate into "droplet nuclei," remain suspended in air for significant periods of time and could be inhaled. Some viruses, such as measles, can be transmitted by droplet nuclei ($Teller$ et al. 2019). At present there is no direct evidence showing SARS-CoV-2 transmission by droplet nuclei. Rather, larger droplets are believed to be the main vector of SARS-CoV-2 transmission, usually by settling onto surfaces that are touched and transported by hands onto mucosal membranes such as the eyes, nose and mouth ($CDC 2020$). The characteristic diameter of large droplets produced by sneezing is $\approx 100 \mu m$ ($Plan J. R. Soc. Interface 2013$), while the diameter of droplet nuclei produced by coughing is on the order of $\approx 1 \mu m$ ($Yang$ et al. 2007). Therefore, N95 masks likely protect against several modes of viral transmission.

5. How similar is SARS-CoV-2 to the common cold and flu viruses?
SARS-CoV-2 is a beta-coronavirus whose genome is a single $\approx 30 \mu m$ strand of RNA. The flu is caused by an entirely different family of RNA viruses called influenza viruses. Flu viruses have smaller genomes ($\approx 14 \mu m$) encoded in 8 distinct strands of RNA, and they infect human cells in a different manner than coronaviruses. The "common cold" is caused by a variety of viruses, including some coronaviruses and rhinoviruses. Cold-causing coronaviruses (e.g. OC43 and 229E strains) are quite similar to SARS-CoV-2 in genome length (within 10%) and gene content, but different from SARS-CoV-2 in sequence ($\approx 50%$ nucleotide identity) and infection severity. One interesting facet of coronaviruses is that they have the largest genomes of any known RNA viruses ($\approx 30 \mu m$). These large genomes led researchers to suspect the presence of a "proofreading mechanism" to reduce the mutation rate and stabilize the genome. Indeed, coronaviruses have a proofreading exonuclease called ExoN, which explains their very low mutation rates ($\approx 10^{-6}$ per site per cycle) in comparison to influenza (nearly 10$^{-3}$ per site per cycle ($Sanjuan et al. 2010$)). This relatively low mutation rate will be of interest for future studies predicting the speed with which coronaviruses can evade our immunization efforts.

6. How much is known about the SARS-CoV-2 genome and proteome?
SARS-CoV-2 has a single-stranded positive-sense RNA genome that codes for 10 genes ultimately producing 26 proteins according to an NCBI annotation ($NC 045512$). How is it that 10 genes code for $\approx 20$ proteins? One long gene, orf1ab, encodes a polyprotein that is cleaved into 16 proteins by proteases that are themselves part of the polyprotein. In addition to proteases, the polyprotein encodes a number of other proteins that are not viral factors but are essential for viral function such as a proofreading exonuclease, and several other non-structural proteins. The remaining genes predominantly code for structural components of the virus: (i) the spike protein which binds the cognate receptor on a human or animal cell; (ii) a nucleoprotein that packages the genome; and (iii) two membrane-bound proteins. Though much current work is centered on understanding the role of "accessory" proteins in the viral life cycle, we estimate that it is currently possible to ascribe clear biochemical or structural functions to only about half of SARS-CoV-2 gene products.

7. What can we learn from the mutation rate of the virus?
Studying viral evolution, researchers commonly use two measures describing the rate of genomic change. The first is the evolutionary rate, which is defined as the average number of substitutions that become fixed per year in strains of the virus, given in units of mutations per site per year. The second is the mutation rate, which is the number of substitutions per site per replication cycle. How can we relate these two values? Consider a single site at the end of a year. The only measurement of a mutation rate in a beta-coronavirus suggests that this site will accumulate $\approx 10^{-5}$ mutations in each round of replication. Each round of replication cycle takes $\approx 10$ hours, and so there are $10^6$ cycles/year. Multiplying the mutation rate by the number of replications, and neglecting the potential effects of evolutionary selection and drift, we arrive at $10^{-3}$ mutations per site per year, consistent with the evolutionary rate inferred from sequenced coronavirus genomes. As our estimate is consistent with the evolutionary rate, we conclude that the virus undergoes near-continuous replication in the wild, constantly generating new mutations that accumulate over the course of the year. Using our knowledge of the mutation rate, we can also draw inferences about single infections. For example, since the mutation rate is $\approx 10^{-6}$ mutations/site/cycle and an mL of sputum might contain upwards of 10$^7$ viral RNAAs, we infer that every site is mutated more than once in such samples.

8. How stable and infectious is the virus on surfaces?
SARS-CoV-2 RNA has been detected on various surfaces several weeks after they were last touched ($Moriarty$ et al. 2020). In the definitions we clarify the difference between detecting viral RNA and active virus. The probability of human infection from such exposure is not yet characterized as experiments to make this determination are very challenging. Nevertheless, caution and protective measures must be taken. We estimate that during the infectious period an undiagnosed infectious person touches surfaces tens of times. These surfaces will subsequently be touched by hundreds of other people. From the basic reproduction number $R_0$ $\approx 2.4$ we can infer that not everyone touching those surfaces will be infected. More detailed bounds on the risk of infection from touching surfaces urgently awaits study.
Glossary

Clinical Measures
Incubation period: time between exposure and symptoms.
Seroconversion: time between exposure to virus and detectable antibody response.

Epidemiological Inferences
$R_0$, the average number of cases directly generated by an individual infection.
Latent period: time between exposure and becoming infective.
Infectious period: time for which an individual is infective.
Interval of half-maximum infectiousness: the time interval during which the probability of viral transmission is higher than half of the peak infectiousness. This interval is similar to the infectious period, but applies also in cases where the probability of infection is not uniform in time.

Viral Species
SARS-CoV-1: a β-coronavirus that caused the 2002 SARS outbreak in China.
MERS: a β-coronavirus that caused the Middle East Respiratory Syndrome outbreak beginning in Jordan in 2012.
MHV: Murine herpes virus, a model β-coronavirus on which much laboratory research has been conducted.
TGEV: Transmissible gastroenteritis virus, a model γ-coronavirus which infects pigs.
229E and OC43: two strains of coronavirus (β- and β- respectively) that are cause a fraction of common colds.

Viral Life-Cycle

Eclipse period: time between viral entry and appearance of intracellular virions.
Latent period (cellular level): time between viral entry and appearance of extracellular virions. Not to be confused with the epidemiological latent period described below.

Burst size: the number of virions produced from infection of a single cell. More appropriately called “per-cell viral yield” for non-lytic viruses like SARS-CoV-2.
Virion: a viral particle.
Polyprotein: a long protein that is proteolytically cleaved into a number of distinct proteins. Distinct from a polypeptide, which is a linear chain of amino acids making up a protein.

Human Biology
Alveolar Macrophage: immune cells found in the lung that engulf foreign material like dust and microbes (“professional phagocytes”)
Pneumocytes: the non-immune cells in the lung.
ACE2: Angiotensin-converting enzyme 2, the mammalian cell surface receptor that SARS-CoV-2 binds.
TMPRSS2: Transmembrane protease, serine 2, a mammalian membrane-bound serine protease that cleaves the viral spike trimer after it binds ACE2, revealing a fusion peptide that participates in membrane fusion which enables subsequent injection of viral RNA into the host cytoplasm.
Nasopharynx: the space above the soft palate at the back of the nose which connects the nose to the mouth.

Notation
Note the difference in notation between the symbol ∼, which indicates “approximately” and connotes accuracy to within a factor 2, and the symbol ~, which indicates “order of magnitude” or accuracy to within a factor 10.

More on definitions and measurement methods

What are the meanings of $R_0$, “latent period” and “infectious period”?
The basic reproduction number, $R_0$, estimates the average number of new infections directly generated by a single infectious person. The 0 subscript connotes that this refers to early stages of an epidemic, when everyone in the region is susceptible (i.e. there is no immunity) and no counter-measures have been taken. As geography and culture affect how many people we encounter daily, how much we touch them and share food with them, estimates of $R_0$ can vary between locales. Moreover, because $R_0$ is defined in the absence of countermeasures and immunity, we are usually only able to assess the effective $R$ ($R_e$). At the beginning of an epidemic, before any countermeasures, $R_e = R_0$. Several days pass before a newly-infected person becomes infectious themselves. This “latent period” is typically followed by several days of infectivity called the “infectious period.” It is important to understand that reported values for all these parameters are population averages inferred from epidemiological models fit to counts of infected, symptomatic, and dying patients. Because testing is always incomplete and model fitting is imperfect, and data will vary between different locations, there is substantial uncertainty associated with reported values. Moreover, these median or average best-fit values do not describe person-to-person variation. For example, viral RNA was detectable in patients with mild symptoms for > 1 week after the onset of symptoms, and more than 2 weeks in patients with severe symptoms (ECDC 2020). Though detectable RNA is not the same as active virus, this evidence calls for caution in using uncertain, average parameters to describe a pandemic. Why aren’t detailed distributions of these parameters across people published? Direct measurement of latent and infectious periods at the individual level is extremely challenging, as accurately identifying the precise time of infection is usually very difficult.

What is the difference between measurements of viral RNA and infectious viruses?
Diagnosis and quantification of viruses utilizes several different methodologies. One common approach is to quantify the amount of viral RNA in an environmental (e.g. surface) or clinical (e.g. sputum) sample via quantitative reverse-transcription polymerase chain reaction (RT-qPCR). This method measures the concentration of copies of viral RNA in a sample. The presence of viral RNA does not necessarily imply the presence of infectious virions. Virions could be defective (e.g. by mutation) or might have been deactivated by environmental conditions. To assess the concentration of infectious virions, researchers typically measure the “50% tissue-culture infectious dose” (TCID50). Measuring TCID50 involves infecting replicate cultures of susceptible cells with dilutions of the virus and noting the dilution at which half the replicate dishes become infected. Viral counts reported by TCID50 tend to be lower than RT-qPCR measurements, which could be one reason why studies relying on RNA measurements (Moriarty et al. 2020) report the persistence of viral RNA on surfaces for much longer times than studies relying on TCID50 (van Doremalen et al. 2020).
It is important to keep this caveat in mind when interpreting data about viral loads, for example a report measuring viral RNA in patient stool samples for several days after recovery (Wu et al. 2020). Nevertheless, for many viruses even a small dose of virions can lead to infection. For the common cold, for example, ~10 TCID50 are sufficient to infect half of the people exposed (Cook et al. 1966).

What is the difference between the case fatality rate and the infection fatality rate?
Global statistics on new infections and fatalities are pouring in from many countries, providing somewhat different views on the severity and progression of the pandemic. Assessing the severity of the pandemic is critical for policy making and thus much effort has been put into quantification. The most common measure for the severity of a disease is the fatality rate. One common reporting measure is the case fatality rate (CFR), which is the proportion of fatalities out of total diagnosed cases. The CFR reported in different countries varies significantly, from 0.3% to about 10%. Several key factors affect the CFR. First, demographic parameters and practices associated with increased or decreased risk differ greatly across societies. For example, the prevalence of smoking, the average age of the population, and the capacity of the healthcare system. Indeed, the majority of people dying from SARS-CoV-2 have a pre-existing condition such as cardiovascular disease or smoking (China CDC 2020). There is also potential for bias in estimating the CFR. For example, a tendency to identify more severe cases (selection bias) will tend to overestimate the CFR. On the other hand, there is usually a delay between the onset of symptoms and death, which can lead to an underestimate of the CFR early in the progression of an epidemic. Even when correcting for these factors, the CFR does not give a complete picture of symptoms as many cases with mild or no symptoms are not tested. Thus, the CFR will tend to overestimate the rate of fatalities per infected person, termed the infection fatality rate (IFR). Estimating the total number of infected people is usually accomplished by testing a random sample for anti-viral antibodies, whose presence indicates that the patient was previously infected. As of writing, such assays are not widely available, and so researchers resort to surrogate datasets generated by testing of foreign citizens returning home from infected countries (Verity et al. 2020) or epidemiological models estimating the number of undocumented cases (Li et al. 2020). These methods provide a first glimpse of the true severity of the disease.

What is the burst size and the replication time of the virus?
Two important characteristics of the viral life cycle are the time it takes them to produce new infectious progeny, and the number of progeny each infected cell produces. The yield of new virions per infected cell is more clearly defined in lytic viruses, such as those infecting bacteria (bacteriophages), as viruses replicate within the cell and subsequently lyse the cell to release a “burst” of progeny. This measure is usually termed “burst size.” SARS-CoV-2 does not release its progeny by lysing the cell, but rather by continuous budding (Park et al. 2020). Even though there is no “burst”, we can still estimate the average number of virions produced by a single infected cell. Measuring the time to complete a replication cycle or the burst size in vivo is very challenging, and thus researchers usually resort to measuring these values in tissue-culture. There are various ways to estimate these quantities, but a common and simple one is using “one-step” growth dynamics. The key principle of this method is to ensure that only a single replication cycle occurs. This is typically achieved by infecting the cells with a large number of virions, such that every cell gets infected, thus leaving no opportunity for secondary infections. Assuming entry of the virus to the cells is rapid (we estimate 10 minutes for SARS-CoV-2), the time it takes to produce progeny can be estimated by quantifying the lag between inoculation and the appearance of new intracellular virions, also known as the “eclipse period”. This eclipse period does not account for the time it takes to release new virions from the cell. The time from cell entry until the appearance of the first extracellular virions, known as the “latent period” (not to be confused with the epidemiological latent period), is important to the duration of the full replication cycle. The burst size can be estimated by waiting until virus production saturates, and then dividing the total virion yield by the number of cells infected. While both the time to complete a replication cycle and the burst size may vary significantly in an animal host due to factors including the type of cell infected or the action of the immune system, these numbers provide us with an approximate quantitative view of the viral life-cycle at the cellular level.
Note that for about 10 out of 45 parameters, the literature values are from other coronaviruses. We await corresponding measurements for SARS-CoV-2.

**Size & Content**

- **Diameter**: (Zhu et al. 2020) - Electron micrographs of negative-stained 2019-nCoV particles were generally spherical with some pleomorphism and varied from about 60 to 140 nm.
- **Volume**: Using diameter and assuming the virus is a sphere: Mass using the volume and a density of ~1 g per mL.
- **Number of surface spikes trimers**: (Neuman et al. 2011) - Our model predicts ~90 spikes per particle.
- **Length of surface spikes trimers**: (Zhu et al. 2020) - Virus particles had quite distinctive spikes, about 9 to 12 nm, and gave virions the appearance of a solar corona.

**Genome**

- **Type**: (Vara-De la Vega et al. 2020) - SARS-CoV-2 virus genome has 10 open reading frames (Fig. 2A) and (Wu et al. 2020) - The 2019-nCoV virus was shown to possess 14 ORFs encoding 27 proteins.
- **Number of codes**: (Wu et al. 2020) - SARS-CoV-2 virus genome has 10 open reading frames (Fig. 2A).
- **Genome length**: (Wu et al. 2020) - The 2019-nCoV virus was shown to possess 14 ORFs encoding 27 proteins.
- **Number of codons**: (Wu et al. 2020) - By aligning with the amino acid sequence of SARS PPLab and analyzing the restriction cleavage sites recognized by 3CLpro and PLpro, we speculated 14 predicted proteolytic sites of 3CLpro and PLpro in SARS-CoV-2 PPLab (Fig. 3B).

**Replication Timescales**

- **Entry into cell**: (Schneider et al. 2012) - Previous experiments had revealed that virus is internalized within 15 min and (Neale et al. 2020) - Within the first 10 min, some virus particles were internalized into vacuoles (arrow) that were just below the plasma membrane surface (Fig. 2, arrows). The observation at 15 min postinfection (p.i), did not differ much from 10 min p.i. (Fig. 4a).
- **Eclipse period**: (Schneider et al. 2012) - SARS-CoV-2 replication cycle from adsorption to release of infectious progeny takes about 7 to 8 h (data not shown).
- **Burst size**: (Iranzo et al. 1976) - “The average per-cell yield of active virus was estimated to be about 6-7×10^4 plaque-forming units.”

**Concentration**

- **Nasopharyngeal, Throat, Stool and Sputum**: (Woo et al. 2020) - Figure 2 and (Kim et al. 2020) - Figure 1 and (Han et al. 2020) - We took the maximal viral load for each patient in nasopharyngeal swabs, throat swabs, stool or in sputum.

**Antibody Response - Seroconversion**

- **Seroconversion time** (time period until a specific antibody becomes detectable in the blood): (Park et al. 2020) - “The median incubation period for being able to transmit; (Li et al. 2020) - In addition, the median estimates for the latent and infectious periods are approximately 3.69 and 3.48 days, respectively; and Table 1 and (Jie et al. 2020) - We use the time it takes the infectiousness to reach half its peak, which happens two days before symptom onset based on Figure 1B. As symptoms arise after 5 days (see incubation period), this means the latent period is about 3 days. (Park et al. 2020) - “The incubation period was estimated to be 3.5 days (95% CI, 4.5 to 5.8 days), and 97.5% of those who develop symptoms will do so within 11.5 days (CI, 8.2 to 15.6 days) of infection. These estimates imply that under conservative assumptions, 101 out of every 10,000 cases (99th percentile, 482) will develop symptoms after 14 days of active monitoring or quarantine” and (Li et al. 2020) - “The mean incubation period was 5.2 days (95% confidence interval [CI], 4.1 to 7.0), with the 95th percentile of the distribution at 12.5 days.”

**Virus Environmental Stability**

- **Half life on surfaces**: (Van Doremalen et al. 2020) - For half-lives we use Supplementary Table 1. For time to decay from ~10^9 to ~10^0 TCID50 / mL or air / m^3 medium, we use the first time the latter reached detection limit in Figure 1A for surfaces. For aerosols, we use ten half-life values (1000-fold decrease from 10^9 to 10^0, meaning 10 halvings of concentration) from Supplementary Table 1. More studies are urgently needed to clarify the implications of virion stability on the probability of infection from aerosols or surfaces.

**References and excerpts**

- “The model estimates for the latent and infectious periods are approximately 3.69 and 3.48 days, respectively; and Table 1 and (Jie et al. 2020) - We use the time it takes the infectiousness to reach half its peak, which happens two days before symptom onset based on Figure 1B. As symptoms arise after 5 days (see incubation period), this means the latent period is about 3 days. (Park et al. 2020) - “The incubation period was estimated to be 3.5 days (95% CI, 4.5 to 5.8 days), and 97.5% of those who develop symptoms will do so within 11.5 days (CI, 8.2 to 15.6 days) of infection. These estimates imply that under conservative assumptions, 101 out of every 10,000 cases (99th percentile, 482) will develop symptoms after 14 days of active monitoring or quarantine” and (Li et al. 2020) - “The mean incubation period was 5.2 days (95% confidence interval [CI], 4.1 to 7.0), with the 95th percentile of the distribution at 12.5 days.”
- **“Characteristic” Infection Progression in a Single Patient**

Future developments in this area will be driven by SARS-CoV-2 RNA was identified on a variety of surfaces in both symptomatic and asymptomatic infected passengers and on surfaces coated on the Diamond Princess but before disinfection procedures had been conducted.

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