DRAFT v1.0

Assessment and Mitigation of Aerosol Airborne SARS-CoV-2 Transmission

Photonics, Physical Chemistry, Atomic, and Condensed Matter Experimental Group Laboratory and Office Environments
Doyle Group
Harvard University

Executive Summary

Knowledge of the transmission pathways for SARS-CoV-2, the virus that causes COVID-19, is incomplete. Current evidence indicates that transmission can occur via both surface contact (fomites) and aerosols/droplets (Singh et al., 2020; Wang et al., 2020). In this report, we consider SARS-CoV-2 transmission via aerosolized particles in Photonics, Physical Chemistry, Atomic, and Condensed Matter Experimental Group labs ("labs") and the offices often connected with those labs ("offices"). However, the presented analysis is often general and can be applied to a variety of working situations.

We review the literature and find that aerosols may be a significant transmission route for SARS-CoV-2. These studies make clear several important factors: both symptomatic and presymptomatic carriers can shed the virus; aerosols can contain active virus for many hours in suspension; and viral spread is dependent on local air flow conditions. We also review the literature regarding the effectiveness of masks at preventing inhalation of virus-laden aerosols. Combining the knowledge on aerosol transmission and mask effectiveness, the typical properties of labs and offices, and a dose-response model, we develop guidelines for laboratory work procedures. The recommended guidelines in this report will also attenuate the large droplet transmission route, although that is not discussed in detail here. Because the air handling and ventilation of labs and offices can differ substantially, we discuss examples for these two work environments.

The analysis presented here is based on several assumptions that are chosen to be conservative. We assume: relatively high SARS-CoV-2 infectivity, setting the relevant infectivity parameters to those of influenza; we consider the infection of a single uninfected ("healthy") person H in an environment of asymptomatic ("sick") carriers S that actively shed virus for 1 full week (except a single healthy person); a person works 8 hours a day, 5 days a week; a healthy person significantly connects (e.g. shares in some way an office or lab) with 3 other people who are shedding virus at different times. (Four people typically work in an experimental subgroup.) We do NOT consider the situation where mixing of air between rooms takes place through the HVAC system. That may be a subject for a future iteration of this report.

We aim to assign an acceptable risk threshold in order to set guidelines. We estimate the level of acceptable risk by comparison with 1) other daily risks excluding contracting COVID-19, 2) work related risks only, excluding contracting COVID-19, and 3) the risk of contracting

COVID-19 outside of work. Our guidelines are based on a threshold of $p=1\%$ chance of contracting COVID-19 at work over the course of six months, assuming that all of the researchers with whom a healthy person connects, will shed virus for one week (each) in a non-overlapping time frame. From a public health perspective, this a risk level of $p = 1\%$ corresponding to a negligible incremental increase in the basic reproduction number $R_0 < 0.001$ (see App. C.4). Adjusting the risk threshold for p to values within a factor of 10 around this value would leave the structure of the guidelines unchanged.

Guidelines

1. In rooms with no HEPA filtration, only one person per room is allowed and a wait time between occupancy by different people is required. This wait time is determined by the fresh air exchange rates for the room ("fresh air changes"). A typical scenario, discussed below, is an office space with ~2 fresh air changes per hour, leading to a ~ 2.5 hour wait time.
2. In rooms with HEPA filtration, for people spaced by sufficient distance (>5 m), and in a way that air flow lines from air output to intake do not mix air from one worker to another within the room, multiple occupancy is allowed. In the case where a new person enters a HEPA filtered room, a waiting period of about 1 air change, 5-10 minutes after the previous occupant exits, is required.

A note on conservative assumptions: We base these guidelines on the idea that even if all researchers contract COVID-19 over the six-month period, any given infected person will shed virus at peak levels for only about one work week. Furthermore, a healthy person, H, will generally connect with at most 3 other people (for a typical subgroup size of 4 people, and assuming negligible mixing between subgroups; for a discussion of auxiliary prep spaces shared between subgroups, including bathrooms, see App. A.5). In the worst case allowed by these assumptions, H would be exposed to 3 weeks of infection risk in the workplace. An *even more* conservative analysis would assume that *all* persons (other than the one healthy person H) are constantly shedding virus at peak levels all the time. This would mean 26 weeks of exposure for a healthy person. Under the "26-week exposure" condition, the wait time indicated in (1) above would be increased to 5 hours if $p<1\%$ is maintained. For (2) above, under the "26-week exposure" condition, the rapid dilution of virus in the air by the HEPA HVAC system is sufficient to ensure $p<1\%$ if H enters at a time 1-2 air change times after S leaves the room (10-15 minutes). In the main text of the document below, we include numbers for both 3 weeks of exposure and 26 weeks of exposure (both over a 6-month period). Another conservative assumption that underlies these guidelines is a risk threshold of $p=1\%$, which if changed to $p=10\%$ and combined with the aforementioned more realistic viral shedding estimate, would indicate a wait time of <1 hour between occupancies of an office space.

Assessment and Mitigation of Aerosol Airborne SARS-CoV-2 Transmission

1. Aerosol Transmission and Physical Spaces

The literature indicates that aerosol transmission of SARS-CoV-2 is likely a significant contributor to the probability of infection. Furthermore, the lifetime of aerosols containing viable virus is long. The filtering and exchange of contaminated air with fresh air will lower the density of viral particles and thus can lower the probability of infection.

1.1 Aerosols provide a transmission route for SARS-CoV-2

Transmission of SARS-Cov-2 can occur even prior to onset of COVID-19 symptoms (Gandhi et al. 2020). In considering airborne spread of SARS-CoV-2, one can allow for two distinct modes of transmission: “droplet sprays” following a sneeze or cough and “microscopic aerosol particles” from evaporated respiratory droplets (Asadi et al., 2020). A general, though not universal, convention is to call particles >5 um droplets and particles < 5 um aerosols. When an infected person coughs, breathes vigorously, or speaks loudly they may shed virus in the form of bio-aerosols ranging from 0.3 to 100 um in diameter (Wang et al., 2020). Ordinary speech can also be a significant source of aerosolized particles (Asadi et al., 2020). Larger droplets are also suspected of providing a transmission route for SARS-CoV-2. However, large droplets > 5 um typically have shorter suspension times in air, < ~10 minutes (Singh et al., 2020). Aerosols in the size range 1-5 um are of particular concern, both because they are respirable and remain in the air for long times, hours or more (Wang et al., 2020; Singh et al., 2020; Meselson, 2020). **Studies have shown that SARS-CoV-2 can remain viable on aerosols for >3 hours, requiring ~13 hours for a 4-log reduction (van Doremalen et al., 2020; FDA, 2020; see Apps. C.2 and C.3).** Thus, the virus remains both active and suspended long enough to be carried by either diffusive or convective flow to other nearby (potentially uninfected) individuals.

While the question of whether aerosolized particles can lead to transmission of SARS-CoV-2 is under active investigation, there is significant circumstantial evidence that this is possible (Brosseau, 2020; Woelfel et al., 2020). A study of patients infected with COVID-19 in Germany found that active shedding of virus from the upper respiratory tract occurred as symptoms developed, a potential source of respirated aerosols (Woelfel et al., 2020). Several case studies have suggested efficient SARS-CoV-2 transmission due to aerosols. A study in Wuhan, China, found significant aerosol spread of SARS-CoV-2 in a restaurant with ~0.8 air changes per hour (Lu et al., 2020; Li et al., 2020). At the same time, the aerosol spread was found to be highly localized to the particular zone covered by a single air-handling unit (AHU), without identified spread to portions of the room under the control of other AHUs or to waiters moving in and out of the region of contaminated air (Li et al., 2020). This observation has important implications for shared work in the same laboratory room, e.g., if sufficient air flow and separation between areas can be maintained when two workers occupy the same room (see Sec. 2). In a hospital setting, the distribution of virus-laden aerosols was found to be somewhat (albeit weakly) determined by airflow patterns in the AHUs (Guo et al., 2020). By contrast, in a different study, very little SARS-CoV-2 RNA was found in negative-pressure, high-air-exchange-rate wings of a hospital (Liu et al., 2020). These studies highlight the importance of convective air currents

influencing the distribution of contaminated aerosols. As will be discussed below, the dominating air patterns differ between laboratory and office settings and must be taken into account in developing mitigation protocols.

1.2 Physical Spaces: Laboratories and Offices

A typical lab room is 500 square feet (~50 m^2) with volume V_{lab} = 200,000 liters. Labs are typically outfitted with air-handling units (AHUs) providing flow rates of ~300-2000 cubic feet per minute (cfm), depending on the room and generally scaling with the room volume. In order to meet temperature accuracy requirements, the air flow rates from lab AHUs result in entire room fresh (outside) air changes every 5-10 minutes and total air changes every 3-5 minutes. In contrast, a typical office room is 200 square feet (~20 m^2) and V_{lab} = 80,000 liters with a fresh air flow rate from the AHU of ~20-50 cfm, with an entire room fresh air change every ~30-60 minutes. AHUs can be equipped with high-efficiency particulate air (HEPA) filters. HEPA filters nominally capture particles of diameter greater than 0.3 μm with >99.97% efficiency (D.B. Day et al., 2018; P. Chuaybamroong, et al). Filtration efficiency depends on particle size. It is not clear whether aerosol transmission of SARS-CoV-2 is caused by suspended viral particle, of size ~50-150 nm (Kim et al., 2020; Cascella et al., 2020), or larger respirable aerosols of size ~1-5 μm (Wang et al., 2020; Singh et al., 2020). However, HEPA filtration efficiency (*f*) is measured to be 99.994% at 50 nm, 99.98% at 0.1-0.2 μm, and >99.996% for particles >0.5 μm (Zhang et al., 2014; Morono et al., 2018). To make conservative estimates below, we assume a nominal penetration value $q = (1-f) \sim 3\times10^{-4}$.

2. General Approach and Model Particulars

Our analytical approach is to 1) use the literature and make conservative assumptions about the infectivity of the SARS-CoV-2 virus, 2) calculate the probability of a healthy person (H) being infected via aerosols by a virus (peak) shedding person (S) under various physical conditions (e.g. one person per room, lab shared by two people, etc.), 3) set a threshold for acceptable probability, *p*, of being infected over the course of 6 months, and 4) devise mitigation approaches to attain that *p*. We choose $p < 1\%$ over six months of work as a target, assuming that one healthy person, H, interacts significantly with up to three virus-shedding persons, S, (for a typical experimental subgroup size of four people). This gives a total exposure to the virus that lasts up to 3 weeks in a six-month period of work. We also describe the stricter protocols that would be required under the less realistic, but more conservative, assumption that H is exposed continuously to the virus for the entire six-month period (26 weeks).

2.1 Model and Examples

We use a dose-response model, described in App. A.1, to assess the risk involved with various modes of laboratory work. This model states that for a viral dose, d, the infection probability is

$$p(d) = 1 - exp(-d/k),$$

where k is taken to be an "infection constant," measured in number of viral particles. The dose d is the number of viral particles inhaled by a person. We take, conservatively, k = 100; see App. A.1. This is equivalent to assuming that each inhaled viral copy incurs a 1% probability of leading to an infection. With this conservative assumption our target of $< 1\%$ probability of infection indicates that on average each researcher inhales less than one viral copy in six months of work. The time scale, *T*, for accumulation of the dose, is needed to calculate quantitative probabilities. One can argue that *T* should correspond to exposure for 5 working days; this is the "natural" time scale for the disease, matching the COVID-19 incubation time, the typical immune response time, and the peak viral shedding time. However, in the conservative model we adopt here, this time drops out of the mathematical analysis as it is assumed that there is one healthy person and all others are actively shedding virus, see below.

Assume there are two people, person S, who is infected, and person H, who is healthy. By breathing, S emits a certain number of SARS-CoV-2 viral particles into the air. As an example, based on the analysis in App. C.1, S exhales ~70 viral particles/minute. This will create (after mixing with a volume of air, filtering, etc.) some local density of viral particle n [viral particle/liter of air] that H inhales. The viral dose that H receives is $d = n \times V_{inh}$, where V_{inh} is the total volume of air inhaled by H. At a typical breathing rate, H will inhale a volume of air per unit time V' ~ 450 l/h. For example, $V_{inh} = 1.8 \times 10^4$ liter during T = 40 hours of work and under these conditions $d_{3week} = 54{,}000n$ and $d_{26week} = 470{,}000n$. The infection probability can be expressed

$$p(n) = 1 - \exp(-(V'T) \times n/k) = 1 - \exp(-Bn)$$

For small probability, this simplifies to

$$p \approx Bn$$

where $B_{3week} = 540$ and $B_{26week} = 4700$.

For easy reference, we supply probability formulas in App. D.1.

Determining n at the location of H depends heavily on the spatial situation (locations and movement of S and H) and the air conditions (mixing and replacement with outside fresh air and filtration). We will assume two baseline situations: "Solo" (S in the room alone, S leaves, H occupies) and "Shared" (S and H are in the same room at the same time).

<u>Solo</u>

Consider first a room with parameters matching those of a typical office. The concentration of viral particles in the air accumulates over a time scale set by the air exchange rate in the room. Assuming rapid mixing of the aerosol (either by convective mixing or diffusion) in typical room with volume $V_{room} = V_{office}$ = 80,000 liters, the maximum density of SARS-CoV-2 is $n \sim 3 \times 10^{-2}$ viral particle/liter accumulating over an air exchange time of 30 mins, giving a 99% likelihood of infection for one week of exposure. Even with use of N95 masks in this scenario, $p \sim$ 40% in one week. This indicates that sharing offices is not possible while maintaining p < 1%, even for a

one-week time scale. However, if S and H do not share the room simultaneously, then the dose to H is reduced by HVAC air exchange. Typically, a room will be traded between H and S one or two times per day. We assume conservatively that H enters a room previously vacated by S no more than four times per day. The time-dependent dilution of the dose is given in App. A.4 and may be used to find the total dose. If H waits 6 (9) air change times before entering, then for 3 (26) weeks of total exposure in a six-month period of work, the probability of infection is $p < 1\%$, requiring, in this example, a 3 (4.5) hour wait time while the room is aired out. Use of masks, either surgical or N95, will shorten the required waiting time.

We separately consider the risk associated with occupying a lab that has been previously occupied by S. In this case, typically $\mathrm{V}_{\mathrm{room}} = \mathrm{V}_{\mathrm{Lab}}$ = 200,000 liters and the air exchange time is approximately 5 minutes. Because labs are HEPA filtered and have high rates of air circulation, the contamination present after S leaves rapidly diminishes (see App. A.3 and App. A.4). With 3 weeks of total exposure in a six-month period of work, the probability of infection is $p < 1\%$ even if H enters immediately after S leaves the room. However, we recommend that H wait for one air exchange time before entering since the local density of virus in some positions (e.g., far from the HVAC inlet) may significantly exceed the average density in the room on time scales shorter than one air exchange time. Waiting one air exchange time is also sufficient to ensure $p < 1\%$ even with 26 weeks of total exposure.

Shared

Now consider the case where S and H are in the same room, but 1) the air is HEPA filtered on a shorter time scale than the air change time discussed in the *Solo* case and 2) S and H are placed far enough apart that they sit in different airstreams formed by the flow of air from the HVAC output to intake openings (see App. A.3 and App. A.4). In this case, H is always breathing air that has been HEPA filtered. For a room with $\mathrm{V}_{\mathrm{room}} = \mathrm{V}_{\mathrm{Lab}}$ = 200,000 liters, and with an air change time for the room of 5 minutes, the air stream input into the HVAC system from S has an approximate viral particle density of n = 2 x 10^{-3} viral particle/liter. A small fraction of this viral particle density will survive HEPA filtering and then be introduced into the airstream occupied by H by the HVAC circulation. Due to filtering and mask wearing, there are dilution factors D that lower the amount of viral particles that H is exposed to (see App. A.2 and App. A.3). For HEPA-filtered air from the HVAC unit, D_{hvac} = *q*, where *q* = 3×10^{-4} is the filter penetration. If both S and H wear surgical (N95) masks, D_{mask} = 0.5 (D_{mask} = 0.1). Then, for small probabilities *p* of H contracting COVID-19, $p_{3week} = 540\, n\times D_{total}$ (see App. D.1) in a six-month period of work, p_{3week} is evaluated to be

a) Surgical, HEPA, $D_{total} = D_{hvac}D_{mask} = 3\times10^{-5}$ → OK: p_{3week} << 1%
b) No masks, HEPA, $D_{total} = D_{hvac} = 3\times10^{-4}$ → OK: p_{3week} < 1%
c) N95, no HEPA, $D_{total} = D_{mask} = 0.1$ → not OK: p_{3week} >> 1%

We note that if H were to reside in the airstream from S (i.e. be in the airstream from S to the input of the HVAC unit) then p~ 20% for one week of exposure to this viral load, which would be

unacceptable. Conclusions in (a), (b), and (c) remain unchanged when considering 26 weeks of exposure. We also note that the above "No masks" case is for illustrative purposes only. All workers should wear masks to mitigate droplet transmission and fomite creation, as well as to attenuate the effects of sneezes and coughs.

2.2 Risk Level

If the risk of contracting COVID-19 in the lab over 6 months is $p < 1\%$, how does this compare to risks from other sources? Although richer comparisons exist, as a base line one can note that the risk of death due to COVID-19 contracted in the laboratory, under the $p = 1\%$ condition, is far smaller than the typical all-cause mortality rate for the typical age of graduate students and postdoc researchers. This level of risk leads to a *marginal* ***fractional*** *increase* in expected mortality in six months of < 2% (see below for detailed estimate). Individuals outside of this age range or with pre-existing medical conditions may be at elevated risk and this situation can be analyzed in the same way.

2.2.1 Comparison to Other Risk Levels

The chosen level of risk (for a COVID-19 contraction risk over 6 months of $p = 1\%$) can be compared to a variety of other standards. See App. B for more details.

1. **Daily risks excluding COVID-19.** Mortality risks may be compared with the all-cause mortality rate of an individual in the typical researcher age demographic, 25-34, excluding COVID-19 and lab-related work. This is approximately 0.05% likelihood of death per 6 month period (Murphy et al. 2015). Estimates of the COVID-19 infection fatality rate (from the Italian outbreak) are ~0.1% for ages 25-34 (Rinaldi et al., 2020). Because our acceptable risk level limits *contraction* of COVID-19 at $p = 1\%$, the 6-month probability of death would be <0.001%, i.e. 2% of the all-cause mortality probability excluding lab-related COVID-19 contraction.
2. **Lab-related risks excluding COVID-19.** Reports of injuries in academic laboratories over the ten-year period of July 2008 - July 2018 give an approximate 0.04% likelihood per person per year of an OSHA reportable injury (U.S. Chemical Safety Board 2018; Widener 2018).
3. **Exposure to COVID-19 outside of lab.** The lack of widespread testing means the prevalence of COVID-19 is likely severely underestimated. Several serological studies have been conducted to attempt to estimate the extent of exposure, with varying results. These have found that between 3% and 30% of the tested populations have been exposed to SARS-CoV-2 (Grzelak et al., 2020). While there are valid concerns about the reliability of the tests and whether the tested populations were representative, there is widespread agreement that the prevalence of COVID-19 is significantly higher than current official counts (Vogel, 2020). One scenario of an essential activity where one can expect a high exposure to SARS-CoV-2 is a trip to the supermarket. Under the same model as we use to assess risk in labs, we estimate that if an individual makes one trip per week and spends one hour each time, then their cumulative probability of infection after six months (26 grocery trips) is approximately 6% (see App. B.4).

3. Discussion and Guidelines

The analyses above make clear the importance of air flow and filtration, and, secondarily, mask use. We recommend mask use primarily to lower the contamination of surfaces and to lower the spread of large droplets. By the analysis presented in this document, the following guidelines indicate laboratory or office work procedures that we calculate lead to a risk of contracting COVID-19 of $p < 1\%$ over 6 months, under the reasonable assumption of 3 total weeks of exposure to an infected person (and also for the conservative assumptions of 26 weeks of exposure):

- **Offices:** Whenever possible, office work will be conducted from home instead of at Harvard. Office rooms shall be single occupancy. Each office space will be evaluated for air flow and size to determine a minimum acceptable time to remain empty before a new researcher may enter. For a typical office room, this will be approximately 2.5 hours (5 hours) for 3 weeks (26 weeks) of total exposure over six months. Masks could be worn in offices to reduce the wait time and minimize surface contamination.
- **Laboratories:** For lab rooms without HEPA filtration, the Office guidelines shall be used. For lab rooms with HEPA filtration, multiple occupancy is allowed under the conditions of large distancing (>5 m) and positioning workers in separate airstreams. Experimental validation of filtration and airstream separation can be performed. Wait time is required between different users accessing a given area of a room. For a typical HEPA filtered lab this would be 1 air change (for either 3 or 26 weeks of exposure in a six-month period). For shared lab resources (e.g. electronics rooms, storage cabinets, chemical rooms, etc.) without HEPA filtration, a wait time of at least 4 air changes will be required (see App. A.5).

We note, as before, all workers should wear masks to mitigate droplet transmission and fomite creation, as well as to attenuate the effects of sneezes and coughs.

Appendix

A.1 Estimation of infection probability from viral load

We can define an infection probability using an exponential dose-response model (T Watanabe, et al 2010). For a viral dose d, the infection probability is defined as

$$p(d) = 1 - exp(-d/k),$$

where k is a virus-dependent single-pathogen infection parameter. Because of a shortage of research studies on SARS-CoV-2, the constant k is not known. For SARS-CoV-1, $k \approx 400$ PFU while a wider variety of viruses have $k \approx 10 - 3,000,000$ PFU (T Watanabe, et al 2010, T. Watanabe, et al 2012), with the higher range applicable to several strains of influenza. For influenza and other RNA viruses, the particle-to-PFU ratio is in the range of 10:1 to 100:1 (Fonville, et al. 2015). A lower particle-to-PFU ratio for a given value of k represents a greater hazard, so we conservatively assume 10:1. Using a conservative value of $k \approx 10$ PFU, we assume $k \approx 100$ viral copies. The only acceptable working conditions are those in which the probability of infection remains low, applicable when $d \ll k$, in which case $p(d) \approx d/k$. With this information, we can estimate a reduction in infection probability as a function of reduction in viral dose as given in Table A.1.1.

Viral load reduction(%) → $p(d_1)/p(d_0)$ (%)↘	**10**	**30**	**50**	**70**	**90**	**99.9**
k = 100	96.5	87.1	73.1	52.1	20.9	0.2
k = 4000	90.2	70.5	50.6	30.5	10.2	0.1

Table A.1.1.The reduction in infection probability as a function of reduction of viral load for two cases of k = 100 (Influenza A) and 4000 (SARS-CoV-1). The initial viral dose is fixed at d_0 = 2.3$\log_{10}$ copies. Note that to achieve a 3$\log_{10}$ reduction in infection probability for this fixed value of d_0, one requires at least a 99.9% reduction in viral load.

One might compare the dose-response model above with a simple model in which each viral copy is associated with a small risk, $1/k$, of becoming infected. Then the total probability of infection after a dose of d viral copies is

$$p(d) = 1 - (1 - 1/k)^d$$

For example, with $k = 100$ the risk of becoming infected is 1% per viral copy. The two formulas above for the dose-response model are equivalent to an extremely good approximation for any $k \geq 50$. Therefore, our target of $< 1\%$ probability of infection means a researcher should not be exposed to even a single viral copy on average in six months of work.

A.2 Mask Filtration Dilution Factor

Density of viral particle without dilution, n_{ND}, is related to that with dilution n through $n = D_{mask} \times D_{hvac} \times n_{ND}$. Here, D_{mask} is the mask attenuation to the inhaled dose and D_{hvac} is the dilution of viral particles in the air, set by air flow, filtering and room volume considerations, treated in App. A.3. The total dilution factor is $D = D_{mask} \times D_{hvac}$. As described below, we determine a working value for D_{mask} for a single wearer to be 0.5 (0.1) for surgical (N95) masks. In the scenarios covered in this document all persons are wearing a mask, so that the value used in calculations is D_{mask} = 0.25 (0.01) for surgical (N95). These are somewhat conservative estimates to accommodate the possibility of imperfect mask use.

Surgical mask usage in the study by Leung et al., 2020 was found to reduce the viral load to 0.3 log-10 copies (the noise level) over a 30 minute period for coronaviruses and to < 2.4 log-10 copies for rhinoviruses. Other studies of surgical masks with filtering material are found to block 50-80% of aerosols < 1 um and ~90% of particles > 1 um (Weber et al., 1993; Chen, et al., 1992) under normal breathing conditions. See Table A.2.1 for a summary of data. Note that it has been found that surgical masks provide essentially *no* reduction in aerosolized virus following coughs or strenuous breathing (Bae et al, 2020). Although properly worn surgical masks and N95 filtering facepiece respirators without exit valves (N95 FFR, or commonly simply 'N95') can reduce the emitted viral load (by factors of ~2-5 and ~10-100, respectively) during normal breathing, their performance is strongly reduced when worn improperly or during strenuous breathing/coughing. N95 masks with exit valves do not significantly reduce the viral load emitted by the wearer in aerosols. N95 masks are found to block >99% of aerosols and droplets if properly sealed (Rengaswamy et al., 2012; Cho et al., 2010). Proper fit and usage must be ensured to achieve nominal mask performance. This is not a trivial matter, as proper usage of N95 masks requires careful training and testing.

	No mask	Surgical mask	N95 mask
Normal breathing	$1.2^{1,*,\dagger}$, $3.3^{1,\ddagger,\dagger}$	$0.3^{1,*,\dagger}$, $0.3^{1,\ddagger,\dagger}$	0.3^{3***}
Coughing	$2.3^{2,**}$	$1.8^{2,**}$	0.3^{3***}

Table A.2.1: Viral load (log-10 copies) produced by an infected person (A) as a function of parameters.

[1] N.H.L. Leung, et al, [2] S. Bae, et al, [3] D. F. Johnson et al 2009

† Patients coughed an average 17 times during 30 min exhaled breath collection

* For particles > 5 μm, upper end of inter-quartile range, $\log_{10}$ particles, 0.3 implies undetected, collected over a 30 min period

‡ For particles ≤ 5 μm, upper end of inter-quartile range, $\log_{10}$ particles, 0.3 implies undetected, collected over a 30 min period
** For all particle sizes per cough, numbers averaged over 4 patients used in study, $\log_{10}$ particles/ml
*** Not done explicitly for SARS-CoV-2 but done for droplets and influenza. Taking 0.3 to imply undetected, $\log_{10}$ particles/ml

The efficiency of filtering out bio-aerosol particles by size for surgical and N95 masks are given in Tables A.2.2 and A.2.3. The average leakage of bio-aerosol particles of all sizes for a sealed surgical mask is ~9% while that of a sealed N95 mask is ~0.5%. In a more realistic scenario, assuming the mask is not perfectly fitted to the user, we see that a surgical mask leaks ~20% while an N95 mask can leak up to 10%. In the case of using a surgical mask without a filter material, the penetration rate can be as high as 80%, shown in the bottom row of table A.2.2.

Aerosol Diameter (μm)	**0.2**	**0.5**	**1.0**	**2.0**
Surgical Mask (Flat, sealed)[1] (%)	12-18	10-12	6-8	3-5
Surgical Mask (Flat, 4 mm leak diameter)[1] (%)	22-28	18-20	12-17	12-15
N 95 mask (sealed)[2] (%)	0.5-2.0	0.5-1.0		
N 95 mask (2x 3 mm diameter leak)[2] (%)	8-10	8-10		
N 95 mask (sealed)[3] (%)			0.1-0.2	0.05-0.1
N 95 mask (40% of the circumference unsealed)[3] (%)			0.6-0.7	0.4-0.5
Surgical mask without filter material [4] (%)	80-85	85-90	83-87	75-80

Table A.2.2. Penetration (Towards Inward Leakage, TIL) of aerosol particles by size through various masks. Data for flow rate of 30 l/min (32 l/min for reference 2) with masks on mannequins. This flow rate represents a typical breathing flow rate for a human. [1] A. Weber, et al (1993), [2] S. Rengaswamy, et al (2012), [3] K.J. Cho, et al (2010), [4] C. Chen, et al (1992)

Aerosol Diameter (μm)	**0.1**	**1.0**
Surgical mask (through filter) (%)	5-8	5-6
Surgical mask (through face seal leakage) (%)	22-47	15-35
N95 mask (through filter) (%)	0.4-0.8	0.2-0.4
N95 mask (through face seal leakage) (%)	2.5-7.2	1.5-4.5

Table A.2.3. Penetration (Towards Inward Leakage, TIL) of aerosol particles by size through various masks. Data was taken on human subjects (25 subjects x 3 repetitions). Read off from the plots in S. A. Grinshpun, et al (2009)

A.3 HVAC Dilution Factors

D_{hvac} parametrizes the dilution of the density of viral particle n through air exchanges and filtering. We treat two cases separately, that of entrained flow in a HEPA filtered room and mixed flow in an unfiltered room, see App. A.4 for more details.

HEPA filtration directly impacts D_{hvac} as aerosols are highly filtered with each full room air change. The HEPA filter has a minimum efficiency for aerosols around 0.1-0.3 μm, where the penetration is $q = 3\times10^{-4}$ (D.B. Day et al., 2018; P. Chuaybamroong, et al; Zhang et al., 2014). HEPA filtration efficiency (*f*) is measured to be 99.994% at 50 nm, 99.98% at 0.1-0.2 μm, and >99.996% for particles >0.5 μm (Zhang et al., 2014; Morono et al., 2018). We take a nominal penetration value $q = (1-f) \sim 3\times10^{-4}$. After one air change time with entrained flow, $D_{hvac} = q$ (see App. A.4).

In the case of mixed flow, without filtration, n decreases exponentially as a function of time. Thus, D_{hvac} is time dependent and decreases in value (that is, improves) over time as air is circulated and exchanged. In a recently vacated (empty) room, the density of viral particle n will decrease as a function of time as fresh air enters and exits the room and the amount of virus particles in the room depends on the air exchange rate in and out of the room (r [1/min]) and *f*.

A.4 Air Flow Conditions

Air flow conditions strongly influence viral particle distribution (L.A. Anchordoqui, et al (2020); Guo, et al (2020)). We consider two environments with different air flow conditions: laboratories and offices.

In a lab (with ~10-20 air changes per hour), flow rates through the HVAC system are high enough to create a steady “drift” of air across the room (from an air inlet to an outlet) that hydrodynamically entrains aerosol particles. The time required for aerosol particles to diffuse across the room is much longer than the drift time for air to travel completely from an inlet to an outlet. In this case, the viral particle density in a room that has been vacated by a shedding individual decreases by a factor of q (due to HEPA filtering) after a duration of $1/r$, where r is the air exchange rate through the HVAC system (not necessarily the rate to introduce fresh air). The dilution factor for viral particle density remaining after time t in this case is $D_{hvac}(t) = e^{r\,ln(q)t}$. For all labs, $r > 10$ per hour, and typically $r = 15-20$ per hour.

In an office setting, flow rates are low enough that some diffusion throughout the room may occur on the time scale of an air exchange. We conservatively assume full mixing of fresh air introduced by the HVAC system and remaining air in the office. In this case, only a fraction of the contaminated air is removed in the time required to introduce a full room volume of fresh air,

and the dilution factor for viral particle density remaining after time t is $D_{hvac}(t) = e^{-rt}$, where r is the fresh air exchange rate. For a typical office, $r \approx 3$ per hour.

In some cases, laboratories have well-defined areas with separate airstreams that do not mix with each other. The degree of isolation between these areas could be confirmed by testing the migration of aerosol particles. If no migration between two separate airstreams occurs, then it is safe for one person to work in each airstream. Separate HVAC inlets and outlets within the same room are typically separated by distance scales > 5 m, so we require individuals working in separate airstreams to remain >5 m apart at all times. (This is much more conservative than the CDC social distancing recommendations to remain >2 m apart (CDC (2020))). These conditions are generally not met in an office setting, so, generally, offices may never be simultaneously occupied.

A.5 Auxiliary prep spaces

We also consider auxiliary prep spaces such as chemical or electronics rooms that will be occupied. Because multiple subgroups share these spaces, they may provide connections between H and a larger set of persons S. On the other hand, H will generally spend less time in these rooms. We conservatively assume that such auxiliary prep spaces have no HEPA filtration and air circulation rates similar to office spaces (~3/hour). We say that H only spends (T_{spent}) 2 hours per day on average in such rooms.

First consider the case that every time H enters the room, it had previously been occupied by S. H enters after a variable wait time T_{wait}. The viral load decreases from the steady state value of $n \sim 3 \times 10^{-2}$ virus particles/liter by a factor of $e^{-rT_{wait}}$. In addition, H only gets exposed to a viral load of $d_0 = ne^{-rT_{wait}}(1 - e^{-rT_{spent}})V'/r$ each time because the air continually circulates during H's occupancy. Here $V' = 450$ L/h is the breathing rate of H (see Sec. 2.1). However, if there are N people besides H who share an auxiliary prep space, and $N_S^{(i)}$ of them are sick on any day (i), the average viral load to H on day (i) is $d^{(i)} = d_0 N_s^{(i)}/N$ since H has an equal chance of entering after any given person whether healthy or sick. Assuming conservatively that all workers besides H become infected at some point in a 26 week work period, and each sheds virus for five days while at work, the total viral load to H is $d = 5d_0$, i.e. equivalent to that due to always entering after an infected person for one week. With that we find that a wait time of 3 air change (~60 minutes) would be sufficient to reduce the 6-month probability to ~1%, and 4 air changes (~80 minutes) sufficient for $p \ll 1\%$.

Bathrooms are also commonly shared by people from multiple groups and subgroup, so the same model applies with different parameters. A typical bathroom with 2000 cubic feet of volume and 6 fresh air changes per hour has a steady state value of virus density of $n \sim 10^{-2}$ viral copies per liter. Assuming each bathroom visit lasts 5 minutes, the viral load on H per visit is $d_0 = 0.040$ provided a healthy person H waits 2 air exchange times (~20 minutes) before entering. With three bathroom visits per day over a six-month working period, the total viral load

is $d = 15d_0 = 0.60$ (see discussion above for auxiliary prep spaces), sufficient to ensure a probability of infection $< 1\%$ over six months of work. If there is zero wait time between occupants, p ~ 4 %.

The risk due to H and S traversing common spaces (e.g. through hallways) can be analyzed using the same methods presented here. However, the time spent in these areas is very short, greatly reducing the risk of infection from these sources.

A.6 Model Parameters

For ease of use, we compile here the parameters assumed in the above analysis.

Parameter	Meaning	Value
D_{mask} (N95)	N95 filter efficiency (single wearer)	0.1
D_{mask} (surgical)	Surgical mask filter efficiency (single wearer)	0.5
$q\ (= 1 - f)$	HEPA filter penetration	3×10^{-4}
k	Single-pathogen infection parameter	100

B.1 Estimated COVID-19 mortality rate

The currently accepted mortality rate for COVID-19 is estimated to be near 1%. Two studies of the outbreak in China placed the case fatality rate at 1.4% (Wu et al., 2020; Verity et al., 2020). The *infection* fatality rate was estimated to be 0.7%, where the lower value is due to more complete estimates on infection prevalence (Verity et al., 2020). Researchers estimated the case fatality rate in the Gangelt municipality to be 0.37% (Streeck et al., 2020). Meanwhile, for the outbreak in Italy an infection fatality rate of 1.3% was determined (Rinaldi et al., 2020). A clear and significant age dependence is seen in fatality rate data: ~0.1-0.3% for ages 20-30, ~0.15-0.35% for ages 30-50, and ~0.7-1.25% for ages 50-60 (Oxford COVID-19 Evidence Survey; Verity et al., 2020; Wu et al., 2020). Note that due to a lack of widespread testing, and because many infections are mild or asymptomatic, the infection fatality rate is significantly lower than inferred by a naive comparison of official counts of confirmed COVID-19 cases and deaths.

B.2 Comparison to other laboratory mortality risks

To provide context for the previous risk analysis, we review typical risks associated with work in a university laboratory, separately considering deaths and OSHA-reportable injuries.

We first consider accidents directly leading to death. In the past 10 years, to our knowledge there have been four laboratory-related accidents resulting in death in US-based academic institutions (Laboratory Safety Institute 2019). Based on the limited number of incidents, physics research represents a larger risk of mortality than accidents in all science labs on average. The large majority of personnel in physics laboratories are graduate students, of which there are approximately 16,000 in any given year in the United States (Nicholson, et al. 2017). Although there are also some non-graduate student personnel in physics laboratories, we believe that this is largely balanced by the fact that not all graduate students work in laboratories (and instead conduct office-based work). Therefore the risk of death in a physics laboratory in American universities over the past decade is approximately $2/(10 \times 16000) \approx 0.001\%$ per researcher per year.

In addition to accidents that directly cause death, researchers may face long-term risk due to exposure to hazardous environments. Although this is difficult to estimate in general, we consider the long-term effects of ionizing radiation on the small subset of researchers who work with radioactive materials. At Harvard University, the department of Environmental Health and Safety sets a limit on radioactive exposure for radiation workers equal to 0.5 rem per year (Harvard University 2002). However, “greater than ninety percent of all users of all radioactive material at Harvard have had an annual dose less than 100 mrem.” We therefore suppose a heavy radiation user at Harvard is exposed to approximately 0.1 rem per year.

The seventh Biological Effects of Ionizing Radiation (BEIR) report has determined an excess lifetime attributable mortality risk of approximately 0.05% per rem of exposure to ionizing radiation (Committee 2006), giving a heavy radiation user at Harvard an expected 0.005% likelihood of death due to exposure-related cancer in their lifetime. Data from other sources gives a similar estimate, but slightly higher for workers aged 20 to 30, because the lifetime risk of developing cancer due to radiation decreases with age of exposure. Using the age-grouped data from (Sodickson et al. 2009) we estimate a heavy radiation user at Harvard to incur a 0.008% likelihood of death due to exposure-related cancer in their lifetime.

All of these mortality risks may be compared with the all-cause mortality rate of an individual in the typical researcher age demographic, 25-34, which is approximately 0.1% likelihood of death per year (Murphy et al. 2015).

B.3 Comparison with other laboratory injury risks

We would also like to estimate the rate of injuries not leading to deaths, which might be compared to the risk of hospitalization due to COVID-19. Because all-cause injury rates in academic laboratories are not available to our knowledge, we consider only chemical accidents leading to injury. These are compiled by the Chemical Safety Board, and rely on the OSHA definition of a reportable injury: “injury or illness that results in loss of consciousness, days away from work, restricted work, or transfer to another job … or illness requiring medical treatment beyond first aid” (OSHA).

In the ten-year period between July 2008 and July 2018, there were an average of 11.3 injuries recorded per year in university settings over the ten-year period of July 2008 - July 2018 (U.S. Chemical Safety Board 2018). Within this period, there were approximately 27,000 chemistry and chemical engineering graduate students in the United States at one time (Widener 2018). As before, we suppose that the number of graduate students is reasonably representative of the population at risk of injury in academic laboratories, giving an approximate 0.04% likelihood per person per year of an OSHA reportable injury.

B.4 Probability of infection outside the lab

We can also compare the acceptable risk to other activities one will usually perform during quarantine. Here we consider grocery shopping at a supermarket. Supermarkets usually use HVAC filters which can go up to 94% filtration efficiency (Service Champions website).

Parameter	Lab	Supermarket
Size (ft^3)	3200	120000
Filtration Efficiency	99.97% HEPA	94% HVAC
Air Exchange Rate (min^{-1})	0.1	0.05

Table B.4.1. Comparing a laboratory with a typical supermarket

With safety guidelines, a typical supermarket has about 100 people inside and we assume that about 2% of the individuals inside are infected at any one time (which would correspond to approximately 50% of other shoppers being infected at some point over six months, provided each person only makes one grocery trip while shedding virus). Assuming no hydrodynamic entrainment of aerosols through the HVAC system and using the parameters in Table B.4.1, with a shedding rate of 70 viral particles/min per infected person (without mask use) the steady state density of SARS-CoV-2 is $n \approx 10^{-3}$ viral particle/L. Supposing that a healthy individual wearing a surgical mask goes to the supermarket once a week and spends 1 hour inside, the probability that the healthy person gets infected after 6 months is 6%.

C.1 Viral Shedding

Studies of viral shedding for aerosols in the <5 μm size range find that the emitted viral load under normal breathing conditions can reach 3.3 log-10 copies over a 30 minute period for a variety of viruses, including coronaviruses, rhinoviruses, and influenza viruses (Leung et al., 2020). There is further evidence that vocalization, loudness of speech and speech "super-emitters" can lead to much higher rates of emission (S. Asadi, et al). In addition, recent studies have shown that the levels of live virus shedding from the nasal cavities of asymptomatic patients can be high (M. Gandhi et al., 2020).

C.2 Estimation of particle suspension time

A straightforward quantity to estimate in the case of aerosol particles suspended in air is the time it takes for the particles to drop on the surface. The expression using Newton-Stokes law for this suspension time is (N. Singh, et al (2020))

$$\tau = 4.5\left(\frac{\eta h}{g\rho r^2}\right)$$

Where, η is the viscosity of air at 25°C = 1.85×10^{-5} kg/m-s, h is the height of the particle, g is the acceleration due to gravity = 10 m/s^2, ρ is the density of water = 1000 kg/m^3 and finally r is the radius of the particle. If we take the height to be 2 m for an average individual, we get the following suspension times as a function of droplet size in Table C.2.1.

Droplet diameter (μm)	0.2	0.5	1	2	5
Suspension time	19 days	1.2 days	18 hours	5 hours	45 minutes

Table C.2.1. Suspension time versus droplet diameter for water droplets in air. The droplets experience the gravitational force in the vertical direction and the suspension time is how long it takes for the particle to reach the ground, 2 m away.

We conclude that for the droplet sizes we are considering for this study, since diffusion timescales are longer than the Newton-Stokes timescales, diffusion becomes negligible and we should concern ourselves only with drift.

C.3 Lifespan of SARS-CoV-2 on aerosols/surfaces According to the FDA, the acceptable level of air purification is a 4-log reduction in SARS-CoV-2 viral activity while for surface decontamination, a 6-log reduction is recommended (FDA, 2020). Recent studies have quantified the duration that the SARS-CoV-2 virus remains viable in different media (N. van Doremalen, et al., 2020). Their findings, translated to a normal viral deactivation time in accordance with FDA guidelines is given in Table C.3.1. We focused on aerosols and material surfaces usually found in a research lab. We note that the viral deactivation time in bio-aerosols, without human intervention, is much longer (13 hours) than entire room air exchange time in the rooms considered in this report. Thus for the purposes of this study, the limiting process will be air filtration. The effect of indirect exposure through surface contact is beyond the scope of this work.

Surface type	Aerosols	Copper	Stainless Steel	Plastic
Deactivation time (hrs/days)	13/0.5	10/0.4	120/5	140/5.8

Table C.3.1. Natural SARS-CoV-2 viral deactivation time for different media. For aerosols, deactivation time corresponds to a reduction of viral load by 4-$\log_{10}$. For surfaces, reduction by

6-$\log_{10}$. Data taken from (N. van Doremalen, et al). An N-$\log_{10}$ decontamination time (t_D) is related to the half life $t_{1/2}$ by $t_D = N t_{1/2}(log_{10}(e) ln(2))$.

The viral load itself is distributed among bio-aerosol particles of sizes ranging from 0.8 to 5.5 μm in the case of normal breathing (L. Morawska, et al) and the number of bio-aerosol particles (of all sizes >150 nm) emitted during exhalations can vary between 38±21 for low emitters to 1500±900 for heavy emitters, cumulatively over a 6 hour period (D.A. Edwards, et al).

C.4 Estimation of basic reproduction number R_0 The basic reproduction number is defined simply as the expected number of cases generated by one infected individual. For a disease to be classified as an epidemic, this number R_0 must be > 1. The currently accepted value of R_0 for COVID-19 is ~5.7 with a serial interval of 6-9 days (S. Sanche, et al 2020). The serial interval is defined as the time interval between the onset of primary cases and the onset of secondary cases. For our simple model of controlled exposure to COVID-19 via lab activities, we can employ a simple "Susceptible-Infected-Removed" (SIR) model to predict the value of R_0 (I. Nesteruk, et al 2020).

Within the simple SIR model, we assume that a closed population of N is distributed amongst susceptible (*S*) individuals, infected (*I*) individuals and removed (*R*) individuals. In addition, we assume an infection probability (*p*) upon contact with an infected person and a rate of contact (*c*). So the net probability of being infected is *pc*.

Next we can define a removal rate (*r*) that is effectively the inverse of the serial interval of the disease. One way to think about it is that if the symptoms take ~5 days to appear, so an infected person cannot be identified and removed from the population faster than that. With these, we can write the rate of change of *S,I* and *R* as

$$dS/dt = -pcSI$$

$$dI/dt = pcSI - rR$$

$$dR/dt = rR$$

The basic reproduction number is tied in with these parameters as

$$R_0 = pc/r$$

Such that for $R_0 > 1$, $dI/dt > 0$ at the onset of an epidemic, as expected. Knowing that in our case study of a return to the lab, the accepted infection probability is *p* ~ 1% over 26 weeks, so the hourly infection probability is ~ 0.001%. The exposure time is 8 hours per day and the serial interval (1/*r*) is ~5 days. Given these numbers, we would predict a marginal increase in R_0 ~ 0.0004, which classifies the lab working environment as non infectious.

D.1 Probability Formulas for Certain Examples

Below are the formulas referred to in Sec. 2.1.
Let us begin by saying that a viral density *n* (per liter) is present in the air that H is breathing.

The breathing rate is 15 times/min and each breath has a volume of 0.5 liter. This means the total volume inhaled by H per hour is V' = 450 litres/hr. This implies that the viral load inhaled by H per hour is 450n.

Now over 1 week, with 8 hours per day and 5 days of work, a total of 40 hours of exposure per week, the total viral load inhaled by H is 1.8×10^4.

Hence the infection probability after a week is (d_{1week}), assuming k=100

$$p(n) = 1 - \exp(-(1.8\times10^4)\times n/k) = 1 - \exp(-180n)$$

And for small p, n is given by

$n = -(6 \times 10^{-3}) \ln(1-p) \sim (6 \times 10^{-3})p$ *(for small p)*

$p = 180n$

Similarly, in the limit of small p, for 3 weeks (120 hours of exposure, d_{3week})

$$p = 540n$$

And for 26 weeks (1,040 hours of exposure, d_{26week})

$p = 5\times10^3\ n$

References (* - not peer reviewed)

***** L A. Anchordoqui, et al. A physicist view of the airborne infection, arXiv:2003.13689

S. Asadi, et al. The coronavirus pandemic and aerosols: Does COVID-19 transmit via expiratory particles? Aerosol Science and Technology 2020, Vol. 54, No. 6, 635–638

S. Bae, et al. Effectiveness of Surgical and Cotton Masks in Blocking SARS–CoV-2: A Controlled Comparison in 4 Patients. Annals of Internal Medicine (2020)

*L. Brosseau, Commentary: COVID-19 transmission messages should hinge on science, Center for Infectious Disease Research and Policy (2020)

*G. Buonanno, et al. Estimation of airborne viral emission: quanta emission rate of SARS-CoV-2 for infection risk assessment, medRxiv 2020.04.12.20062828

M. Cascella et al., Features, Evaluation and Treatment Coronavirus (COVID-19), StatPearls, accessed online 4/28/2020.

*Center for Disease Control, Coronavirus Disease 2019 Symptoms, 2020. URL:https://www.cdc.gov/coronavirus/2019-ncov/symptoms-testing/symptoms.html

*Center for Disease Control, Coronavirus Disease 2019 Social Distancing, 2020, URL:https://www.cdc.gov/coronavirus/2019-ncov/prevent-getting-sick/social-distancing.html

W. Chen, et al. Short-range airborne route dominates exposure of respiratory infection during close contact, Building and Environment, 176 106859 (2020)

K.J. Cho, et al (2010). Large Particle Penetration through N95 Respirator Filters and Facepiece Leaks with Cyclic Flow, The Annals of Occupational Hygiene, Volume 54, Issue 1, January 2010, Pages 68–77

P. Chuaybamroong, et al. Efficacy of photocatalytic HEPA filter on microorganism removal. Indoor Air, 20: 246-254

*Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation, Health risks from exposure to low levels of ionizing radiation: BEIR VII Phase 2, 2006. 10.17226/11340

D. B. Day, et al. Combined use of an electrostatic precipitator and a high-efficiency particulate air filter in building ventilation systems: Effects on cardiorespiratory health indicators in healthy adults. Indoor Air. 2018; 28: 360– 372

D. A. Edwards, et al. Inhaling to mitigate exhaled bioaerosols. PNAS December 14, 2004 101 (50) 17383-17388

*FDA (2020), Enforcement Policy for Sterilizers, Disinfectant Devices, and Air Purifiers During the Coronavirus Disease 2019 (COVID-19) Public Health Emergency

J. M. Fonville, et al. Influenza Virus Reassortment Is Enhanced by Semi-Infectious Particles but Can Be Suppressed by Defective Interfering Particles, 2015. 10.1371/journal.ppat.1005204

M. Gandhi, Asymptomatic Transmission, the Achilles' Heel of Current Strategies to Control Covid-19, NEJM, 10.1056/NEJMe2009758 (2020)

S. Grinshpun et al., Performance of an N95 filterting facepiece particulate respirator and a surgical mask during human breathing: two pathways for particle penetration, J. Occ. Env. Hyg. 6:10 593-603 (2009).

*Grzelak et al., SARS-CoV-2 serological analysis of COVID-19 hospitalized patients, pauci-symptomatic individuals and blood donors, medRxiv doi: 10.1101/2020.04.21.20068858 (2020).

*Z-D Guo, et al. Aerosol and Surface Distribution of Severe Acute Respiratory Syndrome Coronavirus 2 in Hospital Wards, Wuhan, China, 2020. 10.3201/eid2607.200885

Harvard University, Radiation Safety Manual (2002). Accessed online 4/28/2020.

D. F. Johnson, et al., A Quantitative Assessment of the Efficacy of Surgical and N95 Masks to Filter Influenza Virus in Patients with Acute Influenza Infection, 2009. 10.1086/600041

J.-M. Kim et al., Identification of coronavirus isolated from a patient in Korea with COVID-19, Osong Public Health and Research Perspectives 11(1) 3-7 (2020).

*Laboratory Safety Institute, Memorial Wall, Accessed online 4/30/2020.

N.H.L. Leung, et al. Respiratory virus shedding in exhaled breath and efficacy of face masks. Nature Medicine (2020)

*Y. Li et al., Evidence for probable aerosol transmission of SARS-CoV-2 in a poorly ventilated restaurant. medRxiv, doi: 10.1101/2020.04.16.20067728 (2020)

Y. Liu et al., Aerodynamic analysis of SARS-CoV-2 in two Wuhan hospitals, Nature https://doi.org/10.1038/s41586-020-2271-3 (2020).

J. Lu et al., COVID-19 outbreak associated with air conditioning in restaurant, Guangzhou, China, 2020, Emerging Infectious Diseases Journal Vol. 26, No. 7 (2020).

M. Meselson, Droplets and aerosols in the transmission of SARS-CoV-2, NEJM doi: 10.1056/NEJMc2009324 (2020).
L. Morawska, et al. Size distribution and sites of origin of droplets expelled from the human respiratory tract during expiratory activities. Journal of Aerosol Science, Volume 40, Issue 3, March 2009, Pages 256-269

Y. Morono et al., Assessment of capacity to capture DNA aerosols by clean filters for molecular biology experiments, Microbes Environ. Vol 33, No. 2, 222-226 (2018).

*S. Murphy, et al., Mortality in the United States 2014, 2015.

*I. Nesteruk, et al 2020. Statistics based predictions of coronavirus 2019-nCoV spreading in mainland China. medRxiv 2020.02.12.20021931

*S. Nicholson, et al. Roster of Physics Departments with Enrollment and Degree Data, 2017.
Harvard University Radiation Protection Office, Radiation Safety Manual, 2002.

*OSHA, Injury and Illness Recordkeeping and Reporting Requirements. URL:https://www.osha.gov/recordkeeping/

*Oxford COVID-19 Evidence Survey: Global Covid-19 Case Fatality Rates, Oxford Centre for Evidence-Based Medicine, accessed online 4/29/2020.

S. Rengaswamy, et al (2012). Nanoparticle Penetration through Filter Media and Leakage through Face Seal Interface of N95 Filtering Facepiece Respirators, The Annals of Occupational Hygiene, Volume 56, Issue 5, July 2012, Pages 568–580

*G. Rinaldi and M. Paradisi, An empirical estimate of the infection fatality rate of COVID-19 from the first Italian outbreak, medRxiv doi:10.1101/2020.04.18.20070912 (2020).

S. Sanche, et al (2020). High Contagiousness and Rapid Spread of Severe Acute Respiratory Syndrome Coronavirus 2. Emerg Infect Dis. 2020 July

*N. Singh, et al. On the airborne aspect of COVID-19 coronavirus, arXiv:2004.10082

A. Sodickson, et al., Recurrent CT, cumulative radiation exposure, and associated radiation-induced cancer risks from CT of adults, 2009. 10.1148/radiol.2511081296

*H. Streeck et al., Vorlaufiges Ergebnis und Schlussfolgerungen der COVID-19 Case-Cluster-Study (Gemeinde Gangelt), 2020.

*U.S. Chemical Safety Board, Laboratory Incidents January 2001 - July 2018, 2018. URL:https://www.csb.gov/assets/1/6/csb_laboratory_incident_data.pdf

N. van Doremalen, et al. Aerosol and Surface Stability of SARS-CoV-2 as Compared with SARS-CoV-1, N Engl J Med 2020; 382:1564-1567

R. Verity et al., Estimates of the severity of coronavirus disease 2019: a model-based analysis The Lancet Infectious Diseases, Online, 2020.

*G. Vogel, Antibody surveys suggesting vast undercount of coronavirus infections may be unreliable, accessed online 4/28/2020.

*Service Champions website : supermarket HVAC ratings

J. Wang, et al. COVID-19 may transmit through aerosol, Irish Journal of Medical Science, 2020.

T. Watanabe, et al. Development of a Dose-Response Model for SARS Coronavirus, Risk Analysis, Vol. 30, No. 7, 2010

T. Watanabe, et al. Dose-Response Assessment for Influenza A Virus Based on Data Sets of Infection with its Live Attenuated Reassortants, 2012. 10.1111/j.1539-6924.2011.01680.x
Laboratory Safety Institute, Memorial Wall, 2018. URL:https://www.labsafety.org/memorial-wall

A. Weber, et al (1993). Aerosol penetration and leakage characteristics of masks used in the healthcare industry. American Journal of Infection Control, 21, 167-173.

*WHO Situation Report (2020). Coronavirus disease 2019 (COVID-19) Situation Report – 97.

WHO Guidance (2020). Laboratory biosafety guidance related to coronavirus disease 2019 (COVID-19). WHO/WPE/GIH/2020.2
C. Xu, et al (2015). Measuring the exhaled breath of a manikin and human subjects. Indoor Air Volume 25, Issue 2

*A. Widener, By the numbers: Who's going to grad school in chemistry and chemical engineering in the US 2006-16, 2018.

*R. Woelfel et al., Clinical presentation and virological assessment of hospitalized cases of coronavirus disease 2019 in a travel-associated transmission cluster, medRxiv doi:10.1101/2020.03.05.20030502 (2020)

J. Wu et al., Estimating clinical severity of COVID-19 from the transmission dynamics in Wuhan, China. Nature Medicine, Vol 26, 506-510, 2020.

Z. Zhang et al., A blunt sample probe developed for an in situ leak test of HEPA filters in high-level biosafety laboratory, HVAC&R Research 20 221-229 (2014)