Assessment and Mitigation of Aerosol Airborne SARS-CoV-2 Transmission in Laboratory and Office Environments

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Bioaerosols are known to be an important transmission pathway for SARS-CoV-2. We report a framework for estimating the risk of transmitting SARS-CoV-2 via aerosols in laboratory and office settings. High-circulation HVAC systems with HEPA filtration dramatically reduce exposure to the virus in indoor settings, and surgical masks or N95 respirators further reduce exposure. As an example of our risk assessment model, we consider the precautions needed for a typical experimental physical science group to maintain a low risk of transmission over six months of operation.

I. INTRODUCTION

Knowledge of the transmission pathways for SARS-CoV-2, the virus that causes COVID-19, is incomplete. It is currently believed that transmission can occur via both surface contact (fomites) and aerosols/droplets [1–3]. While, to date, there has been no documented case of fomite transmission of SARS-CoV-2 [4], there are a variety of reports of aerosol transmission in social settings [5, 6]. Aerosol transmission is an important pathway especially because both symptomatic and presymptomatic carriers can shed the virus [7] and aerosols can contain active virus for many hours in suspension [8]. Complicating the understanding of viral spread are the effects of local air flow conditions [4, 9]. Understanding of viral transmission dynamics will play a pivotal role in the development of procedures to minimize disease transmission in work settings.

Here we consider SARS-CoV-2 transmission via aerosolized particles. We focus in particular on the environments common in photonics, physical chemistry, atomic physics, and condensed matter physics experimental laboratories ("labs") and the offices that are often connected with those labs ("offices"). Combining the knowledge of aerosol transmission and mask effectiveness, the typical properties of labs and offices, and a simple dose-response model, we develop guidelines for laboratory work procedures. The guidelines developed 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. We present the details of our analysis in the hope that it can be applied by the reader to a variety of other laboratory and office settings.

II. AEROSOL TRANSMISSION AND PHYSICAL SPACES

Here we review literature indicating that aerosol transmission of SARS-CoV-2 is likely a significant contributor to the spread of infection. Furthermore, the lifetime of aerosols containing viable virus is long. Because the filtering and exchange of contaminated air with fresh air will lower the density of viral particles and can thus lower the probability of infection, we also discuss the typical air conditions in laboratory and office settings.

A. Aerosol transmission of SARS-CoV-2

Transmission of SARS-CoV-2 can occur even prior to onset of COVID-19 symptoms [7]. 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 [10]. A general, though not universal, convention is to call particles >5 μm droplets and particles <5 μm 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 μm in diameter [2]. Ordinary speech can also be a significant source of aerosolized particles [10]. Larger droplets are also suspected of providing a transmission route for SARS-CoV-2. However, large droplets >5 μm typically have shorter suspension

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times in air, $\lesssim$10 minutes [1]. Aerosols in the size range 1-5 μm are of particular concern, both because they are respirable and remain in the air for long times [1, 2, 11]. Studies have shown that SARS-CoV-2 can remain viable on aerosols for >3 hours, requiring ∼13 hours for a 4-$\log_{10}$ reduction [8, 12] (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 it is possible [13, 14]. 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 [14]. 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 [6, 15]. 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 [15]. 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. III). In a hospital setting, the distribution of virus-laden aerosols was found to be somewhat (albeit weakly) determined by airflow patterns in the AHUs [16]. 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 [17]. 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.

B. Physical Spaces: Laboratories and Offices

We consider a lab room as having an area of 500 square feet (∼50 m^2) with volume $V_{\rm lab}$ = 200,000 liters. A typical lab room is 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 for many physical science laboratories, 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, we consider a typical office room (which would usually be shared prior to the pandemic) of 200 square feet (∼20 m^2) and $V_{\rm office}$ = 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 [18, 19]. Filtration efficiency depends on particle size. Because virus-laden particles are often expelled from the body in combination with proteins, salts, and water as droplets and aerosols [4], it is not clear whether aerosol transmission of SARS-CoV-2 is caused by suspended individual viral particles, of size ∼50-150 nm [20, 21], or larger respirable aerosols of size ∼1-5 μm [1, 2]. 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 [22, 23]. To make conservative estimates below, we assume a nominal penetration value $q = (1 - f) \sim 3 \times 10^{-4}$, which matches the specified maximum value [4]. We note that to achieve this filtration that the HVAC unit needs to be in good working order. Specifically, the seals around the HEPA filters themselves needs to be adequate. Clean room facilities employ experimental procedures to test such functions.

III. 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 (Sec. III A), (2) calculate the probability of a healthy person being infected via aerosols by a virus shedding person under various physical conditions, e.g., one person per room, lab shared by two people, etc. (Sec. III B), (3) set a threshold for acceptable probability, p, of being infected over the course of 6 months (Sec. III C), and (4) devise mitigation approaches to attain that p (Sec. III D). We choose $p < 1\%$ over six months of work as a target, and compare the corresponding risks to (i) other daily risks excluding contracting COVID-19, (ii) work related risks only, excluding contracting COVID-19, and (iii) the risk of contracting COVID-19 outside of work.

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 and assume that a person works 40 hours per week. We assume that a healthy person significantly connects (e.g. shares in some way an office or lab) with 3 other people who are each shedding virus at different times. This gives a total exposure to the virus that lasts up to 3 weeks in a six-month period of work.

We also describe stricter protocols that would be required under the less realistic, but more conservative, assumption that H is exposed continuously to virus-shedding persons S for the entire six-month period (26 weeks). We do *not* consider the situation where mixing of air between rooms takes place through the HVAC system, which may be an important consideration for insufficiently isolated rooms.

A. Model and Examples

We use a dose-response model [24, 25], 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

$$p(d) = 1 - \exp\left(-d/k\right), \tag{1}$$

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.

Assume there are two people, S and H. By breathing, S emits a certain number of SARS-CoV-2 viral particles into the air. Under normal breathing conditions, S is expected to exhale ∼35-70 viral particles/minute [26, 27]. This will create (after mixing with a volume of air, filtering, etc.) some local density of viral particle n [viral particles/liter of air] that H inhales. The viral dose that H receives is $d = nV_{\rm inh}$, where $V_{\rm inh}$ is the total volume of air inhaled by H. At a typical breathing rate, H will inhale a volume of air per unit time $V' \sim 450$ L/h. For example, $V_{\rm inh} = 1.8 \times 10^4$ L during $T = 40$ hours of work and under these conditions $d_{3\ \rm week}$ =54,000 n and $d_{26\ \rm week}$ =470,000 n. The infection probability can be expressed as

$$p(n) = 1 - \exp\left[-\left(V'T\right) \times n/k\right] = 1 - \exp(-Bn). \tag{2}$$

For small probability, this simplifies to

$$p \approx Bn, \tag{3}$$

where $B_{3\ \rm week} = 540$ and $B_{26\ \rm week} = 4700$. For easy reference, we supply probability formulas in App. D.

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).

B. Probability of aerosol transmission

1. *Solo*

Consider first a room with parameters matching those of a typical office occupied by two persons, S and H. 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 $V_{\rm room} = V_{\rm office} = 80{,}000$ liters, the maximum density of SARS-CoV-2 is $n \sim 3 \times 10^{-2}$ viral particles/liter accumulating over an air exchange time of 30 mins, giving H 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 to H associated with occupying a lab that has been previously occupied by S. In this case, typically $V_{\rm room} = V_{\rm lab} = 200{,}000$ liters and the air exchange time is approximately 5 minutes. Because the labs are assumed in our scenario to be HEPA filtered and have high rates of air circulation, the contamination present

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

TABLE I. Values of model parameters assumed for example risk calculations.

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.

2. *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 Sec. III B 1 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 $V_{\text{room}} = V_{\text{lab}} = 200{,}000$ 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 $n = 2 \times 10^{-3}$ viral particles/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, $D_{\text{hvac}} = q$, where $q = 3 \times 10^{-4}$ is the filter penetration. If both S and H wear surgical (N95) masks, $D_{\text{mask}} = 0.5$ ($D_{\text{mask}} = 0.1$) [28]. Then, for small probabilities p of H contracting COVID-19, $p_{3\,\text{week}} = 540\, n \times D_{\text{total}}$ (see App. D) in a six-month period of work, $p_{3\,\text{week}}$ is evaluated to be

1. HEPA, no mask : $D_{\text{total}} = D_{\text{hvac}} = 3 \times 10^{-4}$ $\rightarrow$ OK : $p_{3\,\text{week}} \ll 1\%$
2. HEPA, N95 : $D_{\text{total}} = D_{\text{hvac}} D_{\text{mask}} = 3 \times 10^{-5}$ $\rightarrow$ OK : $p_{3\,\text{week}} \ll 1\%$
3. No HEPA, N95 : $D_{\text{total}} = D_{\text{mask}} = 0.1$ $\rightarrow$ not OK : $p_{3\,\text{week}} \gg 1\%$

We note that if H were to reside in the same airstream as S (e.g. be in the airstream from S to the input of the HVAC unit) then $p \sim 20\%$ for one week of exposure to this viral load. These conclusions 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. We summarize some important parameters assumed in this model in Tab. I.

C. Choice of 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 baseline 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 post-doctoral 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 their situation should be analyzed accordingly. 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 [29]. Estimates of the COVID-19 infection

fatality rate (from the Italian outbreak) are ∼0.1% for ages 25-34 [30]. 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 [31, 32].

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 [33]. 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 [34]. 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).

From a public health perspective, the risk level of $p = 1\%$ corresponds to a negligible marginal increase in the basic reproduction number, R_0. Adjusting the risk threshold, p, to values within a factor of 10 around 1% would leave the structure of our guidelines unchanged.

D. Guidelines and Discussion

The analyses above make clear the importance of air filtration, and, secondarily, mask usage. 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. The following guidelines correspond to laboratory or office work procedures which, based on the analysis presented here, will 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 should be conducted from home instead of the office. Office rooms shall be single occupancy. Each office space should 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 with ∼2 fresh air changes per hour, 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 "Offices" guidelines shall be used. For lab rooms with HEPA filtration, multiple occupancy is allowed under the conditions of large distancing (e.g >5 m) and positioning workers in separate airstreams. Experimental validation of filtration and airstream separation can be performed. A 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), for long exposure times.

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 for offices (or rooms without HEPA filtration) would be increased to 5 hours if $p < 1\%$ is to be maintained. For the laboratory case, 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). Another conservative assumption that underlies these guidelines is a risk threshold of $p = 1\%$, which if changed to $p = 10\%$ would indicate a wait time of <1 hour is required between occupancy of an office room.

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Appendix A: Risk and mitigation models

1. Estimation of infection probability from viral load

We can define an infection probability using an exponential dose-response model [24]. For a viral dose d, the infection probability is defined as

$$p(d) = 1 - \exp\left(-d/k\right), \tag{A1}$$

where k is a virus-dependent single-pathogen infection parameter. At present, the constant k for SARS-CoV-2 is not known. For SARS-CoV-1, $k \approx 400$ plaque-forming units (PFU) while a wider variety of viruses have $k \approx$ 10-3,000,000 PFU [24, 25], with the higher range applicable to several strains of influenza. For influenza and other RNA viruses, the particle-to-PFU ratio can be in the range of 10:1 to 100:1 [35]. 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 $p(d) \approx d/k$.

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

$$p(d) = 1 - (1 - 1/k)^d. \tag{A2}$$

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.

2. Mask Filtration Dilution Factor

The density of viral particles without dilution, $n_{\rm ND}$, is related to that with dilution n through $n = D_{\rm mask} \times D_{\rm hvac} \times n_{\rm ND}$. Here, $D_{\rm mask}$ is the mask attenuation to the inhaled dose and $D_{\rm hvac}$ 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 $D = D_{\rm mask} \times D_{\rm hvac}$. As described below, we determine a working value for $D_{\rm mask}$ for a single wearer to be 0.5 (0.1) for surgical (N95) masks. These estimates accommodate the possibility of imperfect mask use.

Surgical mask usage in the study by [26] 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 μm and $\sim$90% of particles >1 μm [36, 37] under normal breathing conditions. See Tab. II 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 [38]. 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 $\sim$2-5 and $\sim$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 [39, 40]. 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.

The efficiency of filtering out bio-aerosol particles by size for surgical and N95 masks are given in Tabs. III and IV. The average leakage of bio-aerosol particles of all sizes for a sealed surgical mask is $\sim$9% while that of a sealed N95 mask is $\sim$0.5% . In a more realistic scenario, assuming the mask is not perfectly fitted to the user, we see that a surgical mask leaks $\sim$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 Tab. III.

3. HVAC Dilution Factors

$D_{\rm hvac}$ parametrizes the dilution of the density of viral particles 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 $D_{\rm hvac}$ 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 $q = 3 \times 10^{-4}$ [18, 19, 22],

	No mask	Surgical mask	N95 mask
Normal breathing	$1.2^{*,\dagger}$, $3.3^{\dagger,\ddagger}$ [26]	$0.3^{*,\dagger}$, $0.3^{\dagger,\ddagger}$ [26]	$0.3^{\S}$ [41]
Coughing	$2.3^{\#}$ [38]	$1.8^{\#}$ [38]	$0.3^{\S}$ [41]

TABLE II. Viral load ($\log_{10}$ copies) produced by an infected person (S) as a function of parameters.
* For particles >5 μm, upper end of inter-quartile range. In $\log_{10}$ particles, 0.3 implies undetected, collected over a 30 min period
† Patients coughed an average 17 times during 30 min exhaled breath collection
‡ For particles ≤5 μm, upper end of inter-quartile range. In $\log_{10}$ particles, 0.3 implies undetected, collected over a 30 min period
§ For droplets and influenza. In $\log_{10}$ particles/mL, 0.3 implies undetected.
# For all particle sizes per cough, numbers averaged over 4 patients used in study. In $\log_{10}$ particles/mL.

Aerosol Diameter (μm)	0.2	0.5	1.0	2.0
Surgical mask (Flat, sealed) [36]	12-18	10-12	6-8	3-5
Surgical mask (Flat, 4 mm leak diameter) [36]	22-28	18-20	12-17	12-15
N95 mask (sealed) [39]	0.5-2.0	0.5-1.0		
N95 mask (2×3 mm diameter leak) [39]	8-10	8-10		
N95 mask (sealed) [40]			0.1-0.2	0.05-0.1
N95 mask (40% of the circumference unsealed) [40]			0.6-0.7	0.4-0.5
Surgical mask without filter material [42]	80-85	85-90	83-87	75-80

TABLE III. Penetration (towards inward leakage, TIL) of aerosol particles by size through various masks (in %). Data for flow rate of 30 L/min (32 L/min for reference [39]) with masks on mannequins. This flow rate represents a typical breathing flow rate for a human.

which we take to be the nominal penetration value for our risk analysis. After one air change time with entrained flow, $D_{\text{hvac}} = q$ (see App. A 4).

With or without filtration, n decreases exponentially as a function of time after an infected person leaves. Thus, D_{hvac} is time dependent and decreases in value (that is, improves) over time as air is circulated and exchanged or filtered. The density of remaining virus particles in the room depends on the air exchange rate of the room, r, and possibly on the HEPA filtration efficiency, f.

4. Air Flow Conditions

Air flow conditions strongly influence the distribution of viral particles [16, 44]. 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 $D_{\text{hvac}}(t) = e^{r \ln(q) t}$. Typically r = 15-20/h.

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

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 IV. Data from Ref. [43]. Penetration (towards inward leakage, TIL) of aerosol particles by size through various masks (in %). Data was taken on human subjects (25 subjects × 3 repetitions).

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 $D_{\rm hvac}(t) = e^{-rt}$, where r is the fresh air exchange rate. For a typical office, $r \approx 3$/h.

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 recommend 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 [45]). These conditions are generally not met in an office setting, so, generally, we recommend offices never be simultaneously occupied.

5. Auxiliary prep spaces and common areas

We also consider auxiliary prep spaces such as work spaces with fume hoods or electronics workshops 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 $T_{\rm spent} \sim 2$ 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 $T_{\rm wait}$. The viral load decreases from the steady state value of $n \sim 3 \times 10^{-2}$ virus particles/liter by a factor of $e^{-rT_{\rm wait}}$. In addition, H only gets exposed to a viral load of $d_0 = ne^{-rT_{\rm wait}}\left(1 - e^{-rT_{\rm spent}}\right)V'/r$ each time because the air continually circulates during H's occupancy. Here $V' = 450$ L/h is the breathing rate of H (see Sec. III A). 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 infection 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 subgroups, 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, $p \sim 4\%$.

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 provided they have comparable HVAC conditions.

Appendix B: Mortality rate and comparison to other risks

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% [46, 47]. The *infection* fatality rate was estimated to be 0.7% , where the lower value is due to more complete estimates on infection prevalence [47]. Researchers estimated the case fatality rate in the Gangelt municipality to be 0.37% [48]. Meanwhile, for the outbreak in Italy an infection fatality rate of 1.3% was determined [30]. 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 [46, 47, 49]. 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.

Parameter	Lab	Supermarket
Size (ft^3)	3,200	120,000
Filtration efficiency	99.97% HEPA	94% HVAC
Air exchange rate (min^{-1})	0.1	0.05

TABLE V. Comparing a laboratory with a typical supermarket

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 [50]. 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 [51]. 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 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 [52], giving a heavy radiation user an expected 0.005% likelihood of death due to exposure-related cancer in their lifetime. Risks for workers aged 20 to 30 are slightly higher because the lifetime risk of developing cancer due to radiation decreases for exposure at older ages. Using the age-grouped data from Ref. [53] we estimate a heavy radiation user 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 [29].

3. Comparison with other laboratory injury risks

We also estimate the rate of injuries not leading to deaths, which could 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" [54]. 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 [31]. Within this period, there were approximately 27,000 chemistry and chemical engineering graduate students in the United States at one time [32]. 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.

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 that can go up to 94% filtration efficiency.

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).

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

TABLE VI. 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.

Surface type	Aerosols	Copper	Stainless Steel	Plastic
Deactivation time (hours)	13	10	120	140

TABLE VII. Data from Ref. [8]. Natural SARS-CoV-2 viral deactivation time for different media. For aerosols (surfaces), deactivation time corresponds to a reduction of viral load by 4-$\log_{10}$ (6-$\log_{10}$). An N-$\log_{10}$ decontamination time, t_D, is related to the half life, $t_{1/2}$, by $t_D = Nt_{1/2}\left(\log_{10}(e)\ln(2)\right)$.

Assuming no hydrodynamic entrainment of aerosols through the HVAC system and using the parameters in Tab. V, 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 particles/liter. 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% .

Appendix C: SARS-CoV-2 properties

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 (∼70 copies/minute) for a variety of viruses, including coronaviruses, rhinoviruses, and influenza viruses [26]. This is very similar to independently measured values for influenza, ∼35 viral copies emitted per minute [27]. In addition, recent studies have shown that the levels of live virus shedding from the nasal cavities of asymptomatic patients can be high [7]. There is further evidence that vocalization, loudness of speech and speech "super-emitters" can lead to much higher rates of emission [10].

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 the Newton-Stokes law for this suspension time is [1]

$$\tau = 4.5\left(\frac{\eta h}{g\rho r^2}\right), \tag{C1}$$

where $\eta = 1.85\times10^{-5}$ kg/(m · s) is the viscosity of air at 25°C, h is the height of the particle, g is the acceleration due to gravity, $\rho = 1000\,\mathrm{kg/m^3}$ is the density of water, and r is the radius of the particle. If we take the height to be $h = 2$ m for an average individual, we get the following suspension times as a function of droplet size in Tab. VI. We conclude that for the HVAC conditions considered in this article, emitted particles of size <5 μm can be considered to remain in the air until removed by HEPA filtration or replaced with fresh air.

3. Lifespan of SARS-CoV-2 on aerosols/surfaces

Recent studies have quantified the duration that the SARS-CoV-2 virus remains viable in different media [8]. Their findings, translated to a normal viral deactivation time in accordance with FDA guidelines is given in Tab. VII. We focus 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 the entire room air exchange time for the rooms considered in this report. Thus for the purposes of this risk assessment, the limiting process will be air filtration. The effect of indirect exposure through surface contact is beyond the scope of this work.

The viral load is distributed among bio-aerosol particles of sizes ranging from 0.8 to 5.5 μm in the case of normal breathing [55] 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 [56].

Appendix D: Probability Formulas for Certain Examples

Here we list the formulas referred to in Sec. III A. A viral density n 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 $V' = 450$ liters/hr. With 40 hours of exposure per week, the total viral load inhaled by H is thus $1.8 \times 10^4\, n$ provided n is measured in particles/liter.

Hence the infection probability after 1 week (40 hours of exposure), assuming $k = 100$, is

$$p(n) = 1 - \exp\left[-\left(1.8 \times 10^4\right) n/k\right] = 1 - \exp\left(-180\, n\right). \tag{D1}$$

For small p,

$$n = -\left(6 \times 10^{-3}\right) \ln\left(1 - p\right) \approx \left(6 \times 10^{-3}\right) p \tag{D2}$$

$$\Longrightarrow p \approx 180\, n. \tag{D3}$$

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

$$p \approx 540\, n, \tag{D4}$$

and for 26 weeks (1,040 hours of exposure),

$$p \approx \left(5 \times 10^3\right) n. \tag{D5}$$

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