CORONAVIRUS

Face masks effectively limit the probability of SARS-CoV-2 transmission

Yafang Cheng1±†, Nan Ma2†, Christian Witt3, Steffen Rapp4, Philipp S. Wild4, Meinrat O. Andreae1,5,6, Ulrich Pöschl4, Hang Su7±*

Airborne transmission by droplets and aerosols is important for the spread of viruses. Face masks are a well-established preventive measure, but their effectiveness for mitigating severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) transmission is still under debate. We show that variations in mask efficacy can be explained by different regimes of virus abundance and are related to population-average infection probability and reproduction number. For SARS-CoV-2, the viral load of infectious individuals can vary by orders of magnitude. We find that most environments and contacts are under conditions of low virus abundance (virus-limited), where surgical masks are effective at preventing virus spread. More-advanced masks and other protective equipment are required in potentially virus-rich indoor environments, including medical centers and hospitals. Masks are particularly effective in combination with other preventive measures like ventilation and distancing.

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rirborne transmission is one of the main pathways for the transmission of respiratory viruses, including the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (1). Wearing face masks has been widely advocated to mitigate transmission. Masks are thought to protect people in two ways: (i) source control, reducing the emission and spread of respiratory viruses through airborne droplets and aerosols, and (ii) wearer protection, reducing the inhalation of airborne respiratory viruses.

The effectiveness of masks, however, is still under debate. Compared with N95 or FFP2 respirators, which have very low particle penetration rates (<5%), surgical and similar masks exhibit higher and more variable penetration rates (~30% to 70%) (2, 3). Given the large number of particles emitted upon respiration and especially upon sneezing or coughing (4), the number of respiratory particles that may penetrate masks is substantial, which is one of the main reasons for doubts about their efficacy in preventing infections. Moreover, randomized clinical trials have shown inconsistent or inconclusive results, with some studies reporting only a marginal benefit or no effect of mask use (5, 6). Thus, surgical and similar masks are often considered to be ineffective. On the other hand, observational data show that regions or facilities with a higher percentage of the population wearing masks have better control of COVID-19 (7–9). So how are we to explain these contrasting results and apparent inconsistencies?

In this work, we develop a quantitative model of airborne virus exposure that can explain these contrasting results and provide a basis for quantifying the efficacy of face masks. We show that mask efficacy strongly depends on airborne virus abundance. On the basis of direct measurements of SARS-CoV-2 in air samples and population-level infection probabilities, we find that the virus abundance in most environments is sufficiently low for masks to be effective in reducing airborne transmission.

When evaluating the effectiveness of masks, we want to understand and quantify their effect on the infection probability, \( P_{\text{inf}} \). Assuming that every inhaled single virus (virion) has the same chance to infect a person, \( P_{\text{inf}} \) can be calculated by a single-hit model of infection

\[
P_{\text{inf}} = 1 - (1 - P_{\text{single}})^{N_v}
\]

where \( P_{\text{single}} \) represents the infection probability for a single virus and \( N_v \) represents the total number of viruses to which the person is exposed (10). For airborne transmission, the infection probability \( P_{\text{inf}} \) for a given time period can be plotted as a function of inhaled virus number, \( N_v \).

Figure 1 illustrates the dependence of \( P_{\text{inf}} \) on \( N_v \), based on the single-hit model (Eq. 1) and scaled by the median infectious dose \( ID_{v,50} \), at which the probability of infection is 50% (10). It shows a highly nonlinear sensitivity of \( P_{\text{inf}} \) to changes in \( N_v \). Accordingly, the same percentage of change of \( N_v \) may lead to different changes in \( P_{\text{inf}} \) depending on the absolute level of \( N_v \). In a virus-rich regime, where \( N_v \) is much higher than \( ID_{v,50} \) (Fig. 1, A and B), \( P_{\text{inf}} \) is close to unity and is not sensitive to changes in \( N_v \). In this case, wearing a mask may not suffice to prevent infection. In a virus-limited regime, where \( N_v \) is close to or lower than \( ID_{v,50} \), however, \( P_{\text{inf}} \) strongly varies with \( N_v \), and reducing \( N_v \) by wearing a mask will lead to a substantial reduction in the infection probability (Fig. 1, C and D). Thus, we need to determine the regime of airborne virus abundance to understand mask efficacy.

Respiratory particles, including aerosol particles and larger droplets, can carry viruses and are often used to visualize the transmission of airborne viruses (4). Taking a representative average of respiratory activity (11), we find that a person typically emits a total number of \( \sim 3 \times 10^6 \) particles during a 30-min period (supplementary text, section S1.1). This very large number implies that indoor environments are usually in a respiratory particle–rich regime. Surgical masks with particle collection efficiencies of \( \sim 50\% \) cannot prevent the release of millions of particles per person and their inhalation by others (see green dots in Fig. 1, B and D). In other words, the human-emitted respiratory particle number is so high that we cannot avoid inhaling particles generated by another person, even when wearing a surgical mask. If every respiratory particle were to contain one or more viruses, indoor environments would often be in a virus-rich regime because the median infectious dose \( ID_{v,50} \) for respiratory diseases is typically on the order of a few tens to thousands of viruses (12–14).

But, does a respiratory particle–rich regime actually imply a respiratory virus–rich regime? To answer this question, we investigated characteristic virus distributions in both exhaled air samples and indoor air samples including coronaviruses (HCoV-NL63, -OC43, -229E, and -HKU1), influenza viruses (A and B), rhinoviruses, and SARS-CoV-2 (supplementary text, section S1). We find that usually just a minor fraction of exhaled respiratory particles contains viruses. In contrast to the high number of emitted respiratory particles, the number of viruses in 30-min samples of exhaled air \( (N_{v,30}) \) are typically low, with mean values of \( \sim 53 \) for coronaviruses (HCoV-NL63, -OC43, -229E, and -HKU1), \( \sim 38 \) for influenza viruses (A and B), and \( \sim 96 \) for rhinoviruses (12) (supplementary text, section S1.2, and Fig. 2). Figure 2, A and B, shows the infection probabilities obtained by inserting the number of exhaled viruses \( (N_{v,30}) \) for the number of potentially infected viruses \( (N_{v,30}) \), assuming a characteristic infectious dose of \( ID_{v,50} = 100 \) or 1000 viruses, respectively (12–14). For SARS-CoV-2 in various medical centers, we obtained mean values of \( N_{v,30} \) in the range of \( \sim 1 \) to \( \sim 600 \) (15–18) (supplementary text, section S1.3), which correspond to \( P_{\text{inf}} \) values in the range of \( \sim 0.1\% \) to 10% for \( ID_{v,50} = 1000 \) and \( \sim 1\% \) to 100% for \( ID_{v,50} = 100 \).
The wide range of $N_{v,30}/ID_{v,50}$ and $P_{\text{inf}}$ values demonstrate that both virus-limited and virus-rich conditions can occur in indoor environments.

The high variabilities of $N_{v,30}$ and $P_{\text{inf}}$ shown in Fig. 2, A and B, are consistent with the wide distribution of viral load observed in respiratory tract fluids (19) and need to be considered for estimating population-average infection probabilities, $P_{\text{inf,}\text{pop}}$ (supplementary text, section S4). For this purpose, we modeled $N_v$ for SARS-CoV-2 as lognormally distributed with standard deviations ($\sigma$) in the range of $\approx 1$ to 2 on the basis of recently reported distributions of the viral load of SARS-CoV-2 in respiratory fluids (19) (supplementary text, section S4). As shown in Fig. 2C, the population-average infection probabilities with $\sigma > 0$ are higher than in the case of uniform exposure ($\sigma = 0$) in the virus-limited regime at $P_{\text{inf,}\text{pop}} < \approx 50\%$. In other words, when the population-average infection probability is in the virus-limited regime with $P_{\text{inf,}\text{pop}} < 0.5$ (Fig. 2C), a broader distribution (larger $\sigma$) implies an increase in the fraction of transmission events under virus-rich conditions (e.g., superspreader events), which leads to a reduction of overall mask efficacy.

The basic reproduction number for COVID-19 ($R_0 \approx 2$ to 4) (20) can be related to a basic population-average infection probability, $P_{\text{inf,}\text{pop,}0}$, through $R_0 = P_{\text{inf,}\text{pop,}0} \times c \times d$ (21). With the average duration of infectiousness ($d \approx 10$ days) and average daily numbers of human contacts ($c \approx 10$ to 25 contacts per day) (22, 23), we obtain estimates in the range of $\approx 0.8\%$ to $\approx 4\%$ for $P_{\text{inf,}\text{pop,}0}$, as indicated by the shaded area in Fig. 2C. The low levels of $P_{\text{inf,}\text{pop,}0}$ indicate a widespread prevalence of virus-limited conditions.

Different regimes of abundance are relevant not only for the distinction of respiratory particles and viruses, but also for different types of viruses. For example, viruses with higher transmissibility—i.e., those with higher loads and rates of emission and exhalation, greater environmental persistence, or lower ID$_{v,50}$—may result in a virus-rich regime and lead to higher basic reproduction numbers, as observed for measles and other highly infectious diseases. Our analysis shows that the levels of $P_{\text{inf}}$ and $R_0$ can vary widely for different viruses. This means that aerosol transmission does not necessarily lead to a measles-like high $R_0$ and that relatively low values of $P_{\text{inf}}$ and $R_0$ do not rule out airborne transmission. On the basis of the scaling with ID$_{v,50}$, the curves shown in Figs. 1 to 3 can easily be applied to assess the efficacy of masks and other preventive measures against new and more-infectious mutants of SARS-CoV-2, such as B.1.1.7.

Figure 3 illustrates how the efficacies of surgical masks and N95 or FFP2 masks vary between virus-limited and virus-rich conditions when masks are worn only by infectious
Fig. 2. Infection probabilities and abundance regimes of SARS-CoV-2 and other respiratory viruses. (A and B) Individual infection probabilities \( P_{\text{inf}} \) plotted against inhaled virus number \( N_v \), scaled by characteristic median infectious doses of ID\(_{v,50} = 100 \) or 1000 viruses, respectively. The colored data points represent the mean number of viruses inhaled during a 30-min period in different medical centers in China, Singapore, and the US, according to measurement data of exhaled coronavirus, influenza virus, and rhinovirus numbers (blue circles) \( I_2 \) and of airborne SARS-CoV-2 number concentrations (red symbols) \( I_{25–48} \), respectively. The error bars represent one geometric standard deviation. (C) Population-average infection probability \( P_{\text{inf,pop}} \) curves assuming lognormal distributions of \( N_v \) with different standard deviations of \( \sigma = 0, 1, \) and 2, respectively. The \( x \)-axis represents the mean value of \( \log(N_v/\text{ID}_{v,50}) \). The shaded area indicates the level of basic population-average infection probability, \( P_{\text{inf,pop},0} \), for SARS-CoV-2, as calculated from the basic reproduction number for COVID-19 and estimated values of average duration of infectiousness and daily number of contacts.

persons (source control), only by susceptible persons (wearer protection), or by all persons (universal masking). In Fig. 3A, the population-average infection probability in the case of surgical mask use \( P_{\text{inf,pop,mask}} \) is plotted against the infection probability without masks \( P_{\text{inf,pop}} \). It shows that surgical masking achieves large reductions in infection probability when the maskless infection probability is low but increasingly smaller reductions when the maskless infection probability is high. Figure 3B shows the corresponding mask efficacy, i.e., the percentage reduction of infection probability \( [\Delta P_{\text{inf,pop}}/P_{\text{inf,pop}} = (P_{\text{inf,pop}} - P_{\text{inf,pop,mask}})/P_{\text{inf,pop}}] \). It decreases slowly with increasing \( P_{\text{inf,pop}} \) in the virus-limited regime, exhibits a steep decrease upon transition into the virus-rich regime as \( P_{\text{inf,pop}} \) approaches unity, and goes to zero at \( P_{\text{inf,pop}} = 1 \). Figure 3, C and D, shows equivalent plots for N95 or FFP2 masks.

Figure 3 illustrates that source control alone is more effective than wearer protection alone but that universal masking is the most effective. This is because masks are more effective in removing larger particles (Fig. 4), and freshly generated respiratory particles are usually largest at the source, shrinking upon evaporation in indoor air (20). Figure 3 accounts only for airborne transmission of viruses. When considering other forms of transmission, the relative importance of source control can be even higher (supplementary text, section S5) (20).

The nonlinear dependence of mask efficacy on infection risk differs from the assumption that the percentage change of infection probability as a result of mask use would be proportional to the percentage change of inhaled particle number (20). Under this assumption, wearing a mask would have the same effect on the transmission of a virus disease at any level of infection probability. Our analysis, however, shows that the efficacy of face masks depends strongly on the level of infection probability and virus abundance: Masks reduce the infection probability by as much as their filter efficiency for respiratory particles in the virus-limited regime but much less in the virus-rich regime (Fig. 3). Accordingly, experimental investigations may find low mask efficacies when they are performed under virus-rich conditions. Together with other influencing factors, like consistent and correct mask use (supplementary text, section S7.5), changes between virus-rich and virus-limited conditions may contribute to the divergent results reported from laboratory studies and randomized controlled trials in different environments (20) (supplementary text, section S8). Notably, the increasing effectiveness of mask use at low virus abundance implies synergistic effects of combining masks with other preventive measures that reduce the airborne-virus concentration, such as ventilation and social distancing. For example, ventilation can change an environment from virus-rich to virus-limited conditions, which may be particularly important for medical centers with relatively high SARS-CoV-2 abundances (Fig. 2 and supplementary text, section S6). On the other hand, not only the efficacy of face masks but also the efficacy of distancing may be reduced in virus-rich environments (supplementary text, section S6). The more measures that are used, the more effective each measure will be in containing the virus transmission. If the inhaled dose may also affect the severity of infections (14), as is currently being debated (24), masks may still be useful even if the reduced dose still leads to an infection.

Figure 4 shows the size distribution of respiratory particles emitted by different human activities (25–27). Aerosols are physically defined as airborne solid or liquid particles with
diameters smaller than 100 μm, which can remain suspended over extended periods of time. In medical studies, however, a threshold diameter of 5 μm has often been used to distinguish between a so-called aerosol mode and a so-called droplet mode. Our analysis of measurement data from exhaled and ambient air samples indicates that the so-called aerosol mode (<5 μm) contains more viruses than the so-called droplet mode (>5 μm) (11), although the latter comprises a larger volume of liquid emitted from the respiratory tract (tables S1 and S2). This may be explained by the following mechanisms: a higher viral load occurring in the lower respiratory tract where the smaller aerosol particles are generated (28) or an enrichment of organic surfactants and viruses upon the generation of smaller aerosol particles (29). Enrichment of viruses in the aerosol mode can enhance their transmission because smaller particles remain suspended for a longer time, which leads to stronger accumulation and dispersion in the air. This may cause higher airborne virus concentrations, inhaled virus numbers, and infection risks, especially in densely occupied rooms with poor ventilation and long periods of exposure. Moreover, small aerosol particles have a higher penetration rate and higher probability of reaching the lower respiratory tract (figs. S5 and S6).

Our analysis was focused on respiratory particles and droplets with diameters smaller than 100 μm (traditional physical definition of aerosols (30)). Because of rapid gravitational settling, respiratory droplets larger than 100 μm are removed from the air in seconds, but they may still reach the upper respiratory tract of persons in close contact and cause infections by carrying large numbers of viruses in their very large liquid volume. For example, a single 1-mm droplet may carry as many as ~50,000 viruses in the case of a viral load of 10^8 per milliliter of respiratory fluid, which is realistic and higher than the estimated infectious dose for SARS-CoV-2 (14). Such large droplets, however, are very efficiently (~100%) removed even by simple masks (Fig. 4 and supplementary text, section S3), which further emphasizes the importance and efficacy of face masks for preventing infections. Because of the strong size dependence, and to avoid ambiguities, we suggest that diameter range should be considered to a virus-rich regime in certain indoor environments, such as medical centers treating COVID-19 patients. In such environments, high-efficiency masks and additional protective measures like efficient ventilation should be used to keep the infection risk low. The nonlinear dependence of mask efficacy on airborne virus concentration—i.e., the higher mask efficacy at lower virus abundance—also highlights the importance of combining masks with other preventive measures. Effective
Fig. 4. Volume size distributions of respiratory particles emitted during different respiratory activities with and without masks. (A to D) Distributions for sneezing (A), coughing (B), speaking (C), and breathing (D). The open circles are measurement data obtained without masks, and the solid lines are bi- or trimodal fits to the measurement data (25–27) (supplementary text, section S1.1). The dashed and dotted lines were obtained by scaling with the filter efficiency curves of surgical masks and of N95 or FFP2 masks, respectively (supplementary text, section S3). The symbols \( v_p \) and \( d_p \) represent the volume concentration and diameter of respiratory particles, respectively, and \( dv/d\log D_p \) represents the volume distribution function (supplementary text, section S1.1).

ventilation and social distancing will reduce ambient virus concentrations and increase the effectiveness of face masks in containing the virus transmission. Moreover, high compliance and correct use of masks is important to ensure the effectiveness of universal masking in reducing the reproduction number for COVID-19 (supplementary text, section S7.3, and fig. S11) (20).

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SUPPLEMENTARY MATERIALS
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Figs. S1 to S4
References (32–60)
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