Analytic modeling and risk assessment of aerial transmission of SARS-CoV-2 virus through vaping expirations in shared micro-environments

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Abstract

Background. It is well known that airborne transmission of COVID-19 in indoor spaces occurs through various respiratory activities: breathing, vocalizing, coughing and sneezing. However, there is a complete lack of knowledge of its possible transmission through exhalations of e-cigarette aerosol (ECA), which is also a respiratory activity. E-cigarettes have become widely popular among smokers seeking a much safer way of nicotine consumption than smoking. Due to restrictive lockdown measures taken during the COVID-19 pandemic, many smokers and vapers (e-cigarette users) were confined to shared indoor spaces, making it necessary to assess the risk of SARS-CoV-2 virus aerial transmission through their exhalations. We summarize inferred knowledge of respiratory particles emission and transport through ECA, as well as a theoretical framework for explaining the visibility of exhaled ECA, which has safety implications and is absent in other respiratory activities (apart from smoking). We also summarize and briefly discuss the effects of new SARS-CoV-2 variants, vaccination rates and environmental factors that may influence the spread of COVID-19.

Methods. To estimate the risk of SARS-CoV-2 virus aerial transmission associated with vaping exhalations, we adapt a theoretical risk model that has been used to analyze the risks associated with other respiratory activities in shared indoor spaces. We consider home and restaurant scenarios, with natural and mechanical ventilation, with occupants wearing and not wearing face masks. We consider as “control case” or baseline risk scenario an indoor space (home and restaurant) where respiratory droplets and droplet nuclei are uniformly distributed and aerial contagion risk might originate exclusively from occupants exclusively rest breathing, assuming this to be the only (unavoidable) respiratory activity they all carry on.

Results. If an infected occupant uses an e-cigarette in a home or restaurant scenarios, bystanders not wearing face masks exposed to the resulting ECA expirations face a 1 % increase of risk of contagion with respect the control case. This relative added risk with respect to the control case becomes 5 – 17 % for high intensity vaping, 44 – 176 % and over 260 % for speaking for various periods or coughing (all without vaping). Infectious emissions are significantly modified by mechanical ventilation, face mask usage, vaccination and environmental factors, but given the lack of empiric evidence, we assume as a working hypothesis that all basic parameters of respiratory activities are equally (or roughly equally) affected by these factors. Hence, the relative risk percentages with respect to the control state should remain roughly the same under a wide range of varying conditions. By avoiding direct exposure to the visible exhaled vaping jet, wearers of commonly used face masks are well protected from respiratory droplets and droplet

41 nuclei directly emitted by mask-less vapers.

Conclusions. Compared to the control case of an already existing (unavoidable) risk from continuous breathing, vaping emissions in shared indoor spaces pose just a negligible additional risk of COVID-19 contagion. We consider that it is not necessary to take additional preventive measures beyond those already prescribed (1.5 meters separation and wearing face masks) in order to protect bystanders from this contagion.

42 **Keywords:** SARS-CoV-2 COVID-19, electronic cigarettes, aerosol visibility, risk modeling

43
44 **Highlights**

- 45 • We present the first study modeling of SARS-CoV-2 contagion risk through exposure to
46 exhaled e-cigarette aerosol
- 47 • Viral content of vaping emissions was inferred from data gathered for other respiratory
48 activities. Exclusive rest breathing was assumed as control state to evaluate relative risks
- 49 • Increase of relative risks are 1 % for low intensity, 5-17 % for infrequent high intensity
50 vaping, 44 %, 88 %, 132 %, 176 % and 259 % for speaking 6, 12, 24, 30 minutes and
51 coughing 30 times
- 52 • Relative risks remain stable with respect to mechanical ventilation, wearing face masks,
53 virus variants, vaccination rates and environmental factors
- 54 • Visibility of vaping exhalations allows bystanders to avoid contagion by direct exposure

55 **1. Introduction**

56 The most familiar respiratory activities, breathing, vocalizing, coughing, and sneezing, have
57 been shown to transmit COVID-19, however, there is no data or direct evidence of this trans-
58 mission through the aerosol exhaled by vapers (e-cigarette users). Nevertheless, it cannot be
59 ruled out that vaping (as a respiratory activity like smoking) can plausibly transmit COVID-19
60 through its environmental exhalations. The current COVID-19 pandemic is a very complex and
61 disruptive event involving many factors besides the necessary medical response. This has forced
62 governments to implement prevention and containment measures affecting millions of individu-
63 als, including vapers and smokers finding themselves confined (or forced to spend much time)
64 in shared indoor spaces. Thus, evaluating theoretically the risks involved in this possible conta-
65 gion route is crucial. As a result of the present study, we provide a self-consistent risk model of
66 COVID-19 contagion while using this respiratory route in shared indoor environments, consid-
67 ering factors such as visibility, ventilation, mask usage, SARS-CoV-2 variants, vaccination rates
68 and environmental factors. We undertake a risk assessment of COVID-19 transmission in shared
69 indoor spaces from exhaled aerosols in two settings: a home and a restaurant with natural and
70 mechanical ventilation.

71 The present paper is based on the adaptation to vaping exhalations in a stable environment of
72 the dose-response exponential risk model Watanabe et al. (2010); Sze To and Chao (2010) that

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has been considered mostly for the evaluation of outbreaks of COVID-19 transmission risk in shared indoor spaces, taking into consideration various respiratory activities and environmental parameters (see authoritative review with numerous references in Peng et al. (2021)). As explained in this review, a common set of assumptions in this type of modeling is to consider the “shared space airborne” scenario with individuals sharing air in the same room, all exposed to an infected person in an indoor space regarded as a “box” in which respiratory droplets and their nuclei (known as “aerosols”) have been uniformly distributed throughout the full indoor volume (we consider a home and a restaurant scenarios). In particular, among the risk evaluation studies reviewed in Peng et al. (2021) we adapted to vaping exhalations a simplified and modified version of the exponential dose-response reaction model developed by Buonanno et al. (2020a,b) (hereafter BMS), based on the notion of “*infective quanta*” (the virus concentration needed to infect 63 % of exposed individuals) constructed with data on SARS-CoV-2 infective parameters that was available by the middle of 2020.

Given the lack of previous studies of vaping as a respiratory activity and thus absence of empiric evidence of the type of respiratory particles (droplets or droplet nuclei numbers and diameters) potentially transmitted through exhaled ECA, we inferred this data in a previous paper (see Sussman et al. (2021b)), using cigarette smoking and mouth breathing as useful proxies for the respiratory mechanics and droplet emission that should occur, with transmission distances computed by modeling ECA as an intermittent turbulent jet evolving into a puff. These findings have been incorporated in our adaptation of the BMS risk model to compute the viral quanta emissions that should correspond to exhaled ECA to be able to compare them with those of vocalizing and coughing, all compared with respect to a baseline “control state” defined by the unavoidable respiratory particle transmission from exclusive continuous breathing. As we show in section 7, our approach is consistent with that of BMS, but the interpretation of outcomes is different.

Since wearing face masks has become a universally accepted preventive measure, we discuss the risk modifications involved for masked bystanders exposed to emissions from potentially infected unmasked individuals, as vapers must (at least) momentarily remove their face masks in order to vape. However, the complexity of COVID-19 as an evolving pandemic requires considering other important factors besides face mask wearing, such as SARS-CoV-2 variants and vaccination, as well as environmental factors (whose effects are a more indirect and involve a higher level of speculation). Evidently, as we argue in Section 4, all these factors may modify contagion risks. To address this complexity we assume (see Section 9) as a working hypothesis that all respiratory activities are equally affected by modifications from these factors (given the absence of empiric data on this issue). As a consequence, regardless of the modifications of absolute risks for every respiratory activity, the relative risks referred should remain stable and roughly unchanged with respect to the control state of exclusive breathing.

An important property that distinguishes ECA exhalations from other respiratory activities (save smoking) is visibility, which emerges from the optical properties of ECA droplets (light scattering Ruzer and Harley (2012)). Visibility has significant psychological and safety implications: it allows those surrounding potentially infectious vapers to instinctively place themselves away from the area of direct exposure clearly delineated by the visible exhaled cloud (something impossible to do with expirations from other respiratory activities, except smoking, see Section 3).

Vaping as a substitute of cigarette smoking is part of a tobacco harm reduction strategy that empower users and significantly decrease the harm caused by smoking. However, in an effort to combat the current COVID-19 pandemic, health authorities throughout the world have imple-

mented various containment measures, including curtailing social activities and home confinement recommendations that have forced millions of vapers to share indoor spaces with those who do not smoke or vape. Once under confinement smokers who switched to vaping perceive that their decision to switch to vaping can enhance harm reduction efforts in an important way with respect to those who continue smoking. First, environmental ECA exposes bystanders to a minuscule amount of hazardous compounds in comparison with ETS. Secondly, the time and spatial scope of exposure is very limited, as environmental ECA droplets rapidly evaporate into a fast dispersing gas phase, practically making ECA imperceptible in a time frame below one minute and 2 meters or less from the vaper. As a contrast, ETS exposes bystanders to a significantly larger toxicant charge in whole rooms and for extended periods: up to 40 minutes per cigarette (see comparison between environmental ECA and ETS in Liu et al. (2017); Zhao et al. (2017); Martuzevicius et al. (2019); Palmisani et al. (2019)). As a consequence, smokers who switch to vaping enhance the safety of those with whom they share an indoor space under confinement.

It is important to note that this article only discusses the risk of potential SARS-CoV-2 transmission by exhaled ECA, not with risks of COVID-19 contamination or illness of vapers due to possible effects on the respiratory system of vaping, or other possible health hazards caused by exposure to inhaled or exhaled vapors derived from the use of e-cigarettes to substitute tobacco smoking. As reference on these issues we recommend readers to consult the short review of the literature that we provide in Sussman et al. (2021a), as well as earlier work on the relative safety of e-cigarettes with respect to smoking: Farsalinos and Polosa (2014); RCP (2016); McNeill et al. (2018); NASEM (2018); Polosa et al. (2019).

As a further disclaimer, we will not address the risk of COVID-19 spreading through respiratory droplets transmitted by environmental tobacco smoke (ETS), although our risk assessment of low intensity vaping (practiced by 80 % to 90 % of vapers by puffing with mouth retention) substantially differs from ETS by the complete lack of side stream emissions (originating from the smoker from the burning/smoldering tip of the cigarette that does not enter the respiratory system). However, we emphasize that exposure to ETS indoors is more hazardous than exposure to exhaled ECA in every way except, possibly, for transmission of the SARS-CoV-2 virus.

The methodological structure of the paper is displayed graphically by the diagram of figure 1. In what follows we complement the information provided in this diagram. Theoretical background material is presented in three sections. Two of these sections concern properties of ECA relevant to COVID-19 transmission: section 2 provides a brief summary of results of Sussman et al. (2021b) on emission and transport of respiratory particles (droplets and nuclei), while section 3 examines the visibility of exhaled ECA, commenting on its safety implications in section 10. The third one is section 4, presenting a summary of various factors affecting COVID-19 evolution, mainly the new variants of the virus named with Greek letters, the immunity protection through various vaccines, but also environmental factors, such as air pollution and atmospheric stability that may influence the virus propagation. In Methods we present in section 5 a risk model of SARS-CoV-2 contagion in shared indoor spaces (home and restaurant scenarios, natural and mechanical ventilation) based on an adaptation and simplification of the model proposed by BMS (Buonanno et al. (2020a,b)). Results of the application of the above described risk model are presented together with an extensive discussion in section 6. A complete discussion of these results is given in four sections: a model validation by consistency with the results found by BMS in one scenario and on the Guangzhou restaurant in section 7, a full discussion is provided in section 8 on the effect of face mask wearing, since the risk model was developed without considering this preventive measure, though face masks are seldom worn in a home scenario and compliance with this measure is lax in restaurant scenarios. In section 9

we provide a brief discussion of the validity and robustness of our model in view of effects of SARS-CoV-2 variants and vaccination, as well as environmental factors, while a discussion of safety issues is given in section 10. We show that relative risks are stable under the modification of absolute risks by these factors. The limitations and our conclusion are presented in sections 11 and 12.

2. Background I: Emission and transport of respiratory particles by ECA

2.1. Respiratory particles emission

In order to evaluate infection risks from vaping expirations in shared indoor spaces we need empiric data on relevant parameters of these expirations: tidal expired volume and characteristics of exhaled ECA carrying respiratory droplets, droplet emissions in such expirations and distances along which these droplets should be transported by them. As we argued in Sussman et al. (2021b), the lack of this data requires its inference through theoretical modeling guided by phenomena (on which this data exists) that can serve as proxies for vaping exhalations. Using this inferred outcomes droplet propagation distances can be estimated through a model of a turbulent intermittent jet evolving into a puff.

In what follows we summarize the main results of the theoretical modeling undertaken in Sussman et al. (2021b):

- **'Mouth to Lung' (MTL) low intensity vaping and smoking** The outcomes displayed in Table SM(2) and Table 2 of Sussman et al. (2021b) suggest mean expired tidal volumes of $V_T = 700 - 900 \text{ cm}^3$ potentially carrying $N_p = 6 - 210$ respiratory droplets per exhalation (median $N_p = 39.9$, deviation 67.3), overwhelmingly in the submicron range (typically peaking at $d_p = 0.3 - 0.8 \mu\text{m}$) and droplet number densities well below $n_p = 1 \text{ cm}^{-3}$. As shown in Figure SM(2) of Sussman et al. (2021b), this is the style of vaping involving low powered devices practiced by 80-90 % of vapers in the main consumer markets (the US and the UK). However, the proxies we have used (cigarette smoking and mouth breathing) exhibit a wide individual variation in expired volumes, puffing parameters and droplet emission, all of which should occur also in vaping. Thus, the inferred data we have mentioned excludes the small minority of outlier individuals known as "super emitters" possibly emitting as much as $N_p \sim 1000$ respiratory droplets per exhalation in expired tidal volumes of up to 2 LT.
- **'Direct to Lung' (DTL) high intensity vaping.** It involves high powered tank devices that allow for a wider spectrum of deeper respiratory intensity than MTL vaping. It should involve a higher rate of droplet emission and expired volumes of 2-3 LT. Perhaps the closest analogue to infer its droplet emission rate among the studies listed in Table 2 of Sussman et al. (2021b) is breathing at fractional residual capacity in Almstrand et al. (2010) that reported emission rates of around 1000/LT. However, this style of vaping is practiced by a small minority of vapers (roughly 10-20 %), with its upper end being extreme vaping (the so called "cloud chasers") that is only practiced in competitions or exhibitions. Evidently, this type of extreme vaping cannot be sustained for long periods and is not representative even of even DTL vapers.

While the inferred droplet numbers in the upper end of high intensity DTL vaping can be comparable with low end numbers for vocalizing, the latter involves modes with larger mean diameters

Figure 1: **Methodological structure** . See further explanation in the final paragraph of the Introduction

because of distinct droplet generation processes Asadi et al. (2019); Morawska et al. (2009); Johnson et al. (2011).

2.2. Distance for direct exposure

The distance range that vaping exhalations can transport respiratory droplets provides the spatial scope of direct pathogen exposure through exhaled ECA, modeled in Sussman et al. (2021b) as an intermittent jet evolving as the exhalation ends into a turbulent puff. The scope of direct exposure is the displacement or penetration distance of the jet in the direction of the momentum of the jet at exhalation. The parameters characterizing the jet are expired tidal volume of air diluted ECA mentioned in section 2.1 and exhalation centerline velocities estimated in Sussman et al. (2021b) as $U_0 = 0.5-3$ m/s (MTL vaping) and $U_0 = 1, 5-5$ m/s (DTL vaping), which (assuming horizontal exhalation) yields a distance spread of 0.5-2.0 meters for the MTL vaping and over 2.0 meters for DTL. The maximal penetration goes beyond that afforded by the momentum thrust of the starting jet, with the puff further evolving at lesser speeds. Before the puff stage centerline velocities drop to about 0.2 m/s at different times and distances when fluid injection stops in all cases.

Given its short time duration and close distance scope of the momentum trusted starting jet, the analytic model analyzed in Sussman et al. (2021b) provides a reasonably good inference of the distance and direction that bystanders should keep to minimize the risk of direct exposure. As the jet evolves it mixes with surrounding air, with entrained air reaching about 40 % of the jet mass as exhalation (fluid injection) ends at the transition towards the puff regime Ghaem-Maghani (2006); Ghaem-Maghani and Johari (2010). Since at this point the jet velocities become comparable to typical velocities of ~ 10 cm/s (and up to 25 cm/s) of airflow currents in home environments, even in still air with natural ventilation Matthews et al. (1989); Berlanga et al. (2017), the puff can be easily destabilized by vortex motion generated through turbulent mixing from the large velocity fluctuations produced by the entrainment Wei and Li (2015); Vuorinen et al. (2020).

Once the puff becomes disrupted bystanders face indirect exposure to mostly droplet nuclei, as submicron respiratory droplets evaporate almost instantly as they are exhaled (see Nicas et al. (2005)). Turbulence and thermal buoyancy and stratification become important factors when the jet initial momentum decreases, more so when the vaper (the source) moves or walks Wang and Chow (2011). Mechanical ventilation (mixed or displaced) He et al. (2011); Gao and Niu (2007); Gao et al. (2008) producing a faster disruption and dispersion of the slow moving puff through their own turbulent, thermal stratification and droplet dispersion patterns. In general, the dispersed submicron droplets and droplet nuclei can remain buoyant for hours, with mixing ventilation tending to uniformly spread them, whereas directed ventilation tends to stratify them along different temperature layers. The detailed description of droplet dispersion after the puff disruption is a complicated process that requires computational techniques that are beyond the scope of this paper (see comprehensive analysis in Vuorinen et al. (2020)).

3. Background II: Visualization of the respiratory flow through ECA

As shown in Sussman et al. (2021b), respiratory droplets that would be carried by exhaled ECA should be overwhelmingly submicron (just as ECA droplets). As a consequence, ECA droplets and the much fewer respiratory droplets accompanying them, should follow the fluid flow of ECA approximately as molecular contaminants, thus acting like visual tracers of the expiratory flow Ai et al. (2020); Nazaroff (2004). This visibility is shared with smoking (see Gupta

et al. (2009, 2010); Ivanov (2019)), but is absent in other respiratory expirations (breathing, vocalizing, coughing, sneezing). It has important psychological and safety implications, since bystanders can instinctively detect (and avoid) the area of direct exposure. We provide here a brief discussion of optical properties of aerosols that allow for their visualization (see comprehensive explanation in Ruzer and Harley (2012); Hinds (1999); Kulkarni P and K (2011)).

Visualization and coloring of aerosols follow from the interaction of light with its particulate phase through absorption and scattering (refraction, reflection and diffraction), which depends on the particles' number density n_p , chemical nature and the ratio of their diameters to visible light wavelengths: $\alpha = \pi d_p / \lambda$ with $\lambda = 0.4 - 0.7 \mu\text{m}$. This interaction is described in relatively simple terms for ultra-fine particles ($d_p < 0.05 \mu\text{m}$ or $\alpha \ll 1$) by Rayleigh's molecular scattering theory and for large particles with $d_p > 100 \mu\text{m}$ in terms of geometric optics. Particles with diameters in the intermediate range correspond to Mie scattering theory, which becomes particularly complicated for d_p roughly comparable to λ or $\alpha \approx \pi$, as is the case for ECA and respiratory droplets. Since the latter are liquid droplets, they are in practice non-absorbing so that scattering is the dominant effect.

A simpler approach to aerosol optics follows from the notion of light extinction, the loss of intensity I from absorption and scattering in the direction of an incident parallel non-polarized light beam, with intensity I_0 and cross section distance L within an aerosol. This is described by the extinction coefficient σ_e through the Lambert-Beer (or Bouguer) law

$$I = I_0 e^{-\sigma_e L}, \quad \sigma_e = \frac{\pi}{4} Q_e n \bar{d}^2, \quad (1)$$

where n is the total particle number density, $\bar{d}$ is the mean particle diameter and, for non-absorbing particles, $Q_e \approx Q_{\text{scatt}}$ is the scattering extinction efficiency taken as constant (a valid approximation for a fixed λ and a very small diameter range around $\bar{d}$).

The fact that n in non-biological aerosols (like exhaled ECA) is much larger than in bioaerosols that are just "airborne" without ECA (as in "normal" respiratory activities) explains why exhaled ECA is visible while the bioaerosols are not. To illustrate this point quantitatively, consider the light extinction law (1) for an incident beam with wavelength $\lambda = 0.5 \mu\text{m}$ crossing an exhaled ECA jet (see figure 2 of Sussman et al. (2021b)). From the mean diameters obtained in Sussman et al. (2021b), we have $\bar{d} = \lambda = 0.5 \mu\text{m}$ and $\bar{d} = 0.4 \mu\text{m}$ for respiratory and ECA droplets (the latter just at exhalation before their rapid evaporation), while (from figure 16.2 of Hinds (1999)) we have $Q_e = 2, 3.5$ respectively for ECA and respiratory droplets, as their respective refractive indices are roughly those of water and VG: $m = 1.33, 1.5$. While L is the same (same fluid jet) and $\bar{d}$ and Q_e have comparable values for both types of droplets, the large difference in n makes a significant effect in light transmission through the beam that indicates the aerosol visibility in its specific direction if $I/I_0 < 1$. In numbers: we have $n_p \sim 10^7 \text{ cm}^{-3}$ and $L = 15 \text{ cm}$ for ECA just at exhalation, leading to impaired light transmission, $I/I_0 = 0.51$, while at 1 meter distance after significant dilution: $\bar{d} \sim 0.1, L = 25 \text{ cm}$ and $n_p \sim 10^5 \text{ cm}^{-3}$, we have almost full light transmission $I/I_0 = 0.99$. For respiratory droplets $n_p \sim 0.1 - 1 \text{ cm}^{-3}$, leading to practically full light transmission $I/I_0 \approx 1$ irrespective of L (in the appropriate ranges delineated by the exhaled jet).

Extinction through a parallel beam provides a limited description of light scattering in an aerosol, which occurs through each droplet in all directions. The rigorous description through Mie scattering theory is beyond the scope of this paper, but it is evident that droplet numbers play a significant role since total scattered intensity is basically the sum of scattered intensity from each droplet. The light extinction law (1) is valid under the assumption that photons in

all directions follow classical paths and their scattering complies with a Poisson distribution (a good approximation for sufficiently diluted aerosols). Under these assumptions the mean free path between scattering events is simply (see chapter 13 of Kulkarni P and K (2011)) $\ell = 1/\sigma_e = 1/(C_e n)$, where $C_e \propto \bar{d}^2$ is the extinction cross section per droplet. Evidently, light scattering in the scales of the ECA jet is negligible for bioaerosols with very low n , as the mean free path ℓ becomes extremely large (much larger than the bio-aerosol scale). For $\bar{d} = 0.5 \mu\text{m}$, $Q_e = 2$ and $n \sim 0.1 - 1 \text{ cm}^{-3}$ we have $\ell \rightarrow 10^8 - 10^9 \text{ cm} = 10^3 - 10^4 \text{ km}$, a huge value that explains the practical lack of scattering events in these bio-aerosols.

4. Background III: Viral variants, vaccination and environmental factors

4.1. SARS-CoV-2 variants and vaccines

As of January 19, 2022, more than 9.5 billion COVID-19 vaccine doses have been administered globally WHO (2022), and several evidence demonstrated vaccination safety and effectiveness in reducing COVID-19 burden: Olariu et al. (2021); Ferrara et al. (2022). However, the simultaneous emergence of SARS-CoV-2 variants is a major challenge to the current global vaccination campaign, mostly driven by their higher transmissibility and the possible reduction of neutralizing activity of vaccine-induced antibodies towards novel viral strains, Ferrara et al. (2022); Mistry et al. (2021), particularly the B.1.1.529 variant of concern: Omicron: Dolgin (2022). Preliminary data observed that COVID-19 vaccines maintain relative effectiveness against severe to moderate disease sustained by the circulating variants Mistry et al. (2021), but faster vaccination provision and booster immunization have proved to restore a significant level of protection and are helping control the pandemic.

The further emergence and spread of variants are an expected part of the evolution of SARS-CoV-2. Surveillance systems observed that some antigenically distinct SARS-CoV-2 strains – classified as Variants of Concern (VOC) – exhibit a substantial growth advantage over the ancestral SARS-CoV-2 strain (wild type) and other VOCs Hsu et al. (2022).

Modeling the full extent to which a VOC could infect individuals and/or evade existing vaccine- or infection-derived immunity is challenged by several factors, including computation approaches, changes in community mitigation strategies, vaccination implementation and policies, exposition to different SARS-CoV-2 variants across the previous pandemic waves: Saxena et al. (2021). However, epidemiological data allow at estimating proxy measures of infectiveness dependence of new variants, particularly those which had a major impact on COVID-19 burden, such as B.1.1.7 (Alpha), B.1.617.2 (Delta), and B.1.1.529 (Omicron) VOCs.

The Alpha variant was the first highly impacting VOC to be detected in the United Kingdom in December 2020, and reported to be 50% more contagious than wild type SARS-CoV-2 strain from Wuhan: Yang and Shaman (2021). Estimates of the increased transmissibility found Delta variant to be twice as contagious as the original SARS-CoV-2 strain and around 40 to 60% more transmissible than Alpha: SPIMO (2021). Omicron variant, firstly detected in South Africa in late 2021, was found to be up to 4 times as infectious as the Delta variant among vaccinated people, and has rapidly become the dominant variant in the vast majority of countries: Lyngse et al. (2021). More recent (though speculative) information on the Omicron variant can be found in Koslow (2022); Maxmen (2021).

4.2. Environmental factors

As a disease whose primary cause is overwhelmingly characterized by airborne viral transmission Domingo et al. (2020), there is an abundant literature on numerous factors acting as

secondary effects connected with the physical properties of air that might contribute to enhance or decrease this airborne transmission. In particular, it is well known from previous pandemics that variations of temperature and humidity do affect viral transmission, as low temperatures and humidity allow for respiratory droplets of larger size (and thus with possible higher viral load) to remain more time in the environment and travel longer distances before evaporating into smaller and solid nuclei: Nicas et al. (2005); Diao et al. (2021). Other factors that might affect contagion rates, originating in outdoors but with possible indoor influence, are air pollution and atmospheric dynamics. These topics have become research subjects of interest, though existing studies are still limited in scope and methodology (see comprehensive literature reviews in Bourdrel et al. (2021); Brunekreef et al. (2021)). As all authors recognize, research on environmental effects on viral transmission is still in its infancy, but there is a pressing need to undertake it for guiding and improving a multidisciplinary approach to enhance as much as possible institutional readiness in future pandemics: Coccia (2021c, 2020, 2022). We provide below a brief discussion of these topics.

4.2.1. *Outdoor vs indoor exposure.*

There is a widespread consensus that infected subjects in outdoor spaces represent a negligible proportion with respect to those infected indoors, a consensus based at least on data that includes VOC's before the emergence of the highly contagious omicron variant (see comprehensive review of studies in Dias and Tchepel (2018) and a more recent one during the pandemic in Bulfone et al. (2020) and critical comments in Contini and Costabile (2020)). There is a noticeable heterogeneity of risk estimates, but all reviewed studies based on actual data reviewed in the above cited review report low ratios of outdoor contagion risk, as low as 1/1000 with respect to indoor contagion. Although a large share of indoor PM originates outdoors (40-70 %, depending on the degree of insulation of indoor spaces: housing facilities, offices and business, see Chapter 6 of Ruzer and Harley (2012)), There is a complicated relation (mediated by anthropogenic factors) between the type and chemical composition of PM in outdoors and indoor spaces, including micro-environments Schweizer et al. (2007).

However, while the significant decrease in risk of outdoor exposure to the SARS-CoV-2 virus is accepted, all studies find it extremely difficult to overcome the the near impossibility to determine with minimal certainty if the reported contagions actually happened outdoors, as people (even those working outdoors) normally do spend a non-negligible portion of their time indoors Schweizer et al. (2007). Individuals are often unaware and subjective when asked when and where infections occurred. The prevailing uncertainty is further complicated by the fact that the probability of contagion might be much higher in some outdoor spaces with high occupancy, such as crowded restaurant terraces, outdoor markets, sports stadiums or political demonstrations, events in which it is practically impossible to obtain even inferential risk estimates. Nevertheless, as we argue in Section 9, our risk model is concerned with stable indoor scenarios and thus the complexity of outdoor conditions should bear little influence on the main results.

4.2.2. *Air pollution.*

A large body of literature with diverse content and quality has emerged trying to find links between levels of air pollution and the spread of COVID-19 at regional and even national level. Two comprehensive reviews of this literature up to November 2020 are found in Brunekreef et al. (2021); Walton (2021 [Online]). We describe below the mechanisms with which air pollution can affect COVID-19 transmission:

- 385 • SARS-CoV-2 virus transport through air pollution particulates PM_{10} , $PM_{2.5}$ or even hyper-
386 fine PM. This type of transmission remains very speculative and very likely an extremely
387 remote possibility Dias and Tchepel (2018); Brunekreef et al. (2021); Walton (2021 [On-
388 line]). Air pollution typically originates in open outdoor combustion sources: motor vehi-
389 cles and a wide variety of sources from industrial activity. It can also arise from natural
390 causes (dust and other crustal phenomena), all of which spreading to extremely large dis-
391 persion volumes. The same comments stated before on the complicated relation between
392 outdoor and indoor spaces and sources discussed extensively in Chapter 6 of Ruzer and
393 Harley (2012)) and Schweizer et al. (2007) apply to air pollution, implying a much weaker
394 effect of penetration of air outdoor pollution into indoor spaces (pending on the degree
395 of insulation) where contracting COVID-19 is easier and most frequent. Moreover, the
396 relative number densities make it extremely difficult to regard the transport of viable virus
397 through air pollution a realistic possibility. PM density numbers in polluted areas can be
398 up to 8 orders of magnitude larger than that of respiratory particles from all respiratory ac-
399 tivities Contini and Costabile (2020). Also, viable SARS-CoV-2 in aerosol form seems to
400 have a limited lifetime measures in hours Van Doremalen et al. (2020); Fears et al. (2020).
401 Therefore, even if we assume viral transport following an outdoor to indoor path to be
402 possible (an adventurous guess given the toxic potential of constituents of air pollution:
403 not only PM, but also its gaseous phase Contini and Costabile (2020)), the probability of
404 pollution PM loaded with viable virus entering an indoor space is negligible. Notice that
405 this type of indoor contagion originating outdoors is a very different situation (involving
406 much larger space volume) from that of transport within flats in buildings in a Hong Kong
407 housing complex during the first SARS pandemic McKinney et al. (2006).
- 408 • Biological mechanisms have been identified that account for a link between air pollution
409 (PM and gasses SO_2 , CO, NO_2 and O_3) and respiratory and/or cardiovascular disease
410 Lelieveld et al. (2015), which are related to comorbidities representing major risk factors
411 that increase susceptibility for contagion and evolution towards a more severe COVID-19
412 stage. Many highly polluted areas are characterized by higher frequency of personal con-
413 tacts, mostly business but also social in shared hospitality venues and often the permanent
414 residents belong to lower socioeconomic status (see Contini and Costabile (2020)). Even if
415 wearing face masks, the potential for non-COVID disease is high considering their limita-
416 tions discussed in Section 8. Most studies are of the ecological type that correlate short and
417 long term exposure to averaged air pollution measurements reported in jurisdictions rang-
418 ing from metropolitan areas to districts and provinces within specific countries and even
419 comparing results from countries. As examples of ecological type of studies we have:
420 major urban areas in China: Zheng et al. (2021), county level study covering the United
421 States: Wu et al. (2020), Italy: Conticini et al. (2020); Coccia (2021a); Fattorini and Regoli
422 (2020), Poland: Semczuk-Kaczmarek et al. (2021), 33 European countries: Lembo et al.
423 (2021) and OCDE countries: Barnett-Itzhaki and Levi (2021). Evidently, country wide
424 studies offer a very coarse and limited capacity for the control of confounders.

425 These ecological studies face (specially if they are short term) many uncertainties and major
426 difficulties to disentangle an independent effect from air pollution by controlling for the many
427 confounders due to the differences between jurisdictions in other factors, such as socioeconomic
428 development, mobility of population, climate that might favor thermal inversions, not to mention
429 the fact that many individuals with mild symptoms or asymptomatic are not registered in contact

430 tracings as COVID-19 contagions. All these factors make it is practically impossible to estimate
431 the residual confounding (see Riccò et al. (2020)).

432 Another complication arises from the fact that the constituents of air pollution exhibit signif-
433 icant time and space variation at municipal, regional and national level: Fatimah (2016); Hayes
434 et al. (2013); Crippa et al. (2013). In particular, in the context of COVID-19, inconsistent results
435 (mostly on the abundance of pollutant species allegedly contributing to infection rates) were ob-
436 served in a comparison of ecological studies from four countries: Canada, Italy, England and
437 the United States: Huang et al. (2021). Ecological studies might become more useful if
438 complemented and contrasted with an epidemiological approach based on individual registries of
439 COVID-19 cases, admissions to hospitals and mortality, though in many jurisdictions and coun-
440 tries such data is either absent or scarce or incomplete: Contini and Costabile (2020) (see also
441 extensive discussion of this approach in Walton (2021 [Online])). There is also a deeper theo-
442 retical critique: regression models (in the ecological approach) are not appropriate for epidemi-
443 ological research, instead studies must be based on testing predictive generalizations
444 against data. In other words: science should be complemented (not substituted) by regression
445 techniques: Cox Jr and Popken (2020).

446 4.2.3. *Atmospheric dynamics and stability.*

447 Another environmental factor that might be possibly related to the spread of COVID-19 is
448 atmospheric stability characterized by slow stationary wind patterns, as such conditions might
449 increase or decrease the propagation of airborne virus loaded respiratory particles, either by
450 changes in temperature or humidity and also in population mobility Hassan et al. (2021); Bha-
451 ganagar and Bhimreddy (2020). The fact that COVID-19 had such an alarming outbreak in
452 Northern Italy and Central Spain motivated research on the possible existence special meteoro-
453 logical conditions in these jurisdictions Sanchez-Lorenzo et al. (2021); Coccia (2021a,b). How-
454 ever, research of climatological effects on the spread of COVID-19 face similar methodological
455 problems as those looking at effects from air pollution: basically, the need to regard these effects
456 from a multidisciplinary point of view as summarized and discussed before.

457 5. Methods: Risk model of contagion

458 Having considered the inferred data obtained in Sussman et al. (2021b) on exhaled tidal
459 volumes, emission rates, type of respiratory droplets and exhalation distance spreads, as well as
460 the visibility of these exhalations, we need to evaluate exposure risks of bystanders sharing indoor
461 spaces with infected vapers within the “box” model of a given number of occupants sharing a
462 common indoor space as described in the review Peng et al. (2021). However, as opposed to the
463 approach and most examples contained in this review, we do not seek to describe the conditions
464 for infection outbreaks, but conditions describing a relatively stable environment that can be
465 modeled by taking into account exposure to SARS-CoV-2 viruses potentially carried by a total
466 mass of droplet emission already present in indoor spaces irrespective of whether the exposure
467 is direct or indirect. Still, by using the parameters characterizing the infection outbreak in the
468 famous Guangzhou restaurant we are able in section 7 to provide a good approximation to the
469 estimation of density of infective quanta and infection probability that was estimated by BMS
470 for this event.

471 However, given the visibility of vaping (and smoking) exhalations, it is safe to assume that
472 bystanders sharing indoor spaces with vapers will, extremely likely, avoid direct exposure to the

Figure 2: **Quanta emission rates.** The curves display ER_q (quanta/hour) as a function of viral load c_v (RNA copies/mL) for various expiratory activities: rest breathing (br), low and high intensity vaping (vp), speaking (bottom to top) 10, 20, 30, 40% of the hour (sp), coughing (cf) and speaking 100% of the time (sp100). Numerical values of ER_q for $c_v = 10^7$ RNA copies/mL (vertical line) are listed and discussed in the text. The ratios between these activities and rest breathing (taken as the case control scenario) is displayed in figure 5.

exhaled jet and thus will be mostly subjected to indirect exposure to (mostly) droplet nuclei dispersed by surrounding air currents once the jet has become a puff subjected to thermal buoyancy and turbulent mixing. It is also important for the consistent risk model that we require to examine viral exposure of a total mass of droplet emission for other expiratory activities (breathing, vocalizing, coughing, sneezing) under the same assumptions, though for these activities avoidance of direct exposure is not instinctive because the exhalations are not visible.

The data inferred in Sussman et al. (2021b) considered generic respiratory particles (droplets and droplet nuclei) without reference to a specific pathogen/disease and have not evaluated infection risks of exposed susceptible individuals. We undertake now this evaluation, referring specifically to the available information on the parameters of the SARS-CoV-2 virus, assuming as well that emitted respiratory droplets or droplet nuclei potentially carrying this virus have been dispersed uniformly throughout a given indoor micro-environment.

Besides visibility, another extremely important feature that fully characterizes exposure risks from vaping expirations is the significant shortening of exposure time because of their intermittent and episodic nature: an infectious vaper (symptomatic or not) would emit respiratory droplets only while vaping (120-200 daily exhalations Dautzenberg and Bricard (2015); Cahours and Prasad (2018)), whereas the same vaper will emit respiratory droplets continuously just by normal rest breathing (17,000–29,000 daily exhalations for 12-20 breaths per minute for healthy adults).

5.1. Infective quanta

To evaluate indirect exposure risks from vaping we simplify and adapt the analytic risk model of two articles by Buonanno, Morawska and Stabile (hereafter BMS) Buonanno et al. (2020a),

cited in the review Peng et al. (2021). BMS have examined the potential SARS-CoV-2 virus transmission in various indoor micro-environments (see also their previous paper Buonanno et al. (2020b) and further updated information in Peng et al. (2021)). BMS develop this model by means of Montecarlo simulations in which variability of droplet emission rates and exposure parameters is described by suitable probability distributions. Our approach is to assume median values for these variables (50 percentiles) of these distributions, similar to their approach in their previous paper Buonanno et al. (2020b). This is justified because our aim is to evaluate the risks from indoor COVID-19 transmission from vaping, speaking and coughing (all episodic or intermittent expirations) in comparison with what can be denoted as a “control case” scenario of risks in a space where the infectious vapor is only rest breathing (a continuous expiration). We are not aiming at providing a full comprehensive risk analysis for each respiratory activity separately under more realistic conditions (something that would justify a full separate study in itself). BMS consider the notion of an infective “quantum”: the dose of airborne respiratory droplet nuclei necessary to infect 63 % of exposed susceptible individuals. They introduce the “quantum emission rate” ER_q (emitted quanta per hour) for various respiratory expirations

$$ER_q = \frac{c_v}{c_{RNA} c_{PFU}} \times f_{br} V_T C_d, \quad (2)$$

where c_v is the viral load (RNA copies/mL) in the sputum of a SARS-CoV-2 infected person (symptomatic or not), c_{RNA} is the number of RNA copies per PFU (plaque forming unit) needed to generate infection and c_{PFU} is quanta-to-PFU conversion parameter, f_{br} is the number of breaths per hour and V_T the tidal exhaled volume, C_d is the droplet volume concentration (in mL/m³, hence $C_d V_T$ is the total volume of exhaled droplets in mL). BMS define the product “ $IR = V_T \times f_{br}$ ” as an “inhalation rate”, but it can also be used as an exhalation rate expressible in units m³/h.

For the infection parameters BMS consider values that have emerged from recent data: $c_v = 10^7$ RNA copies/mL (average in the range $10^3 - 10^{11}$), $c_{RNA} = 1.3 \times 10^2$ RNA copies/PFU and $c_{PFU} = 2.1 \times 10^2$ PFU/quanta. For the droplet volume concentration they take as reference an experimental value that incorporated dehydration effects in droplets associated with loud speech Stadnytskyi et al. (2020) (see Bake et al. (2019) for a review of this type of observational studies of droplet/nuclei emission as well as formation mechanism in the upper and lower respiratory tracts). Then, using experimental data from Morawska et al. (2009) to scale this reference to other respiratory expirations, leading to the following values (in mL/m³)

$$C_d = 2 \times 10^{-2} \text{ (loud speech), } 6 \times 10^{-3} \text{ (normal speech), } 2 \times 10^{-3} \text{ (rest breathing),} \quad (3)$$

5.2. Adaptation of quanta estimations to vaping

In order to fit vaping expirations into these values we need to make some assumptions on the involved parameters, besides considering the effects on exposure from the time duration of expiratory activities. In particular, we need to evaluate their mean quanta emission rate *only* in the times when they occur and compare with the rates of normal rest breathing (which takes place all the time). To simplify matters, we assume that c_v , c_i and $f_{(br)}$ are largely unaffected by the timing of these expiratory activities. We have then

- **Low intensity MTL Vaping.** A vaper breathes $N_{(tot)}$ times in (say) one hour and of these breaths $N_{(vp)}$ coincide with vaping expirations (puffs), the expression for ER_q in (2) must be

modified as

$$\text{ER}_{q(\text{vp})} = \frac{c_v f_{\text{br}}}{c_{\text{RNA}} + c_{\text{PFU}}} \left[\frac{N_{(\text{vp})}}{N_{(\text{tot})}} V_{T(\text{vp})} C_{d(\text{vp})} + \left(1 - \frac{N_{(\text{vp})}}{N_{(\text{tot})}} \right) V_{T(\text{br})} C_{d(\text{br})} \right], \quad (4)$$

where $N_{(\text{vp})}$, $N_{(\text{tot})}$ are the number of vaping puffs and total number of breaths per hour, $V_{T(\text{br})}$, $V_{T(\text{vp})}$ and $C_{d(\text{vp})}$, $C_{d(\text{br})}$ are the tidal volumes and droplet volume concentration for vaping and rest breathing. For low intensity MTL vaping we assume a tidal volume of $V_T = 750 \text{ cm}^3$ supported by data inferred and discussed in Sussman et al. (2021b), while for droplet volume concentration we assume $C_d = 3 \times 10^{-3} \text{ mL/m}^3$, a plausible value denoting emissions slightly above rest breathing but below normal speech in (3), fitting the ‘whispered counting’ data of Morawska et al. (2009). For the number of breaths we can take the average values of 160 daily puffs in a 16 hour journey Dautzenberg and Bricard (2015); Cahours and Prasad (2018) and breathing frequency of $f_{\text{br}} = 16/\text{min}$ (in the range 12-20), so that $N_{(\text{tot})} = 960 \text{ breaths/h}$ and $N_{(\text{vp})} = 10 \text{ breaths/h}$.

- **High intensity DTL vaping.** We assume $V_T = 2000 \text{ cm}^3$ as an average tidal volume. However, there is ambiguity in inferring a value for droplet volume concentration because of insufficient data on how much the larger tidal volume and deeper inhalation of DTL vaping can modify respiratory droplet numbers and diameters. As mentioned in section 2 of Sussman et al. (2021b), higher powered devices associated with DTL vaping tend to increase ECA droplet sizes and diameters Lechasseur et al. (2019); Floyd et al. (2018) but it is not certain if this applies to respiratory droplets. However, as mentioned in section 3.3.2 of Sussman et al. (2021b), speech involves droplet generating mechanisms that are distinct from those of breathing Asadi et al. (2019); Morawska et al. (2009); Johnson et al. (2011), resulting in higher rate of droplet emission even with a tidal volume only slightly larger than the breathing rest value of $400 - 600 \text{ cm}^3$ Bailey and Hoit (2002); Hoshiko (1965). Thus, we have two plausible options to account for a higher total volume of exhaled droplets $V_d = V_T C_d$: it may follow simply from a larger V_T with the same value $C_d = 3 \times 10^{-3} \text{ mL/m}^3$ of low intensity vaping, or we might assume the larger value of C_d for normal speech in (3). Instead of choosing one option, we will keep the continuous range of $C_d = 3 - 6 \times 10^{-3} \text{ mL/m}^3$. Regarding the number of breaths we can assume the same values as low intensity vaping: $N_{(\text{tot})} = 960 \text{ breaths/h}$ and $N_{(\text{vp})} = 10 \text{ breaths/h}$.
- **Normal speech.** The equation for ER_q in (2) needs to be modified in a similar way as (4), replacing the droplet volume concentration C_d with the value for normal speech in (3) and we take as tidal volume the value $V_T = 600 \text{ cm}^3$, roughly 10 % larger than the average rest value Bailey and Hoit (2002); Hoshiko (1965). To incorporate the timing we replace $N_{(\text{vp})}$ with a number count of breaths coinciding with a given percentage of an hour interval spent on continuously speaking in a given indoor environment. For 5, 10, 20, 30, 40 % of the hour (960 total breaths) we have $N_{(\text{sp})} = 48, 96, 192, 288, 384 \text{ breaths/h}$.
- **Coughing.** The emission data from coughing in Morawska et al. (2009) is comparable to that of ‘unmodulated vocalization’ (repeating the vowel “aahh”). Hence, we can use (4) with the value for droplet concentration volume of loud speaking in (3) as a proxy for coughing, while for coughing tidal volume we have $V_T = 1400 \text{ cm}^3$ Gupta et al. (2009). Assuming a cough every 2 and 3 minutes, $N_{(\text{vp})}$ is replaced by $N_{(\text{cf})} = 20, 30$.

Considering the plausible assumptions stated above, we display in figure ?? the logarithmic plots of quanta emission rate ER_q from an infectious individual as a function of viral load c_v , for rest

576 breathing, low and high intensity vaping, speaking for 10 %, 20 %, 30 % and 100 % of the time,
 577 as well as coughing every 2 and 3 minutes. The numerical values of ER_q in quanta per hour for
 578 $c_v = 10^7$ RNA copies/mL are

$$\begin{aligned} ER_{q(br)} &= 0.3416, & ER_{q(vpL)} &= 0.3562, & ER_{q(vpH)} &= 0.3727 - 0.4139, \\ ER_{q(sp10)} &= 0.5063, & ER_{q(sp20)} &= 0.6610, & ER_{q(sp30)} &= 0.8158, \\ ER_{q(sp40)} &= 0.9705, & ER_{q(cf)} &= 1.2637, & ER_{q(sp100)} &= 1.8216, \end{aligned} \quad (5)$$

579 where the symbols br, vpL, vpH, sp10, sp20, sp30, sp40, sp100 and cf respectively de-
 580 note breathing, vaping low and high intensity, speaking 10, 20, 30, 40, 100% of the hour and
 581 coughing 30 times. In particular, for low and high intensity vaping ER_q is very close to the
 582 control case of rest breathing (almost indistinguishable for low intensity vaping), while even
 583 speaking 10 % of the hour (6 minutes) yields a larger ER_q value than the upper end of high in-
 584 tensity vaping. Also, normal speech for a full hour (not uncommon) produces a higher quanta
 585 emission than coughing 30 times

586 5.3. Exponential dose-response risk model

587 In order to evaluate a time dependent risk for expiratory activities that incorporates quanta
 588 emission rates and indoor environment variables, BSM consider the “dose response exponen-
 589 tial model” given in terms of the the density of the quanta $n(t)$ in units quanta/m³ under the
 590 assumption that $n(0) = 0$ (no exposure at initial time $t = 0$)

$$R = 1 - \exp \left[-IR \int_0^T n(t) dt \right] = 1 - \exp \left[-\frac{IR [ER_q N T - n(T) V]}{IVVR V} \right], \quad (6)$$

$$n(t) = \frac{ER_q N}{IVVR V} [1 - \exp(-IVVR t)], \quad (7)$$

591 where V is the volume (m³) of the indoor micro-environment, N is the number of exposed suscep-
 592 tible individuals, IR is the inhalation rate (m³/h) of these individuals and $IVVR$ is the infectious
 593 virus removal rate, which which BMS take as the sum of three factors: $IVVR = AER + \kappa + \lambda_0$,
 594 where AER is the ventilation air exchange rate, κ is the particle deposition on surfaces and λ_0 is
 595 the virus inactivation (all of these quantities given as h^{-1}).

596 We evaluate in section 6 the risk R for vaping exhalations and other respiratory activities,
 597 aiming at the evaluation of their relative risk with respect to the control state of continuous
 598 breathing, assuming a home and restaurant scenarios with natural and mechanical ventilation.

599 6. Results: Risk evaluation

600 To evaluate the risk of exposure to respiratory droplets carried by vaping exhalations in indoor
 601 environments we have adapted in section 5 the “dose response exponential model” developed by
 602 Bounnano, Morawska and Stabile (BMS) Buonanno et al. (2020a)¹. Specifically, we evaluate
 603 equation (6) that defines the risk R (as a fraction < 1) for the value $IR = 0.96$ m³/h taken from the
 604 previous paper of BMS Buonanno et al. (2020b) and justified as a level of physical activity half
 605 way between standing and light activity. For the remaining parameters BSM assume the range

¹We do not assume face mask wearing in this subsection. Effects of face masks are discussed in section 8.

606 $\text{AER} = 0.2 - 0.5/h$ for natural ventilation and $\text{AER} = 9.6/h$ for a restaurant scenario with mixed
 607 ventilation. BMS compute the deposition rate by dividing typical gravitational settling velocity
 608 for supermicron particles (10^{-4} m/s) by the height of emission (1.5 m), leading to $\kappa = 0.24/h$,
 609 while for the viral inactivation they take the measured aerosolized SARS-CoV-2 virus mean life
 610 of 1.1 hours Van Doremalen et al. (2020) and even longer periods Fears et al. (2020), leading to
 611 $\lambda_0 = 0.63/h$. We consider the following home and restaurant indoor scenarios:

- 612 • Home scenario. We assume one infectious vaper and three exposed susceptible family
 613 members ($N = 3$). Total exposure time $T = 12$ h. Indoor volume 125 m^3 (small 50 m^2
 614 apartment with roof height of 2.5 m). For natural ventilation: $\text{AER} = 0.2/h$ we have $\text{IVVR} =$
 615 $1.07/h$.
- 616 • Restaurant, natural ventilation with open door. Thirty costumers ($N = 30$), total exposure
 617 time $T = 3$ h. Air exchange rate $\text{AER} = 0.5/h$, indoor volume 300 m^3 (100 m^2 area with
 618 roof height of 3 m), results in $\text{IVVR} = 1.37/h$
- 619 • Same restaurant endowed with mechanical ventilation: $\text{AER} = 9.6/h$ (taken from Bu-
 620 nanno et al. (2020b)), results in $\text{IVVR} = 10.47/h$

621 The infection risk R for home and restaurant scenarios is plotted in figures 3 and 4 as a function
 622 of time for breathing, low and high intensity vaping, various percentages of time spent speaking
 623 and coughing every 2 minutes, considering natural and mechanical ventilation. As expected
 624 from the quanta emission rates displayed in figure ??, the exposure time of different expirations
 625 is a crucial factor in computing R . As expected, the risk factor R increases with exposure time
 626 T , displaying an approximately growing linear dependence that keeps the same shape but is
 627 markedly decreased with mechanical ventilation in in each scenario: R decreases to one third
 628 (25 % to 8 %) after 12 hours exposure in a home environment with air exchange rate of 3/h and
 629 one fifth (25 % to 5 %) after 3 hours exposure in a restaurant environment with air exchange rate
 630 of 9.6/h. However, the key point is not the absolute values of $R(T)$ but its comparison for various
 631 respiratory activities and the control state of exclusive normal rest breathing. Figures 3 and 4
 632 reveal that exposure to vaping expiration (vaper doing 10 puffs per hour) poses an infection risk
 633 to bystanders that is very close to that from the control case scenario. In fact, for low intensity
 634 vaping the infection risk $R(T)$ is practically indistinguishable from the control case and even for
 635 high intensity vaping it is well below that from the same person speaking and coughing. Speaking
 636 only for 10 % of the time (6 minutes per hour) already yields a higher infection risk than high
 637 intensity vaping, while speaking 30 – 40 % of the hour yields up tp 4 times the infection risk,
 638 which is roughly the values plotted in figure 5.

639 A good inference of the risk from intermittent and episodic expiratory activities (vaping,
 640 speaking, coughing) relative to the control case scenario of exclusive rest breathing (a continuous
 641 expiration) is furnished by the ratio $R_{(A)}/R_{(\text{br})}$, where $A = \text{vp, sp, cf}$ (see (5)). Plotting this ratio
 642 from (6)–(7) for every expiratory activity yields near constant curves around the values of the
 643 quotients $\text{ER}_{q(A)}/\text{ER}_{q(\text{br})}$ (see numerical values in (5)). This is not surprising since ER_q is the only
 644 variable in R that characterizes the infectious person (the other variables characterize the indoor
 645 micro-environment and the exposed susceptible persons). Hence, given the same indoor micro-
 646 environment and same number of susceptible individuals, we consider risks relative to the control
 647 case scenario of rest breathing in terms of the ratio of quanta emission. Using (4) we have

$$\varepsilon = \frac{\text{ER}_{q(A)}}{\text{ER}_{q(\text{br})}} = 1 + \left(\frac{\mathcal{V}_{d(A)}}{\mathcal{V}_{d(\text{br})}} - 1 \right) \approx \frac{R_{(A)}}{R_{(\text{br})}}, \quad (8)$$

Figure 3: **Infection risk in a home environment.** The curves display R as a function of exposure time T from (6). The abbreviations br, vpl, vpH, sp10, sp20, sp30, sp40 and cf stand for rest breathing, vaping low intensity, vaping high intensity (upper end option), speaking for 10, 20, 30, 40, % of time and coughing. Notice the dramatic reduction of R achieved by mechanical ventilation (moderate air exchange rate of 3/h). Also: the curves for the risks from vaping (full range of intensities) are practically indistinguishable from that of the case control scenario of rest breathing (red circles). These curves show the same order of magnitude values as those of figure 3 of BMS for their scenarios A-1 and A-2

Figure 4: **Infection risk in a restaurant.** The same abbreviations as in figure ?? plus sp100 (speaking 100 % of the time, a possible outcome when spending 3 hours in a restaurant). As in figure 3, mechanical ventilation (air exchange rate 9.6/h) achieves a dramatic reduction of R and the curves for the risks from vaping are practically indistinguishable from the curve of the control case scenario of rest breathing (red circles). These curves are comparable on orders of magnitude to those of figure 3 of BMS for their scenario C in the time frame of 3 hours

Figure 5: **Added percentage risks of expiratory activities with respect to the control case scenario of rest breathing.** The percentage values with respect to the control case are: low intensity vaping 1.3 % (vpL), high intensity vaping 5.2-17.7 % (vpH), speaking 44% (sp10), 88% (sp20), 132% (sp30), 176% (sp40) for 10%, 20%, 30%, 40% of time, coughing 259% 30 times per hour (cf). These values were obtained from $(\varepsilon - 1) \times 100$ for ε defined for these expiratory activities by (8)–(10).

where $\mathcal{V}_{d(A)} = V_{T(A)} C_{d(A)}$ is the total exhaled droplet volume (in mL) for each expiratory activity “A”. Since $N_{(br)} = N_{(tot)}$, then for a heavy breathing activity in intense aerobic exercise ε might grow only because of the much larger tidal volume. However, for a truly intermittent expiration like vaping we have $N_{(vp)}/N_{(br)} \ll 1$ and thus $\varepsilon \approx 1$ holds even if we have $\mathcal{V}_{d(A)}/\mathcal{V}_{d(br)} \gg 1$ (large exhaled amount of droplets as with the large tidal volumes in extremely intense vaping). For the values of tidal volume and droplet volume concentration we have used the numerical values in (5), we have the following relative risks

$$\varepsilon = 1.25 \times \frac{N_{(vpL)}}{N_{(br)}} \quad (\text{low intensity vaping}), \quad \varepsilon = 5 - 11 \times \frac{N_{(vpH)}}{N_{(br)}} \quad (\text{high intensity vaping}), \quad (9)$$

$$\varepsilon = 3.6 \times \frac{N_{(sp)}}{N_{(br)}} \quad (\text{speaking}), \quad \varepsilon = 28 \times \frac{N_{(cf)}}{N_{(br)}} \quad (\text{coughing}), \quad (10)$$

which provides an intuitive indication of the added exposure risks relative to the control case from the different expiratory activities.

We display in figure 5 the numerical values of ε , as an added risk with respect to the control case for various expiratory activities with respect to the continuous presence of risk from rest breathing and under the assumptions we have used. These numbers clearly reflect the effects of the intermittence or duration time of each activity. Under normal vaping conditions (10-15 puffs per hour) the added risk of low intensity vaping respect to the control scenario of exclusive rest breathing is of the order of $\sim 1\%$ (since $\varepsilon - 1 \sim 10^{-2}$). For high intensity vaping it is $\sim 5 - 17\%$, given the ambiguity in the range of $\mathcal{V}_d = V_T C_d$, still it is of the order of $\varepsilon - 1 \sim 5 \times 10^{-2} - 10^{-1}$, also a low added risk since the low value of $N_{(vp)}/N_{(br)}$ compensates for the large exhaled tidal volume.

665 Notice that the added risk respect to the control case grows to $\sim 40\%$ just for talking for 10 %
 666 of the time and easily reaches 176 % if talking 40 % of the time. Coughing is also intermittent,
 667 possibly even more intermittent than vaping, but its large amount of exhaled droplets (large factor
 668 of 28 in (10)) can offset this effect. For speaking ε can be large even if normal speech involves
 669 a tidal volume close to rest breathing, but it also involves a much larger amount of time (larger
 670 number of breaths in typical conversation).

671 7. Discussion I: Comparison with results of BMS

672 Since we have adapted the risk model developed by BMS to be able to incorporate vaping
 673 exhalations in the potential indoor transmission of COVID-19, it is important to compare our re-
 674 sults with theirs with the parameters characteristic of their scenarios, bearing in mind as well that
 675 we are considering median values of the main parameters (roughly equivalent to 50th percentiles)
 676 instead of a statistical distribution to account for their variability. Comparison with BMS also
 677 serves a sort of validation of our approach, since the values ER_q in quanta per hour for $c_v = 10^7$
 678 RNA copies/mL in (5) cover the range of magnitudes as the 50 percentiles values reported for
 679 oral breathing and various forms of vocalizing in Table 2 and figure 1a of BMS, as well as the
 680 respiratory activities listed in their Table 1.

681 The first sign that we have obtained analogous risk factors R comes from comparing our
 682 figures 3 and 4 with figure 3 of BMS that also plots R vs time, but with the axes inverted: vertical
 683 and horizontal axis respectively corresponding time and R . Notice that the relation between R
 684 and time in the scenarios A-1, A-2 and C of BMS depicted in their figure 3, which are roughly
 685 comparable to our home and restaurant scenarios, show same orders of magnitude as in our
 686 figures 3 and 4.

687 We can also approximate the “scenario D” of BMS, defined as an infected subject singing or
 688 speaking loudly in an 800 m^3 room with healthy subjects listening at a sedentary activity level.
 689 Considering the value $ER_{q(\text{sp100})} = 1.8216$ for quanta emission per hour when speaking 100 % of
 690 the time, with $N = 10$ listeners, $IVVR = 1.37/\text{h}$ and $IR = 0.96\text{ m}^3/\text{h}$, we can produce the three
 691 graphs displayed in Figure 6 for the density of quanta $n(t)$, as well as the dose of quanta D_q and
 692 infection probability P_{inf} (as percentage), defined as

$$D_q(ER_q, T) = IR \int_0^T n(t)dt, \quad P_{\text{inf}} = 1 - \exp(-D_q). \quad (11)$$

693 These graphs provide a good approximation to the 50th percentile curves (thick curves) displayed
 694 in figure 1 of BMS. However, the interpretation is different: for BMS the spread of values in
 695 that figure correspond to percentile levels representing variation of parameter values in a normal
 696 statistical distribution associated with Montecarlo simulations, whereas in our case it corresponds
 697 to the median value (50-percentile) for various respiratory activities that would be carried on by
 698 the emitter: breathing, vaping (circles), talking 10 % (sp10), 40 % (sp40) and 100 % (sp100)
 699 of the time. Notice that (as expected) for the three quantities plotted the best approximation to
 700 the 50-percentile curve in the three panels of figure 1 of BMS is the emitter speaking 100 % of
 701 the time (sp100 thick curve). From the spread of the curves in our figure 6) we could argue
 702 that assuming mean values of infective parameters the spread of COVID-19 transmission in this
 703 scenario would have been greatly diminished if the infected person had spoken (or sung) for only
 704 10 % or 40 % of the time instead of 100 %. Notice also that the curve for vaping is practically
 705 indistinguishable with that of breathing.

Figure 6: **Scenario D of BMS.** The panels depict the density of quanta $n(ER_q)$, the dose of quanta $D_q(ER_q)$ and probability of infection P_{inf} (in %) from the data of scenario D described by Table 1 of BMS. The curves correspond from bottom to top to breathing, vaping (circles), speaking 10 %, 40 % and 100 % of the time (thick curve). The latter curve, as expected from this scenario, provides a good approximation to the 50-percentile curves of Figure 1 of BMS

706 We can also provide a reasonable approximation to the estimation made by BMS of the well
 707 known surge of infections in the Guangzhou restaurant in China. For this case we take the known
 708 data $N = 20$, $V = 97 \text{ m}^3$ and assume $IR = 0.96$, together with natural ventilation $IVVR = 0.67/\text{h}$
 709 (values taken from Peng et al. (2021)). As shown in the graphs of $n(t)$ and P_{inf} (in percentages)
 710 displayed in the two panels of Figure 7, the best match with the curves of figure 4a of BMS
 711 correspond to the emitter speaking 40 % of the time (sp40), which is a reasonable proportion
 712 of time for speaking during a meal. As in the previous example of scenario D of BMS, we can
 713 also argue that under the assumption of 50-percentile values of infective parameters the spread
 714 of COVID-19 would have been greatly reduced if the infected patrons had spoken only 10 % of
 715 the time (sp10 curve) or remained silent (even if he/she had vaped).

716 8. Discussion II: Effects of face mask wearing

717 In our risk evaluation in a home and restaurant scenarios in section 6 we did not assume face
 718 mask wearing by emitters and receivers of infective quanta. However, this fact has little real
 719 life relevance in our risk evaluation for the home scenario because face mask are rarely worn
 720 at home (even under confinement). Assuming that containment measures permit that bars and
 721 restaurants remain open, our risk evaluation also remains roughly valid for such venues (if vaping
 722 is allowed), even if vaping necessarily requires the vaper to remove the face mask (at least for
 723 the brief time lapse of intermittent puffs). In fact, eating and drinking in a restaurant scenario
 724 also require face mask removal, which in a convivial atmosphere (with conversations accompany
 725 eating and drinking) should involve quanta emissions by mask-free patrons whose duration is
 726 likely to exceed the strict time needed to eat and drink. However, it is still necessary to examine
 727 exposure risks in hypothetical indoor scenarios in which universal face mask wearing is strictly
 728 enforced. In particular, we need to emphasize the effects on those face masks that are usually
 729 worn at a community level: surgical masks and/or those made of cotton and other fabrics.

Figure 7: **Outbreak at the Guangzhou restaurant.** The panels depict density of quanta and probability of infection for the data of the Guangzhou restaurant. The curves correspond from bottom up to breathing, vaping (circles), speaking 10 %, 40 % and 100 % of the time (sp10, sp40, sp100). The thick curve (speaking 40 % of time) provides the best fit to the curves of Figure 4 of BMS.

Face masks, in their multiple designs and fiber characteristics (see review in Tcharkhtchi et al. (2020)), filter aerosol particles through various physical processes: gravitational sedimentation, inertial impaction, interception, diffusion and electrostatics, each of which govern and/or becomes dominant in specific ranges of particle sizes, airflow, leaks and environmental factors according to aerosol filtering theory (see Hinds (1999)). The key issue we need to assess is how much cotton and surgical masks protect (in terms of filtering efficiency) their wearers from *inward* emission when they are exposed to *outward* quanta emissions by potentially infectious individuals not wearing face masks (as vapers when vaping). It is especially useful to compare this protection with respect to the one they would get when exposed to emitters who are also masked (*i.e. reciprocal masking*).

Filtering efficiency is high in outward emission for N95 respirators (over 90 %) and slightly less so (74 %) in surgical masks in human emitters breathing, speaking and coughing Asadi et al. (2020), with decreasing diameters of filtered droplets, though in these experiments droplet counts excluded ultra-fine droplets below $0.3 \mu m$ and leaks were not evaluated. Similar results were obtained in laboratory conditions with a non-biological aerosol, though leaking decreased efficiency in surgical and cotton masks between one half and two thirds Drewnick et al. (2021).

Evidently, face masks also protect their wearers from inward emissions, as revealed by the following two laboratory experiments:

- In Sickbert-Bennett et al. (2020) two human subjects in different body postures, wearing well fit N95 respirators and surgical masks (tied with stripes or ear loops), were exposed to a non-biological polydisperse aerosol ($d_p = 0.02 - 3.00 \mu m$) released in a chamber at concentrations between $2000-5000 cm^{-3}$. Fitted filtration efficiency was above 95 % in all N95 respirators, 71.5 % for the surgical mask tied with stripes and 38.1 % for the one fit with ear lobes. Efficiency decreased for the latter to 21.2 % when the subjects turned their head left or right, showing the effects of leaks.
- In Ueki et al. (2020) a bio-aerosol generated by a nebulizer emitted at flow velocity of 2 m/s, simulating a mild cough airflow, was used to examine virus penetration (in terms of virus titer) between two mannequins separated at distances of 50 and 100 cm, in experiments wearing loose and fit N95 respirators, surgical and cotton masks. The filtering

efficiency was measured in terms of the detected percentage of virus titer in the receiving mannequin with respect to the titer in the emitting one. For 10^5 PFU (plaque forming units) when the receiver was wearing different face masks and the emitter was unmasked filtering efficiency was: 17 %, 47 %, 57 % and 79 % for the cotton mask, surgical mask, loose and fit N95 respirators respectively. When both mannequins wore a surgical mask the filtering efficiency significantly rose to 60 %, 71 %, 69 % and 92 %. Efficiency was about 10 % lower for 10^8 PFU. Virus titers decreased to 45 % and 31 % when mannequins (both unmasked) were placed at distances of 50 cm and 100 cm with respect to their values at 25 cm separation.

Both laboratory experiments describe, in spite of their idealization, the relatively low protection afforded by cotton and surgical masks to bystanders exposed to either unmasked emitters at close range and/or high droplet concentrations, flow and virus titer, which are precisely the conditions characteristic of direct exposure that would likely affect bystanders (wearing such masks) placed at the source (close to the mouth) and in the direction of the jet exhaled by an infectious vapor or by someone infectious breathing, talking or coughing without wearing a mask.

However, as opposed to masked bystanders exposed to mask-less emissions from other respiratory activities, we showed in section 3 that bystanders close to a vapor in shared indoor spaces can avoid instinctively the spatial zone of direct exposure that is clearly delineated by the visible emission jet exhaled by a vapor. These are evidently very different exposure conditions from those of the experiments described above, as bystanders wearing cotton or surgical masks located outside the exhaled jet would be subjected only to indirect exposure to a very low concentration of submicron droplet nuclei (the so-called “aerosols”) dispersing through air currents at ambient velocities that depend on the ventilation regime (roughly below 10-20 cm/s with natural ventilation Matthews et al. (1989); Berlanga et al. (2017)). In fact, while SARS-CoV-2 transmission through indirect exposure to these submicron droplets and droplet nuclei (what the WHO and most medical literature denotes as “aerosols”) is an undeniable fact, its scope and reach and its relation to occupancy and ventilation is still controversial NAS (2020); Jayaweera et al. (2020); Shiu et al. (2019); Sommerstein et al. (2020).

In particular, a much decreased airflow in indirect exposure implies a much decreased face velocity U_f , the air velocity at the mask surface obtained by dividing the air flow (LT/min) over the mask surface area. For the range of flow of rest breathing 10-25 LT/min, we have $U_f = 6 - 12$ cm/s (see Drewnick et al. (2021)), which is qualitatively analogous to characteristic velocities of indoor circulation currents with natural ventilation. Intuitively, under these low intensity flow conditions the masks capture more particles (droplets and nuclei) because the permanence time of the latter favors the capture mechanisms that are dominant for the particle diameter $d_p > 0.3 \mu\text{m}$: inertial impaction and interception. The dependence of the percentage of filtration efficiency E on U_f when these mechanisms are dominant is given by $E \propto [1 - \exp(-U_f^{-4/9})] \times 100$ for a broad range of filtering parameters (see equations 9.19 and 9.35 of Hinds (1999)), which for $d_p > 0.3 \mu\text{m}$ yields $E > 90 \%$ (see figures 9.9 and 9.10 of Hinds (1999)). The same arguments should apply to indirect exposure to drifting droplet nuclei from other respiratory activities that are sufficiently small to remain buoyant for long times.

As a consequence, universal wearing of cotton and surgical face masks offers a significantly higher level of protection against indirect exposure to small droplets and nuclei spread once respiratory droplets have evaporated. Evidently, it would be extremely complicated to adapt the relative risk model we have presented in section 5 to these conditions, since we would have to re-calculate in terms of the filtering efficiency of the face masks the quanta emission assigned to

805 the control state of breathing emitters, of the comparative emitters talking and coughing, as well
806 as the exposed receivers. However, incorporating this complexity might not be worthwhile after
807 all, given the fact that the added contribution of vaping to the overall respiratory droplet emission
808 from the control state of breathing remains vary small.

809 9. Discussion III: How robust is our relative risk model?

810 The results of our analysis, as listed in detail in section 6 and figures 3, 4 and 5, reveal
811 that vaping expirations (by being intermittent and with low emission rates close to breathing)
812 represent a minor relative risk increase of exposure to SARS-COV-2 transmission with respect to
813 the control state: low intensity vaping (practiced by 80-90 % of vapers) involves a 1 % increase
814 of the risk while high intensity vaping involves an increase of risk of 5-17 % (the uncertainty
815 follows from the lack of a precise inference on its droplet emission rate). As a comparison (see
816 figure 5) speaking 6-24 minutes per hour increase the risk by 44–176 % and coughing 2 times
817 per minute in an hour by 259 %.

818 How stable are these results under the enormously varied conditions that result from the com-
819 plexity of the COVID-19 global pandemic? As we argued in Sections 6, 7 and 8, exposure time,
820 face mask wearing and ventilation introduce significant modifications on the the (approximately
821 linear) dependence of infective absolute risk R exposure time T , though these environmental ef-
822 fects should produce the same modification to all respiratory activities (at the very least there is
823 no evidence to sustain a contrary argument), thus the comparative relative risks plotted in figure
824 5 should remain roughly constant. This is consistent with the fact that figures 3 and 4, which
825 display $R(T)$ for the various respiratory activities under natural and mechanical ventilation for
826 the same micro-environment conditions (under the parameters used by BMS), which show that
827 the slopes for vaping (low and high intensity) in all cases are practically indistinguishable with
828 the slope for the the breathing control state.

829 As argued extensively in Section 4, several important factors that modify contagion risks arise
830 as new information on the biological evolution of the SARS-CoV-2 virus emerges, leading to new
831 SARS-CoV-2 variants that are more infectious than the original strain, though this increase in risk
832 can be greatly mitigated by increasing vaccination rates at least in more developed countries.
833 Being an airborne transmitted disease mechanical ventilation is an important modifying factor
834 on the computation of computation of the risk of infection $R(T)$, but risk estimates might also
835 affect natural ventilation estimates through local variations of temperature and humidity, which
836 (pending on local climate) might also affect indoor spaces. Other factors that might modify
837 contagion risks are atmospheric conditions and air pollution. However, the primary and best
838 known and studied cause of contagion remains airborne transmission in indoor spaces, while
839 research on the effects from environmental factors originating outdoors remaining uncertain and
840 speculative (see discussion in Section 4). Nevertheless, it is necessary and important to assess
841 the stability and robustness of our relative model with respect to the all the above mentioned
842 elements of the complexity of COVID-19.

843 The key argument that sustains the robustness of the model is the hypothesis that all res-
844 piratory activities are equally affected by the different factors of COVID-19 complexity that we
845 mentioned above. In other words, there is no evidence that a more infectious variant will generate
846 on infected individuals more (or less) emission of respiratory droplets only for a given respiratory
847 activity (say, speaking) and not in another one. In more technical terms this can be appreciated
848 from equation (2) that defines the most basic parameter of our analysis, the quantum emission

rate ER_q (emitted quanta per hour). A more infectious SARS-CoV-2 variant should change only the infective parameters in this equation:

- c_v , the viral load (in RNA copies/mL) in the sputum of an infected person,
- c_{RNA} , the number of RNA copies per PFU needed to generate infection,
- c_{PFU} , the quanta-to-PFU conversion parameter,

but should not have any effect on the parameters specific to respiratory activities: f_{br} (number of breaths per hour) and V_T (tidal exhaled volume), nor on C_d , the droplet volume concentration (in mL/m³), or $C_d V_T$, the total volume of exhaled droplets in mL. We remark that there is an immense individual variation in these parameters, with some individuals being recognized as “super spreaders”, but there is no evidence that different SARS-CoV-2 variants have any influence on these parameters, which largely depend on physiological characteristics of individuals and of mechanisms for generating respiratory droplets.

A more infectious variant should imply either more virus loaded respiratory particles in a given emission or the same number of such particles but each carrying a heavier viral load (as seems to be the case with the Delta variant, see Section 4). Another possibility regarding the efficient transmissibility of the Omicron variant would be from its enhanced ability to infect evading SARS-CoV-2 immunity caused by either vaccination or past infection Koslow (2022). In all cases these possibilities imply modifications of the numerical values of the infective parameters, but not of the parameters of the respiratory activities. This in turn would modify the numerical evaluation of ER_q in equation (5) but in a way that is proportionate to the parameters of the respiratory activities. This would modify the absolute contagion risk in (6)-(7) that explicitly depends on the numerical values of ER_q for the different respiratory activities. However, these modification would not affect the relative risk defined with respect to the control case of exclusive rest breathing: $R_{(A)}/R_{(br)}$, where $A = vp, sp, cf$ defines the respiratory activity (see (5)), so that plotting (6)–(7) will yield near constant curves around the values of the quotients $ER_{q(A)}/ER_{q(br)}$. The near equivalence of the ratio of quanta emission to the ratio of contagion risk associated to the control state should yield the same values in equation (8) and thus we would obtain basically the same results as displayed in figure 5.

Whether our hypothesis of independence of infective parameters on the respiratory activities holds or not should be verified empirically. The effects from environmental factors, such as temperature, humidity, air pollution and atmospheric dynamics, should also be independent of the respiratory activities. As more information and data emerge from the new variants, it might be possible to design the appropriate experiments for testing this hypothesis. However, as long as there is no contrary evidence, we believe that this is a valid working hypothesis and that the results we have obtained should be robust with respect to the complexity that characterizes the COVID-19 pandemic.

10. IV: safety considerations

As mentioned in the introduction, available evidence suggests that aerial COVID-19 contagion has occurred through a wide spectrum of environmental conditions between direct exposure to “droplets” (droplets with $d_p > 5 \mu m$) and indirect exposure to “aerosols” (small droplets and droplet nuclei $d_p < 5 \mu m$) (see NAS (2020); Jayaweera et al. (2020); Shiu et al. (2019); Sommerstein et al. (2020) and further upgrade in Peng et al. (2021)). However, the distinction between

891 “droplets” and “aerosols” has practically no effect for the risk evaluation we have undertaken,
892 since bystanders easily avoid direct exposure by virtue of the visibility of vaping and our goal has
893 been the evaluation of relative risks of respiratory activities in comparison with a well defined
894 control state of pure breathing.

895 10.1. Safety measures

896 As shown in section 3, vaping expirations potentially carrying the SARS-CoV-2 virus are
897 visible (as opposed to other respiratory activities). Besides the evident psychological dimension
898 of this flow visualization, there are safety implications: vapers and those surrounding them have
899 a clear, instinctive and immediate delineation of the flow’s horizontal and vertical distance reach
900 and spreading direction along the exhaled jet. From the outcomes of the hydrodynamical anal-
901 ysis carried in Sussman et al. (2021b), we can recommend as a basic safety measure to avoid
902 direct exposure (irrespective of face mask wearing) by keeping a 2 meter distance away from the
903 vaper (when vaping) in the direction of the visible jet. However, notice that exhalation ranges
904 above 2 meters are unusual, as 80-90 % of vapers use low powered devices whose exhaled jets
905 reach well below 2 meters (and typically exhaling with a 30 degrees downward angle, see Suss-
906 man et al. (2021b)). In other directions away from the jet (even at close distance) the exposure
907 is indirect, but nevertheless as a safety measure it is prudent to maintain 2 meters of separation
908 in all directions from anyone vaping when not wearing a face mask. Notice that these recom-
909 mended safety measures coincide with the standard social separation recommendations adopted
910 worldwide Hsiang et al. (2020).

911 10.1.1. Face masks.

912 In computing exposure risks in section 6 we did not consider face mask wearing. This is
913 justified because face masks are not usually worn in a home scenario. Even in a restaurant or
914 bar scenario, patrons are likely to remain mask-less for extended periods because masks must
915 be removed for eating and drinking (as with vaping). As we discuss in section 8, face masks
916 of common usage (surgical and cotton) afford limited protection to bystanders wearing them
917 subjected to direct exposure to respiratory droplets from mask-free emitters. However, once
918 outside the direct exposure zone (visible and delineated for vaping), bystanders wearing common
919 usage face masks would be facing practically the same risks from indirect exposure as in spaces
920 in which no vaping took place.

921 10.1.2. Lockdown vs opening.

922 Risk assessments are essential to provide evidence based support for preventive and mitigat-
923 ing policies that have been proposed and enacted worldwide (see review Hsiang et al. (2020)).
924 These assessments are sensitive to the wide variety of rapidly changing pandemic conditions and
925 scenarios. High levels of severity characterized by frequent contagion rates can be addressed by
926 lockdowns contemplating different levels and stages of home confinement. Under these condi-
927 tions the risk assessment for the home scenario that we presented is particularly relevant, as a
928 large number of vapers and smokers become home bound for a range of large periods. As we
929 argue in Sussman et al. (2021a), the risk assessment undertaken in the present paper provides
930 valuable information for safety policies in this scenario: low intensity vaping only produces a
931 minuscule ($\sim 1\%$) extra contagion risk with respect to the control case scenario of continuous
932 breathing. Safety interventions should consider that abstention from vaping would not produce a
933 noticeable safety improvement, but could generate an undesired level of stress and anxiety under

934 long term confinement. High intensity vaping produces a higher increase of relative risk, but
935 still well below speaking and coughing. Notice that face masks are seldom worn in home bound
936 scenarios of family clusters.

937 10.1.3. SARS-CoV-2 variants and vaccination.

938 Evidently, safety considerations must vary following the biological evolution of the SARS-
939 CoV-2 virus, as well as on the response to it by means of vaccination process and other non-
940 pharmaceutical preventive and containment measures. More infectious new SARS-CoV-2 vari-
941 ants necessarily require improving safety considerations, as absolute risks increase for all respi-
942 ratory activities, including vaping, but this assessment depends on whether exposed individuals
943 or the vaper have been vaccinated. Safety considerations under these constantly varying condi-
944 tions can become complicated and strongly dependent on vaccination rates and face mask usage
945 which vary for different countries. However, our safety considerations that come from our main
946 result still holds: in comparison with vocalizing and coughing, vaping represents a small increase
947 of contagion risk in indoor spaces with respect to the control state of exclusive breathing.

948 11. Limitations

949 The COVID-19 pandemic is a global dynamical phenomenon of high complexity and multi-
950 ple regional variations, different responses and attitudes, whose study and understanding requires
951 a dedicated multidisciplinary approach. For these reasons it is practically impossible for any
952 risk model to be applicable everywhere and at all times, but this complexity does not invalidate
953 simplified or idealized risk models that cover only specific issues of the pandemic. While this
954 article has presented a consistent model of COVID-19 contagion risks in two indoor scenarios
955 for vaping, as a respiratory activity comparable to other such activities (breathing, vocalizing,
956 coughing), it is important to describe its main limitations.

957 *Lack of empiric data.* Given the lack of experimental and observational data on respiratory
958 droplets carried by exhaled ECA, we had to consider as basic input for the risk model the data
959 inferred in Sussman et al. (2021b) on the basis of theoretical speculation from the physical and
960 chemical properties of ECA and extrapolation from available data on other expiratory activities
961 (cigarette smoking and mouth breathing with a mouthpiece) that can serve as reasonable proxies
962 for vaping. Evidently, the present paper inherits another important limitations of Sussman et al.
963 (2021b): the oversimplification of vaping styles by classifying a complex usage pattern into two
964 categories, “low” and “high” intensity” vaping, which cannot capture the full range and scope of
965 individual vaping habits.

966 *Oversimplification of infective parameters and individual variability.* The rates of emitted
967 droplets inferred for vaping are rough average estimates gathered from outcomes reported in
968 breathing studies (see Table 2 of in Sussman et al. (2021b)), involving a wide variety of subjects,
969 including both healthy and individuals affected by respiratory conditions (not by SARS-CoV-2).
970 Also, we did not considered the small minority of outlier individuals who are super spreaders
971 emitting significantly larger numbers of droplets Asadi et al. (2019). Also, the data on infective
972 SARS-CoV-2 parameters gathered by BMS that we use in section 5 is also subjected to uncer-
973 tainties that they specifically recognize. In fact, numerous aspects associated with the spreading
974 and infection details of the SARS-CoV-2 virus remain uncertain and subject to large (often un-
975 explained) individual and environmental variability (a good summary of these uncertainties is
976 found in Klompas et al. (2020); Morawska and Milton (2020); Morawska and Cao (2020); NAS

(2020); Peng et al. (2021)). However, in order to be able to model a possible (previously unexplored) route of droplet transmission and possible infection, it is necessary and unavoidable to simplify this complexity and lack of data to obtain plausible order of magnitude estimates that can be verified once empiric evidence is available.

Oversimplification of the risk model. The adapted BMS risk model that we presented in section 5 is also simplified (see details in section 2.1.4 of BMS). While it fulfills our aim of providing a rough comparative estimation of relative risks with respect to the control case of continuous rest breathing, we do recognize its limitations: the risks are evaluated for a single vaper in highly idealized micro-environments, assuming constant infection parameters and inhalation rates (which BMS also assume), ignoring as well probability distributions of the quanta emission rates that convey individual variation on infection susceptibility and other parameters (which the model of BMS does incorporate). A more elaborate and complete approach should include a more robust methodology to quantify exposure risks to intermittent and sporadic sources, as for example in Nazaroff (2004); Ai et al. (2019) or a more complete description of environmental parameters as in Peng et al. (2021). This task is left for a future analysis.

SARS-CoV-2 variants and vaccination and environmental factors. In the computation of the quantum emission rate we used numerical values of infective parameters that were valid in 2020, before the massive vaccination effort and when there were fewer variants. However, these parameters are certainly varying in more infectious variants of the SARS-CoV-2 virus that have emerged. Incorporating the dynamical complexity of the COVID-19 pandemic would necessarily require evaluating absolute risks for each emerging SARS-CoV-2 variant, considering vaccination rates at each jurisdiction and a number of other factors and elements that we did not include in our analysis. However, as we argued in Section 9, we assumed as working hypothesis that this multifactorial dynamic evolution characterizing the pandemic should equally affect all respiratory activities. Therefore, while exposure and absolute risks associated to each respiratory activity will necessarily vary along a complicated pattern, the relative risks of respiratory activities with respect to a control state defined by exclusive rest breathing should remain relatively stable.

Environmental factors. We did not consider environmental factors, some of which can influence indoors transmission of the virus (temperature and humidity), while others (air pollution and atmospheric dynamics) some originate outdoors but might also affect indoor spaces. Air pollution affects global health conditions (in general) and thus it might play a role also on the spread and evolution of the pandemic. However, as we argued in Section 4, contagion risks in large open outdoor spaces are way below the indoor airborne transmission, the main cause of COVID-19 contagion. The influence of these environmental factors on COVID-19 contagion is constrained by the complexity of the wide variation of conditions that occur in air dynamics between outdoor and indoor spaces. Also, research on how and to what degree these factors influence the spread of COVID-19 is still in its early stages, and thus results from many studies are still uncertain and speculative, but further advancing this research might be very useful to promote environmental measures to handle future challenges to global health.

12. Conclusion

We have presented in this paper a risk analysis of COVID-19 contagion through direct and indirect exposure to the SARS-CoV-2 virus potentially carried by respiratory droplets and droplet nuclei that would be carried by ECA (e-cigarette aerosol) exhaled by vapers in shared indoor spaces (home and restaurant scenarios). This risk analysis is based on suitable adaptations of

the risk model presented by BMS (see Buonanno et al. (2020a,b)) that incorporates experimental data on SARS-COV-2 infective quanta: we consider vaping expirations characterized by the respiratory parameters inferred in Sussman et al. (2021b), we also considered the quantitative effects of exposure from the characteristic duration times of vaping and of other expiratory activities (breathing, vocalizing and coughing). In particular, given the fact that breathing is a continuous (and unavoidable) expiratory activity, we considered the rate of infective quanta of pure breathing (without vocalizing, coughing or vaping) as a “control state” that serves as reference to evaluate comparative risks for the rest of the inspiratory activities. To complement this risk analysis we also discussed the visibility of vaping expirations (Section 3), the usage of face masks in the indoor scenarios under consideration (Section 8), the possible effects in risk evaluation of new variants of the SARS-CoV-2 virus and vaccination rates, as well as environmental factors (Section 4).

Vaping expirations represent a minimal increase of risk with respect to continuous breathing in home and restaurant scenarios with natural and mechanical ventilation (1 % and 5–17 % for low and high intensity vaping). Visibility of vaping expirations is protective, as it allows avoidance of the high risk of direct exposure to droplets and droplet nuclei potentially carrying the SARS-CoV-2 virus. Those sharing indoor spaces with vapers do not require extra safety interventions besides those already recommended for the general population: wearing face masks and keeping a separation distance of 1.5-2 meters to avoid direct exposure. Setting aside harms from environmental tobacco smoke unrelated to COVID-19, these recommendations should also apply to sharing an indoor space with a smoker.

The rapidly varying complexity of the COVID-19 global pandemic makes it very challenging to elaborate a risk model without making simplifying assumptions that may allow for results that can be valid in a wide range of conditions. In this paper we considered as a working hypothesis the assumption that all modifying factors (virus variants, vaccination rates and environmental condition) act on equal form on all respiratory activities. This means that (for example) a new infectious variant like Omicron is bound to produce the same extra contagion risk for breathing, vocalizing, coughing and vaping. While this hypothesis needs to be tested, as far as we are aware, there is no contrary evidence that would show that a given modifying factor acts differently for different respiratory activities. By taking this hypothesis as valid, we can state that while absolute contagion risks for every individual respiratory activity are necessarily dependent on widely varying specific local conditions, the contagion risk of these activities with respect to the control state should remain relatively stable in all conditions. Notice that the rapid evolution of the pandemic places severe limitations on the validity of the results from all types of studies. Nevertheless, the idealized risk model that we have presented can be a useful first step to advance in our understanding of risks of contagion from airborne transmission of the SARS-CoV-2 virus and pandemics that might occur in the future.

List of figure captions

Figure 1, **Methodological structure** . See further explanation in the final paragraph of the Introduction

Figure 2, **Quanta emission rates**. The curves display ER_q (quanta/hour) as a function of viral load c_v (RNA copies/mL) for various expiratory activities: rest breathing (br), low and high intensity vaping (vp), speaking (bottom to top) 10, 20, 30, 40 % of the hour (sp), coughing (cf) and speaking 100 % of the time (sp100). Numerical values of ER_q for $c_v = 10^7$ RNA

copies/mL (vertical line) are listed and discussed in the text. The ratios between these activities and rest breathing (taken as the case control scenario) is displayed in figure 5.

Figure 3, Infection risk in a home environment. The curves display R as a function of exposure time T from (6). The abbreviations br, vpL, vpH, sp10, sp20, sp30, sp40 and cf stand for rest breathing, vaping low intensity, vaping high intensity (upper end option), speaking for 10, 20, 30, 40, % of time and coughing. Notice the dramatic reduction of R achieved by mechanical ventilation (moderate air exchange rate of 3/h). Also: the curves for the risks from vaping (full range of intensities) are practically indistinguishable from that of the case control scenario of rest breathing (red circles). These curves show the same order of magnitude values as those of figure 3 of BMS for their scenarios A-1 and A-2.

Figure 4, Infection risk in a restaurant. The same abbreviations as in figure ?? plus sp100 (speaking 100 % of the time, a possible outcome when spending 3 hours in a restaurant). As in figure 3, mechanical ventilation (air exchange rate 9.6/h) achieves a dramatic reduction of R and the curves for the risks from vaping are practically indistinguishable from the curve of the control case scenario of rest breathing (red circles). These curves are comparable on orders of magnitude to those of figure 3 of BMS for their scenario C in the time frame of 3 hours.

Figure 5, Added percentage risks of expiratory activities with respect to the control case scenario of rest breathing. The percentage values with respect to the control case are: low intensity vaping 1.3 % (vpL), high intensity vaping 5.2-17.7 % (vpH), speaking 44% (sp10), 88% (sp20), 132% (sp30), 176% (sp40) for 10%, 20%, 30%, 40% of time, coughing 259% 30 times per hour (cf). These values were obtained from $(\varepsilon - 1) \times 100$ for ε defined for these expiratory activities by (8)–(10).

Figure 6, Scenario D of BMS. The panels depict the density of quanta $n(ER_q)$, the dose of quanta $D_q(ER_q)$ and probability of infection P_{inf} (in %) from the data of scenario D described by Table 1 of BMS. The curves correspond from bottom to top to breathing, vaping (circles), speaking 10 %, 40 % and 100 % of the time (thick curve). The latter curve, as expected from this scenario, provides a good approximation to the 50-percentile curves of Figure 1 of BMS.

Figure 7, Outbreak at the Guangzhou restaurant. The panels depict density of quanta and probability of infection for the data of the Guangzhou restaurant. The curves correspond from bottom up to breathing, vaping (circles), speaking 10 %, 40 % and 100 % of the time (sp10, sp40, sp100). The thick curve (speaking 40 % of time) provides the best fit to the curves of Figure 4 of BMS.

List of abbreviations and symbols

Acronyms and abbreviations

ECA,	E-Cigarette Aerosol
COVID-19	Coronavirus disease 2019
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2

1105	BMS	Bounnano, Morawska & Stabile
1106	MTL	Mouth to Lung (vaping style)
1107	DTL	Direct to Lung (vaping style)
1108	RNA	Ribonucleic acid
1109	PFU	Plaque forming units
1110	br	Breathing
1111	vp	Vaping
1112	vpL	Vaping low intensity
1113	vpH	Vaping high intensity
1114	sp10	Speaking 10% of time
1115	sp20	Speaking 20% of time
1116	sp30	Speaking 30% of time
1117	sp40	Speaking 40% of time
1118	sp100	Speaking 100% of time
1119	cf	coughing
1120	<i>Units</i>	
1121	LT	Litters
1122	mL	mililiters
1123	m	meters
1124	cm	centimeters
1125	μm	micrometers
1126	s	seconds
1127	h	hour

1128 *Variables*

1129	V_T	Tidal volume
1130	N_p	Number of particles
1131	d_p	Diameter of particles
1132	n_p	Number of particles
1133	U_0	Exhalation velocity
1134	α	diameter to wavelength ratio
1135	L	Cross section distance within the aerosol
1136	λ	light wavelength
1137	I	Beam intensity
1138	I_0	Incident beam intensity
1139	σ_c	Extinction coefficient
1140	Q_c	Scattering extinction coefficient
1141	$\bar{d}$	Mean particle diameter
1142	ℓ	Mean free path between scattering events
1143	ER_q	Quanta emission rate
1144	c_v	Viral load (RNA copies per mL)
1145	c_{RNA}	Number of RNA copies per PFU
1146	c_{PFU}	Quanta to PFU conversion parameter
1147	f_{br}	Number of breaths per hour
1148	C_d	Droplet volume concentration (mL/m ³)
1149	IR	Inhalation rate
1150	$N_{(tot)}$	Total number of breaths per hour
1151	$N_{(vp)}$	Number of breaths/puffs per hour for vaping
1152	$N_{(sp)}$	Number of breaths per hour while speaking
1153	$\mathcal{V}_d$	Total volume of droplets
1154	R	Risk for expiratory activities
1155	T	Exposure time
1156	n	Density of quanta

1157	N	Number of exposed susceptible individuals
1158	V	Volume of indoor space
1159	IVVR	Infective virus removal rate
1160	AER	Air Ventilation exchange rate (per hour)
1161	κ	Particle deposition on surfaces (per hour)
1162	λ_0	virus inactivation (per hour)
1163	D_q	dose of quanta (quanta)
1164	P_{inf}	Probability of infection (percentage)
1165	U_f	Face velocity
1166	E	Efficiency of filtration

1167 **Declarations**

1168 Ethics approval and consent to participate

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1178 **Competing interests**

1179 RAS has no competing interests to declare.

1180
1181 EG is currently employed by Myriad Pharmaceuticals, an independent company that man-
1182 ufactures e-liquids and vaping devices in New Zealand. She also provides consultancy
1183 work on research and development, regulatory affairs support, and formulation to several
1184 independent vaping companies in the Pacific Region. In the past she has worked for several
1185 pharmaceutical companies, including GlaxoSmithKline and Genomma Lab. She is also a
1186 member of the standards committee of the VTANZ and UKVIA.

1187
1188 RP is full tenured professor of Internal Medicine at the University of Catania (Italy) and
1189 Medical Director of the Institute for Internal Medicine and Clinical Immunology at the

1190 same University. In relation to his recent work in the area of respiratory diseases, clinical
 1191 immunology, and tobacco control, RP has received has received lecture fees and re-
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 1198 ists Network (IRIS) initiative. He has also served as a consultant for Pfizer, Global Health
 1199 Alliance for treatment of tobacco dependence, CV Therapeutics, Boehringer Ingelheim,
 1200 Novartis, Duska Therapeutics, ECITA (Electronic Cigarette Industry Trade Association,
 1201 in the UK), Arbi Group Srl., and Health Diplomats. RP has served on the Medical and
 1202 Scientific Advisory Board of Cordex Pharma, Inc., CV Therapeutics, Duska Therapeutics
 1203 Inc, Pfizer, and PharmaCielo. RP is also founder of the Center for Tobacco prevention
 1204 and treatment (CPCT) at the University of Catania and of the Center of Excellence for the
 1205 acceleration of Harm Reduction (CoEHAR) at the same University, which has received
 1206 support from Foundation for a Smoke Free World to conduct 8 independent investigator-
 1207 initiated research projects on harm reduction. RP is also currently involved in the following
 1208 pro bono activities: scientific advisor for LIAF, Lega Italiana Anti Fumo (Italian acronym
 1209 for Italian Anti-Smoking League), the Consumer Advocates for Smoke-free Alternatives
 1210 (CASAA) and the International Network of Nicotine Consumers Organizations (INNCO);
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1220 Author contributions

1221 Conceptualization, EG and RAS and; methodology, RAS.; investigation, RAS, EG and
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1228 References

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1257 URL [https://www.europarl.europa.eu/RegData/etudes/STUD/2021/658216/IPOL_STU\(2021\)658216_EN.pdf](https://www.europarl.europa.eu/RegData/etudes/STUD/2021/658216/IPOL_STU(2021)658216_EN.pdf)

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