Complex Models and Simple Results in Airborne Disease Transmission

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SUMMARY
The SARS-CoV-2 pandemic energized all fields related to indoor air, including exposure modelling. The confluence of scientific disciplines that responded to the devastation of COVID-19 has created a need to be precise with language. Exposure is presented as the rate of dose accumulation via air, and dose as the product of the dosing rate and the dosing duration. From this model of dose as the environmental determinant of infection, complexities are acknowledged, and simplifications are sought. The analysis asserts that as models become more complex and accurate by inclusion of real variables and dynamics, their results often converge to simpler, narrower solution spaces. For larger sample sizes, uncertainty in dose was found to be lower than the uncertainties in exposure and in exposure duration when dose is their product. The present analysis examines some common assumptions, like complete mixing, and their criticisms toward providing what is hoped are useful perspectives.

KEYWORDS
CFD; COVID-19; complete mixing; air travel; Wells-Riley

1 INTRODUCTION

The recognition that SARS-CoV-2 has a significant airborne infection path has focused attention on contaminant transport models in the occupied built environment. The inaccuracy of complete mixing became a concern of medical doctors, public health authorities and virologists whereas it had previously only bothered ventilation engineers. The Wells-Riley model calculates infection probability from a normalized dose, termed quanta. This classical approach lost favour when freighted by complete mixing and the notion that complete mixing is never strictly true. However, in the paper on the 1974 measles outbreak in an elementary school, Riley differentiated the children's exposures into three subenvironments (Riley, Murphy, and Riley 1978). There is no limit on the number of divisions this model can use in capturing concentration variability. Miller's investigation of the Skagit County Chorale superspreading event used a type of Wells-Riley model. The authors took the wide spatial distribution of cases to justify a complete mixing assumption, which then allowed first order removal of infectious respiratory aerosol (by various processes) over time (Miller et al. 2021; Sze-To and Chao 2010; Bazant and Bush 2021). Their estimate of an emission rate of 970 quanta/hr analysed along with viral shedding rates from the literature led to good agreement with an infectious dose of 1 virion. The current work examines models from the simple to the complex to demonstrate that the inclusion of greater physical realism can lead to a relatively uniform airborne exposure field and that the resulting dose of infectious aerosol due to various durations of exposure can have a tighter statistical distribution than its factors. This perspective can balance the tendency of groups of diverse experts to generate notional complexities. Since understanding exposure, dose, and risk is pursued often for the purpose of controlling dose, the effect of modelling errors on dose and dose reduction is analysed.

2 METHODS

Definitions

Airborne *Concentration*, $C = C(x,y,z,t)$, can be mass of vapor, aerosol, droplets, or quanta per volume of contaminated air. Other than for quanta, the units of C are [M/L^3], with mass, M and length, L. Airborne *Exposure*, $E = E(C)$ is often presumed equal to airborne concentration in the breathing zone and given as a time-weighted average of C. A more careful consideration of particle dynamics and physiology motivates defining E as a function of concentration that depends on respiration rate ([L^3/T] with time, T) and other factors. The units of E are then [M/T], and E is a mass intake rate. *Dose*, D is a function, f, of exposure level and duration.

$$D = \int_0^T f(E,t)dt \tag{1}$$

If exposure is assumed to be constant w.r.t. time over the duration, T, or if the time-weighted average exposure is used, then

$$D = \int_0^T f(E,t)dt = \left\{\frac{1}{T}\int_0^T E(t)dt\right\} T = E\,T, \tag{2}$$

with unit [M]. One way to distinguish exposure and dose is to think of exposure as the *Dose Accumulation Rate*. Concentration [M/L^3], exposure [M/T], and dose [M] are related quantities, listed in increasing order of relevance to infection. *Probability of Infection* is a function, g, of dose or $P_I = g(D)$. *Risk* is often used to mean likelihood, but it might be better defined to consider both probability of infection and severity of infection outcomes.

Wells-Riley

This airborne infection model treats E as quanta intake rate, with one quanta of infectious particles being sufficient for infection (an infection probability of 63%). The quanta intake rate,

$$E = \frac{Iqp}{Q} \tag{3}$$

E is the rate of quanta intake, Iqp/Q, with p the pulmonary flow or respiration rate and Q the ventilation rate to the space.

Probability of not getting infected is modeled as a Poisson distribution, with the expected value of number of infections created by quanta dose λ set to zero.

$$P(X = x) = e^{-\lambda}\left(\frac{\lambda^x}{x!}\right) \tag{4}$$

No infection is X = 0.

$$P'(X = 0) = e^{-\lambda}\left(\frac{\lambda^0}{0!}\right) = e^{-\lambda} \tag{5}$$

For zero quanta, the probability of not getting infected is 100%.

$$P'(0) = e^{-0} = 1 \tag{6}$$

For one quanta, the probability of not getting infected is 37%.

$$P'(0) = e^{-1} = 0.37 \tag{7}$$

At a quanta dose λ, the probability of getting infected is the complement of the probability of not getting infected

$$P(X = 1) = 1 - P'(X = 0) \tag{8}$$

The analysis also used the Taylor expansion to write

$$P = 1 - e^{-\lambda} = 1 - \left[1 - \lambda + \frac{\lambda^2}{2!} - \frac{\lambda^3}{3!} + \cdots\right] \tag{9}$$

and note that, for small λ,

$$P \approx \lambda \tag{10}$$

Distribution of the product of exposure and duration

Dose as D = E T is treated as the product of two independent normal random variables toward understanding the level of uncertainty that can be expected in D, compared to that in E and in T.

$$[D] = [ET] \tag{11}$$

CFD Modelling of an Airport Boarding Bridge

A steady-state RANS k-ε simulation was performed of a notional passenger boarding bridge, with 18 occupants. This is the enclosed walkway connecting an airport gate area to an aircraft. A typical decision in modeling occupied environments is whether to address the thermal situation. Cool supply air and warm passengers as boundary conditions were compared to the isothermal case, based on exposure of 17 susceptibles to the passive scalar emission of one infector.

3 RESULTS and DISCUSSION

Efforts at infection control seek a small dose and a low probability of infection. Computing both sides of Equation 10 if, for example, $\lambda = 0.3$, gives $0.26 \approx 0.3$, which is a reasonable statement, and equation 10 gets more accurate as λ approaches zero. This shows that successful controls can focus on quanta dose because it is essentially the infection probability. The opposite situation of poorly controlled dose corresponds to values of λ that are not especially large. An infection probability of 90% results from a quanta of 2.3. Discerning exactly how much greater than this the true dose is does not really help from a control point of view.

The complete mixing assumption more specifically refers to uniform concentration or exposure, and thus it can undervalue exposures near sources. When Riley related the number of measles infections to the ventilation rate in the rooms and buses of an elementary school, the situation was probably not complete mixing but a uniform rate of dose accumulation within these compartments, as children's movements through the exposure field essentially sampled the spatial average.

Table 1 shows how this error propagates to infection probability. Note that the error in infection probability is always less than the error in exposure, but asymptotes to equal as dose goes to zero, i.e. when it no longer matters.

Table 1. Effect of Exposure Error on Infection Probability.

E_{CM}/E_{TRUE} (hr^{-1})	Duration (min)	$\lambda_{CM}/\lambda_{TRUE}$	P_{CM}/P_{TRUE} (%)
20/60	15	5/15	99.3/100.0
12/48	15	3/12	95.0/100.0
4/8	15	1/2	63.2/86.5
1/2	15	0.25/0.5	22.1/39.3
0.4/0.8	15	0.1/0.2	9.52/18.1
0.1/0.2	15	0.025/0.05	2.47/4.88

In constructing dose, exposure and duration are equally important. Variability in each is a concern. However, since infection probability (or ID50 and TCID50) is based on dose, variability in exposure and in duration cause trouble only to the extent they create variability in their product. Equation 11 shows the distribution [*D*] as the product distribution [*E T*]. Note that the distribution of a product differs from the product of distributions.

Consider the assumption that the mean, μ, standard deviation, σ, and number of observations, *N*, of [*E*] and [*T*] are equal, then the standard deviation of the dose (their product distribution) is

$$\sigma_D = \sigma_{ET} = \sqrt{({\sigma_E}^2 + {\mu_E}^2)({\sigma_T}^2 + {\mu_T}^2) - (\mu_E \mu_T)2} = \sqrt{(\sigma^2)(\sigma^2 + 2\mu^2)} \qquad (12)$$

The standard error of *D* is then

$$SE_D = \frac{\sigma_{ET}}{\sqrt{N_D}} = \frac{\sqrt{(\sigma^2)(\sigma^2+2\mu^2)}}{N} \qquad (13)$$

Where $N_D = N^2$. The number of observations that should be attributed to *D* requires explanation. Since we are interested in dose, we observe the product *E T* and there are N_D of these. Since we know *D* is constructed from pairs of *E* and *T*, we discern that there are *N* values of each of those. For E and T the standard error is

$$SE = \frac{\sigma}{\sqrt{N}} \qquad (14)$$

The question is, can SE_D be less than SE?

$$\frac{\sqrt{(\sigma^2)(\sigma^2+2\mu^2)}}{N} < \frac{\sigma}{\sqrt{N}} \qquad (15)$$

Rearranging Equation 15 yields that

$SE_D < SE$ when

$$N > \sigma^2 + 2\mu^2 \, , \qquad (16)$$

which shows we can know D with greater certainty than E or T. For example, a variance of 4 and a mean of 5 would require N > 54, and a variance of 1 and a mean of 2 would need an N of only 10. The dependence of N on the size of measured values shows that normalization and choice of units could play a role in these analyses.

CFD models are clearly more complex than well-mixed models and have levels of fidelity in time-dependence, turbulence, heat, and particle dynamics (Ai and Melikov 2018). In a boarding bridge—the pedestrian connector between gate area and aircraft cabin—variability in exposure from one infector decreased, as realistic mixing mechanisms were added.

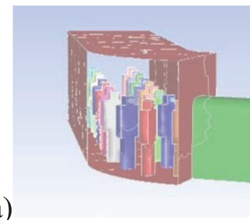
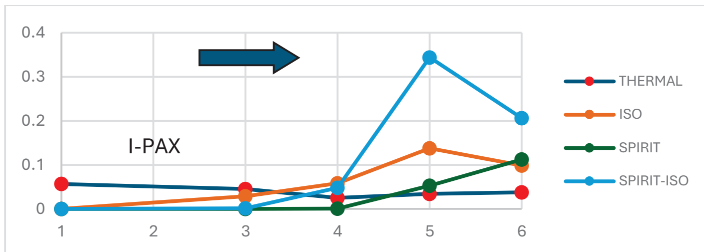
a)  b) 

Figure 1. a) geometry of a boarding bridge with 18 occupants as it curves into the cabin. b) Breathing zone exposure varied going from the entrance to the exit of the boarding bridge in a CFD model, with the infector (I-Pax) at position 2. Ventilation is directional, indicated by the blue arrow, from the gate to the cabin. Inclusion of body and thermal effects (Thermal case) resulted in uniform exposure, but without these effects (Spirit-Iso case) the exposure plume remained intact to decrease the breathing zone exposure nearby and increase it downstream.

4 CONCLUSIONS

Researchers, designers, regulators, and operators of occupied spaces heard conflicting guidance during the pandemic about Wells-Riley and complete mixing. Because mixing processes in fluid flows lead to greater homogeneity in fluid characteristics, a relatively uniform dose can be the end result of a realistic process. The sum of notional complexities asserted by experts created enough uncertainty to delay or reduce policy actions and to undermine public adherence. Perfect is the enemy of good, and complex models can reveal the wisdom of simple models.

Disclaimer

The findings and conclusions in this report are those of the author and do not necessarily represent the official position of the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention.

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