THE LANCET
Infectious Diseases

Supplementary appendix

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Supplement to: BaxA, Bax CE, Stadnytskyi V, Anfinrud P. SARS-CoV-2 transmission via speech-generated respiratory droplets. *Lancet Infect Dis* 2020; published online Sept 11. https://doi.org/10.1016/S1473-3099(20)30726-X.

Appendix

Below, we address the issues Abbas and Pittet[1] raised with respect to the various aspects of our work.[2,3]

"*First, the main assumption in the model is that dehydration is key to reducing the diameter of the expelled droplets, allowing droplets to become aerosols.*"

Dehydration of droplets is not an assumption but a scientific fact. Water is known to evaporate. The dehydration kinetics of small droplets and their transformation into aerosol particles is a well-studied and never-before questioned scientific observation.[2,3] Likewise, the fact that droplets shrink in diameter when their total mass decreases due to water evaporation is not an assumption.

"*The experiment was done in an environment with a relative humidity of 27%, which is below the minimum recommended indoor relative humidity of 40%*"

The relative humidity (RH) of 27% in which our measurements were conducted is 3 percentage points below the CDC guidelines for healthcare facilities (30%-60%) [4] and within the RH reported for common office buildings (20%-40%).[5] We assume that the authors were more concerned with the effect of RH on the rate of evaporation from respiratory droplets. Again, while the laws of physics dictate that higher RH increases the time needed for evaporation of water, and thereby decreases the time needed for a droplet's fall to ground, to first order this time scales with (1-RH) and has no significant impact for any of the small droplets observed in our work. For example, droplets of 20 micron diameter will fully dry out at 50% RH in *ca* 30 ms,[6] many orders of magnitude faster than the time needed to fall to ground if they were to remain fully hydrated.

"The duration of recorded speech was 25 s, but the results were artificially extrapolated to 1 min."

As clearly described in the Results section and Figure 1 of our PNAS article,[7] light scattering observations resulted from 25 seconds of recorded speech. As explicitly stated in the discussion, these results were used to **estimate** the number of potential virions emitted in one minute of speaking. There is nothing artificial about normalizing measured results to standard units. For example, we could have stated "an average of 17 virions per second over a period of 25 seconds" but such a number would suggest a precision higher than warranted, considering the wide variation in viral load and the fractional uncertainty in the diameter of the fully hydrated particles. Instead, our reported "1000 per minute" provides an order of magnitude estimate.

"*in the 2.33 min preceding the beginning of the speech, we counted at least 12 instances where flying particles were observed*"

Indeed, even when using a high-efficiency particulate air filter, infiltration of particles from outside sources contributed to our low background particle count rate. As indicated in Figure 1A of our PNAS article,[7] the decay of observed particles returns to this low background of 0.4 particles per frame (for the green curve; smallest particles). This was explicitly stated in the figure legend and was used when fitting the decay curves.

"*the authors used fluorescent green light to illuminate particles*"

Nowhere did we state or suggest that fluorescent light was involved in any of our measurements. The laser used in our study generated coherent green light with a wavelength of 532 nm. Laser light is not fluorescent. Our measurements recorded green light scattered from particles passing through the light sheet.

"*No report of the loudness, measured in decibels, was found in either manuscript, although in the videos it seems that in some cases the study participant was shouting, so the claim of normal speech is dubious.*"

Evidently, Abbas and Pittet failed to read the legend for Figure 1 in our PNAS paper[7] which reports that the speaker used "a loud (maximum 85 dB_B at a distance of 30 cm; average 59 dB_B)." Notably, the average loudness (59 dB) is consistent with the CDC's definition of conversational volume (60 dB).[8] The "shouting" we assume the authors refer to is when the speaker had the mouth covered by a washcloth,[9] which was used to illustrate that even when shouting, the number of detectable speech-generated droplets remains close to background levels when wearing a mouth cover.

"The authors were mistaken when stating that high viral loads were found in asymptomatic patients while referring to the study by Wolfel and colleagues. Only one patient reported being asymptomatic in the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) outbreak in Bavaria, Germany, and that patient was not included in Wolfel and colleagues' study, which included only hospitalised patients."

Evidently, Abbas and Pittet misread or misunderstood the results reported by Wölfel et al.[10] Wölfel et al. report the high viral loads of nine individuals. This patient population was a *subset* of the 16 COVID-19 cases reported at the end of January 2020 in Bavaria, Germany by Boehmer et al.[11] We thank Abbas and Pittet for referring us to Boehmer et al.,[11] whose report provides further evidence that presymptomatic spread occurs, with at least one patient, but probably five additional patients, being infected by a presymptomatic carrier. Moreover, it is important to note that Wölfel's patients were hospitalized not for the severity of symptoms, but preemptively on the basis of a positive COVID-19 test.[11] In fact, 4/9 patients (44%) never had a cough (Figure 2 a,d,f,i)[10] and Wölfel et al. state that "The clinical courses in the patients under study—all of whom were young- to middle-aged professionals without notable underlying disease—were mild".[10] Indeed, patient no. 16 did not exhibit symptoms during his hospitalization (Fig.2 and Table 2).[10]

"*the presence of a fan at the bottom of the black box during the speech and for 10 s after the end of speech does not represent real-life conditions*"

In "real-life" conditions, exhaled air emerges with high humidity at a temperature near 37 ºC and rapidly mixes with room air, as demonstrated by Schlieren images.[12] Speaking into an enclosure creates thermal and humidity gradients that are nominally eliminated by operating the internal fan for a short period of time during and after speaking. Using the fan to achieve a more homogeneous distribution of droplets **prior** to the actual decay time measurement does not influence the rate at which droplet nuclei subsequently disappear from view. The dispersion of speech droplets achieved by operating the fan for a short period of time is not unlike that produced by air currents generated when a person walks past a speaker. To suggest that use of the fan does not represent real-life conditions is as perplexing as it is irrelevant.

"In the abstract of one of the articles, it is stated that asymptomatic transmission is plausible, but its role has not been clearly elucidated and indeed is highly disputed".

We stated that transmission by asymptomatic COVID-19 carriers is plausible. There is now an abundance of evidence for this observation in the scientific literature,[13] as acknowledged by WHO on July 8, 2020.[14] While the nuances between presymptomatic and asymptomatic transmission, or even oligosymptomatic transmission, had not yet been extensively discussed within the context of transmission when our work[7] was submitted and published, the key point remains that disease carriers with no symptoms, that is, subjects who are by definition unlikely to be coughing or sneezing, may be transmitting the virus via speaking. The number of speech droplets observed in our studies, and in particular for the fraction that is sufficiently small to remain airborne for many minutes, is far higher than was previously considered by the medical community.[15] Multiple studies have shown that the oropharyngeal viral load in asymptomatic or presymptomatic patients is similar to that of symptomatic patients,[16,17] with infectivity appearing to peak prior to onset of symptoms.[10,18-20]

"Second, the authors assumed an average viral load in saliva of 7 × 10⁶ copies per mL on the basis of a prospective study wherein viral load was measured in sputum. Thus, they assume that viral load in sputum is the same as in saliva."

Wolfel et al. report throat viral loads as high as 7×10^8 copies per throat swab. Considering a throat swab to contain *ca 100-150* μL of oral fluid, the viral load was as high as ~7×10^9 copies per mL. Wolfel et al. also explicitly state "There were no discernible differences in viral loads or detection rates when comparing naso- and oropharyngeal swabs (Fig. 1b)". Sputum consists of "lower respiratory tract secretions along with nasopharyngeal and oropharyngeal secretions, cellular debris, and microorganisms".[21] As mentioned above, speech droplets originate from oral fluid, both at the vocal folds (mostly sputum) and at the front of the oral cavity (mostly saliva).[22] Oropharyngeal swabs represent an intermediate location. Vowel sounds have been associated with high levels of small speech droplets[23] and are minimally modulated by other narrow passages before entering the atmosphere. These droplets therefore are generated at the same physical location as cough droplets. It is important to note that the viral load of a disease carrier, that is, whether it is high or low, equally impacts the probability of disease transmission through the airborne, large-droplet, and fomite pathways. The **relative** probability of transmission through these pathways is primarily defined by the likelihood that secreted virions reach the respiratory tract of a bystander, not by the viral load of the droplets. Only if Abbas and Pittet wish to argue that the fecal route dominates disease transmission does the **absolute** viral load of respiratory fluid secretions become relevant.

"The group also assume that every RNA copy detected is a potentially infectious virion"

Nowhere did we state or assume that every RNA copy detected is a potentially infectious virion. Viability of excreted virions will modulate all pathways equally. Indeed, as highlighted in Figure 1f and 1g of Wolfel et al.,[10] the ability to culture virus from respiratory secretions rapidly decreases after onset of symptoms whereas viral loads decrease substantially slower, indicating that a progressively smaller fraction of virions is viable in culture as the infection progresses. However, the viability of virions modulates all transmission pathways equally.

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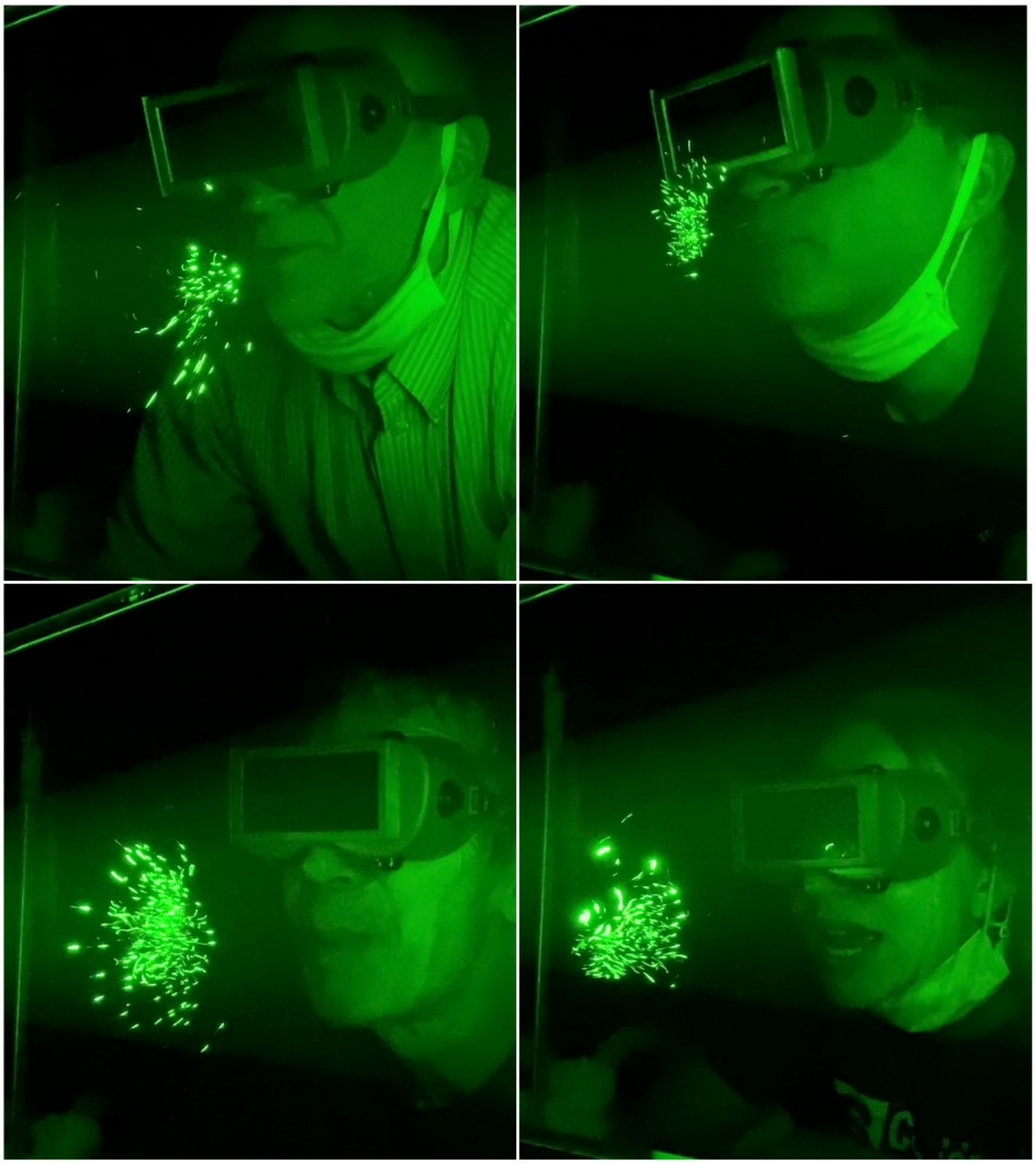

Figure 1. Speech droplets emitted by four persons when speaking the phrase “Spit happens” with the face positioned about 10-15 cm behind a thin sheet of intense green laser light. Flashes correspond to speech droplets crossing the light sheet. Individual frames shown here were extracted from a video recorded at 24 frames per second, available at https://doi.org/10.5281/zenodo.3935894