



## Impulse Dispersion of Aerosols during Singing and Speaking: A Potential COVID-19 Transmission Pathway

To the Editor:

Group singing events have been associated with several outbreaks of infection during the coronavirus disease (COVID-19) pandemic (1). This link supports the possibility that aerosols are partly responsible for person-to-person infection. This study aims to analyze the impulse dispersion dynamics of aerosols in professional singers concerning the differences between singing a text, singing a vowel, or speaking at different levels of loudness.

Some of the results of these studies have been previously reported in the form of a preprint (<https://doi.org/10.1101/2020.07.21.20158832>).

### Methods

After ethical approval (LMU-20-395), 10 healthy (by medical history, acute infection questionnaire, singing voice handicap index, and spirometry) professional singers from the Bavarian Radio Choir (five female and five male; mean [SD] age,  $44 \pm 11$  yr) were asked to perform the melody from Beethoven's "Ode to Joy" to the original text "Freude schöner Götterfunken, Tochter aus Elysium" in the key of D major, starting on F#3 for the male voices and F#4 for female voices (task "melody and text" [MT]). Moreover, the singers were asked to read out the text (T) at a comfortable pitch and to vocalize only the melody (M) without text on the vowel [ə]. All three tasks were performed with soft (−) and loud (+) phonation. Thus, the following six tasks were performed: MT+, MT−, M+, M−, T+, and T−. In addition, a 6-second exhalation and a coughing task were performed.

Preceding the phonation of all tasks, the subjects were asked to inhale 0.5 L of the fume of an e-cigarette, filled only with the basic liquid (50%:50% glycerin:propylene glycol, Lyneden Vox e-cigarette; Lynden GmbH, controlled by a ZAN 100 spirometer; Oberthulba). According to Ingebrethsen and colleagues (2), the particles generated in e-cigarettes have a diameter in the range of aerosols at 250–450 nm.

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Three full HD Sony HDC 1700R cameras recorded the experiment from side (camera 1) and top view (camera 2) perspectives. All measurements were performed in a Bavarian Broadcasting television network studio (dimensions, 27 m × 22 m × 9 m). The walls were at least 4 m away and covered in black. The smoke was illuminated with three spotlights positioned in a distance of at least 3.5 m. Before each task, the studio was aired out. After aeration, all people present were instructed not to move for another 2 minutes. The temperature was measured at mean (SD) 23.27°C (0.46) and the relative humidity at 46.12% (0.95).

The cloud of smoke was segmented in each video frame using a threshold-based region-growing algorithm yielding the area of the cloud and its contour as a function of time in three dimensions from the mouth of the singer (*x*-dimension to the front, *y*-dimension from the left to the right, and *z*-dimension from the bottom to the top). The dimensions of the region of interest (ROI) were  $ROI_x \times ROI_y \times ROI_z = 260 \text{ cm} \times 270 \text{ cm} \times 180 \text{ cm}$  for camera 1 and  $190 \text{ cm} \times 270 \text{ cm} \times 180 \text{ cm}$  for camera 2.

### Results

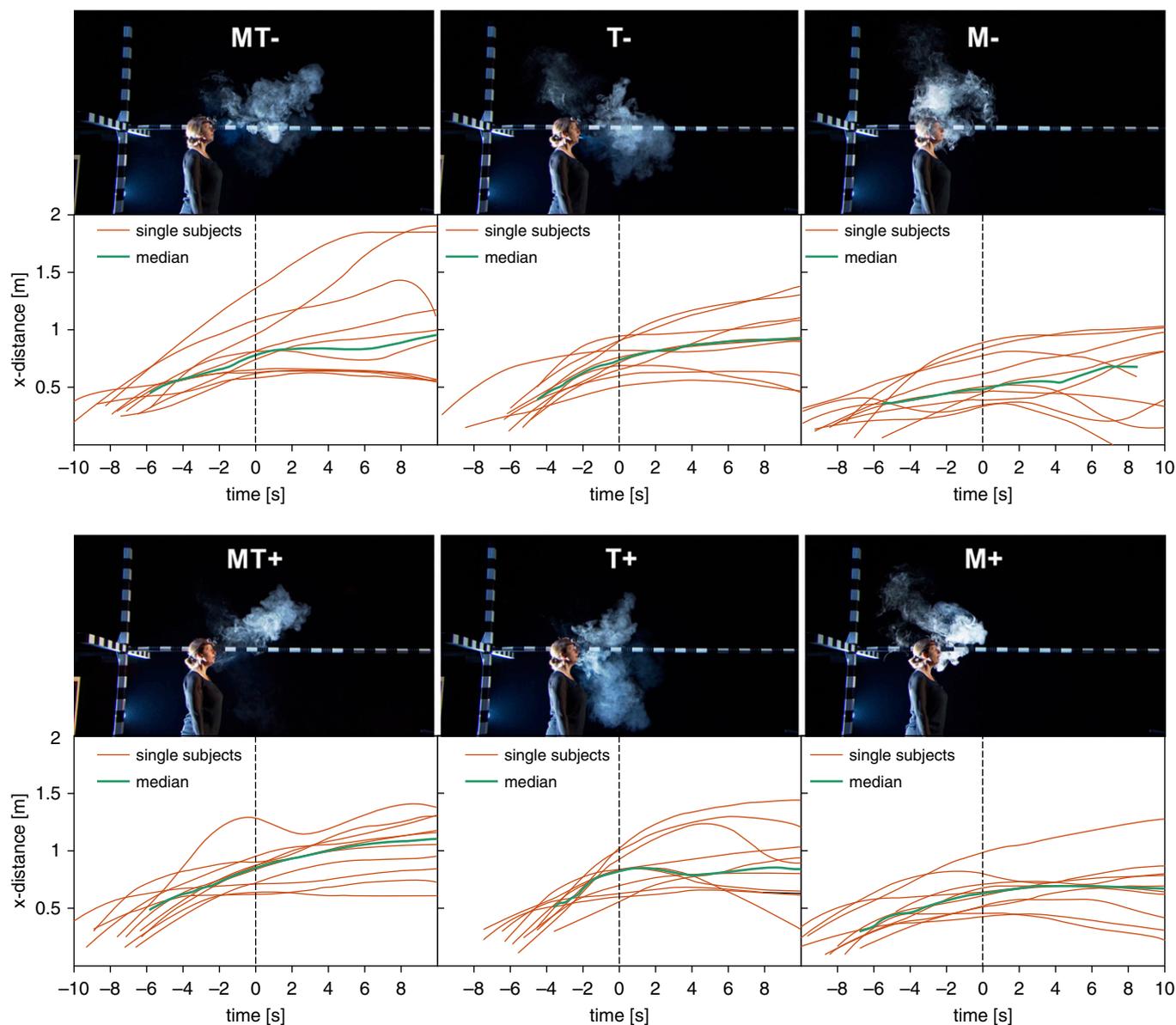
The impulse dispersion in *x*-direction was found to be greater than in *y*- or *z*-direction. The median distance to the front was 0.86 m for MT+, 0.78 m for MT−, 0.82 m for T+, and 0.74 m for T− at the end of the tasks. The M tasks revealed distinctly lower values with 0.62 m (M+) and 0.49 m (M−), respectively (Friedmann/Wilcoxon/Bonferroni-correction *P* values:  $MT_{\pm}$  vs.  $M_{\pm} = 0.003$ ,  $T_{\pm}$  vs.  $M_{\pm} = 0.015$ , and  $MT_{\pm}$  vs.  $T_{\pm} =$  nonsignificant). The intersubject variability was large, ranging from 0.61 m to 1.36 m for the MT tasks (Figure 1). Once a task was completed, the motion of the aerosol cloud decreased with an additional median movement to the front (*x*-direction) between 0.04 m and 0.11 m for all tasks 3 seconds after the end of task. The dispersion to the side was much less (Figure 2). However, the distances in *y*-direction show a lateralization imbalance for some subjects, presumably because of a small convective flow generated by the singer's motion immediately before the beginning of the task. The *y*-diameter between left and right exhibited median values between 0.57 m and 0.88 m at the end of the task.

The sound pressure level was  $MT- = 57.08 \text{ dB(A)}$ ,  $MT+ = 67.75 \text{ dB(A)}$ ,  $T- = 44.69 \text{ dB(A)}$ ,  $T+ = 65.32 \text{ dB(A)}$ ,  $M- = 61.74 \text{ dB(A)}$ , and  $M+ = 73.12 \text{ dB(A)}$  at 1.5 m distance. Although there was a tendency for loud tasks to show different dispersion patterns than the soft ones, statistical analysis failed to show significance ( $Loud_{MT,T,M}$  vs.  $Soft_{MT,T,M}$  Wilcoxon  $P = 0.069$ ).

With regard to both breathing and coughing tasks, detected distances were much greater than all phonation related tasks. After 6 seconds of exhalation, the median distance in the *x*-direction was 1.19 m (maximum 1.71 m), and after coughing, the median distance in the *x*-direction was 1.32 m (maximum 1.89 m).

### Discussion

Although the median distance to the front reached values <1 m for the MT+ and MT− tasks, many subjects reached greater distances of up to 1.4 m. The dispersion distance to the side was much lower. Because of the maximum dispersion, no distances lower than 2–2.5 m between persons to the front and 1.5 m to the side should be recommended as safety distance. However, safety is not only



**Figure 1.** Exemplary images demonstrating the dispersion of a female subject at the end of all tasks (melody and text, text, and melody). Below each image, the diagrams show the temporal dispersion to the front ( $x$ -direction). The upper row refers to the soft ( $-$ ) versions of the tasks, and the lower row refers to the loud ( $+$ ) versions of the tasks. The red curves represent the individual subjects, and the green curve represents the median. The 0 point on the  $x$ -axis in the time scale refers to the end of the task. M = melody; MT = melody and text; T = text.

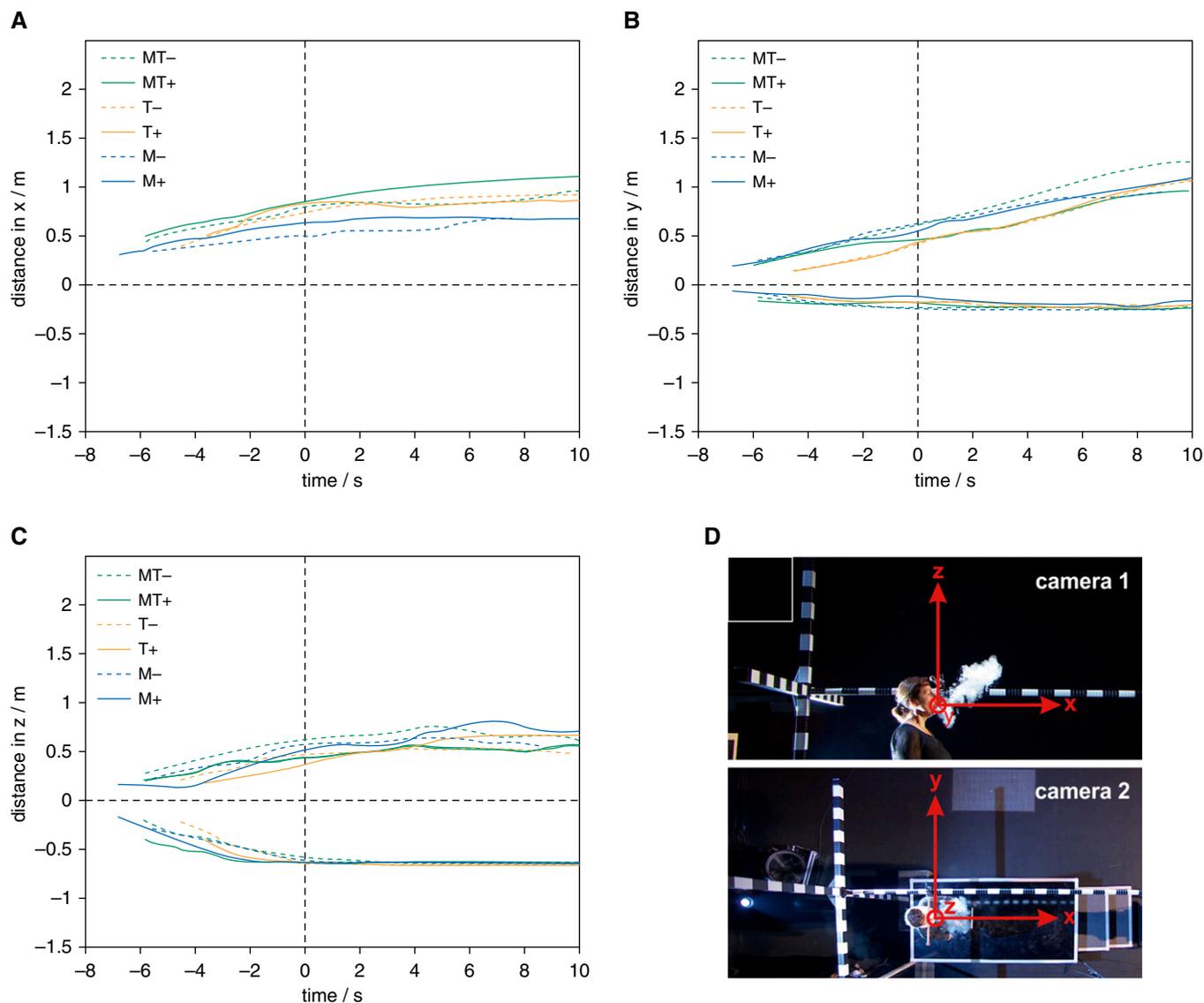
dependent on the measured near field under controlled laboratory conditions but also on the accumulation of aerosols over time during phonation and the convective flow in realistic environments. Therefore, a continuous ventilation and/or filtration of the air volume during singing could diminish the amount of aerosols and therefore reduce the risk of infection transmissions. Furthermore, wearing masks could affect the speed of aerosol dispersion; however, it could also restrain the articulation.

The softer tasks showed a tendentially lower dispersion to the front than the louder tasks. Loudness is dependent on the transglottal pressure difference (3), which generates greater airflow. In agreement with previous studies (4, 5), the largest

frontal dispersion was found for coughing. Furthermore, Asadi and colleagues found that the absolute aerosol production was greater for louder phonation (6). As a consequence, the potential transmission risk appears increased for loud phonation, resulting from both the absolute aerosol production and the tendentially greater dispersion distance to the front.

#### Limitations

The generalization of this study is limited by its inclusion of only professional singers and a consequently low number of subjects. Also, the gas from the e-cigarette might have influenced the singing. Lastly, the study used an artificially added aerosol with a comparable



**Figure 2.** (A–C) Median traces for the x-dimension (front) (A), y-dimension (left–right) (B), and z-dimension (up–down) (C) and all tasks. The 0 point in the time scale refers to the end of the task. The different colors refer to the three tasks (green: melody and text, yellow: text, and blue: melody). The solid lines show the loud (+) tasks, and the dashed lines the soft (–) tasks. (D) The directions in the two camera perspectives. M = melody; MT = melody and text; T = text.

amount of aerosols for each task. The real number of aerosols expelled during phonation is, however, much lower. It has been found that for voiced counting, the number of expelled droplets with sizes of  $0.3\text{--}20\ \mu\text{m}$  was  $0.322\ \text{cm}^{-3}$  and was approximately three times higher during singing (7). ■

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## Novel Documentation of Onset and Rapid Advancement of Pulmonary Arterial Hypertension without Symptoms in BMPR2 Mutation Carriers: Cautionary Tales?

To the Editor:

Mutations in the *BMPR2* (bone morphogenetic protein receptor type 2) gene account for most heritable pulmonary arterial hypertension (PAH) cases. However, pathologic expression only occurs in approximately 42% of female, and 14% of male, carriers, with onset of *BMPR2*-associated PAH (*BMPR2*+PAH) varying across the lifespan (1). Limited data exist regarding the

onset of pulmonary artery pressure (PAP) elevation, etiologies for phenotypic expression, and the optimal frequency of screening. We report two cases that highlight challenges for adolescents and young adults at risk of *BMPR2*+PAH.

### Case 1

A healthy 17-year-old female, whose case timeline is detailed in Figure 1, was evaluated because of a family history of *BMPR2*+PAH. At initial evaluation, she had no signs or symptoms of PAH, a normal physical exam, and a history of regular exercise without exertional symptoms. Her echocardiogram demonstrated normal biventricular function and no evidence of right ventricular pressure (RVP) elevation. Genetic testing confirmed the presence of a pathogenic *BMPR2* gene mutation (c.1125\_1128 + 17del [p.Glu376del]) predicted to alter splicing of intron 8. Because of familial concern, hemodynamic evaluation by cardiac catheterization was performed and revealed a normal cardiac index (CI) (3.4 L/min/m<sup>2</sup>), mean PAP (15 mm Hg), and indexed pulmonary vascular resistance (PVRI) (2.7 Wood units [WU]).

Six months later, echocardiography demonstrated normal RVP estimate and normal biventricular function. Fifteen months following catheterization, and 9 months following her last normal echocardiogram, she presented with a syncopal episode and increasing dyspnea over the preceding 6 weeks after starting college. Echocardiography showed mild right ventricular dilation with elevated RVP. Her laboratory evaluation was remarkable only for a slight elevation in white blood cell count of unclear significance. Evaluation for pulmonary embolism with D-dimer,  $\dot{V}/Q$  scan, and single-photon emission computed tomographic/computed tomographic scan was negative. Of note, she had an etonogestrel/ethinyl estradiol vaginal ring in place for birth control, which had been used since before her genetic testing with negative pregnancy tests. She denied illicit drug use. Catheterization revealed a CI of 3.9 L/min/m<sup>2</sup>, mean PAP of 52 mm Hg, and PVRI of 5.1 WU, unresponsive to acute vasodilator treatment. Sildenafil and ambrisentan were initiated. Repeat catheterization 6 months later demonstrated a CI of 3.5 L/min/m<sup>2</sup>, PAP of 40 mm Hg, and PVRI of 4.8 WU. Selexipag was added.

### Case 2

A healthy 16-year-old male presented for evaluation because of a family history of *BMPR2*+PAH associated with a heterozygous missense mutation in *BMPR2* (c.354T>G [p.Cys118Trp]). Of note, he had an echocardiogram at age 12 for murmur, which showed normal anatomy and function. At initial evaluation, he reported a history of albuterol-responsive wheezing, occasional chest pains but none in over a year, and an admitted lack of interest in physical fitness. His exam was notable for a body mass index of 35.3 kg/m<sup>2</sup> (>97th percentile) and a pronounced second heart sound. His echocardiogram demonstrated normal heart structure, normal biventricular function, mild septal flattening, and elevated tricuspid regurgitant velocity gradient suggestive of RVP elevation (~50 mm Hg). Routine fasting bloodwork for new PAH diagnoses (including HIV, C-reactive protein, and complement levels) were normal except for a blood glucose of 115 mg/dl consistent with a diagnosis of prediabetes.

Cardiac catheterization revealed a CI of 2.5 L/min/m<sup>2</sup>, mean PAP of 38 mm Hg, and PVRI of 10.9 WU, unresponsive to acute vasodilator testing. He was started on sildenafil. Comprehensive workup confirmed the family's *BMPR2* gene mutation. At

All authors have made substantial contributions to the conception of the work, have revised the work critically for important intellectual content, have approved the final version to be published, and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. This letter has a related editorial.

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