

“Gesundheit!” Sneezing, Common Colds, Allergies, and *Staphylococcus aureus* Dispersion

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Background. *Staphylococcus aureus* is among the most important pathogens in today’s hospital setting.

Methods. The effects of sneezing on the airborne dispersal of *S. aureus* and other bacteria were assessed in 11 healthy nasal *S. aureus* carriers with experimentally induced rhinovirus colds. Airborne dispersal was studied by volumetric air sampling in 2 chamber sessions with and without histamine-induced sneezing. After 2 days of preexposure measurements, volunteers were inoculated with a rhinovirus and monitored for 14 days. Daily quantitative nasal- and skin-culture samples for bacteria and nasal-culture samples for rhinovirus were obtained, cold symptoms were assessed, and volunteer activities were recorded during sessions.

Results. All participants developed a cold. Sneezing caused a 4.7-fold increase in the airborne dispersal of *S. aureus*, a 1.4-fold increase in coagulase-negative staphylococci (CoNS), and a 3.9-fold increase in other bacteria ($P < .001$). An additional 2.83 colony forming units (cfu) of *S. aureus*/m³/min, 3.24 cfu of CoNS/m³/min, and 474.61 cfu of other bacteria/m³/min were released per sneeze. Rhinovirus exposure did not change the frequency of sneezing or airborne dispersal. Having respiratory allergies increased the spread of *S. aureus* by 3.8-fold during sneezing sessions ($P < .001$).

Conclusion. Nasal *S. aureus* carriers disperse a significant amount of *S. aureus* into the air by sneezing. Experimental colds do not alter bacterial dispersal, but respiratory allergies multiply the effect of dispersing *S. aureus*.

The act of sneezing is a unique reflex and is a common manifestation of both colds and nasal allergies. Sneezing has also been interpreted as a favorable sign in numerous cultures [1–3], although it has also been associated with danger. Around 580 CE, when the bubonic plague raged in Rome, Pope Gregory the Great incited his people to say “may God bless you” when a person sneezed, to protect the blessed individual from death by the plague [3]. In modern times, it still remains unclear what the actual impact of sneezing is on the

airborne dispersal of bacteria. In particular, to our knowledge, there have been no studies to date that have linked sneezing to nosocomial infections caused by *Staphylococcus aureus* or other bacteria. In this regard, sneezing appears to be somewhat neglected, despite its being a frequent event associated with a variety of conditions, such as common colds and allergic rhinitis.

We previously determined that common colds caused by rhinovirus increased the airborne spread of *S. aureus*, coagulase-negative staphylococci (CoNS), and other upper-respiratory-tract bacteria (the “cloud” phenomenon) [4–7]. Consequently, we wanted to determine the impact of sneezing specifically on the airborne dispersal of these bacteria and to investigate the potential interaction between common colds and sneezing with regard to bacterial dissemination.

PARTICIPANTS AND METHODS

Participants. A total of 593 medical (first and second year) and undergraduate students at Wake Forest University were screened for nasal *S. aureus* carriage by

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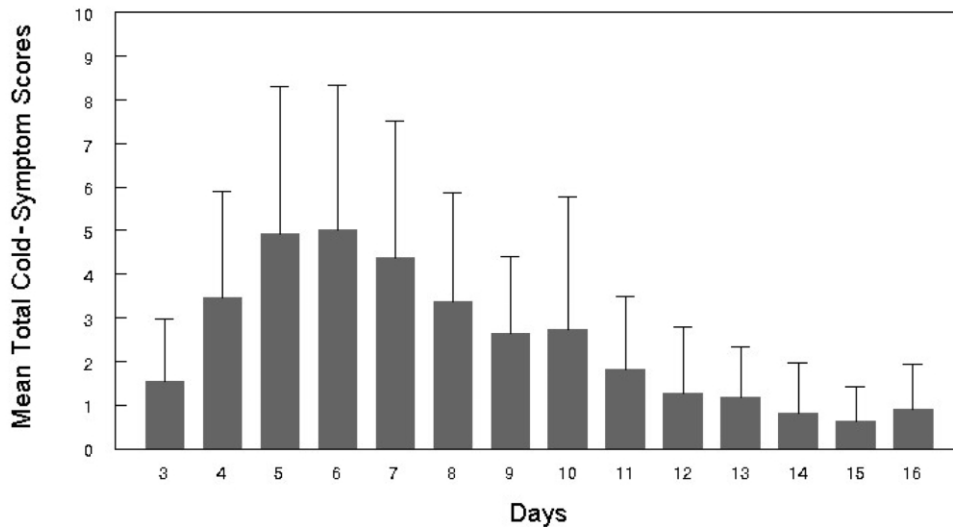


Figure 1. Daily mean total cold-symptom scores for all participants after rhinovirus exposure

nasal cultures from October 2000 to April 2004 [8]. Some 120 (20%) carried *S. aureus* for at least 4 weeks, as confirmed by follow-up nose swabs every 2 weeks. Eleven healthy nasal *S. aureus* carriers were subsequently recruited for participation in the study on the basis of susceptibility (serum-neutralizing antibody titer $\leq 1:4$) to 1 of 3 available challenge rhinoviruses.

A self-administered questionnaire regarding the medical and medication history of the participants was obtained from all individuals. Each participant provided informed consent, and the human-experimentation guidelines of the US Department of Health and Human Services and those of the Wake Forest University School of Medicine (Winston-Salem, NC) were followed in the conduct of the study.

Microbiologic techniques. The methods used for the collection of mucosal and air cultures, virus challenge, and the assessment of rhinovirus infection and illness have been described elsewhere in detail [7]. In short, samples for quantitative nasal, pharynx, and skin (both axillae, palms of the hands, and groin) culture were obtained from each participant by swabbing. After surface plating and incubation for 24–48 h at 35°C, the total number of colonies and those that had morphological characteristics consistent with *S. aureus* and CoNS were counted as colony-forming units and evaluated by Gram’s stain, catalase test, and coagulase test (Staphaurex; Murex Biotech).

Air-sample cultures were performed in an airtight chamber ~ 110 ft³ (3.1 m³) in volume that was built around the front of a class II biological safety hood (Purifier; Labconco) with sufficient room for a volunteer to sit in front of the workbench. Three 2-stage and one 6-stage Andersen air samplers were used to measure airborne bacterial particles by impact on blood agar plates (sample volume, 28.3 L [1 ft³]/min; Andersen Instru-

ments) [9]. The agar plates were incubated for 24 h at 35°C, then held for an additional 24 h at room temperature [10]. Colonies were counted and evaluated as described above. One colony of *S. aureus* and CoNS from each site cultured, if available, was stored in 95% tryptic soy broth with 5% glycerol at -70°C for molecular typing.

To test the recovery of *S. aureus* isolates in the test chamber, a saline solution that contained a defined concentration of *S. aureus* bacteria was dispersed into the air using a Collison nebulizer [11]. Air samples were collected using the Andersen air samplers for 20 min, and the recovery rate was calculated as the mean of 3 independent chamber runs. On average, 8 cfu/m³/min of *S. aureus* had to be dispersed by the nebulizer to recover 1 cfu of *S. aureus*.

Molecular typing of selected *S. aureus* isolates was performed using the restriction endonuclease *Sma*I followed by pulsed-field gel electrophoresis (PFGE; BioRad) [12]. Cluster analysis of the banding patterns was used to determine clonal similarity (BioNumerics software version 3.0; Applied Math). Strains were considered to be different if they showed >3 fragment differences [13]. Isolates from the dominant strains were tested for methicillin susceptibility using the disk-diffusion test (1 μg of methicillin; control *S. aureus* strain ATCC 25923) [14].

Study design. Daily bacterial air-sample cultures were performed for 16 consecutive days, with participants sitting in the chamber for two 20-min sessions, preceded by a control run without participants. In all, there were 528 chamber sessions. During the first or “nonsneezing” session, participants were wearing sterile garb (a fluid-resistant surgical gown [Medline Industries] over street clothes, surgical gloves [Ansell Healthcare Products], shoe covers [Spunbond Gripper; Medline In-

dustries], and a bouffant cap [Spunbond Sheer-Fit; Medline Industries]). This was followed by a histamine-induced sneezing session with new sterile garb, gloves, shoe covers, bouffant cap, and the administration of intranasal histamine (25 mg/mL). The latter was applied to the anterior nares with a sterile cotton swab and distributed by gently pressing the exterior walls of the nose together [15]. This sequence of sessions was repeated on all 16 days. During chamber sessions, the number of sneezes, coughs, nose blows, talking, and unusual activities of the participant were recorded by directly observing study personnel. The participants were asked to avoid brisk body movements and to sneeze or cough in the direction of the safety hood without covering the facial area.

Participants were exposed after day 2 to 1 of 3 rhinovirus serotypes (Hanks, 39, or 16) at an intranasal dose of ~100 TCID₅₀, on the basis of their serosusceptibility (serum neutralizing antibody titers ≤1:4 to the challenge virus) [7, 16]. The measurements of bacterial dispersal were repeated daily. Infection was determined by virus isolation from nasal lavage samples and by measurement of homotypic serum-neutralizing antibody titers on paired acute and convalescent (4-week) specimens. To assess illness, 8 symptoms (sneezing, rhinorrhea, nasal obstruction, sore or scratchy throat, cough, malaise, chilliness, and headache) were each evaluated daily on a scale of 0 to 4 [17]. The total symptom score is the sum of the individual symptom ratings. A cold was defined as being present if a subject had a total symptom score of ≥6 and rhinorrhea on ≥3 days and/or the subjective impression of a cold [17].

Data analysis. Data analyses were performed using STATA software (version 9.1; StataCorp). The outcome was defined as the daily dispersal of airborne bacteria—such as *S. aureus*, CoNS, and other bacteria—at the individual level over the course of 16 consecutive days, in colony-forming units per cubic meter per minute. The 2 study sessions with or without exposure to histamine served as a categorical exposure variable. Model building and testing were based on random-effects neg-

ative binomial distribution for overdispersed count data [18, 19]. Effect measures are expressed as incidence rate ratios (IRRs). This distribution accounts for between-individual heterogeneity in rates of airborne bacteria, which can lead to overdispersion and an excess of zeros found in the outcome distributions. The random-effects negative binomial distribution also accounts for clustering due to repeated observations occurring within an individual over time and across the sessions.

The actual model building was based on a hierarchical backward-elimination strategy that pared down from the most complex model to a final model. Potential risk factors influencing the exposure outcome association included the modifiable factors exposure to rhinovirus and allergies and the fixed factors sex and age. Effect-measure modification was assessed using the composite Wald test with an α -level set at .05 and Akaike's [20] and Schwartz's [21] information criteria [22]. The change-in-estimate technique was chosen to test for confounding, with a preset threshold of 10%. After calculation of the final models, model diagnostics were applied to detect potential influential cases. Because of the clustered nature of the data set, each individual participant was removed 1 at a time from the final models, and the change-in-estimate effects were determined. The threshold was preset at a 20% change-in-estimate effect.

RESULTS

Participants and measures of illness/infection. Two women and 9 men (mean age, 24 years; range, 19–29 years) were studied. One volunteer smoked. All participants had ≥1 symptom and met the criteria for having a cold. Figure 1 depicts the daily total cold symptom scores after exposure to rhinovirus. Table 1 lists the number of participants affected by specific cold symptoms and the maximum symptom scores during the 5 days after rhinovirus exposure.

Volunteers most often had high symptom scores for nasal congestion, sore throat, and runny nose, followed by coughing,

Table 1. Frequency of specific cold symptoms and maximum symptom scores in 11 individual participants during the 5 days after rhinovirus exposure.

Symptom	Study day 3		Study day 4		Study day 5		Study day 6		Study day 7	
	Volunteers affected	Maximum score	Volunteers affected	Maximum score	Volunteers affected	Maximum score	Volunteers affected	Maximum score	Volunteers affected	Maximum score
Sneezing	0	0	3	1	2	1	2	1	1	1
Cough	1	1	2	1	2	2	3	2	5	1
Runny nose	3	1	4	4	4	4	4	4	4	3
Nasal congestion	3	1	8	3	8	3	7	3	7	3
Sore throat	5	2	7	2	8	3	8	4	7	3
Headache	0	0	1	1	4	2	3	2	2	2
Malaise	2	1	4	2	6	2	6	2	5	2
Chills	0	0	0	0	1	1	1	2	1	1

NOTE. Data are no. affected, unless otherwise indicated. Symptom scores: 0, absent; 1, mild; 2, moderate; 3, severe; 4, very severe.

Table 2. Comparison of bacterial carriage before and after rhinovirus exposure.

Organism, exposure status	Nose			Pharynx			Skin		
	Mean ± SD, cfu	IRR (95% CI)	<i>P</i>	Mean ± SD, cfu	IRR (95% CI)	<i>P</i>	Mean ± SD, cfu	IRR (95% CI)	<i>P</i>
<i>Staphylococcus aureus</i>									
Before	5428.55 ± 13,301.61	1.00		37.27 ± 116.06	1.00		273.64 ± 622.18	1.00	
After	4492.78 ± 8666.59	1.02 (0.76–1.38)	.888	60.60 ± 269.13	1.51 (0.79–2.89)	.215	1896.21 ± 8186.62	2.06 (1.41–3.03)	<.001
CoNS									
Before	3341.09 ± 4281.88	1.00		127.64 ± 446.26	1.00		7892.18 ± 24,985.84	1.00	
After	5514.16 ± 10,171.64	0.84 (0.67–1.06)	.141	91.90 ± 401.84	1.00 (0.43–2.35)	.999	11,618.18 ± 44,210.35	1.12 (0.85–1.48)	.428
Other bacteria									
Before	1501.00 ± 3376.88	1.00		50,925.00 ± 45,531.75	1.00		4699.00 ± 16,844.50	1.00	
After	540.46 ± 2454.49	0.36 (0.19–0.69)	.002	72,062.79 ± 130,444.00	0.90 (0.68–1.20)	.463	2632.94 ± 9134.24	1.01 (0.71–1.46)	.939
All bacteria									
Before	5707.75 ± 3920.62	1.00		51,151.25 ± 45,410.22	1.00		15,759.50 ± 38,386.93	1.00	
After	9967.29 ± 10,463.62	1.19 (0.92–1.55)	.175	72,271.11 ± 130,414.80	0.89 (0.67–1.19)	.437	20,998.43 ± 63,020.66	1.22 (0.88–1.69)	.232

NOTE. Data are based on random-effects negative binomial regression models. CoNS, coagulase-negative staphylococci; IRR incidence rate ratio.

Table 3. Mean airborne dispersal of *Staphylococcus aureus*, coagulase-negative staphylococci (CoNS), and other bacteria during study sessions, before and after rhinovirus exposure.

Organisms	Nonsneezing sessions		Sneezing sessions	
	Before rhinovirus exposure	After rhinovirus exposure	Before rhinovirus exposure	After rhinovirus exposure
<i>S. aureus</i>	0.06 ± 0.21	0.50 ± 1.16 ^a	7.76 ± 12.10	15.44 ± 38.24 ^a
CoNS	15.87 ± 29.04	31.80 ± 52.55 ^a	34.13 ± 47.09	47.53 ± 81.47 ^a
Other bacteria	13.42 ± 20.25	44.94 ± 282.78 ^a	1503.01 ± 2134.77	2522.64 ± 4141.83 ^a

NOTE. Data are mean ± SD cfu/m³/min. No bacteria were detected during the control sessions.

^a *P* > .05, sessions before vs. after rhinovirus exposure, random-effects negative binomial models.

malaise, and headache. Sneezing and chills were reported to be less frequent and less severe.

Seroconversion with an antibody increase of ≥4 dilutions occurred in 6 volunteers. All participants were infected and had at least 1 nasal-wash sample positive for the challenge rhinovirus after exposure.

Sneezing and other events. During the histamine-induced sneezing sessions, volunteers sneezed 5.4 ± 4.4 times before rhinovirus exposure and 4.9 ± 2.8 times after exposure, respectively (*P* = .2622). Sneezing events were rare during the nonhistamine sessions (4 sneezes in 176 sessions). Nose blowing occurred more frequently during the sneezing sessions than during the nonsneezing sessions (1.61 ± 1.38 vs. 0.01 ± 0.15; *P* < .0001), but this did not change significantly after rhinovirus exposure (*P* = .1736). Coughing events were uncommon, with a total of 183 coughs during 352 chamber sessions and with no apparent difference between sessions or rhinovirus exposure (*P* = .7722). Reading was the only additional activity noted.

Mucosal/skin cultures. All participants carried *S. aureus* in their nares before and after rhinovirus exposure (table 2). The mean number of *S. aureus* in the nose or pharynx did not change significantly after rhinovirus exposure (IRR, 1.02; *P* = .888). However, skin carriage with *S. aureus* doubled from before to after rhinovirus exposure (*P* < .001), whereas nose carriage with “other bacteria” decreased by 64% (*P* = .002).

None of the other bacteria groups changed because of rhinovirus exposure.

Airborne dispersal of *S. aureus* and other upper-respiratory-tract bacteria. A total of 788,179 cfu were detected in air cultures, including 2130 cfu of *S. aureus* (0.3%) and 12,205 cfu of CoNS (1.6%). Table 3 lists the mean counts of *S. aureus*, CoNS, and other bacteria for the sneezing and nonsneezing sessions before and after rhinovirus exposure.

Tables 4 and 5 list the final models after testing for effect-measure modifiers and confounders. The comparison between sneezing and nonsneezing sessions showed a 4.71-fold increase in *S. aureus*, a 1.43-fold increase in CoNS, and a 3.92-fold increase in other bacteria associated with sneezing (*P* < .0001). Individual *S. aureus* dispersal during the sneezing sessions varied from 0 to 1.77 to 278.96 cfu/m³/min. The presence of respiratory allergies multiplied the effect measure for *S. aureus* from a 4.71-fold to a 17.45-fold increase in airborne *S. aureus* dispersal in allergic participants (table 5). The histamine-induced frequency of sneezing did not differ between allergic and nonallergic participants (average number of sneezes per session, 5.15 for allergic participants vs. 4.70 for nonallergic participants). However, the airborne release of *S. aureus* by a single sneeze was significantly higher in allergic than in nonallergic participants (IRR_{nonallergic participants}, 1.13 [95% confidence interval {CI}, 1.095–1.18] [*P* < .001] vs. IRR_{allergic participants}, 1.34

Table 4. Estimated regression coefficients and SEs from final models for all outcome variables.

Variable	<i>Staphylococcus aureus</i>		CoNS		Other bacteria	
	Coefficient ± SE	<i>P</i>	Coefficient ± SE	<i>P</i>	Coefficient ± SE	<i>P</i>
Sneezing	1.55 ± 0.19	<.001	0.36 ± 0.09	<.001	1.36 ± 0.25	<.001
Allergies	1.64 ± 0.45	<.001
Age	−0.15 ± 0.03	<.001
Sex	0.14 ± 0.22	.517
Sneezing ^a and allergies	1.32 ± 0.47	.005
Sneezing ^a and age	0.22 ± 0.03	<.001
Sneezing ^a and sex	0.63 ± 0.28	.023

NOTE. CoNS, coagulase-negative staphylococci.

^a Based on random-effects negative binomial regression models.

Table 5. Estimated incidence rate ratios (IRRs) comparing sneezing and nonsneezing sessions and 95% confidence intervals (CIs) from final models, with effect modification for all outcome variables presented in table 4.

Organism, model	IRR (95% CI)
<i>Staphylococcus aureus</i>	
Allergies	17.45 (7.48–40.71)
No allergies	4.71 (3.27–6.78)
CoNS	
Mean age +1 year	1.79 (1.40–2.26)
Mean age	1.43 (1.19–1.71)
Other bacteria	
Male sex	7.22 (5.76–9.07)
Female sex	3.92 (2.39–6.41)

NOTE. Data are based on random-effects negative binomial regression models. $P < .001$ for all outcome variables shown. CoNS, coagulase-negative staphylococci.

[95% CI, 1.27–1.41] [$P < .05$]). Effect-measure modification was also noted for CoNS by age, with a decrease in airborne dispersal of CoNS with older age. Male participants shed larger amounts of other bacteria into the air than female volunteers. No influential cases were detected when model diagnostics were used.

On average, 1 sneeze expelled 2.83 cfu of *S. aureus*/m³/min, 3.24 cfu of CoNS/m³/min, and 474.61 cfu of other bacteria/m³/min over and above the existing dispersal. The percentage of particles $< 5 \mu\text{m}$ carrying *S. aureus* increased from 16.2% during nonsneezing sessions to 37.6% during sneezing sessions ($P = .0593$). A modest increase was observed in other bacteria (42.9%–49.4%; $P = .0827$), whereas the percentage of CoNS did not change (30.5%–29.5%; $P = .7740$). Rhinovirus exposure led to a similar increase in small particles carrying *S. aureus* (0%–16.7% for nonsneezing sessions, $P = .0161$; 23.9%–38.6% for sneezing sessions, $P = .0773$). CoNS and other bacteria did not change significantly in the nonsneezing sessions. During the sneezing sessions, there was a statistically significant decrease in small particles for other bacteria (64.5%–48.0%; $P < .001$) but not for CoNS ($P = .1730$).

Sneezing, the common cold, and airborne bacterial dispersal.

Inoculation with rhinovirus at day 2 led to a 1.4–8.3-fold increase in the airborne spread of *S. aureus*, CoNS, or other bacteria (table 3). However, the changes were not statistically significant in random-effects negative binomial models ($P > .05$). Furthermore, the cold-symptom score was also not associated with a change in the dispersal of bacteria into the air.

***S. aureus* characterization.** A total of 813 of 2130 cfu of *S. aureus* (38%) were studied by PFGE. The clonal similarity of airborne and skin/mucosal *S. aureus* isolates was 84%–100% in 9 volunteers. The remaining 2 volunteers carried 2 distinct *S. aureus* strains, each with a dominating clone accounting for

60% and 78%, respectively, of *S. aureus* isolates. All *S. aureus* strains were sensitive to methicillin.

DISCUSSION

Sneezing is a common physiological reflex that involves the sudden, uncontrolled, forcible expulsion of air through the nose and mouth. The causes are numerous and include allergic rhinitis, vasomotor rhinitis, nasal irritation, drug withdrawal, exposure to bright light, gastric distension, epilepsy, psychogenic factors, sexual excitement, menstruation, chilling, cervical tuberculous lymphadenitis, and the common cold [23]. In particular, the impact of the latter on sneezing and the airborne dispersal of bacteria provoked our interest, given that each adult has an average of 2 colds each year [24]. We focused primarily on *S. aureus*, because it is among the most common nosocomial pathogens in the United States [25, 26].

Early studies demonstrated the large quantities of bacteria spread by sneezing into the environment, compared with other activities, such as quietly breathing, talking, or coughing. Hare and Mackenzie [27] found a 95-fold increase in all airborne bacteria due to sneezing: a 50-fold increase from talking to sneezing and a 34-fold increase from coughing to sneezing. However, sneezing did not yield the airborne dispersal of *S. aureus*. By contrast, Duguid and Wallace [28] found *S. aureus* in the air after induced sneezing in 2 volunteers. In the present study, we used more-sophisticated measuring techniques to show that sneezing causes a highly significant increase in *S. aureus*, CoNS, and other bacteria in environmental samples. It is noteworthy that the range of responses varied widely among the individual participants, from < 1 to 279 cfu of *S. aureus*/m³/min during sneezing sessions. Five volunteers dispersed > 60 cfu of *S. aureus*/m³/min, whereas the remaining 6 volunteers did not expel > 5 cfu of *S. aureus*/m³/min. This is likely explained by the higher frequency of sneezing in the first volunteer group, in which an average of 6.5 sneezes/session was seen, compared with 3.8 sneezes/session in the latter group ($P < .05$). It must also be noted that no other cold symptom or activity was statistically related to this increase.

The actual number of bacteria set free by a single sneezing event may be a key parameter in risk assessment for cross-infection in nosocomial settings. Our results demonstrate that 1 sneeze expels, on average, mostly other bacteria, such as α -hemolytic streptococci (474.61 cfu/m³/min), but also 3.24 cfu of CoNS/m³/min and 2.83 cfu of *S. aureus*/m³/min. No clear threshold exists for the airborne infective dose of *S. aureus*. However, Forster and Hutt [29] conducted several experiments in which they inoculated skin lesions with broth dilutions that contained *S. aureus*. The lowest amount that produced infection in a standard skin lesion was 15 cfu of *S. aureus*/mL. Previous tests in our airtight chamber revealed that, for every colony-forming unit recovered by Andersen samplers, 8 cfu have to

be dispersed into the air by the participant. Given that 2.83 cfu of *S. aureus*/m³/min were recovered per sneeze, ~23 cfu/m³/min were expelled into the air by 1 sneeze. Thus, a sneeze could deposit a significant number of *S. aureus* on a patient. Furthermore, it is also possible that airborne *S. aureus* can be transferred to and colonize the mucus membranes of persons in close proximity. However, to our knowledge, no data are available to date supporting this route of cross-colonization.

Multivariate analyses revealed several effect-measure modifiers for the different species of bacteria and groups. The presence of respiratory allergies led to an increase in airborne *S. aureus*. Shiomori et al. [30] found an association between perennial allergies and higher rates of *S. aureus* carriage. These higher rates may lead to increased airborne dispersal. This is supported by the results of Bassetti et al. [5], which showed a significant increase in airborne spreading patterns in a person with symptomatic allergic rhinitis. Independent predictors were sneezing and the number of tissues used. In our study, the increased spread was associated not with sneezing frequencies or nasal or skin carriage rates but with the amount of organisms expelled by a sneeze. One can speculate about differences in the intensities of the sneezes in allergic individuals, the mucous membranes, or mucus itself promoting the release of *S. aureus*. Further studies are necessary to clarify the underlying mechanisms of allergies of the respiratory tract and *S. aureus* airborne dispersal. This applies also to the effects of age and sex on the airborne spread of CoNS and other bacteria.

When investigating the transmission of pathogens via air, the distance between source and potential target is crucial. Because of the closed environment of our test chamber, it was not possible to investigate how far *S. aureus* or other bacteria could be spread from participants. However, measurement of the particle sizes revealed that sneezing caused a significant increase in particles <5 µm in size carrying *S. aureus*, from 16.2% to 37.6%. It is well known that organisms attached to these particle sizes are transmissible via the airborne route because of their capability to float through the air [31–33]. Therefore, prolonged and wide dispersal of *S. aureus* by air after sneezing can be assumed. Once in the environment, *S. aureus* isolates can survive for >60 days on common hospital materials, which presents an immanent risk for environment-to-person transmission [34, 35].

A further objective of our study was to determine the impact of a common cold on the airborne spread of upper-respiratory-tract bacteria during sneezing. We compared the number of infection-related and histamine-induced sneezing events during the chamber sessions before and after exposure to rhinovirus, and we found no significant change in frequency. This was also confirmed by the overall low score of sneezing symptoms over the course of a 24-h period that was reported daily by the participants. Induction of a common cold was not an independent risk factor for the airborne dispersal of *S. aureus*,

CoNS, and other bacteria in the comparison of nonsneezing with sneezing sessions. One has to mention that previous findings indicated a significant increase in the airborne dispersal of *S. aureus* due to a rhinovirus challenge [4, 6]. This was not confirmed in the present study, where the main focus was to study the effect of sneezing. The implementation of preventive measures for persons with mild to moderate common colds because of this higher bacterial air load appears to be unnecessary, unless sneezing is part of their symptom complex.

The main source of airborne *S. aureus* could not be conclusively determined. High clonal similarity with >84% for single strain carriers did not allow us to distinguish between organisms generated from the nose, pharynx, or skin. However, when we compared the airborne spreading patterns of bacteria with their natural colonization areas, *S. aureus* and other bacteria usually found on mucus membranes were more frequently expelled during sneezing sessions than were skin colonizers, such as CoNS.

It remains unknown whether other viruses, such as coronaviruses or influenza virus, might cause similar results. In addition, the significant increase detected in the group of other bacteria with ~475 cfu/m³/min per sneeze warrants further study, given that pathogens such as penicillin-resistant pneumococci, *Neisseria meningitidis*, and *Streptococcus pyogenes* can inhabit the human respiratory tract and might be similarly spread by airborne route [36–38].

In conclusion, our findings suggest that sneezing contributes to the risk of cross-infection by airborne transmission of *S. aureus*, CoNS, and probably other upper-respiratory-tract bacteria, and they should be taken into consideration in future investigations of outbreaks. Mild to moderate common colds do not appear to increase the risk of bacterial spread unless sneezing is a part of the syndrome. However, individuals with respiratory allergies pose a potential source for airborne *S. aureus* spread, particularly when they sneeze.

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