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A new method of assessing aerosolized bacteria generated during orthodontic debonding procedures

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Introduction: The main objective of this study was to assess the efficacy of a new and innovative method of harvesting bacteria that are aerosolized during orthodontic debonding. Additionally, the protection efficacy of several commercially available masks from such aerosols was assessed in a pilot study. **Methods:** Twenty-six subjects were debonded during aerosol sampling, by using an innovative collection system to harvest bonding dust liberated during debonding. Dark-field microscopy, gram-stain microscopy, and chemical identification were used to determine speciation of the collected aerosol from 23 subjects. Three additional subjects were used to test 3 commercial dental or protective masks to determine whether they provide effective protection from the aerosol. **Results:** Twenty-one species of oral bacteria were identified by the new sampling technique. Two of the 3 masks that were tested offered no protection against the aerosolized bacteria. **Conclusions:** A new and effective method for collecting airborne bacteria is presented. Some conventional dental masks offer no protection from aerosolized organisms liberated during debonding procedures. Further assessment of mask efficacy is ongoing. (Am J Orthod Dentofacial Orthop 2008;133:S79-87)

Respiratory diseases are responsible for significant morbidity and mortality in humans. In 2001, chronic lower respiratory disease was listed as the fourth leading cause of death in the United States, claiming 123,000 lives, whereas pneumonia and influenza together contributed to 62,000 deaths per year as the seventh leading cause of death.[1] Although the precise relationship of aerosolized pathogens to the lower respiratory death rate is unclear, a suggested mechanism of infection is contamination of the lower airway epithelium by microorganisms from aerosolized droplets.[2] Aerosols that compromise the respiratory system have also been implicated in other diseases, such as asthma, heart disease, and cancer.[3,4]

The body is assaulted by pathogenic microorganisms many times each day. Subsequent development of infection depends on 2 factors: the host's susceptibility to disease and the organism's ability to cause disease.

The host's susceptibility depends on the integrity of defensive factors such as nasal hairs, convoluted nasal passages, the mucous lining of the nasal turbinates, secretory immunoglobulin A and antibacterial substances such as lysozymes in respiratory secretions, cilia, and the mucous lining of the trachea. Reflexes such as sneezing, coughing, and swallowing are also defensive. Macrophage activity engulfs particles that have escaped physical defenses. Florae of the nasopharynx and oropharynx also prevent colonization of the upper respiratory tract from invading microbes. However, these normal florae can cause disease for unknown reasons and in unknown circumstances, probably because of physical damage to the respiratory epithelium or decreased host immunity.

The microorganism's ability to cause disease is based on several factors. Adherence capability defines the microorganism's ability to adhere to the respiratory tract mucosa and subsequently colonize. *Staphylococcus aureus* is an example of a species whose cell wall contains lipoteichoic acid and certain M proteins that can adhere to respiratory membranes. Toxin production is another function that enhances virulence. It has been shown that lifeguards working at indoor swimming pools can develop granulomatous lung disease associated with endotoxin-producing microbes emitted by water spray equipment.[5] Among others, *S aureus* and beta-hemolytic staphylococcus produce extracellular enzymes that damage host cells or tissues including phagocytotic cells.

There are 3 routes for oral microorganisms to reach the lower respiratory tract: hematogenous spread, aspiration, and inhalation. Factors such as nasal vs mouth breathing, preexisting airway disease, hygroscopic nature of the particles, and breathing patterns are factors that determine the depth of penetration of airborne particles.[6] It has been well documented that dental procedures can introduce oral pathogens into the bloodstream or lymphatic system via direct hematogenous spread or aspiration to cause various medical conditions, including bacteremia,[7] aspiration pneumonia,[8] coronary heart disease,[9] preterm low birth weight,[10] infective endocarditis,[11] gastrointestinal infections,[12] and osteogenic and prosthetic implant infections.[13]

The pulmonary defense system usually maintains the 300 million sterile alveoli in the lower respiratory tract, but systemic outcomes are more commonly seen in elderly, institutionalized people or patients with poor oral hygiene or periodontitis.[14] One study estimated that a third of lung abscesses are caused by oral microorganisms after direct aspiration of saliva or dental plaque or from blood dissemination of septic emboli.[15] It has also been found that aspiration of upper airway material occurs more often while a patient is in a state of depressed consciousness—eg, in 45% of healthy subjects during sleep or impaired consciousness.[16]

Changes in physical condition might also influence one's predisposition to disease. For example, a recent study suggested that age-related decline of respiratory tract ciliary function can also contribute to greater host susceptibility to respiratory infections.[17] The association of oral bacteria and systemic diseases is further supported when oral hygiene was implemented to reduce colonizing bacteria. Yoneyama et al[18] demonstrated that the risk of pneumonia could be reduced by 1.67 times in the intervention group practicing improved oral hygiene compared with that of the control group.

Sama et al[19] found that two-thirds of 469 patients had no evidence of previous asthma diagnosis. New-onset cases were more likely attributed to infection (59%) than to allergy (14%). The overall frequency for work-related asthma was approximately 5%. The conclusion of the study was that most clinicians ignore the contribution of occupational and workplace exposures as etiology of adult-onset asthma.

The American Thoracic Society Statement of 2003 further emphasized that the occupational component of adult-onset asthma and chronic obstructive pulmonary disease is authentic, but the recognition is difficult for 2 reasons. "These are multifactorial diseases strongly associated with nonoccupational exposures," and "the occupational dose-response for obstructive airway disease is complex."[20] Both environmental and genetic factors play roles in etiology. The literature review for the statement showed that 15% of both work-related asthma and chronic obstructive pulmonary disease involves a cost estimate of nearly $7 billion in the United States alone.[20]

It is estimated that about 150 billion microorganisms can be found in 1 g of crevicular fluid collected from a dental patient with poor oral hygiene, and about 6 billion microorganisms are present in 1 mL of saliva.[21] One study showed that as many as 100,000 bacteria per cubic foot of aerosols can be generated from a patient's mouth that normally contains 300 to 500 different species of bacteria.[22] Despite the lack of definitive epidemiologic studies that link dental aerosols to disease transmission, the reported resurgence of bacterial pathogens with airborne transmissibility such as tuberculosis has raised concerns about potential risks to health care providers as a result of inhalation of aerosol contamination during dental procedures. It has been reported that dental aerosols can contaminate the skin and mucous membranes of the mouth, respiratory passages, and eyes of dental personnel.[23] For example, higher prevalences of aerosol-related symptoms such as nasal, ocular, and skin discharges have been observed in dental hygienists.[24]

The concern in dentistry is contamination by aerosolization. An aerosol is defined as a gas generated by coughing, sneezing, or any act that expels oral fluids into the air. If these particles consist of microorganisms or their by-products—ie, endotoxins or mycotoxins—the aerosol is called a bioaerosol. It can consist of cell particles, droplets, and airborne dust and debris. Infectious bioaerosols, on the other hand, are those that are generated as respirable particles. The infectious potential of aerosols depends on several factors, including aerosol generation, particle size and concentration, viability of the contaminating microorganisms, infectivity and virulence, airflow, climate, and host susceptibility.[25]

Inhalation of the larger 10 to 15 μm droplet nuclei particles is more closely related to upper respiratory infections, whereas the smaller 0.5 to 3.0 μm droplet nuclei tend to accumulate in the lower respiratory tract (1 μm = 1/25 thousandth of an inch). Most respiratory infections transmitted through inhalation of droplet nuclei are less than 5 μm in diameter. It was reported that most dental aerosol droplets have a diameter of 5 μm or less and are concentrated within 2 feet (approximately 61 cm) of the patient's mouth.[26]

Particles of 2.5 μm in diameter can circulate for days in moderately turbulent air, potentially prolonging their harmful effects.[27]

The disease potential to health care providers and patients from inhalation of aerosolized oral bacteria generated from dental procedures is unclear. A clinical study showed that airborne infections and tuberculosis occurred more often in dentists than in people in other occupations.[28] Dental students have also been reported to

contract more tuberculosis than medical students.[28] Nonetheless, no epidemiologic study on direct transmission of tuberculosis to dental workers from contaminated aerosols has been documented. Similarly, there is no evidence that hepatitis B virus or human immunodeficiency virus infection can be transmitted through aerosol inhalation. However, a new study has suggested that severe acute respiratory syndrome can be transmitted by aerosols.[29]

In 2000, Greco and Greene[30] found that most inanimate particles of aerosolized composite resin liberated during grinding are mainly 0.1 to 0.2 μm. These could impose hazardous health conditions for the orthodontist and staff, especially if combined with plaque and debris. They also found that airborne particle liberation secondary to the high speed of bonding resin, ceramic, glass ionomer cements, microetch material, and self-cured acrylic generates a persistent, respirable cloud near the operator and the patient. These particles are not filtered by the mucociliary defenses of the pulmonary tree.

Greco and Greene[30] also noted that the microetch particle sizes are definitely of respirable size (<10 μm). Dustless and "dusty" alginate use generates a respirable cloud that also persists for at least several minutes in tranquil air. These preliminary results showed that 2 commonly used clinical masks are ineffective in protecting the operators from particulate aerosols generated during routine orthodontic procedures; they do not block particles of less that 5 μm. This is a concerning finding because 10-μm particles can become lodged in the upper respiratory tract, and particles of 2.5 μm or smaller tend to be deposited in the terminal bronchioles and alveoli.[31]

In general, aerosolized bacteria appear to be composed of predominantly grampositive species. Some bacteria that have been implicated in airborne transmission in health care facilities include grampositive, group A streptococci, *S aureus*, *Neisseria meningitis*, and *Bordetella pertussis*. Toroglu et al[32] also found the most common microorganisms in the aerosol spray during orthodontic debonding procedures to consist of grampositive species such as streptococcus, neisseria, staphylococcus and others in 43%, 18%, and 17% proportions, respectively. In a subsequent study, they found that hepatitis B surface antigen and hepatitis B DNA were detected in the debonding aerosol of some but not all carriers.[32] Nonetheless, this study confirmed that the debonding procedure should always be considered a potential health hazard.

There has been little investigation about the effect of aerosols generated during orthodontic procedures on patients and professionals, however. Because fixed orthodontic appliances tend to interfere with conventional oral care, higher rates of intraoral pathogen colonization might result. The risk of disease transmission from infectious aerosols generated during orthodontic proce-

Fig 1. Porthole to accommodate high-speed handpiece was cut into 1 side of resuscitation mask, allowing movement of handpiece without loss of aerosol.

dures is reaffirmed when hepatitis B virus DNA in the aerosol sample from hepatitis B virus carriers during orthodontic debonding procedure was detected.[32]

We used a novel system developed to collect aerosolized bacteria generated during orthodontic debonding procedures. We assessed the presence of bioaerosols and subsequently liberated speciate bacteria during the removal of orthodontic appliances. Also, a pilot study of the protective efficacy of several commonly used dental masks was assessed.

MATERIAL AND METHODS

The following new procedure for bacterial sampling was used.

Twenty-three patients for this portion of the study were debonded in the private office of the principle investigator (P.M.G.). The bonding resin used was chemically cured composite adhesive (Reliance Phase II; Reliance Orthodontic Products, Itasca, Ill).

Patient exclusion criteria were no signs of respiratory infection, no antibiotic therapy at least 2 weeks before debanding, no steroid use during orthodontic therapy, no dental prophylaxis within 30 days preceding debanding, and at least 10 brackets per arch present.

A modified bleeding index was recorded before sampling. The patient's age and total number of teeth to be debonded were also recorded.

Baseline ambient air samples were collected in an enclosed room measuring 10 × 10.5 × 11.5 feet with a transparent vinyl resuscitation mask (CE 0086; Ace Surgical Supply, Brockton, Mass) connected to a 3- to 6-in sterile latex rubber surgical tube of 1/4 × 3/32-in diameter, fitted to an industrial air-suction pump (SKC Model 224-XR; SKC, Inc, Eighty Four, Pa). The mask had a porthole on its right side that intimately accommodated a high-speed handpiece (Fig 1). The hole was

Fig 2. Mask touched subject's face lightly and was held in position by operator.

cut to permit free rotation of the handpiece, yet allowed only minimal air to escape at the junction of the mask and handpiece. The pump was connected to a sterilized 0.22 μm millipore filter mounted in an ethylene oxide sterilized 37-mm cassette with support pad. Pump intake was calibrated at 4 mL per minute to simulate the average normal adult breathing flow rate. Two control samples were collected at the head of the dental chair in the same room that was used to debond the patient.

The brackets were removed from the teeth with standard orthodontic debonding pliers. The patient was not permitted to rinse but could expectorate after the brackets were removed.

The resuscitation mask contacted the subject's face lightly and was kept in position by the investigator (Fig 2).

The sampler was activated, and the remaining bonding resin was removed from the patient's teeth by using the high-speed handpiece operating at 30,000 rpm without suction or water coolant. A spiral fluted carbide finishing bur (FG #7901; SS White Burs, Inc, Lakewood, NJ) was used for all debonding procedures. The patient was instructed to remain silent during the entire procedure. The sampler was turned off as soon as bonding resin removal was completed. The total time of sampler operation was recorded.

The patient was asked to remain still for 10 minutes. The cassettes were sealed and transported to an off-site oral microbiologic laboratory. Each cassette was opened in the laboratory within 5 hours in a contained, aerobic chamber. Each filter was placed on a blood agar plate. The plates were inverted and aerobically incubated at 35°C for 4 days. The colonies were then identified by an oral microbiologist, aerobically incubated for another 2 to 4 days, and assessed with

dark-field microscopy for determination of morphotype and bright-field microscopy to observe gram-stained smears. Growth viability of the colonies was then determined. Microorganisms were identified and compared with the control sample to determine genus and species.

The first 11 samples were collected with the industry-approved technique for particulate sampling. This method used a 37-mm metallic cyclone (aluminum funnel) connected via a vinyl hose leading to an autoclaved 0.22-μm filter mounted in factory-sterilized plastic cassettes. These filters allowed only bacteria of 0.22-μm diameter or less to be retained for future culture and assay. It was difficult to determine whether the samples were contaminated because the autoclaved filter and cassettes, which were assembled before sampling by the investigator using a dental mask and gloved hands, became contaminated during handling. The cassettes could not be autoclaved without perishing because they could not tolerate the heat and pressure of the autoclave.

The rest of the samples were then collected by using cassettes with 0.22-μm filters that were placed into the cassettes before ethylene oxide sterilization. This was accomplished by arrangement with a hospital facility to ensure full sterilization. Sterilized rubber tubing replaced the vinyl tubing. Additionally, the collection cyclone was replaced by a modified vinyl resuscitation mask that was disinfected by thorough washing in antibacterial soap. The flexible resuscitation mask effectively adapted to the patient's lower facial contours during debonding.

After it was established that the collection technique was effective in harvesting airborne microbes, 2 commonly used dental masks, Fluid Resistant Molded Face Mask (3M ESPE Dental Products, St Paul, Minn) and Allegiance Caliber Brand Instagard Face Mask (Allegiance Healthcare, McGaw Park, Ill), were tested for efficacy in preventing bacteria from operator inhalation (Fig 3). Additionally, the industrial N-95 mask (MSA Affinity Plus, model #PL-200-2; Approved Gas Masks, Beltsville, Md) from a local hardware store, but also used by health care professionals treating severe acute respiratory syndrome and H5N1 avian influenza patients, was tested. The mask tests were accomplished by the placement of a 37-mm diameter mask remnant over the collection filter in the cassette for use. The assembly was then sterilized with ethylene oxide. A different mask type was used for each of the 3 subjects. Sampling was then conducted as listed above, by using a split-mouth design as follows.

All bands and bonds were removed with pliers. Half of the subject's mouth (right or left side, both maxil-

Fig 3. Protective capabilities of 2 commercial dental masks were tested.

lary and mandibular teeth) was then sampled during removal of bonding resin as previously described, by using the sterilized cassette (without mask remnant) prepared with the identical filter assembly as described above.

The first cassette was then removed, and a second sterilized cassette was attached to the mask and pump. This cassette contained 1 of the 3 commercial dental masks placed at the intake port of the cassette lumen so that incoming air passed through the mask material and then to the filter via the suction generated by the pump. Bonding resin was then removed with the handpiece at the other side of the mouth. This technique was intended to determine whether microbes were effectively trapped by the dental mask before reaching the sampling filter.

The cassettes were then sent to the laboratory for study of growth viability under anaerobic conditions

followed by inspection by the microbiologist to determine differences in quantity of bacteria colony formation with and without the mask material.

The collected samples were cultured on the same day as collection. Each filter was placed on a blood agar plate that was inverted and anaerobically incubated at 35°C for approximately 4 days. The agar plates were then removed from the incubator for inspection. The filter was removed, and the colonies were identified by an oral microbiologist using a 2-times magnification lens. The total number of each species type was recorded as the number of colony-forming units. The same procedure was repeated for all collected filters. Each representative colony was subsequently cultured onto another agar plate and reincubated for another 2 to 4 days for further microbiologic identification with the following testing methods: dark-field microscopy, gram-stain microscopy, speciation via chemical identification method, and growth viability under aerobic conditions. These 4 techniques are summarized below.

In dark-field microscopy, from a freshly grown 4-day culture, a single isolate was removed from the agar plate with a sterile loop and smeared onto a glass slide with 1 drop of distilled water. The slide was examined under a 1000-times dark-field microscope for bacterial morphotyping (eg, cocci, rod). All identifications were conducted by the same oral microbiologist.

In the gram-stain microscopy, from a freshly grown 4-day culture, a single isolate was removed from the agar plate and smeared onto a glass slide for gram staining. After the sample was dried, the cells were fixed and stained with crystal violet basic dye for 1 minute. This dye is absorbed by all bacteria in a similar manner. The slides were then treated for an additional minute with a mordant (I2-KI mixture) to fix the stain, washed briefly with 95% alcohol for 10 seconds to destain the sample, and then counterstained with a paler dye of different color (safranin) for 30 seconds. Grampositive organisms retain the initial violet stain, but gramnegative organisms are decolorized by the organic solvent and thus display pink counterstain. All bacteria slides were then examined under the oil immersion lens for gross identification of the species.

For growth viability under aerobic conditions, a single isolate was removed from the agar plate with a sterile loop and transferred onto another agar plate, which was then aerobically incubated at 35°C. Confirmation of growth was documented after 4 days of incubation.

In the chemical identification method, from a freshly grown 4-day culture, a single isolate was removed from the agar plate with a sterile loop and transferred into growth broth and subjected to a 24-hour

Table I. Sampling profile (excluding 3 patients involved in mask study)

Subject	Age (y)	Bonds removed (n)	Bands removed (n)	Bleeding index	Sampling time (min)
1	14	14	10	21	10
2	14	14	12	52	10
3	20	24	2	18	10
4	29	15	8	13	12
5	50	13	8	17	17
6	33	12	9	12	17
7	14	15	9	9	10
8	14	14	11	53	5
9	47	15	2	16	17
10	Control	0	0		10
11	Control	0	0		10
12	18	12	12	10	7
13	13	12	14	16	6
14	16	12	14	5	5
16	13	12	5	19	6
17	16	12	16	5	5
17B	13	12	10	15	5
18	56	15	4	1	7
19	36	16	4	6	6
20	14	12	14	7	5
21	66	16	6	18	7
22	18	12	10	24	6
23	15	12	15	25	4
24	14	13	9	24	4
25	Control	0	0		6
26	Control	0	0		6

Table II. Bacterial populations collected, including controls

Sample number	Species	% of species
1	*S haemolyticus*	50
	S capitis capit	25
	Propionibacterium acnes	25
2	*S warneri*	33.33
	S epidermidis	33.33
	S saprophyticus	16.67
	P acnes	16.67
3	*Actinomyces viscosus*	50
	S hyicus	25
	S aureus	25
4	*S cohnii-cohnii*	42.86
	A viscosus	21.43
	Streptococcus pneuomoniae	14.29
	S epidermidis	7.14
5	*S cohnii-cohnii*	40
	S warneri	20
	A viscosus	20
	Streptococcus pneumoniae	20
6	*S epidermidis*	50
	S hominis homin	50
7	*P acnes*	25
8	*S hominis novo*	50
	S warneri	50
9	*P acnes*	100
10 (control, colonies on edges only)	*S xylosus*	37.5
	S aureus	37.5
	P acnes	17.5
	Acinetobacter lwoffii	17.5
17	*Streptococcus mitis*	67
	A viscosus	33
17B	*A viscosus*	20
	S simulans	20
	S epidermis	20
	S auricularis	20
	Species	20
18	*S epidermis*	57
	A viscosus	27
	S aureus	10
	Streptococcus mitis	3
	Flavobacterium breve	3
19	*S aureus*	25
	S auricularis	25
	S cohnii-cohnii	25
	A viscosus	25
20	Unidentifiable G(+) anaerobic rods	100
	S hyicus	50
21	*S cohnii-cohnii*	37.5
	S auricularis	12.5
22	Negative (no growth)	0
23	*S cohnii-cohnii*	40
	S epidermidis	20
	Streptococcus milleri group	20
	P acnes	20
24	*S xylosus*	75
	Leuconostoc sp (cocci)	50

incubation period before chemical testing. Each species was subjected to a panel of wells (MicroScan: Rapid Anaerobe ID Panel and MicroScan: Pos Combo Panel Type 12; DADE Behring, West Sacramento, Calif) to increase identification accuracy. The sample was placed in each well, and the composite reaction of the sample generated a numeric code that was correlated to positive species identification.

RESULTS

Twenty-three patients were sampled (age range, 13–66 years) with the new collection method. Table I summarizes their ages, number of brackets removed, sample collection time, and bleeding indexes. The average age was 25.62 years, and the average number of bonds removed was 13.22 per patient. Total bonds removed were 304. Table II lists the aerosolized bacterial colonies collected from each patient.

Two ethylene-oxide sterilized cassettes (numbers 25 and 26) that served as controls operated under pump intake. Cassette 25 was exposed to the debanding room (operatory) ambient air only, and cassette 26 was attached to the resuscitation mask and worn in the op-

Fig 4. Control and experimental cassettes were indistinguishable in bacterial colony growth, with abundant colonization observed in each.

Fig 5. Industrial mask prevented colonization on agar plates, demonstrating protection from aerosolized bacteria.

eratory by the investigator to detect exhaled microbes. These cassettes were subjected to the same processes as all other cassettes and had no bacterial growth.

The 2 commercially marketed dental masks tested offered no protection from aerosolized bacteria liberated during orthodontic debonding. The control cassettes and the experimental cassettes were indistinguishable in terms of bacterial colony growth, with abundant colonization on each agar plate (Fig 4).

The industrial mask, however, prevented contamination and colonization of the plates, demonstrating effective protection from at least aerosolized oral bacteria (Fig 5).

DISCUSSION

Oral bacteria and pathogens have been implicated as potential sources of systemic infections. Improved methodology in collection of oral microbiota liberated during debonding procedures is needed to assess risks to both patients and clinicians. This investigation has shown that our sampling technique was effective and useful in collection of aerosolized bacteria.

With the sampling method described here, comparison of the control cassettes to the debond cassettes disclosed significant aerosolization of oral and pharyngeal bacteria. This finding agrees with Toroglu et al[32] and Ireland et al[31] In our investigation, the duration of the postdebonding bacterial cloud was not determined. Greco and Greene,[30] however, demonstrated that the particulate cloud persisted for more than 10 minutes after debonding, increasing the risks to clinician and patient. Although many species collected were nonpathogenic in situ, bacterial seeding of systems such as the respira-

tory tract and eyes can be pathogenic due to endotoxin production or compromised host defense.

Because of the numerous species in the oral and pharyngeal flora, relatively few species were identified. However, identification was conducted in a clinical oral microbiologic laboratory that is used to target periodontal pathogens, and thus identification techniques for many other pathogens were unavailable. It is logical to assume that additional species of bacteria, viruses, and other microbes were aerosolized but unidentified.

Several potentially pathogenic species aerosolized and isolated in this study were[33]: *Propionibacterium acnes:* causes uveitis and endophthalmitis; *Actinomyces viscosus:* 1 of several species causing actinomycosis, which involves chronic granulomatous lesions of the thoracic cavity; *Streptococcus mitis:* associated with subacute bacterial endocarditis; *S cohnii-cohnii:* causes ophthalmia; and other staphylococcus species that cause many human infections.

The wide age range of the subjects (13-66 years; mean, 25.62 years) might be related to the variation of bacterial florae identified in this study. Mask efficacy was found to be variable. Classification of mask efficacy is with particle filtration efficiency (PFE) or bacterial filtration efficiency (BFE). PFE ratings refer to the mask's ability to trap particles with a fixed nonviable particle size of 0.1 to 1.0 μm. BFE ratings are correlated to the mask's ability to trap viable particles of 1 to 5 μm. The catalogs of 2 large dental supply companies showed 61 and 70 different clinical masks. Although the cost per mask varied between $ 0.20 to $1.09, there was no correlation between PFE or BFE and mask cost. Other dental supply catalogs list masks predominantly by BFE. It is doubtful whether many dental practitioners are aware of PFE and

BFE ratings as measures of mask efficacy. The masks in this study were those used in the principal investigator's office, because of familiarity with the manufacturer's name and advice of supply representatives. Based on our results, masks with PFE or BFE of at least that of the N-95 are advisable (95% filtration rate at 0.3 μm). PFE and BFE ratings and the efficacy values we determined are independent of mask adaptation. Facial hair and the adaptation of the mask to the operator's face also affect protection levels.[34] Finally, mask users must be aware that viruses are significantly smaller than 0.1 to 5.0 μm and are commonly aerosolized.[26] Thus, clinicians should consider the use of high-speed suction during all debonding procedures, but reduction of aerosolized viruses by high-speed suction has yet to be verified.

CONCLUSIONS

A new method of bacterial sampling was introduced to collect aerosol generated during orthodontic debonding procedures and will be used in future studies of airborne aerosols. The diameters of the bacterial species collected by this technique are sufficiently small to be inhaled and deposited in the alveolar spaces of patients and clinicians. In a pilot study of 2 brands of commonly used dental masks, the clinician remained unprotected from aerosolized bacteria because of the mask pore size exceeded the bacterial diameter. An industrial mask was effective in preventing transmission of aerosolized bacteria. Clinicians should choose a protective mask with the most efficient PFE and BFE rating and the best facial adaptation possible. Further mask efficacy study is ongoing.

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