Received: 22 May 2020 | Revised: 22 June 2020 | Accepted: 23 June 2020

DOI: 10.1002/JPER.20-0395

JOURNAL OF Periodontology

REVIEW

Demystifying the mist: Sources of microbial bioload in dental aerosols

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Abstract

The risk of transmitting airborne pathogens is an important consideration in dentistry and has acquired special significance in the context of recent respiratory disease epidemics. The purpose of this review, therefore, is to examine (1) what is currently known regarding the physics of aerosol creation, (2) the types of environmental contaminants generated by dental procedures, (3) the nature, quantity, and sources of microbiota in these contaminants and (4) the risk of disease transmission from patients to dental healthcare workers. Most dental procedures that use ultrasonics, handpieces, air-water syringes, and lasers generate sprays, a fraction of which are aerosolized. The vast heterogeneity in the types of airborne samples collected (spatter, settled aerosol, or harvested air), the presence and type of at-source aerosol reduction methods (high-volume evacuators, low volume suction, or none), the methods of microbial sampling (petri dishes with solid media, filter paper discs, air harvesters, and liquid transport media) and assessment of microbial bioload (growth conditions, time of growth, specificity of microbial characterization) are barriers to drawing robust conclusions. For example, although several studies have reported the presence of microorganisms in aerosols generated by ultrasonic scalers and high-speed turbines, the specific types of organisms or their source is not as well studied. This paucity of data does not allow for definitive conclusions to be drawn regarding saliva as a major source of airborne microorganisms during aerosol generating dental procedures. Well-controlled, large-scale, multi center studies using atraumatic air harvesters, open-ended methods for microbial characterization and integrated data modeling are urgently needed to characterize the microbial constituents of aerosols created during dental procedures and to estimate time and extent of spread of these infectious agents.

KEYWORDS
evidence-based dentistry, infection control, microbiology, saliva

1 | INTRODUCTION

Aerosols, especially those created during dental procedures, have recently taken front and center stage in the news, driven by fears of transmission of SARS-CoV-2 virus. However, COVID-19 is just the latest episode of a century-long assault of the human race by zoonotic respiratory viruses, and a much longer assault by several respiratory

bacterial pathogens; tuberculosis, and bacterial pneumonia, to name but a few. The proximity of the nasopharynx and lower respiratory tract to the oral cavity creates an open communication channel for movement of viruses and bacteria from these areas into the mouth. In this scenario, aerosol generating dental procedures on patients with infectious respiratory diseases become sources of contagion. In an immunocompetent individual, the risk of spread of infection by aerosolized particles is largely driven by the kinetics of the aerosol, presence of pathogen in the aerosol source, the type of pathogen, frequency of exposure, and the infectious dose.

As dental professionals, it behooves us to protect ourselves, our patients and our staff from occupationally acquired diseases. The purpose of this review, therefore, is to examine what is currently known regarding the physics of aerosol creation, the types of aerosols generated by dental procedures, the nature, quantity, and sources of microbiota in these aerosols and the probability of disease transmission from patients to dental healthcare workers.

2 | THE CHARACTERISTICS OF AEROSOLIZED PARTICLES

In an attempt to establish context for reviewing the literature on dental aerosols, we begin this review by examining the reasons why definitions of aerosols vary widely. In general, aerosols refer to particles suspended in gas. Although aerosols may be generated from a multitude of events, such as combustion, evaporation, industrial work etc., we will focus on aerosols generated in the healthcare environment.

In 1934, Wells pioneered the concept that airborne infections can be transmitted either as droplets or as aerosols.[1] According to his work, droplets are defined as those with particle sizes > 5 μm and typically carried on heavy colloids like mucus or saliva. Droplets cannot remain suspended in air for long or travel long distances, hence, they are spread by close contact with (typically 1 m) and in the presence of, the host. However, according to Wells, droplets < 100 μm dry out before falling ≈2 m to the ground. When these droplets evaporate, they can be carried on airborne vectors and become aerosols. He estimated the particle size in aerosols to be < 5 μm (sometimes called droplet nuclei) and stated that these particles can stay airborne for long periods of time, carry viable pathogen as payload and settle on surfaces distant from the source (which is then referred to as a fomite). The vectors can be natural, namely, mist, fog, and vapor or anthropogenic, for example, smoke, dust, smog, and of particular importance to us, dental aerosol. However, in certain cases, for example, high ambient temperature or high airflow, large droplets can evaporate and acquire aerosol-like properties. Because of their size, they can carry larger payloads than droplet nuclei (see below).

Aerosols have also been classified based on their deposition patterns. For example, using a semi-empirical model, the International Commission on Radiological Protection (ICRP) estimated that particles between 1to 10 μm or < 0.5 μm are most likely to deposit in the tracheobronchial and pulmonary regions of the lungs, whereas particles ≤5 μm have the highest probability of entering the lower airways of the average adult during oral inhalation.[2] Because the nose offers a greater filtration efficiency than the mouth, only particles ≤3 μm have a high probability of entering the lower airways during nose breathing. Particles with diameters between 1 and 3 μm or <0.5 μm have the greatest probability of entering the lung, thereby the highest potential of initiating an infection at this site. The Infectious Diseases Society of America (IDSA) has defined "respirable particles" as having a diameter of ≤10 μm and "inspirable particles" as having a diameter between 10 μm and 100 μm, nearly all of which are deposited in the upper airways.[3] Other studies on infectious disease transmission indicate that droplets >5 μm are trapped in the upper respiratory tract whereas droplets ≤5 μm can be inhaled into the lower respiratory tract.[4] In this review, we will use the 10 μm diameter to distinguish between aerosolized and non-aerosol particles, because they have important implications for time of settling, penetration depth into airways and requirements for PPE.

Another important characteristic of aerosolized particles that impacts their definition is settling time. In still air, it has been estimated that particles 0.5 μm take 41 hours to settle over a distance of 5 feet, and that the time exponentially decreases as the size increases. For example, 1 μm sized particles take 12 hours to settle whereas 10 μm take 8.2 minutes and 100 μm take a mere 5.8 seconds.[5] However, this characteristic is heavily influenced by the direction and velocity of air currents (such as those created by foot traffic, opening of doors, position and setting of room air circulation systems etc.), humidity, the forces of attraction/repulsion between aerosolized particles and the size of the agglomerates/coaggregates (see below). In the presence of turbulence, particles nearer the floor continue to follow the settling times described above, but other factors begin to influence those that are two feet or more above the surface, for example, particle impaction, electrostatic forces etc.

When vector particles and aerosol droplets collide with each other, they might coalesce or coaggregate, changing the particle size, in which case, the classifications described above do not apply anymore. In certain situations, these aggregates break down into numerous smaller conglomerates, generating a new generation of payload. Together, these collisions randomly create a heterogeneous

mixture of large and small particles with highly variable electrical charges, aerodynamic diameter, diffusion dynamics, and terminal velocity.[6] It is therefore unsurprising that, in real life scenarios, each aerosol responds in a highly variable manner to gravitational forces. Temperature and humidity of the environment, and the superimposition of new aerosol further impact aerosol dynamics.[6]

The characteristics and behavior of aerosolized particles are important determinants of defining an aerosol, and for this reason, definitions have to be contextualized. For example, size and penetrability-based definitions have important implications for selecting appropriate face masks, while settling-characteristics-based definitions are impactful in deciding nature and time of surface decontamination. Hence, studies on aerosol transmission must account for these confounding variables in order to be interpreted in the appropriate clinical context. As we shall see below, most studies on aerosol generating medical/dental procedures (AGM/DP) have used simplistic calculations, for example, estimating particle size to compute aerodynamic diameter (this has limited use outside of regular sized particles such as inhalable drugs) and applying Stokes' law to calculate terminal velocity of a particle in a fluid (the assumptions of Stokes' law fail for particles <1 μm).[6]

3 | METHODS TO INVESTIGATE AEROSOLS

One of the most important considerations in any study is the investigational methodology. Early studies employed impaction on solid and liquidized interfaces to measure aerosol volume and properties.[7,8] Advances in visualization technology have enabled greater temporal and spatial visualization of aerosol generated particles and their trajectories. Among the various methodologies used to visualize aerosols, laser capture imaging, particle counters, air samplers, and droplet capture methods are the most popular.[9] Similarly, methodologies for microbial characterization have demonstrated tremendous advances from the early days of culturing and microscopy to targeted methods such as polymerase chain reaction (PCR) to quantitative PCR to collectively sequencing entire microbial communities.[10-12] A third component is development of computational models of human behavior and predicting patterns and paths of spread.[13]

Although these advances in pathogen detection, airflow measurement, and disease modeling have had a major impact on understanding the spread of diseases such as Ebola[14] and changed our perception of older diseases such as tuberculosis and measles,[15] several questions still remain to be addressed. For instance, although molecular microbiology has allowed us to identify infectious agents earlier and at much lower concentrations, it is not unclear if these doses are clinically relevant, how the relevance is modified by the type of populations (adult versus children, immunocompetent versus compromised, ambulatory versus hospitalized, and individual versus group living) and most importantly, how many of these organisms are viable.[16] Similarly, the very act of air-sampling can generate an aerosol as well as destroying the organisms being captured.[17] Importantly, computer machine learning relies on large and granular datasets for accuracy, and when studies from the field are unable to capture all the required components, the model is not reflective of real-life scenarios.

Thus, any investigation of aerosol characteristics must use well-validated methods of aerosol capture, incorporate appropriate positive and negative controls to allow standardization of microbial payload, and be sufficiently powered to reduce the "noise" generated by random behavior of aerosol particles. Most importantly, they must be quantitative, because pathogen dose is an important element of infectivity. As we will see in the next few sections, much of what we currently know about dental aerosols falls far short of the most basic principles of scientific rigor and reproducibility.

4 | THE ORAL CAVITY AS A RESERVOIR FOR VIRUSES IN HEALTH AND DISEASE

Until recently, the viral constituents of the oral microbiome had only been examined in the context of their ability to cause disease and spread contagion. We now know that viruses are normal inhabitants of the healthy oral microbiome,[18,19] and that a diverse population of both DNA and RNA viruses is found in saliva and subgingival plaque of healthy individuals.[20] The most common oral viruses are cytomegalovirus, herpesvirus one through nine and papilloma virus.[21] The types of viruses that inhabit an individual are highly subject-specific, much more so than the types of bacteria.[22] The oral virome also demonstrates significant gender-specificity.[19] The type of viral exposure an individual has had, and the nature of the shared living environment are two major determinants of individual viral signatures.[23] It is also established that the majority of viral particles are derived from gram-positive and gram-negative bacteriophages rather than free-living viruses.[18] Once acquired, these viruses demonstrate remarkable colonization stability in the absence of extraneous influences such as local or systemic disease.[22] Studies exploring the role of saliva as a diagnostic tool for viral diseases such as dengue, West Nile, SARS, chikungunya, MERS-CoV, Ebola, Zika,

and Yellow Fever have further expanded our knowledge of non-oral viruses.[24] Most of these investigations have reported that whereas viral RNA and viable virus were detected in saliva early in the course of disease, viral shedding did not persist after resolution of symptoms.[25,26] However, influenza A and B were detected in 20 to 60% of asymptomatic individuals.[27] Taken together, these studies suggest that (a) the oral viral community is acquired through a non-random process of microbial assembly that is partly dictated by individual genotype (b) viral communities are temporally stable once acquired and (c) exogenous viruses are present in saliva during acute phase infection, but most do not persist following resolution of disease.

5 | THE ORAL CAVITY AS A HOST FOR RESPIRATORY BACTERIAL PATHOGENS

Like viruses, respiratory bacterial pathogens have been detected in saliva during acute and symptomatic phases of respiratory illnesses,[28,29] as well as in institutionalized and hospitalized, elderly individuals.[30,31] However, unlike viruses, certain bacterial respiratory pathogens have been identified in the oral cavities of systemically healthy and asymptomatic individuals, especially smokers.[32,33] For instance, bacteria such as *Streptococcus pneumoniae* can be isolated more frequently and consistently from saliva than from naso-pharyngeal or oro-pharyngeal swabs.[34] These pathogens are known to reside in the subgingival crevice, the buccal mucosa and saliva.[28,35-39]

However, exogenous pathogens are not dominant members of the oral microbiome, which is one of the most diverse in the human body with over 20 billion microbial cells.[40] Moreover, in states of health, a robust interbacterial interaction limits or reduces colonization with exogenous pathogens. For instance, bacteriocins such as LS1 (produced by the oral commensal *Lactobacillus salivarius)* contribute to controlling the growth of *S. aureus* and *S. pneumoniae*,[41,42] and hydrogen peroxide (which is produced by several commensal species) prevents colonization by *Serratia marcescens*, *S. agalactiae*, *S. pneumoniae*, *Haemophilus influenzae* and MRSA.[43,44]

In summary, a large body of evidence supports saliva as a potential source of respiratory pathogens, however, many of these studies lack quantitative data. Therefore, there is an urgent need for studies that quantify the salivary bioload of these species in non-infected individuals and for investigations on whether these microbial loads are high enough to create a biologically relevant infectious dose.

6 | AEROSOL GENERATION DURING PHYSIOLOGICAL ACTIVITY

Although AGM/DP have been implicated in spread of viral contagion, it must be remembered that aerosols are generated during normal physiological activities such as breathing, talking, coughing, and sneezing. Studies on healthy volunteers have demonstrated that mouth breathing produces 1-98 particles per liter,[45] with a median diameter of 0.3 μm; with only about 2% of the particles >1μm and none > 5 μm.[46,47] During speaking, 1 to 50 particles in the 1 μm range are emitted per second (0.06 to 3 particles per liter)[48]; with some "super-seeders" expelling as many at 200 particles per second while speaking loudly. Singing creates six times as many droplet nuclei as talking and is equivalent to coughing.[49] Sneezing can expel nearly 40,000 droplets between 0.5 to 12 μm at speeds of almost 100 m/sec, while coughing may generate up to 3000 droplet nuclei.[50,51] Collectively, studies such as these demonstrate that healthy individuals generate particles sized between 0.01 and 500 μm, underlining the fact that dispersal of expelled particles does not occur exclusively by airborne or droplet transmission but by both mechanisms concurrently.

Although healthy and diseased individuals generate aerosols during normal activities, evidence that these aerosols contain an infectious agent is equivocal. For example, *Streptococcus pneumoniae, Staphylococcus aureus, Methicillin-resistant Staphylococcus aureus* (MRSA)*, Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Acinetobacter baumannii, Stenotrophomonas maltophilia, Haemophilus influenzae, Legionella pneumophila, Mycoplasma pneumonia, Chlamydia pneumonia, and Mycobacterium tuberculosis* can be detected in 36% of patients with symptomatic respiratory diseases.[10] However, although 89% of nasal swabs were positive for live virus in 142 patients diagnosed with influenza A, only 39% of individuals exhaled live viral particles in their breath,[52] and the number of particles shed declined significantly within 3 days of onset of symptoms.[12,52] Importantly, these particles failed to land on targets placed at a distance of 0.1 and 0.5 m.[53] Furthermore, when a patient wore a surgical mask, it reduced the viral shedding in aerosol by 3.4 fold.[54] On the other hand, *P. aeruginosa* can travel 4 m and persist in the aerosol for 45 minutes subsequent to a coughing episode.[11] Wearing surgical masks for 10 to 40 minutes reduced the levels of respiratory pathogens by more than four-fold.[55]

Collectively, there is a large body of evidence that patients in the acute phase of respiratory infections are capable of disseminating large numbers of airborne microorganisms during activities such as breathing, talking, singing, coughing, and sneezing. This shedding can be

mitigated by the simple act of wearing a mask and is effective against viral as well as bacterial pathogens.

7 | AEROSOL GENERATING MEDICAL AND DENTAL PROCEDURES (AGMP AND AGDP)

The SARS-1, 2009 H1N1 MERS, Ebola and Zika outbreaks were instrumental in drawing attention to medical aerosols as sources of infection to health-care personnel. Two broad categories of AGMP have been documented in the literature: those that induce the patient to express the contents of the lower respiratory tract by stimulating cough reflex (sputum induction), and those that mechanically disrupt the contents of the respiratory tract. The latter procedures typically include intubation/extubation, cardiopulmonary resuscitation, bronchoscopy, noninvasive ventilation, tracheotomy, airway suctioning, manual ventilation, and administering oxygen or nebulized medication.[56] All these procedures are conducted on patients who are typically experiencing active disease, and therefore, the aerosols and droplets generated from sites with active pathogen colonization could potentially contain high numbers of respiratory pathogens. However, even though MAGP have been the subject of at least 400 different studies, questions still remain regarding the amount of aerosols generated, the size and concentration of medically aerosolized particles, and whether such aerosols could transmit viable pathogens to HCP or to other patients. For instance, the review by Davies et al.[56] and by O'Neil et al.[57] suggests that although the potential for aerosol production exists with AGMP, there is little evidence that these procedures actually do create aerosols.

During dental procedures, the "wet environment" created by saliva and water coolant combined with high-speed instrumentation generates a large spray which disperses in many forms as dictated by the physics of aerosol creation (see section on characteristics of aerosols, above). Thus, the spray can initially take the form of spatter, droplets, droplet-nuclei, a true aerosol, or some combination thereof; and continue to evolve based on room temperature, humidity, airflow dynamics, electrostatic forces etc. The term "dental aerosol", therefore, is somewhat of a misnomer, because it does not encompass the various airborne particles that can be created during an AGDP. To avoid confusion, we will use the word spray unless the study specifically measured aerosols.

There are four main sources of dental sprays: air-water syringes, ultrasonic instruments, high-speed turbines, and lasers. There is no literature on sprays from air-water syringes, so we will examine the evidence from the rest of instruments below.

7.1 | Ultrasonic instrumentation

The quantity of sprays, spatter, or aerosol generated by ultrasonics, the distance travelled by the aerosolized particles and their composition have been studied using air samplers,[58-60] bacterial growth medium placed at strategic locations,[58-67] filter paper strips (with and without dye) on the patient and operator,[68,69] and heme-detectors.[70] Sprays are generated during all types of procedures using ultrasonic instruments, whether it be supragingival scaling, subgingival scaling of periodontally diseased teeth or endodontic instrumentation. The amount of spatter and aerosol generated by sonic, ultrasonic or piezoelectric devices and distance travelled by airborne particles from these devices is similar or comparable.[58,64,71,72] These sprays expose the inhabitants of the operatory to 1.86×10^5 particles per cubic meter of space, and the contaminants settle to a great extent on the dominant arm of the operator, and eyewear and chest of the patient and to a lesser extent on the non-dominant arm and chest of the operator and assistant.[66,68,69,73] They can also be detected as far away as 2 to 11 m from the treatment site.[59,66] However, in the absence of a coolant, the aerosol is limited to an 18 inch radius.[72] The levels of aerosolized particles return to preoperative levels within 30 minutes[68] to 2 hours.[60] In summary, there is unequivocal evidence that some of the spray from all types of ultrasonic devices is converted to aerosol, and while the spatter settles on the person of the operator, assistant and patient, the aerosolized particles can travel much larger distances and settle up to 2 hours after creation.

7.2 | High-speed handpiece

High speed handpieces can generate spatter containing blood and other components,[59,61,63,74,75] and the amount of microbial bioload varies with the tooth being treated.[74] as well as the caries level of the patient.[65] It has been reported that microbial fallout from restorative procedures can extend up to 1.5 to 2 m, however, this study did not report the type of evacuators that were used during the procedures.[76]

7.3 | Laser instrumentation

When a laser is used to cauterize blood vessels and incise tissue by vaporization, it generates a gaseous material known as surgical smoke plume, which is composed of 95% water. The remaining 5% has been reported to contain blood, particulate and microbial matter.[77] The particle

size generated by lasers ranges from 0.1 to 2 μm. All Class IV lasers (surgical lasers) carry the risk of plume hazard. Although there is no evidence on lasers used in dental operatories, *Escherichia coli, Staphylococcus aureus*, human papillomavirus, human immunodeficiency virus, and hepatitis B virus have been detected in surgical laser plumes used in dermatology and otolaryngology.[78]

8 | IS SALIVA THE PRIMARY SOURCE OF PATHOGENS IN DENTAL AEROSOLS?

Although every single study to date has demonstrated that all forms of mechanical instrumentation in the oral cavity create aerosols and spatter with a significant bioload, critical gaps in knowledge still exist. The first of these is the source of the aerosolized microbiota. It is easy to point to saliva as a source. If this were indeed true, then one would expect a high degree of variability in the clinical studies because of differences in salivary volume, flow rate and composition between patients. However, all the literature detailed in this review report remarkably homogeneous findings in terms of aerosol volume, quantity of contagion, and distance and time of spread. This is in spite of variability in operators, instruments, procedures, subject characteristics, and data collection methods.

Moreover, if saliva were the source of microbiota in dental aerosols, one would expect a certain level of microbial heterogeneity between the studies. However, the bacteria most frequently identified in all studies were *Staphylococcus aureus*, beta hemolytic *Streptococci, Escherichia coli*, spore-forming bacteria, fungi belonging to the genera *Cladosporium* and *Penicillium*, and *Micrococccus*[66,79-81]; all of which are environmental species. In parallel, a study of dental unit water reservoirs revealed the presence of *Staphylococcus aureus*, beta hemolytic *Streptococci, Escherichia coli, Ralstonia pickettii, Sphingomonas paucimobilis, Brevundimonas vesicularis, Moraxella lacunata, Moraxella* spp., *Stenotrophomonas maltophilia, Micrococcus luteus, Micrococcus lylae, Staphylococcus cohnii, Staphylococcus hominis ss novobiosepticus, Staphylococcus* spp., *Streptococcus* spp.; actinomycetes, and *Streptomyces albus*.[82] Another study documented the presence of high levels (10^5 CFUs) of *Legionella, Pseudomonas* and non-tuerculous mycobacteria in water lines.[83] Thus, there is plausible evidence to suggest that water might contribute to a large fraction of the microbial payload in dental aerosols.

This plausibility is further supported by the fact that ultrasonic devices and high-speed handpieces use water as a coolant with a typical flow rate of 10 to 40 mL per minute,[84] whereas the flow rate of saliva during the same time period is 0.4-0.5 mL.[85] Thus, the dilution ratio varies between 1:20 to 1:100. That is not to say that saliva does not contribute to the microbial payload in aerosols. In fact, a strong correlation was observed between the number of decayed teeth in a patient and the levels of beta hemolytic streptococci on the operator's mask,[65] and reductions in aerobic and anaerobic colony forming units (CFUs) have been reported following pre-procedural mouth rinsing.[65,86-88] However, as described above, most the culturable bacteria identified thus far in dental aerosols are of environmental origin, bacterial profiles in aerosols demonstrate remarkably low "noise" between studies and the dilution factor because of water coolants is very high. In the absence of evidence demonstrating a salivary source for these bacteria, the microbial similarities between water lines and aerosols is the only evidence that can be brought to bear upon this argument.

Although the amount to effort invested in studying dental aerosols is commendable, these studies suffer from critical flaws in design and methodology that preclude robust decision making.[8] For instance, none of the studies used a control group where the aerosol was generated in the absence of a patient. This would provide invaluable information on the source of the microbial payload. There is also incredible diversity in the methodologies used. For instance, several studies originating from the Indian subcontinent and South East Asia have not used any form of aspiration of oral fluids, whereas most studies from Europe and the United States have used high or low volume aspirators. Because the amount of aerosol directly correlates with the partial pressure of fluid in the mouth, this important variable does not allow for comparisons to be made between studies. Perhaps the most important gap in knowledge stems from the use of rudimentary cultivation-based approaches to characterize microbiota. Such approaches have created very simplistic views of the microbial contaminants (e.g. gram positive versus gram negative, gross counts of CFU, catalase activity, and other such basic characterizations), have hampered our ability to pinpoint the source of the aerosol and completely ignored the viral, fungal and other constituents of the microbial payload. Hence, these studies have allowed room for liberal interpretation of the data, and in some instances, this has served to create a certain level of misinformation.

9 | DISEASE TRANSMISSION TO DENTAL HEALTHCARE PERSONNEL AND PATIENTS

Before we examine the statistics on cross-infection in dental settings, it must be acknowledged that lack of reporting poses a huge barrier to obtaining accurate data. An excellent review by Volgenant et al.[89] examines the

several potential routes of transmitting infections in the dental office. These include blood-borne, contact, and aerosol transmission. Several instances of transmission of blood-borne pathogens to patients and health-care personnel have been documented. These are attributable both to poor infection control practices,[90] as well as to blood exposure accidents.[91] However, the risk appears to be very low, with only five cases reported between 2003 and 2016.[92] Aerosol-transmitted diseases have been documented, although dental unit water lines appear to be the microbial source.[93,94] Especially, legionellosis has been connected to dental treatment in two case reports.[95,96] Moreover, dentists in certain areas have been shown to have higher antibody levels to *Legionella* when compared to non-dental professionals,[97] adding further credence to dental unit water lines as the source of aerosol microorganisms.

10 | SUMMARY AND CONCLUSIONS

A careful and contextualized review of the currently available evidence on dental aerosols reveals the following:

1. Viral shedding occurs in saliva during acute phases of all respiratory diseases, and influenza viruses have been reported in post-recovery and asymptomatic patients.
2. Respiratory bacterial pathogens are present in saliva of asymptomatic individuals; however, their relative abundances are very low.
3. Aerosols are generated by all individuals during all times of the day during all types of activities.
4. The microbial payload in physiological aerosols correlates with disease severity for respiratory diseases.
5. Aerosols are created during most dental procedures. The four main aerosol emitting devices are ultrasonics, handpieces, air-water syringes, and lasers.
6. There is little evidence to definitively implicate saliva as the primary source of bacteria in these aerosols. Although absence of evidence is not evidence of absence, the available evidence currently points to environmental sources, particularly dental unit water lines, as a major basis of aerosol bacteria in the dental environment.

Large-scale, multi center studies using atraumatic air harvesters and integrated data modeling that is superimposed on a geographic map of the physical space have enabled the medical community to identify patterns of aerosol spread, model disease transmission, and create human and instrument flow paths to reduce risk of infection.[98] Similar studies to determine the creation and spread of aerosols during dental procedures and to estimate time and extent of spread are urgently needed.

AUTHOR CONTRIBUTIONS

Purnima S. Kumar and Kumar Subramanian contributed equally to the literature review, and writing and reviewing the manuscript.

CONFLICT OF INTEREST

The authors have no conflicts of interest or financial relationships impacting this manuscript.

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REFERENCES

1. WELLS WF. ON AIR-BORNE INFECTION*: STUDY II. DROPLETS AND DROPLET NUCLEI. *Am j epidemiol* 1934;20:611-618.
2. International Commission on Radiological Protection. Human respiratory tract model for radiological protection. *Ann ICRP* 1994;66:1-3.
3. *Institute of Medicine. Preventing Transmission of Pandemic Influenza and Other Viral Respiratory Diseases: Personal Protective Equipment for Healthcare Personnel: Update 2010. In:* 2 Understanding the risk to healthcare personnel. Washington, DC: The National Academies Press, 2011.
4. Atkinson J, Chartier Y, Pessoa-Silva CL. Natural ventilation for infection control in health-care settings. In: atkinson J, Chartier Y, Pessoa-Silva CL. Geneva: World Health Organization. 2009.
5. Baron P. Generation and behavior of airborne particles (aerosols). In: powerpoint presentation. US Department of Health and Human ServicesCenters. 2010.
6. Hinds WC. Aerosol technology: properties, behavior, and measurement of airborne particles. 1999.
7. EICHENWALD HF, Kotsevalov O, Fasso LA. The cloud baby: an example of bacterial-viral interaction. *Am J Diss Child*. 1960;100:161-173.
8. Gralton J, Tovey E, McLaws M-L, Rawlinson WD. The role of particle size in aerosolised pathogen transmission: a review. *J Infect*. 2011;62:1-13.
9. Tang JW. Investigating the airborne transmission pathway – different approaches with the same objectives. *Indoor Air*. 2015;25:119-124.
10. Zheng Y, Chen H, Yao M, Li X. Bacterial pathogens were detected from human exhaled breath using a novel protocol. *J Aerosol Sci*. 2018;117:224-234.
11. Knibbs LD, Johnson GR, Kidd TJ, et al. Viability of pseudomonas aeruginosa in cough aerosols generated by persons with cystic fibrosis. *Thorax*. 2014;69:740-745.
12. Hatagishi E, Okamoto M, Ohmiya S, et al. Establishment and clinical applications of a portable system for capturing influenza viruses released through coughing. *PLoS One*. 2014;9:e103560.
13. Choi JI, Edwards JR. Large-eddy simulation of human-induced contaminant transport in room compartments. *Indoor Air*. 2012;22:77-87.
14. Leffel EK, Reed DS. Marburg and Ebola viruses as aerosol threats. *Biosecur Bioterror*. 2004;2:186-191.

15. Urbanowski ME, Ihms EA, Bigelow K, Kübler A, Elkington PT, Bishai WR. Repetitive Aerosol Exposure Promotes Cavitary Tuberculosis and Enables Screening for Targeted Inhibitors of Extensive Lung Destruction. *J Infect Dis*. 2018;218:53-63.
16. Tang JW, Li Y, Eames I, Chan PKS, Ridgway GL. Factors involved in the aerosol transmission of infection and control of ventilation in healthcare premises. *The J hosp infect*. 2006;64:100-114.
17. Verreault D, Moineau S, Duchaine C. Methods for sampling of airborne viruses. *Microbiol Mol Biol Rev*. 2008;72:413-444.
18. Pride DT, Salzman J, Haynes M, Rohwer F, Davis-Long C, White RA. Evidence of a robust resident bacteriophage population revealed through analysis of the human salivary virome. *ISME J*. 2012;6(5):915-926.
19. Pride DT, Sun CL, Salzman J, et al. Analysis of streptococcal CRISPRs from human saliva reveals substantial sequence diversity within and between subjects over time. *Genome Res*. 2011;21:126-136.
20. Pérez-Brocal V, Moya A. The analysis of the oral DNA virome reveals which viruses are widespread and rare among healthy young adults in Valencia (Spain). *PLOS ONE*. 2018;13: e0191867.
21. Dabdoub SM, Ganesan SM, Kumar PS. Comparative metagenomics reveals taxonomically idiosyncratic yet functionally congruent communities in periodontitis. *Scientific Reports*. 2016;6:38993.
22. Abeles SR, Robles-Sikisaka R, Ly M, et al. Human oral viruses are personal, persistent and gender-consistent. *ISME J*. 2014;8:1753-1767.
23. Robles-Sikisaka R, Ly M, Boehm T, Naidu M, Salzman J, Pride DT. Association between living environment and human oral viral ecology. *ISME J*. 2013;7:1710-1724.
24. Niedrig M, Patel P, El Wahed AA, Schädler R, Yactayo S. Find the right sample: a study on the versatility of saliva and urine samples for the diagnosis of emerging viruses. *BMC Infect Dis*. 2018;18:707.
25. Vetter P, Fischer WA, 2nd, Schibler M, Jacobs M, Bausch DG, Kaiser L. Ebola virus shedding and transmission: review of current evidence. *J Infect Dis*. 2016;214:S177-S184.
26. Korhonen EM, Huhtamo E, Virtala AM, Kantele A, Vapalahti O. Approach to non-invasive sampling in dengue diagnostics: exploring virus and NS1 antigen detection in saliva and urine of travelers with dengue. *J Clin Virol*. 2014;61:353-358.
27. Suda Y, Nagatomo M, Yokoyama R, et al. Highly sensitive detection of influenza virus in saliva by real-time PCR method using sugar chain-immobilized gold nanoparticles; application to clinical studies. *Biotechnol Rep (Amst)*. 2015;7:64-71.
28. To K, Yip C, Lai C, et al. Saliva as a diagnostic specimen for testing respiratory virus by a point-of-care molecular assay: a diagnostic validity study. *Clin Microbiol Infect*. 2019;25:372-378.
29. Y-g Kim, SG Yun, Kim MY, et al. Comparison between saliva and nasopharyngeal swab specimens for detection of respiratory viruses by multiplex reverse Transcription-PCR. *JClin Microbiol*. 2017;55:226-233.
30. Tada A, Shiiba M, Yokoe H, Hanada N, Tanzawa H. Relationship between oral motor dysfunction and oral bacteria in bedridden elderly. *Oral Surg, Oral Mede, Oral Pathol, Oral Radiol, Endod*. 2004;98:184-188.
31. Tada A, Hanada N, Tanzawa H. The relation between tube feeding and pseudomonas aeruginosa detection in the oral cavity. *J Gerontol A, Biol Sci Med Sci*. 2002;57:M71.
32. El Ahmer OR, Essery SD, Saadi AT, et al. The effect of cigarette smoke on adherence of respiratory pathogens to buccal epithelial cells. *FEMS Immunol Med Microbiol*. 1999;23:27-36.
33. Mason M, R PreshawP, M NagarajaH, N DabdoubS, M RahmanA, Kumar P. S. The subgingival microbiome of clinically healthy current and never smokers. *Isme J*. 2015;9:268-272.
34. Krone CL, Wyllie AL, van Beek J, et al. Carriage of streptococcus pneumoniae in aged adults with influenza-like-illness. *PLOS ONE*. 2015;10:e0119875.
35. Heo SM, Sung RS, Scannapieco FA, Haase EM. Genetic relationships between Candida albicans strains isolated from dental plaque, trachea, and bronchoalveolar lavage fluid from mechanically ventilated intensive care unit patients. *J Oral Microbiol*. 2011;3.
36. Paju S. Scannapieco FA. Oral biofilms, periodontitis, and pulmonary infections. *Oral Diseases*. 2007;13:508-512.
37. Russell SL, Boylan RJ, Kaslick RS, Scannapieco FA, Katz RV. Respiratory pathogen colonization of the dental plaque of institutionalized elders. *Special care in dentistry: official publication of the American Association of Hospital Dentists, the Academy of Dentistry for the Handicapped, and the American Society for Geriatric Dentistry*. 1999;19:128-134.
38. Scannapieco FA, Stewart EM, Mylotte JM. Colonization of dental plaque by respiratory pathogens in medical intensive care patients. *Critical care medicine*. 1992;20:740-745.
39. Shay K, Scannapieco FA, Terpenning MS, Smith BJ, Taylor GW. Nosocomial pneumonia and oral health. *Spec Care Dentist*. 2005;25:179-187.
40. Loesche WJ. *Dental caries: a treatable infection*:Springfield; 1982.
41. Busarcevic M, Kojic M, Dalgalarrondo M, Chobert JM, Haertle T, Topisirovic L. Purification of bacteriocin LS1 produced by human oral isolate lactobacillus salivarius BGHO1. *Oral microbiology and immunology*. 2008;23:254-258.
42. Kang MS, Lim HS, Oh JS, et al. Antimicrobial activity of lactobacillus salivarius and lactobacillus fermentum against staphylococcus aureus. *pathog dis*. 2017;75.
43. Traub WH, Spohr M. Hydrogen peroxide-mediated antagonism against serratia marcescens by streptococcus mitis. *Zentralbl Bakteriol Mikrobiol Hyg A*. 1983;254:326-332.
44. Uehara Y, Kikuchi K, Nakamura T, et al. Inhibition of methicillin-resistant staphylococcus aureus colonization of oral cavities in newborns by viridans group streptococci. *Clin infect dis*. 2001;32:1399-1407.
45. Morawska L, Johnson GR, Ristovski ZD, et al. Size distribution and sites of origin of droplets expelled from the human respiratory tract during expiratory activities. *J Aerosol Sci*. 2009;40:256-269.
46. Johnson GR, Morawska L. The Mechanism of Breath Aerosol Formation. *J Aerosol Med Pulm Drug Deliv*. 2009;22:229-237.
47. Papineni RS, Rosenthal FS. The size distribution of droplets in the exhaled breath of healthy human subjects. *J Aerosol Med*. 1997;10:105-116.
48. Asadi S, Wexler AS, Cappa CD, Barreda S, Bouvier NM, Ristenpart WD. Aerosol emission and superemission during human speech increase with voice loudness. *Scientific Reports*. 2019;9:2348.

49. Loudon RG, Roberts RM. Singing and the dissemination of tuberculosis. *Am Rev Respir Dis*. 1968;98:297-300.
50. Cole EC, Cook CE. Characterization of infectious aerosols in health care facilities: an aid to effective engineering controls and preventive strategies. *Am J Infect Control*. 1998;26: 453-464.
51. Keene CH. Airborne, Hygiene Contagion air. William firth wells. *J School Health*. 1955;25:249-249.
52. Yan J, Grantham M, Pantelic J, et al. Infectious virus in exhaled breath of symptomatic seasonal influenza cases from a college community. *Proc Natl Acad Sci USA*. 2018;115:1081-1086.
53. Tang JW, Gao CX, Cowling BJ, et al. Absence of detectable influenza RNA transmitted via aerosol during various human respiratory activities–experiments from Singapore and Hong Kong. *PLoS One*. 2014;9:e107338.
54. Milton DK, Fabian MP, Cowling BJ, Grantham ML, McDevitt JJ. Influenza virus aerosols in human exhaled breath: particle size, culturability, and effect of surgical masks. *PLoS Pathog*. 2013;9:e1003205.
55. Wood ME, Stockwell RE, Johnson GR, et al. Face masks and cough etiquette reduce the cough aerosol concentration of pseudomonas aeruginosa in people with cystic fibrosis. *Am J Respir Criti Care Med*. 2018;197:348-355.
56. Davies A, Thomson G, Walker J, Bennett A. A review of the risks and disease transmission associated with aerosol generating medical procedures. *J Infection Prevention*. 2009;10:122-126.
57. O'Neil CA, Li J, Leavey A, et al. Characterization of aerosols generated during patient care activities. *Clin Infect Dis*. 2017;65:1335-1341.
58. Gross KB, Overman PR, Cobb C, Brockmann S. Aerosol generation by two ultrasonic scalers and one sonic scaler. A comparative study. *J Dent Hyg*. 1992;66:314-318.
59. Grenier D. Quantitative analysis of bacterial aerosols in two different dental clinic environments. *Appl Environ Microbiol*. 1995;61:3165-3168.
60. Dutil S, Meriaux A, de Latremoille MC, Lazure L, Barbeau J, Duchaine C. Measurement of airborne bacteria and endotoxin generated during dental cleaning. *J Occup Environ Hyg*. 2009;6:121-130.
61. Bennett AM, Fulford MR, Walker JT, Bradshaw DJ, Martin MV, Marsh PD. Microbial aerosols in general dental practice. *Br Dent J*. 2000;189:664-667.
62. Legnani P, Checchi L, Pelliccioni GA, D'Achille C. Atmospheric contamination during dental procedures. *Quintessence Int*. 1994;25:435-439.
63. Osorio R, Toledano M, Liebana J, Rosales JI, Lozano JA. Environmental microbial contamination. Pilot study in a dental surgery. *Int Dent J*. 1995;45:352-357.
64. Rivera-Hidalgo F, Barnes JB, Harrel SK. Aerosol and splatter production by focused spray and standard ultrasonic inserts. *J Periodontol*. 1999;70:473-477.
65. Serban D, Banu A, Serban C, Tuta-Sas I, Vlaicu B. Predictors of quantitative microbiological analysis of spatter and aerosolization during scaling. *Rev Med Chir Soc Med Nat Iasi*. 2013;117:503-508.
66. Singh A, Shiva Manjunath RG, Singla D, Bhattacharya HS, Sarkar A, Chandra N. Aerosol, a health hazard during ultrasonic scaling: a clinico-microbiological study. *Indian J Dent Res: official publication of Indian Society for Dental Research*. 2016;27:160-162.
67. Timmerman MF, Menso L, Steinfort J, van Winkelhoff AJ, van der Weijden GA. Atmospheric contamination during ultrasonic scaling. *J Clin Periodontol*. 2004;31:458-462.
68. Veena HR, Mahantesha S, Joseph PA, Patil SR, Patil SH. Dissemination of aerosol and splatter during ultrasonic scaling: a pilot study. *J Infect Public Health*. 2015;8:260-265.
69. Watanabe A, Tamaki N, Yokota K, Matsuyama M, Kokeguchi S. Use of ATP bioluminescence to survey the spread of aerosol and splatter during dental treatments. *J Hosp Infect*. 2018;99:303-305.
70. Barnes JB, Harrel SK, Rivera-Hidalgo F. Blood contamination of the aerosols produced by in vivo use of ultrasonic scalers. *J Periodontol*. 1998;69:434-438.
71. Graetz C, Plaumann A, Bielfeldt J, Tillner A, Salzer S, Dorfer CE. Efficacy versus health risks: an in vitro evaluation of power-driven scalers. *J Indian Soc Periodontol*. 2015;19:18-24.
72. Harrel SK, Barnes JB, Rivera-Hidalgo F. Aerosol and splatter contamination from the operative site during ultrasonic scaling. *J Am Dental Assoc*. 1998;129:1241-1249.
73. Huntley DE, Campbell J. Bacterial contamination of scrub jackets during dental hygiene procedures. *J Dent Hyg*. 1998;72:19-23.
74. Bentley CD, Burkhart NW, Crawford JJ. Evaluating spatter and aerosol contamination during dental procedures. *J Am Dent Assoc*. 1994;125:579-584.
75. Yamada H, Ishihama K, Yasuda K, Hasumi-Nakayama Y, Shimoji S, Furusawa K. Aerial dispersal of blood-contaminated aerosols during dental procedures. *Quintessence Int*. 2011;42:399-405.
76. Rautemaa R, Nordberg A, Wuolijoki-Saaristo K, Meurman JH. Bacterial aerosols in dental practice – a potential hospital infection problem?. *J Hosp Infect*. 2006;64:76-81.
77. Kokosa JM, Eugene J. Chemical composition of laser-tissue interaction smoke plume. *J Laser Applications*. 1989;1:59-63.
78. Contaminants BargmanHLaser-generatedAirborne. *J Clin Aesthet Dermatol*. 2011;4:56-57.
79. Hallier C, Williams DW, Potts AJ, Lewis MA. A pilot study of bioaerosol reduction using an air cleaning system during dental procedures. *Br Dent J*. 2010;209:E14.
80. Teanpaisan R, Taeporamaysamai M, Rattanachone P, Poldoung N, Srisintorn S. The usefulness of the modified extra-oral vacuum aspirator (EOVA) from household vacuum cleaner in reducing bacteria in dental aerosols. *Int Dent J*. 2001;51:413-416.
81. Kobza J, Pastuszka JS, Bragoszewska E. Do exposures to aerosols pose a risk to dental professionals?. *Occup Med (Lond)*. 2018;68:454-458.
82. Szymańska J, Sitkowska J. Bacterial contamination of dental unit waterlines. *Environ Monit Assess*. 2013;185:3603-3611.
83. Rowland BM. Bacterial contamination of dental unit waterlines: what is your dentist spraying into your mouth?. *Clin Microbiol Newsletter*. 2003;25:73-77.
84. Lea SC, Landini G, Walmsley AD. Thermal imaging of ultrasonic scaler tips during tooth instrumentation. *J Clin Periodontol*. 2004;31:370-375.
85. Iorgulescu G. Saliva between normal and pathological. Important factors in determining systemic and oral health. *J Med Life*. 2009;2:303-307.
86. Gupta G, Mitra D, Ashok KP, et al. Efficacy of preprocedural mouth rinsing in reducing aerosol contamination produced

by ultrasonic scaler: a pilot study. *J Periodontol*. 2014;85:562-568.
87. Klyn SL, Cummings DE, Richardson BW, Davis RD. Reduction of bacteria-containing spray produced during ultrasonic scaling. *Gen Dent*. 2001;49:648-652.
88. Shetty SK, Sharath K, Shenoy S, Sreekumar C, Shetty RN, Biju T. Compare the effcacy of two commercially available mouthrinses in reducing viable bacterial count in dental aerosol produced during ultrasonic scaling when used as a preprocedural rinse. *J Contemp Dent Pract*. 2013;14:848-851.
89. Volgenant CMC, de Soet JJ. Cross-transmission in the dental office: does this make you ill. *Curr Oral Health Rep*. 2018;5:221-228.
90. Weaver JM. Confirmed transmission of hepatitis C in an oral surgery office. *Anesth Prog*. 2014;61:93-94.
91. Pervaiz M, Gilbert R, Ali N. The prevalence and underreporting of needlestick injuries among dental healthcare workers in pakistan: a systematic review. *Int J Dent*. 2018;2018:9609038.
92. Cleveland JL, Gray SK, Harte JA, Robison VA, Moorman AC, Gooch BF. Transmission of blood-borne pathogens in US dental health care settings: 2016 update. *J Am Dent Assoc*. 2016;147:729-738.
93. Jensen ET, Giwercman B, Ojeniyi B, et al. Epidemiology of pseudomonas aeruginosa in cystic fibrosis and the possible role of contamination by dental equipment. *J Hosp Infect*. 1997;36:117-122.
94. Peralta G, Tobin-D'Angelo M, Parham A, et al. Notes from the field: mycobacterium abscessus infections among patients of a pediatric dentistry practice–Georgia. *MMWR Morbidity and mortality weekly report*. 2015;65:355-356.
95. Schonning C, Jernberg C, Klingenberg D, et al. Legionellosis acquired through a dental unit: a case study. *J Hosp Infect*. 2017;96:89-92.
96. Ricci ML, Fontana S, Pinci F, et al. Pneumonia associated with a dental unit waterline. *Lancet (London, England)*. 2012;379:684.
97. Petti S, Vitali M. Occupational risk for Legionella infection among dental healthcare workers: meta-analysis in occupational epidemiology. *BMJ Open*. 2017;7:e015374.
98. Judson SD, Munster VJ. Nosocomial transmission of emerging viruses via aerosol-generating medical procedures. *Viruses*. 2019;11:940.

How to cite this article: Kumar PS, Subramanian K. Demystifying the mist: Sources of microbial bioload in dental aerosols. *J Periodontol*. 2020;91:1113–1122.
https://doi.org/10.1002/JPER.20-0395