Healthcare-associated viral and bacterial infections in dentistry

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Infection prevention in dentistry is an important topic that has gained more interest in recent years and guidelines for the prevention of cross-transmission are common practice in many countries. However, little is known about the real risks of cross-transmission, specifically in the dental healthcare setting. This paper evaluated the literature to determine the risk of cross-transmission and infection of viruses and bacteria that are of particular relevance in the dental practice environment. Facts from the literature on HSV, VZV, HIV, Hepatitis B, C and D viruses, *Mycobacterium* spp., *Pseudomonas* spp., *Legionella* spp. and multi-resistant bacteria are presented. There is evidence that Hepatitis B virus is a real threat for cross-infection in dentistry. Data for the transmission of, and infection with, other viruses or bacteria in dental practice are scarce. However, a number of cases are probably not acknowledged by patients, healthcare workers and authorities. Furthermore, cross-transmission in dentistry is under-reported in the literature. For the above reasons, the real risks of cross-transmission are likely to be higher. There is therefore a need for prospective longitudinal research in this area, to determine the real risks of cross-infection in dentistry. This will assist the adoption of effective hygiene procedures in dental practice.

Keywords: *cross-transmission*; *cross-infection*; *dentistry*; *bacteria*; *viruses*; *healthcare-associated infections*

The oral cavity is a natural habitat for a large number of microorganisms. This ecological niche can be a reservoir for opportunistic and pathogenic microorganisms that can pose a risk for cross-contamination and infection and may even cause systemic infections. This is of particular importance in the case of routine dental practice, as the risk of exposure to microorganisms in the oral cavity is increased due to the open and invasive nature of the procedures.

It is important to consider that the pathways of contamination can be bidirectional. An infectious microorganism may be transferred from the patient to members of the dental team, but also vice versa, e.g. through the hands of the dental team. Moreover, another infectious association is the transfer of pathogens from patient to patient, without the mediation of the dental staff, but rather through a surface located in the dental practice, or a device or instrument used during dental procedures. This can apply in the case of inadequate sterilization of the dental instruments or disinfection of the dental unit. The possibility also exists that pathogens present in dental unit waterlines (DUWLs) could be spread by aerosols created by dental hand-pieces, presenting a risk for both the patient and members of the dental team.

There are a number of possible means by which transmission of viral and bacterial pathogens can occur in the dental practice. The patient's own saliva and blood are major vectors of cross-transmission. Blood-borne contamination can occur by exposure to the infectious material through non-intact skin and mucosal lesions. The highest infectious risk of this type is associated with accidental punctures by contaminated needles or injuries by sharp instruments. Insufficient cross-contamination control, such as improperly sterilized dental instruments, is also a possible device-borne means of pathogen

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transmission. Emanation of the pathogens through the spray of the hand-pieces of the dental unit can also be considered an air-borne or water-borne means of transmission, which may affect both the patient and the dental team. Air-borne infections can also occur via an inefficient ventilation system in the dental practice environment, whereby contaminated air may be withheld or recycled. Overall, the risk of any such transmission depends on the dose of the pathogens transmitted, the virulence of the pathogen, as well as the frequency or probability of exposure to the infectious material and the state of the host immune responses.

The aim of this position paper on healthcare-associated infections in dentistry was primarily to evaluate cross-transmission risks of relevant viral and bacterial infections based on the evidence available in the current literature.

Viruses

Herpes viruses

Herpes viruses are ubiquitous human pathogens which can all be found in the oral environment. Cross-transmission risks in dentistry are mainly related to herpes simplex virus type 1 (HSV-1) and type 2 (HSV-2) and to varicella-zoster virus (VZV). Oral secreta can also be infectious in the subclinical phase of herpes virus infections and constitute a risk of cross-transmission.

Herpes simplex virus (HSV)

In Europe, the age standardized seropositivity for HSV-1 ranges from 52% in Finland to 84% in Bulgaria and the Czech Republic. For HSV-2 the seroprevalence ranges from 4% in England and Wales to 24% in Bulgaria (1). Seropositivity increases with age. In the US on average 60.3% of the population is infected with HSV-1 (2). The seroprevalence of HSV-1 is 92% and of HSV-2 is 13.2% in rural parts of East China (3). Twenty to forty percent of the seropositive subjects have recurrent infections in the oral region and asymptomatic shedding is common (4). Manipulation of the oral region, endotracheal intubation and immune-suppression are health or medical care-associated factors known to provoke oral reactivation of HSV-1 (5, 6). Previously, HSV-1 primary infections were commonly acquired in childhood. According to recent literature, however, a large proportion of young adults in Western countries today are not infected with HSV-1 (1).

HSV is highly contagious and is transmitted via exposure of the mucosa or skin to infectious secreta or contents of an infectious HSV blister. In the periphery, HSV-1 is most commonly associated with mucosal infections of the oral region and HSV-2 with genital infections, although both viruses are detected in either anatomical region. In the oral region, HSV-1 primary infection causes gingivostomatitis in 1–10% of patients and labial herpes or intraoral herpetic ulcers are typical symptoms of reactivation (7).

Unless adequate personal protection is worn, the dental team and the patient are at risk of being exposed to HSV via direct contact with herpetic ulcers or infectious splatters from herpetic lesions or saliva (8). This could result in mucosal or skin infections, keratitis or herpetic whitlow. Outside the body HSV is inactivated within hours and is easily inactivated by disinfectants, such as alcohols.

There are only a few reports of HSV cross-infection in the dental practice. The frequency of herpetic whitlow was observed to be higher among practicing dentists compared to the normal population (9). However, reports confirming the transmission of infection from the patient to the dental team infection are available in the literature (8). Also the cross-infection of HSV from dental team to patients has been shown. A dental hygienist with a herpetic whitlow, who did not use gloves routinely, infected 20 out of 46 patients (10).

A large portion of the adult population is infected with HSV-1 and reactivations resulting in subclinical or symptomatic infection are frequent. However, little research is published on the cross-transmission and infection of HSV-1 through the dental practice.

Varicella-zoster virus (VZV)

Chickenpox is the manifestation of VZV primary infection. About 90% of unvaccinated children acquire chickenpox before school age (11). In the periphery, VZV reactivation typically manifests as zoster of the skin or mucosa affecting 1–3 dermatomes in an episode. 20–30% of people are estimated to have zoster during lifetime (12). VZV can be also asymptomatically shed in saliva. Immune suppressed patients are at increased risk for disseminated disease and in non-immune pregnant women fetal infections are possible.

VZV is a highly contagious virus. It is transmitted through direct contact to the blisters or exposure to infectious droplets from saliva or the respiratory system. Airborne transmission is also possible (13).

Cross-transmission during dental care has not been reported, but VZV is known to cause healthcare-associated infections. After inadvertent exposure in the hospital environment, the transmission rate has been reported to be 4.5–29% (14). Respiratory secreta can already be contagious 2–4 days before varicella rash eruption. In addition to rashes, secreta of a zoster patient may also be contagious (15). VZV DNA has been detected in the environmental contact area of a zoster patient but also in the air-conditioner filter in a zoster patient's room, indicating a possibility of spread by aerosols from zoster patients (15, 16).

There is no evidence of VZV cross-transmission within dental healthcare but reports from other medical fields

indicate that VZV infection constitutes a risk for healthcare-associated transmission of VZV in the dental practice as well.

Human immunodeficiency virus (HIV)

In the 2010 UNAIDS report the total number of people infected with HIV worldwide in 2009 was estimated to be 33.3 million (0.8% of the global population). The HIV prevalence ranges from 0.2% in Central and Western Europe to 5% in Sub-Saharan Africa.

In prospective studies the risk of HIV infection after the percutaneous exposure to HIV infected blood was estimated to be 0.3% (17, 18). After mucous membrane exposure the risk is approximately 0.09% (19). The risk of acquiring HIV after exposure to tissues or other body fluids of an HIV infected person is not quantified, but is thought to be substantially lower (18). The risk of an infection correlates with the volume of blood exposure and blood viral counts (20). Transmission of HIV via saliva is considered unlikely due to low salivary viral titers, low numbers of CD4-positive target cells and the presence of anti-HIV antibodies and salivary antiviral factors protecting oral tissues (21).

Until December 2002, there were 344 published cases worldwide in which healthcare workers were infected with HIV as a result of their profession (22). Of these, 106 were documented to result from occupational exposure. For dental professionals eight possible occupationally acquired HIV infections were published, although no cases were confirmed (22). In four cases transmission of HIV from a healthcare worker to a patient was reported; a dentist in Florida, USA (23), a nurse in France (24), an orthopedic surgeon in France (25) and an obstetrician in Spain (26). Transmission of HIV from patient to patient has been reported, particularly as a result of poor infection control in developing countries and in developed countries in recipients of blood and blood products (27). There are no reports of patient to patient transmission of HIV in the dental practice.

The risk of transmission of HIV through the dental practice appears to be low; however, the data may not provide the complete picture. For instance, no occupationally acquired HIV infections were reported in Asia or parts of Africa and South America, yet the prevalence of HIV in some of these areas is high (28). Given that the risk of exposure to HIV is expected to be higher in areas where the prevalence of HIV infection is high, one would have anticipated reported cases of healthcare associated HIV transmission from these areas too.

Hepatitis B virus

The prevalence of chronic Hepatitis B virus (HBV) infections (positive for surface antigen HBsAg) ranges from <2% in Europe and the USA to 8% in Asia.

However, the prevalence of people that have experienced an HBV infection at some stage (positive for antibodies to HBc) ranges from 4–15% in low endemic countries to 40–90% in high endemic countries (29).

Before HBV vaccination was available, HBV infection was considered to be an occupational risk for healthcare workers and laboratory personnel (30). Following the introduction of the vaccine in 1983, the percentage of dentists with serological evidence of an HBV infection declined, especially in young dentists (31). However, even after the vaccine became available, healthcare providers remained at risk for HBV infection (32). The risk of an infection with HBV highly depends on the volume of blood and on the Hepatitis B e antigen (HBeAg) status of the source (33). The risk of HBV transmission to a healthcare worker after the percutaneous exposure to HBeAg- and HBsAg-positive blood is approximately 30%. The risk of HBV transmission after the percutaneous exposure to HBsAg-positive, HBeAg-negative blood is 1–6% (34). Besides through percutaneous injuries, HBV infection can also result from (in)direct blood or body fluid exposure through inoculation into cutaneous scratches, abrasions, burns or on mucosal surfaces (35). HBV has demonstrated the ability to survive and remain infectious in dried blood at room temperature on environmental surfaces for at least 1 week and probably longer (36).

The US Centers for Disease Control (CDC) described 300 patients that were infected through HBV-infected healthcare workers, including dentists and oral surgeons (34). Perry et al. (37) reported that a total of 12 healthcare workers had infected 91 patients with HBV between 1991 and 2005. The percentage of patients infected by the healthcare workers was 2.96%. None of the healthcare workers were dentists or members of the dental team. Recently, patient-to-patient transmission of HBV in an oral surgery practice was demonstrated using molecular techniques (38). In this case an older woman that had oral surgery contracted an acute Hepatitis B virus infection 2 months after her visit to the oral surgeon. The source turned out to be a woman that had oral surgery on the same date, in the same room, with the same hospital staff, 2 hours before the index patient. Other patients that were treated on the same day and the hospital staff that worked on that day were not infected. Interestingly, the CDC did not find breaches in standard infection control practices (38).

Vaccination of healthcare workers for HBV has greatly reduced the risk of transmission of HBV. However, HBV is highly infectious and vaccination for HBV is not standard for healthcare workers throughout the world. Moreover, patient-to-patient transmission of HBV has recently been proven. The risk of transmission of HBV through the dental practice remains an issue.

Hepatitis C virus

The prevalence of Hepatitis C virus (HCV) infection ranges from 0.1–1% in Northern Europe, 0.2–1.2% in Central Europe and 2.5–3.5% in Southern Europe (39). However, the prevalence of HCV infection can be as high as 26% in the southern parts of Italy (40). In the eastern part of Europe the prevalence of HCV infection was reported to be 0.9–4.9% of blood donors and 1–10% of healthcare workers (41). High prevalence of HCV outside of Europe is found in Egypt (14.9%), Taiwan (4.4%), Vietnam (2–2.9%) and Pakistan (3%) (42). Here the regional differences are large as well. The prevalence rate of HCV is 30% in the Punjab region in Pakistan.

Relatively little is known about the occupational risk of HCV infection. The average transmission rate of HCV after exposure is about 0.5% (43), much lower than for HBV and slightly higher than for HIV. The risk of transmission of HCV was significantly associated with hollow-bore needle placement in the source patient's artery or vein, deep injury and the sex of the healthcare worker. All cases of HCV transmission occurred after percutaneous exposure to viraemic blood or body fluids and the risk of transmission increased with the HCV viral load (44).

Perry et al. (37) reported a total of 11 HCV-positive healthcare workers that infected 38 patients between 1991 and 2005. The HCV-infected healthcare workers did not include dentists or oral surgeons. One study (45) assessed the risk of HCV infection among dentists in New York. Dentists and especially oral surgeons were at higher risk of having anti-HCV antibodies in their blood than matched blood donors. The higher risk for HCV infection was only attributed to occupational exposure. In the Netherlands, two cases of Hepatitis C virus infection resulting from blood exposure accidents have been documented in healthcare workers (46, 47). Occasionally, cases of acute HCV infection are attributed to blood exposure accidents. In a UK dental practice 1.2% of the staff (none of the dentists) were positive for HCV antibodies. Since the prevalence of HCV in the British population is 0.08–0.55%, the dental healthcare workers have a slightly higher risk of HCV infection compared to the general population (48).

In the general population the prevalence of HCV-infected patients is relatively low. In combination with a low average transmission rate, the risk of HCV transmission in the dental practice appears to be low. Research seems to support this conclusion. However, little is known about the transmission of HCV in comparison to HBV and HIV. Vaccination for HBV does not protect against infection with HCV.

Hepatitis D virus

Hepatitis D virus (HDV) infection can only occur in patients that are HBV co-infected. The incidence of Hepatitis D virus infection is not known, but is thought to be at about 5% of HBV carriers. In the literature, there is a lack of available reports regarding HDV infections in dentistry. One paper reported an outbreak of HDV in the 1980s where dentists were involved (49).

Bacteria

Viruses are mainly associated with a specific disease whilst for many bacteria this is not the case. Often transmission of bacteria does not result in a real infection, especially when opportunistic bacteria are involved. In the following sections, some severe pathogens are discussed that are potentially transmitted in the dental office. Furthermore, special forms of commensal bacteria that are (multi)-resistant to antibiotics are discussed, since they can result in severe and even lethal infections.

Mycobacterium ssp.

Mycobacterium tuberculosis causes the disease tuberculosis (TB). Other *Mycobacterium* spp. such as *M. bovis* are also capable of causing the disease. TB continues to be a major health burden in the twenty-first century with an estimated 1.7 million deaths every year (50). In the UK the number of reported cases of TB have been rising in recent years (51). Furthermore, the emergence of multi-drug resistant strains of *M. tuberculosis* (MDR-TB) is a major problem in the successful treatment and control of TB (52). Transmission of *M. tuberculosis* occurs through aerosols generated by coughing, sneezing and speaking. *M. tuberculosis* can remain airborne within small droplets for several hours and susceptible individuals can still become infected (52). It is thought that as few as 1–5 bacilli are required to initiate infection (53). Only about 30% of those exposed to TB are infected, 90% of them latently. In latent infection the bacteria remain viable in the body for many years without causing an active infection but retain the ability to activate under favorable conditions. It is not possible to predict who will develop active TB at some point (54). However, risk factors include immune-compromising situations such as chronic inflammatory diseases, during antitumor necrosis factor therapy, diabetes/obesity or a HIV co-infection (55). Other risk factors include alcoholism, and poor nutrition (54).

The possibility of *M. tuberculosis* transmission within dental settings has been subject to risk assessments by the CDC. Most dental clinics in the US were considered to fall within a very low risk category (56, 57). Despite the putative low risk, evidence for the transmission of TB in dental practice is present. In the UK a report documented cases of intraoral and pulmonary TB in patients that had been infected by their dental surgeon (58). All of the patients had tooth extractions performed by the dental surgeon who had active pulmonary TB. Furthermore,

dental team-to-dental team and possibly patient-to dental team TB infection was documented (59, 60). Hospital dental staff may be at increased risk of exposure to TB (60, 61).

The risk of TB transmission in the dental practice appears to be low. However, transmission of TB remains possible, primarily through patients from high risk areas in the world or from patients with reactivated TB infections such as the present-day elderly who may have been exposed to TB in their youth.

Pseudomonas spp.

The opportunistic pathogen *Pseudomonas aeruginosa* is frequently recovered from DUWLs where it can form biofilms in the tubing. *Pseudomonas* spp. may pose an important health hazard, particularly for immune-compromised patients. *P. aeruginosa* is associated with many types of infection, including hospital-acquired pneumonia (62), skin infections (63), urinary tract infections (64), burns (65), eye infections (66) and blood stream infections (67). Furthermore, *P. aeruginosa* is increasingly becoming multi-drug resistant, thus negatively affecting patient outcomes (68).

P. aeruginosa was isolated in 25% of studied DUWLs in Canada (69) and in 8% of DUWLs in Switzerland, where the dental chair units older than 5 years were contaminated significantly more often than new units (70). Patients with cystic fibrosis, known to be exceptionally susceptible to *Pseudomonas* spp. infections, were investigated for the presence of *P. aeruginosa* after a dental visit. *P. aeruginosa* was isolated from up to 5% of DUWLs, but only one patient tested positive in sputum for genotypically matched *P. aeruginosa* (71). Others reported two patients with solid tumors that developed gingival abscesses with pyocin-matched *P. aeruginosa* after dental treatment. Furthermore, 78 subjects with no underlying medical condition were transiently colonized for 3–5 weeks with *P. aeruginosa* when treated in a unit with contaminated water (72).

Given that the incidence of *Pseudomonas* spp. in DUWLs is high but that there are limited reported cases of infection following exposure to DUWLs, it is apparent that the risk of infection is very low. Although reports in this area are conflicting (73, 74); DUWL endotoxin from gram-negative bacteria such as *Pseudomonas* spp., has been suggested to complicate the healing processes after periodontal surgery operations (75) and to induce asthma exacerbations (74). Therefore, further studies will be needed to verify this potential health hazard.

Legionella spp.

Legionella spp. can cause a severe form of pneumonia in humans: Legionnaires' disease, and a less severe flu-like disease: Pontiac fever. *Legionella pneumophila*, particularly *L. pneumophila* serogroup 1, is the most common etiological agent of Legionnaires' disease although other species of the *Legionella* genus can also be causal (76). Inhalation of aerosols contaminated with *Legionella* spp. acts as the main route of transmission (77). Legionnaires' disease outbreaks have frequently been associated with contaminated aerosol-producing water systems such as showers, humidifiers and water-cooling towers. *Legionella* spp. can readily multiply in water at temperatures between 25 and 45°C (78), especially in stagnant water and they grow in biofilms. Legionnaires' disease is considered to be an important nosocomial infection, about 11.5% of reported Legionnaires' disease cases in the UK between 1980 and 2009 were hospital-acquired (UK's Health Protection Agency, January 2011). Outbreaks of Legionnaires' disease in hospitals have been predominantly associated with contaminated potable water supplies (79).

Legionella spp. presents a potential risk in the dental clinic where contaminated DUWLs could act as a source of infection to both dental staff and patients. DUWLs harbor microbial biofilms that are seeded by the mains water supply and can provide a suitable environment for the multiplication of *Legionella* spp. within protozoa (80). Studies of contamination of DUWLs frequently detected *Legionella* spp. among the bacteria present, although their prevalence varies substantially between different studies (81–84). In particular, prevalence rates vary with differing geographic location (80). While the prevalence of *Legionella* spp. in UK DUWLs varies between 0.37 and 1.19%, similar studies in the US show prevalence rates of 68% (83, 85), although differences in methodology could be responsible for these differences. The number of colony forming units (CFU's) of *Legionella* spp. found in DUWLs has been reported to be around 10^2–10^5 CFU/ml. The infective dose of *Legionella* spp. is thought to be greater than 10^5 CFU/ml (86).

Recently a case was published of a patient that was infected with *L. pneumophila* serogroup 1 which originated from a dental office in Italy (87). After hospitalization because of the pulmonary problems, the patient died from this infection. By using molecular typing methods, it was clearly shown that the source of the *Legionella* infection was the DUWL in a dental practice, where both the tap water and the unit water were contaminated. It is remarkable that only one patient acquired this *Legionella*. Even Pontiac fever was not found within the other patients in this office. This is the first time that Legionnaires' disease resulting from visits to, or treatment in, a dental clinic was documented. To date, there are no known cases of Pontiac fever in patients, resulting from visits to, or treatment in, a dental clinic. This would indicate that the risk to patients posed by *Legionella* spp. from DUWLs is low. However the risk is not absent and infection can have dramatic consequences. In addition, cases of Pontiac fever that appear 1 week after a dental

visit may not be recognized as such and the link between disease and dental treatment is not acknowledged.

There is a single documented fatal case of Legionnaire's disease in a US dentist. In this case the infection was attributed to exposure of the dentist to DUWL aerosols containing *Legionella* spp. (81). *L. pneumophilia* and *L. longbeachae* were detected in the dentist's lung tissue and in the DUWLs, however, the dentist's domestic water supplies also had very low levels of *Legionella* spp. The evidence in this case was not conclusive. Studies have found dental staff to have higher serum levels of antibodies specific to *Legionella* spp. than members of the general public (88, 89). This is indicative of an increased occupational exposure to *Legionella* spp. through contaminated aerosols.

Despite this, no other cases of Legionnaires' disease or Pontiac fever shown to result from occupational exposure have been documented in dental staff. However, the lack of investigation into the DUWLs of the dental clinics in other cases of Legionnaire's disease or Pontiac fever in dental staff could have led to under-reporting.

Multi-resistant bacteria

Multi-resistant bacteria pose a major health risk and are increasing the cost of healthcare worldwide (90). Multi-resistant bacteria are mainly transmitted by direct contact or indirectly via contaminated surfaces. Increased risk of colonization by multi-resistant bacteria is associated with long hospital stays, living in a nursing home, institutional and international patient transfers, surgical procedures and the presence of invasive devices, severe medical conditions, immune suppression and antimicrobial therapy (91, 92). Currently, the most problematic healthcare-associated multi-resistant species are methicillin-resistant *Staphylococcus aureus* (MRSA), extended-spectrum β-lactamase (ESBL)-producing *Enterobacteriaceae* and carbapenemase-producing gram-negative bacteria (93).

S. aureus asymptomatically colonizes the oropharynx or the skin of 20–30% of people and it has been isolated from various oral infections (94). *S. aureus* is a commensal bacterium, but is also responsible for skin infections, septicaemia, endocarditis, osteomyelitis, pneumonia and toxic shock syndrome (95). Among hospitalized patients receiving treatment for oral conditions, oral cancer patients have been reported to be at increased risk for MRSA colonization (96)–(98). Carriage rate is influenced by the age and the overall disease status of the host (99). MRSA is resistant to all β-lactam antibiotics including methicillin, cloxacillin, dicloxacillin, cephalosporins and carbapenems and may also display resistance to some other antibiotics.

During the past 10 years, community-acquired MRSA (CA-MRSA) infections have been increasing (100). Studies on the oral or nasopharyngeal MRSA colonization rate report a prevalence of up to 26%, depending on

the population (101). In a recent literature review, MRSA carriage in healthcare workers was found to be 4.6%, which is above the rate of the normal population (102). In most studies, MRSA carriage in dental healthcare workers has not been above the level of the normal adult population (101). In the greater Houston metropolitan area 4.2% of dentists and 1.5% of dental hygienists were positive for MRSA (103) whereas only 1.5% of the non-institutionalized US citizens are colonized with MRSA (104). Others showed that 21% of dental students were found to be carrying MRSA in their nose (105). MRSA carriage in dental staff could pose a risk for transmission of MRSA to the patients or co-workers. In the UK, a dentist carrying MRSA was proven to have transmitted MRSA to two patients having oral surgery (106). During 1 year of surveillance in a dental hospital clinic, 8 (out of 140) patients were colonized or infected with MRSA after treatment. The air–water syringe and the chair arm of the dental chair were contaminated with MRSA. The antibiograms revealed that the isolated MRSA strains were similar between patients and the contaminated chair and it was concluded that the patients were colonized or infected by (parts of) the dental chair (107). Furthermore, MRSA has been isolated in dental clinics outside of the hospital environment (108). Moreover, devices such as dental impression guns were reported to be contaminated with MRSA in routine use (109). Dental impressions and gypsum casts were also shown to be heavily colonized by MRSA (110).

Bacteria in the family *Enterobacteriaceae* can acquire the ability to produce the enzyme extended-spectrum β-lactamases (ESBL) making them multi-resistant to penicillins, second and third generation cephalosporins and monobactams (111).

Carbapenemase-producing gram-negative bacteria such as *Klebsiella pneumoniae* carbapenemase (KPC) are the most recent and serious threat of healthcare-associated infections since they are resistant to all β-lactam antibiotics and they display co-resistance to other antibiotics. They have spread particularly via international hospital transfers from epidemic areas. In addition, *Enterobacteriaceae* harboring New Delhi metallo-β-lactamase-1 (NDM-1) have been repeatedly introduced in Europe via patient transfers (92).

In healthy individuals oral colonization by *Enterobacteriaceae* or *Pseudomonas* spp. is usually transient (112). Even transiently colonizing bacteria can be part of the flora of oral cavity related abscesses and source of cross-transmission (113). ESBL-producers were detected in oropharyngeal samples of 40% of the inhabitants in an Italian long-term-care facility (114) and in the throat of patients in intensive care units (115, 116). There are no published reports available on the possible association of ESBL- or carbapenemase-producers and oral infections

nor evidence for transmission of these bacteria through the dental practice.

Hence, MRSA has been shown to be transmitted in dental care. However, evidence for the transmission of ESBL- and carbapenemase-producers does not exist yet, although transmission in the dental practice is possible.

General considerations and concluding remarks

This paper aimed to report and discuss the current literature on the transmission of relevant viruses and bacteria in dentistry. All viruses and bacteria discussed above can be, and most have been, proven to be transmitted to the patient or the dental team in the dental practice. It appears that the transmission of, and infection with, Hepatitis B virus poses the greatest risk for both patients and the dental team, based on the incidence and risk of transmission. Literature on the transmission of the other bacteria and viruses is scarce and it seems that the risk for transmission resulting in an infection with these microorganisms is low. It should be noted, however, that transmission of pathogens may result in an asymptomatic infection that can last weeks or even months until symptoms appear (Table 1). In addition, it is likely that some patients that visit the dental practice are not aware of their infectious status and may carry an asymptomatic infection. There is a lack of prospective longitudinal studies that investigate the incidence of healthcare-associated infections in patients after dental treatment.

Another important point to keep in mind is that the likelihood of healthcare-associated infections, particularly in dentistry, of being detected, reported, documented and published is small (37). Therefore, healthcare-associated infections are under-reported in literature from the developed world (27, 117). In developing countries the risk of transmission of several microorganisms, for instance the blood borne viruses, is high since a large section of the population is infected. However, literature on the subject from these countries is scarce.

Table 1. Incubation period of microorganisms associated with healthcare infections in dentistry

Microorganism	Incubation period
Herpes simplex virus	Up to 2 weeks
Varicella zoster virus	2–3 weeks
Hepatitis B/C/D virus	Up to 6 months
HIV/AIDS	Months–years
Mycobacterium tuberculosis	Up to 6 months
Pseudomonas spp.	3–10 days
Legionella spp.	2–19 days
Staphylococcus aureus	4–10 days

Based on the number of reported cases, most of which were of blood borne viruses, the actual risk for developing an infectious disease through the dental practice appears to be low. However, the real transmission rate of the viruses and bacteria that are discussed in this paper is probably higher. As long as accurate data are absent, the dental team should be fully aware of the risk of dissemination of potentially hazardous microorganisms and ensure that efficient cross-infection control procedures are well in place (118). Every member of the team must follow the standard procedures required to prevent the transmission of microorganisms. Besides preventing disease by vaccination, these include hand hygiene, personal barrier protection, instrument disinfection and sterilization protocols, surface decontamination strategies, approaches to maintain the quality of DUWLs, as well as the emergency procedures in case of accidents that would increase the risk of cross-transmission. These procedures substantially lower the risk of the transmission of microorganisms. Every patient should be treated as potentially infectious. The dental team should be acquainted with the biological principles behind these procedures. The cross-infection control regulations should undergo regular monitoring and need to be subjected to revision whenever necessary.

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The authors declare no conflicts of interest.

References

1. Pebody RG, Andrews N, Brown D, Gopal R, De Melker H, Francois G, et al. The seroepidemiology of herpes simplex virus type 1 and 2 in Europe. Sex Transm Infect 2004; 80: 185–91.
2. Karjala Z, Neal D, Rohrer J. Association between HSV1 seropositivity and obesity: data from the National Health and Nutritional Examination Survey, 2007–2008. PLoS One 2011; 6: e19092.
3. Lin H, He N, Su M, Feng J, Chen L, Gao M. Herpes simplex virus infections among rural residents in eastern China. BMC Infect Dis 2011; 11: 69.
4. Wheeler CE, Jr. The herpes simplex problem, J Am Acad Dermatol 1988; 18: 163–8.
5. Kameyama T, Sujaku C, Yamamoto S, Hwang CB, Shillitoe EJ. Shedding of herpes simplex virus type 1 into saliva. J Oral Pathol 1988; 17: 478–81.
6. Hanley PJ, Conaway MM, Halstead DC, Rhodes LV, III, Reed J, III. Nosocomial herpes simplex virus infection associated with oral endotracheal intubation. Am J Infect Control 1993; 21: 310–6.
7. Spruance SL. Herpes simplex labialis. In: Sacks SL, Straus SE, Whiteley RJ, Griffiths PD, eds. Clinical management of herpes viruses. Amsterdam: IOS Press; 1995. p. 3–42.
8. Browning WD, Mccarthy JP. A case series: herpes simplex virus as an occupational hazard. J Esthet Restor Dent 2012; 24: 61–6.

9. Rowe NH, Heine CS, Kowalski CJ. Herpetic whitlow: an occupational disease of practicing dentists. J Am Dent Assoc 1982; 105: 471–3.

10. Manzella JP, Mcconville JH, Valenti W, Menegus MA, Swierkosz EM, Arens M. An outbreak of herpes simplex virus type I gingivostomatitis in a dental hygiene practice. JAMA 1984; 252: 2019–22.

11. Wolleswinkel-Van Den Bosch JH, Speets AM, Rumke HC, Gumbs PD, Fortanier SC. The burden of varicella from a parent's perspective and its societal impact in the Netherlands: an Internet survey. BMC Infect Dis 2011; 11: 320.

12. Gnann JW, Jr, Whitley RJ. Clinical practice. Herpes zoster. N Engl J Med 2002; 347: 340–6.

13. Gershon AA, Gershon MD, Breuer J, Levin MJ, Oaklander AL, Griffiths PD. Advances in the understanding of the pathogenesis and epidemiology of herpes zoster. J Clin Virol 2010; 48(Suppl 1): S2–7.

14. Langley JM, Hanakowski M. Variation in risk for nosocomial chickenpox after inadvertent exposure. J Hosp Infect 2000; 44: 224–6.

15. Yoshikawa T, Ihira M, Suzuki K, Suga S, Tomitaka A, Ueda H, et al. Rapid contamination of the environments with varicella-zoster virus DNA from a patient with herpes zoster. J Med Virol 2001; 63: 64–6.

16. Suzuki K, Yoshikawa T, Tomitaka A, Matsunaga K, Asano Y. Detection of aerosolized varicella-zoster virus DNA in patients with localized herpes zoster. J Infect Dis 2004; 189: 1009–12.

17. Bell DM. Occupational risk of human immunodeficiency virus infection in healthcare workers: an overview. Am J Med 1997; 102: 9–15.

18. Henderson DK, Fahey BJ, Willy M, Schmitt JM, Carey K, Koziol DE, et al. Risk for occupational transmission of human immunodeficiency virus type 1 (HIV-1) associated with clinical exposures. A prospective evaluation. Ann Intern Med 1990; 113: 740–6.

19. Ippolito G, Puro V, De Carli G. The risk of occupational human immunodeficiency virus infection in health care workers. Italian multicenter study. The Italian Study group on occupational risk of HIV infection. Arch Intern Med 1993; 153: 1451–8.

20. Cardo DM, Culver DH, Ciesielski CA, Srivastava PU, Marcus R, Abiteboul D, et al. A case-control study of HIV seroconversion in health care workers after percutaneous exposure. Centers for disease control and prevention needlestick surveillance group. N Engl J Med 1997; 337: 1485–90.

21. Shugars DC, Wahl SM. The role of the oral environment in HIV-1 transmission. J Am Dent Assoc 1998; 129: 851–8.

22. Health Protection Agency. Occupational transmission of HIV. Summary of published reports. March 2005 edition. Available from: http://www.hpa.org.uk/webc/HPAwebFile/HPAweb_C/1194947320156

23. Ciesielski C, Marianos D, Ou CY, Dumbaugh R, Witte J, Berkelman R, et al. Transmission of human immunodeficiency virus in a dental practice. Ann Intern Med 1992; 116: 798–805.

24. Astagneau P, Lot F, Bouvet E, Lebascle K, Baffoy N, Aggoune M, et al. Lookback investigation of patients potentially exposed to HIV type 1 after a nurse-to-patient transmission. Am J Infect Control 2002; 30: 242–5.

25. Lot F, Seguier JC, Fegueux S, Astagneau P, Simon P, Aggoune M, et al. Probable transmission of HIV from an orthopedic surgeon to a patient in France. Ann Intern Med 1999; 130: 1–6.

26. Bosch X. Second case of doctor-to-patient HIV transmission. Lancet Infect Dis 2003; 3: 261.

27. Mccarthy GM, Sali CS, Bednarsh H, Jorge J, Wangrangsimakul K, Page-Shafer K. Transmission of HIV in the dental clinic and elsewhere. Oral Dis 2002; 8(Suppl 2): 126–35.

28. Health Protection Agency. Occupational transmission of HIV: summary of published reports. March 2005 edition. Data to the end of December 2002. Health Protection Agency Centre for Infections and Collaborators; 2005. Available from: http://www.hpa.org.uk/webc/HPAwebFile/HPAweb_C/1194947320156

29. Margolis HS, Alter MJ, Hadler SC. Viral hepatitis. In: Evans AS, Kaslow RA, eds. Viral infections of humans, fourth ed. New York: Plenum Medical Books Co; 1997. p. 363–418.

30. Hadler SC, Doto IL, Maynard JE, Smith J, Clark B, Mosley J, et al. Occupational risk of hepatitis B infection in hospital workers. Infect Control 1985; 6: 24–31.

31. Cleveland JL, Siew C, Lockwood SA, Gruninger SE, Gooch BF, Shapiro CN. Hepatitis B vaccination and infection among US dentists, 1983–1992. J Am Dent Assoc 1996; 127: 1385–90.

32. Gerberding JL. Incidence and prevalence of human immuno-deficiency virus, hepatitis B virus, hepatitis C virus, and cytomegalovirus among health care personnel at risk for blood exposure: final report from a longitudinal study. J Infect Dis 1994; 170: 1410–7.

33. Werner BG, Grady GF. Accidental hepatitis-B-surface-antigen-positive inoculations. Use of e antigen to estimate infectivity. Ann Intern Med 1982; 97: 367–9.

34. US Public Health Service. Updated US public health service guidelines for the management of occupational exposures to HBV, HCV, and HIV and recommendations for postexposure prophylaxis. MMWR Recomm Rep 2001; 50: 1–52.

35. Lauer JL, Vandrunen NA, Washburn JW, Balfour HH, Jr. Transmission of hepatitis B virus in clinical laboratory areas. J Infect Dis 1979; 140: 513–6.

36. Bond WW, Favero MS, Petersen NJ, Gravelle CR, Ebert JW, Maynard JE. Survival of hepatitis B virus after drying and storage for one week. Lancet 1981; 1: 550–1.

37. Perry JL, Pearson RD, Jagger J. Infected health care workers and patient safety: a double standard. Am J Infect Control 2006; 34: 313–9.

38. Redd JT, Baumbach J, Kohn W, Nainan O, Khristova M, Williams I. Patient-to-patient transmission of hepatitis B virus associated with oral surgery. J Infect Dis 2007; 195: 1311–4.

39. Esteban JI, Sauleda S, Quer J. The changing epidemiology of hepatitis C virus infection in Europe. J Hepatol 2008; 48: 148–62.

40. Bellentani S, Miglioli L, Masutti F, Saccoccio G, Tiribelli C. Epidemiology of hepatitis C virus infection in Italy: the slowly unraveling mystery. Microbes Infect 2000; 2: 1757–63.

41. Naoumov NV. Hepatitis C virus infection in Eastern Europe. J Hepatol 1999; 31(Suppl 1): 84–7.

42. Sievert W, Altraif I, Razavi HA, Abdo A, Ahmed EA, Alomair A, et al. A systematic review of hepatitis C virus epidemiology in Asia, Australia and Egypt. Liver Int 2011; 31(Suppl 2): 61–80.

43. Jagger J, Puro V, De Carli G. Occupational transmission of hepatitis C virus. JAMA 2002; 288: 1469; author reply 1469–71.

44. Yazdanpanah Y, De Carli G, Migueres B, Lot F, Campins M, Colombo C, et al. Risk factors for hepatitis C virus transmission to health care workers after occupational exposure: a European case-control study. Clin Infect Dis 2005; 41: 1423–30.

45. Klein RS, Freeman K, Taylor PE, Stevens CE. Occupational risk for hepatitis C virus infection among New York City dentists. Lancet 1991; 338: 1539–42.

46. Weegink CJ, Sentjens RE, Van Der Heyden JF, Chamuleau RA, Tytgat GN, Beld MG. A physician with a positive hepatitis C virus RNA test after a needlestick injury. Eur J Gastroenterol Hepatol 2003; 15: 1367–9.

47. Berger CM, Leentvaar-Kuijpers A, Van Doornum GJ, Coutinho RA. Accidentele blootstelling aan bloed en risico op transmissie van virusinfecties bij verschillende beroepsgroepen in Amsterdam, 1986–1996. Ned Tijdschr Geneeskd 1998; 142: 2312–4.

48. Lodi G, Porter SR, Teo CG, Scully C. Prevalence of HCV infection in health care workers of a UK dental hospital. Br Dent J 1997; 183: 329–32.

49. Porter S, Scully C, Samaranayake L. Viral hepatitis. Current concepts for dental practice. Oral Surg Oral Med Oral Pathol 1994; 78: 682–95.

50. Lawn SD, Zumla AI. Tuberculosis. Lancet 2011; 378: 57–72.

51. Abubakar I, Lipman M, Anderson C, Davies P, Zumla A. Tuberculosis in the UK – time to regain control. BMJ 2011; 343. doi: 10.1136/bmj.d4281.

52. Carvalho AC, Migliori GB, Cirillo DM. Tuberculosis in Europe: a problem of drug resistance or much more? Expert Rev Respir Med 2010; 4: 189–200.

53. Small PM, Fujiwara PI. Management of tuberculosis in the United States. N Engl J Med 2001; 345: 189–200.

54. Gengenbacher M, Kaufmann SH. Mycobacterium tuberculosis: success through dormancy. FEMS Microbiol Rev 2012; 36: 514–32.

55. Barry CE, III, Boshoff HI, Dartois V, Dick T, Ehrt S, Flynn J, et al. The spectrum of latent tuberculosis: rethinking the biology and intervention strategies. Nat Rev Microbiol 2009; 7: 845–55.

56. Cleveland JL, Gooch BF, Bolyard EA, Simone PM, Mullan RJ, Marianos DW. TB infection control recommendations from the CDC, 1994: considerations for dentistry. United States Centers for Disease Control and Prevention. J Am Dent Assoc 1995; 126: 593–9.

57. Cleveland JL, Robison VA, Panlilio AL. Tuberculosis epidemiology, diagnosis and infection control recommendations for dental settings: an update on the Centers for disease control and prevention guidelines. J Am Dent Assoc 2009; 140: 1092–9.

58. Smith WH, Davies D, Mason KD, Onions JP. Intraoral and pulmonary tuberculosis following dental treatment. Lancet 1982; 1: 842–4.

59. Cleveland JL, Kent J, Gooch BF, Valway SE, Marianos DW, Butler WR, et al. Multidrug-resistant Mycobacterium tuberculosis in an HIV dental clinic. Infect Control Hosp Epidemiol 1995; 16: 7–11.

60. Harlow RF, Rutkauskas JS. Tuberculosis risk in the hospital dental practice. Spec Care Dentist 1995; 15: 50–5.

61. Cadmus SI, Okoje VN, Taiwo BO, Van Soolingen D. Exposure of dentists to Mycobacterium tuberculosis, Ibadan, Nigeria. Emerg Infect Dis 2010; 16: 1479–81.

62. Fujitani S, Sun HY, Yu VL, Weingarten JA. Pneumonia due to Pseudomonas aeruginosa: part I: epidemiology, clinical diagnosis, and source. Chest 2011; 139: 909–19.

63. Wu DC, Chan WW, Metelitsa AI, Fiorillo L, Lin AN. Pseudomonas skin infection: clinical features, epidemiology, and management. Am J Clin Dermatol 2011; 12: 157–69.

64. Mittal R, Aggarwal S, Sharma S, Chhibber S, Harjai K. Urinary tract infections caused by Pseudomonas aeruginosa: a minireview. J Infect Public Health 2009; 2: 101–11.

65. Branski LK, Al-Mousawi A, Rivero H, Jeschke MG, Sanford AP, Herndon DN. Emerging infections in burns. Surg Infect (Larchmt) 2009; 10: 389–97.

66. Willcox MD. Pseudomonas aeruginosa infection and inflammation during contact lens wear: a review. Optom Vis Sci 2007; 84: 273–8.

67. Van Delden C. Pseudomonas aeruginosa bloodstream infections: how should we treat them? Int J Antimicrob Agents 2007; 30(Suppl 1): S71–5.

68. Hirsch EB, Tam VH. Impact of multidrug-resistant Pseudomonas aeruginosa infection on patient outcomes. Expert Rev Pharmacoecon Outcomes Res 2010; 10: 441–51.

69. Barbeau J, Tanguay R, Faucher E, Avezard C, Trudel L, Cote L, et al. Multiparametric analysis of waterline contamination in dental units. Appl Environ Microbiol 1996; 62: 3954–9.

70. Barben J, Kuehni CE, Schmid J. Water quality in dental chair units. A random sample in the canton of St. Gallen. Schweiz Monatsschr Zahnmed 2009; 119: 976–85.

71. Jensen ET, Giwercman B, Ojeniyi B, Bangsborg JM, Hansen A, Koch C, et al. Epidemiology of Pseudomonas aeruginosa in cystic fibrosis and the possible role of contamination by dental equipment. J Hosp Infect 1997; 36: 117–22.

72. Martin MV. The significance of the bacterial contamination of dental unit water systems. Br Dent J 1987; 163: 152–4.

73. Scannapieco FA, Ho AW, Ditolla M, Chen C, Dentino AR. Exposure to the dental environment and prevalence of respiratory illness in dental student populations. J Can Dent Assoc 2004; 70: 170–4.

74. Pankhurst CL, Coulter W, Philpott-Howard JN, Surman-Lee S, Warburton F, Challacombe S. Evaluation of the potential risk of occupational asthma in dentists exposed to contaminated dental unit waterlines. Prim Dent Care 2005; 12: 53–9.

75. Putnins EE, Di Giovanni D, Bhullar AS. Dental unit waterline contamination and its possible implications during periodontal surgery. J Periodontol 2001; 72: 393–400.

76. Newton HJ, Ang DK, Van Driel IR, Hartland EL. Molecular pathogenesis of infections caused by Legionella pneumophila. Clin Microbiol Rev 2010; 23: 274–98.

77. Gomez-Valero L, Rusniok C, Buchrieser C. Legionella pneumophila: population genetics, phylogeny and genomics. Infect Genet Evol 2009; 9: 727–39.

78. Wadowsky RM, Wolford R, Mcnamara AM, Yee RB. Effect of temperature, pH, and oxygen level on the multiplication of naturally occurring Legionella pneumophila in potable water. Appl Environ Microbiol 1985; 49: 1197–205.

79. Sabria M, Yu VL. Hospital-acquired legionellosis: solutions for a preventable infection. Lancet Infect Dis 2002; 2: 368–73.

80. Pankhurst CL, Coulter WA. Do contaminated dental unit waterlines pose a risk of infection? J Dent 2007; 35: 712–20.

81. Atlas RM, Williams JF, Huntington MK. Legionella contamination of dental-unit waters. Appl Environ Microbiol 1995; 61: 1208–13.

82. Challacombe SJ, Fernandes LL. Detecting Legionella pneumophila in water systems: a comparison of various dental units. J Am Dent Assoc 1995; 126: 603–8.

83. Walker JT, Bradshaw DJ, Bennett AM, Fulford MR, Martin MV, Marsh PD. Microbial biofilm formation and contamination of dental-unit water systems in general dental practice. Appl Environ Microbiol 2000; 66: 3363–7.

84. Veronesi L, Capobianco E, Affanni P, Pizzi S, Vitali P, Tanzi ML. Legionella contamination in the water system of hospital dental settings. Acta Biomed 2007; 78: 117–22.

85. Pankhurst CL, Coulter W, Philpott-Howard JJ, Harrison T, Warburton F, Platt S, et al. Prevalence of legionella waterline contamination and Legionella pneumophila antibodies in general dental practitioners in London and rural Northern Ireland, Br Dent J 2003; 195: 591–4; discussion 581.

86. Pankhurst CL. Risk assessment of dental unit waterline contamination. Prim Dent Care 2003; 10: 5–10.

87. Ricci ML, Fontana S, Pinci F, Fiumana E, Pedna MF, Farolfi P, et al. Pneumonia associated with a dental unit waterline. Lancet 2012; 379: 684.

88. Fotos PG, Westfall HN, Snyder IS, Miller RW, Mutchler BM. Prevalence of Legionella-specific IgG and IgM antibody in a dental clinic population. J Dent Res 1985; 64: 1382–5.

89. Reinthaler FF, Mascher F, Stunzner D. Serological examinations for antibodies against *Legionella* species in dental personnel. J Dent Res 1988; 67: 942–3.

90. European Centre for Disease Prevention and Control. Antimicrobial resistance surveillance in Europe 2010. Annual Report of the European Antimicrobial Resistance Surveillance Network (EARS-Net). Stockholm: ECDC; 2011. Available from: http://ecdc.europa.eu/en/publications/Publications/1111_SUR_AMR_data.pdf.pdf

91. Safdar N, Maki DG. The commonality of risk factors for nosocomial colonization and infection with antimicrobial-resistant *Staphylococcus aureus*, enterococcus, gram-negative bacilli, *Clostridium difficile*, and Candida. Ann Intern Med 2002; 136: 834–44.

92. Grundmann H, Livermore DM, Giske CG, Canton R, Rossolini GM, Campos J, Vatopoulos A, Gniadkowski M, Toth A, Pfeifer Y, Jarlier V, Carmeli Y, the CNSE Working Group. Carbapenem-non-susceptible Enterobacteriaceae in Europe: conclusions from a meeting of national experts. Euro Surveill 2010; 15(46): pii=19711. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19711

93. Wieler LH, Ewers C, Guenther S, Walther B, Lubke-Becker A. Methicillin-resistant staphylococci (MRS) and extended-spectrum beta-lactamases (ESBL)-producing Enterobacteriaceae in companion animals: nosocomial infections as one reason for the rising prevalence of these potential zoonotic pathogens in clinical samples. Int J Med Microbiol 2011; 301: 635–41.

94. Smith AJ, Robertson D, Tang MK, Jackson MS, Mackenzie D, Bagg J. *Staphylococcus aureus* in the oral cavity: a three-year retrospective analysis of clinical laboratory data. Br Dent J 2003; 195: 701–3; discussion 694.

95. O'Leary E, Lockhart DE, Smith A. Coming to a practice near you? Community-acquired methicillin resistant *Staphylococcus aureus* (CA-MRSA). Dent Update 2011; 38: 254–6, 259–60.

96. Parton M, Beasley NJ, Harvey G, Houghton D, Jones AS. Four cases of aggressive MRSA wound infection following head and neck surgery. J Laryngol Otol 1997; 111: 874–6.

97. Mitchell TE, Pickles JM. Four cases of aggressive MRSA wound infection following head and neck surgery. J Laryngol Otol 1998; 112: 422.

98. Supriya M, Shakeel M, Santangeli L, Ah-See KW. Controlling MRSA in head and neck cancer patients: what works? Otolaryngol Head Neck Surg 2009; 140: 224–7.

99. Smith AJ, Jackson MS, Bagg J. The ecology of S*taphylococcus* species in the oral cavity. J Med Microbiol 2001; 50: 940–6.

100. Kock R, Becker K, Cookson B, Van Gemert-Pijnen JE, Harbarth S, Kluytmans J, et al. Methicillin-resistant *Staphylococcus aureus* (MRSA): burden of disease and control challenges in Europe. Euro Surveill 2010; 15: 19688.

101. Petti S, Polimeni A. Risk of methicillin-resistant *staphylococcus aureus* transmission in the dental healthcare setting: a narrative review. Infect Control Hosp Epidemiol 2011; 32: 1109–15.

102. Albrich WC, Harbarth S. Health-care workers: source, vector, or victim of MRSA? Lancet Infect Dis 2008; 8: 289–301.

103. Reddy Y. Prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) nazal colonization among dental professionals (MPH thesis). Ann Arbor, MI: ProQuest; 2010.

104. Gorwitz RJ, Kruszon-Moran D, Mcallister SK, Mcquillan G, Mcdougal LK, Fosheim GE, et al. Changes in the prevalence of nasal colonization with *Staphylococcus aureus* in the United States, 2001–2004. J Infect Dis 2008; 197: 1226–34.

105. Roberts MC, Soge OO, Horst JA, Ly KA, Milgrom P. Methicillin-resistant *Staphylococcus aureus* from dental school clinic surfaces and students. Am J Infect Control 2011; 39: 628–32.

106. Martin MV, Hardy P. Two cases of oral infection by methicillin-resistant *Staphylococcus aureus*. Br Dent J 1991; 170: 63–4.

107. Kurita H, Kurashina K, Honda T. Nosocomial transmission of methicillin-resistant *Staphylococcus aureus* via the surfaces of the dental operatory. Br Dent J 2006; 201: 297–300; discussion 291.

108. Motta RH, Groppo FC, Bergamaschi C, de C, Ramacciato JC, Baglie S, De Mattos-Filho TR. Isolation and antimicrobial resistance of *staphylococcus aureus* isolates in a dental clinic environment. Infect Control Hosp Epidemiol 2007; 28: 185–90.

109. Westergard EJ, Romito LM, Kowolik MJ, Palenik CJ. Controlling bacterial contamination of dental impression guns. J Am Dent Assoc 2011; 142: 1269–74.

110. Egusa H, Watamoto T, Abe K, Kobayashi M, Kaneda Y, Ashida S, et al. An analysis of the persistent presence of opportunistic pathogens on patient-derived dental impressions and gypsum casts. Int J Prosthodont 2008; 21: 62–8.

111. Grover SS, Sharma M, Pasha ST, Singh G, Lal S. Antimicrobial susceptibility pattern and prevalence of extended spectrum beta-lactamase (ESBLs) producing strains of *Klebsiella pneumoniae* from a major hospital in New Delhi. J Commun Dis 2004; 36: 17–26.

112. Mobbs KJ, Van Saene HK, Sunderland D, Davies PD. Oropharyngeal Gram-negative bacillary carriage: a survey of 120 healthy individuals. Chest 1999; 115: 1570–5.

113. Mehra P, Murad H. Maxillary sinus disease of odontogenic origin. Otolaryngol Clin North Am 2004; 37: 347–64.

114. March A, Aschbacher R, Dhanji H, Livermore DM, Bottcher A, Sleghel F, et al. Colonization of residents and staff of a long-term-care facility and adjacent acute-care hospital geriatric unit by multiresistant bacteria. Clin Microbiol Infect 2010; 16: 934–44.

115. Murk JL, Heddema ER, Hess DL, Bogaards JA, Vandenbroucke-Grauls CM, Debets-Ossenkopp YJ. Enrichment broth improved detection of extended-spectrum-beta-lactamase-producing bacteria in throat and rectal surveillance cultures of samples from patients in intensive care units. J Clin Microbiol 2009; 47: 1885–7.

116. Roshan M, Ikram A, Mirza IA, Malik N, Abbasi SA, Alizai SA. Susceptibility pattern of extended spectrum ß-lactamase producing isolates in various clinical specimens. J Coll Physicians Surg Pak 2011; 21: 342–6.

117. Doebbeling BN, Vaughn TE, McCoy KD, Beekmann SE, Woolson RF, Ferguson KJ, et al. Percutaneous injury, blood exposure, and adherence to standard precautions: are hospital-based health care providers still at risk? Clin Infect Dis 2003; 37: 1006–13.

118. Molinari JA. Infection control: its evolution to the current standard precautions. J Am Dent Assoc 2003; 134: 569–74; quiz 631–2.

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