ELSEVIER

JOURNAL OF ADOLESCENT HEALTH

www.jahonline.org

Original article

Tongue Piercing: The Impact of Material on Microbiological Findings

AQ: 1 AQ: 2 Ines Kapferer, M.D.[a,*], Ulrike S. Beier, M.D.[a], Rutger G. Persson, Ph.D.[b,c]

[a] *Department of Restorative and Operative Dentistry, Dental School, Innsbruck Medical University, Innsbruck, Austria*
[b] *Laboratory of Oral Microbiology, School of Dental Medicine, University of Bern, Bern, Switzerland*
[c] *Department of Periodontology, School of Dental Medicine, University of Bern, Bern, Switzerland*

Article history: Received 22 July 2010; Accepted 21 October 2010
Keywords: Adolescent, Body piercing/adverse effects, Humans, Tongue piercing, Tongue/microbiology

A B S T R A C T

Purpose: Biofilms on oral piercings may serve as a bacterial reservoir and lead to systemic bacteremia or local transmission of pathogenic microbiota. The use of piercing materials which are less susceptible to biofilm accumulation could contribute to prevention of problems. The present study investigated whether there are microbiological differences in bacterial samples collected from tongue piercings made of different materials.
Methods: A total of 85 subjects with tongue piercings participated in this study. After a baseline dental examination, sterile piercings of four different materials were randomly allocated to the study subjects. After 2 weeks, microbiologic samples were collected and processed by checkerboard deoxyribonucleic acid-deoxyribonucleic acid hybridization methods.
Results: About 28.8% of subjects reported 61 lingual recessions (1.91 ± .96 mm), whereas 5% reported tooth chipping on one tooth each. With the exception of *Aggregatibacter actinomycetemcomitans* (Y4), *Fusobacterium nucleatum* species, and *Parvimonas micra*, bacteria associated with periodontitis were not commonly found in the samples from studs or piercing channels. Of the 80 bacterial species, 67 were found at significantly higher levels ($p < .001$) in samples from stainless steel than from polytetrafluoroethylene or polypropylene piercings.
Conclusion: The low bacterial counts from piercing channels suggest that having a tongue pierced would not contribute to an increased risk for oral infection. The present study demonstrated that studs made of steel might promote the development of a biofilm, whereas those made of polytetrafluoroethylene or polypropylene may be rather inert to bacterial colonization. The finding of *Staphylococci* on steel and titanium studs may suggest an elevated risk for complication if the piercing channel is infected.

Body piercing and other body modifications have increased tremendously in popularity in recent years [1], especially among teenagers and young adults in the industrial world. Oral piercing mostly involves the lips, tongue, and cheeks, with tongue being the most commonly pierced intraoral site [2,3]. In a cross-sectional household survey of 10,503 British adults, the prevalence of tongue piercing was 6.5% in those aged 16–24 [3]. From a medical perspective, the use of tongue jewellery cannot be considered a harmless fashion trend as it can produce undesired local and general effects [4]. Early complications include bacterial infection, pain, swelling, prolonged bleeding, and difficulties in swallowing, speech, and mastication [5]. Late complications include chipped and fractured teeth, gingival trauma, localized periodontitis, persistent difficulties in oral functions, and swallowing of the device [5]. The published data on medical implications of tongue piercing mainly Includes case reports and a limited number of clinical studies [4–7]. Therefore, many biological questions related to these foreign bodies, such as biofilm formation, remain unaddressed. With infections being one of the most frequent piercing complications [8], biofilm formation on oral piercing is a fundamental issue. Additionally, biofilms on oral piercings may serve as a bacterial reservoir and lead to systemic bacteremia and even septic complications. The piercing procedure exposes the piercee to a high risk of infection because the oral cavity harbors a huge amount of bacteria [4]. The high

* Address correspondence to: Ines Kapferer, M.D., Department of Restorative and Operative Dentistry, Dental School, Innsbruck Medical University, Reichenauerstraβe 46, 6020, Innsbruck, Austria.
E-mail address: ines.kapferer@gmx.net

1054-139X/$ - see front matter © 2010 Published by Elsevier Inc. on behalf of Society for Adolescent Health and Medicine.
doi:10.1016/j.jadohealth.2010.10.008

vascularity of the area is a further aspect to be considered [4]. Although complications such as infective endocarditis [9], epidural abscess [10], chorioamnionitis [11], herpes simplex virus hepatitis [12], hepatitis C virus infection [13], toxic shock syndrome [14], and cerebellar brain abscess [15] are rare, they are dangerous complications. Additionally, biofilms on oral piercings may serve as reservoirs for bacteria associated with periodontitis, because of the anaerobic condition in the piercing channel [4]. Thus, the use of piercing materials less susceptible to biofilm accumulation could contribute to alleviation or even prevention of problems. Currently, there are no data on the additional role of the piercing material in plaque accumulation on oral piercings.

Stainless steel (SS), titanium (Ti), polytetrafluoroethylene (PTFE), and the polypropylene Borealis A/S Bormed HD810MO (PP) are commonly used as piercing materials. The surfaces of SS as well as Ti are well known for good mechanical properties, high corrosion resistance, and excellent biocompatibility [16]. PTFE is an autoclavable synthetic polymer consisting of carbon and fluorine [17]. Bormed is a heat and radiation sterilizable PP homopolymer designed for medical applications [18]. These four piercing materials differ in surface roughness R_a (which is highest for PTFE), in wettability (which is lowest in PTFE), and surface chemistry (unpublished observations). Materials like gold and silver – popular in other body regions – are only of scarce usage with regard to piercing in the oral cavity, and thus they have not been considered in this study.

The present study aimed to assess microbiological findings in association with tongue piercing in a population obtained from a nondental setting. It was hypothesized that there are microbiological differences in bacterial samples collected from tongue piercings made of different materials. It was also hypothesized that the piercings carry the same characteristic bacteria as found in the piercing channels and that the biofilm on the tongue is independently similar to the other study locations.

Methods

Ethical considerations

The Ethics Committee of Innsbruck Medical University, Austria, approved the study. The study was conducted in accordance with the Helsinki Declaration of 1975, as revised in 2000. All subjects signed informed written consent before investigation. The study was performed in 2008 at the Department of Operative and Preventive Dentistry, Innsbruck Medical University, Innsbruck, Austria. At the conclusion of the clinical examination, participants obtained appropriate compensation, and were informed about their oral status and any diagnosed muco-gingival lesions. Subjects with diagnosed pathological conditions were offered appropriate treatment.

Study subjects

Posters and flyers were dispersed on the university campus, in high schools, and vocational schools in Innsbruck, Austria, to recruit subjects for this study. The study cohort included 85 subjects with tongue piercing. The piercing had to be in situ for at least 6 months. The following exclusion criteria were applied: pregnancy and lactating women, medication with an effect on gingival tissues, antibiotic medication in the last 6 months or need for antibiotic prophylaxis, chlorhexidine use in the last 6 months, nonplaque induced gingival disease, and earlier diagnosis of periodontitis.

Questionnaire

Participants were asked to complete a questionnaire to determine demographic and medical data, smoking habits, characteristics of the piercing device worn, and postpiercing complications. If tooth chipping was found during clinical examination, subjects were asked to provide information about the circumstances under which the chipping occurred.

Clinical examination

Clinical periodontal conditions were recorded at six sites per tooth, excluding wisdom teeth. Probing depth (PD) was measured with a pressure-calibrated probe (ClickProbe 1395, KerrHawe, Bioggio, CH) to the nearest millimeter. Bleeding on probing (BOP) [19] was recorded dichotomously. Presence or absence of plaque was measured using the plaque control record [20]. The amount of recession was measured from the cementoenamel junction to the free gingival margin at six sites per tooth. Clinical attachment level was calculated by adding the amount of recession and PD. One investigator (I.K.) performed all measurements and collected all samples.

After the periodontal examination, the personal piercings of the study subjects were substituted by one of the test piercings of four commonly used piercing materials: Ti, SS, PP, PTFE. Randomization was performed before experiments by computer-generated randomization (Excel, Microsoft Corp., Redmond, WA), and piercing materials were allocated to the study subjects in the chronological order of appointment. The length of the stud for each subject was measured. Piercings were cut to the appropriate length for PP and PTFE. Different lengths of piercings were available to the investigator for Ti and SS. All piercings were packaged individually and sterilized (121°C, 20 minutes, steam autoclave Belimed KHS 2000 [Belimed Sauter AG, Sulgen, CH]) before the experiments. Packages were only opened at the visit when the devices were placed and carefully handled to prevent contamination during clinical manipulations. Special attention was paid to prevent damage to the channel tissue when removing and inserting the studs. Study subjects had the piercing 2 weeks in situ. The test piercings were then removed. The piercings with adhering biofilms were placed in 1.5 mL PBS in deoxyribonuclease free laboratory tubes (natural flat cap microcentrifuge tubes, Starlab GmbH Ahrensburg, Germany), and sonicated for 30 seconds to disperse adhering bacteria. The efficacy of the removal of the bacteria has been tested in preliminary studies through scanning electron microscopy. The studs were removed and the solutions were assayed for bacterial identification. At the laboratory, .15 mL Tris ethylenediaminetetraacetic acid (EDTA) buffer (10 mM Tris–HCl, 1.0 mM EDTA, pH 7.6) and .10 mL .5 M NaOH were added to each Eppendorf tube. A swab was used to collect microbiological samples from the tongue. An endodontic paper point size 55 (Absorbent Paper Points, Dentsply/Maillefer, Ballaigues, CH) was inserted in the piercing channel and kept in situ for 20 seconds. Efforts were made to move the paper point against the channel linings of the piercing locations after the stud had been removed. The collected paper points were placed in individual dry Eppendorf tubes (1.5-mL natural flat cap deoxyribonucleic acids (DNAs) and ribonucleic acids free micro-centrifuge tubes, Starlab, Ahrensburg, Germany).

Please cite this article in press as: Ines Kapferer, et al., Tongue Piercing: The Impact of Material on Microbiological Findings, J Adolesc Health (2010), doi: 10.1016/j.jadohealth.2010.10.008

Table 1
Reference bacteria strains included in the DNA-DNA checkerboard analysis

Species	Collection	Species	Collection
Actinomyces israelii	ATCC 12102	*Lactobacillus jensenii*	GUH 160339
Actinomyces naeslundii (type I + II)	ATCC 43146	*Lactobacillus vaginalis*	GUH 078092
Actinomyces neuii	GUH 550898	*Leptotrichia buccalis*	ATCC14201
Actinomyces odontolyticus	ATCC 17929	*Mobiluncis curtisii*	GUH 070927
Aggregatibacter actinomycetemcomitans (a)	ATCC29523	*Mobiluncus mulieris*	GUH 070926
Aggregatibacter actinomycetemcomitans (Y4)	ATCC 43718	*Neisseria mucosa*	ATCC 33270
Aerococcus christensenii	GUH 070938	*Parvimonas micra*	ATCC 19696
Aanaerococcus vaginalis	GUH 290486	*Peptoniphilus* sp.	GUH 55097
Atopobium parvulum	GUH 160323	*Porphyromonas endodontalis*	ATCC 35406
Atopobium vaginae	GUH 010535	*Porphyromonas gingivalis*	ATCC 33277
Bacteroides ureolyticus	GUH 080189	*Prevotella bivia*	GUH 450429
Bifidobacterium biavatii	GUH 071026	*Prevotella disiens*	GUH 190184
Bifidobacterium bifidum	GUH 070962	*Prevotella intermedia*	ATCC 25611
Bifidobacterium breve	GUH 080484	*Prevotella melaninogenica*	ATCC 25845
Bifidobacterioum longum	GUH 180689	*Propionibacterium acnes* (type I+II)	ATCC 11727/2
Campylobacter gracilis	ATCC 33236	*Proteus mirabilis*	GUH 07092
Campylobacter rectus	ATCC 33286	*Pseudomonas aeruginosa*	DSMZ 50071
Campylobacter showae	ATCC 51146	*Selenomonas noxia*	ATCC 43541
Capnocytophaga gingivalis	ATCC 33612	*Staphylococcus anaerobius*	DSMZ 20714
Capnocytophaga ochraceae	ATCC 335945	*Staphylococcus aureus*	ATCC 25923
Capnocytophaga sputigena	ASTCC 33612	*Staphylococcus aureus* (yellow)	GUH 070921
Corynebacterium nigricans	GUH 450453	*Staphylococcus aureus* (white)	GUH 070922
Cornerbacterium aurimucosum	GUH 071035	*Staphylococcus epidermidis*	GUH 130381
Dialister sp.	GUH 071045	*Staphylococcus haemolyticus*	DSMZ 20263
Escherichia coli	GUH 070903	*Streptococcus agalactiae*	GUH 230282
Eikenella corrodens	ATCC 23834	*Streptococcus anginosus*	ATCC 33397
Enterococcus faecalis	GUH 170812	*Streptococcus constellatus*	ATCC 27823
Enterococcus faecalis	ATCC 29212	*Streptococcus gordonii*	ATCC 10558
Fusobacterium nucleatum nucleatum	ATCC 25586	*Streptococcus intermedius*	ATCC 27335
Fusobacterium nucleatum polymorphum	ATCC 10953	*Streptococcus mitis*	ATCC 49456
Fusobacterium nucleatum naviforme	ATCC 49256	*Streptococcus oralis*	ATCC 35037
Fusobacterium periodonticum	ATCC 33693	*Streptococcus pneumoniae*	DSMZ 11866
Gardnerella vaginalis	GUH 080585	*Streptococcus sanguinis*	ATCC 10556
Haemophilus influenzae	ATCC 49247	*Streptococcus mutans*	ATCC 25175
Helicobacter pylori	ATCC 43504	*Tannerella forsythia*	ATCC 43037
Lactobacillus acidophilus	ATCC 11975	*Treponema denticola*	ATCC 35405
Lactobacillus crispatus	GUH 160342	*Treponema socranskii*	D40DR2
Lactobacillus gasseri	GUH 17085	*Varibaculum cambriense*	GUH 070917
Lactobacillus iners	GUH 160334	*Veillonella parvula*	ATCC 10790

ATCC = American Type Culture Collection; D: sample from Forsyth Institute, Boston, MA; GUH = Ghent University Hospital Collection, Ghent, Belgium.

Microbiological processing

The microbiological samples were frozen at −20°C. Samples were sent to the oral microbiology laboratory at the University of Bern, Switzerland, and then processed within 3 months. The swabs were transferred to deoxyribonuclease free laboratory tubes (natural flat cap microcentrifuge tubes, Starlab GmbH Ahrensburg, Germany) with 350 μL Tris EDTA buffer (10 m*M* Tris-HCl, 1.0 mM EDTA and pH 7.6). The swabs were carefully rotated in the buffer solution and then squeezed against the tube walls to recover as much bacterial material in the solution as possible. The swab was then removed. Subsequently, 200 μL of freshly made .5 M NaOH was then added to each vial. The content was then aliquoted in two equal portions in laboratory tubes. Samples were processed as described for the checkerboard DNA-DNA hybridization method described elsewhere [21–24]. The tubes of collected paper points were sonicated for 20 seconds and the paper points were removed. The remaining content was pipetted on to slots and processed as described for the checkerboard DNA-DNA hybridization method [21–24]. Information on the species used in the present study for the checkerboard method is listed (Table 1). Signals were detected by chemiluminescence using the Storm Fluor-Imager (Storm 840, Amersham Biosciences, Piscataway, NJ) with a setup of 200 μm and 600 V. The digitized information was analyzed by a software program (ImageQuant, Amersham Pharmacia, Piscataway, NJ) allowing comparison of the density 19 sample-lanes against the two standard-lanes (10^5 or 10^6 cells) and converted to absolute counts by comparisons with these standards. Relative microbial counts were used when different sampling sites (piercing channel, stud, tongue) were compared. The surface area of each of the test studs was defined (circumference × length) and used for normalization of microbiological data (cells/mm^2) to compare biofilms on different piercing materials.

Statistical analysis

Wilcoxon signed-ranks test, χ^2 test, and Mann–Whitney test were used for statistical analysis of microbiological findings. Adjustment for multiple comparisons was made and a statistically significant difference was defined by $p < .001$. For clinical data, standard descriptive statistics were used to summarize the variables studied. Unless otherwise stated, results are expressed as mean ± standard deviation (SD). Variations in demographic and clinical data between groups were assessed by χ^2 and Kruskal–Wallis test. The data analysis was performed with a

Please cite this article in press as: Ines Kapferer, et al., Tongue Piercing: The Impact of Material on Microbiological Findings, J Adolesc Health (2010), doi: 10.1016/j.jadohealth.2010.10.008

Table 2
Subjects' demographic background and clinical data

	Stainless steel (n = 20)	Titanium (n = 20)	Polypropylene (n = 20)	PTFE (n = 20)
Mean age ± SD	22.7 ± 3.7	23.4 ± 4.6	20.9 ± 7.6	20.8 ± 6.8
Gender				
Male, n (%)	2 (10)	3 (15)	2 (10)	5 (25)
Female, n (%)	18 (90)	17 (85)	18 (90)	15 (75)
Smokers (life time exposure)				
Nonsmokers, n (%)	6 (30)	8 (40)	6 (30)	5 (25)
Light smokers, n (%)	0 (0)	0 (0)	0 (0)	0 (0)
Moderate/heavy smokers, n (%)	14 (70)	12 (60)	14 (70)	15 (75)
Characteristics of the stud				
Mean time since piercing, months ± SD	60.1 ± 50.9	41.5 ± 32.4*	59.4 ± 36.3	73.8 ± 31
Length of the stud[a], mm ± SD	16.4 ± .8	17.7 ± 1**	16.4 ± 4.5	17.2 ± 3.7
Surface area, mm^2 ± SD	86.5 ± 12.3	88.7 ± 5	82.7 ± 4.1	89.7 ± 10.3
Clinical data				
Probing depth, mm ± SD	1.9 ± .5	1.9 ± .6	1.9 ± .6	2 ± .4
Clinical attachment level, mm ± SD	1.9 ± .6	2 ± .6	2 ± .5	2.1 ± .3
Plaque control record, % ± SD	39.7 ± 19.7	34 ± 18.8	31.9 ± 20.1	32 ± 17
BOP, % ± SD	11.8 ± 17.8	11.4 ± 15.3	12.7 ± 14.8	15.3 ± 14.3

* $p = .03$,
** $p = .003$.
[a] Length of the subject's own stud and the test stud.

statistical software package (SPSS 17.5 for MAC computers, SPSS Inc., Chicago, IL).

Results

Subject characteristics

Subject characteristics are presented in Table 2. Five subjects were excluded because of antibiotic medication during the study period. None of the subjects presented with clinical evidence of periodontitis or other exclusionary criteria. A total of 80 subjects (68 women, 12 men) completed the study. All participants were Caucasians, aged 16–36 years (mean age ± SD = 22.74 ± 4.47), among whom 31.25% had never smoked, 0% were light smokers (1–912 packs lifetime exposure), and 68.75% were moderate to heavy smokers (>912 packs lifetime exposure). The average time since piercing at examination day was 60.05 ± 38.69 months (range: 6 months–14.25 years, median: 60 months).

Clinical data

Clinical data are shown in Table 2. No subject presented with localized periodontitis as late complication of the tongue piercing. A total of 23 subjects (28.8%) reported 61 lingual recessions (1.91 ± .96 mm), which might be late complications of the tongue piercings, with 39 gingival recessions located lingually on lower incisors. Four subjects (5%) reported tooth chipping on one tooth each. All of them could exactly report the situation, about how the tooth chipping occurred because of biting on the tongue piercing. One subject had tooth chipping on six teeth, which could not be related to the piercing. No patient had hyperplastic tissues around the piercing. There was no case with swelling or keloid scarring around the piercing.

Microbiological analysis

Relative microbial counts showed statistically significant differences between the study locations tongue, piercing channel, and piercing stud (Table 3). Most bacterial species were found at significantly higher proportions ($p < .001$) in samples from the tongue than from the piercing channel (35/80) and the stud (42/80). On the contrary, of the 80 species, 18 were found at significantly higher proportions in samples from the piercing channel than from the tongue. These included *A actinomycetemcomitans bY4, Campylobacter gracilis, C rectus, C showae, Capnocytophaga ochraceae, Capnocytophaga sputigena, Eubacterium saburreum*, *Fusobacterium nucleatum species nucleatum*, *F nucleatum species polymorphum, Leptotrichia buccalis, P micra, Staphylococcus anaerobius, S aureus, S haemolyticus, Streptococcus anginosus, Streptococcus intermedius*, *Streptococcus mutans*, and *Treponema denticola* (Table 3). Additionally, statistical analysis identified significantly higher bacterial proportions ($p < .001$) from studs than from tongue samples for six species. These included the following species*: A actinomycetemcomitans bY4, Capnocytophaga gingivalis, C gracilis, C rectus, Propionibacterium acnes*, and *S haemolyticus* (Table 3).

Of those 80 species, eight were presented with significantly higher proportions ($p < .001$) from studs than from piercing channels (*Actinomyces naeslundii type I and II, A odontolyticus, Eikenella corrodens, Escherischia coli, Proteus mirabilis, Selenomonas noxia, Streptococcus sanguinis*, and *Veilonella parvula*) (Table 3).

Analysis of bacteria identified from piercing studs according to the piercing material

There were no statistically significant differences between groups in relation to age, gender, smoking status, or clinical baseline data (PD, clinical attachment level, plaque control record, and BOP). Statistically significant differences between groups in piercing characteristics were identified (Table 2): in the Ti group, the studs were significantly longer ($p = .003$), but the duration with piercing was significantly lower ($p = .03$) than in the other groups. No statistically significant differences were found between groups when comparing microbial counts from the tongue or the channel.

Comparing normalized microbial counts from the studs (cells/mm^2) regarding the material identified that the total microbial load was significantly higher ($p < .001$) on SS piercings

Please cite this article in press as: Ines Kapferer, et al., Tongue Piercing: The Impact of Material on Microbiological Findings, J Adolesc Health (2010), doi: 10.1016/j.jadohealth.2010.10.008

Table 3
Relative microbial counts of samples from tongue, piercing channel, and piercing studs; median (25. percentile; 75. percentile)

Tongue		Piercing channel		Studs	
Bacterial species	Relative microbial counts (%)	Bacterial species	Relative microbial counts (%)	Bacterial species	Relative microbial counts (%)
V parvula	22.36 (16.97; 27.32)	*S haemolyticus*	14.6 (8.47; 22.03)	*V parvula*	21.48 (4.9; 43.95)
P melaninogenica	4.45 (2.88; 6.72)	*P micra*	6.49 (3.63; 9.80)	*S haemolyticus*	6.59 (.40; 17.26)
A vaginae	3.84 (1.19; 8.51)	*C showae*	5.20 (2.58; 8.25)	*E corrodens*	3.11 (1.54; 8.29)
S pneumoniae	3.73 (2.90; 4.52)	*V parvula*	4.20 (2.44; 10.06)	*S pneumoniae*	2.31 (0; 4.72)
S haemolyticus	3.73 (2.20; 4.46)	*F periodonticum*	3.03 (1.93; 5.13)	*S oralis*	1.59 (0; 2.59)
S oralis	3.09 (2.38; 3.76)	*P melaninogenica*	2.46 (1.54; 3.69)	*C showae*	1.23 (0; 2.99)
P aeruginosa	2.90 (2.08; 3.70)	*A a (b) Y4*	2.37 (1.46; 3.48)	*A a (b) Y4*	1.18 (0; 4.17)
F periodonticum	2.55 (1.40; 3.27)	*S mutans*	2.33 (1.74; 2.94)	*N mucosa*	1.04 (0; 2.83)
Aodontolyticus⁎	2.35 (1.74; 2.86)	*L. buccalis*	2.28 (1.47; 2.86)	*P melaninogenica*	1.03 (0; 10.31)
L gasseri	2.11 (.86; 6.52)	*F n sp. nucleatum*	2.18 (1.98; 2.76)	*P micra*	.98 (0; 2.91)
C showae	2.05 (.75; 3.71)	*S oralis*	1.91 (1.43; 2.94)	*P mirabilis*	.94 (0; 2.29)
S mitis	1.99 (1.53; 2.72)	*C rectus*	1.82 (.98; 2.48)	*C rectus*	.93 (.44; 1.65)
L acidophilus	1.90 (1.56; 2.14)	*C gingivalis*	1.73 (1.20; 2.79)	*L gasseri*	.84 (0; 2.01)
E corrodens	1.65 (1.15; 3.99)	*S intermedius*	1.70 (0; 3.35)	*Dialister sp.*	.83 (.44; 1.52)
N mucosa	1.62 (.66; 7.64)	*C gracilis*	1.67 (1.22; 2.54)	*F periodonticum*	.83 (.35; 2.32)
P micra	1.52 (.75; 2.52)	*S aureus*	1.64 (.96; 2.24)	*S noxia*	.79 (0; 1.74)
Dialister sp.	1.40 (.44; 2.98)	*F n sp. polymorphum*	1.55 (.88; 2.56)	*F n sp. naviforme*	.75 (0; 1.41)
E coli	1.39 (.59; 2.75)	*S anginosus*	1.43 (1.11; 1.91)	*A vaginae*	.73 (0; 1.41)
P mirabilis	1.37 (.81; 3.65)	*A a (a)29,523*	1.39 (1.10; 1.96)	*L jensenii*	.69 (0; 1.81)
L jensenii	1.28 (.43; 2.12)	*S mitis*	1.33 (0; 3.39)	*E coli*	.68 (.29; 1.55)
A a (a)29523	1.24 (1.05; 1.72)	*S anaerobius*	1.31 (.69; 1.91)	*L buccalis*	.68 (0; 1.50)
L iners	1.18 (.59; 2.29)	*C sputigena*	1.20 (.85; 1.50)	*S mitis*	.66 (0; 1.37)
S noxia	1.10 (.74; 2.49)	*T denticola*	1.18 (0; 1.91)	*F n sp. polymorphum*	.64 (0; 1.45)
L vaginalis	1.05 (.69; 2.62)	*E saburreum*	1.11 (.13; 1.85)	*A naeslundii 1 and 2*	.64 (0; 1.68)
E saburreum	1.02 (.29; 1.55)	*S constellatus*	1.09 (.60; 1.62)	*C gingivalis*	.61 (0; 1.79)
S sanguinis	.95 (.69; 1.22)	*F n sp. naviforme*	.99 (.54; 1.39)	*G vaginalis*	.62 (0; 2.72)
S gordonii	.95 (.67; 1.22)	*Dialister sp.*	.89 (.11; 1.29)	*F n sp. nucleatum*	.59 (0; 1.41)
S aureus	.94 (.56; 1.13)	*A vaginae*	.85 (.49; 1.29)	*S sanguinis*	.58 (0; 1.17)
A naeslundii 1 and 2	.89 (.75; 1.13)	*Peptoniphilus sp.*	.80 (0; 1.97)	*E saburreum*	.55 (0; 1.49)
P intermedia	.88 (.55; 1.27)	*L acidophilus*	.73 (.42; 1.06)	*S aureus*	.52 (0; .98)
S mutans	.85 (.47; 1.21)	*G vaginalis*	.72 (0; 1.56)	*S epidermis*	.49 (0; 1.30)
H pylori	.84 (.23; 2.19)	*L jensenii*	.72 (0; 1.79)	*S anaerobius*	.49 (.17; .73)
S anginosus	.84 (.55; 1.11)	*H pylori*	.71 (.27; 1.02)	*S anginosus*	.47 (0; .98)
A a (b)Y4	.81 (.62; .99)	*A vaginalis*	.71 (.48; 1.26)	*L vaginalis*	.41 (0; .83)
S anaerobius	.78 (.39; 1.01)	*S epidermis*	.68 (.07; 1.19)	*A odontolyticus*	.41 (.05; .63)
S. epidermis	.75 (.49; 1.96)	*E corrodens*	.64 (.44; .97)	*T socranscii*	.38 (0; .92)
F n sp. naviforme	.73 (.49; .97)	*L gasseri*	.63 (0; 1.70)	*H pylori*	.37 (0; .61)
B ureolyticus	.73 (.27; 1.48)	*E faecalis*	.62 (.31; 1.14)	*C sputigena*	.36 (.13; .77)
G vaginalis	.67 (.33; 7.49)	*B ureolyticus*	.60 (.36; .99)	*Strept gordonii*	.35 (0; 1.81)
S agalactiae	.66 (.35; 1.13)	*P intermedia*	.58 (.37; 1.26)	*L iners*	.34 (0; 1.26)
S intermedius	.66 (.51; .88)	*T socranscii*	.52 (0; 1.05)	*L acidophilus*	.31 (0; .55)
C nigricans	.65 (.29; 1.80)	*P mirabilis*	.52 (0; .85)	*S agalactiae*	.30 (0; 1.33)
F n sp. polym.	.62 (.42; .87)	*S aureus* (yellow)	.52 (0; .81)	*B longum*	.29 (0; .52)
L buccalis	.62 (.46; .83)	*P anaerobius*	.48 (0; 1.69)	*P disiens*	.27 (0; 1.12)
A israelii	.61 (.48; .78)	*S gordonii*	.48 (0; .71)	*B ureolyticus*	.26 (0; .58)
P bivia	.61 (.24; 1.52)	*S agalactiae*	.47 (0; .72)	*A vaginalis*	.25 (0; .63)
F n sp. nucleatum	.58 (.44; .81)	*P gingivalis*	.45 (0;.79)	*P anaerobius*	.23 (0; .57)
T socranscii	.58 (.35; 1.54)	*P disiens*	.43 (0; .73)	*H influenza*	.23 (0; .54)
B longum	.54 (.32; 8.43)	*P bivia*	.41 (0; .67)	*S aureus* (white)	.20 (0; .39)
T forsythia	.52 (.39; .67)	*S aureus* (white)	.37 (0; .73)	*E faecalis*	.16 (0; .34)
C gingivalis	.49 (.37; .69)	*P acnes*	.35 (.18; .59)	*P aeruginosa*	.16 (0; .35)
S constellatus	.49 (.39; .67)	*L iners*	.34 (0; .67)	*C gracilis*	0 (0; 2.65)
P gingivalis	.48 (.41; .63)	*L vaginalis*	.32 (.16; .45)	*P intermedia*	0 (0; 1.07)
C rectus	.47 (.38; .89)	*H influenza*	.27 (0;.48)	*S intermedius*	0 (0; .76)
E faecalis	.46 (.25; 1.69)	*L cispatus*	.26 (.15; .42)	*S mutans*	0 (0; .71)
P disiens	.42 (.18; 2.54)	*B longum*	.24 (.09; .38)	*A israelii*	0 (0; .67)
P anaerobius	.41 (.28; .68)	*C ochracea*	.07 (0; .85)	*A a (a) 29,523*	0 (0; .54)
A parvulum	.378 (.13; 1.29)	*N mucosa*	0 (0; 7.45)	*S constellatus*	0 (0; .48)
L cispatus	.37 (.15; .63)	*P aeruginosa*	0 (0; 4.08)	*P bivia*	0 (0; .40)
H influenza	.34 (.16; 1.13)	*S pneumoniae*	0 (0; 1.51)	*P gingivalis*	0 (0; .39)
A christensenii	.34 (.15; 1.53)	*T forsythia*	0 (0; 1.47)	*A parvulum*	0 (0; .33)
A neuii	.33 (.15; 1.76)	*S sanguinis*	0 (0; 1.23)	*P endodontalis*	0 (0; .31)
C pseudogenitalium	.33 (.17; 1.32)	*E coli*	0 (0; .71)	*P acnes*	0 (0; .27)
C sputigena	.31 (.25; .45)	*A christensenii*	0 (0; .66)	*C nigricans*	0 (0; .26)
C gracilis	.30 (.17; .75)	*A naeslundii 1 and 2*	0 (0; .55)	*Peptoniphilus sp.*	0 (0; .25)
S aureus (white)	.30 (.19; 1.47)	*S noxia*	0 (0; .39)	*B breve*	0 (0; .21)
M curtisii	.29 (.17; .49)	*B biavatii*	0 (0; .36)	*L cispatus*	0 (0; .19)
P acnes⁎	.29 (.24; .47)	*V cambriensis*	0 (0; .32)	*A neuii*	0 (0; .17)
A vaginalis	.26 (.14; 1.47)	*P endodontalis*	0 (0; .22)	*C pseudogenitalium*	0 (0; .15)

Please cite this article in press as: Ines Kapferer, et al., Tongue Piercing: The Impact of Material on Microbiological Findings, J Adolesc Health (2010), doi: 10.1016/j.jadohealth.2010.10.008

Table 3
Continued

Tongue		Piercing channel		Studs	
Bacterial species	Relative microbial counts (%)	Bacterial species	Relative microbial counts (%)	Bacterial species	Relative microbial counts (%)
B bifidum	.26 (.14; .52)	*A israelii*	0 (0; .18)	*B biavatii*	0 (0; .14)
B breve	.24 (.14; .58)	*A odontolyticus*	0 (0; 0)	*M curtisii*	0 (0; .13)
S aureus (yellow)	.23 (.09; 1.31)	*A neuii*	0 (0; 0)	*M mulieris*	0 (0; .11)
Peptoniphilus sp.	.22 (.16; 1.14)	*A parvulum*	0 (0; 0)	*T denticola*	0 (0; .09)
T denticola	.19 (.14; .32)	*B bifidum*	0 (0; 0)	*C ochracea*	0 (0; .04)
M mulieris	.17 (.101; .31)	*B breve*	0 (0; 0)	*T forsythia*	0 (0; 0)
B biavatii	.17 (.11; .41)	*C nigricans*	0 (0; 0)	*A christensenii*	0 (0; 0)
P endodontalis	.16 (.09; .29)	*C pseudogenitalium*	0 (0; 0)	*B bifidum*	0 (0; 0)
V cambriensis	.13 (.08; .23)	*M curtisii*	0 (0; 0)	*S aureus* (yellow)	0 (0; 0)
C ochracea	0 (0; .79)	*M mulieris*	0 (0; 0)	*V cambriensis*	0 (0; 0)

(median: .82 × 10^4 cells/mm^2) than on all other studs (Ti: .34 × 10^4 cells/mm^2, PP: .09 × 10^4 cells/mm^2, PTFE: .02 × 10^4 cells/mm^2). Of the 18 species 13 were found at significantly higher levels ($p < .001$) on SS than on all other materials. These included: *Actinomyces neuii, A odontolyticus, A israelii, A naeslundii type I and II, Bifidobacterium breve, Corynebacterium sputigena, Fusobacterium periodonticum, Lactobacillus iners, L vaginalis, Peptostreptococcus anaerobius, Prevotella disiens, S noxia, and V parvula* (Table 4). Additionally, of the 80 species, 67 were found at significantly higher levels ($p < .001$) in samples from SS than from PTFE and PP (Table 4). Twenty-eight of these species were also found at significantly higher levels ($p < .001$) in samples from Ti than from PTFE and PP (Table 4). There were no statistically significant differences between samples from PTFE and PP.

Discussion

Previous studies [5,7,25,26] have shown that the occurrence of gingival recession is one of the main effects of the use of tongue piercings, whose prevalence can vary from 19.2% to 55%. The present study demonstrated the occurrence of lingual recession in 28.8%, and a prevalence of tooth chipping in 5%. A higher incidence of tooth fractures (19.2%–26.7%) has been reported in previous studies [5–7]. These reported differences can be attributed to the different methodologies used, and possible co-factors like piercing material, barbell stem length, and time of wear [5]. Additionally, we had a very young study population (22.74 ± 4.47), which might be a bias of the study. Given the fact that the popularity of having piercings in oral tissue parts is more common among young subjects, the authors considered that the present study population represented the sub-population at the greatest risk for complications with piercing elements.

Consistent with the age of the study subjects, the periodontal health was excellent and no subject presented with periodontitis. The extent of gingivitis as defined by BOP was low and supported by the fact that dental plaque deposits were also low, suggesting a good oral hygiene level among the study subjects. This might explain the low prevalence of gingival recessions. Additionally, it seems reasonable that the counts of bacteria associated with periodontitis should be low. Nevertheless, the periopathogenic bacteria *A actinomycetemcomtans* (Y4), *F nucleatum* species, *P micra*, and *T denticola* were found at significantly higher proportions ($p < .001$) in samples from the piercing channel than from the tongue. This is in accordance with Ziebolz et al [4], who collected microbiological samples from the surface of 12 tongue piercings and analyzed them for the presence of 11 periodontopathogenic bacteria. Therefore, in subjects with periodontitis, the piercing might provide an additional reservoir for periopathogenic bacteria and should be removed in the course of periodontal treatment.

Recent data suggest that the colonization of bacteria from the back of the tongue seems to change in children and adolescent subjects approaching a microbiota similar to that in adults, and with the presence of bacteria associated with periodontitis [27]. Similar bacteria can be identified from bacterial samples obtained from the back of the tongue and in periodontal pockets (sulci) [28]. Such studies have focused on the 40 bacterial species assessed with the checkerboard hybridization assay [22]. In the present study, the assay was expanded to include other bacteria that are known to be associated with biofilm development on foreign metallic and plastic surfaces.

The dominating bacteria from the tongue samples did not include species associated with periodontal disease. As expected, we found not only *Streptococcus oralis, V parvula*, and *Neisseria mucosa*, but also *Streptococcus pneumoniae, S haemolyticus*, and *Paeruginosa. S pneumoniae* is commonly found in the upper respiratory tract. *S haemolyticus* and *P aeruginosa* could potentially be important pathogens in biofilm development on foreign materials inserted in the oral cavity, such as tongue and lip piercing devices, or dental implants [29,30], and in association with periodontitis [31]. *Staphylococci* sp. have been associated with infections at dental Ti implants [32–34].

Of the 80 species, 67 were found at significantly higher levels ($p < .001$) in samples from SS than from PTFE and PP. These included periodontopathogenic bacteria like *Porphyromonas gingivalis, Prevotella intermedia, F nucleatum* sp., *C rectus* and *P micra*, as well as bacteria associated with systemic infections (e.g., *S aureus*, *E corrodens*, alpha- and beta-hemolytic *Streptococci, Enterococcus faecalis, Haemophilus influenza*, and *P aeruginosa*). Also for local piercing infections, the most commonly found causal agents are *S aureus* and *Pseudomonas* sp. [35].

In conclusion, the present study demonstrated low bacterial counts at studs and in piercing channels. Different groups of bacteria are found at higher counts on studs and piercing channels. We confirmed that differences in bacterial colonization patterns occur for different stud materials. Studs made of SS might promote the development of a biofilm, whereas studs made of PTFE and PP may be rather inert to bacterial colonization. The finding of *Staphylococci* on SS and Ti studs may

Please cite this article in press as: Ines Kapferer, et al., Tongue Piercing: The Impact of Material on Microbiological Findings, J Adolesc Health (2010), doi: 10.1016/j.jadohealth.2010.10.008

Table 4
Distribution of the 67 bacterial species found at significantly higher levels ($p < .001$) in samples from stainless steel than from PTFE and PP; median (25. percentile; 75. percentile)

Bacterial species	Stainless steel counts $\times 10^7$	Titanium counts $\times 10^7$	Polypropylene counts $\times 10^7$	PTFE counts $\times 10^7$
A israelii[ab]	3.98 (2.98; 6.19)	1.23 (0; 2.14)	0 (0; 0)	0 (0; 0)
A naeslundii 1 and 2[ab]	12.56 (7.51; 17.40)	3.41 (1.71; 7.72)	0 (0; 0)	0 (0; 1.19)
A neuii[b]	1.61 (.81; 2.14)	0 (0; .41)	0 (0; .20)	0 (0; 0)
A odontolyticus[ab]	6.55 (5.13; 11.33)	2.21 (1.59; 4.91)	0 (0; .61)	0 (0; 0)
A parvulum	2.92 (1.82; 4.63)	0 (0; 2.14)	0 (0; 0)	0 (0; 0)
A vaginae	6.40 (4.76; 14.78)	4.68 (2.12; 2.14)	0 (0; 2.54)	0 (0; 0)
B biavatii	.99 (.68; 1.69)	.18 (0; 2.14)	0 (0; .54)	0 (0; 0)
B bifidum	1.40 (0; 2.56)	0 (0; 0)	0 (0; 0)	0 (0; 0)
B breve[b]	2.17 (1.12; 2.63)	.51 (0; .79)	0 (0; 0)	0 (0; 0)
B longum	5.47 (1.99; 10.04)	1.23 (.69; 2.95)	0 (0; 1.35)	0 (0; .49)
B ureolyticus	2.24 (1.12; 5.56)	1.22 (.5; 2.14)	.40 (0; .80)	0 (0; .47)
C gingivalis[a]	12.22 (8.92; 22.97)	6.85 (4.32; 8)	0 (0; 0)	0 (0; 2.43)
C nigricans	2.65 (1.37; 4.64)	0 (0; 1.522)	0 (0; 0)	0 (0; 0)
C pseudogenitalium	1.66 (0; 3.66)	0 (0; .93)	0 (0; 0)	0 (0; 0)
C rectus	6.88 (4.07; 15.42)	3.75 (1.967; 7.04)	.58 (0; 2.59)	0 (0; 1.545)
C showae	13.18 (7.23; 28.49)	8.65 (3.06; 18.42)	0 (0; 4.12)	0 (0; 1.00)
C sputigena[b]	13.06 (4.51; 17.66)	3.92 (1.57; 2.14)	0 (0; 2.278)	0 (0; 0)
Dialister sp.	6.30 (3.42; 11.21)	2.77 (1.46; 10.44)	1.44 (0; 2.49)	.67 (0; 1.87)
E coli[a]	10.04 (5.28; 17.02)	3.60 (2.14; 7.34)	0 (0; 1.37)	0 (0; 0)
E corrodens	19.09 (15.44; 40.09)	13.05 (7.62; 2.14)	1.21 (0; 7.62)	0 (0; 0)
E faecalis[a]	2.42 (1.71; 5.69)	.85 (0; 2.24)	0 (0; .64)	0 (0; 0)
E saburreum	5.16 (3.31; 11.49)	1.71 (.95; 5.14)	.39 (0; 1.04)	0 (0; 1.19)
F n sp. naviforme (vincentii)	8.24 (4.29; 17.07)	3.12 (1.76; 2.14)	.29 (0; 1.70)	0 (0; .49)
F nucleatum sp. nucleatum	8.45 (3.47; 12.12)	3.78 (2.27; 2.14)	0 (0; 1.934)	0 (0; 0)
F n sp. polymorphum[a]	10.69 (8.29; 20.12)	4.14 (1.76; 2.14)	0 (0; 0)	0 (0; 0)
F periodonticum[ab]	11.51 (5.98; 19.58)	4.15 (2.36; 2.14)	0 (0; .87)	0 (0; 0)
G vaginalis	5.91 (3.29; 14.63)	2.33 (1.26; 7.58)	.72 (0; 2.42)	0 (0; 2.22)
H influenza[a]	2.09 (.75; 2.89)	1.10 (.46; 2.14)	.16 (0; .46)	0 (0; .25)
H pylori	2.30 (1.550; 5.71)	1.21 (.81; 2.14)	.48 (0; .98)	0 (0; .62)
L acidophilus[a]	9.01 (6.81; 12.74)	4.45 (1.99; 9.156)	0 (0; 0)	0 (0; 0)
L buccalis	6.73 (4.4; 13.24)	2.98 (1.39; 4.74)	1.57 (0; 2.87)	0 (0; 0)
L cispatus[a]	7.34 (5.53; 13.20)	3.77 (1.65; 2.14)	0 (0; 0)	0 (0; .84)
L gasseri[a]	3.79 (2.70; 7.74)	1.39 (1.05; 2.14)	.70 (.46; 1.51)	0 (0; 0)
L iners[ab]	3.73 (1.57; 5.71)	2.24 (.91; 2.14)	0 (0; .88)	.49 (0; .89)
L vaginalis[ab]	1.06 (.75; 2.41)	.67 (0; 2.14)	.49 (.22; .98)	0 (0; 0)
M curtisii	1.25 (.47; 2.11)	0 (0; .81)	0 (0; 0)	0 (0; 0)
N mucosa	37.63 (15.96; 109.01)	16.79 (0; 45.56)	0 (0; 8.589)	0 (0; 0)
P acnes	1.68 (0; 2.26)	.79 (0; 1.756)	0 (0; 0)	0 (0; 0)
P aeruginosa	14.11 (0; 31.92)	5.56 (0; 13.08)	0 (0; 0)	0 (0; 0)
P bivia	3.67 (1.83; 7.73)	0 (0; 1.61)	0 (0; 0)	0 (0; 0)
P disiens	4.06 (1.93; 13.14)	.91 (0; 2.01)	0 (0; .59)	0 (0; .89)
P endodontalis	1.68 (1.9; 3.39)	0 (0; 1.445)	0 (0; 0)	0 (0; 0)
P gingivalis	2.55 (1.46; 5.53)	.61 (0; 2.04)	0 (0; 0)	0 (0; 0)
P intermedia[a]	5.47 (1.40; 10.76)	.994 (0; 4.48)	0 (0; 0)	0 (0; 0)
P melaninogenica[a]	20.21 (9.12; 36.03)	4.53 (3.27; 17.26)	.72 (0; 1.95)	0 (0; .55)
P micra	13.25 (7.06; 28.14)	6.20 (3.66; 14.92)	0 (0; 2.45)	0 (0; 0)
P mirabilis[a]	9.33 (4.19; 17.01)	4.04 (1.59; 7.29)	.789 (0; 1.45)	.53 (0; 1.29)
Peptoniphilus sp.	1.37 (.55; 2.30)	.26 (0; 1.15)	0 (0; 0)	0 (0; 0)
S oralis[a]	2.30 (1.74; 4.24)	1.25 (.93; 2.14)	.86 (0; 2.67)	0 (0; .37)
S agalactiae[a]	3.98 (2.64; 6.51)	2.49 (0; 3.59)	.39 (.06; .87)	0 (0; 1.44)
S anaerobius	3.77 (3.09; 4.51)	2.35 (1.2; 2.14)	0 (0; 1.58)	0 (0; .93)
S anginosus	3.99 (2.49; 6.05)	1.79 (0; 2.14)	0 (0; 1.26)	0 (0; .62)
S aureus	1.48 (1.12; 4.79)	.61 (.52; 2.14)	0 (0; 1.42)	0 (0; .29)
S aureus (white)[a]	0 (0; 1.99)	0 (0; 2.15)	0 (0; .46)	0 (0; 0)
S constellatus[a]	3.87 (3.20; 8.31)	1.40 (1.10; 2.14)	0 (0; 0)	0 (0; .62)
S epidermis[a]	5.28 (4.19; 8.03)	6.17 (2.26; 2.14)	.52 (.345; .81)	0 (0; 0)
S gordonii[a]	12.05 (8.86; 27.66)	9.53 (6.32; 2.14)	0 (0; 0)	1.77 (0; 7.96)
S intermedius	6.57 (4.75; 7.43)	4.52 (0; 8.34)	0 (0; 0)	0 (0; .77)
S mitis	2.74 (1.83; 5.64)	1.58 (0; 3.82)	0 (0; 1.85)	0 (0; 0)
S noxia[ab]	11.50 (7.34; 16.56)	6.61 (3.34; 18.32)	0 (0; 1.26)	0 (0; 1.00)
S pneumoniae[a]	13.30 (11.13; 20.64)	6.88 (4.71; 14.59)	0 (0; 2.89)	0 (0; 0)
S sanguinis[a]	5.1 (3.23; 10.91)	5.41 (2.41; 9.934)	0 (0; 1.25)	0 (0; 0)
T forsythia	0 (0; 4.07)	0 (0; .52)	0 (0; 0)	0 (0; 0)
T socranscii	3.39 (2.42; 5.03)	1.52 (.64; 2.14)	.38 (0; .99)	0 (0; .37)
V parvula[a]	134.47 (51.26; 198.23)	34.44 (19.02; 67.38)	5.19 (.59; 9.70)	0 (0; 3.36)

[a] Bacterial species additionally found at significantly higher levels ($p < .001$) in samples from Ti than from PTFE and PP.
[b] Bacterial species additionally found at significantly higher levels ($p < .001$) in samples from SS than from Ti.

Please cite this article in press as: Ines Kapferer, et al., Tongue Piercing: The Impact of Material on Microbiological Findings, J Adolesc Health (2010), doi: 10.1016/j.jadohealth.2010.10.008

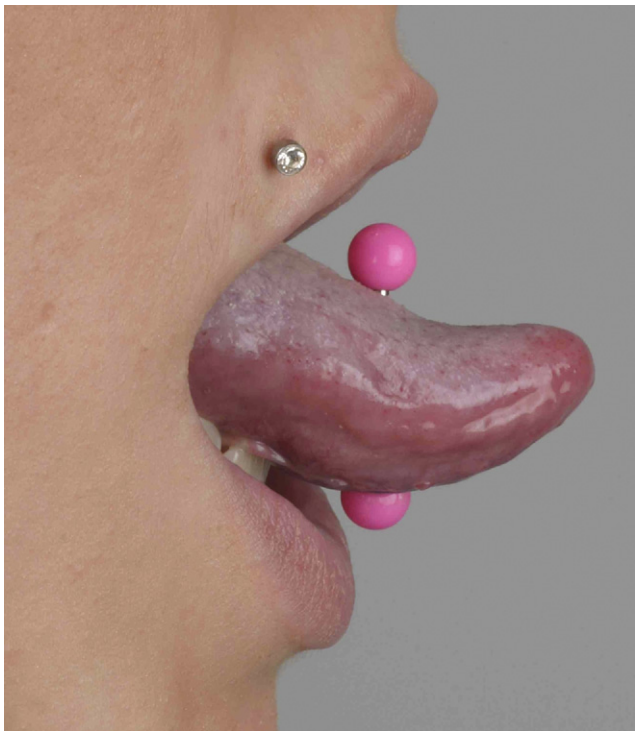

Figure 1. Piercing of the tongue (and the upper lip). For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.

suggest an elevated risk for complication if the piercing channel is infected. Figure 1.

Acknowledgments

The authors appreciate the laboratory work performed by Ms Marianne Weibel and Ms Regula Hirschi-Imfeld, Laboratory for Oral Microbiology, Department of Periodontology, School of Dental Medicine, University of Bern, Switzerland. This study was supported by a grant from the Austrian Society of Periodontology.

References

[1] Stirn A. Body piercing: Medical consequences and psychological motivations. Lancet 2003;361:1205–15.
[2] Price SS, Lewis MW. Body piercing involving oral sites. J Am Dent Assoc 1997;128:1017–20.
[3] Bone A, Ncube F, Nichols T, Noah ND. Body piercing in England: A survey of piercing at sites other than earlobe. BMJ 2008;336:1426–8.
[4] Ziebolz D, Hornecker E, Mausberg RF. Microbiological findings at tongue piercing sites: Implications to oral health. Int J Dent Hyg 2009;7:256–62.
[5] Campbell A, Moore A, Williams E, et al. Tongue piercing: Impact of time and barbell stem length on lingual gingival recession and tooth chipping. J Periodontol 2002;73:289–97.
[6] De Moor RJ, De Witte AM, De Bruyne MA. Tongue piercing and associated oral and dental complications. Endod Dent Traumatol 2000;16:232–7.
[7] Pires IL, Cota LO, Oliveira AC, et al. Association between periodontal condition and use of tongue piercing: A case-control study. J Clin Periodontol 2010;37:712–8.
[8] Gold MA, Schorzman CM, Murray PJ, et al. Body piercing practices and attitudes among urban adolescents. J Adolesc Health Care 2005;36:352–3.
[9] Armstrong ML, DeBoer S, Cetta F. Infective endocarditis after body art: A review of the literature and concerns. J Adolesc Health 2008;43:217–25.
[10] Bruns AS, Sood N. Community-acquired methicillin-resistant *Staphylococcus aureus* epidural abscess with bacteremia and multiple lung abscesses: Case report. Am J Crit Care 2009;18:86–8.
[11] Jadhav AR, Belfort MA, Dildy GA, 3rd. Eikenella corrodens chorioamnionitis: Modes of infection? Am J Obstet Gynecol 2009;200:e4–5.
[12] Lakhan SE, Harle L. Fatal fulminant herpes simplex hepatitis secondary to tongue piercing in an immunocompetent adult: A case report. J Med Case Reports 2008;2:356.
[13] Delarocque-Astagneau E, Pillonel J, De Valk H, et al. An incident case-control study of modes of hepatitis C virus transmission in France. Ann Epidemiol 2007;17:755–62.
[14] Bader MS, Hamodat M, Hutchinson J. A fatal case of *Staphylococcus aureus*: Associated toxic shock syndrome following nipple piercing. Scand J Infect Dis 2007;39:741–3.
[15] Martinello RA, Cooney EL. Cerebellar brain abscess associated with tongue piercing. Clin Infect Dis 2003;36:e32–34.
[16] Carlsson L, Rostlund T, Albrektsson B, et al. Osseointegration of titanium implants. Acta Orthop Scand 1986;57:285–9.
[17] Anonymous. TeflonO industrial [online]. Available at: http://www2.dupont.com/Teflon_Industrial/en_US/index.html. Accessed November 12, 2009.
[18] Anonymous. Polypropylene BormedTM HD810MO. Available at: http://www.borealisgroup.com/datasheets/10015489. Accessed November 12, 2009.
[19] Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. Int Dent J 1975;25:229–35.
[20] O'Leary TJ, Drake RB, Naylor JE. The plaque control record. J Periodontol 1972;43:38.
[21] Katsoulis J, Heitz-Mayfield LJ, Weibel M, et al. Impact of sample storage on detection of periodontal bacteria. Oral Microbiol Immunol 2005;20:128–30.
[22] Socransky SS, Smith C, Martin L, et al. "Checkerboard" DNA-DNA hybridization. BioTechniques 1994;17:788–92.
[23] Socransky SS, Haffajee AD, Smith C, et al. Use of checkerboard DNA-DNA hybridization to study complex microbial ecosystems. Oral Microbiol Immunol 2004;19:352–62.
[24] Persson GR, Hitti J, Paul K, et al. *Tannerella forsythia* and *Pseudomonas aeruginosa* in subgingival bacterial samples from parous women. J Periodontol 2008;79:508–16.
[25] Levin L, Zadik Y, Becker T. Oral and dental complications of intra-oral piercing. Dent Traumatol 2005;21:341–3.
[26] Kieser JA, Thomson WM, Koopu P, Quick AN. Oral piercing and oral trauma in a New Zealand sample. Dent Traumatol 2005;21:254–7.
[27] Papaioannou W, Gizani S, Haffajee AD, et al. The microbiota on different oral surfaces in healthy children. Oral Microbiol Immunol 2009;24:183–9.
[28] Do Nascimento C, Sato S, Mardegan Issa JP, et al. DNA checkerboard method for bacterial detection of microbiota from teeth and tongue biofilms. A preliminary study. Minerva Stomatol 2008;57:561–7.
[29] Rampioni G, Schuster M, Greenberg EP, et al. Contribution of the RsaL global regulator to *Pseudomonas aeruginosa* virulence and biofilm formation. FEMS Microbiol Lett 2009;301:210–7.
[30] Van de Velde T, Thevissen E, Persson GR, et al. Two-year outcome with Nobel Direct implants: A retrospective radiographic and microbiologic study in 10 patients. Clin Implant Dent Relat Res 2009;11:183–93.
[31] Persson GR, Weibel M, Hirschi R, Katsoulis J. Similarities in the subgingival microbiota assessed by a curet sampling method at sites with chronic periodontitis. J Periodontol 2008;79:2290–6.
[32] Kronstrom M, Svenson B, Hellman M, Persson GR. Early implant failures in patients treated with Branemark System titanium dental implants: A retrospective study. Int J Oral Maxillofac Implants 2001;16:201–7.
[33] Salvi GE, Furst MM, Lang NP, Persson GR. One-year bacterial colonization patterns of *Staphylococcus aureus* and other bacteria at implants and adjacent teeth. Clin Oral Implants Res 2008;19:242–8.
[34] Pye AD, Lockhart DE, Dawson MP, et al. A review of dental implants and infection. J Hosp Infect 2009;72:104–10.
[35] Guiard-Schmid JB, Picard H, Slama L, et al. Piercing and its infectious complications. A public health issue in France. Presse Med 2000;29:1948–56.

Please cite this article in press as: Ines Kapferer, et al., Tongue Piercing: The Impact of Material on Microbiological Findings, J Adolesc Health (2010), doi: 10.1016/j.jadohealth.2010.10.008