3489 Assessment of Passive Smoking and Risk for Periodontal Disease

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Tobacco smoking has been implicated as a significant risk factor for both prevalence and severity of periodontal disease. However, this relationship has been examined in history of current and former smokers. On the other hand, the effect of passive smoking on periodontal disease has not yet been studied. Therefore, we examined the relationship between periodontal disease and passive cigarette smoking utilizing data from the Third National Health and Nutrition Examination Survey (NHANES III) conducted in 1988 to 1994, a national representative sample of 11,162 non-institutionalized subjects ages 20-90 years. Passive smoking was ascertained as exposure to tobacco smoke in the household. Periodontal status was determined by measurement of clinical attachment loss (CAL) in two sites of all teeth in two randomly selected quadrants, only subjects with at least six natural remaining teeth were included in the analysis. The effect of passive smoking was examined on severity of periodontal disease defined as mean CAL and presence of gingival bleeding ≥ 20% of gingival units. A weighted logistic regression model showed that for subjects who were exposed to household tobacco smoke, the risk for more severe CAL (≥ 1.5 mm mean CAL) was significantly increased compared to those who were non- exposed with odds ratio of 3.0 (95% CI 2.6 - 3.5) after adjusting for age, gender, race, education , income and diabetes mellitus. Important known risk factors for periodontal disease. In addition, a similar model indicated that individuals exposed to passive smoking were at higher risk for having greater gingival bleeding compared to those non-exposed, the odds ratio was 1.4 (95% CI 1.2 - 1.5). Results from this study indicate that passive smoking is associated with severity of periodontal disease, providing additional evidence for the negative effect of tobacco smoke on the periodontium. Longitudinal studies are needed to further examine the role of passive smoking on periodontal disease. Supported by USPHS Grant No. DE04898.

3490 Effects of Smoking and Treatment Status on Periodontal Marker Bacteria

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Smoking is recognized as a risk factor in the development of periodontitis. We were able to examine the influence of treatment status and of smoking status on the occurrence of specific microbiological marker bacteria in two groups of periodontal patients. We employed a commercial immunoassay (Evalusite, Kodak Canada Inc.) for semi-quantitative detection of the microbial marker species *Prevotella intermedia, Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans* in pocket samples. We report findings for 55 patients of F-ME and 179 patients of MHM tested with the immunoassay. Patients were identified as smokers on the basis of a history of any smoking within the preceding 6 months (F-ME 13 Smokers, 42 Non-Smokers, MHM 70S, 109 NS). Patients with a history of regular periodontal maintenance treatment were assigned to a Recall group. Patients with no history of active periodontal maintenance were assigned to a New treatment group (F-ME 19 New, 36 Recall, MHM 100 N, 79 R).

Smoking and treatment status were unrelated to gender and age of the patient. Smoking and treatment status were also unrelated to average depths of sampling sites chosen for the study. We found consistent and significant trends (χ^2 analysis for trend $p < 0.05$) toward increased proportions of marker-free individuals in treated versus untreated non-smoking patients that were not observed in smokers. This effect was seen consistently for *P. gingivalis* and *P. intermedia*, but only in the F-ME patients for *A. actinomycetemcomitans*. By averaging measurements of all 3 markers for each patient we found that 36 to 50% of treated non-smokers present with microbial marker-free dentitions while 12 to 25% of treated smokers had marker-free mouths. Clearance of microbial markers can be observed in treated non-smokers. Smoking appears to interfere with the ability of periodontal patients to suppress the three marker organisms following conventional periodontal therapy. This effect of smoking complicates periodontal therapy through an inhibition of the beneficial effects of treatment on the clearance of microbial marker organisms.

3491 Association of Periodontal Disease and Chronic Lung Disease: Analysis of NHANES III.

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Associations between poor oral hygiene, periodontal disease and chronic lung disease (CLD) have recently been reported. The goal of the present study was to evaluate the potential associations between respiratory diseases and oral health status by examining data from the National Health and Nutrition Examination Survey III (NHANES III). The database from NHANES III, which documents the general health and nutritional status of individuals randomly selected from the continental United States from 1988 to 1994, was analyzed. Of the subjects surveyed, 13,792 having an age ≥20 years and with at least 6 natural teeth received a standardized dental examination. A history of pneumonia, asthma, bronchitis and/or emphysema were obtained from the self-reported survey questionnaire. In addition, a dichotomized variable (CLD) was constructed by combining those who had either chronic bronchitis and/or emphysema. Unweighted analyses were used for initial examination of the data, and a weighted analysis using WesVar statistical software was performed in a final logistic regression model adjusting for age, gender, race and ethnicity, education, income, frequency of dental visits, smoking and alcohol consumption. Results found the age of all subjects was 44.4 ± 17.8 years (mean and S.D.), the age of subjects with CLD was 51.2 ± 17.9 years, and the mean age of subjects without CLD was 43.9 ± 17.7 years. Those with CLD had more periodontal attachment loss (AL) than those without CLD (1.48 ± 1.35 mm vs. 1.17 ± 1.09, p= 0.0001). Subjects with mean AL ≥2.0 mm had a higher risk of CLD than those who had mean AL < 2.0 mm (odds ratio of 1.43, 95% confidence intervals of 1.08-1.90). The findings of the present analysis support recently published studies that suggest a relationship between periodontal attachment loss and CLD. (Supported by NIDR grant DE12386).

3492 Preliminary Evaluation of Periodontal Status of WIHS Women.

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Two year longitudinal data from a multi center, national cohort of 539 HIV⁺ and 142 HIV⁻ women participating in the Women's Interagency HIV Study (WIHS) was collected using the following indices: gingival banding (GB), papillary assessment (PA), plaque index (PI), pocket depth (PD) and loss of attachment (LA). Results: Mean of teeth-surfaces/subject- HIV⁻ 46.41 (SD=1.05), HIV⁺ 43.15 (SD= 5.4). Mean PD - HIV⁻ 2.07mm (SD=.04) and HIV⁺ 2.07mm (SD= .02). Average LA- HIV⁻ 1.85mm (SD= .08) and HIV⁺ 1.82mm (SD=.04). Maximum PD- HIV⁻ 4.10mm (SD=.12) and HIV⁺ 3.81mm (SD= .05). Maximum LA- HIV⁻ 4.52mm (SD=.19) and HIV⁺ 4.12mm (SD=.10). PI declined over time for both groups, faster for the HIV⁻ (P= .001). PD analysis showed no difference between the two groups. GB was evaluated as present or not, and 12% of both groups had it. HIV⁻ women had a declining prevalence over time (P= .0306) while GB increased in HIV⁺ (P= .0538). Difference between them was highly significant (P=.0087). With PA, 91% had erythema with no difference between the two groups and no significant differences over time. 50 to 70% had edema with tendency to increase over time which was significantly higher (P= .0001) in HIV⁺. Based on the limitations of this analysis and the short time of evaluation (4 six month-visits) the conclusions are: a. The progress of chronic periodontitis does not appear to be influenced by the HIV infection, its complications and therapy. b. HIV⁻ women had better plaque control over time. c. GB had a higher incidence over time in HIV⁺ women; d. Papillary erythema had the highest common prevalence (91%) and edema (50 to 70%) increased significantly in time in HIV⁺ women.

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3493 Risk Factors for Incident Periodontitis in HIV Infection.

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The purpose of this study was to conduct an exploratory bivariate analysis of clinical and socio-demographic risk factors for new and progressive periodontal disease in persons with HIV infection. The study consisted of 180 dentate HIV-infected participants of a longitudinal study who returned for their first annual follow-up visit. Loss to follow-up was 55%, 20% of which were deaths. There were no significant differences in mean, severity, or extent of periodontitis measures at baseline for those returning and those lost to follow-up. Of those returning, 76% were males, 63% were Black. Median age at follow-up was 39 years (range 19-65) and mean CD4+ cell count was 307 cells /µl (SD = 255). Six sites for all erupted teeth, excluding 3rd molars, were measured by one calibrated examiner at baseline and follow-up for probing pocket depth (PPD) and recession (REC). Clinical attachment level (CAL) was calculated from PPD and REC, and attachment loss (ALOSS) was the difference between baseline and follow-up measures of CAL. Incident PPD and ALOSS required an increase of 3mm from baseline to follow-up in at least one site. Incident REC required an increase of 2mm from baseline to follow-up. Forty-four percent of participants had incident PPD, 53% incident ALOSS, and 47% incident REC. Significant predictors of incident periodontitis were similar for all measures in bivariate analyses: refractory or recurrent candidiasis and self-report of current tobacco use. Candidiasis at baseline and follow-up increased the relative (RR) risk of incident ALOSS 1.8 times (95% CI=1.4, 2.3) and doubled the RR of incident PPD (RR=2.0, 95% CI = 1.5, 2.8) and REC (RR=2.1, 95% CI=1.6, 2.7). Self-reported tobacco use increased the RR over two-fold for incident ALOSS (RR=2.6 95%CI= 1.4, 4.6), REC (RR=2.2, 95%CI=1.2,4.0), and PPD (RR=2.4, 95%CI=1.3, 4.5). Neither HIV therapeutic medications nor CD4+ cell counts modified the risk of incident periodontitis. This analysis suggests that recurrent or refractory candidiasis and tobacco use may be risk factors for incident periodontitis in HIV infection. Supported by NIH DE11369

3494 Effect of systemically administered metronidazole on subgingival plaque composition.

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The purpose of the present investigation was to evaluate changes in the subgingival microbiota during and after the systemic administration of metronidazole. Nine subjects with adult periodontitis entered the study and after baseline clinical and microbiological monitoring, received full mouth SRP and metronidazole at the dosage of 250 mg 3 times a day for 14 days. Clinical measures included % of sites with plaque, gingival redness, BOP and suppuration as well as pocket depth and attachment level. Subgingival plaque samples were taken from the mesial surface of up to 28 teeth in each subject at baseline and at 90 days. In addition, plaque samples were taken from 2 randomly selected teeth at 3, 7 and 14 days during and after antibiotic administration. Counts of 40 subgingival species were determined using checkerboard DNA–DNA hybridization and a Storm Fluorimager. Significance of differences for each species and clinical measures over time were determined using the Quade test.

Mean pocket depth (mm) was significantly reduced from 3.3 ± 0.2 at baseline to 3.0 ± 0.2 at 90 days ($p < 0.05$). Further, the subjects exhibited a marked reduction in the levels and proportions of 3 species of the "red complex", *B. forsythus, P. gingivalis* and *T. denticola* during the antibiotic administration which was maintained for 90 days. For example, counts (x 10^5, ± SEM) of *P. gingivalis* fell from 7.8 ± 5.0 to 0.1 ± 0.04 during antibiotic administration and remained at approximately this level at 90 days. *B. forsythus* also showed a dramatic reduction in counts during the short term phase, from 1.5 ± 0.3 to 0.04 ± 0.02 with a slight increase after 3 months to 0.12 ± 0.04. Some members of the "orange complex" such as *F. nucleatum* ss. *vincentii, F. nucleatum* ss. *nucleatum, F. periodontaticum* and *P. nigrescens* were inhibited by metronidazole for 14 days but started to increase as soon as the antibiotic was withdrawn. Counts of *Actinomyces* and *Streptococcus* sp. did not change over time. The systemic administration of metronidazole had a selective effect on the subgingival microbiota, affecting species most strongly associated with adult forms of periodontal disease such as *B. forsythus, P. gingivalis* and *T. denticola*.

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3495 Systemic metronidazole kills bacteria found within the dentinal tubules.

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Bacteria are known to invade the dentinal tubules of root surfaces. This location could serve as a reservoir from which these bacteria could recolonize the root surfaces following mechanical debridement. Four double-blind studies have established that systemic metronidazole plus scaling and root planing is statistically superior to systemic placebo plus scaling and root planing in reducing pocket depths, causing an increase in attachment levels and in reducing the need for access surgery. Part of the success of metronidazole could be that it penetrates the dentinal tubules either from the pulp side or from the plaque side and kills these bacteria in situ. Pilot studies showed that metronidazole and hydroxy-metronidazole could be found in pulp tissue (5 teeth) of 2 individuals who took metronidazole the day before extraction, but that only 9 pulps of 42 examined (15 patients) had traces of metronidazole, when the metronidazole was taken 1 hour prior to extraction. In the present investigation, patients were identified who had teeth scheduled for extraction for periodontal purposes. They were then randomly given unmarked bottles containing 14 tablets of either metronidazole (500mg) or placebo and instructed to take the tablets b.i.d. for one week. Compliance with this regimen was poor, and only 8 subjects, 4 in each group, completed the protocol giving rise to 14 teeth that were extracted. The teeth were debrided and then split in half using aseptic techniques. The pulp as well as 2 to 4 layers of dentine, beginning from the pulp side of the root and progressing to the plaque side were cultured using a quantitative anaerobic procedure. The pulps from the metronidazole treated patients averaged 706 ± 1219 CFUs/mg wet weight compared to 5,514 ± 6,342 CFUs/ mg in the placebo treated patients. In the dentinal layer adjacent to the pulp there were 44 ± 93 CFUs/mg in the metronidazole group compared to 849 ± 1545 in the placebo treated patients. These data suggest that one reason for the efficacy of systemic metronidazole in the treatment of periodontal disease is its ability to penetrate, via the pulp, the dentinal tubules, killing in situ those bacteria present within the tubules. Supported by AADR Student Research Fellowship

3496 The Effect of Scaling, Root planing and Systemic Metronidazole in Surgically Treated Refractory Periodontitis in a Five Year Study.

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The aim was to determine the effect of scaling, root-planing and systemic metronidazole in surgically treated refractory periodontitis subjects. The material consisted of 64 subjects, 37 smokers and 27 non-smokers, mean age 41.3 (±2.9 SD) yr. After initial scaling and root planing the subjects were randomly allotted to experimental or placebo groups: 400 mg metronidazole each or a placebo was administered at 8-hour intervals for one week. All subjects were treated with scaling and root planing every 6 months during 5 years. Periodontal surgery was performed by modified Widman flap operations. Clinical parameters and percentage of remaining bone, (BH%) were determined at the start and end of the study. Paired Students *t* - test and Mann Whitney test were the statistics used.

During the 5-years, 16 subjects required only non-surgical therapy and 48 periodontal surgery complemented with scaling and root planing. The number of teeth included in the periodontal surgery were not significant (NS) between experimental and placebo groups. BH% increased significantly ($p<0.05$) between the start and end of the study for non-smokers in the experimental and placebo groups from mean 80.5 (±2.0 SE) % to 82.7 (±1.6 SE) % and 81.1 (±0.9 SE) % to 82.8 (±0.8 SE) %, respectively. The changes in BH% between the start and end of the study in smokers in the experimental as well as in the placebo group were not significant (NS).

The subjects who required surgery had significantly more ($p<0.01$) and deeper pockets ($p<0.01$) at baseline than those who only got non-surgical treatment.

Conclusions: Decisive factors in the sustained long-term improvement are probably regular follow-ups examinations at 6-month intervals for oral hygiene, scaling and root planing.

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