

Apatzidou DA, Riggio MP, et al. Impact of smoking on the clinical, microbiological and immunological parameters of adult patient with periodontitis. J Clin Periodontol 2005; 32:973-83.

Purpose: This study was done to assess the impact of smoking on the clinical indices, the humoral immune response and the subgingival microflora of patients before and after periodontal therapy.

Materials and Methods: 40 patients out of 58, age 31 to 70 yr old, took part in this study, 18 were excluded on the basis of intake of antibiotics and failure of attending appointment, 15 smokers and 25 nonsmokers. Each patient had at least 2 non adjacent sites per quadrant with a PD of 5mm or over and radiographic evidence of bone loss with no history of systemic disease or antibiotic therapy within the last 3 months or during the course of study. Cigarette smoking status was self reported by the patient at the screening visit and was confirmed by the cotinine enzyme inhibition assay (CEIA). Subjects were considered smokers if they had been smoking 5 or more cigarettes a day. After initial visit, patients were randomly allocated into one of the two treatment groups based on a pre determined randomization list made by computer and baseline measurements were recorded. Subsequently, same day full mouth S/RP or quadrant S/RP at 2 weekly intervals were performed on each patient under local anesthesia using an assortment of periodontal curettes and ultrasonic scalers. Pockets were irrigated with saline. During the 6 month of study, OHI was re-enforced as needed. Full mouth periodontal pocket chartings were completed at baseline (BAS) and at 6 month reassessment (RAS). PD and CALs were determined at 6 sites/tooth using PCP 12 probe. BOP was recorded as absent or present. 1 site/quadrant with the deepest PD and not less than 5mm deep and with no endodontic or furcation involvement was selected from each patient at BAS for selected site clinical assessment. At each selected site, the modified gingival index MGI and PeI, BOP, PD and relative attachment level (RAL) were recorded. Each site was measured twice to assess the variability of probing measurement. GCF samples were harvested and subgingival plaque samples were collected for the detection of 5 putative periodontal pathogens. *A.a*, *P. gingivalis*, *T. forsythensis*, *P. intermedia* and *T. denticola* by polymerase chain reaction (PCR). Blood samples were collected from all participants to determine the serum antibody titer.

Findings: At baseline, the whole mouth clinical parameters were similar for smokers and non smokers. Similarly, no significant differences between the 2 groups were found at RAS. At selected sites a compromised treatment outcome was seen similar in terms of PD reduction and gain in RAL. At RAS, smokers presented higher PD and attachment loss than did non smokers. Lower MGI but similar PII were found for smokers at BAS. At this point BOP was less severe in smoker than non smokers. Also, GCF volume was significantly lower for smokers. After treatment no statistical difference was found between 2 groups while the longitudinal study shows that clinical parameters within each group showed significant improvement in all of them. No significant differences in the detection of putative periodontal pathogens in subgingival plaque existed between smokers and non smokers. A consistent trend was noted in that smokers had lower sera IgG antibody titer to these organisms before and after treatment for *A.a*.

Conclusions: This study shows that smokers with periodontal disease have a suppressed inflammatory response and seems to have an altered antibody response to

antigenic challenge than non smokers. The subgingival microflora of smokers appears similar to the non smokers.

Shimazaki Y, Saito T, Kiyohara Y et al. Relationship between drinking and periodontitis: the Hisayama study. J Periodontol 2005; 76:1534-41.

Purpose: to examine the dose-response relationship between drinking and severity of periodontitis.

Materials and Methods: 961 individuals aged 40-79 years were included in this study, which was a part of Hisayama health study. Participants reported their daily alcohol intake on a self-administered questionnaire. Following categories were used: non-drinker (0 g/day), light drinker (0.1-14.9g/day), moderate drinker (15-29.9g/day), and heavy drinker (>30g/day).

Periodontal status was evaluated based on probing depth (PD) and clinical attachment loss (CAL). PD categories: none= no teeth with $PD \geq 4$ mm, low=0.1-19.9% teeth with $PD \geq 4$ mm, mid=20-34.9% teeth with $PD \geq 4$ mm, high= $\geq 35\%$ teeth with $PD \geq 4$ mm. CAL categories: none= no teeth with $CAL \geq 5$ mm, low=0.1-9.9% teeth with $CAL \geq 5$ mm, mid=10-21.9% teeth with $CAL \geq 5$ mm, high= $\geq 22\%$ teeth with $CAL \geq 5$ mm

Findings: Univariate analysis: the more the subjects' alcohol consumption, the greater proportion of their teeth had $PD \geq 4$ mm and $CAL \geq 5$ mm.

Multivariate analysis: moderate drinker (15-29.9g alcohol per day) has odd ratio = 2.7, 95% confidence interval vs. non-drinkers. Heavy drinkers (>30g alcohol per day) odd ratio =2.5 95% CI vs. non-drinkers. Both moderate and heavy drinkers had a higher risk of generalized periodontal disease (>35% of dentition with $PD > 4$ mm) versus non drinkers. No significant relationship found in the multivariate analysis between drinking and CAL.

Conclusions: Authors felt that moderate or heavy drinking is associated with periodontitis. The study data suggest that subjects who drink >15g of alcohol per day increased risk with generalized periodontitis.