Loss of Teeth and Coronary Heart Disease

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Purpose: This study examined the possible association of three dental factors with total mortality and death from coronary heart disease. ***Materials and Methods:*** Samples from two studies were combined, for a total of 2,613 individuals aged 25 to 79 years; a total of 353 deaths occurred, of which 82 were from coronary heart disease. The hazard ratio was calculated for total and coronary heart disease mortality by regression for the dental components; conventional risk factors were controlled for in a stepwise manner. ***Results:*** For total and coronary heart disease mortality, associations with both edentulousness and number of years of edentulism were statistically significant until smoking was added into the analysis; then, all significance was lost. When the effect of the oral parameters was studied in relation to total and coronary heart disease mortality, after adjusting for age and gender, there was a significant hazard ratio for total mortality, but only for edentulousness. When examined by stepwise regression of the coronary heart disease risk factors, all significance of risk from the three oral parameters was lost, smoking having the largest effect of all risk factors. ***Conclusion:*** Number of remaining teeth, edentulousness, and number of years of edentulism were not independent risk factors for total or coronary heart disease mortality, but they were surrogate markers for the risk from smoking. *Int J Prosthodont 2004;17:441–446.*

Interest in periodontal disease as a possible contributing factor to coronary heart disease (CHD) has increased in recent years. A number of studies have implicated periodontal disease in the development of CHD, although they are far from being conclusive.[1–8] A meta-analysis showed a weak association between periodontal disease and the risk of CHD, although that study could not exclude the contribution of other cardiovascular risk factors to this association.[9] The role of inflammation as a causal or contributing factor in the development of arteriosclerosis has attracted increased interest in recent years.[10] Smoking is a known major risk factor for CHD. Its effect on the periodontium, leading to periodontitis, periodontal infection, and eventually loss of teeth, is generally accepted.[11–15]

An initial study found significant associations among: *(1)* being edentulous, *(2)* number of years of edentulism, and *(3)* number of remaining teeth, as well as smoking, age, gender, level of education, and geographic location.[11,16] The authors' hypothesis was that by correlating the three odontologic factors mentioned above, the nature of a presumed association with accepted risk factors for general death and death from CHD may be revealed.

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Materials and Methods

The populations used for this study have been described earlier.[11,16–19] In 1985 to 1987, an oral examination was carried out on a randomized population sample known as "Group V," which was invited for general health examination at the Heart Disease Preventive Clinic of the Icelandic Heart Society. The sample consisted of 1,025 men and women, aged 52 to 79 years, who had been living in Reykjavík on December 1, 1966, according to the national register. A trained nurse carried out the oral examination, registering missing and remaining teeth, using a light and dental mouth mirror. Participants also answered 33 questions on oral health and habits, asked by a trained interviewer, along with the standard questionnaire of the Icelandic Heart Society.[11,17,18] In 1989 to 1990, the authors participated in a second study under the auspices of the Heart Preventive Clinic. The sample was from the MONICA Project (MONItoring trends and determinants in CArdiovascular disease) of the World Health Organization and consisted of 1,548 men and women, aged 25 to 74 years, who, according to the national register, had been living in the respective areas on December 1, 1987.[16,19]

A trained oral hygienist using a dental probe and a mouth mirror performed an oral examination. Complete dentures; removable partial dentures; decayed, missing, and filled teeth; and fixed partial dentures were recorded. In the latter assessment, the Community Periodontal Index of Treatment Needs (CPITN) was included in the examination using the standard method.[20] A trained interviewer asked the same 33 questions on oral health and habits as had been used in the previous investigation.

Combined results from the oral examinations of both investigations, as well as results from oral and general health questionnaires, were compared with officially recorded death diagnoses of those who had died since the examinations were performed. For administrative reasons, records of final diagnosis of death were only available to the authors up to 1998. The national recording system has a 2-year lag regarding official causes of death. Hence, number of deaths without official diagnoses was given separately to the end of 2000.

Cox regression analysis was applied to assess the predictive power of the three variables—*(1)* number of remaining teeth based on the oral examinations; *(2)* complete edentulousness ("edentulous" was interpreted as not a single tooth remaining); and *(3)* number of years of edentulism (based on the answer to the interviewer)—on risk of death from all causes and from CHD, respectively. First, each of the three variables was tested, controlling only for age and gender. Second, each variable was tested while controlling stepwise for the following variables: cholesterol, systolic blood pressure, education (two groups), and smoking (three groups). A 0/1 variable was included for testing risk differences between the two studies. A proportional hazard ratio was tested for the three oral variables and for the 0/1 variable. The two continuous variables, number of years of edentulism and number of remaining teeth, which both included 0 values, were used as linear variables. Possible nonlinearity was tested by categorizing the variables into four values (three dummy variables). Levels of significance were chosen as $P < .05$.

Information on any death was available until the end of 2000, whereas diagnosis for the official cause of death was only available from the national registry until the end of 1998. For these reasons, follow-up was from 8 to 15 years.

Results

The proportional hazards assumption held for all three oral variables ($P > .10$ in all cases). The same applied to the 0/1 variable representing the two studies ($P > .05$). To examine whether these samples were representative for the general population and had power to detect an association with the endpoints under investigation, total and CHD mortality, the relationship of the CHD risk factors previously shown to correlate with dental status in these samples was examined.

The dental characteristics of the samples revealed that the rate of edentulousness was higher for women than for men and differed markedly between the two samples for the men but not for the women (Table 1).

Total mortality and death from CHD were higher for the men.[11] Approximately 3% of both genders of the combined groups had suffered death from CHD (1.5% of women, 5.0% of men). Total death in the sample was barely 14% (women 10%, men 17%; Table 1).

To examine the possible relationship of the dental status with CHD mortality and total mortality, the two cohorts were combined to increase the number of endpoints. There was no indication of differences in risk between the two studies ($P > .50$ in all cases). All of the above-mentioned factors were associated with total CHD mortality, with the exception of cholesterol and higher levels of education (the explanation is most likely thinning of the data[11,16]) (Table 2).

There was a significant hazard ratio for all three oral factors examined—number of remaining teeth, edentulousness, and number of years of edentulism—for total mortality, but edentulousness was the only oral factor that was also significantly associated with CHD death (Table 3). According to log likelihood, number of years of edentulism was the strongest of the three oral variables in predicting total mortality, whereas edentulousness was the strongest in predicting CHD mortality.

Table 1 Dental Characteristics and Total and Coronary Heart Disease (CHD) Mortality of Cohorts Examined

	Men				Women			
Age (y)	n	Edentulous (%)	Total death*	CHD death†	n	Edentulous (%)	Total death*	CHD death†
Group V‡								
52–54	83	19	7	2	75	28	4	2
55–64	236	34	48	12	211	39	15	1
65–74	156	56	64	25	162	68	41	3
75–79	40	65	31	8	60	72	37	7
MONICA§								
25–34	105	3	0	0	148	0	0	0
35–44	144	6	2	0	163	7	0	0
45–54	165	10	5	2	172	22	3	0
55–64	149	30	18	3	198	51	11	2
65–74	157	30	39	10	189	71	28	5
Total	1,235	31	214	62	1,378	39	139	20

*Until the end of 2000.
†Until the end of 1998.
‡For Group V, the men were examined in 1985 and the women in 1986.
§The MONICA cohort was examined in 1990.

Table 2 Characteristics and Hazard Ratio of All Causes of Mortality and Coronary Heart Disease (CHD) Mortality Adjusted for Some Established Risk Factors*

			All causes of mortality		CHD mortality	
Risk factor	Mean	Standard deviation	Hazard ratio	95% CI	Hazard ratio	95% CI
Age (at measurement, in y)	55.4	13.4	1.12	1.11–1.14	1.12	1.08–1.16
Female (%)	52.0	–	0.64	0.51–0.82	0.30	0.17–0.52
Smoking (%)						
Never smoked	40.8	–	1.00	–	1.00	–
Former smoker	28.7	–	1.46	1.11–1.91	2.00	1.08–3.73
Current smoker	30.5	–	2.25	1.72–2.95	3.77	2.02–7.01
Education (%)						
Primary school only	27.8	–	1.00	–	1.00	–
Secondary school or more	72.2	–	0.79	0.63–0.99	0.73	0.46–1.15
Cholesterol (mmol/L)	6.3	1.3	0.96	0.87–1.05	1.09	0.91–1.32
Systolic blood pressure (mm Hg)	132.0	20.0	1.01	1.00–1.02	1.02	1.01–1.03

*Results of a multivariable Cox regression.
CI = confidence interval.

Table 3 Hazard Ratio for All Causes of Mortality and Coronary Heart Disease (CHD) Mortality for Dental Factors with Stepwise Adjustment of Risk Factors

	Edentulousness*			No. of years of edentulism†			No. of remaining teeth‡		
Risk factor	Hazard ratio	95% CI	*P*	Hazard ratio	95% CI	*P*	Hazard ratio	95% CI	*P*
All causes of mortality									
Age and gender	1.37	1.09–1.72	.007	1.009	1.003–1.016	.003	0.985	0.973–0.996	.01
+ cholesterol	1.38	1.10–1.73	.006	1.010	1.003–1.016	.003	0.984	0.973–0.996	.01
+ blood pressure	1.35	1.07–1.69	.010	1.009	1.003–1.016	.003	0.986	0.974–0.998	.02
+ education	1.30	1.05–1.64	.020	1.008	1.002–1.016	.008	0.987	0.975–0.999	.03
+ smoking	1.15	0.94–1.50	.150	1.007	1.001–1.013	.030	0.991	0.979–1.003	.14
CHD mortality									
Age and gender	1.88	1.15–3.07	.010	1.008	0.996–1.021	.200	0.975	0.950–1.001	.06
+ cholesterol	1.86	1.14–3.04	.010	1.008	0.995–1.021	.220	0.976	0.950–1.002	.07
+ blood pressure	1.78	1.08–2.91	.020	1.008	0.995–1.022	.200	0.975	0.950–1.001	.06
+ education	1.70	1.03–2.81	.040	1.007	0.994–1.020	.300	0.977	0.952–1.003	.09
+ smoking	1.46	0.88–2.43	.150	1.004	0.991–1.018	.550	0.983	0.958–1.009	.20

*Prevalence 34.8%.
†Mean 8.6 years (standard deviation 14.5).
‡Mean 12.7 (standard deviation 11.6).
CI = confidence interval.

To evaluate if the dental components were independent risk factors or if the other risk factors could explain this effect, risk factors were added one by one to the calculation (Table 3). Upon sequential addition of the risk factors, the significance of the association with CHD was lost. For edentulousness, smoking was the most important factor explaining the association with CHD mortality.

For CHD death, edentulousness was significant as a risk factor until smoking was added; then, all association was lost, suggesting that smoking explains the association of edentulousness with CHD death in these samples. For total mortality, all oral factors examined showed significant association with the endpoints. However, when the CHD risk factors were added stepwise to the analysis, the association with total mortality remained significant for both edentulousness and number of years of edentulism until smoking was added.

Discussion

This study further examined some conventional risk factors for CHD and three oral factors—number of remaining teeth, edentulousness, and number of years of edentulism—in relation to total and CHD mortality. This was done with two combined samples from the prospective Reykjavík study and a follow-up after a single examination in the MONICA survey.[17–19] The main finding was a significant hazard ratio for all three dental components with respect to total mortality, but only for edentulousness with respect to CHD mortality. This relationship, however, was explained by other CHD risk factors, smoking in particular.

In the samples studied, we have previously shown a strong association between conventional risk factors for CHD, such as smoking, and dental pathology.[11] This is in agreement with other studies, where oral factors have increasingly been associated with CHD.[12] The relationship seems to be confined to inflammation such as periodontitis, infection, and loss of teeth.[21–28] It was thus of interest to further explore whether there was an association of these oral factors of pathology with total and CHD mortality.

Because of the small sample size and relatively few endpoints, the two cohorts were combined. The main weakness of this study concerns the few endpoints (n = 353), particularly only 82 CHD deaths in a sample of a total of 2,613 individuals. Although the sample material was derived from two sources, the relationship between the risk factors and total and CHD mortality showed the same trend for most of the risk factors as in the Reykjavík study, a population study of approximately 23,000 individuals.[17,18] This suggests a reasonable statistical power to examine the effect of the oral components studied on the endpoint mentioned above in these samples.

When hazard ratios for the three odontologic variables (number of remaining teeth, complete edentulousness, and number of years of edentulism) were tested against total mortality on one hand and CHD mortality on the other, adjusting for age and gender, all three stood out as significant predictors regarding total mortality, with number of years of edentulism as the strongest predictor. When examined for CHD mortality, the predictive power was confined to edentulousness. The importance of the odontologic factors was reduced as the other risk factors were sequentially added to the analysis. All three odontologic variables finally lost their significance as predictors when smoking was added to the model. This indicates the confounding effect of smoking habits and is in accordance with the findings reported by Hujoel et al.[29] In most studies, it has been extremely difficult to separate smoking from the combined risk of CHD.[10,30–33]

This does not, however, exclude the possibility that dental and general oral health may play a role as part of the factors leading to CHD and/or death of the respective individuals. On several occasions, loss of teeth has been shown to be more common among those who suffer from CHD and die from the disease compared to those who suffer other causes of death.[6,28,34]

It has been shown that poor dental health and edentulousness lead to a change in the composition of the diet, with avoidance of vegetables and other products that are more difficult to chew and consumption of more refined and processed food.[35,36] Whether this, for example, leads to obesity and increased risk of CHD remains to be determined.

It can also be speculated that the foundation for CHD among edentulous people may have been laid before they actually became edentulous. Therefore, dental and periodontal disease cannot be excluded. The beneficial effect of certain food products, such as fish and vegetables, has also been suggested.[37,38] Loss of teeth may be caused by periodontal disease and/or dental caries. In normal function, at the time of removal of the teeth, and/or during periodontal treatment or other common interventions, potentially dangerous microorganisms may gain access to the bloodstream and later cause damage that could lead to a fatal outcome.[1–8,39–41]

It cannot be denied that among those who are edentulous, individuals who have been more careless with their health in general may be more numerous.[21,35–44] Thus, this group may contain more of those with poor hygiene habits and higher consumption of alcohol and tobacco. Controlling for these previous events is not possible, at least not with the present material.

When the CPITN was tested against death and history of CHD, it did not give any statistically significant results to either parameter (data therefore not presented).

The nature of the samples studied has to be kept in mind as well as the relatively few people who have died within the MONICA part of the investigation during the last decade. Therefore, at the moment, no statement can be made regarding a possible connection between

periodontal health and causes of death, be it CHD compared with the accepted risk factors in multivariate analysis or the confounding factor of smoking habits.

Murine experiments in which the animals have been infected by oral administration of *Porphyromonas gingivalis* have shown accelerated early atherosclerosis in the experimental animals. How and when these results can be explained has been a matter of discussion.[45,46] From the present data, however, it is clear that edentulousness per se as well as the length of time for which the individual has been edentulous correlate with increased likelihood of the subject suffering from premature death from any cause, and probably CHD as well. Nevertheless, smoking may account for all the mortality found to associate with oral/dental status of the cohorts examined. This leaves us with the intriguing possibility that oral disease such as periodontitis and its consequences, like loss of teeth, may be one way in which smoking exerts its effect by persistently maintaining an inflammatory/infected state.[47–49]

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Literature Abstract

Influence of implant length and diameter on stress distribution: A finite element analysis

Implant size influences the area of possible retention in bone; factors such as occlusion, masticatory force, number of implants, and implant position within the prosthesis affect the distribution of stress on adjacent bone. The purpose of this study was to determine which length and diameter of implants would best dissipate stress using a mathematical simulation of stress distribution around implants. Implant models with diameters of 3.6 mm and lengths of 8 mm, 10 mm, 12 mm, 14 mm, 16 mm, 17 mm, and 18 mm were used to investigate the influence of length as a factor. The influence of diameter was modeled by implants with a length of 12 mm and diameters of 2.9 mm, 3.6 mm, 4.2 mm, 5.0 mm, 5.5 mm, 6.0 mm, and 6.5 mm. The selected 3-D models represented commonly available submerged titanium solid cylinder-shaped dental implants without threads (IMZ; ITI Bonefit) with bioactive coating. 3-D loading of the implants with forces of 17.1 N, 114.6 N, and 23.4 N in a lingual, axial, and distomesial direction, respectively, simulated average masticatory force in a natural, oblique direction. The von Mises equivalent stress (MPa) at the implant-bone interface was computed using finite element analysis (FEA). The relative stress acting in the bone around the implant with a diameter of 4.2 mm was 31.5% smaller than the reference implant (diameter of 3.6 mm). Further stress reduction with the 5.0-mm implant represented an additional 16.4%. The use of an implant with a diameter of 6.5 mm resulted in reduction of the maximum stress values by almost 60%. The model for implants with the same diameter (3.6 mm) but different lengths showed a substantially lower effect of length than diameter. The authors concluded that an increase in the implant diameter decreased the maximum von Mises equivalent stress around the implant neck more than an increase in the implant length, as a result of a more favorable distribution of the simulated masticatory forces.

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