

## Periodontal health, dental caries, and metabolic control in insulin-dependent diabetic children and adolescents

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### Abstract

*The purpose of this investigation was to examine the relationship between dental health and metabolic control in insulin-dependent diabetic children. Thirty diabetic children and adolescents (ages 4-19 years) were matched by age, sex, and race to 30 healthy children. Metabolic control was determined for each diabetic child by percentage of glycosylated hemoglobin, HbA<sub>1c</sub>; 14 children were judged to be well controlled (HbA<sub>1c</sub> ≤ 10%); 16 were poorly controlled (HbA<sub>1c</sub> > 10%). Plaque index, gingival index, loss of periodontal attachment, and presence of caries were recorded for each subject. Poorly controlled diabetic children displayed an increased plaque index. The gingival index of diabetics in poor control was significantly higher than that of healthy children. Prevalence of caries was the same for all children in the study.*

Despite the ubiquitous nature of the complications of insulin-dependent diabetes, relatively few studies have examined the complications of diabetes manifested in oral tissues of diabetic children. Dental disease in the diabetic child can increase the difficulty of maintaining metabolic control (Gottsegen 1983). Insulin-dependent diabetics with periodontal disease have been reported to have more hospital admissions and require more insulin than diabetics without periodontal disease (Williams and Mahan 1960).

Diabetes appears to act as a modifying and probably accelerating factor in periodontitis (Gottsegen 1983). Diabetic children are reported to have an increased prevalence of periodontal disease.<sup>1</sup> However, only one group of investigators has compared the prevalence of periodontal disease in poorly controlled and well controlled diabetic children (Gislen et al. 1980). The degree of diabetic control may influence the biological mechanisms which cause increased periodontal disease in diabetics.

There is general agreement that diabetic subjects have the same caries prevalence as healthy controls

(Gottsegen 1983). The diabetic child's decreased daily intake of refined carbohydrate and the universal availability of fluoride have a protective effect against caries-promoting factors like increased salivary glucose and decreased flow of saliva.

The purpose of the present study was to examine the relationship between dental health and metabolic control in diabetic children.

### Materials and Methods

#### Subject Population

The insulin-dependent diabetic participants in the study were patients of the Pediatric Diabetic Outpatient Clinic of Strong Memorial Hospital (SMH), Rochester, New York. Prior to commencement of the study, approval for it was granted by the Committee for Investigations Involving Human Subjects of SMH.

The level of glycosylated hemoglobin (HbA<sub>1c</sub>), which was the main criterion used to judge metabolic control, was determined for each diabetic patient. The HbA<sub>1c</sub> level in a nondiabetic ranges from 5.0 to 7.5% (Sperling 1983). A diabetic with an HbA<sub>1c</sub> level of less than 10% was considered to be well controlled; a level of more than 10% indicated poor to fair control (Sperling 1983). A combination of daily insulin injections and blood glucose monitoring, dietary restrictions, exercise, and stress management, were the means used to achieve diabetic control for each child.

A child had to fulfill the following criteria for inclusion in the study: (a) confirmed diagnosis of diabetes; (b) absence of systemic illnesses other than diabetes; (c) not taking any medications other than insulin; (d) not wearing fixed orthodontic appliances.

The nondiabetic control subjects in the study were regular attenders at the Pediatric Dentistry Department of the Eastman Dental Center (EDC). The study was approved by the Institutional Review Board of EDC.

For control subjects a medical history with no suggestion of diabetes and a negative family history were accepted as adequate evidence that diabetes was not present.

<sup>1</sup> Bernick et al. 1975; Cianciola et al. 1982; Falconbridge et al. 1981; Ringelberg et al. 1975.

## Oral Examination

Periodontal health was recorded at six sites for each fully erupted, nonmobile tooth: distobuccal, midbuccal, mesiobuccal, mesiolingual, midlingual, and distolingual cervical areas. The gingival index for each site was the sum of the gingival inflammation index and the gingival bleeding index. If the scoring of any site was uncertain, a score of "zero" was assigned (i.e., healthy). Gingival inflammation was scored using the criteria of Meitner et al. (1979). The gingival margin of each site was examined and classified as either not inflamed (pale pink color = 0) or inflamed (change in color from normal pale pink = 1).

Before the gingival bleeding index was determined, the tooth surfaces at each site were scraped for soft plaque. The amount of plaque was recorded after an explorer was scraped twice over the tooth surface (Kjellman et al. 1970). If plaque was present on the explorer, the surface was given a score of 1. If no plaque was present, the surface was scored "zero." The plaque index was recorded as the percentage of plaque-bearing surfaces. This method of determining plaque index (Kjellman et al. 1970) did not require the use of a disclosing agent and was, therefore, well accepted by all subjects.

The gingival bleeding index was determined by inserting a periodontal probe into the gingival sulcus of the six representative areas until slight resistance was felt (Nowicki et al. 1981). The probe was rubbed against the inner aspect of the gingiva for one back-and-forth motion over a distance of approximately 2 mm and the site was examined in 10-15 sec for bleeding. A site with no bleeding was given a score of "zero;" if bleeding was present, the site was given a score of 1. The gingival bleeding index was recorded as the percentage of bleeding surfaces. A standard technique (Ramfjord 1967) was used to measure loss of attachment.

Decayed, filled, and missing teeth were recorded. The standard criteria of Radike (1968) were used to diagnose caries. Instruments used for the examination were a front surface mirror<sup>a</sup> and a double-ended "piano wire" explorer.<sup>c</sup>

The only examiner was the principal investigator who was standardized to the scoring system by calibration with a dentist experienced in using the scoring system. After being standardized, the principal investigator's reliability was verified.

Least squares means adjusted for differences in age and sex among the groups were computed for the clinical indices for each group of subjects (i.e., poorly controlled diabetics, well controlled diabetics and healthy subjects). Analysis of covariance was per-

<sup>a</sup> Williams periodontal probe - Henry Schein Co; Port Washington, NY.

<sup>b</sup> Size 3 - Henry Schein Co; Port Washington, NY.

<sup>c</sup> Starlite 5 - Star Dental Manufacturing; Valley Forge, PA.

formed to determine the relationship between subject group and the clinical indices. Newman-Keuls nonsignificant groupings were determined if subject groups were found to be significantly ( $P \leq 0.05$ ) associated with a clinical index.

## Results

Thirty diabetic children and adolescents, matched as closely as possible by age, sex, and race to 30 healthy control subjects, were included in the study (Table 1).

TABLE 1. Age Distribution of Subjects

	≤ 12 Years	12-15 Years	≥ 15 Years	Total Number of Subjects
Females				
Diabetic	8	8	2	18
Nondiabetic	9	6	2	17
Males				
Diabetic	2	6	4	12
Nondiabetic	5	6	2	13

Fourteen diabetic subjects (5 males, 9 females) had good metabolic control with an HbA<sub>1c</sub> of  $8.7 \pm 0.2\%$  (mean  $\pm$  standard error). Sixteen diabetic subjects (7 males, 9 females) with an HbA<sub>1c</sub> of  $11.9 \pm 0.4\%$  (mean  $\pm$  standard error) were designated as poorly controlled.

Plaque scores were significantly higher in poorly controlled diabetics in comparison to the other two groups (Table 2, top of page 285). The poorly controlled diabetic group also had a significantly higher mean gingival index than the healthy subjects (Table 2). The gingival index of the well-controlled diabetics was not significantly different from either group. Bleeding and inflammation indices were not significantly different among the groups; healthy subjects had the least amount of bleeding and inflammation; and the poorly controlled subjects demonstrated the highest gingival inflammation index (Table 2). The poorly controlled diabetics also demonstrated the strongest correlation between plaque and gingival index (Table 3, 285). The correlation between plaque index and gingival index was highly significant ( $P \leq 0.01$ ) only for poorly controlled diabetics.

Two subjects, both of whom were diabetic, had measurable loss of gingival attachment. One subject, a 17-year-old black male diabetic in poor control, had 4 sites of attachment loss with a mean loss of attachment of 1.5 mm per site. The other subject, a 17-year-old white male diabetic in good metabolic control, had 2 sites of attachment loss with a mean loss of attachment of 1 mm per site.

No differences in mean numbers of decayed, missing, or restored surfaces were apparent among the three groups of subjects (Table 2). Means were corrected for differences in fluoridation of community water supplies.

## Discussion

Differences in amount of plaque between subject groups were determined because plaque is the prime agent in the etiology of periodontal disease. The authors' observation that the poorly controlled diabetic group had significantly more plaque than well-controlled diabetics and healthy subjects was consistent with a previous investigation (Falconbridge et al. 1981), but contrasted with other studies that found no difference in amount of plaque between diabetic and healthy children (Bernick et al. 1975; Cianciola et al. 1982). None of these previous investigators considered degree of metabolic control of diabetes when plaque indices were compared. The present study supports the results of a study involving animals which found that alloxan-induced diabetic rats had more plaque than healthy controls (McNamara et al. 1977).

Poor oral hygiene contributes to accumulation of plaque, but increased plaque in the poorly controlled diabetic subjects was probably a result of other factors besides inadequate oral hygiene. Decreased salivary flow in poorly controlled diabetics may have resulted in less effective cleansing and a decreased supply of antibacterial substances (Conner et al. 1970; Harrison, 1985). In addition, elevated salivary glucose concentration (Tenovuo et al. 1985; Harrison, 1985) ensured that an increased supply of fermentable carbohydrate was continually available to plaque bacteria. The combination of decreased salivary flow, increased salivary glucose, and poor oral hygiene contributed to an increased accumulation of plaque in poorly controlled diabetics.

TABLE 3. Spearman Correlations between Clinical Indices—Plaque Index, Gingival Bleeding, Gingival Inflammation, and Gingival Index—for Each Subject Group

Indices	Bleeding	Inflammation	Gingival Index
Diabetics in poor control (N = 16)			
Plaque	0.57*	0.86**	0.78**
Bleeding		0.67**	0.88**
Inflammation			0.88**
Diabetics in good control (N = 14)			
Plaque	0.35	0.59*	0.52
Bleeding		0.87**	0.92**
Inflammation			0.97**
Non-diabetic subjects (N = 30)			
Plaque	0.41*	0.37	0.36
Bleeding		0.78**	0.85**
Inflammation			0.98**

\*  $P \leq 0.05$ .

\*\*  $P \leq 0.01$ .

TABLE 2. Least Squares Means\* ( $\pm$  SD) for Clinical Indices of All Subjects

Clinical Index	Diabetics		Healthy Subjects (N = 30)	Newman-Keuls Nonsignificant Groupings†
	Poor-fair Control (N = 16)	Good Control (N = 14)		
Plaque index	0.89 $\pm$ 0.07	0.75 $\pm$ 0.08	0.70 $\pm$ 0.07	[0][1, 2]
Bleeding index	0.29 0.06	0.30 0.07	0.19 0.06	[1, 0, 2]
Inflammation index	0.83 0.08	0.69 0.09	0.64 0.08	[0, 1, 2]
Gingival index	0.56 0.06	0.51 0.07	0.41 0.06	[0, 1][1, 2]
DMFS	5.6 $\pm$ 1.3	4.8 $\pm$ 1.3	5.0 $\pm$ 1.1	[0, 2, 1]

\* Adjusted for any imbalances in age, sex, and community water fluoridation.

† Newman-Keuls groupings ranked beginning with group with highest mean index; groups in separate brackets significantly different ( $P \leq 0.05$ ); groups placed together in brackets not significantly different. Group 0 = poorly controlled diabetics; group 1 = well-controlled diabetics; group 2 = healthy subjects.

The increased gingival index observed in the poorly controlled diabetic group in the present study confirmed and extended the results of previous investigators<sup>2</sup> who reported more severe gingivitis in diabetic than in healthy children. The observation by Gislén et al. (1980) that poorly controlled diabetics had more severe gingivitis than well-controlled diabetics or healthy subjects also was confirmed in the present study.

Two general theories may explain increased periodontal disease in response to plaque in the poorly controlled diabetic group: (a) the microbial composition of the plaque of poorly controlled diabetics may be more pathogenic than that of the other two groups; (b) the resistance of the periodontal tissues to virulence factors of plaque bacteria may be diminished.

Several components of host resistance to bacteria, including disturbed functioning of neutrophils (Bissada et al. 1982) and altered collagen metabolism (Kaplan et al. 1982) have been reported to be compromised in diabetics. The burden of an increased and possibly more pathogenic plaque in a compromised host may help explain increased gingivitis in poorly controlled diabetic children.

Because prevalence of caries is significantly correlated with fluoride in the water supply and because about 25% of the poorly controlled diabetics did not have access to a fluoridated water supply, the mean DMFS scores were adjusted to account for differences in fluoride in the water supply. No difference in DMFS scores between subject groups was found, which confirmed the consensus from previous investigations that caries prevalence is no greater in diabetic than in healthy children.<sup>3</sup> Furthermore, the similar DMFS scores verified the results from an earlier investigation that found the degree of metabolic control was not associated with presence of caries (Sterky et al. 1971). The protective effects of fluoride and decreased dietary refined carbo-

<sup>2</sup> Bernick et al. 1975; Cianciola et al. 1982; Falconbridge et al. 1981; Gislén et al. 1980; Ringelberg et al. 1975.

<sup>3</sup> Bernick et al. 1975; Falconbridge et al. 1981; Kjellman et al. 1970; Ringelberg et al. 1975; Sterky et al. 1971.

hydrates appeared to prevail against caries-promoting factors such as decreased flow of saliva and increased salivary glucose (Harrison 1985), resulting in no difference in prevalence of caries.

## Conclusions

The conclusions of this study are summarized as follows:

1. Poorly controlled diabetic children displayed an increased plaque index.
2. Diabetic children in poor control demonstrated more severe gingivitis than healthy children.
3. Prevalence of caries was similar for diabetic children and healthy control subjects.

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