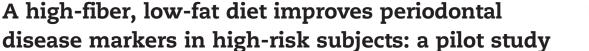


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#### ABSTRACT

Periodontal disease is related to aging, smoking habits, diabetes mellitus, and systemic inflammation. However, there remains limited evidence about causality from intervention studies. An effective diet for prevention of periodontal disease has not been well established. The current study was an intervention study examining the effects of a highfiber, low-fat diet on periodontal disease markers in high-risk subjects. Forty-seven volunteers were interviewed for recruitment into the study. Twenty-one volunteers with a body mass index of at least 25.0 kg/m<sup>2</sup> or with impaired glucose tolerance were enrolled in the study. After a 2- to 3-week run-in period, subjects were provided with a test meal consisting of high fiber and low fat (30 kcal/kg of ideal body weight) 3 times a day for 8 weeks and followed by a regular diet for 24 weeks. Four hundred twenty-five teeth from 17 subjects were analyzed. Periodontal disease markers assessed as probing depth (2.28 vs 2.21 vs 2.13 mm; P < .0001), clinical attachment loss (6.11 vs 6.06 vs 5.98 mm; P < .0001), and bleeding on probing (16.2 vs 13.2 vs 14.6 %; P = .005) showed significant reductions after the test-meal period, and these improvements persisted until the follow-up period. Body weight (P < .0001), HbA1c (P < .0001), and high-sensitivity C-reactive protein (P = .038) levels showed improvement after the test-meal period; they returned to baseline levels after the follow-up period. In conclusion, treatment with a

Abbreviations: AUC, area under the curve; BMI, body mass index; BOP, bleeding on probing; CAL, clinical attachment loss; GCF, gingival crevicular fluid; hs-CRP, high-sensitivity C-reactive protein; OGTT, oral glucose tolerance test; PD, probing depth.

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high-fiber, low-fat diet for 8 weeks effectively improved periodontal disease markers as well as metabolic profiles, at least in part, by effects other than the reduction of total energy intake.

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#### 1. Introduction

Periodontal disease is a chronic disorder generally characterized by the inflammation and breakdown of tooth-supporting tissues. The breakdown of tooth-supporting tissues by the progression of periodontal disease is a major factor of tooth loss in middle-aged and elderly people [1].

The main cause of periodontal disease is plaque, but other factors such as age, smoking, and alcohol intake affect the health of the gums [1]. In addition to these conventional risk factors, obesity [2–4], diabetes mellitus [5,6], systemic inflammation [7], and dysregulation of adipocytokines [8–10] are also recognized as risk factors for periodontal disease. Recently, periodontal disease has been recognized as a risk factor for diabetes and cardiovascular disease, through chronic inflammation [5,6]. Although a relationship between periodontal disease and diabetes has been recognized, the causal link between the 2 remains unclear because most studies on the topic are observational studies. Few intervention studies have been conducted (such as smoking cessation) that have reported a successful reduction in the severity of periodontal disease and the onset of periodontal disease [11].

There are few reports that have been published that have tested the effects of nutritional intervention on periodontal disease in obese or diabetic subjects. One randomized controlled trial has reported that intervention with a customized diet (increased consumption of fruits, vegetables, and whole grains) did not improve periodontal disease markers in patients with adult periodontitis [12]. Jenzsch et al [13] indicated that wholesome nutritional intervention for 12 months reduced clinical probing depth (PD) and gingival inflammation in patients with metabolic syndrome who had chronic periodontitis. However, the mechanisms underlying the improved periodontitis observed with nutritional intervention were not evaluated.

A high-fiber diet has been reported to improve glucose excursion after a meal [14], systemic inflammation [15,16], and dysregulation of adipocytokines [17,18]. In addition, intervention with a low-fat diet decreased body weights in overweight and obese individuals [19].

We hypothesized that dietary intervention improves periodontal disease markers in high-risk subjects through reduced systemic inflammation by body weight reduction. In this study, we examined the effects of a high-fiber, low-fat diet on periodontal disease markers in mildly obese and/or prediabetic subjects.

## 2. Methods and materials

#### 2.1. Subjects

Subjects were recruited with a local advertisement at a company in Takatsuki in Japan. The inclusion criteria for the

study were people aged 35 to 60 years with a body mass index (BMI) of at least  $25.0\,\mathrm{kg/m^2}$  or a plasma glucose level at 2 hours after a 75-g oral glucose tolerance test (OGTT) of at least 120 mg/dL. Exclusion criteria were medication for dyslipidemia, hypertension, diabetes, and constipation; the use of corticosteroids; and subjects with abnormal thyroid function. The nature and potential risks of the study were explained to all subjects, and written informed consent was obtained. The Ethics Committee of Shiga University of Medical Science approved the study protocol (date of Ethics Commission approval: July 26, 2005; Protocol No. 17-39). The study duration was July 2005 to June 2006.

## 2.2. Study design

The test-meal period was preceded by a 2- to 3-week run-in period. At the beginning of the run-in period, subjects were advised to have a standard diet consisting of 28 to 30 kcal/kg of ideal body weight (protein 15%, fat 25%, carbohydrate 60%) by a dietician. During the run-in period, every subject kept a 7day food record with a digital camera to determine individual energy intake [20]. At the end of the run-in period, OGTT was performed. Before glucose load was assessed, a blood sample was taken to measure insulin (chemiluminescent enzyme immunoassay), glucose (hexokinase G6PD UV method), HbA1c (latex agglutination turbidimetry method), lipids, high-sensitivity C-reactive protein (hs-CRP; latex photometric immunoassay), tissue plasminogen activator inhibitor-1 (latex photometric immunoassay), fibrinogen (method for measuring coagulation time), leptin (double-antibody radioimmunoassay), and adiponectin (latex photometric immunoassay) [21,22]. Blood pressure, body weight, and waist circumference were also measured. After the run-in period, subjects consumed the provided ready-made retort pouch test meal consisting of high-fiber and low-fat food, which provided a total energy of 30 kcal/kg of ideal body weight, 3 times a day for 8 weeks. After the test-meal period, subjects were allowed to return to their regular dietary habits without limitation. During the follow-up period, every subject kept a 7-day food record by digital camera to determine individual energy intake [20]. At the end of the test meal and the followup periods, the same examinations as the run-in period were administered. These examinations were conducted at the Shiga University of Medical Science Hospital. The subjects were given advice on maintaining their usual lifestyle habits, such as physical activity, throughout the study.

### 2.3. Periodontal examination

At the end of each study period, periodontal examination was performed by a single dentist. Clinical measurements were performed at 6 sites per tooth at the mesiobuccal, buccal, distobuccal, mesiolingual, lingual, and distolingual tooth surfaces of all teeth, except the third molars, as previously described [23]. Briefly, PD was measured from the gingival margin to the base of the clinical pocket with the probe tip

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