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Dietary ratio of n-6 to n-3 polyunsaturated fatty acids and periodontal disease in community-based older Japanese: A 3-year follow-up study

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ABSTRACT

The longitudinal relationship between dietary n-6 to n-3 PUFAs ratio and periodontal disease in 235 Japanese subjects for whom data were available for the years 2003–2006 was investigated. PUFAs intake was assessed at baseline with a brief-type self-administered diet history questionnaire. Full-mouth periodontal status, measured as the clinical attachment level (CAL), was recorded at baseline and once a year for 3 years. The number of teeth with a change in the loss of CAL ≥ 3 mm at any site over a year was calculated as 'periodontal disease events'. Poisson regression analysis was conducted, with dietary n-6 to n-3 PUFAs ratio as the main predictor, to estimate its influence on periodontal disease events.A high dietary n-6 to n-3 PUFAs ratio was significantly associated with greater number of periodontal disease events. The findings suggest the dietary n-6 to n-3 PUFAs ratio is associated with periodontal disease among older Japanese.

1. Introduction

Periodontal disease is defined as an inflammatory condition of the gingival tissues, characterized by loss of attachment of the periodontal ligament and the bony support of the tooth. Periodontal disease is one of the main risk factors for tooth loss in older people [1,2]. Tooth loss has been associated with a sub-optimum intake of some nutrients and changes in food preference [3,4].

Diet plays an important role in terms of optimizing health and disease prevention. Polyunsaturated fatty acids (PUFAs) are of major importance in normal physiological functions linked to membrane integrity and regulatory cell signals [5]. Two groups of PUFAs, n-3 series and n-6 series, have attracted a great deal of attention with regard to health and diseases [6]. Linoleic acid (LA; 18:2n-6) represents the basis of the n-6 family and α-linolenic acid (ALA; 18:3n-3) represents the basis of the n-3 family. Both LA and ALA are metabolized to longer-chain fatty acids of 20 and 22 carbon atoms. LA is metabolized to arachidonic acid (AA; 20:4n-6), and ALA to eicosapentaenoic acid (EPA; 20:5n-3) and docosahexaenoic acid (DHA; 22:6n-3), increasing the chain length and degree of unsaturation by adding extra double bonds to the carboxyl end of the fatty acid molecule. These two types of PUFA are not interconvertible, are metabolically and functionally distinct, and often have important opposing physiological functions [7,8].

Previous studies have shown that diets rich in n-6 PUFAs create an inflammatory environment that may increase the risk of chronic diseases associated with an inflammatory state [7,8]. On the other hand, higher intakes of n-3 PUFAs appear to reduce the risk of many diseases, including coronary heart disease [9], type 2 diabetes [10], rheumatoid arthritis [11], asthma [12], depression [13], and cancer [14]. Both PUFAs compete for the activity of a fatty acid desaturase and the overabundance of dietary LA gives a quantitative advantage that limits the conversion of ALA to EPA *in vivo* [15]; therefore, it has been suggested that a balanced ratio of these two types of PUFAs may be necessary for the prevention and treatment of chronic diseases [7,8,]. A high n-6 to n-3 PUFA ratio, i.e. n-6 PUFAs consumed in much larger quantities than n-3 PUFAs, is characteristic of modern day diets [16]. In observational and experimental studies, a high ratio of n-6 to n-3 PUFAs has an adverse influence on health [17,18]. It is still debated which explains the PUFA effect on health and diseases more

Abbreviations: PUFA, polyunsaturated fatty acid; LA, linoleic acid; ALA, α-linolenic acid; AA, arachidonic acid; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; BDHQ, brief-type self-administered diet history questionnaire; CAL, clinical attachment level; BMI, body mass index; HbA1c, hemoglobin A1c; HDL-C, high-density lipoprotein cholesterol; RR, relative risk; CI, confidence intervals; PG, prostaglandin; LT, leukotriene; IL, interleukin; TNF-α, tumor necrosis factor-α

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significantly, the n-6 to n-3 PUFA ratio or the absolute amount of each PUFA [19].

Recently, we demonstrated an inverse independent relationship of dietary DHA intake with periodontal disease progression in a small sample study ($n=36$) [20]; however, information on n-6 PUFA intake was not available in the previous study; therefore, it was not possible to assess both the effects of the dietary n-6 to n-3 PUFA ratio and each intake of n-3 PUFAs and n-6 PUFAs on periodontal disease in one study. So far, there have been no reports in the literature on the relationships of the dietary ratio of n-6 to n-3 PUFAs to changes in the periodontal condition over time.

Investigating the relationship between the dietary n-6 to n-3 PUFA ratio and periodontal disease is important to address the value of the dietary n-6 to n-3 PUFA ratio and to understand the potential role of dietary modification in the prevention and treatment of periodontal disease and the ultimate prevention of tooth loss through periodontal disease. Consequently, the hypothesis of the present study is that a high dietary ratio of n-6 to n-3 PUFAs may contribute to periodontal disease progression. This study aimed to determine if there is a relationship between the dietary n-6 to n-3 PUFA ratio and periodontal disease progression in community-dwelling older Japanese.

2. Materials and methods

2.1. Study population (Fig. 1)

The current investigation was a subset study of the Niigata study over the study period of 2003–2006. The original participants in the Niigata study included 600 randomly selected 70-year-old Japanese individuals who were residents of Niigata city, Japan in 1998. All participants were fully informed of the purpose of the survey and consented to participate. Details of the sampling methodology and selection have been previously published [21].

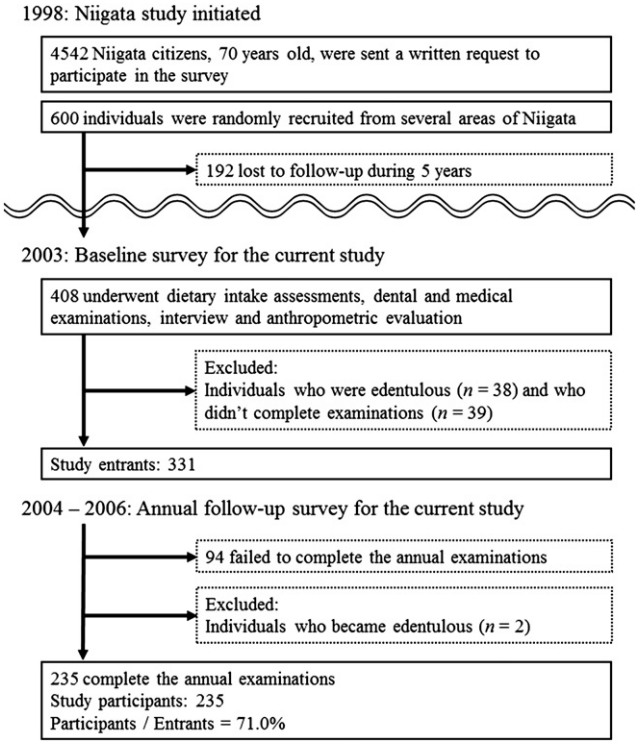

Fig. 1. Flow diagram of the study.

Five years after the Niigata study was initiated, 408 subjects who were 75 years of age in 2003 underwent dietary intake assessments, dental and medical examinations, interview and anthropometric evaluation as part of the baseline assessment for the current study. At baseline, people who were already edentulous ($n=38$) or who did not submit complete data ($n=39$) were excluded, leaving 331 eligible subjects to enter the study. Study entrants underwent annual follow-up examinations including dental assessment. During the study period (2003–2006), 94 failed to complete the annual examinations and 2 became edentulous. Data were therefore analyzed for 235 participants (121 men, 114 women) examined as dentate in 2006. The examination protocol used for this study was reviewed and approved by the Ethics Committee of the Faculty of Dentistry, Niigata University.

2.2. Dietary intake assessment

Dietary habits during the preceding month were assessed with a brief-type self-administered diet history questionnaire (BDHQ) [22]. This validated retrospective method of dietary assessment is based on a the food frequency quesionnaire method [23]. Responses to the BDHQ were checked for completeness and, where necessary, clarified by direct questioning of the subject.

The BDHQ is a questionnaire that inquires about the frequency of consumption of a total of 56 food and beverage items, with specified serving sizes described in terms of the natural portion or the standard weight and volume measurement of servings commonly consumed in general Japanese populations. The BDHQ was developed based on a comprehensive version of a self-administered diet history questionnaire [24]. Estimates of mean daily intake for energy (kcal), total PUFAs, total n-3 PUFAs, ALA, EPA, DHA, total n-6 PUFAs, LA, AA were calculated using an ad hoc computer algorithm for the BDHQ, which was based on the Standard Tables of Food Composition in Japan [25]. Values of dietary PUFA intake were energy-adjusted (i.e., amount per 1000 kcal for PUFA). Information on dietary supplement use was not available to the investigators and therefore PUFA intake from dietary supplements was not incorporated into the analysis.

2.3. Dental examination

Dental examinations were carried out at baseline (2003) and once a year for 3 years. The same methods as the baseline survey were used in follow-up dental examinations (2004–2006). Numbers of teeth present were counted and periodontal conditions were assessed for participants with at least one remaining tooth. The periodontal condition, measured as the clinical attachment level (CAL), was recorded. Teeth were probed at six sites per tooth for all teeth present, and measurements were recorded approximately to the nearest millimeter. A change in the loss of attachment of 3 mm or greater in 1 year at any site was considered as a periodontal disease progression [26]. Teeth with periodontal disease progression were excluded from additional-year assessments. Finally, the cumulative numbers of teeth with periodontal disease progression over 3 years per person were calculated as 'periodontal disease events' [20]. Clinical periodontal parameters were recorded by calibrated examiners as previously reported [21].

2.4. Interview, anthropometric evaluation and blood pressure determination

An interview was conducted to obtain information regarding smoking habits. Participants were classified as non-, previous-, or current-smoker according to their smoking histories. Information about oral hygiene habits, namely, the status of visits to a dentist