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Atherogenic cholesterol-rich diet and periodontal disease

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ABSTRACT

Objective: This study investigated the effect of an atherogenic cholesterol-rich diet (AT) on the alveolar bone loss in rats with ligature-induced experimental periodontitis (EP).
Methods: Female Wistar adult rats were assigned either a control (Co) or an AT diet fed for 9 weeks. The AT diet was high in saturated fat, cholesterol and energy. At week 2, animals were subjected to a unilateral ligature (L) around the left first molar (Co + L and AT + L). The contra lateral first right molar (not ligated) of both groups (Co and AT) were used as untreated controls. At week 9, blood was drawn, rats were euthanized, hemi-mandibles removed and stained digital photographs (buccal and lingual surfaces) and radiographs were obtained for quantification of alveolar bone loss (ABL). The ABL was determined by distance and area methods (mm^2) and X-rays were used for periodontal bone support (PBS), (%).
Results: Rats in the AT group exhibited a 17% increase in energy intake, gained significant body weight and showed the highest serum total-cholesterol (T-C) and non-high density lipoprotein-cholesterol (HDL-C) levels ($p < 0.001$). The amount of lost periodontal bone was the greatest in AT + L rats. AT feedings significantly increased the buccal area and distance of bone loss when compared with the unligated-teeth ($p < 0.001$). The rats in the AT + L group also achieved the lowest percentage of PBS ($p < 0.001$). The AT and Co + L rats showed similar PBS. This method more clearly elucidated the effect of the cholesterol-rich AT, with and without the influence of molar ligature, compared to the morphometric analysis.
Conclusion: The alveolar bone loss of EP was magnified by ingestion of an atherogenic diet high in saturated fatty acids and cholesterol.

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1. Introduction

Periodontal disease (PD) or periodontitis extensively affects human populations and is one of the main risk factor for tooth loss.[1,2] There is a high prevalence of PD among American adults[3] and the CDC has estimated that up to half of the population may have PD.[4] Similarly a high prevalence of PD was also found in other populations.[5] PD is characterized by destruction of supporting structures of the teeth, including the periodontal ligament, alveolar bone and gingival tissues leading to a breakdown of the cementum due to the inflammatory condition of the soft tissues surrounding the teeth.[6] PD is closely associated with food intake, particularly with sub-optimal intake of nutrients with well-established roles in formation and maintenance of structural components of oral tissue, i.e. fluoride,[7] vitamin C,[8] calcium[9] vitamin A[10] and carbohydrates.[11]

The development of PD may also be interrelated with dietary lipids, cholesterol and saturated fat intake.[12,13] The incidence of PD has also been associated with enhanced concentration of proatherogenic plasma-lipids, with significant differences in total-cholesterol (T-C), triacylglycerols, and very low density lipoprotein-cholesterol (VLDL-C),[14–16] as well as with atherosclerotic cardiovascular disease.[17] PD is accompanied by chronic bacterial infections which have been considered as a possible culprit of the increased risk of atherosclerosis and coronary heart disease.[18] The chronic bacterial infections in PD increase the levels of serum and gingival crevicular fluid proinflammatory cytokines.[19] Bacterial translocation from periodontal pockets yields the systemic release of inflammatory cytokines, leading to an atherogenic profile[19] associated with hyperlipidemia.[20,21] Experimental studies in rats have also demonstrated that pulpal and periodontal diseases increased serum lipid levels[21] and PD treatment demonstrated beneficial effects on the metabolic control of hypercholesterolemia in patients[21] with improvement of systemic inflammatory markers.[17]

In previous studies we demonstrated that rats fed atherogenic diets (AT) had an inadequate tibiae sub-endochondral ossification and mineralization,[22] and dietary enrichment with mono-unsaturated fatty acid (MUFA) oils could not prevent the negative effect on bone mass and could not mitigate impact on serum atherogenic lipoproteins.[23] Therefore, this study was designed to elucidate the effects of AT feedings on the onset and the progression of experimentally induced PD by molar ligature in rats.

2. Materials and methods

2.1. Animals

Twenty female adult Wistar rats (initial body weight: 152.1 ± 9.2 g; aged: 44 ± 2 days, were obtained from the animal laboratory of the Department of Biochemistry, Faculty of Dentistry, University of Buenos Aires, Argentina. Animals were housed in galvanized cages with meshed floors in order to maintain hygienic conditions and to avoid coprophagy. Rats were kept in individual cages and exposed to a 12-h light/dark cycle throughout the study. Room temperature was maintained at 21 ± 1 °C with a humidity of 50–60%. The rats were maintained in accordance with the USA National Institutes of Health Guide for the Care and Use of Laboratory Animals.[24] The protocols for these experiments were approved by the University of Buenos Aires, Argentina and by the local Committee of the Faculty of Dentistry, University of Buenos Aires.

At baseline, rats were randomized into two groups of 10 animals each. They were fed ad libitum one of the two diets for 9 weeks. The controls (Co) were fed standard chow pellets and AT rats were fed high levels of saturated fatty acids and energy. The details of the diets fed are shown in Table 1. Both diets were processed to produce pellets with similar grain sizes. At week 2, blood was obtained and serum T-C levels were determined in Co and AT groups; this confirmed the presence of hypercholesterolemia in AT rats.

Body weights (W) were measured every 4 days, after a fasting period of 2–4 h. A Mettler PC 4000 scale was used to measure W with an accuracy of ±1 mg. Daily food intake was recorded as kcal per 100 g of body weight per day (kcal/100 g W/day).

Table 1 – Ingredient and nutrient composition of the experimental diet given to rats for 9 weeks.

Ingredient (g/100 g)	Co	AT
Energy		
kcal	293.0	327.4
kJ	1226.7	1370.7
Protein (mix of corn, wheat, soybean, fish and meat flour)	18.0	15.00
Fat (acid hydrolysis; mix of corn oil and fish oil)	7.00	15.00
Cholesterol	0.02	1.42
Total polyunsaturated fatty acids (linoleic acid + omega-3 fatty acids)	3.14	2.45
Total saturated fatty acids	1.96	8.50
Total monounsaturated fatty acids	1.90	4.05
Fibre crude	4.00	4.00
Ash	5.20	4.42
Calcium	1.04	0.884
Phosphorous	0.64	0.544
Potassium	0.96	0.816
Magnesium	0.16	0.136
Sulphur	0.128	0.1088
Sodium	0.16	0.136
Chlorine	0.24	0.204
Iron (ppm)	80	68
Copper (ppm)	7.6	6.46
Zinc (ppm)	40	34
Selenium (ppm)	0.32	0.272
Manganese (ppm)	24	20.4
Iodine (ppm)	0.48	0.408
Water	24.0	24.0
Starch	39.5	33.1

Co, control group; AT, atherogenic cholesterol-rich diet group; Co and AT rats were fed freely.