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Effect of whole foods and dietary patterns on markers of subclinical inflammation in weight stable overweight and obese adults – A Systematic Literature Review

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Reduction of subclinical inflammation is a potential target for chronic disease management. Adiposity is a known modifier of meta-inflammation, however the influence of dietary factors is less clear. This review examines the evidence from human trials evaluating effects of wholefoods or dietary patterns on circulating inflammatory markers in weight stable overweight and obese adults. It is the first review to investigate the effect of diet on inflammation, independent of changes in adiposity. This review was conducted using the Cochrane Collaboration Handbook for Systematic Reviews of Interventions, and data sources included Ovid MEDLINE, EMBASE, CINAHL and Cochrane. Quality of studies was evaluated using the Cochrane Collaboration's Risk of Bias Assessment tool. Twenty-eight studies were included assessing the effect of 17 different foods and dietary patterns over 38 inflammatory markers. Overall, wholefoods and dietary patterns were not found to have significant effects on inflammatory markers in weight stable overweight and obese adults. While wholegrains, soy, dairy, citrus fruits, nuts and chocolate, ginger and high total antioxidant capacity diets showed potential to improve inflammatory profiles, inconsistent findings made it difficult to produce definite conclusions. Study design characteristics contributing to these inconsistencies are discussed and recommendations for future research in this area are presented.

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Periodontal bone loss is not modulated by weight gain in an experimental mouse model of periodontitis

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Obesity is a risk factor for poor oral health. Here we test the hypothesis that weight gain due to high carbohydrate diets affects the inflammatory response in a mouse model of periodontitis.

300 male C57BL/6 mice (8 wk old) were fed ad libitum one of 15 isocaloric diets composed of differing percentages of total net metabolisable energy: protein (5, 10, 15%), carbohydrate (75, 70, 65%), fat was fixed at 20%. Mice were maintained on experimental diets for 18–19 weeks. The maxilla were dissected, stained and dried before taking standardised photographs. Periodontal bone loss was assessed by measurements of 3 positions of the second molar on both sides of the maxilla. Data were analysed, and 'Geometric Framework' surfaces generated using two generalised additive mixed models (GAMMs). In all models, the response variable was alveolar bone loss, and the identity of the animal from which the measurement was fitted as a random effect.

Increasing intake of total carbohydrates was associated with a linear increase in alveolar bone loss in mice ($p < 0.01$). On the other hand, protein intake had no effect on bone loss ($p = 0.29$). The mean concentration of IL-1β in gingival tissues was 1.046 ± 0.565 ng/mg total protein. There was a positive correlation between dietary carbohydrate content and pro-inflammatory cytokine IL-1β, although the effect was not statistically significant ($p = 0.08$). There was no evidence that weight moderates the effect of diet on bone loss (linear mixed model, $p = 0.47$).

This is the first study to explore macronutrient composition and types of carbohydrate on the development of natural occurring periodontitis. In the experimental model used an association between weight gain and the periodontitis associated bone loss was not found. However, increased carbohydrate intake significantly increased bone loss. Further research needs to show if weight gain is a risk factor for inflammatory diseases.

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The designer cytokine IC7Fc improves metabolic homeostasis in *db/db* mice

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Background: gp130 receptor ligands represent a therapeutic strategy for the treatment of type 2 diabetes (T2D) [1,2]. Accordingly, we have generated a novel chimeric cytokine, termed IC7Fc, and previously demonstrated it improves metabolic homeostasis in diet-induced obesity and insulin resistance. Whether IC7Fc improves glycemia in a mouse model of human T2D is unknown. Accordingly, in this study, we administered IC7Fc to the leptin receptor-deficient (*db/db*) mouse, which is an appropriate pre-clinical model to study the efficacy of anti-diabetic drugs.

Basic methodologies: Male diabetic *db/db* mice and their lean controls (*db/+*) received a single dose of IC7Fc (1 mg/kg i.p.) or saline in the fed state at 7, 13 and 17 wk of age as the animals transitioned from β-cell compensation (7 wk) to β-cell failure (17 wk). Blood glucose, plasma insulin and body composition were measured.

Major findings: The acute administration of IC7Fc reduced ($P < 0.001$) blood glucose in 7 wk old *db/db* mice within 90 min compared to saline treated *db/db* mice. The actions of IC7Fc progressively diminished along with the age-dependent progression of the disease such that at 17 wk of age, IC7Fc was ineffective in reducing blood glucose. Remarkably, we observed that a single dose of IC7Fc decreased ($P < 0.05$) total body mass, fat mass and fasting insulin 10 d post injection, effects not observed in age-matched lean (*db/+*) control mice.