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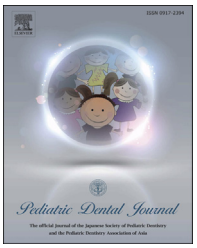

Review

Impact of a high-fat diet on bone health during growth

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ABSTRACT

Background: An inappropriate eating habit is a relatively easily modified risk factor for obesity and osteoporosis in adults. The consumption of high-fat foods is known to induce obesity. Although numerous studies have documented a relationship between high-fat diet (HFD)-induced obesity and osteoporosis, no consensus has been reached. In addition, few data on the relationships between mandibular properties and an HFD in the growth period are available.

Objective: This review aims to summarize current findings related to these issues, focusing on the influence of an HFD on mandibular health, including mechanisms of periodontal disease development.

Main results: Recent data suggest that HFD-induced obesity has a negative impact on the mandible in mice. The loss of trabecular bone and reduction of cortical bone growth in mice with HFD-induced obesity reflect a state of noninvasive and noninfective inflammation.

Authors' conclusions: These results are related to the potential association between metabolic stress and systemic inflammatory changes occurring in bone and other tissues.

Contents

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1. Introduction

The prevalence of overweight and obesity is increasing worldwide [1]. In 2015, 107.7 million children and 603.7 million adults were obese. Since 1980, the prevalence of obesity has doubled in more than 70 countries and has increased continuously in most other countries. Although the prevalence of obesity is lower among children than among adults, the rate of increase in childhood obesity has been greater than the rate of increase in adult obesity in many countries [2]. In recent decades, the eating habits of children and adolescents have undergone many changes due to lifestyle diversification in many countries. [3,4]. In Japan, the National Health and Nutrition Survey showed that mean fat intake increased from 25.8 g/day in 1961 to 57.0 g/day in 2015 [5]. Overweight children are at greater risk of developing type 2 diabetes, hypertension, and hyperlipidemia, which in turn increase the risk of cardiovascular disease later in life.[6]

The development of obesity and osteoporosis in adults can be traced to dietary intake and physical activity during childhood and adolescence. Inappropriate nutritional intake is a relatively easily modified risk factor for obesity and osteoporosis. The relationship between the consumption of high-fat foods and obesity is well established [7], but whether it affects bone architecture in childhood and adolescence remains controversial.

In this review, I summarize the effects of high-fat diet (HFD)-induced obesity on bone health, including the health of alveolar and periodontal bone, and examine the relationship between leptin and bone mass using current findings from human and animal studies. The mechanisms of bone loss due to HFD-induced obesity are also discussed.

2. High-fat diet–induced obesity and bone mass

Although a relationship between obesity and osteoporosis has been proposed in the clinical literature [8–10], no consensus has been reached. Several researchers have observed an increased fracture incidence in obese adolescents and children compared with age-matched controls [8,9]. Additionally, bone fragility may occur in obese children and adolescents because of malnutrition [10].

In animal studies, a positive correlation between obesity and long-bone density was found in 4-week-old male mice fed an HFD for 19 weeks [11], whereas obesity was correlated negatively with long-bone mass in 6-week-old male mice fed an HFD for 14 weeks and 7-week-old male mice fed an HFD for 24 weeks [12,13]. Bartelt et al. showed that HFD-induced obesity did not significantly affect long-bone mass in 4-week-old male mice treated for 16 weeks [14]. They suggested that conflicting results from previous studies regarding the relationship between HFD-induced obesity and bone mass were attributable to differences in the fatty acid profiles of the diets. Several studies have shown associations between the fatty acid compositions of diets and bone health. Weiss et al. reported that a higher ratio of linoleic acid (n-6) to α-linolenic acid (n-3) was associated with detrimental effects on bone health in humans [15], and Watkins et al. showed that a lower ratio of dietary n-6/n-3 was associated with the promotion of bone formation in rats [16]. Bartelt et al. proposed that more precise data could be obtained by considering the fatty acid ratio of dietary components when choosing standard diets and HFDs [14]. However, regardless of the changes in bone mass, many studies have shown significant increases in the size and number of adipocytes in the long-bone marrow of mice with HFD-induced obesity [13,14,17–19]. These findings suggest that HFD-induced obesity causes significant bone loss in mice, due mainly to resorptive changes in the trabecular architecture caused by the increase in, and enlargement of, adipocytes in bone marrow. However, the factors mediating such environmental changes in bone marrow, and the ways in which those mediators and bone-marrow adiposity affect bone metabolism, remain unclear.

In micro–computed tomography analyses, 7-week-old male mice fed an HFD for 4 weeks showed significantly reduced trabecular bone volume, cortical bone thickness, and cortical bone cross-sectional area in the mandible compared with control mice fed a standard diet. In addition, significant decreases in cortical bone density in HFD-fed mice relative to age-matched controls were observed after 12 weeks of HFD treatment. Although cortical bone formation in the mandible was slower in HFD-fed mice than in control mice, bone formation on the periosteal surface increased with age in both groups for 12 weeks [20]. These data support the difference in responses of trabecular and cortical bone to diet-induced obesity; bone loss at these two sites is regulated differentially in mice.

3. Relationships among a high-fat diet, leptin, and bone mineral density

A clinical investigation of the relationship between obesity and osteoporosis suggested that adipose tissue influences bone mineral density through the production of hormones and adipokines, such as leptin [10]. Generally, leptin is known to be an important circulating signal that inhibits food intake and enhances energy expenditure through its actions in the brain [21]. Therefore, several researchers have suggested that an HFD plays a key role in the development of leptin resistance in animals with HFD-induced obesity [22,23]. In confirmation of this proposed role, Choi et al. showed that energy expenditure was lower in mice fed an HFD than in those few a low-fat diet, despite the similarity in intake between the two groups [24].

The relationship between leptin and bone is complex, with diverging effects depending on whether central or peripheral mechanisms are in operation [25,26]. Centrally, leptin has been shown to inhibit bone formation through a hypothalamic relay, and this effect is suppressed by β blockers [27,28]. Peripherally, leptin has a positive effect on bone in rats [29]; an in vitro study also showed that leptin promotes increased production of the potent antiresorptive factor osteoprotegerin by osteoblasts [30].

Studies involving children have documented a direct relationship between the serum leptin concentration and bone mass, but conflicting findings have been reported [31–33]; in one study, the serum leptin concentration was not related to bone mineral density in boys or girls [34].

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