OPEN ACCESS Freely available online

Journal of Osteoporosis and Physical Activity

Short Communication

Molecular Links between Obesity and Periodontitis

Filippova Dmitry*

Department of Medicine, McMaster University, Hamilton, ON, Canada

DESCRIPTION

Obesity and periodontitis are both common health concerns that have brought about economic and societal burdens around the world. They have laid out negative connections between bone metabolism and obesity, obesity and Diabetes Mellitus (DM), and DM and periodontitis. In the oral cavity, bone metabolic disorders fundamentally show an increased risk for periodontitis and alveolar bone loss. Obesity is characterized as someone with a body mass index of >30 kg/m^2 with extreme body fat accumulation prompting excessive health risks [1-3]. According to the WHO, the prevalence of obesity is expanding around the world. The pathogenesis of various diseases, including Type 2 Diabetes Mellitus (T2DM), cardiovascular disease, cancer growth, and osteoporosis, is related to obesity. Osteoporosis appears as weakened bone quality and increased risk of fracture, bringing about a decrease in mobility and quality of life.

The bone loss affected by obesity is expressed as different severities of osteoporosis and could include alveolar bone resorption. As per the 2017 periodontal diseases order, obesity was recognized as a huge metabolic disorder that is related to loss of periodontal tissues, and an increased risk of periodontitis in fat people, recommending a comorbidity impact among heftiness and periodontitis. Specifically, inflammatory environment and imbalanced bone homeostasis affected by obesity were broadly recognized [4]. With the rising trends of obesity around the world, there is presently insufficient evidence for clinicians on the most effective method to manage both health problems in fat people with periodontitis.

Bone is an organ that is permanently in a process of resorption followed by remodeling/reconstruction as its major natural cycle over the course of life. Bone homeostasis includes a balance between bone development and resorption, which are designed by osteoblasts and osteoclasts. In certain cohorts, the level of total fat mass is strongly and inversely connected with the Bone Mineral Density (BMD) and complete bone mineral substance. Obesity impacts bone homeostasis by weakening the balance between osteoblast and osteoclast activities and increasing bone resorption. Purposes behind this impact could be that excess fat in bone marrow, with an enlarged adipocyte number and size, prompts adjusted Bone Marrow Stem Cells (BMSCs) separation with decreased osteoblasts.

Li et al. described increased periodontal inflammatory reaction and bone loss, which were both seen after infusion of bacterial lipopolysaccharides in the jaws of obese aminals nourished from an HFD, compared with a normal control diet [5].

CONCLUSION

Of interest, it was described that alveolar bone density loss could be set off by obesity without the need for periodontitis induction, (i.e., pathogenic microorganism immunization or ligature) in both mature and growing animals to work with the advancement of periodontal disease. In the improvement of diet-prompted obesity, another animal study showed impaired trabecular bone architecture and periosteal bone development in the beginning phase of HFD treatment (4 weeks), followed by a diminishing in cortical bone density in the alveolar bone region with expanded serum leptin levels. In spite of the fact that there is an absence of evidence to determine the causal-effect relationship, the association between obesity and periodontal disease is demonstrated. The connections between weight, bone health, and periodontitis are complex, and alveolar bone loss is probably going to be directed by both systemic and periodontal changes in obesity.

REFERENCES

1. Raisz LG. Pathogenesis of osteoporosis: Concepts, conflicts, and prospects. J Clin Invest. 2005;115: 3318–3325.
2. Drake MT, Clarke BL, Lewiecki EM. The pathophysiology and treatment of osteoporosis. Clin Ther. 2015;37: 1837–1850.
3. Demontiero O, Vidal C, Duque G. Aging and bone loss: New insights for the clinician. Ther Adv Musculoskelet Dis. 2012;4: 61-76.
4. Feskanich D, Willett W, Colditz G. Walking and leisure-time activity and risk of hip fracture in postmenopausal women. JAMA. 2002;288: 2300–2306.
5. Li M, Nichol P, Lee L, Levin RM. Estrogen modulates permeability and prostaglandin levels in the rabbit urinary bladder. Prostaglandins Leukot Essent Fatty Acids. 2009;80: 125-129.

Correspondence to: Filippova Dmitry, Department of Medicine, McMaster University, Hamilton, ON, Canada, E-mail: dmitry.fil@nioch.nsc.ca

Received: 12-Jul-2022, Manuscript No. JOPA-22-18796; **Editor assigned**: 15-Jul-2022, PreQC No. JOPA-22-18796 (PQ); **Reviewed:** 29-Jul-2022, QC No. JOPA-22-18796; **Revised:** 05-Aug-2022, Manuscript No. JOPA-22-18796 (R); **Published:** 16-Aug-2022, DOI: 10.35841/2329-9509.22.10.316

Citation: Dmitry F (2022) Molecular Links between Obesity and Periodontitis. J Osteopor Phys Act. 10: 316.