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A comprehensive Review: Changes to the Upper Airway and Bone Mass as a Result of Weight Loss

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Introduction

Obesity has been identi ed as one of the main causal and perpetuating factors associated with sleep apnea.2 In an epidemiological study conducted in the city of So Paulo, where the prevalence of OSA was 32.8%, 60% of the volunteers were overweight. 3 Obstructive Sleep Apnea (OSA) is characterized by upper airway obstruction during sleep, resulting in periods of apnea, oxyhemoglobin desaturation, and frequent awakenings [1].

e mechanisms underlying OSA and obesity are still poorly understood. Imaging studies have shown that an increase in adipose tissue in the cervicofacial region reduces the volume of the upper airway. Another mechanism would be an increase in visceral fat, which would reduce the volume of the lung and the upper airway, which would support increased pharyngeal collapsibility and reduce caudal tracheal traction.

Literature Review

It is unclear how obesity and losing weight a ect bone health in humans. Obesity, on the other hand, does not result in the same bone changes as weight loss does in rodents and humans. Obesity is usually linked to more bone mass in humans, which is made worse by getting older and going through menopause. In contrast, sex, age, and mechanical load all have an impact on the degree and duration of bone mass loss in obese rodents. In spite of these di erences, rodents are frequently used as models for the human situation [2]. We focus on the applicability of ndings from animal models in this review, which provides a summary of the existing knowledge regarding the e ects of obesity and weight loss on bone mass in humans and rodents. en, we talk about how human skeletal health, obesity, and weight loss can be better understood through the use of animal models. In particular, we draw attention to the aspects of the study design that ought to be taken into consideration in order to maximize the rodent models of obesity and weight loss's translatableness. Particularly, the animals' sex, age, and nutritional status ought to match those of interest to humans [3].

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obesity to maximize translatability. However, even in situations where translatability is low, animal models may still be useful for mechanistic research into things like hormonal interactions and changes in bone phenotype. Additionally, the goal of intervention studies using anti-obesity drugs on obese animals is a return to normal bone mass, regardless of whether it is increased or decreased; consequently, the speci c changes at baseline may not be important for translation.

Conclusion

Weight-bearing and non-weight-bearing bones may be a ected di erently by a reduction in body weight following calorie restriction. According to some studies, the weight-bearing bones in rats are primarily a ected. Following calorie restriction, the femoral and tibial trabecular bone volume and cortical cross-sectional area decreased, while the lumbar vertebrae did not change when the bone properties were measured using histological methods. However, non-weight-bearing bones showed more pronounced changes, according to others. In a similar vein, gastric surgery resulted in a greater volume loss of trabecular bone in the lumbar vertebrae than in the tibia . Weight-bearing bones appear to be particularly a ected by a decrease in body weight in mice, with the femur experiencing a greater reduction in trabecular bone mass than the lumbar vertebrae, regardless of whether the decrease in body weight was caused by food restriction or gastric surgery.

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Conflict of Interest

None

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