

Wolski Dariusz, Bednarski Jerzy, Łuszczewska Sierakowska Iwona, Michalik Joanna, Wawrzyniak Agata, Nowicki Grzegorz Józef, Bodajko Grochowska Anna, Kosiński Jakub. Relationships between body weight and percentage body fat in the body and the development of osteopenia and osteoporosis. *Journal of Education, Health and Sport*. 2018;8(9):727-737 eISSN 2391-8306. DOI <http://dx.doi.org/10.5281/zenodo.1413373> <http://ojs.ukw.edu.pl/index.php/johs/article/view/5925>

The journal has had 7 points in Ministry of Science and Higher Education parametric evaluation. Part b item 1223 (26/01/2017).
1223 Journal of Education, Health and Sport eissn 2391-8306 7

© The Authors 2018;

This article is published with open access at Licensee Open Journal Systems of Kazimierz Wielki University in Bydgoszcz, Poland
Open Access. This article is distributed under the terms of the Creative Commons Attribution Noncommercial License which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author (s) and source are credited. This is an open access article licensed under the terms of the Creative Commons Attribution Non commercial license Share alike. (<http://creativecommons.org/licenses/by-nc-sa/4.0/>) which permits unrestricted, non commercial use, distribution and reproduction in any medium, provided the work is properly cited.

The authors declare that there is no conflict of interests regarding the publication of this paper.

Received: 02.08.2018. Revised: 18.08.2018. Accepted: 11.09.2018.

Relationships between body weight and percentage body fat in the body and the development of osteopenia and osteoporosis

**Dariusz Wolski¹, Jerzy Bednarski², Iwona Łuszczewska–Sierakowska²,
Joanna Michalik³, Agata Wawrzyniak¹, Grzegorz Józef Nowicki³,
Anna Bodajko–Grochowska⁴, Jakub Kosiński⁵**

¹Department of Animal Anatomy and Histology, Faculty of Veterinary Medicine, University of Life Sciences in Lublin

²Department of Human Anatomy, I Faculty of Medicine with the Dental Department, Medical University of Lublin

³Department of Oncology and Environmental Health Care, Faculty of Health Sciences, Medical University of Lublin

⁴Department of Lung Diseases and Pediatric Rheumatology, II Chair of Pediatrics, II Faculty of Medicine with an English Department

⁵Department of Family Medicine and Community Nursing, Medical University of Lublin

Abstract

Obesity nowadays is a significant problem in developing countries and developed ones. Both the percentage of adipose tissue in the body and the proportion of muscle tissue affect the condition of the skeletal system. The common origin of adipose tissue and muscle tissue shows

that overweight and obesity are not indifferent to the metabolism of bone tissue. Both malnutrition and obesity can lead to unfavorable health effects, contributing to the development of bone disorders and the occurrence of osteopenia, osteopenia with sarcoidosis, osteoporosis or osteoarthritis. Increased percentage of adipose tissue and/or muscle tissue during menopause may have an osteoprotective effect and thus prevent or relieve the effects of menopause in women or andropause in men. Research aimed at measuring the content of adipose tissue as a supplement to other diagnostic tests may contribute to the early detection and even prevention of osteoporosis.

Key words: BMI, obesity, osteoporosis, osteopenia

Introduction

Obesity is a non-existent problem, in other words, and comorbidities, as well as health problems. Obesity, according to the World Health Organization (WHO), to increase the accumulation of fat data in the body that has overwhelmed the health event [1]. Excessive accumulation of fat functions in the body may lead to the development of so-called Metabolic Syndrome, which is characterized by a complex metabolism (called Metabolic Syndrome), which works in the following way: diabetes, type 2 diabetes, shotguns, atherosclerosis, type 2 diabetes, shooting, night sleep apnea, urolithiasis bile or a number of bone-joint diseases, including osteoarthritis, osteopenia and osteoporosis [2,3]. Data reflecting the next expiration date of this product. It is estimated that after 700 million years obesity was diagnosed, along with approximately 700 million obesity was diagnosed along with the procedure in this area [1].

During embryonic development, both adipose tissue and bone tissue are derived from the same germ layer - mesenchyma. Due to the common origin, both tissues have many connections, which determines their integrity in physiological conditions and pathological conditions [4,5]. Adipose tissue is not only a source of energy in the body, but what is very important is an important organ of internal secretion. Thanks to the strongly developed network of blood vessels surrounding the adipocytes, characterized by high permeability, rapid and continuous exchange of substances with blood is ensured [6]. As an endocrine organ in the body it is the source of many factors, such as hormones (leptin, adiponectin, resistin, visfatin, estrogens, androgens), cytokines (IL-6, TNF- α) and enzymes and other substances related to bone tissue

metabolism [7, 8].

Diagnosis of obesity

For many years, the view was dominated that determining the body mass index (BMI) is the simplest anthropometric indicator helpful in determining and classifying obesity. This index is obtained by the quotient of the body mass expressed in kilograms and the height expressed in meters raised to the second power. This is not a perfect method, but it is a reference method approved by the WHO. According to the proposed classification, overweight begins when the BMI value falls within the limits of 25.0-29.9, where the adopted norm is a value in the range 18.5-24.9. When the value of the index rises above 30.0, then we speak about obesity, which can be divided into degrees. The first degree of obesity is characterized by a BMI value in the range of 30.0-34.9, II degree in the range of 35.0-39.9, and the highest third degree of obesity is confirmed by the value of the body mass index equal to or greater than 40.0 [1]. Due to the high risk of error, other methods of determining obesity have been developed, and one of the most accepted methods is the method based on the location of the largest amount of body fat. With this method, central obesity, otherwise known as visceral obesity or abdominal obesity, has been distinguished, where the waist circumference is taken into account. However, the waist circumference is racially diverse, which is why three subpopulations (American, European and Asian) have been identified in which the comparison is made. The second type of obesity is the ilio-femoral obesity, where the measure determining the degree of obesity is the hip circumference and thigh circumference. It is assumed that the index obtained from measurements of waist and hip circumferences and thigh circumference, after appropriate conversion, should not exceed 0.8 in women and 0.9 in men. Particular attention should be paid to the fact that these methods can be used in adults, not in children and adolescents during the growth period. This is mainly due to differences that occur during growth and development and differences in the metabolism of young and mature individuals. Body composition in children differs significantly from the composition that occurs in humans after the growth.

A frequent mistake in the diagnosis of obesity is the situation where children and adolescents are examined with methods assigned to adults. It is allowed to use this type of classification in some cases in adolescents, where the increase can be considered almost completed. For the diagnostics of obesity in children and adolescents, percentiles [1.4] are used.

Consequences of obesity development and bone tissue

Links between adipose tissue and bone tissue metabolism should be considered taking into

account the type and location of adipose tissue. Percentage of subcutaneous fat is the highest percentage in the body fat mass (about 65-70%). The content of this tissue in the course of individual life is subject to the greatest fluctuations and depending on age and sex may appear in various parts of the body. Subcutaneous fat is the main reservoir of energy in the body, from which it is possible to obtain fatty acids that can be used in metabolic processes. The content of this fraction of adipose tissue undergoes greater fluctuations during the individual life, in comparison with visceral fat, which is located mainly in the abdominal cavity and contributes to the development of abdominal obesity mainly in men [9]. Its percentage share is sexually diverse and in males accounts for about 20% of the total fat content in the body, while in women it is only 5-8%. The specific function of this tissue is to induce abdominal obesity as a result of its excessive accumulation. This is particularly the case in male subjects and in female postmenopausal females. It is the main source of triglycerides and LDL cholesterol fractions that can contribute to the development of atherosclerosis. In addition, visceral fat secretes significantly more adipocytokines than subcutaneous fat (e.g., adiponectin, resistin, IL-6), which have direct or indirect effects on the bone tissue metabolism [10].

When increasing the content of adipose tissue in the body, its functional changes occur, namely increased secretion of leptin and resistin and to reduce the secretion of adiponectin. In addition, adipocytes become the source of, among others proinflammatory cytokines (e.g., IL-6, TNF- α). Visceral fat is a source of free fatty acids (WKT). WKT contribute to reducing the sensitivity of target tissues to insulin, stimulating gluconeogenesis and increased synthesis of very low density lipoproteins (VLDL) leading to the development of insulin resistance. In addition to insulin resistance, obesity may secondarily cause hormonal disturbances in the form of hypothalamo-pituitary-adrenal axis hyperthyroidism, lead to excess testosterone in women, and deficiency in men and cause disorders in the synthesis and secretion of GH [11, 12].

Estrogens and androgens play a key role in the physiology of bone tissue in both females and males. They stimulate osteoblasts to synthesize many factors conditioning the intensification of osteosynthetic processes. They also inhibit the synthesis of many factors responsible for the resorption effect [13, 14]. Estrogen is attributed to the essential function of the inhibitor of bone resorption processes and to the androgens of the bone-forming stimulating factor [15]. The management of sex hormones is also significantly influenced by conditions of overweight or obesity in both men and women. In the case of men with abdominal obesity, testosterone and testosterone-binding proteins are lowered. Restoration of the appropriate testosterone concentration, through treatment with testosterone-containing preparations, leads to the reduction of body fat and improves insulin sensitivity. Testosterone additionally reduces lipid

uptake and enhances lipolysis in visceral fat. In contrast, in women, obesity is accompanied by a decrease in the sex hormone binding protein, which in turn contributes to the increase in the estradiol fraction in the blood. In the case of obese women after menopause, the estrogen concentration is closely correlated with the degree of obesity and fat content [16].

There are reports on the protective effect of high BMI (associated with increased body fat) in the development of osteoporosis and osteoporotic fractures in both women and men over 50 years of age. It turns out that with BMI below normal, along with weight loss, BMD decreases [17,18], and the loss of bone mass associated with age, bone mechanical strength and decrease in total bone mineral content (BMC) are closely correlated with the total fat content in the body [19].

Fat tissue and osteopenia and sarcopenia

According to scientists, not only the state of muscle mass, but also the body weight and percentage of body fat affect the metabolic state of the bone tissue. A recently identified syndrome, manifested by the impaired quality of bone tissue, is the OSO - obesity with osteopenia and sarcopenia (osteosarcopenic obesity) [20]. Coexistence of decreased bone mineral density (BMD), decreased muscle mass and strength, and obesity were determined for postmenopausal women. It was found that such a condition is manifested clinically obesity (eg BMI higher than 30 kg / m² or fat content higher than 30%) [21]. The results of the studies defining the SPA correlate with the results of the work in which it was found that in obese people, despite the fact that their muscles have to carry more weight, muscle strength may be reduced [22]. This affects the mobility, which is impaired, which translates into an increased risk of falling, and thus contributes to the emergence of life-threatening fractures. In the SPA, lower body fat content is found in the lower limbs, which causes slower movements and reduces the speed of movement [23]. According to Liu et al., BMD defined for the femoral neck begins to decrease with the percentage of adipose tissue above 33%, while BMD in the spine - when it exceeds 38% [24]. This is reflected in the worse results of patients with OSO than in the case of people with obesity only, in whom the handshake is much weaker, the speed of movement and the coordination of the body [21].

The reason for this may be the fact that obesity predisposes to a sedentary lifestyle, unfavorable mechanical stimulation of the bones and muscle exercise [25]. Another dependence that may influence the development of OSO is the secretion of pro-inflammatory cytokines, such as tumor necrosis factor or interleukin-6, which negatively affects the muscular system by the adipose tissue [26]. The work of Kersente on energy requirements and leptin, which may affect

the skeleton, are also important in determining the impact on bone tissue [27]. The dependence between the action of leptin and adiponectin - hormones, the level of which directly connects to the development of adipose tissue, also dealt with Lecke et al. They demonstrated the influence of these hormones on the bone in obesity [28]. The correlation between adipose tissue and bone tissue may also be demonstrated by the fact that adipocytes and osteoblasts are derived from the same progenitor cell - the above-mentioned mesenchymal stem cell [29].

The definition of SPAs allows the assessment of persons at risk of fracture in a broader context that has not yet been covered by overweight patients. It was believed that obesity could protect against fractures, among others through higher weighting of the skeleton, which could stimulate bone formation [30]. Despite this, Zhao et al. After correlating the effect of bone stimulation by the load proved that increased body mass negatively interacts with the quality of bone tissue [31]. Another factor suggesting a positive effect of obesity was the fact that the fatty padding could act as a buffer against the force acting on the bone tissue during the fall, and thus protect it from breaking. In addition, many papers have shown that increased body mass and BMI correlate with increased BMD and BMC, and that weight loss causes impairment of bone structure [32.33]. This dependence was to occur regardless of age, even during adolescence [34].

Therefore, it was postulated that the reverse situation, i.e. malnutrition, should predispose to fractures. Traditionally, people who are malnourished and their BMI is below 20kg / m² are considered to be at risk of fracture. This parameter has been found in FRAX calculator. The dependence was based on 12 cohort studies [17]. According to De Laet et al. With a BMI of 20kg / m² compared to a BMI of 25kg / m², the relative risk of a hip fracture rises twice. BMI is independent of the patient. BMI is independent of the bone mineral density. BMI is most related to the hip, however. Researchers prefer BMI to be more useful than traditional weight, which can vary greatly in different populations around the world. De Laet et al., However, do not relate to the relationship between the body and the risk of fracture. Researchers also do not know that BMI is a large impact on fractures.

Fat tissue and osteoporosis

In the study of osteoporosis, conflicting reports on the effect of fat content and / or muscle mass on osteoporosis are available in the literature. Lau et al. Demonstrated that the increase in bone resorption processes by increasing bone turnover in postmenopausal women leads to a reduction in BMD in conditions of reduced body fat content [35]. Christensen et al. Pointed out that the increase in the content of muscle tissue in the body leads to fluctuations in the BMD and BMC

values compared to the control group, and above all the increased content of muscle tissue leads to an increase in BMC [36]. The changes that occur with age in bone tissue depend on a number of factors related to the fact that after reaching peak bone mass (PBM), there is an advantage of resorption processes over bone formation processes, which predisposes to the occurrence of osteoporosis, as it has this place in postmenopausal women. Age-related changes are associated with lowering estrogen levels in women and androgens in men, a decrease in the number of osteoblasts with a progressive decrease in bone marrow adipocytes, which is associated with a lower level of differentiation of mesenchymal stem cells (MSC) towards adipocytes [37, 38]. Increased body mass, with a BMI over 25 kg / m² in the elderly, exerts an osteoprotective effect, where significantly higher BMD values of femurs were found compared to people with a lower value of this index, as demonstrated by Zhao et al. [31]. Opposing dependencies showed Greco et al., Who found that overweight people have lower BMD values in femurs compared to people with normal BMI [14], which proves that increased body weight does not remain indifferent to bone tissue metabolism. In addition, in women with post-menopausal obesity in pQCT studies, higher values of volumetric tibial and humeral bone mineral density were demonstrated compared to women in the same period of life with normal body weight [39].

In studies on the links between body weight and the development of osteoporosis, in addition to the percentage of adipose tissue, attention is increasingly focused on the content of muscle tissue. In perimenopausal women, low muscle mass is closely related to lower BMD values, and some scientists believe that the positive effect of increased body weight in counteracting osteoporosis is associated mainly with increased muscle content, not fat tissue [40,41]. In postmenopausal women with increased body mass, associated with both the higher proportion of muscle tissue and the percentage of adipose tissue, there was a reduced risk of fractures resulting from osteoporosis [42,43], which was not demonstrated in studies in adolescents and adults up to 35 years of age life [44,45].

There is an additional aspect of increased body weight, independent of whether the increase in BMI is due to an increased proportion of muscle mass or percentage of body fat. Increased body weight in older people effectively contributes to lowering the risk of developing osteoporosis by increasing the mechanical load on the bone. The increase in the strength of long bones occurs through the intensification of osteosynthetic processes and inhibition of osteoblast apoptosis, and this directly contributes to the increase in BMD [46], which is a different character of Zhao et al. Mentioned above, who proved reverse dependence [31].

Summary

In conclusion, both obesity and malnutrition and lipid parameters may modulate the properties of the human body, including bone parameters. It is connected with the fact that the percentage of adipose tissue, especially when it exceeds the norms, should be an integral part of diagnostics towards osteoporosis and sarcopenia [47]. Particular attention should be paid to the analysis of body composition, remembering that not only the content of adipose tissue should be taken into account, but also the proportions between the content of adipose tissue and muscle mass, which turns out to be also a key role in the systemic processes.

Literature

1. World Health Organization: Obesity: Preventing and Managing the Global Epidemic (Who Technical Report Series). World Health Organization, 2000
2. Hutley L, Prins JB. Fat as an endocrine organ: Relationship to the metabolic syndrome. *Am J Med Sci* 2005, 330: 280-289
3. Tatoń J, Czech A, Bernas M. Otyłość- zespół metaboliczny. PZWL, Warszawa, 2007
4. Siemińska L. Adipose tissue. Pathophysiology, distribution, sex differences and the role in inflammation and cancerogenesis. *Endokrynol Pol* 2007, 4: 42-50.
5. Reid IR. Relationships between fat and bone. *Osteoporosis Int* 2008, 19: 595-606.
6. Rajala MW, Scherer PE. Minireview: The adipocyte - at the crossroads of energy homeostasis, inflammation, and atherosclerosis. *Endocrinol* 2003, 144: 3765–3773.
7. Skowrońska B, Fichna M, Fichna P. Rola tkanki tłuszczowej w układzie dokrewnym. *Endokrynologia, Otyłość i Zaburzenia Przemiany Materii* 2005, 1, 3: 21-29.
8. Bieńko M, Lis A, Wolski D, et al. Relationship between fat tissue and bone tissue. *Med Weter* 2016, 72, 4: 217-221.
9. Fantuzzi G: Adipose tissue, adipokines, and inflammation. *American Academy of Allergy, Asthma, and Immunology* 2005, 5: 911-919.
10. Cao JJ. Effects of obesity on bone metabolism. *Journal of Orthopaedic Surgery and Research* 2011, 6, 30: 1-7.
11. Trayhurn P, Wood IS. Adipokines: inflammation and the pleiotropic role of white adipose tissue. *The British journal of nutrition* 2004, 92: 347-355
12. Farooqi S, O’Rahilly S. Genetics of obesity in humans. *Endocrinology Review* 2006, 27, 7: 710-718.
13. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. *J Clin Endocrinol metabol* 2004, 89: 2548-2556.
14. Greco EA, Fornari R, Rossi F, et al. Is obesity protective for osteoporosis? Evaluation of bone mineral density in individuals with high body mass index. *Int J Clin Pract* 2010, 64, 6 :817-20.
15. Saxon LK, Turner CH. Estrogen receptor beta: antimechanostat? *2005 Bone*, 36, 185-192.
16. Demissie M., Milewicz A. Zaburzenia hormonalne w otyłości. *Diabetol Prak* 2003, 4, 3, 207-209.
17. De Laet C, Kanis JA, Oden A, et al. Body Mass index as predictor of fracture risk: a meta-analysis. *Osteoporos Int* 2005, 16, 1330-1338.
18. Villareal DT, Fontana L, Weiss EP, et al. Bone mineral density response to caloric restriction-induced weight loss or exercise-induced weight loss: a randomized controlled trial. *Arch Intern Med* 2006, 166: 2502-2510.
19. Shapses SA, Sukumar D. Bone metabolism in obesity and weight loss. *Annu rev Nutr* 2012, 32: 287-309.
20. Ormsbee MJ, Prado CM, Ilich JZ, et al. Osteosarcopenic obesity: the role of bone, muscle, and fat on health. *J Cachexia Sarcopenia Muscle* 2014 5, 3:183-92.
21. Ilich JZ, Inglis JE, Kelly OJ, et al. Osteosarcopenic obesity is associated with reduced handgrip strength, walking abilities, and balance in postmenopausal women. *Osteoporos Int* 2015, 26, 11: 2587-95.
22. Newman AB, Kupelian V, Visser M, et al. Sarcopenia: alternative definitions and associations with lower extremity function. *J Am Geriatr Soc* 2003, 51, 11:1602-9.
23. Shin H, Liu PY, Panton L, et al. Physical performance in relation to body composition and bone mineral

- density in healthy, overweight, and obese postmenopausal women. *J Geriatr Phys Ther* 2014, 37, 1: 7-16.
24. Liu PY., Ilich JZ., Brummel-Smith K, et al. New insight into fat, muscle and bone relationship in women: determining the threshold at which body fat assumes negative relationship with bone mineral density. *Int J Prev Med* 2014, 5, 11: 1452-63.
 25. Rolland Y, Lauwers-Cances V, Cristini C, et al. Difficulties with physical function associated with obesity, sarcopenia, and sarcopenic-obesity in community-dwelling elderly women: the EPIDOS (EPIDemiologie de l'OSteoporose) Study. *Am J Clin Nutr* 2009, 89, 6: 1895-900.
 26. Cesari M, Kritchevsky SB, Baumgartner RN, et al. Sarcopenia, obesity, and inflammation--results from the Trial of Angiotensin Converting Enzyme Inhibition and Novel Cardiovascular Risk Factors study. *Am J Clin Nutr* 2005, 82, 2: 428-34.
 27. Karsenty G, Oury F. The central regulation of bone mass, the first link between bone remodeling and energy metabolism. *J Clin Endocrinol Metab* 2010, 95, 11: 4795-801.
 28. Lecke SB, Morsch DM, Spritzer PM. Leptin and adiponectin in the female life course. *Braz J Med Biol Res* 2011, 44, 5 :381-7.
 29. Akune T, Ohba S, Kamekura S, et al. PPARgamma insufficiency enhances osteogenesis through osteoblast formation from bone marrow progenitors. *J Clin Invest* 2004, 113, 6: 846-55.
 30. Ilich-Ernst J, Brownbill RA, Ludemann MA, et al. Critical factors for bone health in women across the age span: how important is muscle mass? *Medscape Womens Health* 2002, 7, 3: 2-8.
 31. Zhao LJ, Jiang H, Papiasian CJ, et al. Correlation of obesity and osteoporosis: effect of fat mass on the determination of osteoporosis. *J Bone Miner Res* 2008, 23, 1: 17-29.
 32. Felson DT, Zhang Y, Hannan MT, et al. Effects of weight and body mass index on bone mineral density in men and women: the Framingham study. *J Bone Miner Res* 1993, 8, 5:567-73.
 33. Ravn P, Cizza G, Bjarnason NH, et al. Low body mass index is an important risk factor for low bone mass and increased bone loss in early postmenopausal women. Early Postmenopausal Intervention Cohort (EPIC) study group. *J Bone Miner Res* 1999, 14, 9:1622-7.
 34. Sabatier JP, Guaydier-Souquières G, Benmalek A, et al. Evolution of lumbar bone mineral content during adolescence and adulthood: a longitudinal study in 395 healthy females 10-24 years of age and 206 premenopausal women. *Osteoporos Int* 1999, 9, 6:476-8.
 35. Lau EM, Chan YH, Chan M, et al. Vertebral deformity in Chinese men: prevalence, risk factors, bone mineral density, and body composition measurements. *Calcif Tissue Int* 2000, 66, 47-52.
 36. Christensen P, Riecke BF, Bliddal H, et al. Improved nutritional status and bone health after diet-induced weight loss in sedentary osteoarthritis patients: a prospective cohort study. *Europ J Clin Nutr* 2012, 66, 504-509.
 37. Gimble JM, Zvonic S, Floyd ZE, et al. Playing with bone and fat. *J Cell Biochem* 2006, 98, 251-266.
 38. Jilka RL, Weinstein RS, Parafitt AM, et al. Quantifying osteoblast and osteocyte apoptosis: challenges and rewards. *J Bone Miner Res* 2007, 22, 192-151.
 39. Sornay-Rendu E, Boutroy S, Vilayphiou N, et al. In obese postmenopausal women, bone microarchitecture and strength are not commensurate to greater body weight: the Os des Femmes de Lyon (OFELY) study. *J Bone Miner Res* 2013, 28, 7:1679-87.

40. Sowers MF, Kshirsagar A, Crutchfield MM, et al. Joint influence of fat and lean body composition compartments on femoral bone mineral density in premenopausal women. *Am J Epidemiol* 1992, 136: 257-265.
41. Salamone LM, Glynn N, Black D, et al. Body composition and bone mineral density in premenopausal and early perimenopausal women. *J Bone Miner Res* 1995, 10: 1762-1768.
42. Chen Z, Lohman TG, Stini WA, et al. Fat or lean tissue mass: Which one is the major determinant of bone mineral mass in healthy postmenopausal women? *J Bone Miner Res* 1997, 12: 144-151.
43. Douchi T, Yamamoto S, Oki T, et al. Difference in the effect of adiposity on bone density between pre- and postmenopausal women. *Maturitas* 2000, 34: 261-266
44. Goulding A, Jones JE, Taylor RW, et al. Bone mineral density and body composition in boys with distal forearm fractures: a dual-energy x-ray absorptiometry study. *J Pediatr* 2001, 139: 509-515.
45. Janicka A, Wren TA, Sanchez MM, et al. Fat mass is not beneficial to bone in adolescents and young adults. *J Clin Endocrinol Metab* 2007, 92: 143-147.
46. Lenchik L, Register TC, Hsu FC, et al. Adiponectin as a novel determinant of bone mineral density and visceral fat. *Bone* 2003, 33: 646-651.
47. Domiciano DS, Figueiredo CP, Lopes JB, et al. Discriminating sarcopenia in community-dwelling older women with high frequency of overweight/obesity: the São Paulo Ageing & Health Study (SPAH). *Osteoporos Int* 2013, 24, 2:595-603.