ISSN 2618-1576 Vol. 4, Issue 5: 39-43, DOI: 10.25236/FSR.2022.040510

Effects of high-intensity interval exercise on obesity

Jin Zhan, Ruan Ling*, Wang Guanghua, Wu Rongping

Department of Physical Education, Xi 'an Shiyou University, Xi'an, China
**Corresponding author*

Abstract: *With the increasing improvement of people's living standards, obesity has become a chronic disease all over the world, which can make people's physical shape and various physical indicators abnormal, resulting in a variety of complications, posing a threat to people's lives. Osteoporosis (OP) is one of the complications caused by Osteoporosis, which is mainly characterized by decreased bone density and easy to fracture. Obese people with OP have a higher probability of fracture than normal people. Once fracture occurs, they will lose their ability to exercise and may even threaten their lives. Exercise is one of the measures against obesity. High-intensity Interval Training (HIIT) has been widely used in the field of weight loss, and the right amount of exercise can prevent OP. In this paper, a large number of literature was reviewed to find out the possible mechanism of HIIT in preventing and treating OP in obese people, so as to reduce the probability of obesity and obese people suffering from OP.*

Keywords: *high-intensity interval exercise; Obesity; Osteoporosis; Inflammatory cytokines*

1. Introduction

As a chronic disease spreading all over the world and becoming more and more serious, obesity poses a great threat to people's life. One study found that the BMI of boys and girls increased by 0. 32 and 0. 40kg/m per decade, respectively, from 1975 to 2016, following a continuous study of 24. 1 million adolescents aged 5 to 17. The number of obese people is gradually increasing each year, and the number of deaths due to other complications of obesity is also increasing year by year. According to the White Paper on Osteoporosis Prevention and Treatment in China, 70, 000 people in China suffer from OP, and another 210 million people have lower than normal bone mineral density. Physical exercise is one of the most effective ways to improve obesity, among which HIIT is more easily accepted and adhering to by obese people because of its characteristics of short exercise time, small space occupation and high energy consumption [1], so it is widely used in weight loss to achieve people's goal of weight loss. There have been extensive studies on HIIT abroad. Based on previous studies, this paper proved that HIIT as an intervention measure for obesity can prevent OP in obese people , focusing on the OP in overweight people through the reduction of fat.

2. The origin and development of HIIT

HIIT was used 100 years ago by a track and field athlete in track training [2]. In 1930, exercise physiology expert Hans showed that HIIT combined with a well-timed recovery could improve cardiorespiratory function more effectively. Since then, it has been added to the training of sports on a large scale [3]. In the 1960s and 1970s, researchers began to study HIIT in greater depth. Physiologist Astrand[4] proposed a method of interval training with a critical speed and a speed between VO2max (maximum oxygen uptake, VO2max) (90% ~ 95% VO2max) for a long time. Astrand et al. [4, 5] coordinated exercise and relaxation to enable exercisers to achieve the best physical state. Christensen4 et al. [6] proposed the method of conducting extremely short interval training with 100% VO2max. During 1970-1980, HIIT was explored from the perspective of human body and exercise, and a comparative analysis was made between HIIT and exercise under high lactic acid for a long time [7], and no significant difference was found between them. In the 1980s and 1990s, there was less research on interval training because of the different direction of research. In recent years, HIIT research has set off a new upsurge of research. According to statistics, from 2013 to 2020, there will be 220 domestic research papers and 470 foreign academic papers. Research results at home and abroad reflect that HIIT can improve the functions of human organs, tissues and systems in many aspects.

ISSN 2618-1576 Vol. 4, Issue 5: 39-43, DOI: 10.25236/FSR.2022.040510

3. The relationship between fat and OP

OP is caused by a decrease in mineral content in the bone and damage to the internal structure of the bone, mainly slowing down bone metabolism and bone strength below normal levels. Causing obesity mainly depends on eating type imbalance, the energy that the human body uses is lower than the energy that eating produces, the remaining energy is the liver synthesizes triglyceride to transport to adipose cell through the blood, bring about adipose accumulation to build fat. On the relationship between the two, there are two kinds of opposed views, one view is that with obesity can reduce the probability of OP, and two views are: (1) the overweight participants stress than general people's congress, for the stimulation of bone can accelerate the formation of bone, and inhibit bone absorption, helps increase bone density and minerals; Another explanation is that body mass consists of not only fat, but also muscle and bone, and muscle mass may have a greater impact on body mass compared with bone density due to higher fat density. This considered obesity is not an increase in fat, but an increase in muscle mass [8]. (2) Obesity can lead to the occurrence of OP. Some scholars pointed out that type 2 diabetes mellitus (T2DM) is one of the inducement factors of OP, because long-term hyperglycemia may lead to bone tissue dysfunction, thus increasing the risk of OP [9]. Some scholars believe that although the increase of fat content will improve the strength of bone, it has a certain limit for human body. When the increase of fat content reaches a certain level, it will have a negative effect on bone, resulting in OP, and then the deformation of bone. And different locations of fat have different effects on bones[10].

4. Effects of HIIT on OP in obese patients

4.1. The effect of obesity on OP

Kathryn[11] believed that obesity is one of the risk factors for fracture, and the relationship between obesity and fracture risk may be determined by the location of fat distribution. Although greater body weight is beneficial for bones, different fat distribution can alter the relationship. Studies have found that increased fat mass and body fat percentage are positively correlated with bone mineral density, obesity can increase bone mineral density, and higher bone mineral density can reduce the occurrence of OP [12]. Another study found that obesity was highly correlated with OP, and the increase of abdominal fat was negatively correlated with bone mineral density. The more abdominal fat accumulated, the greater the impact on bone mineral density, and the corresponding increase in the prevalence of OP. AbDOMinal fat is composed of visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT). Studies have shown that VAT has a negative effect on OP, while SAT and OP have a positive effect [11]. Vat can secrete a large amount of Leptin (LEP). Other studies have found that LEP receptors exist on the surface of osteoblast (OB) and chondrocytes, and LEP can reduce differentiation of osteoclast (OC) and accelerate OB division, mineralization of bone minerals, and accelerate collagen synthesis [13]. LEP in the human body is positively correlated with body mass and fat content. Compared with normal body weight, serum LEP level of obese patients is significantly higher. ObESE individuals secrete more LEP due to fat, which has a protective effect on OP. However, obese people have LEP resistance, so LEP cannot play its due role [14], the mechanism may be that LEP passes through the blood-brain barrier. It enters the skull and activates the OB-Rb receptor in the hypothalamus, so that the hypothalamus is stimulated to secrete inhibitory OB factor [15]. VAT can also cause inflammation and promote the secretion of inflammatory cytokines. Inflammation plays a key role in OP. Inflammatory cytokines produced by fat cells link obesity and its metabolism-producing diseases, Tumor necrosis factor-α (TNF-α), interleukin (interleukin) and TNF-α were the main inflammatory cytokines. IL-1β, IL-6, transforming growth factor-β (TGF-β), IL-8, IL-L0, etc. [16-19]. The enhanced expression of IL-6 could reduce bone mineral density. Among them, the GG genotype of -634C/G polymorphism in IL-6 may play a role in reducing bone mineral density, interfering with normal bone metabolism, leading to the increase of OP[20] and TNF-α concentration, enhancing bone resorption, increasing the activity of OC, and then inducing OP. Studies have found that in menopausal women with OP, TNF-α can induce the formation of OC by activating NF-κB (enhanced κ-light chain of nuclear factor-activated B cells, NF- κB) and PI3K/Akt (phosphatidlinositol 3-excitol/protein kinase B, PI3K/Akt) signaling pathways and synergically promoting RANKL (nuclear factor-isogenic B receptor activator ligand, RANKL). Moreover, interference with PI3K/Akt completely blocked the synergistic effect of TNF-α on NF-κB activation and OC generation [21]. Fas can bind to the Fas receptor on the cell surface and mediate cell apoptosis. RANKL at high concentration can inhibit the concentration of Fas protein (TNF receptor superfamily member 6, Fas) and mRNA, the activation of caspase-3 of Fas and the death of OC induced by Fas. However, RANKL decreased Fas expression in mature OC and increased Fas expression in OC by NF-κB[22] .

ISSN 2618-1576 Vol. 4, Issue 5: 39-43, DOI: 10.25236/FSR.2022.040510

Therefore, OP is closely related to the location of obesity, and visceral fat is negatively correlated with OP. The mechanism is that the increase of adipocytes will secrete more inflammatory cytokines, and IL-6 and TNF-α in inflammatory cytokines will lead to the decrease of bone density, thus inducing OP.

4.2. Impact of HIIT on OP

The World Health Organization has given three recommendations to prevent OP: calcium supplementation, moderate exercise and diet regulation, and even gave specific exercise prescriptions to OP patients. From the perspective of biomechanics, stimulation of muscle contraction can reduce bone loss. From a medical perspective, normal muscle content is conducive to the maintenance of normal bone mass [23]. From the perspective of genes, IGF-1 (growth promoting factor, IGF-1) in muscle tissue can promote bone development [24]. Combined with all aspects of the study, sports has a positive effect on bone mineral density, and moderate load exercise can prevent bone loss and/or cause a small amount of bone mass increase [25], that is to say HIIT can inhibit the occurrence of the OP, but HIIT's influence on the bone with two different points of view, a view that HIIT for bone presents the negative effect, Specifically, the loss of bone mineral salt, reduction of bone area, and decrease of bone density increase the incidence of OP. Another believes that HIIT can improve bone mineral density and reduce the incidence of OP . Because the two views adopt different exercise programs, the results are also different. In summary, HIIT can increase bone density while also preventing OP.

4.3. HIIT works by reducing the impact of fat on OP

In this paper, we summarize previous conclusions to prove that HIIT can reduce fat and play a role in preventing OP. HIIT can reduce fat in obese people of all ages. For example, Liang Jinyu et al. conducted HIIT intervention on 18 obese children and found that HIIT can reduce the weight, body fat percentage and VAT of obese children, indicating that HIIT has a significant effect on obesity. Wang Jingjing et al. conducted HIIT intervention on 24 obese women and found that it had a significant effect on reducing body fat, especially abdominal fat. Buckinx et al. conducted HIIT on 30 elderly obese people for 12 weeks, and found that the elderly obese people had significantly less fat [26]. [27] HIIT can effectively reduce abdominal fat, reduce the incidence of T2DM, dyslipidemia and hypertension, and also reduce metabolic disorders and improve the incidence of related diseases [28]. HIIT can increase insulin sensitivity, reduce insulin resistance [29 30], reduce triglyceride (TG) , and increase the rate of fat decomposition, thereby reducing fat. HIIT can effectively reduce inflammation and have a positive impact on the body [31]. HIIT can also improve the expression of TNF-α and IL-6 in LEP and inflammatory cytokines, reduce their expression [32-34], inhibit the -634C/G polymorphism of GG genotype expression in IL-6, reduce TNF-α, inhibit the NF-κB and PI3K/Akt signaling pathway and synergistic effect with RANKL. Reduced OC formation and low RANKL concentration could not inhibit Fas protein and mRNA levels, Fas induced caspase-3 activation, Fas action and Fas mediated OC death, thus accelerating Fas induced OC death and ultimately reducing the incidence of OP (Figure 1).

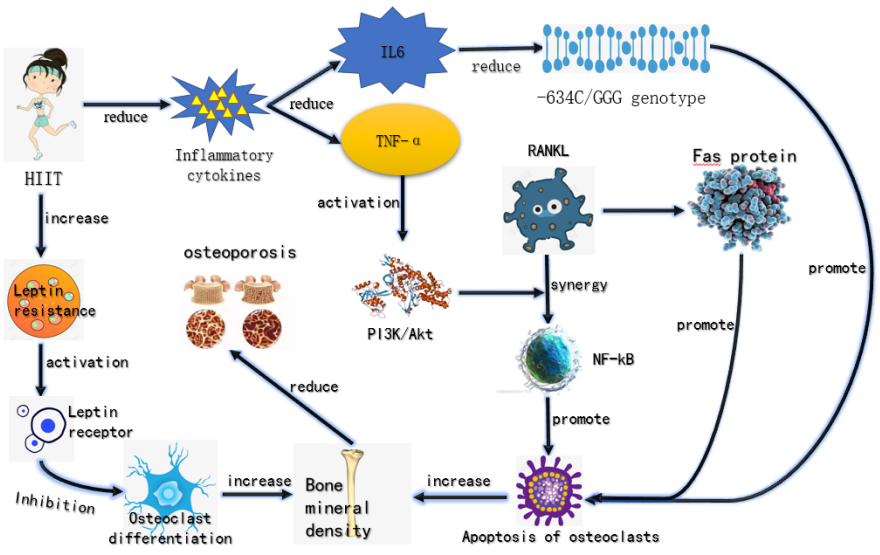

Figure 1: The mechanism of high intensity interval exercise on osteoporosis

ISSN 2618-1576 Vol. 4, Issue 5: 39-43, DOI: 10.25236/FSR.2022.040510

To sum up, HIIT can remove body fat more effectively from many aspects, reduce body fat rate, reduce complications caused by obesity, reduce fat in internal organs, increase the secretion of lipohydrolytic hormone, reduce fat from the inside out, and improve health. At the molecular level, HIIT can reduce inflammatory factors produced by obesity, including IL-6 and TNF-α, so as to strengthen bone and reduce the occurrence of OP.

5. Conclusion

This review is based on previous studies on HIIT as an exercise intervention to reduce the prevalence of OP in obese people. Compared with other methods to prevent OP in obese people, HIIT has its unique role. While preventing obesity, it can also improve the cardiopulmonary function, reduce the body fat rate, reduce the complications of obesity, and improve the body metabolism, etc.

From the perspective that fat can inhibit OP, although fat can inhibit the incidence of OP, but for human health is not good, obesity can lead to a variety of complications, but also can lead to metabolic disorders, and then threaten people's lives. From the point of view of human physiology, reducing fat is beneficial to the body. Exercise can not only reduce fat, but also increase bone density and reduce the incidence of OP. At present, it is the best choice for both reducing fat and preventing OP. HIIT can reduce the percentage of body fat in the abdomen, which is extremely harmful to the human body and easily causes OP. Therefore, HIIT plays a positive role in preventing OP in obese people, strengthening physical fitness and reducing other diseases caused by obesity. HIIT can alleviate the release of inflammatory factors such as IL-6 and TNF-α caused by obesity, promote the apoptosis of OC and increase bone density, and ultimately reduce the incidence of OP in obese people.

This paper only verified HIIT prevention of OP in obese people, and there is no training program for HIIT prevention of OP in obese people, especially the specific high-intensity training program for the elderly with a high incidence of OP. This aspect is still a vacancy and needs further study.

References

[1] Thum Jacob S, Parsons Gregory, Whittle Taylor, et al. (2017) High-Intensity Interval Training Elicits Higher Enjoyment than Moderate Intensity Continuous Exercise. PloS one , 12(1) :e0166299
[2] Billat L V. (2001) Interval training for performance: a scientific and empirical practice. Special recommendations for middle- and long-distance running. Part I: aerobic interval training. Sports medicine (Auckland, N. Z.) , 31(1):13-31
[3] Daniels J, Scardina N. (1984) Interval training and performance. Sports medicine, 1(4):327-34
[4] Astrand I, Astrand P O, Christensen E H, et al. (1960) Intermittent muscular work. Acta physiologica Scandinavica, 25(48):448-53
[5] Astrand I, Astrand P O, Christensen E H, et al. (1960) Myohemoglobin as an oxygen-store in man. Acta physiologica Scandinavica , 25(48):454-60
[6] Christensen E H, Hedman R, Saltin B. (1960) Intermittent and continuous running. (A further contribution to the physiology of intermittent work.). Acta physiologica Scandinavica, 30(50):269-86
[7] Stephen Seiler, Ken J. Hetlelid. (2005) The Impact of Rest Duration on Work Intensity and RPE during Interval Training. Medicine & Science in Sports & Exercise, 37(9):1601-7
[8] Vandewalle S, Taes Y, Van Helvoirt M, et al. (2013) Bone size and bone strength are increased in obese male adolescents. The Journal of clinical endocrinology and metabolism, 98(7):3019-28
[9] Napoli Nicola, Chandran Manju, Pierroz Dominique D, et al. (2017) Mechanisms of diabetes mellitus-induced bone fragility. Nature reviews. Endocrinology, 13(4):208-219
[10] Jennifer S. Walsh, Tatiane Vilaca. (2017) Obesity, Type 2 Diabetes and Bone in Adults. Calcified Tissue International, 100(5):528-535
[11] H. E. Meyer, W. C. Willett, A. J. Flint, et al. (2016) Abdominal obesity and hip fracture: results from the Nurses' Health Study and the Health Professionals Follow-up Study. Osteoporosis International, 27(6):2127-36
[12] Neglia Cosimo, Argentiero Alberto, Chitano Giovanna, et al. Diabetes and Obesity as Independent Risk Factors for Osteoporosis: Updated Results from the ROIS/EMEROS Registry in a Population of Five Thousand Post-Menopausal Women Living in a Region Characterized by Heavy Environmental Pressure. [J]. International journal of environmental research and public health, 2016, 13(11):1067.
[13] Rudolph L. Leibel M. D. (2020) The Role of Leptin in the Control of Body Weight. Nutrition Review, 60:S15-9
[14] Abhiram Sahu. (2003) Leptin signaling in the hypothalamus: emphasis on energy homeostasis and

ISSN 2618-1576 Vol. 4, Issue 5: 39-43, DOI: 10.25236/FSR.2022.040510

leptin resistance. Frontiers in Neuroendocrinology, 24(4):225-53
[15] Tang Zi-Hui, Xiao Peng, Lei Shu-Feng, et al. (2007) A bivariate whole-genome linkage scan suggests several shared genomic regions for obesity and osteoporosis. The Journal of clinical endocrinology and metabolism, 92(7):2751-7
[16] Bogusław Czerny, Adam Kaminski, Mateusz Kurzawski, et al. (2009)The association of IL-1β, IL-2, and IL-6 gene polymorphisms with bone mineral density and osteoporosis in postmenopausal women. European Journal of Obstetrics and Gynecology, 149(1):82-5
[17] Andrews Nancy C. (2004) Anemia of inflammation: the cytokine-hepcidin link. The Journal of clinical investigation, 113(9):1251-3
[18] Friebe H, Peters A. (2005) Obesity and osteoporosis. Der Orthopade , 34(7):645-51
[19] Jasminka Z. Ilich, Owen J. Kelly, Youjin Kim, et al. (2014) Spicer. Low-grade chronic inflammation perpetuated by modern diet as a promoter of obesity and osteoporosis. Archives of Industrial Hygiene and Toxicology, 65(2):139-48
[20] Yan L, Hu R, Tu S, et al. (2015) Meta-analysis of association between IL-6 -634C/G polymorphism and osteoporosis. Genetics and molecular research : GMR, 14(4):19225-32
[21] Li Zha, Li He, Yijian Liang, et al. (2018) TNF-α contributes to postmenopausal osteoporosis by synergistically promoting RANKL-induced osteoclast formation. Biomedicine & Pharmacotherapy, 102:369-374
[22] Xiaojun Wu, George Pan, Margaret A McKenna, et al. (2005) RANKL Regulates Fas Expression and Fas-Mediated Apoptosis in Osteoclasts. Journal of Bone and Mineral Research, 20(1):107-16
[23] Sarah L. Manske, Steven K. Boyd, Ronald F. Zernicke. (2011) Vertical ground reaction forces diminish in mice after botulinum toxin injection. Journal of Biomechanics, 44(4):637-43
[24] J. Banu, L. Wang, D. N. Kalu. (2003) Effects of Increased Muscle Mass on Bone in Male Mice Overexpressing IGF-I in Skeletal Muscles. Calcified Tissue International, 73(2):196-201
[25] Prince Richard L, Smith Margaret, Dick Ian M, et al. (1991) Prevention of Postmenopausal Osteoporosis:A Comparative Study of Exercise, Calcium Supplementation, and Hormone-Replacement Therapy. The New England Journal of Medicine, 325(17):1189-95
[26] F. Buckinx, P. Gaudreau, V. Marcangeli, G. El Hajj Boutros, et al. (2019) Muscle adaptation in response to a high-intensity interval training in obese older adults: effect of daily protein intake distribution. Aging Clinical and Experimental Research, 31(6):863-874
[27] F. Maillard, S. Rousset, B. Pereira, et al. (2016) High-intensity interval training reduces abdominal fat mass in postmenopausal women with type 2 diabetes. Diabetes and Metabolism, 42(6):433-441
[28] Klara J. Rosenquist, Alison Pedley, Joseph M. Massaro, et al. (2013) Visceral and Subcutaneous Fat Quality and Cardiometabolic Risk. JACC: Cardiovascular Imaging, 6(7):762-71
[29] Ningning Wang, Yang Liu, Yanan Ma, et al. (2017) High-intensity interval versus moderate-intensity continuous training: Superior metabolic benefits in diet-induced obesity mice. Life Sciences, 15(191):122-131
[30] de Matos Mariana Aguiar, Vieira Dênia Vargas, Pinhal Kaio Cesar, et al. (2018) High-Intensity Interval Training Improves Markers of Oxidative Metabolism in Skeletal Muscle of Individuals With Obesity and Insulin Resistance. Frontiers in physiology, 31(9):1451
[31] Ali Mohammad Alizadeh, Amin Isanejad, Sanambar Sadighi, et al. (2019) High-intensity interval training can modulate the systemic inflammation and HSP70 in the breast cancer: a randomized control trial. Journal of Cancer Research and Clinical Oncology, 145(10):2583-2593
[32] Khalafi Mousa, Symonds Michael E. (2020) The impact of high-intensity interval training on inflammatory markers in metabolic disorders: A meta-analysis. Scandinavian journal of medicine & science in sports, 30(11):2020-2036
[33] Gerosa-Neto José, Antunes Barbara M M, Campos Eduardo Z, et al. (2016) Impact of long-term high-intensity interval and moderate-intensity continuous training on subclinical inflammation in overweight/obese adults. Journal of exercise rehabilitation, 12(6):575-580
[34] Leggate Melanie, Carter Wayne G, Evans Matthew J C, et al. (2012) Determination of inflammatory and prominent proteomic changes in plasma and adipose tissue after high-intensity intermittent training in overweight and obese males. Journal of applied physiology, 112(8):1353-60