Biomechanics of Bone: Factors That Contribute to Osteoporosis and Fractures and How to Combat This Risk

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Abstract: Reducing the risk of fracture and osteoporosis is particularly important as they have been a worldwide health challenge, resulting in increased mortality and economic loss. According to relevant data, there are about 23,700,000 people getting disease because of fracture and osteoporosis, which proves the harmlessness of it (Al Anouti, F.et al, 2019). Bone delicacy is closely related with the chance of break and osteoporosis, bone delicacy or bone quality is influenced by a assortment of variables, including bone density, vitamin D, external forces and bone aging (Fonseca, et al, 2014). This paper addresses the factors that influence bone fragility from a biomechanical perspective and suggests appropriate solutions to reduce the risk of fracture and osteoporosis due to bone fragility. It was concluded that the natural aging of bones is the main factor among many others, while the lack of vitamin D and external forces affect bone density to accelerate this process.The risk of fracture and osteoporosis is reduced by appropriate exercise and moderate intake of vitamin D.

1 INTRODUCTION

World widely, osteoporosis causes nearly 10 million fractures each year, with an osteoporotic fracture occurring every 3 seconds (Chao, et al, 2004). And there is the academic result showing that the goal of treating bone fragility is to increase strength and reduce fragility (Turner, 2002). In the real implementations, the following questions are shown for this research topic. First and foremost, there are few normal people except professional doctors knowing the real principle of how fracture and osteoporosis form and cause relevant bone diseases. Besides, people who are getting these kinds of diseases do not know much about Vitamin. As a result, the lack of this information results in a more serious degree of fracture and osteoporosis. Last but not least, there is little attention and focus on this kind of problem in the whole society, which requires more relevant researches to change it. Based on these conditions, this research focuses on the factors that influence bone fragility from a biomechanical perspective and suggests appropriate solutions to reduce the risk of fracture and osteoporosis due to bone fragility. To make the research aims in detail, the following questions can be achieved.

RQ1. What are factors that influence bone fragility from a biomechanical perspective?

RQ2. What are key factors that reduce the risk of fracture and osteoporosis due to bone fragility?

Based on the information above, the significance of this research can be summarized in the following two aspects. On the one hand, there will be more information for normal people to protect their bones in daily life based on the solutions and other recommendations mentioned in this research. Therefore, this social problem can get solved more. On the other hand, this research can provide evidence for more professional and longer-term academic research in the future.

2 DECLINE IN BONE MASS DUE TO AGEING

Osteoporosis as a bone malady is clinically characterized by decreased bone quality and a propensity to break (Fonseca, et al, 2014). Several studies have shown that skeletal changes are age-related which maturing can bring almost a huge number of skeletal changes within the tissue and basic levels (Abraham, et al, 2016). Ageing causes a

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slowing of metabolism andisfortune of calcium from the skeleton, leading to a general decline in bone mechanics and a consequent expanded hazard of break.

In expansion to this, age-related skeletal changes are not simply a metabolic issue; the capacity of tissues to stand up to break is decided by their particular composition and structure. 1 Bone could be a composite fabric comprising primarily of sort I collagen, with a little sum of other non-collagenous proteins and proteoglycans, and hydroxyapatite gems develop amid biomineralisation.

This property of the bone strands permits them to retain stretch through versatile misshapening and to resist tall loads some time recently break. The mineral stage is basically capable for the capacity to stand up to distortion (hardness), whereas the collagen strands can assimilate vitality (durability). Changes in either composition may therefore affect the mechanical properties of the bone and thus the risk of fracture. 4The two-phase composition of minerals and proteins in bone moreover gives it a special combination of tall quality and durability. While quality decides the most extreme stack that the bone can withstand, sturdiness decides the bone's capacity to scatter vitality, stand up to basic harm, and subsequently trigger bone reconstruction to repair microdamage to damaged bone tissue. Bone toughness decreases with age and disease, thereby increasing the risk of fracture (Abraham, et al, 2016); (Hernandez, Keaveny, 2006). When strain reaches a critical limit that cannot be tolerated, damage gradually develops within the material as microcracks develop, which can be shown in the following figure.

Figure 1: The comparison between different conditions of bones.

The quality and durability of bone are hence profoundly subordinate on the capacity of the bone to disseminate the stresses that cause expanded strain, and on the microstructural properties that prevent crack extension. Aging and disease lead to an increase in intracortical porosity. Changes in collagen and minerals in bone. As we age, bone cell traps become smaller and more spherical. Experimental studies have shown that as porosity increases, the fracture toughness of bone decreases significantly and the mineral content of bone increases (Hemmatian, et al, 2017); (Ural, Vashishth, 2007).

Estrogen shows up to be a major controller of skeletal digestion system not as it were in ladies but also in men. 8 Bone deficiency is partly caused by a deficiency of sex hormones. In particular, post-menopausal women have reduced levels of oestrogen, resulting in an overall negative balance between bone resorption and formation rates. The impaired bone structure may be due to a reduced ability of osteoblasts to control local osteoblast and/or osteoclast recruitment. Indeed, there is strong evidence that reduced osteoclast sensitivity is associated with age-related bone loss. For instance, people who are aging are prone to getting rheumatism (Al Anouti, et al, 2019). Thus, oestrogen deficiency and impaired osteocyte mechanosensitivity may be major risk factors for osteoporotic fractures (Fonseca.et al, 2014).

The shape of osteoblasts and their traps changes considerably with age. As osteocytes can directly sense matrix strain through the cell body, changes in osteocyte morphology may lead to alterations in osteocyte mechanical sensitivity. Thus, the load-adaptive response of osteocytes may change with age, even when mechanical load remains constant. Although substantial quantitative data are lacking, there is evidence that osteocyte traps

become smaller and more rounded with age (Fonseca.et al, 2014).

3 VITAMIN D AND BONE FRAGILITY

In studies of skeletal biomechanics in small animals, a low calcium diet (LCD), reduced calcium absorption and increased loss have been found to be some of the important mechanisms that may contribute to bone loss (Jiang.et al, 1997); (Vashishth, 2008). Bone as a living tissue is more accurately described as a mineralized tissue, and its complexity is reflected in the fact that it undergoes morphological changes in order to constantly adapt to metabolic and structural demands. All changes in the morphology of the external bone occur on the surface of the periosteum, where complex anabolic and catabolic processes take place (Roberts, et al, 2004). Thus, metabolic changes caused by food intake and exercise can affect the chemistry of the periosteal surface.

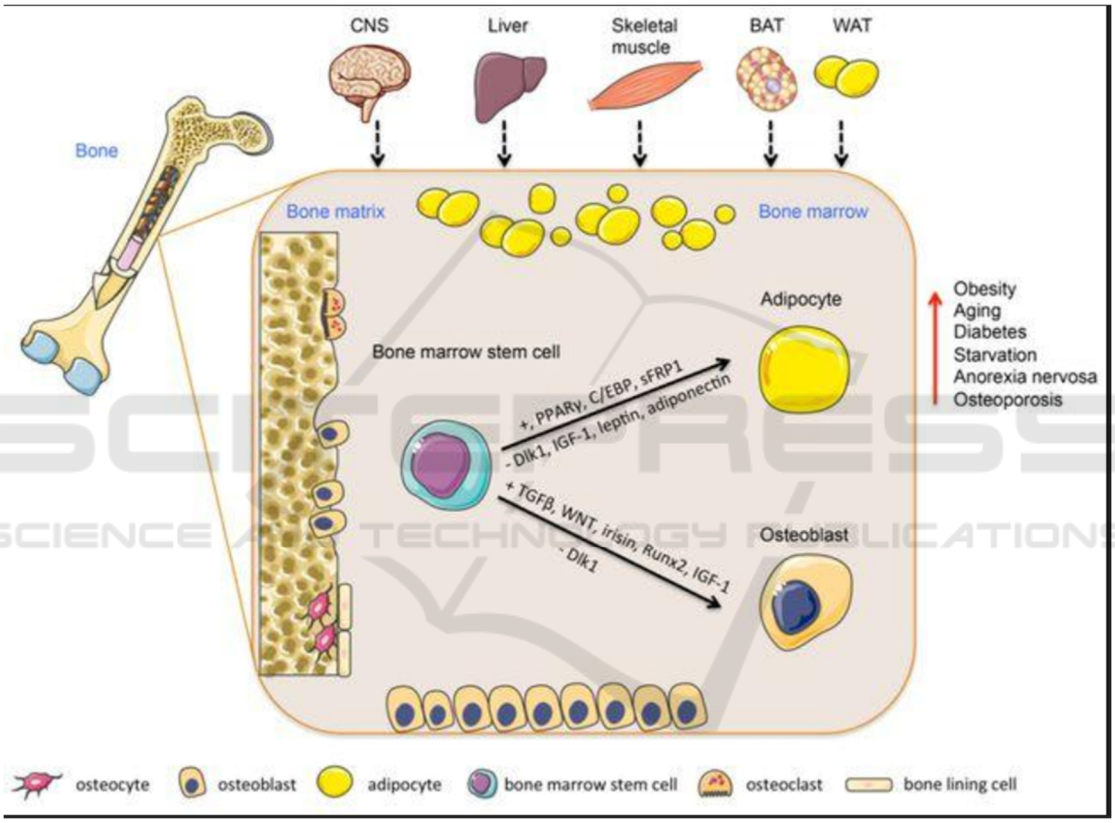

Figure 2: How Vitamin D influences bones (Al Anouti, F.et al, 2019).

Vitamin D insufficiency may be a broad clutter that plays an imperative part in human bone wellbeing. Vitamin D lack causes maturing of the human skeleton and increments the hazard of break. In the presence of vitamin D deficiency, fracture susceptibility is mainly associated with defects in the mineralisation of the collagen matrix (bone-like material). Vitamin D is broad in nature and one of its parts in vertebrates and people is to advance the retention of calcium and phosphorus so that bones can mineralise appropriately. Vitamin D deficiency in childhood predisposes to rickets, and defects in growth plate cartilage and bone mineralisation lead to altered long bone morphology, resulting in curvature and deformity.In grown-ups, vitamin D lack leads to osteochondrosis, a condition of flawed mineralisation where the recently shaped bone lattice (osteoid) falls flat to mineralise, driving to bone torment, muscle shortcoming and an expanded hazard of bone deformation and break (Busse, et al, 2013).

Vitamin D deficiency is a low serum 25(OH)D3 concentration associated with reduced serum 1,25-(OH)2D3 and calcium absorption. In turn, low

blood calcium leads to increased secretion of parathyroid hormone (PTH), which promotes the production of 1,25-(OH)2D3(5). As a result, serum 1, 25-(OH) 2D3 concentrations return to normal but are accompanied by higher serum parathyroid hormone concentrations, suggesting secondary hyperparathyroidism. Increased serum parathyroid hormone concentrations have been reported to stimulate the rate of bone conversion, leading to a reorganisation of bone structure. Secondary hyperparathyroidism has therefore been suggested to be a major factor in the increased susceptibility to fracture due to vitamin D deficiency. Treatment with vitamin D3 and calcium does significantly reduce the incidence of non-vertebral fractures. However, this was only achieved with little change in bone mineral density (BMD) and serum parathyroid hormone (PTH) concentrations, suggesting that other factors play a role in reducing fracture risk. Vitamin D deficiency increases bone turnover to maintain normal calcium levels in the body, producing bone-like bone that never mineralises because of an overall calcium imbalance. The deterioration in mechanical properties due to vitamin D deficiency is associated with the accumulation of large amounts of osteoid on the bone surface and impaired resorption below the bone surface, as evidenced by the accumulation of unreconstructed traps of highly mineralised bone cells. Localised tissue ageing is a key cause of the increased risk of fracture in vitamin D deficiency osteochondrosis, and tissue ageing affects the resorption energy of bone by limiting bone plasticity (Busse, et al, 2013).

4 METHODS OF REDUCING THE RISK OF FRACTURES

Mechanical and biophysical stimulation can be effective in promoting fracture healing in elderly patients under less than ideal circumstances. Different stimuli may limit their association with specific healing mechanisms. However, accurate repositioning is necessary for fracture healing, regardless of the method of fixation used. Misalignment of the fracture site will result in delayed healing, deformed healing, or no healing. When elderly patients with long bone fractures are unable to perform the necessary rehabilitation program, including partial weight-bearing exercises, after fracture fixation, adjunctive physical or biophysical stimulation can be applied to promote bone healing and improve the quality of life of these bedridden or wheelchair-bound patients.

Different types of stimulation have been shown to be effective for fresh fractures or delayed healing. As a result, more basic science research and clinical trials are needed to make these potentially powerful alternative medicine modalities more reliable. Through signaling transducer design, tissue response monitoring, dose and signal optimization, and individualized and knowledge-based treatment protocols for each patient and the fracture involved. Considering all these special factors, the outcome of fracture treatment in elderly patients and patients with osteoporosis should not be different from other fracture patients. Coordinated research and development in relevant biomechanical areas is needed to prepare us for the exponential growth of the global aging population in the coming decades (Chao, et al, 2004).

Bones benefit from regular physical activity. Athletes typically have higher bone mass than sedentary individuals, and prospective studies have shown that exercise increases bone mass in humans and experimental animals and experimental animals (Turner, 2002). Although the increase in bone density due to exercise is evident at younger ages, the increase is small in adults. Despite this apparently small effect, sedentary behavior is a known risk factor for hip fracture, with men and women who exercise regularly having up to half the risk of hip fracture than sedentary men and women. This reduction in fracture risk in physically active adults must then be achieved by altering other meaningful attributes that have an effect on bone strength independent of BMD, as well as other non-skeletal variables that significantly affect fracture occurrence (e.g., fall risk), if only a slight increase in BMD is obtained through exercise. Most exercise intervention studies have shown that exercise programs are either ineffective or have only a small benefit in improving bone mineral density (BMD) in patients with osteoporosis. Physical activity has the potential to improve bone quality and reduce fracture risk by influencing each of these determinants. These findings have meaningful clinical implications because they highlight the fact that exercise interventions may benefit patients with osteoporosis by improving other determinants of bone strength, even if they do not lead to improvements in BMD (Hemmatian, et al, 2017).

5 CONCLUSION

This paper focuses on on the factors that influence bone fragility from a biomechanical perspective and suggests appropriate solutions to reduce the risk of fracture and osteoporosis due to bone fragility. It can be concluded that the natural aging of bones is a major factor among many others, and lack of vitamin D and external forces affecting bone density accelerate this process. Aging slows down the body's metabolism and thus affects the absorption of vitamin D, which is an important component involved in bone metabolism. Excessive physical work depletes the durability of the bones and the rate of bone metabolism does not keep up with the rate of depletion, leading to fractures. There are two ways to reduce the brittleness of bones and make them stronger. Firstly, effective distribution of bone mass can minimize the overuse of the bones. It is important not to overuse a particular bone, but to distribute the load on the bone appropriately. Secondly, improving the material properties of bone tissue can also make bone stronger at the tissue level, controlling diet and exercising sensibly to improve bone mass.

Here we have only discussed the general framework of factors influencing osteoporosis and fracture but we lack a quantitative analysis. As bone fragility is ultimately a biomechanical event, further research directions for this project should be based on quantitative biomechanical tests to achieve more intuitive data-based conclusions. The biomechanical effects of bone mass can be quantified by analysing the relationship between bone biomechanical properties and bone density, with biomechanical tests on bone at different physical scales (<1mm, 1mm, 1cm, etc.). Furthermore, data from analysis of the relevant literature suggest that changes in bone biomechanical properties with ageing, osteoporosis or drug treatment remain unclear. We propose to use the framework presented here, which represents the basic bone biomechanical principles and will provide new insights.

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