The relationship between bone mineral density, vitamin D and cortisol in healthy subjects of different ages

Introduction

Vitamin D is a prohormone, a substance that the body converts to a hormone, and skin makes vitamin D after exposure to sunlight. It is absorbed from certain foods, such as dairy products and certain oily fish, such as salmon, mackerel, and sardines. By binding to a protein called the Vitamin D receptor, has its effects and this receptor is found in nearly every cell in the body and affects many different body processes.

Mawer & Davies [1] in their study have pointed out that Vitamin D and cortisol are known to influence bone mineralization, and that both also play a role in body composition. A clear role is played by vitamin D in increasing bone mineral density (BMD), particularly through the regulation of calcium homeostasis.

According to Holick et al study 2009 [2], dietary vitamin D as well as vitamin D synthesized by skin tissues from sunlight are activated by conversion to vitamin D3, which then enters blood circulation and is either stored in adipose cells or travels to the liver to be hydroxylated to 25-hydroxyvitamin D (25(OH)D).

Rezumat:

Acest articol este o trecere în revistă a datelor din literatura de specialitate cu privire la aspectele particulare ale densității minerale osoase la bărbați și femei de vârste diferite. Sunt discutate mecanismele de acțiune ale vitaminei D și ale cortizolului în mineralizarea osoasă. Concluzii: 1. Vitamina D joacă un rol important în creșterea DMO, în particular prin reglarea homeostaziei Ca. 2. La fetele tinere nu există o relație semnificativă între măsurătorile DEXA ale DMO și nivelul serum de 25(OH)D. 3. La persoanele vârstnice de sexe diferite peste 65 de ani, nivelul serum de 25(OH)D s-a correlat negativ cu măsurătorile DEXA ale DMO, circumferința taliei, măsurarea stratului adipos. 4. La femeile între 42 și 61 de ani s-a observat o corelație inversă între nivelul coloanei lombare și DMO la nivelul coloanei lombare și scăderea DMO la nivelul coloanei lombare.

Cuvinte cheie: densitate minerală osoasă, vitamina D, cortizol

Abstract:

This article is a review of the literature data on the particular aspects of bone mass density in men and women of different ages. There are discussed the mechanisms of action of Vitamin D and Cortisol in bone mineralization. Conclusions: 1. A clear role is played by vitamin D in increasing bone mineral density (BMD), particularly through the regulation of calcium homeostasis. 2. In young females, there were no significant relationship between DEXA-measured BMD and serum 25(OH)D. 3. In healthy males and females aged 65 years and older, serum 25(OH)D was found to be significantly negatively correlated with BMI, waist circumference, and skin-fold caliper measurements. 4. In healthy women aged 42-61 years, it was found a significant inverse correlation between fasting serum cortisol and BMD of the lumbar spine, total femur, and femoral neck. 5. In healthy men and women aged 61-73 years, there was a statistically significant positive relationship between elevated serum cortisol and decrease in lumbar BMD.

Keywords: bone mineral density, vitamin D, Cortisol
D), the form indicative of vitamin D status. In the next stage, 25(OH)D travels to the kidneys to be hydroxylated to calcitriol, or 1,25-dihydroxyvitamin D (1,25(OH)2D), which works in the small intestine to regulate absorption of dietary calcium and at bone to influence bone-forming osteoblast and bone-degrading osteoclast activity and regulate uptake of calcium and phosphorus.

Arunabh et al [3] in their study have shown an inverse association between serum vitamin D and body fat. Lenders et al [4] in their study pointed out that even the mechanism by which vitamin D may influence body fat accumulation is not completely understood, in their in vitro studies have suggested that adipocyte production is inhibited by 1,25(OH)2D.

Kelly & Gimble’s [5] and Kong & Li [6] in their studies two using animal preadipocytes and Khimiphong et al [7] and Shi et al [8] studies using human preadipocytes have pointed out that 1,25(OH)2D had a significant negative effect on adipose cell differentiation.

According to Kelly & Gimble’s [5] opinion, it is considered that a common parent cell gives rise to both osteoblasts and preadipocytes and in the presence of 1,25(OH)2D, osteoblast production is increased and adipocyte production is inhibited. According to these findings, it has been suggested a direct relationship between the roles that vitamin D plays in body fat and bone density regulation.

Schwarz et al [9] study has pointed out that Cortisol, which is released by the hypothalamic-pituitary-adrenal axis, is increased during physical and psychological stress. Bedford & Barr [10] study emphasized a positive association between cortisol secretion and delayed or restricted eating patterns.

According to Schwartz et al [9] studies, this phenomenon could be attributed to the role of ghrelin in cortisol production although not fully understood.

According to Borer et al [11] study, Ghrelin, which is a hormone secreted during fasting, stimulates the release of adrenocorticotropic hormone from the pituitary gland, which signals the hypothalamic pituitary-adrenal axis to produce cortisol.

As Canalis et al [12] and Schwartz et al [9] pointed out in their studies, there are multiple mechanisms by which cortisol may act to lower BMD, such as an impairment of dietary calcium absorption in the small intestine, inhibition of calcium reabsorption at the renal tubules, stimulation of resorption of bone calcium, and inhibition of sex hormones in females.

Also, Canalis et al [12] in his in vitro studies suggested that cortisol acts to inhibit periosteal cell proliferation and cell differentiation of osteoblasts.

Even if the mechanism by which cortisol may influence body fat is not well understood, Mead et al [13] in his in vitro experiments had revealed that cortisol increases formation and activity of lipoprotein lipase (LPL), a hormone that aids in the catabolism of dietary triglycerides to one monoacylglycerol molecule and two free fatty acids. According to the same author [13], LPL are engaged in promoting the uptake of free fatty acids into cells, including adipocytes.

Dimtriou et al [14] in his study has shown that chronically elevated cortisol is associated with increased body weight and body fat, particularly of the abdomen.

Rodriguez et al [15] in their study have pointed out a lower BMD in older female while having sufficient dietary intake of calcium, vitamin D, and energy and adequate sun exposure are associated with higher BMD in athletes as well as in the general healthy population.

According to Nicolas et al [16] study, stress to bone in the form of weight-bearing physical activity increases bone density by upregulating osteoblast activity.

Porter et al [17] in his study pointed out that Overuse syndrome, characterized by repeated exertion of the same body part, attenuates the positive effects of physical activity on bone and represents a significant contributor to stress fracture risk in athletes.

According to Angeline et al [18] most studies of athletes confirm the positive influence of vitamin D on bone, but not all studies of vitamin D and bone health show a clear relationship. Kremer et al [19] study on 90 healthy females aged 16-22 years found no significant relationship between DEXA-measured BMD and serum 25(OH)D.

Vitamin D and Body Composition

Kremer et al [19] studies in non-athlete subjects has pointed out that increased adiposity has been associated with vitamin D insufficiency, but the relationship in athletes was not clear. His study on 90 healthy average females aged 16-22 years revealed a strong inverse correlation between serum 25(OH)D and body fat mass measured by DEXA.

In a study on 410 healthy women aged 20-80 years, Arunabh et al [3] also found a significant inverse correlation between serum 25(OH)D and BF% measured by DEXA. Snijder et al [20] in 453 healthy males and females aged 65 years and older, serum 25(OH)D was found to be significantly negatively correlated with BMI, waist circumference, and skin-fold caliper measurements.

Parikh et al [21] in his study performed on 302 healthy men and women aged 18-71 years, found that serum 25(OH)D was significantly negatively correlated with BMI and body fat measured by DEXA, and significantly lower serum 25(OH)D levels were observed in the 152 obese subjects compared to those of normal weight.

Lenders et al [4] study done on 58 obese adolescents aged 13-17 years revealed a significant negative correlation between serum 25(OH)D and fat mass measured by DEXA.

According to Canalis et al [12] study, the osteoporotic effects of glucocorticoid use are well established.

Most of the research on the interaction between endogenous cortisol and bone focuses on aging, non-athlete populations.

Van Schoor et al [22] has made an assessment of 502 older men and women, as part of the Longitudinal Ageing Study Amsterdam, revealed a significant negative association between serum fasting cortisol and DEXA measured BMD of the hip, femoral neck, trochanteric region, intertrochanteric region, and lumbar spine in women, but no relationship was seen in men.

Bedford & Barr [10] have made an assessment of 132 healthy, normal-weight women aged 19-35 years and found a significant inverse correlation between urinary cortisol and DEXA-
Dennison et al. [23] made an assessment of 34 healthy men aged 61-72 years and they revealed significant inverse correlations between serum cortisol and BMD of the lumbar spine and three of five femoral sites, as well as significant positive correlations between serum cortisol and rates of lumbar, femoral, and trochanteric bone loss over four years [Dennison et al., 1999].

Osella et al. [24] have made in their study an analysis of 82 healthy women aged 42-61 years reported a significant inverse correlation between fasting serum cortisol and BMD of the lumbar spine, total femur, and femoral neck.

Reynolds et al. [25] study on 247 healthy men and women aged 61-73 years observed a statistically significant positive relationship between elevated serum cortisol and decrease in lumbar BMD over four years in men, as well as significantly lower mean BMD of the femoral neck in women with elevated cortisol compared to women with normal cortisol levels.

### References:


