Relationships Between Serum Cortisol, Vitamin D, Bone Mineral Density, and Body Composition in National Team Figure Skaters

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CHAPTER I

Introduction

Figure skaters have relatively high rates of stress fractures and have also been shown to be at increased risk for certain nutrition-related issues, including food and energy restriction and suboptimal intakes of micronutrients (Porter et al., 2007). Figure skating requires competitors to have low body weight and to maintain a fit appearance, but is also physically demanding, requiring difficult spins and jumps that produce high impact to bones through jump landings (Dubravcic-Simunjak et al., 2003). The aesthetic demands of the sport predispose figure skaters to the same dietary concerns that may have negative effects on performance and bone health. Vitamin D and cortisol are known to influence bone mineralization, and recent research suggests that both also play a role in body composition.

Vitamin D plays a clear role in increasing bone mineral density (BMD), particularly through the regulation of calcium homeostasis (Mawer & Davies, 2001). Dietary vitamin D as well as vitamin D synthesized by skin tissues from sunlight are activated by conversion to vitamin D3, which 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), the form indicative of vitamin D status (Holick, 2009). Then 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.

Although there are few total studies in this area, research has consistently shown an inverse association between serum vitamin D and body fat (Arunabh et al., 2003; Kremer et al., 2009; Lenders et al., 2009; Parikh et al., 2004; Snijder et al., 2005). Although the mechanism by which vitamin D may influence body fat accumulation is not completely understood, in vitro studies have suggested that adipocyte production is inhibited by 1,25(OH)₂D. Four in vitro studies, two using animal preadipocytes and two using human preadipocytes, observed that 1,25(OH)₂D had a significant negative effect on adipose cell differentiation (Kelly & Gimble, 1998; Kong & Li, 2006; Nimitphong et al., 2012; Shi et al., 2001). It is believed that a common parent cell gives rise to both osteoblasts and preadipocytes (Kelly & Gimble, 1998; Vu et al., 1996). In the presence of 1,25(OH)₂D, osteoblast production is increased and adipocyte production is inhibited. These findings suggest that the roles vitamin D plays in body fat and bone density regulation are interrelated.

Cortisol, which is released by the hypothalamic-pituitary-adrenal axis, is increased during physical and psychological stress (Schwarz et al., 2011). Additionally, a positive association has been observed between cortisol secretion and delayed or restricted eating patterns; the majority of research was performed with female adult subjects (Anderson et al., 2002; Bedford & Barr, 2010; McLean et al., 2001; Puttermann & Linden, 2006; Rideout et al., 2006; Tomiyama et al., 2010). This phenomenon, although not fully
understood, has been attributed to the role of ghrelin in cortisol production (Schwarz et al., 2011). Ghrelin, 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 (Borer, 2003).

There are multiple mechanisms by which cortisol may act to lower BMD, including impairment of dietary calcium absorption in the small intestine, inhibition of calcium reabsorption at the renal tubules, stimulation of resorption of bone calcium, and, in females, inhibition of sex hormones (Canalis et al., 2007; Schwarz et al., 2011; Van Schoor et al., 2007). In vitro studies suggest that cortisol acts to inhibit periosteal cell proliferation and cell differentiation of osteoblasts (Canalis et al., 2007; Pereira et al., 2001).

The mechanism by which cortisol may influence body fat is not well understood. In vitro experiments have 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 (Mead et al., 2002). LPL works to promote the uptake of free fatty acids into cells, including adipocytes. Studies have shown that chronically elevated cortisol is associated with increased body weight and body fat, particularly of the abdomen (Dimitriou et al., 2003; Duclos et al., 2001; Purnell et al., 2004).
General Factors Associated with Bone Mineral Density

Nutritional inadequacies as well as non-nutritional factors are known determinants of BMD in both athletes and non-athlete populations. Being older and being female have been associated with lower BMD, 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 (American Dietetic Association, 2005; Rodriguez et al., 2009). Stress to bone in the form of weight-bearing physical activity increases bone density by upregulating osteoblast activity (Duncan et al., 2002; Etherington et al., 1996; Nichols et al., 2007). An assessment of 36 adolescent female figure skaters and 22 age-matched controls showed significantly higher calcaneus BMD in skaters than in controls. The same assessment revealed no significant differences in calcaneus BMD between the 10 skaters who had sustained heel fractures and the 26 who had not (Oleson et al., 2002). No equivalent studies have been performed in male skaters. Out of 211 singles skaters assessed at two international competitions, 19.8% of females and 13.2% of males sustained at least one stress fracture by the age of 18 (Dubravcic-Simunjak et al., 2003). The same assessment found that 100% of male and female skaters had previously suffered some injury from overuse. 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 (Dubravcic-Simunjak et al., 2003; Porter et al., 2007). Abnormal menstrual status poses a threat to BMD as well. Low estrogen causes an increase in bone calcium resorption in both males and females (Syed & Khosla, 2005). Although no studies have examined the effects of abnormal menstrual status on BMD and fracture risk in figure skaters, this
relationship is well documented in athletes of varied disciplines (Nichols et al., 2007; Redman & Loucks, 2005; Rencken et al., 1996).

**Vitamin D and Bone Mineral Density**

Vitamin D status is a known determinant of BMD in the general population (Mawer & Davies, 2001; Pekkinen et al., 2012) particularly through adolescence. In athletes, vitamin D may influence performance not only due to its role in bone metabolism; vitamin D also influences muscle function, prevention of acute and chronic illness, and body fat metabolism (Hamilton, 2011; Holick, 2009; Kremer et al., 2009; Lenders et al., 2009; Willis et al., 2008). Vitamin D status can be impacted by factors other than dietary intake of vitamin D. Increasing age and female gender are negatively associated with vitamin D status, as is having darker skin pigmentation (Angeline et al., 2013; Gennari, 2001). Lack of sunlight exposure caused by indoor training may predispose figure skaters to vitamin D insufficiency (Larson-Meyer & Willis, 2010). Most studies of athletes confirm the positive influence of vitamin D on bone (Angeline et al., 2013). However, not all studies of vitamin D and bone health show a clear relationship. An assessment of 18 male ballet dancers found no significant correlation between serum 25(OH)D and BMD, and no significant difference in fracture prevalence between dancers with sufficient and insufficient vitamin D status (Ducher et al., 2011). An assessment of 90 healthy females aged 16-22 years found no significant relationship between DEXA-measured BMD and serum 25(OH)D (Kremer et al., 2009). No studies have examined the relationship between vitamin D status and BMD in elite figure skaters.
**Vitamin D and Body Composition**

In non-athlete subjects, increased adiposity has been associated with vitamin D insufficiency, but the relationship in athletes is not clear. An assessment of 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 (Kremer et al., 2009). An assessment of 410 healthy women aged 20-80 years also found a significant inverse correlation between serum 25(OH)D and BF% measured by DEXA (Arunabh, Pollack, Yeh, & Aloia, 2003b). 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 (Snijder et al., 2005). In 302 healthy men and women aged 18-71 years, 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 (Parikh et al., 2004). An assessment of 58 obese adolescents aged 13-17 years revealed a significant negative correlation between serum 25(OH)D and fat mass measured by DEXA (Lenders et al., 2009). No studies have examined the relationship between vitamin D status and BF% in elite figure skaters.

**Cortisol and Bone Mineral Density**

The osteoporotic effects of glucocorticoid use are well established (Canalis et al., 2007; O’Brien et al., 2004). The majority of the research on the interaction between endogenous cortisol and bone focuses on aging, non-athlete populations. 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 (Van Schoor et al., 2007). An assessment of 132 healthy, normal-weight women aged 19-35 years found a significant inverse correlation between urinary cortisol and DEXA-measured total body and lumbar spine BMD values (Bedford & Barr, 2010). An assessment of 34 healthy men aged 61-72 years 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). 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 (Osella et al., 2012). A cohort study of 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 (Reynolds et al., 2005). There are no studies that examine the relationship between fasting serum cortisol and BMD in elite figure skaters.

**Cortisol and Body Composition**

The current understanding of the role of cortisol in athletes is mainly limited to its effects on bone. The interaction between cortisol and body fat has not been thoroughly investigated, especially in athletes. Most of the literature examines the relationship
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Duclos, M., Gatta, B., Corcuff, J.-B., Rashedi, M., Pehourcq, F., & Roger, P. (2001). Fat distribution in obese women is associated with subtle alterations of the


Vitamin D and Disease Prevention

Why is vitamin D important for health?

Vitamin D is a hot topic in the news, thanks to reports claiming its many health benefits. There is controversy, though, over the effects of vitamin D on health other than the bones. Doctors have long known that vitamin D helps the body absorb calcium and is vital for strong, healthy bones. In fact, a lack of vitamin D can contribute to weak bones in people who have osteoporosis. Severe vitamin D deficiency can cause rickets in children and osteomalacia ("soft" bones) in adults.

Yet, vitamin D is more than a vitamin. It really is a prohormone, a substance that the body converts to a hormone. The skin makes vitamin D after exposure to sunlight. We also absorb vitamin D from certain foods, such as dairy products and certain oily fish, such as salmon, mackerel, and sardines. Vitamin D has its effects by binding to a protein (called the vitamin D receptor). This receptor is present in nearly every cell in the body and affects many different body processes.

The problem is that many people do not make enough vitamin D by exposure to sunlight* or get enough through their diets. They may need supplements to raise their vitamin D level.

Health care providers can measure vitamin D in the body with a blood test. This screening test is for people at risk of vitamin D shortage.

What is the new thinking about vitamin D?

Some studies show that low blood levels of vitamin D may raise the risk of chronic (long-term) health problems, and that getting more vitamin D may lower those disease risks. Health problems that may be linked to low vitamin D include these, among many others:

- Some cancers
- Heart disease
- Diabetes (high blood sugar)
- Obesity
- Muscle weakness

However, it is not clear if a relationship between non-bone diseases and low vitamin D levels means that low vitamin D causes those diseases. It also is not clear if taking extra vitamin D can protect against chronic diseases. Therefore, The Endocrine Society asked a panel of experts to prepare a Scientific Statement that would assess scientific evidence about non-skeletal effects of vitamin D. The panel reviewed published studies for some of the health problems linked to low vitamin D. This fact sheet gives an overview of their conclusions, by type of health problem.

Does low vitamin D cause diabetes and obesity?

Studies show that people who have low vitamin D levels are more likely to be obese. However, they do not show that low vitamin D causes obesity. In fact, because vitamin D can become “trapped” in body fat, obesity may cause low vitamin D.

People with low vitamin D, studies show, are likelier to have type 2 diabetes, pre-diabetes, and the metabolic syndrome. (This syndrome is a cluster of risk factors that raise the chance of developing diabetes, heart disease, and stroke.) These diseases are even more likely to affect children and teens with low vitamin D than adults.

Yet, studies have not revealed whether the health problem (such as diabetes) or the low vitamin D came first. Also, most studies have found that vitamin D supplements had no effect on blood sugar. Because these diseases are more likely in people who are obese, it may be the obesity that contributes to vitamin D deficiency.

* You should limit your exposure to sunlight to reduce the risk of skin cancer. You should also know that sunscreens interfere with your body’s ability to make vitamin D.
Some studies have linked low vitamin D levels in the blood to a raised risk of cancer and dying of cancer (all types). Other studies have found that the higher the vitamin D intake from supplements, the lower the risk of certain types of cancer. These include

- Colorectal
- Breast
- Prostate

However, we do not yet know if vitamin D supplementation lowers the chance of getting cancer. To date, most studies have not had enough subjects or a long enough follow-up. Also, results have conflicted. Some studies show a benefit from vitamin D, and others find no benefit in reducing cancer risk.

Of concern is some studies suggest that high blood levels of vitamin D (40 ng/mL or above) may raise the risk of some cancers (cancer of the pancreas and esophagus).

Based on current scientific publications, the panel of experts could not answer the questions here with an absolute “yes” or “no.”

In reviewing published studies, the experts gave the most weight to the “gold standard” for most medical research: randomized clinical trials. In these human studies, researchers randomly assign subjects to one of two or more treatments, and they control for factors that could bias the results. Only randomized clinical trials can find a cause and effect. Observational studies, which simply observe what people are doing and what the outcomes are, often are larger than clinical trials. But, they can show only a relationship between a treatment (or prevention) and an outcome, not cause. For example, observational studies show that more educated people live longer, but that doesn’t mean a better education causes longer life. Instead, the opportunities that come with better education, such as jobs, health care, and income, may be more likely to lead to a longer life.
There were not enough randomized trials on this topic. More randomized studies in large numbers of people are needed before experts can conclude that vitamin D offers preventive and therapeutic benefits for a wide range of chronic non-bone diseases. At least one large randomized clinical trial has already begun to look at whether taking a high daily dose of vitamin D can lower the risk of cancer, heart disease, and stroke. Other studies are looking at whether vitamin D supplementation early in life can prevent type 1 diabetes in children.

**Should I take high-dose vitamin D to prevent chronic disease?**

The Endocrine Society has advised how much vitamin D people should get to protect their bone health. (See the Hormone Health Network’s Patient Guide to Vitamin D Deficiency.) It does not, however, recommend a high dose of vitamin D to try to prevent non-bone disease, improve quality of life, or extend life. Until more and better scientific data become available, talk to your doctor about whether to test your vitamin D level and how much vitamin D you need.

**Resources**

Hormone Health Network (www.hormone.org)
- Search under vitamin D

Endocrine Society (www.endocrine.org)
- The Nonskeletal Effects of Vitamin D: An Endocrine Society Scientific Statement