Vitamin D Supplementation Causes a Decrease in Blood Cholesterol in Professional Rowers

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(Received October 6, 2015)

Summary In the skin vitamin D3 is synthesized from cholesterol, which leaves the question whether a feedback mechanism controlling the level of blood cholesterol exists. Here we investigate the effects of vitamin D3 supplementation on serum lipids in professional rowers. The rowers were divided into two groups following the same training schedule for 4 wk: one received placebo (TP) while the second received 5,000 IU of vitamin D3 every day (TD3). Plasma total antioxidant status, total triglycerides (TG), total cholesterol (TC), high-density lipoprotein (HDL)-cholesterol (HDL-C), low-density lipoprotein (LDL)-cholesterol (LDL-C) and 25-hydroxyvitamin D (25-OH-D3) were determined in pre- and post-intervention. The ratios of TC/HDL-C and LDL-C/HDL-C were also calculated. Furthermore, maximal oxygen uptake was also measured at baseline. There were significant decreases over time in the TD3 group in TC 186 ± 18 vs 163 ± 21 (p < 0.05) and HDL-C; LDL-C also decreased, but the changes were not statistically significant. Moreover, the supplementation caused a significant rise in blood 25-OH-D3 (+98%). Neither training nor vitamin D3 supplementation had an effect on total antioxidant status. In conclusion, the alterations in the lipoprotein profile seen in this study would suggest that effects of regular exercise on lipoprotein profile may linked to vitamin D3 status.

Key Words HDL, LDL, training, cholesterol, rowers

As vitamin D3 is synthesized from 7-dehydrocholesterol, it is possible that our body compensates for vitamin D3 deficiency by increasing cholesterol synthesis. Based on the gathered data, we hypothesized that beneficial effects of exercise on the lipid profile may depend on the status of vitamin D.

Although chronic exercise is generally believed to improve the lipid profiles it is not clear whether this is due to exercise training or other determinants. There are some reports demonstrating that athletes do not differ from non-athletes in serum triacylglycerol (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL) or low-density lipoprotein cholesterol (LDL) concentrations if both groups are characterized by similar body fat (1). The effects of exercise on lipid profiles are equivocal. For example, 12 wk of high-intensity circuit training led to a decrease in TC, LDL and TG concentrations in overweight middle-aged subjects (2). In another study, insulin sensitivity and serum lipid levels were positively affected by exercise training in subjects with chronic heart failure. Interestingly, celiprolol, a vasodilating beta1-selective adrenoceptor antagonist, elevates this effect (3). This data indicates that the results of physical exercise on lipid profile can be modified by other factors. In particular, physical activity has been suggested to positively influence blood vitamin D (25-OH-D3). The reason for this is not clear but it may be due to the time spent outdoors and lower body fat (4). Vitamin D is closely associated with calcium metabolism, but it can also influence several processes by modulating the expression of 900 genes (5). It may also affect the lipid concentrations due to its effects on insulin sensitivity (6) and is associated with lower prevalence of metabolic syndrome (7). However, there are no data showing that vitamin D can improve insulin sensitivity in well-trained athletes as exercise has positive effects on insulin signaling too. Previous studies of the effects of supplemental vitamin D3 on circulating lipids produced inconsistent results. For example, in a large, randomized clinical trial, Rajpathak et al. reported <5% changes in levels of circulating lipids, including cholesterol and triglycerides, after daily supplementation of 1.0 g elemental calcium and 400 IU vitamin D3 for 5 y (8). However, only the combined effects of calcium and vitamin D were assessed in the above study and the dose of vitamin D was low. The study from Canadian Health Measures Survey showed that plasma 25-hydroxyvitamin D (25-OH-D) is inversely associated with triglycerides and total cholesterol (9). In a study performed on mice with

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hypercholesterolemia, an active form of 1,25(OH)2D3 has been shown to reduce the levels of plasma and liver cholesterol by increasing the presence of the hepatic protein Cyp7a1 (10).

To the best of our knowledge, there are no studies of the combined effects of sport training and vitamin D3 supplementation on lipid profile. Vitamin D deficiency is quite common among athletes living in northern latitudes (11–14). In this paper, the effects of vitamin D3 supplementation and 4 wk of training on lipid profile in young rowers were studied. All in all, we report that 4 wk of the training significantly decreased blood cholesterol and had no effect on triglyceride concentration, in rowers supplemented with vitamin D3.

**METHODS**

**Participants.** Sixteen elite, light-weight rowers took part in our experiment. They were divided into 2 groups. The TD3 group was supplemented with vitamin D3 (n=8), while TP was the placebo group (n=8). Two participants dropped out due to infections, one from TD3 and one from TP. Both groups followed the same training schedule and diet. The average training experience of the participants lasted 8 y. All participants completed the same training program during the preparation period. The 4-wk training included 45 units, all together around 60 h of effective exercise such as rowers’ ergometer (Concept II), running, swimming and strength training. None of the rowers took part in a regatta competition during the training program. All subjects were fully informed of any risks before giving their written agreement to participate in this experiment. The study was approved by the Ethical Committee of the Regional Medical Society in Gdansk, according to the Declaration of Helsinki.

**Blood sampling and 25-OH-D3 analysis.** Blood samples were taken from the antecubital vein into single-use containers with an anticoagulant (EDTA K2). After collection, the samples were immediately stored at a temperature of 4°C. Within 10 min, they were centrifuged in a temperate of 4˚C. Within 10 min, they were centrifuged at 3,000 g and 4°C for 10 min. Aliquots of the plasma were stored at −80°C. Red blood cells counts [10^6 /μL] (RBC), hematocrit [%] (Hct) and blood hemoglobin concentration (g/dL) (Hb), LDL, HDL and TC were determined from the venous blood samples by conventional methods using a Biosystems S.A, Analyzer A25 (Costa Brava, Barcelona, Spain). Plasma 25-OH-D3 concentration was performed by standard method of Architect-System (Abbot, Wiesbaden, Germany). Plasma total antioxidant status (TAS) was determined using a kit (TAS, Cat No. NX 2332; Randox Laboratories, London, UK) according to the manufacturer’s protocol.

**Aerobic power.** All of the subjects performed a continuous graded exercise test on a rowing ergometer (model-C, Concept2, Morrisville, VT) with a gas analyzer (Oxycon-Mobile, Erich Jaeger GmbH, Hoechberg, Germany) to determine the maximal oxygen uptake. The atmospheric conditions in the laboratory were temperature of 20°C, 56% humidity, and atmospheric pressure of 991 Hpa. The probe began with 3 min of rowing, with a load of 170 W. After this phase, the workload was systematically increased by 30 W every 3 min until exhaustion. The highest value of the oxygen uptake maintained for 15 s was considered to be the maximal oxygen uptake (VO2max) (15). Indicators such as HR, VO2, VCO2, and RER were obtained as described before (16).

**Vitamin D3 supplementation.** Each subject was given a vitamin D3 bottle (Vigantol Merck) and was asked to take 10 droplets per day (around 5,000 IU/d) in the morning for the 4 wk of the study. The placebo group received similar-looking bottles containing vegetable oil. The dispensation procedure was random and double-blind. Supplementation and testing were carried out in late winter, when natural levels of 25-OH-D3 were likely to be minimal. During the study, subjects were asked several times if they had taken the vitamins as instructed. All subjects reported that they had followed the instructions. It was confirmed by the measurements of 25-OH-D3 before and 4 wk after supplementation.

**Statistical analysis.** The results are expressed as mean values and standard deviations. The Shapiro–Wilk test was used to assess the homogeneity of dispersion from the normal distribution. The Levene test was used to verify the homogeneity of the variance. For homogenous results, an analysis of variance (ANOVA) for repeated measurements and post-hoc HSD Tukey test for equal sample sizes were performed to identify significantly different results. For heterogeneous results, an ANOVA Friedman’s test and right post-hoc test were applied. The significance level was set at p<0.05. The results were analyzed using Statistica 9.0 software.

**RESULTS**

The basic anthropometric characteristics of the subjects are summarized in Table 1. VO2max was measured at baseline to better characterize the subjects. In addi-
tion the effects of training on blood lipids were expected to depend on fitness level.

One of the main goals of this study was to evaluate the effects of professional rowers’ training and vitamin D₃ supplementation on lipid profile. First of all the 25-OH-D₃ was determined at the baseline to estimate if our athletes were vitamin D deficient. In most studies vitamin D deficiency is defined when serum concentration of 25-OH-D₃ is below 50 nmol/L. Among 14 athletes, a concentration lower than 50 nmol/L was observed in 12 of them. Accordingly, data presented in Table 2 demonstrates that, at the baseline, in both the control and vitamin D₃ supplemented groups, concentrations of 25-OH-D₃ were far below the deficiency threshold. This data indicated that almost all athletes were vitamin deficient in March when study was initiated. In addition, serum total cholesterol, triglycerides, LDL and HDL cholesterol were measured. No significant differences were found between the two groups at baseline. Four weeks of vitamin D₃ supplementation (5,000 IU/d) significantly raised serum 25-OH-D₃ to 89.1±21.9 nmol/L, while in the control group a nonsignificant increase was observed (Table 2). Four weeks of vitamin D₃ supplementation induced changes in blood lipid profile. A significant drop in total and HDL cholesterol was observed. Some decrease in LDL cholesterol was also noticed, but the changes were not significant (p=0.79). In the TP group no changes in lipid profile were observed (Table 2). In addition, neither training nor vitamin D₃ supplementation had an effect on total antioxidant status (TAS). Moreover, the increase of CRP was noticed in both groups and vitamin D₃ supplementation had no influence (Table 2).

**DISCUSSION**

For the first time the present study demonstrates that exercise, when accompanied by vitamin D supplementation, leads to a significant decrease in blood total cholesterol in young, well-trained men. Interestingly, a small drop in LDL and a significant one in HDL cholesterol were found, and non-significant changes in the HDL-to-LDL ratio after vitamin D supplementation were observed. Some previous studies are partially in agreement with our data, as it has been shown that plasma 25-OH-D was inversely associated with insulin, insulin resistance, triglycerides, total cholesterol, low-density lipoprotein cholesterol, and the ratio of total to high-density lipoprotein cholesterol (9). In addition serum 25-OH-D level was inversely correlated with the LDL-C/HDL-C ratio and TG values in Japanese men, independent of the visceral fat area and cardiorespiratory fitness (17).

On the other hand, our data demonstrated that vitamin D₃ supplementation had no effects on blood triglycerides. In addition, calcium and vitamin D₃ supplementation were shown to reduce blood triglycerides but did not change the level of blood cholesterol (18). These effects were not seen with vitamin D₃ alone, indicating that calcium is the main factor responsible for the observed changes (18). It is important to note that in the study mentioned above, low doses of vitamin D₃ had been used (800 IU per day) and no effect of the supplementation on blood 25-OH-D concentration had been measured. In our study, 5,000 IU of vitamin D₃ per day was used and we demonstrated that after 4 wk of supplementation, a significant rise in plasma 25-OH-D₃
occurred. A study performed on pregnant women with gestational diabetes mellitus supported this observation. In women who received two doses of vitamin D3 equal to 50,000 IU during a six-week period, a significant drop in LDL and total cholesterol without changes in HDL was observed. Meanwhile in the placebo group no changes in lipid profile over the period of the study were found (19). In a study of 185 men and 173 women, the levels of plasma apolipoprotein A-I, which is the main protein component of HDL, had a significant positive correlation with 25-OH-D3 levels in both sexes (r = 0.316 in men, r = 0.274 in women) (20). On the other hand, HDL cholesterol was positively correlated with 25-OH-D3 in women only (r = 0.264). The effect of 25-OH-D3 on HDL and apo A-I was independent of serum calcium levels. In addition, a low level of 25-OH-D3 has been associated with lower HDL cholesterol (21). Thus, in this project we hypothesized that rising blood 25-OH-D3 would be accompanied by an increase in HDL cholesterol; however this was not the case. Despite all of this data, it is not clear what the effect of vitamin D status on plasma lipid profile is.

Regular exercise has been associated with an improved lipid profile in young and old subjects (22). Moreover, athletes are characterized by lower total blood cholesterol and LDL, and higher HDL concentration compared to non-athletes (23). On the other hand, there are several studies, which have found that habitual physical activity and blood lipid concentrations show little association (24, 25). The study was performed on highly trained athletes during the preparation season who were characterized by normal lipid profiles. It has been suggested that the effect of exercise on LDL-C and TG can be greater when baseline values are above the clinically recommended normal range (26). We observed changes in lipid profile after training and vitamin D supplementation despite our subjects’ normal lipid profile at baseline. In conclusion, this is the first study which demonstrated that regular high intensity training, when accompanied by vitamin D supplementation, improved lipid profiles in young men. Possibly, some equivocal effects of training on lipid profiles can be resolved when the status of vitamin D is considered.

Limitations included no diet control, relatively small sample size and relatively short follow-up period.

Acknowledgments

This study was funded by a grant from the Polish Ministry of Science and Higher Education RSA205552. The authors declare that there are no conflicts of interest. We thank Derek Singh Gill for language assistance. ZJ, JK performed the experiment and manuscript preparation, KK, JA-data elaboration and manuscript preparation.

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