Effects of Jump Training on Bone and Calcium and Phosphorus Metabolism in Rats Fed a Phosphorus Enriched Diet

Guodong Wang, Akiko Honda, Yoshihisa Umemura

Laboratory for Exercise Physiology and Biomechanics, School of Health and Sport Sciences, Chukyo University, 101 Tokodachi, Kaizu-cho, Toyota 470-0393, Aichi, Japan

ABSTRACT

Wang GD, Honda A, Umemura Y. Effects of Jump Training on Bone and Calcium and Phosphorus Metabolism in Rats Fed a Phosphorus Enriched Diet. JEPonline 2016;19(5):123-135. This study examined the effects of high-impact and low-repetition jump training on the tibia bone and calcium and phosphorus-related factors in rats fed a phosphorus enriched diet. Forty-two male Wistar rats were assigned to four groups: normal diet sedentary control group; normal diet jump exercise group; high-phosphorus diet sedentary control group, and high-phosphorus diet jump exercise group. Rats in the two exercise groups underwent jump exercise for 2 wks. Data are presented as means ± SD. Two-way (diet x exercise) ANOVA and three-way ANOVA (diet x exercise x time points) were used. There was a significant positive exercise main effect on bone mineral content and bone mineral density of the tibia without any significant dietary main effect and interaction. Serum osteocalcin showed a significant exercise main effect and significant interaction. Serum inorganic phosphorus and fibroblast growth factor 23 were markedly increased by the high-phosphorus diet and suppressed by exercise. Serum total calcium was decreased by exercise. The results indicate that high-impact exercise has the potential not only to create local bone, but also to change calcium and phosphorus metabolism even under a high phosphorus diet.

Key Words: Jump Training, Bone Mass, High Phosphorus, Calcium and Phosphorus Metabolism
INTRODUCTION

In modern diet consumption habits, the intake of phosphorus is increasing according to the effects of processed foods and food additives, and is considered to have an impact on bone metabolism (5). In fact, Heaney (9) and Schürch and colleagues (22) indicate in their work that bone development is suppressed by excessive intake of phosphorus. It may be important to clarify whether exercise is effective for increasing the bone mineral content (BMC) under the modern nutritional status with increased intake of phosphorus. There have been several previous studies on this issue. In Bégot et al. (3), run training was proven to be effective for increasing or maintaining the BMC that has been changed by an unbalanced diet, such as a high-phosphorus diet or a low-calcium diet, although the BMC did not reach the normal level measured in the control groups. However, it is important to point out that a running movement was evaluated in their study, which provided the limbs with some elements of aerobic exercise as well as mechanical stress. In agreement, Ju et al. (16) demonstrated that local impact accompanied by muscle contraction during jumps plays an important role in enhancing trabecular bone mass compared with impact expressed as the ground reaction force.

In the previous studies, high-impact exercise has been shown to be very beneficial for increasing and maintaining BMC and bone mineral density (BMD) (1,10-12,27). It has also been recognized that high-impact exercise of appropriate intensity with low frequency stimulates mineralization, and therefore bone development (13,28). Umemura et al. (29) found that just five jumps per day increased bone mass and breaking force in rats, and that only 10 repetitions per day of jump exercise promoted an osteogenic response within a short time period from the onset of training. Jump training imposes great mechanical stress on the lower limbs with little aerobic or anaerobic exercise factors.

The effects of mechanical stress on bone are local, and the effects of jump training in rats mainly appear in the bones of the lower limbs. However, if deposition of bone calcium and phosphorus is significantly increased by adaptation of the local bone, this may possibly affect the metabolism of calcium and phosphorus in the whole body due to the increase of 1,25-(OH)_{2}D_{3} level by the stimulation of exercise (15). Recent studies have identified a new hormone-like substance, designated fibroblast growth factor 23 (FGF23) (8,17,21,30). The studies show that FGF23 is secreted from bone cells into the blood to reach the whole body. Furthermore, it primarily acts on the kidney to promote excretion of phosphorus by inhibiting the reabsorption of phosphorus while suppressing the activation of vitamin D. Therefore, it is reasonable to conclude that the whole-body metabolism of calcium and phosphorus is adjusted proactively by bone cells. We also considered that mechanical stress may induce changes in metabolism of calcium and phosphorus by stimulating bone cells.

Thus, the purpose of this study was to examine the effects of mechanical stress generated by high-impact and low-repetition jump training on the properties of the tibia bone in rats fed a phosphorus-enriched diet. This study also focused on the effects of mechanical stress on the metabolism of calcium and phosphorus. For these purposes, we measured bone mineral content, bone mineral density, bone strength, stiffness, and the blood parameters of serum concentrations of FGF23, inorganic phosphorus, total calcium, 1,25-(OH)_{2}D_{3}, parathyroid hormone (PTH), and osteocalcin. We hypothesized that jump exercise would change the metabolism of calcium and phosphorus in rats when compared to the rats in the control
groups. In addition, BMC, BMD, and bone strength would increase even under the condition of a high-phosphorus diet.

METHODS

Subjects
Forty-two male Wistar rats aged 8 wks were purchased from Japan SLC. The rats were housed individually in standard cages under constant temperature (23 ± 1°C) and 12-h/12-h light/dark cycle. The rats were fed a high-phosphorus diet (P/Ca = 2.0) or normal diet (P/Ca = 1.0), with the diet and water provided ad libitum for 1 wk before starting the experiment. As the basic feed, CE-2 powdered food was purchased from CLEA Japan Inc. The high-phosphorus diet (P/Ca = 2.0) was formulated by adding dehydrated sodium dihydrogen phosphate and dextrin to the basic feed (Table 1). To make the sodium content consistent with the high-phosphorus diet, sodium chloride was added to the basic feed, in addition to a small amount of dextrin, and the P/Ca ratio was set at 1.0 in the normal diet (Table 1). The study was conducted with approval from the Chukyo University Animal Care and Use Committee.

Table 1. Mineral Components of the Basic Feed (CE-2) and Additives to 100 g of the Basic Feed.

<table>
<thead>
<tr>
<th>Minerals</th>
<th>Normal Diet (P/Ca = 1.0)</th>
<th>High-Phosphorus Diet (P/Ca = 2.0)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic Feed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ca (g)</td>
<td>1.06</td>
<td>1.06</td>
</tr>
<tr>
<td>P (g)</td>
<td>0.99</td>
<td>0.99</td>
</tr>
<tr>
<td>Mg (g)</td>
<td>0.34</td>
<td>0.34</td>
</tr>
<tr>
<td>K (g)</td>
<td>1.02</td>
<td>1.02</td>
</tr>
<tr>
<td>Mn (mg)</td>
<td>9.96</td>
<td>9.96</td>
</tr>
<tr>
<td>Fe (mg)</td>
<td>30.64</td>
<td>30.64</td>
</tr>
<tr>
<td>Cu (mg)</td>
<td>0.71</td>
<td>0.71</td>
</tr>
<tr>
<td>Zn (mg)</td>
<td>5.45</td>
<td>5.45</td>
</tr>
<tr>
<td>Na (g)</td>
<td>0.31</td>
<td>0.31</td>
</tr>
<tr>
<td>Additives to Basic Feed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NaH₂PO₄.2H₂O (g)</td>
<td>0.35</td>
<td>5.38</td>
</tr>
<tr>
<td>NaCl (g)</td>
<td>1.89</td>
<td>0</td>
</tr>
<tr>
<td>Dextrin (g)</td>
<td>3.76</td>
<td>0.62</td>
</tr>
</tbody>
</table>

Procedures
At the beginning of the preliminary feeding period, all rats were trained to jump from the bottom of a wooden box (height = 40 cm) surrounded by a four-way plate, to catch the top of the box with their forelimb, and to climb up to the top edge. For the initial training, an
electrical stimulation device (SEN-3301; Nihon Kohden) was installed at the bottom of the box to make the rats jump in response to electrical stimulation. Once the rats had become accustomed to the training after 1 to 2 days, electrical stimulation was not used thereafter. Subsequently, the rats in the two dietary groups were further assigned to a normal diet sedentary control group (NC; N = 10), normal diet jump exercise group (NE; N = 10), high-phosphorus diet sedentary control group (HC; N = 11), and high-phosphorus diet jump exercise group (HE; N = 11). After the 1-wk preliminary feeding period, the rats in the two exercise groups underwent 2 wks of jump exercise at the age of 9 wks (height = 40 cm; 20 jumps per day; 5 d·wk⁻¹). The rats in the sedentary groups were allowed to move freely in their cages for 2 wks.

Body weight was measured weekly using an electronic balance (GX-4000; Kensei Industry). Tail artery blood samples were collected for measurement of FGF23 concentrations before the jump exercise and after 1 wk of jump exercise. After 2 wks of jump exercise, the rats were exsanguinated under isoflurane anesthesia. The blood samples were centrifuged and the serum samples were frozen. Thereafter, the concentrations of osteocalcin, FGF23, PTH, 1,25-(OH)₂D₃, total calcium, and inorganic phosphorus were measured using the frozen serum samples.

Inorganic phosphorus was measured by the direct molybdate method (Inorganic phosphorus HR-II; Wako Pure Chemical Industries, Ltd.), and total calcium was measured by the orthocresolphthalein complexone method (Serotec Ca-AL; Serotec Company). 1,25-(OH)₂D₃ was measured by a two-antibody method according to a radioimmunossay (1,25(OH)₂D RIA Kit [FR]SRL; Immunodiagnostic Systems Limited). FGF23 was measured using an enzyme-linked immunosorbent assay (FGF23 ELISA Kit; Kainos Laboratories Inc).

The left tibia was removed and its soft tissue was carefully peeled to avoid damage to the bone. The length of the left tibia was measured with a Vernier caliper, and the BMD and BMC were measured by dual-energy X-ray absorptiometry (DSC-600EX; Aloka). In the latter measurements, X-rays were irradiated in the direction of the outer surface of the tibia corresponding to the outside of the hind limb (lateral side) of the rat. Using a bone strength-testing machine (TK-252C/RDT; Muromachi Kikai), the tibia was subjected to three-point support fracture experiments to measure the bone strength. The distance between the fulcrum of both ends was set at 16 mm, and a plunger was displaced at a speed of 10 mm·min⁻¹ in the neutral position from the top to break the test bone. The break energy was measured as the greatest bending load to cause breakage by the plunger. For the breakage, the plunger was set at the mid-shaft of each bone. In addition, the stiffness was calculated by the relationship between the power that needed to be applied to the bone and the distortion of the bone.

**Statistical Analyses**

The data are presented as means ± SD. Two-way (diet x exercise) ANOVA and three-way ANOVA (diet x exercise x time points) were used to examine the individual main effects and interaction between the factors. The Tukey–HSD test was used to examine the significance of differences among the groups. All statistical analyses were performed with SPSS 17.0 for Windows (SPSS Inc). The significance level of P<0.05 was used.
RESULTS

All rats completed the treatments without injury, soreness, or illness as determined by the research personnel. The final body weights were increased in all four groups after the 2-wk period with and without jump exercise, but did not differ significantly among the groups (Table 2). The interaction and main effects of diet and jump training were not significant at any age.

Effects on Bone Properties
For BMC, there was a main effect of jump training after just 2 wks, but the diet did not (P=0.854), and there was no interaction between jump training and diet (P=0.082). A significant difference was observed between the sedentary and jump training groups for the high-phosphorus diet, with no significant difference (P=0.283) between the corresponding groups for the normal diet (Figure 1).

Figure 1. Effects of Jump Training and High-Phosphorus Diet on the BMC of the Tibia Evaluated by Dual X-Ray Absorptiometry. NC = normal diet sedentary control group (N = 10); NE = normal diet jump exercise group (N = 10); HC = high-phosphorus diet sedentary control group (N = 11); HE = high-phosphorus diet jump exercise group (N = 11). ANOVA reveals significant main effects of exercise (**P<0.000)

For BMD, after only 2 wks there was a main effect of jump training, but the diet did not (P=0.934), and there was no interaction between jump training and high-phosphorus diet (P=0.476). Similarly, a significant difference was observed between the sedentary and jump training groups for the high-phosphorus diet, but no significant difference was found for the corresponding groups for the normal diet (Figure 2).
Figure 2. Effects of Jump Training and High-Phosphorus Diet on the BMD of the Tibia Evaluated by Dual X-Ray Absorptiometry. NC = normal diet sedentary control group (N = 10); NE = normal diet jump exercise group (N = 10); HC = high-phosphorus diet sedentary control group (n = 11); HE = high-phosphorus diet jump exercise group (N = 11). ANOVA reveals significant main effects of exercise (**P = 0.002).

Regarding bone length, after the 2-wk experiment, there were no main effects of diet and jump training, and no interaction between high-phosphorus diet and jump training (Table 2). For maximum load, after 2 wks of the high-phosphorus diet and jump training, there were no main effects of diet and jump training, and no interaction between high-phosphorus diet and jump training (Table 2).

Table 2. Body Weight, Bone Lengths and Maximum Loads after Two Weeks.

<table>
<thead>
<tr>
<th>Group</th>
<th>NC (N = 10)</th>
<th>NE (N = 10)</th>
<th>HC (N = 11)</th>
<th>HE (N = 11)</th>
<th>Main Effect</th>
<th>Exercise</th>
<th>Feed</th>
<th>Inter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body w</td>
<td>352 ± 18</td>
<td>338 ± 10</td>
<td>339 ± 18</td>
<td>338 ± 11</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Bone l</td>
<td>41.2 ± 1.5</td>
<td>40.8 ± 0.3</td>
<td>40.9 ± 0.5</td>
<td>41.2 ± 0.7</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Max l</td>
<td>79.8 ± 6.9</td>
<td>83.0 ± 4.2</td>
<td>80.9 ± 5.7</td>
<td>84.4 ± 8.9</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Body w = Body weight (g); Bone l = Bone length (mm); Max l = Max load (N/mm). NC = normal diet sedentary control group; NE = normal diet jump exercise group; HC = high-phosphorus diet sedentary control group; HE = high-phosphorus diet jump exercise group; NS = not significant; Inter = interaction Data represent means ± SD.

Effects on Blood Parameters
For FGF23, we performed a three-way ANOVA (exercise x feed x time point) followed by a two-way ANOVA for specific time points (1 wk and 2 wks). There was no interaction observed.
between jump training and high-phosphorus diet, but the main effects of jump training and high-phosphorus diet were significant. In the high-phosphorus diet groups, the FGF23 concentration was significantly increased at 1 wk after the start of the experiment. Although the concentration of FGF23 was elevated after 2 wks, it was significantly repressed by jump training (Figure 3).

For osteocalcin, there was a main effect of jump training and an interaction after just 2 wks, but there was no main effect of diet (P=0.094). The normal diet caused no changes in the concentration of osteocalcin. Meanwhile, the concentration of osteocalcin was low in the high-phosphorus diet group but elevated by jump training, and a significant difference was only observed between the two groups on the high-phosphorus diet (Table 3).

Regarding serum inorganic phosphorus, the interaction of jump training and high-phosphorus diet was not significant, but the main effects of jump training and high-phosphorus diet were significant, in that jump training reduced the concentration of serum inorganic phosphorus (Table 3). For serum total calcium, only the main effect of training was significant, with the serum concentration of total calcium being decreased by training, and the main effect of diet and interaction were not significant (Table 3).

For 1,25-(OH)$_2$D$_3$, an interaction of jump training and high-phosphorus diet and a main effect of jump training were not observed, but the main effect of high-phosphorus diet was significant. Although there was no significant difference between the groups on the normal diet and high-phosphorus diet, the concentrations of 1,25-(OH)$_2$D$_3$ in the two groups on the high-phosphorus diet were higher than those in the two groups on the normal diet (Table 3).
Regarding PTH, main effects of jump training and high-phosphorus diet were not observed, and there was no interaction between jump training and high-phosphorus diet (Table 3).

Table 3. Serum Levels of Osteocalcin, Calcium, Inorganic Phosphorus, 1,25-(OH)\(_2\)D\(_3\) and PTH after Two Weeks.

<table>
<thead>
<tr>
<th>Group</th>
<th>NC (N = 10)</th>
<th>NE (N = 10)</th>
<th>HC (N = 11)</th>
<th>HE (N = 11)</th>
<th>Main Effect</th>
<th>Feed</th>
<th>Inter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oste</td>
<td>.65 ± .13</td>
<td>.67 ± .08</td>
<td>.52 ± .10</td>
<td>.68 ± .09†</td>
<td>P=0.007</td>
<td>NS</td>
<td>P=0.029</td>
</tr>
<tr>
<td>Cal</td>
<td>9.90 ± 0.59</td>
<td>9.27 ± .52</td>
<td>9.95 ± .98</td>
<td>9.45 ± .28</td>
<td>P=0.008</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Inor p</td>
<td>8.94 ± 1.49</td>
<td>7.74 ± .77</td>
<td>10.25 ± 2.10</td>
<td>8.90 ± .95</td>
<td>P=0.007</td>
<td>P=0.008</td>
<td>NS</td>
</tr>
<tr>
<td>1,25-D</td>
<td>174.4 ± 36.4</td>
<td>182.6 ± 38.4</td>
<td>201.5 ± 39.7</td>
<td>211.4 ± 37.0</td>
<td>NS</td>
<td>P=0.022</td>
<td>NS</td>
</tr>
<tr>
<td>PTH</td>
<td>16.83 ± 15.04</td>
<td>7.36 ± 4.75</td>
<td>55.32 ± 72.8</td>
<td>26.60 ± 25.44</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Oste = Osteocalcin (pg/ml); Cal = Calcium (mg/ml); Inor p = Inorganic phosphorus (mg/ml); 1,25-D = 1,25-(OH)\(_2\)D\(_3\) (pg/ml); PTH (pg/ml). NC = normal diet sedentary control group; NE = normal diet jump exercise group; HC = high-phosphorus diet sedentary control group; HE = high-phosphorus diet jump exercise group; NS = not significant; Inter = interaction. ANOVA reveals significant main effects of exercise and diet and interaction of exercise and feed (†significant difference (P = 0.004) compared with HC group by the Tukey–HSD test)

**DISCUSSION**

In this study, 8-wk-old rats (jump exercise start at the age of 9 wks) were used for comparisons of a normal diet (P/Ca = 1.0) and high-phosphorus diet (P/Ca = 2.0), and the effects of 2 wks of jump training on bone and calcium and phosphorus metabolism. In order to investigate the adaptive changes of bone properties and parameters in blood, we chose the 2-wk period of the experiment. As a result, even under the high-phosphorus diet, just 2 wks jump training increased the BMC and BMD of the lower limbs as we hypothesized, although we did not find significant differences in the bone strength of the tibia. Also, we found that the influence of the high-phosphorus diet with P/Ca = 2.0 did not affect the body weight. In addition, because serum phosphorus was increased by the high-phosphorus diet, FGF23 was increased. However, FGF23 was decreased by jump training to some extent.

In previous studies (16,20,29), jump training was demonstrated to be effective for increasing BMC, bone morphology, and bone strength. Furthermore, in the study by Bégot et al. (3) the combination of run training with an unbalanced diet (high-phosphorus and/or low-calcium diet) was found to be effective for improving the microscopic bone structure of the tibia and increasing its BMC. In the present study, we investigated the effects of a combination of jump training and high-phosphorus diet on bone in rats. Compared with running exercise, the difference was that jump exercise has less aerobic exercise factors. In addition, given the 20 jumps a day, the jump training can be characterized as having less impact on the metabolism of the whole body and yet, it has the ability to increase BMC and bone strength even under a high-phosphorus diet without a body weight change because the number of training times was lower compared with other training periods.
This study found that the effects of jump training on bone were markedly observed by the significant increases in BMC and BMD even under the high-phosphorus diet without significant weight changes in rats by carrying out 2 wks of jump training with only 20 jumps per day, compared with rats under a normal diet. In a previous study (12), about 19.2% of the BMC of the tibia was increased by jump training compared with the control group, while increases of about 4.5% (normal diet) and 10.8% (high-phosphorus diet) were observed in the present study. These changes were lower than that in the previous 8-wk study, which is likely due to the shorter experimental period.

Regarding the maximum load, an interaction and main effects of high-phosphorus diet and jump training were not seen. From the viewpoint of the bone parameters, the BMC seems to be very susceptible to jump training than the bone length and bone strength. In addition, we considered that the properties of bone have a process of adaptation when subjected to external forces and that the period of 2 wks may not allow for the adaptation of bone strength. If the experiment is longer, such as 3 wks or 4 wks, adaptive changes in bone strength is more likely to occur. Also, another consideration is the negative effects of high phosphorus diets and the positive effects of jump training on bone strength that offset each other.

Osteocalcin is a protein that accounts for 25% of the non-collagenous proteins in bone. It is considered to contribute to the maintenance of homeostasis of mineral formation and calcium ions, and is also a negative regulator of bone formation (7,19). Because osteocalcin is produced by osteoblasts during the mineralization period, it is often used as a marker for the bone formation process. It has been observed that higher serum osteocalcin levels are relatively well-correlated with increases in BMD during treatment with anabolic bone formation drugs for osteoporosis, such as teriparatide (4). Hence, osteocalcin has been used as a preliminary biomarker for the effectiveness of a given drug on bone formation. For example, with the aim of studying the effectiveness of a glycoprotein called lactoferrin on bone formation, osteocalcin was used as a measure of osteoblast activity (4). In the present study, when comparing the serum osteocalcin levels with their respective control groups, there was no significant difference for the normal diet, but there was a significant difference for the high-phosphorus diet. Thus, there may be a better effect of exercise on bone formation with a high-phosphorus diet than with a normal diet.

In this study, the serum phosphorus levels were significantly increased by the high-phosphorus diet, but significantly decreased when the rats were subjected to jump training. It was inferred that the jump training inhibited bone resorption and contributed to the decrease in the serum phosphorus level. The jump training also significantly reduced the level of serum calcium, but this was not affected by the high-phosphorus diet. It is likely that the decreases in serum phosphorus and calcium induced by the jump training were caused by deposition in the bone during the process of bone formation.

Although there was no significant difference (P=0.065) in PTH, its concentration was increased by the high-phosphorus diet compared with the normal diet. Because of the high serum PTH, the synthesis of 1,25-(OH)_{2}D_{3} might have been promoted for bone formation, leading an increase in serum 1,25-(OH)_{2}D_{3} (6,14). There was no direct impact of the jump training on the serum concentrations of PTH and 1,25-(OH)_{2}D_{3}.
Regarding serum FGF23, from the results of the analysis, the concentration of FGF23 was significantly increased at 1 wk after the start of the experiment under the high-phosphorus diet, but was not changed under the normal diet. This is thought to be caused by the raised concentration of serum phosphorus under the high-phosphorus diet (2,26). However, the jump training had no significant influence on FGF23 at this time point. At 2 wks, the serum FGF23 level was markedly elevated compared with that at 1 wk under the high-phosphorus diet. But, the level was significantly restricted by jump training in both the high-phosphorus diet and normal diet groups compared with their respective sedentary groups. Accordingly, the low serum FGF23 level at 2 wks was caused by the jump training that acts directly on the bone cells and is thought to inhibit the secretion of FGF23. There is also a possibility that the concentration of FGF23 was reduced by the decline in serum phosphorus caused by the jump training.

Limitations of the Study

We are aware of several limitations to this study. First, we used young growing rats (9 wks of age) in this experimental design because it was reported that young animals usually have a greater potential for bone recovery after immobilization (24). Furthermore, other experimental studies (13,29) have shown that the bones of growing rats exhibited a better response to high-impact exercise. Therefore, the results might have been different if adult rats had been used. Second, we only looked at bone formation marker data and did not determine bone resorption marker data, which might have reflected the result that the exercise had a better effect on bone formation under the high-phosphorus diet than under the normal diet. Finally, although it was shown that even in the high-phosphorus state jump training increased BMC after only 2 wks of jumps at 20 times per day, we do not clearly know why there was no increase in the maximum load of the tibia. In previous studies (18,23,25), micro-CT analysis was used to clarify the bone responses to different exercise patterns or different magnitudes of load based on the bone microstructure. Thus, it is believed that we might have clarified the reason why the maximum load was not enhanced by jump training if three-dimensional micro-CT analysis had been used simultaneously, or it may be necessary to carry out additional studies using three-dimensional micro-CT analysis.

CONCLUSIONS

The findings from this study indicate that it is possible to increase the BMC and BMD of the tibia by just 2 wks of jump training even under a high-phosphorus diet (P/Ca = 2.0). Although the serum phosphorus level was significantly increased by the high-phosphorus diet, it was significantly reduced by the jump training. The serum calcium level was significantly reduced by the jump training, but was not affected by the high-phosphorus diet. The serum FGF23 level increased because of the high serum phosphorus level, but declined when subjected to the jump training.
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Address for correspondence: Guodong Wang, Laboratory for Exercise Physiology and Biomechanics, School of Health and Sport Sciences, Chukyo University, 101 Tokodachi, Kaizu-cho, Toyota Zip Code: 470-0393, Aichi, Japan. Email: wgdmyt@hotmail.com

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