Effects of exhaustive high-intensity intermittent exercise on serum parathyroid hormone

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Abstract
Increased serum parathyroid hormone (PTH) during moderate-intensity exercise has been reported, suggesting that such exercise may stimulate bone resorption. This study was undertaken to observe the effects of exhausting high-intensity intermittent exercise (HIIE) on serum PTH and on blood parameters that may affect PTH secretion during exercise. Seven young trained adults exercised on 2 days after overnight fasting. On the HIIE day, they performed 6–7 exhausting bouts of 20-sec bicycle exercise (intensity, 170% VO2max) with intervening 10-sec rests. On the moderate-intensity exercise (MIE) day, the subjects biked for 60 min at 70% VO2max. The peak lactate concentration in blood after the HIIE was 15.2 ± 1.3 mmol/l. The blood lactate concentration at the end of the MIE was 2.2 ± 0.9 mmol/l. The HIIE significantly reduced the serum PTH (Pre: 30 ± 5 pg/ml, 10 min post-HIIE: 22 ± 4 pg/ml, p < 0.05), whereas the MIE significantly elevated the serum PTH. The HIIE induced a significant increase in serum ionized Ca (iCa); but MIE did not affect iCa. The serum cortisol concentration post-MIE was significantly higher than that observed pre-exercise; no changes from the pre-exercise value were noted post-HIIE. The serum phosphate concentration immediately post-HIIE increased significantly to the same level as that post-MIE. No changes in serum C-terminal telopeptide of Type I collagen (a marker of bone resorption) was observed after the HIIE or MIE. Although these results do not identify stimulator(s) for PTH secretion during HIIE and MIE, they indicate that HIIE does not induce an exercise-induced increase in PTH (which might deteriorate bone metabolism).

Keywords: bicycle exercise, 170% VO2max, CTX, cortisol, phosphate, Tabata training

Introduction
It has been reported that during moderate-intensity prolonged exercise, the serum level of parathyroid hormone (PTH) is elevated1–3. Since PTH acutely stimulates the resorption of Ca from bone, repeated disruptions of Ca homeostasis during this type of exercise may contribute to bone loss. For athletes such as long-distance runners who intend to improve their performance by increasing their aerobic power with running training, the training can be beneficial for improving their aerobic fitness, but could be hazardous for their bone health. In fact, male competitive cyclists were reported to reduce bone density after 1 year of intensive training and competition3). Regarding the change in serum PTH during exercise, Scott et al.4 reported that 1 hr of running at 75% of the maximal oxygen uptake (VO2max) elevated the serum PTH concentration, whereas no increase was observed after 60 min at 55% or 65% VO2max. This suggests that there may be an exercise-intensity threshold that stimulates the secretion of PTH. Interestingly, Cunningham et al. reported that after young adults engaged in a running exercise that exhausted them within 60–130 sec, their serum PTH concentrations were not significantly changed5–6. The exercise intensity that exhausts subjects within 60–130 sec is speculated to correspond to a supramaximal intensity (160%–120% VO2max, respectively).

These findings led us to hypothesize that supramaximal exercise may not induce an increase in serum PTH. In addition, we reported that training using such high-intensity (170% VO2max) intermittent exercise (HIIE) improved the VO2max to a degree that was comparable to that observed after conventional endurance training adopting 1 hr exercise at 70% VO2max7. If this hypothesis is correct, high-intensity exercise may be as effective as conventional moderate-intensity exercise for increasing cardiovascular fitness, but with less disruption of bone metabolism, thereby potentially having a more favorable effect on bone health. We thus conducted the present study to compare changes in the serum PTH concentration after a high-
intensity intermittent exercise (for details, see Appendix) and moderate-intensity prolonged exercise (MIE), which has been conventionally recommended as a tool to elevate aerobic power.

Subjects and Methods
The protocols for the experiments were approved by the Ethics Committee of Ritsumeikan University (BKC 2015-001), and the experiments were conducted in accord with the Declaration of Helsinki. All subjects were given an oral and written briefing of the study, and each of the subjects provided written informed consent to participate.

All bicycle experiments and pretests were conducted on a mechanically braked cycle ergometer (Monark, Stockholm, Sweden) at 90 repetitions per minute (rpm).

Subjects
Seven trained young men (age 21 ± 1 yr) who belonged to the varsity triathlon team volunteered to participate in the study after they provided written informed consent. Each subject passed a comprehensive physical examination. The subjects’ height, weight, and VO2max measured during a cycle ergometer exercise were 169.6 ± 6.7 cm, 61.1 ± 4.6 kg, and 58.3 ± 3.7 ml/kg/min, respectively.

Pretest
Since the exercise intensity used in the present investigation is expressed relative to the VO2max, that value was established during screening tests of the subjects conducted over 3–5 days, as follows. First, to determine a linear relationship between the submaximal intensity of cycling (watts) and the steady-state oxygen uptake (L min⁻¹) for each individual, the oxygen uptake was measured during the last 2 min of 6–9 different 10-min bouts of cycling at a constant power between 35% and 90% of the estimated VO2max. Next, to determine the VO2max, the oxygen uptake was measured during the last two or three 30-sec intervals during several bouts of supramaximal-intensity exercises that exhausted the subjects within 2–4 min. The criterion for exhaustion was that the subject was unable to maintain the pedaling frequency at or above 85 rpm near the end of the bout. After confirming a leveling-off in the oxygen uptake despite an increase in the exercise intensity, the highest oxygen uptake (L min⁻¹) measured during the 30-sec interval was taken as the subject’s VO2max⁷¹⁰.

To determine 170% VO2max and the corresponding cycling exercise intensity, a linear extrapolation was carried out using the established relationship between the power and steady-state oxygen uptake described above. Oxygen demand (L min⁻¹) of the 170% VO2max was taken as 1.70 times the VO2max (L min⁻¹), and the corresponding cycling power was determined from the linear relationship⁸⁻¹⁰.

Exercise test
High-intensity intermittent exercise (HIIE)
After an overnight fast, the subjects arrived at the laboratory at approx. 8:00 a.m. After the subjects rested for 20 min, blood samples were taken, followed by 10 min of warm-up cycling exercise at 50% VO2max. Ten min after the conclusion of the warm-up exercise, the subjects conducted the exhausting HIIE consisting of 6–7 sets of high-intensity exhausting exercise with a 10-sec rest between the exercise bouts (HIIE). The exercise intensity for HIIE was 170% of the subject’s VO2max. The work rate of the HIIE for the bicycle ergometer was 384 ± 55 watt (mean ± SD). The criterion for exhaustion was identical to that described above. Our earlier investigation demonstrated that training using this HIIE maximally improves both the aerobic and anaerobic energy-releasing systems simultaneously⁷¹¹.

Moderate-intensity exercise (MIE)
After reporting to the laboratory at approx. 8:00 a.m. after an overnight fast, the subjects rested for 30 min and then started exercise for 60 min on a cycle ergometer at 70% VO2max. The work rate of the MIE for the bicycle ergometer was 170 ± 22 watts (mean ± SD).

The order of the HIIE and MIE exercise tests was randomized, and the tests were conducted ≥1 week apart.

Blood sampling and analysis
For the HIIE test, blood was collected from each subject’s antecubital vein immediately before (Pre1) and 10 min after the warm-up exercise (Pre2) and at 0, 10, 20, 30, 60, and 90 min after the completion of the HIIE. In addition, for determining the peak blood lactate concentration after HIIE, blood was sampled from the subject’s fingertip immediately and 3, 6, 9 min after the HIIE. The highest value was reported as the peak lactate concentration after the HIIE for each subject.

For MIE test, intravenous blood samples from the subjects were taken before (Pre1), 30 min after the start of MIE (MIE30), and at 0, 10, 20, 30, 60, and 90 min after the MIE. Only the blood lactate value measured from intravenously sampled blood immediately after the end of the MIE is reported as representative of the lactate concentration after the MIE.

The blood iCa value and hematocrit (Hct) were measured immediately after the collection of blood samples, using a cartridge-based whole blood analyzer (iSTAT; Abbott, East Windsor, NJ, USA). The blood lactate concentration was measured using a Lactate Pro 2 test meter (LT-1730; Arkray, Kyoto, Japan) immediately after the blood sampling. The serum levels of intact PTH and cortisol were analyzed in duplicate by an electro-chemiluminescence immunoassay (ECLIA) method. The serum phosphate concentration was measured using a Quantichrom Phosphate Assay Kit (DIPI-500; BioAssay Systems, Hayward, CA, USA). The serum level of C-terminal
telopeptide of Type I collagen (CTX; a marker of bone resorption) was measured in duplicate by an enzyme-linked immunosorbent assay (ELISA) (Nordic Bioscience Diagnostics, Herlev, Denmark).

**Gas analysis**

For the quantification of oxygen uptake during the subjects’ exercise, the fractions of oxygen and carbon dioxide in the expired air were measured by a mass spectrometer (Arco 2000; Arcosystems, Chiba, Japan). The gas volume was measured by a gasometer (Shinagawa Seisakusho, Tokyo, Japan).

**Statistical analysis**

Values are expressed as the mean ± SD. All analyses were performed using Sigma Plot 12 software (Systat, San Jose, CA). A two-way analysis of variance (ANOVA) was performed for analysis of the differences in blood parameters between the HIIE and MIE experiments. Differences were considered significant when p < 0.05.

**Results**

Subjects’ heart rates at the end of the HIIE and MIE were 174 ± 6 bpm and 163 ± 16 bpm, respectively. The rating of perceived exertion (RPE) at the end of the HIIE and MIE was 20 ± 0 and 14 ± 3, respectively. The peak lactate concentration after the HIIE was 15.2 ± 1.3 mmol/l. The lactate concentration at the end of the MIE was 2.2 ± 0.9 mmol/l.

The serum PTH concentration before exercise did not differ between the HIIE and MIE. The PTH concentration observed 10 min after the HIIE was significantly less than the Pre1 value of HIIE (p < 0.05) (Fig. 1). The serum PTH concentrations at other time periods were not significantly different from the Pre1 value. For MIE, significant increases from Pre1 were observed immediately and 10 min after the exercise at p < 0.05 and 0.001, respectively. The serum PTH concentrations observed immediately and 10 min after exercise were significantly higher than that observed at the same time point of the HIIE (p < 0.001).

The serum iCa values obtained before exercise did not significantly differ between the HIIE (Pre1 and Pre2) and the MIE (Pre1) (Fig. 2). The serum iCa level 30 min after the start of the MIE (MIE30) did not significantly differ from Pre1 measured for the MIE. Immediately after the HIIE, the iCa level increased significantly (p < 0.001). After this period, no significant differences from the Pre1 value were observed. For MIE, no changes in the iCa values were noted during the experimental period between Pre1 and 90 min after the exercise. The iCa level immediately and 10 min after the HIIE was significantly higher than that observed at the same period of MIE (p < 0.001 and 0.05, respectively).

The serum phosphate concentration observed immediately and 10 min after the MIE was significantly higher than that observed before the exercise (p < 0.001) (Fig. 3). A significant increase from the pre-exercise value was only noted immediately after the HIIE (p < 0.001). The serum phosphate concentration observed at 60 min and 90 min after the HIIE was significantly lower than that measured before the exercise (p < 0.001). The serum phosphate concentrations observed 30, 60, and 90 min after the HIIE were significantly lower than those at the

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![Fig. 1](image-url)  
**Fig. 1** Effects of HIIE and MIE on serum PTH. Values are mean ± SD. *A significant decrease from Pre1 at p < 0.05 (HIIE). ! and !!!: Significant increases from Pre1 at p < 0.05 and 0.001, respectively (MIE). ###: p < 0.001 between HIIE and MIE at the indicated time points.
same timepoints of MIE at $p < 0.001$, 0.01, and 0.001, respectively.

The serum cortisol concentration immediately and 10 min after the MIE was significantly higher than that observed before the exercise at $p < 0.05$ and $p < 0.001$, respectively, whereas no significant changes from the pre-exercise value were noted after the HIIE (Fig. 4). The serum cortisol concentration gradually and significantly decreased after both the MIE and HIIE ($p < 0.05$–0.001). The serum cortisol concentration measured immediately after the HIIE was significantly lower than that observed at the same time point of MIE ($p < 0.05$).

There was no significant difference in the serum CTX concentration between the HIIE and MIE at pre-exercise (Fig. 5). No changes in CTX were observed in response to either HIIE or MIE.

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**Fig. 2** Effects of HIIE and MIE on serum iCa. Values are mean ± SD. ***A significant increase from Pre1 at $p < 0.001$ (MIE). # and ###: Significant differences between HIIE and MIE at the indicated time points at $p < 0.05$ and 0.001, respectively.

**Fig. 3** Effects of HIIE and MIE on serum phosphate. Values are mean ± SD. ***A significant decrease from Pre1 at $p < 0.001$ (HIIE). !!!: Significant increase from Pre1 at $p < 0.001$ (MIE). #, ##, and ###: $p < 0.05$, 0.01, and 0.001 between HIIE and MIE at the indicated time points, respectively.
Discussion

Our present findings demonstrated that high-intensity intermittent exercise does not stimulate PTH secretion, suggesting that this type of exercise may not induce a disruption of bone metabolism. The results showed that during the HIIE and at 90 min of recovery, subject serum PTH concentration was not increased, suggesting that HIIE may not increase bone absorption, which can lead to the bone loss. This result is in accord with previous studies. Kristoffersson et al. reported that a modified Wingate exercise (a method for measuring anaerobic capacity) did not significantly affect subject serum PTH concentration up to 60 min after the exercise\(^4\). Cunningham et al. also reported an absence of significant changes in serum PTH after a supramaximal short exhausting exercise (time to

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**Fig. 4** Effects of HIIE and MIE on serum cortisol. Values are mean ± SD. *, **: Significant decreases from Pre1 at p < 0.001 and 0.05, respectively (HIIE). ! and !!!: Significant increases from Pre1 at p < 0.05 and 0.001, respectively (MIE). #: p < 0.05 between HIIE and MIE at the indicated time points.

**Fig. 5** Effects of HIIE and MIE on serum C-terminal telopeptide of Type I collagen (CTX). Values are mean ± SD.
exhaustion: 60–130 sec\textsuperscript{5,6}. Takeda et al. reported that immediately after a high intensity intermittent exercise, consisting of three 10-sec sets of maximal cycling exercise with 2-min rest between bouts, PTH decreased significantly\textsuperscript{15}. In this previous study\textsuperscript{15}, the PTH concentration was transiently increased 15 min after exercise, but had returned to the pre-exercise value 30 min post-exercise. Collectively, the past and present findings indicate that it is reasonable to conclude that supramaximal intensity exercise stimulates little, if any, PTH secretion during and after exercise.

It is of interest that we observed herein that PTH was significantly reduced 10 min after the HIIE. Kristoffersson et al. reported that 5 min after 30 sec of modified Wingate exercise, the plasma PTH level was not significantly changed (−18%) with a significant elevation of ionized Ca (pre: 1.23 mmol/l, post: 1.30 mmol/l)\textsuperscript{4}. In the present investigation, the serum PTH was not changed immediately after the HIIE (total duration of the exercise test: <4 min) when iCa was elevated, but the serum PTH was significantly reduced 10 min after the HIIE (−27%) with elevated iCa compared to the pre-exercise value, suggesting that 10 min of high serum iCa might reduce the secretion of PTH.

Takeda et al. reported that PTH decreased significantly immediately after a high intensity intermittent exercise consisting of 3 sets of a 10-sec maximal cycling exercise with 2-min rest between bouts (total duration of the exercise test: 4 min 30 sec), with a significant elevation of Ca\textsuperscript{15}. Cunningham et al. also reported that not 1 min after, but 5 min after a short exhausting exercise (duration: 60–130 sec), the PTH concentration tended to be decreased; statistical results were not reported because only five subjects were studied\textsuperscript{5}. Collectively, these study results suggest that there may be a delayed (~5 min) decrease in serum PTH after high-intensity exercise that causes an increase in iCa.

Several studies pointed out that the iCa value adjusted by the hemoconcentration is appropriate in terms of discussing iCa as a stimulating factor for PTH secretion\textsuperscript{1-3}). Herein, the adjusted iCa value at the end of the MIE (0.90 ± 0.05 mmol/l) was significantly lower than that of the pre-exercise value (1.12 ± 0.02 mmol/l) (p < 0.001), suggesting that decreased iCa might stimulate PTH secretion. However, the adjusted iCa observed 10 min after the HIIE (0.94 ± 0.07 mmol/l) (p < 0.001) was also significantly lower than the pre-exercise value (1.12 ± 0.05 mmol/l) (p < 0.001), whereas the adjusted PTH value (18 ± 3 pg/mL) observed at the same time point was still significantly less than the pre-exercise value (35 ± 7 pg/mL) (p < 0.01). These results might not support our hypothesis that PTH secretion is affected directly by iCa, expressed as either raw or adjusted values. However, since the maintenance of iCa by infusion\textsuperscript{19} and calcium supplementation\textsuperscript{17} avoided PTH secretion during moderate-intensity prolonged exercise, Ca availability could be related to increased PTH secretion during such exercise. Further research is necessary to determine the relationship between iCa and PTH secretion during various types of exercises.

The HIIE used in the present investigation was shown to maximally stimulate anaerobic energy release\textsuperscript{6,11}, resulting in a high blood lactate concentration and low pH. Acidosis is a stimulus for PTH secretion\textsuperscript{17,18}. It is thus reasonable to speculate that such exercise may evoke high PTH secretion. However, in the present investigation, no changes in PTH were observed after the HIIE. The blood lactate concentration (~15 mmol/L) was similar to that observed by Kristoffersson et al.\textsuperscript{14}. The results of these two studies might indicate that the effect of elevated iCa for inhibiting the secretion of PTH is stronger than the stimulatory effect of low pH.

Townsend et al. suggested that an exercise-induced increase in serum phosphate may elevate PTH\textsuperscript{10}. Another study suggested a direct relationship between submaximal exercise intensity (−20%−−80% VO\textsubscript{2}max) and serum phosphate concentration\textsuperscript{5,6}. After short-duration exhausting supramaximal exercises, phosphate was significantly increased and PTH tended to increase, whereas the 30-sec Wingate exercise did not affect the serum PTH or phosphate levels\textsuperscript{5,6}. In the present investigation, subjects’ serum phosphate increased at the end of both the HIIE and MIE. However, the serum phosphate concentration was decreased thereafter and lower than the pre-exercise level. Further, the phosphate concentration after the HIIE was significantly lower than that after the MIE. These results may indicate that in terms of a less-negative stimulation of PTH and subsequent bone absorption, HIIE is more protective on bone compared to MIE.

PTH secretion might be stimulated by the β-adrenergic system, which is augmented by intense exercise\textsuperscript{21}. Since catecholamines were not measured in the present study, it is not known whether this influenced the subjects’ PTH concentration.

The PTH secretion response induced by high-intensity exercise may depend on the gender, age, and training status of the person engaged in the exercise. In terms of gender, Kohrt et al. reported that in adults, no gender difference was observed in the increased PTH concentration after moderate-intensity prolonged exercise\textsuperscript{22}. Further, since the response to high-intensity intermittent exercise in female adolescent athletes\textsuperscript{15} seems to be no different from that observed in the young subjects of the present investigation, we speculate that gender may not affect the PTH responses to high-intensity intermittent exercise. In terms of training status and age, the PTH response to acute exercise was reported to be unaffected by training status in young subjects\textsuperscript{23,24}, although the PTH response in older men was shown to be enhanced after training\textsuperscript{25}. The training status of the present subjects was that of endurance-trained triathletes. Therefore, the suggestions and conclusions derived from the present investigation may
be applicable to sedentary and moderately trained young men and women, and possibly to adolescent athletes.

Scott et al. reported that the PTH and CTX responses to exercise are exercise intensity-dependent. Exercise at 75% $\text{VO}_2\text{max}$ (but not 55% or 65%) induced PTH secretion and an elevation of serum CTX, suggesting that a threshold for PTH secretion may exist between 65% and 75% $\text{VO}_2\text{max}$. An exercise-induced increase in PTH would be expected to activate bone resorption. Evidence for this would be an increase in serum CTX, which is a relative index of bone resorption rather than a quantitative estimate of Ca mobilized from bone. No changes in serum CTX were observed after the HIIE in the present investigation. In another study, the subjects’ serum CTX values were also unchanged up to 60 min after 30 sec of exercise, suggesting that brief supramaximal exercise does not activate bone absorption. However, since the effects of such exercise may continue for a longer period, further research on the total balance in bone absorption and synthesis should be conducted.

No changes in serum CTX were observed after the MIE in the present investigation. Because the intensity of this bout was 70% of the $\text{VO}_2\text{max}$, this observation is consistent with the finding reported by Scott et al. that running at 75% of the $\text{VO}_2\text{max}$, but not 55% or 65%, resulted in increases in PTH and CTX.

Brahm et al. reported that exhausting running exercise lasting ~35 min increased the serum PTH concentration despite a significant increase in the serum Ca concentration. The lactate concentration was 10.6 mmol/L and the Hb concentration changed by 15.1%, and both of these findings are comparable to the present results. The Brahm et al.’s study also demonstrated that the blood levels of carboxy-terminal propeptide of type I procollagen (PICP) (a bone formation marker) and carboxy-terminal cross-linked telopeptide of type I collagen (ICTP) (a bone resorption marker) did not change up to 30 min after the exercise, but were significantly increased 24 hr after the exercise, demonstrating an increased bone turnover rate after the exercise. Thorsen et al. also reported that after 45 min jogging at 50% $\text{VO}_2\text{max}$, elevations were observed in both ICTP and PICP at 24 and 72 hr after the exercise, and they observed a strong positive correlation between serum PICP and ICTP throughout this time interval. Brahm et al. reported that they observed a net release of both PICP and ICTP from the exercising thigh. These markers were not measured in the present study, but future studies should evaluate these two markers up to 24 hr after HIIE.

Sustained elevations in PTH are normally associated with increased bone resorption, whereas daily injections that produce a transient spike in PTH lead to an increase in bone formation. The mechanisms explaining these paradoxical effects of PTH on bone have not yet been fully clarified. It thus remains unclear whether an exercise-induced increase in PTH contributes to favorable or unfavorable changes in bone metabolism.

Bone absorption may be induced by an increased concentration of blood cortisol. After the MIE in the present study, subjects’ serum cortisol concentration increased, suggesting that bone absorption may occur after such exercise. Since no changes from the pre-exercise value were observed in the serum cortisol concentration at the end of the HIIE and until 90 min after the HIIE, HIIE might be a preferred form of exercise for bone health. Earlier studies suggested that during exercise lasting 30–120 min, the blood cortisol concentration increases if the exercise intensity is >60% $\text{VO}_2\text{max}$; whereas prolonged exercise at the intensity <50% $\text{VO}_2\text{max}$ lasting >2 hrs evokes the secretion of both adrenocorticotropic hormone (ACTH) and cortisol when the blood glucose concentration decreases to <3.3 mM. Since the intensity of the MIE in the present investigation (70% $\text{VO}_2\text{max}$) was >60% $\text{VO}_2\text{max}$ and the exercise time was >30 min, the increase that we observed in the serum cortisol concentration was consistent with previous studies. However, even though the exercise intensity of the HIIE (170% $\text{VO}_2\text{max}$) was far above the threshold, no change in the serum cortisol concentration was observed after the HIIE. No data on ACTH (which triggers cortisol secretion) were available in our study, and it is thus difficult to discuss this issue. One possibility is that 4 min might be too short to induce pituitary ACTH production and secretion and the consequent increase in blood cortisol. The decrease in cortisol during recovery following both the MIE and HIIE may reflect the circadian rhythm of cortisol at that time of day.

Many elite endurance athletes suffer bone injuries that prevent them from training and competing. If the exercise-induced increase in PTH leads to an excessive activation of bone absorption during conventional training regimens that are composed mainly of moderate-intensity long distance running, this could contribute to the risk of bone injury. Therefore, if the responses of PTH to high-intensity exercise are determined to be favorable in terms of how they affect bone metabolism, endurance athletes may be able to optimize their cardiorespiratory fitness and reduce their risk of bone injury by using high-intensity intermittent exercise training (HIIT). This unique and potential feature might be another favorable characteristic of HIIE in terms of training exercise.

An animal study demonstrated some beneficial effects of high-intensity intermittent swimming training on the bone structure and strength of ovariectomized rats. Another study suggested that more HIIT seems to be a protective factor against injuries in junior elite orienteering athletes. These findings might help design training programs for elite long-distance runners who run longer at moderate intensity and frequently suffer from stress fractures.
Study limitations

Since the present investigation was not an intervention study, the conclusions that can be drawn based on the findings are weak; however, some epidemiological studies suggest that the volume (running distance) of training is more closely related to incidents of bone fracture than the intensity (running speed). Further investigations comparing the incident rates of bone fracture among elite endurance athletes who engage in HIIT and those who do not for their training may verify the findings provided by the present investigation.

Conclusion

Our present findings demonstrate that, in contrast to more prolonged moderate-intensity exercise, brief supermaximal exercise does not stimulate the PTH secretion that may deteriorate bone metabolism.

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Conflicts of Interests

The authors have no conflict of interests to report. The results of the study are presented clearly, honestly, and without fabrication or inappropriate data manipulation.

Author Contributions

JH, TS and IT conceived and designed the research. JH, TS and KT performed the experiments. JH and TS analyzed the data. JH, TS and IT interpreted the results of the experiments. JH and KT prepared the figures and tables. JH and TS drafted the manuscript. JH, IT and WMK edited and revised the manuscript. JH, TS and IT interpreted the results of the experiments. JH and TS and IT conceived and designed the research. JH, TS and IT interpreted the results of the experiments. JH and TS analyzed the data. JH, TS and IT interpreted the results of the experiments.

Appendix. Supplemental Data

Appendix regarding the history behind the Tabata training is available.

References


