ORIGINAL ARTICLE

Response of bone metabolism related hormones to a single session of strenuous exercise in active elderly subjects

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Objective: To evaluate the effect of strenuous exercise on bone metabolism and related hormones in elderly subjects.

Methods: Twenty one active elderly subjects (11 men and 10 women; mean age 73.3 years) showing a mean theoretical Vo_{2max} of 151.4% participated. Concentrations of plasma ionised calcium (iCa), serum intact parathyroid hormone (iPTH), 25-hydroxyvitamin D (25(OH)D), and 1.25-dihydroxy-vitamin D3 ($1.25(OH)_2D3$), as well as the bone biochemical markers type I collagen C-telopeptide for bone resorption and osteocalcin and bone alkaline phosphatase for bone formation, were analysed before and after a maximal incremental exercise test.

Results: At basal level, iPTH was positively correlated with age ($r=0.56$, $p<0.01$) and negatively correlated with 25(OH)D ($r=-0.50$; $p<0.01$) and $1.25(OH)_2D3$ ($r=-0.47$; $p<0.05$). Moreover, 25(OH)D and $1.25(OH)_2D3$ levels were negatively correlated with age ($r=-0.50$, $p<0.01$ and $r=-0.53$, $p<0.01$, respectively). After exercise, iCa and 25(OH)D decreased ($p<0.001$ and $p=0.01$, respectively) while iPTH increased ($p<0.001$). The levels of $1.25(OH)_2D3$, bone biochemical markers, haematocrit, and haemoglobin were unchanged. The variations in iCa and 25(OH)D were not related to age and/or sex. The iPTH variation was directly related to basal iPTH levels ($p<0.01$) and indirectly related to age.

Conclusions: In active elderly subjects, strenuous exercise disturbed calcium homeostasis and bone related hormones without immediate measurable effect on bone turnover. Although an increase in iPTH could have an anabolic action on bone tissue, our findings from our short term study did not allow us to conclude that such action occurred.

Osteoporosis, which is a major public health problem, can be defined as a diffuse skeletal disease with reduced bone mass and altered bone micro-architecture as its main structural features.[1] Both abnormalities result in increased fragility of the skeleton, and hip fracture is the most serious complication with associated high rates of morbidity and mortality.[2] Osteoporosis is a multifactorial process that depends on several environmental factors, such as dietary calcium deficiency[3] and genetic influences.[4]

Improving nutritional habits, particularly dietary calcium intake, and increasing the level of physical activity have been suggested as practical strategies for the non-pharmacological prevention of osteoporosis.[5] Physical exercise has different beneficial effects on the skeleton according to the period of life in which it is undertaken: it optimises peak bone mass in growing children[6] and maintains bone mass or reduces the bone loss rate in the elderly.[7] The beneficial effect of physical activity on bone mineral density (BMD) was particularly well demonstrated in power trained athletes engaged in high impact or strengthening exercises,[8] but such an activity level is not conceivable in fragile elderly subjects. Furthermore, the search for a relationship between physical activity and bone density in osteoporotic patients has not always led to consistent findings. Although most authors have reported the beneficial effects of exercise on bone mass,[9 10] others have not.[11] These conflicting results may be related to selection differences (age, gender, subject's initial BMD), the explored bone sites,[5 9] or the duration and type (intensity, mechanical loading, and so on) of the exercise program.[9] To optimise the effect of physical activity on bone health, a better understanding of the bone tissue responses to specific mechanical stimuli is therefore needed. More precise definition of the biomechanical specifications, as well as the intensity, duration, and frequency of the exercise to be prescribed, would most likely lead to more efficient prevention of bone loss.

By consensus, BMD is usually measured to estimate bone health and may be the primary indicator of the risk of osteoporotic fractures.[12] However, although BMD measurements provide a static representation of bone mineral status, they cannot be used to evaluate the slight bone metabolic changes induced by a single episode of physical exercise. Biochemical markers, on the other hand, can be used to assess dynamic changes in bone turnover and appear to be sensitive enough to determine the bone response to a given exercise. Brahm *et al*[13] demonstrated favourable systemic effects of physical exercise on bone metabolism using serum bone markers, while Wallace *et al*[14] showed that endurance exercise transiently activates bone and collagen turnover. In elderly subjects, bone markers are used especially for monitoring treatment for osteoporosis.[15] Few reports, however, are available concerning the effects of physical exercise on bone markers in a healthy aged population.[16] Moreover, the immediate effect of exercise on calcium homeostasis and bone turnover has never been evaluated in this population, even though the data obtained from bone marker based

Abbreviations: $1.25(OH)_2D3$, 1.25-dihydroxy-vitamin D3; 25(OH)D, 25-hydroxyvitamin D; B-ALP, bone alkaline phosphatase; BMD, bone mineral density; BMI, body mass index; bpm, beats per minute; CTX, type I collagen C-telopeptide; CV, coefficient of variation; Hb, haemoglobin; Hct, haematocrit; HR, heart rate; iCa, ionised calcium; iPTH, intact parathyroid hormone; IRMA, immunoradiometric assay; OC, osteocalcin; RER, respiratory exchange ratio; RIA, radioimmunoassay; Vco_2, CO_2 output; VE, ventilation; Vo_2, oxygen consumption

investigating methods would provide useful information for the design of therapeutic programs to improve bone health.

The aim of our study was to determine to what extent a single session of brisk walking exercise affected bone metabolism related hormones in active elderly subjects. Normal walking is generally not considered to be a high impact loading activity, but it is the only physical exercise commonly practiced by the elderly. Moreover, we examined whether the response was sex or age dependent, and whether it was likely to provide early indications of the effects of physical exercise (if any) on the bone biochemical markers.

METHODS

Subjects

Twenty one physically active elderly subjects, 11 men and 10 women (mean age 73.3 years, range 60–88) who were free of any limiting orthopaedic conditions, underwent medical screening that included a medical history, a physical examination, and an electrocardiogram. The non-inclusion criteria were medical treatment known to affect bone metabolism, osteoporotic hip or vertebral fractures, cardiovascular disease, diabetes mellitus, smoking, and excessive alcohol intake. Ethical approval was obtained from the local ethics committee and informed consent was given by the subjects.

The subjects were asked to complete a series of questionnaires concerning their medical history and physical activity level. This last was assessed with a questionnaire specifically adapted for the elderly,[17] with different scores to quantify household, sports, and other physically active leisure time activities, together resulting in a total activity score. The questionnaire provided a method for classifying elderly subjects into categories of high, medium, and low physical activity, with cut off points of 16.5 and 9.4.[17] Answers to routine queries about known metabolic disorders, current medication, diet, possible vitamin and mineral supplementation, and smoking/alcohol consumption were also collected.

Experimental protocol

The experiments were carried out in two sessions separated by at least 4 days and never more than 7 days. During the first session, the subjects' preferred walking speed was determined. The second session was devoted to a maximal incremental exercise test.

Preferred walking speed determination

The subjects began treadmill walking at the lowest familiarisation speed (0.67 m s^{-1}), which was then slowly increased until each subject subjectively identified his or her preferred walking speed. This speed was maintained for 1 min and was then modified slightly. The subject was again asked to evaluate the speed and adjustments were made according to the subject's directives. This procedure was repeated starting with the highest familiarisation speed (1.56 m s^{-1}) and gradually reducing to the preferred speed. The final preferred walking speed was considered to be the mean of the two speeds selected by the subject during both the increasing and decreasing speed trials.[17 18] During this session and the exercise test, the subjects were secured continuously by a cross-belt fixed to the handrails in such a way that arm swing was not impeded.

Maximal incremental exercise test

The subjects arrived at the laboratory at 8.00 a.m. after a 12 h overnight fast. At 8.30 a.m., a resting blood sample was drawn for analysis of biochemical parameters. The subjects then performed a maximal incremental exercise test at the individually determined preferred walking speed as previously described.[18] After a 3 min standing rest period, they were asked to walk for 5 min at 0% grade for warm up. The grade was then increased by 1–2% each minute until exhaustion, resulting in a test duration of between 8 and 12 min. During the test, oxygen consumption (Vo_2), CO_2 output (Vco_2), and ventilation (VE) were analysed breath by breath using an on line system (LE 200 CE, Jaeger, Hoechberg, Germany) and averaged every 20 s. Cardiac activity was continuously monitored using a 12 lead electrocardiogram (Oxycon Pro, Jaeger). Theoretical Vo_2 maximum (Vo_{2max} th) values were obtained using reference equations specifically developed in older adults aged 55–86 years during a treadmill maximal exercise test, according to age and sex.[19] Different equations were thus specifically developed for men (Vo_{2max} th (ml kg^{-1} min^{-1}) = 44.23–0.31×age) and women (Vo_{2max} th ml kg^{-1} min^{-1}) = 36.63−0.25×age. Vo_{2max} was considered to be attained if the subject reported a feeling of fatigue and if one of the following criteria was reached: (a) a plateau in oxygen uptake concurrent with continuing increase in exercise intensity or (b) respiratory exchange ratio (RER) greater than 1.0 and heart rate (HR) within 5 beats per minute (bpm) of the theoretical age specific maximal HR = (−0.84×age)+217.31 for men and HR = (−0.91×age)+221.7 for women.[19]

Sample collection

Blood samples (20 ml) were collected in sterile chilled tubes, at rest and just after the maximal incremental exercise test. The samples were allowed to clot at room temperature and were then centrifuged at 2500 rpm for 10 min at 4°C. Serum samples were stored at −80°C until analysis.

Biochemical assays

All samples were run in duplicate and, to reduce inter-assay variation, all the serum samples were analysed in a single session.

Calcium homeostasis

Ionised serum calcium (iCa) was measured by an ion-selective electrode (BGE, Electrolytes Instrumentation Laboratory, Lexington, MA). Intact parathyroid hormone (1–84) (iPTH) was measured by an immunoradiometric assay (IRMA) (N-tact PTH SP, DiaSorin, Stillwater, MN). The intra- and inter-assay coefficients of variation (CVs) were 3.6% and 3.4%, respectively. The sensitivity of the test was 0.7 pg ml^{-1} with no cross-reaction with human PTH fragments. The reference range for iPTH in our laboratory was 10–55 pg ml^{-1}. The level of 25-hydroxyvitamin D (25(OH)D) was measured by radioimmunoassay (RIA) (25-hydroxyvitamin D RIA kit, Nichols Institute Diagnostics, Paris, France). The intra- and inter-assay CVs were 5% and 8.1%, respectively. Assay sensitivity was $<$1.2 ng ml^{-1}. Serum 1.25-dihydroxy-vitamin D3 ($1.25(OH)_2D3$) was measured by RIA (1.25-dihydroxyvitamin D RIA kit, Nichols Institute Diagnostics). The sensitivity of the assay was 2.1 pg ml^{-1}. The intra- and inter-assay CVs were 5% and 10.8%, respectively. The reference range for $1.25(OH)_2D3$ in our laboratory was 20–66 pg ml^{-1}.

Bone biochemical markers

Markers of bone formation

Serum osteocalcin (OC) was measured by IRMA assay (Elsa-OST-NAT, CIS Biointernational, Gif/Yvette, France). The intra- and inter-assay CVs were below 5% and the sensitivity was 0.3 ng ml^{-1}. The reference range for serum OC in our laboratory was 5–20 ng ml^{-1}. Serum bone alkaline phosphatase (B-ALP) was measured by IRMA assay (Tandem-R Ostase, Hybritec, San Diego, CA). The sensitivity of the assay was 0.2 ng ml^{-1}, and the intra- and inter-assay CVs were less

Table 1 Characteristics of the study participants

Variables	Mean (SD)	Range
Age (year)	73.3 (9.1)	60–88
Weight (kg)	65.8 (13.2)	47–89
Height (cm)	166.3 (9.2)	152–180
BMI (kg m^{-2})	23.6 (2.9)	18.7–31.1
Vo_{2max} (l min^{-1})	2.06 (0.77)	1.16–3.49
Vo_{2max} (ml min^{-1} kg^{-1})	30.5 (7.1)	18.3–45.1
Vo_{2max} th (ml min^{-1} kg^{-1})	20.1 (3.5)	14.6–25.6
% Vo_{2max} th (ml min^{-1} kg^{-1})	151.4 (12.5)	125–195
Exercise duration (min)	9.9 (1.4)	8–13
Maximal slope (%)	15 (5)	5–24
Physical activity score	20.1 (7.1)	11.9–38.6

Data are presented as means (SD). BMI, body mass index; Vo_{2max}, maximal O_2 uptake. Vo_{2max} th, theoretical maximal O_2 uptake.

than 7% and 9%, respectively. The reference range for serum B-ALP in our laboratory was 4–15 ng ml^{-1}.

Marker of bone resorption

Serum type I-C telopeptide breakdown products (CTX) were measured by ELISA (CrossLaps ELISA, Osteometer, Rodovre, Denmark). The intra- and inter-assay CVs were less than 5.7% and 9.4%, respectively, and the detection limit was 0.5 µg l^{-1}. The reference range for CTX was <5500 pmol l^{-1} (manufacturer's specification).

Other parameters

Haematocrit (Hct) and haemoglobin (Hb) were determined by routine laboratory tests at rest and during exercise to ensure that measurements of metabolite and hormone concentrations were not influenced by changes in plasma volume.

Statistical analysis

All data are expressed as means (SD). The Gaussian distribution of variables was assessed by the Shapiro-Wilk statistical test. The differences between baseline pre-exercise and post-exercise data were assessed with Student's paired *t* test. When the *t* test was significant, the effect of independent variables such as sex and age was assessed by ANCOVA. When the ANCOVA test revealed that the independent variable significantly contributed to the outcome, Pearson correlation was performed to examine the degree of association between the variables. A level of $p<0.05$ was considered significant. SAS software, version 8.2 (SAS Institute, Cary, NC) was used for statistical analysis.

RESULTS

The anthropometric data and the parameters of physical fitness of the participants are shown in table 1. Vo_{2max} represented 150% of the predicted value, highlighting the high physical fitness of our population. According to the physical activity questionnaire previously established,[17] all subjects were classified in the medium or high physical activity categories.

Response of biochemical parameters to exercise

The variations in the parameters of calcium homeostasis are shown in table 2. The concentrations of iCa and 25(OH)D were decreased significantly after exercise ($p<0.001$ and $p = 0.01$, respectively) compared with pre-exercise measurement. The values of $1.25(OH)_2D3$, Hct, and Hb were unchanged ($p>0.05$). The concentrations of iPTH were significantly increased after exercise ($p<0.001$).

The values of the post-exercise bone biochemical markers (CTX, OC, B-ALP) were unchanged compared with pre-exercise values (table 3).

Basal levels of hormones and bone biochemical markers

All basal hormonal and bone biochemical markers were found to be within the normal ranges (tables 2 and 3). After stratification for gender, there were no significant differences between males and females.

Relationships between age, anthropometric data, and biochemical parameters at baseline

Table 4 shows the different relationships between age, body mass index (BMI), and the various biochemical parameters at baseline. Age was positively correlated with iPTH ($p<0.01$, $r = 0.56$) (fig 1) and negatively correlated with 25(OH)D ($p<0.01$, $r = -0.51$) and $1.25(OH)_2D3$ ($p<0.01$, $p = -0.53$). A negative relationship was found between iPTH and 25(OH)D ($p<0.01$, $r = -0.51$), $1.25(OH)_2D3$ ($p<0.05$, $r = -0.47$), and BMI ($p<0.05$, $r = -0.43$). Correlations were also found between markers at baseline (OC *v* B-ALP: $p<0.001$, $r = 0.76$; OC *v* CTX: $p<0.01$, $r = 0.82$; B-ALP *v* CTX: $p<0.01$, $r = 0.75$). There were no correlations between markers of bone turnover and age, anthropometric data (weight, height, BMI), parameters of physical fitness (Vo_{2max} and Vo_{2max}/kg), or physical activity score.

Effects of age and sex on the variation in biochemical parameters during exercise

The variations (Δ) in iCa and 25(OH)D were not related to age or sex. A negative correlation ($r = -0.46$, $p = 0.035$) was found between ΔiPTH and age. However, since we found that basal iPTH levels were also correlated with age ($r = 0.56$, $p<0.01$) (table 4), a stepwise linear regression analysis was performed to determine the contribution of possible independent variables (age and basal iPTH) to ΔiPTH. In fact, only basal iPTH levels ($p<0.01$) were independently related to ΔiPTH.

DISCUSSION

To our knowledge, this investigation is the first to examine the effects of a single session of high intensity physical exercise on the parameters of calcium homeostasis and the

Table 2 Parameters of calcium homeostasis before and after exercise

Biochemical parameters	Pre-exercise	Post-exercise	%Δ	p value	Normal range
Calcium homeostasis					
iCa (mmol l^{-1})	1.185 (0.027)	1.150 (0.034)	−3	<0.001	1.10–1.25
25(OH)D (pg ml^{-1})	23.4 (11.2)	21 (9.3)	−10.3	0.013	16–28
$1.25(OH)_2D3$ (pg ml^{-1})	46 (12.5)	46.1 (12.9)	0.2	0.900	20–66
iPTH (pg ml^{-1})	28.9 (9.7)	36.2 (11.8)	25	<0.001	10–55
Other parameters					
Hct (%)	43.1 (3.7)	43.4 (3.6)	0.7	0.850	39–54
Hb (g dl^{-1})	14 (1.2)	14 (1.1)	0	0.958	13–18

Data are expressed as means (SD). $1.25(OH)_2D3$, 1.25-dihydroxy-vitamin D3; 25(OH)D, 25-hydroxyvitamin D; Hb, haemoglobin; Hct, haematocrit; iCa, ionised calcium; iPTH, intact parathyroid hormone; %Δ, per cent change from pre- to post-exercise.

Table 3 Biochemical markers of bone turnover before and after exercise

Biochemical parameters	Pre-exercise	Post-exercise	p value	Normal range
Bone resorption marker				
CTX (pmol l^{-1})	5998 (3045)	5959 (2866)	0.945	<5500
Bone formation markers				
OC (ng ml^{-1})	12.7 (5.5)	12.5 (5.3)	0.627	5–20
B-ALP (ng ml^{-1})	13.1 (4.8)	13.2 (4.7)	0.606	4–16

Data are expressed as means (SD). B-ALP, bone alkaline phosphatase; CTX, type I collagen C-telopeptide; OC, osteocalcin; %Δ, per cent change from pre- to post-exercise.

bone biochemical markers in a large elderly population. The main findings were that in elderly physically active subjects: (a) strenuous exercise induced a decrease in iCa and 25(OH)D concentrations associated with a concomitant increase in iPTH concentration; (b) the variations in these parameters, with the exception of iPTH, were independent of sex and age; and (c) no modification concerning bone turnover, as evaluated by bone biochemical markers, was observed.

Basal calcium homeostasis and bone remodelling

At baseline, the major factor found to influence hormonal values was age. A relative consensus concerning the increase in serum iPTH with age in the general sedentary population has emerged from the literature,[20 21] probably related to vitamin D status.[22] The similar finding in our study suggests that maintaining a high level of physical activity is not sufficient to compensate for the hormonal profile alteration related to age. Indeed, in our study, an inverse relationship was found between the basal 25(OH)D, 1.25$(OH)_2$D3, and iPTH levels. The gradual reduction in 1.25$(OH)_2$D3 production with advancing age seems to be multifactorial and due to a reduction in 1 α-hydroxylase synthesis associated with the reduced sensitivity of this enzyme to PTH,[23] a reduced capacity of the skin to produce vitamin D, and lack of exposure to sunlight.[24] In agreement with the majority of studies, we found no relationship between serum iCa and age.[23] Moreover, no gender related difference in calciotropic hormonal status or bone turnover was found.

Response of calcium homeostasis to strenuous exercise

The study showed that a brief incremental exercise test induced a significant variation in the parameters of calcium homeostasis. The rise in serum iPTH level may directly reflect the decrease in serum iCa level, the main regulating factor of PTH secretion. Brent *et al*[25] found a strong relationship between extracellular iCa concentration and PTH secretion. This relationship was represented by an inverse sigmoidal curve, suggesting that a slight modification in iCa concentration induces large reciprocal changes in serum PTH. The net changes in these parameters cannot be attributed to haemoconcentration, since no variation in Hct or Hb was observed. It is more probable that the metabolic acidosis induced by strenuous exercise was the main factor inducing the calcium metabolism disturbance. Ashisawa *et al*[26] reported an increase in urinary calcium excretion after a single bout of resistance exercise through a decrease in renal calcium reabsorption. These alterations were not accompanied by an increase in osteoclast activity. The imbalance between urinary Ca excretion and Ca release from bone may induce a net decrease in serum iCa level. In addition, Lopez *et al*[27] demonstrated that acidosis may also have a direct effect on the increase in PTH secretion independently of calcium level.

The findings reported in the literature concerning the effect of physical activity on PTH and iCa values are conflicting and the degree of variation might reflect differences in the intensity and duration of the exercise. Our results were, nevertheless, in agreement with those of Bouassida *et al*[28] who found a decrease in iCa levels and a rise in PTH levels in young subjects during continuous exercise at 70 and 80% of Vo_{2max}. These findings were also observed by Nishiyama *et al*[29] in athletic and non-athletic young men after short and moderately intense exercise. In contrast, Kristoffersson *et al*[30] found increased iCa levels after short term maximal work, without significant changes in the serum PTH levels. PTH has been shown to act directly on osteoblasts, whereas its action on osteoclasts is mediated by local factors.[31] Animal experiments have demonstrated that intermittent administration of PTH increased bone mass and improved trabecular bone microarchitecture, whereas continuous administration by infusion at the same mean rate leads to a net loss of bone mass and altered bone structure.[32] In accordance with these experimental results, a therapeutic protocol with daily injections of PTH produced the largest gain in trabecular bone mass and reduced the risk of fracture in osteoporotic patients.[33] Although our results seem to lend support to these findings, the small iPTH variation and the short duration of our study make it difficult to interpret the increase in iPTH during physical exercise as a potential factor to improve bone health. The physiological and clinical significance of the iPTH variation related to strenuous exercise thus remains to be determined.

Table 4 Pearson correlation coefficient of the relationships between age, BMI, and basal biochemical parameter levels

Parameter	BMI	iCa	25(OH)D	1.25$(OH)_2$D3	iPTH	OC	B-ALP	CTX
Age	−0.378	−0.165	−0.504**	−0.533**	0.558**	0.036	0.055	0.196
BMI		0.037	0.075	0.154	−0.427*	−0.128	−0.073	−0.344
iCa			0.175	0.263	−0.341	0.273	0.281	0.345
25(OH)D				0.585**	−0.506**	−0.410*	−0.364	−0.584
1.25$(OH)_2$D3					−0.468*	−0.154	−0.238	0.342
iPTH						0.136	0.148	0.075
OC							0.755***	0.821**
B-ALP								0.750**

B-ALP, bone alkaline phosphatase; BMI, body mass index; CTX, type I collagen C-telopeptide; iCa, ionised calcium; iPTH, intact parathyroid hormone; OC, osteocalcin.
*p<0.05; **p<0.01; ***p<0.001.

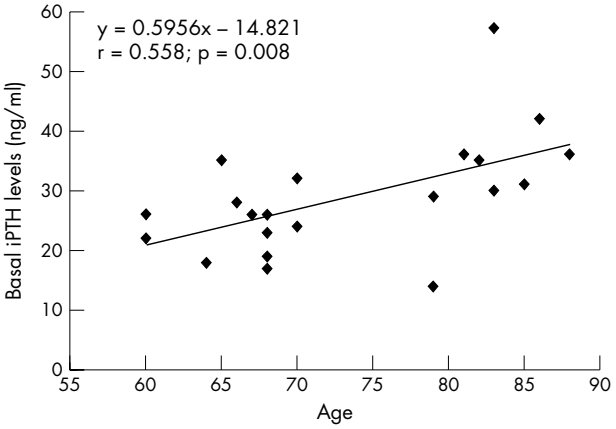

Figure 1 Relationship between age and basal iPTH levels. iPTH, intact parathyroid hormone.

We were unable to determine precisely why the 25(OH)D level decreased during exercise. It is probable that the increase in iPTH stimulates the kidney production of $1.25(OH)_2D3$, the most biologically active metabolite of vitamin D from the precursor 25(OH)D. However, as no variation in $1.25(OH)_2D3$ was observed, this assumption remains purely speculative and we cannot rule out the possibility that the change in 25(OH)D levels resulted from a modification in metabolic clearance or degradation.

Response of bone markers to strenuous exercise

Bone biochemical markers provide a new way to examine the response of bone cells to exercise. Nevertheless, we could not show any significant changes in either serum CTX, which indicates resorption activity,[34] or serum B-ALP and serum OC, which are considered to reflect newly synthesised bone.[35 36] It is widely acknowledged that long term physical activity with high mechanical loading produces an increase in bone mass, especially at the load bearing bone sites.[37 38] In our experiment, specific physical exercise did not induce any variation in bone biochemical markers, probably because the ground reaction forces generated by brisk walking, that is, approximately 1.1 times body weight,[39] were insufficient to produce an immediate measurable bone response. Our results were, nevertheless, in accordance with the majority of studies that have investigated the immediate response of bone markers to short and intense or moderate exercise bouts.[30 40–42] Therefore, the relatively short duration of exercise (range 8–13 min), as well as the short time of post-exercise investigation,[40 41] could also explain why no significant bone marker response was observed. Moreover, it seems probable that the bone tissue of our elderly population had become less responsive to exercise.[43] This was confirmed by Wallace *et al*[14] who stated that age is probably a negative determinant of the bone marker response to exercise. However, given the complexity of the bone marker response to physical exercise, these results should be cautiously interpreted. Further investigations of the effects of some of the physical activity related factors, such as clearance or/and release of bone markers, must be carried out.

CONCLUSION

In active elderly subjects, calcium homeostasis and bone metabolism related hormones were noticeably modified after a single session of strenuous physical exercise. These modifications were mainly characterised by a decrease in iCa and an increase in iPTH levels. Although a transitional increase in iPTH level may have a potential anabolic effect on bone health, the specific iPTH variation observed after short duration exercise did not allow us to draw a definitive conclusion. Further investigations are required to elucidate the physiological and clinical significance of the observed endocrine modifications. Finally, no measurable effect on bone turnover could be demonstrated immediately after strenuous physical exercise.

What is already known on this topic

Although bone mineral density measurements provide a static representation of bone mineral status, they cannot be used to evaluate the slight bone metabolic changes induced by a single episode of physical exercise.

What this study adds

In active elderly subjects, calcium homeostasis and bone metabolism related hormones were noticeably modified after a single session of strenuous physical exercise.

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