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ORIGINAL ARTICLE

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Acute effects of a single session of aerobic exercise with or without weight-lifting on bone turnover in healthy young women

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Abstract This study aimed to investigate the acute effects of exercise on bone turnover and to determine whether brisk walking with or without weight-lifting makes a difference on bone metabolism. Nine healthy women performed two exercise bouts: brisk walking on a treadmill for 30 min (E), and similar exercise carrying 5 kg of weight in a backpack (WE). Serum parathyroid hormone (PTH), osteocalcin (OC), calcitonin (CT), procollagen type 1 carboxy terminal propeptide (PICP), procollagen type 1 amino terminal propeptide (PINP), type 1 collagen carboxy terminal telopeptide (ICTP), total alkaline phosphatase (ALP), and urine deoxypyridinoline (D-Pyr) levels were studied. Resting values served as control. Significant variances were observed only in serum ALP and PTH values. Variances in ALP values within subjects after exercise were statistically significant (analysis of variance in repeated measurements [AVRM], $P = 0.000$). E caused a significant decrease, while WE caused a significant increase in ALP values at the 24th h (Bonferroni pairwise comparisons tests [BPC *t*-test]: $P = 0.028$, $P = 0.000$, respectively). Variances in PTH values within subjects after exercise were statistically significant (AVRM, $P = 0.029$), while diurnal variation was not significant ($P = 0.981$). E caused significant alterations in PTH levels (an increase at the 30th min, turned towards baseline at the 45th min) (BPC *t*-test, $P = 0.007$). WE also caused alterations in PTH levels, though insignificant (BPC *t*-test, $P = 1.00$). Brisk walking for 30 min has stimulating effects on bone turnover by various mechanisms without any additive effect of weight bearing.

Key words Acute effect · Aerobic exercise · Bone turnover

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Introduction

Osteoporosis (OP) is a systemic disease characterized by increased fracture risk as a consequence of low bone mass and microarchitectural deterioration of bone tissue. Osteoporotic fractures are associated with increased morbidity and mortality rates, which establish OP as a major public health-care concern. Therefore, diagnosis, treatment and especially prevention of osteoporosis are of particular importance.[1–3]

It is stated in several studies that physical activity has positive effects on the skeleton. Physical activity transmits mechanical loads to the skeleton via gravitational forces and muscular pull at sites of attachment.[3] Osteogenic response is suggested to be site-specific to the anatomic sites at which the mechanical strains occur.[4–6] Optimal stimulus for skeletal development is especially achieved by weight-bearing exercises.[3] Weight-bearing stimulus can be produced by both resistance exercises and aerobic exercises.[7] It is reported that appropriate overload for improving bone mass must induce forces greater than those experienced by activities of daily living.[3]

Biochemical markers of bone remodeling, a dynamic process of breakdown, and renewal of bone in order to maintain mechanical integrity of skeleton provide a dynamic measurement of skeletal status.[8] Parathyroid hormone (PTH) and calcitonin (CT) are also involved in bone metabolism and may be helpful in evaluating the effects of exercise.[9,10] Walking has been reported to have beneficial effects on skeletal mass.[11–13] This study aimed to investigate the acute effects of a single session of brisk walking on bone turnover and to determine whether additive weight-lifting would cause observable differences on bone metabolism,

Table 1. Measured parameters and apparatus

	Kit	Firm
Total alkaline phosphatase	Aeroset Alkaline Phosphatase Reagent	Abbott Diagnostics, Abbott Park, IL, USA
Osteocalcin	Biosource h-OST, Immunoradiometric assay (IRMA)	Biosource Europe, Nivelles, Belgium
Calcitonin	Biosource CT-US.-IRMA	Biosource Europe, Nivelles, Belgium
Parathyroid hormone	Gamma-BCT Intact PTH IRMA	Immuno Diagnostic Systems, Bolton, UK
Procollagen type 1 carboxy terminal propeptide	Unique PICP, Radioimmune assay (RIA)	Orion Diagnostica, Espoo, Finland
Procollagen type 1 amino terminal propeptide	Unique PINP, RIA	Orion Diagnostica, Espoo, Finland
Type 1 collagen carboxy terminal telopeptide	Unique ICTP, RIA	Orion Diagnostica, Espoo, Finland
Deoxypyridinoline	Gamma-BCT DPD, RIA	Immuno Diagnostic Systems, Bolton, UK

since it has been stated that forces greater than those experienced by daily living activities are required for improving bone mass.[3] Biochemical markers of bone remodeling, PTH, and CT were used in evaluating the acute response of bone tissue to a single-session of aerobic exercise.

Materials and methods

This study was conducted in Gazi University School of Medicine Department of Physical and Rehabilitation Medicine.

Subjects

Nine healthy and sedentary premenopausal women aged between 26 and 33 years volunteered in the study. Mean age of the subjects was 28.0 ± 2.2 years, height 164.1 ± 7.2 cm, and body mass index 19.8 ± 1.6 (kg/m^2). None of the subjects had any disease or had been consuming any drugs that could affect bone metabolism. All subjects gave informed consents and local Ethical Committee approved the study.

Experimental design

Two "single" exercise bouts were carried out with 7 days' interval in between: brisk walking on a treadmill for 30 min at submaximal intensity (Group E), and similar exercise carrying 5 kg of weight in a backpack (Group WE). On three occasions (resting, E, and WE), fasting blood samples for PTH, CT, osteocalcin (OC), procollagen type 1 carboxy terminal propeptide (PICP), procollagen type 1 amino terminal propeptide (PINP), and type 1 collagen carboxy terminal telopeptide (ICTP) were collected at initially, at the 30th, and 45th min; for alkaline phosphatase (ALP) initially and the 24th h; urine samples for deoxypyridinoline (D-Pyr) initially, at the 1st, and 24th hs. D-Pyr measurements were corrected by using urinary creatinine values. Resting values were obtained at the same scheduled intervals without performing any exercise and served as control group (Group C). All exercise bouts were carried out between 09:00 and 11:00 h. There was an interval of 7 days between these three experiments (resting, E, and WE) and experiments were conducted at the same hour of the day for the same subject in order to eliminate the diurnal variation factor.

Intensity of aerobic exercise was defined in terms of the individual's heart rate. Maximum heart rates (HR_{max}) of subjects were calculated by using formula: $HR_{max} = 220 -$ age. Targeted heart rate for aerobic exercise at submaximal intensity was 60–85% of HR_{max}.[14]

Testing

Measured parameters in blood and urine samples are given in Table 1. Blood samples were collected from the antecubital vein at scheduled intervals and centrifuged instantly at room temperature. Plasma obtained was frozen at –85°C for subsequent analysis. Urine samples were also obtained at scheduled intervals; centrifuged instantly and was frozen at –85°C. To prevent interpretation errors, all blood and urine samples were analyzed in the same run by the same investigator.

Statistical analysis

The statistical analyses were performed using the Statistical Package for the Social Sciences Package for Windows (SPSS, Chicago, IL, USA). Within-subject variations (exercise × diurnal variation) were assessed initially by using analysis of variance in repeated measurements (AVRM) tests and subsequently by Bonferroni pairwise comparisons (BPC) *t*-tests. Significance level was set at $P < 0.05$ for all statistical analyses.

Results

Statistically significant variances were observed only in serum ALP and PTH values. No statistically significant variances were observed in OC, CT, PICP, PINP, and ICTP values. Diurnal variation was not observed in any of the studied parameters (Table 2). Statistical analysis of deoxypyridinoline values was not performed since standard deviations were too high.

Table 2. Change in bone metabolism markers

	Diurnal variation (*P* value)	Brisk walking (E + WE) (AVRM, *P* value)	E (BPC *t*-test, *P* value)	WE (BPC *t*-test, *P* value)
Total alkaline phosphatase	–	0.000*	0.028*	0.000*
Parathyroid hormone	0.981	0.029*	0.007*	1.00
Osteocalcin	0.059	0.849	–	–
Calcitonin	0.107	0.320	–	–
Procollagen type 1 amino terminal propeptide	0.515	0.314	–	–
Procollagen type 1 carboxy terminal propeptide	0.867	0.843	–	–
Type 1 collagen carboxy terminal telopeptide	0.894	0.681	–	–

E, brisk walking on a treadmill for 30 min at submaximal intensity; WE, brisk walking on a treadmill for 30 min at submaximal intensity carrying 5 kg of weight in a backpack; AVRM, analysis of variance in repeated measurements; BPC *t*-tests, Bonferroni pairwise comparisons tests
* $P < 0.05$

Table 3. Alkaline phosphatase values (U/l)

Experiments	Baseline Mean ± SD	24th h Mean ± SD
Group C	64.8 ± 12.9	61.6 ± 12.5
Group E	61.1 ± 12.1	53.8 ± 14.7*
Group WE	52.6 ± 10.6	57.3 ± 17.4*

Group C, control group; Group E, brisk walking on a treadmill for 30 min at submaximal intensity; Group WE, brisk walking on a treadmill for 30 min at submaximal intensity carrying 5 kg of weight in a backpack
* $P < 0.05$

Table 4. Parathyroid hormone values (pmol/l)

Experiments	Baseline Mean ± SD	30th minute Mean ± SD	45th minute Mean ± SD
Group C	2.2 ± 2.2	2.7 ± 1.6	2.2 ± 1.3
Group E	2.6 ± 1.9	5.1 ± 2.3*	2.7 ± 1.52*
Group WE	2.5 ± 1.3	5.0 ± 1.1	2.4 ± 1.5

Group C, control group; Group E, brisk walking on a treadmill for 30 min at submaximal intensity; Group WE, brisk walking on a treadmill for 30 min at submaximal intensity carrying 5 kg of weight in a backpack
* $P < 0.05$

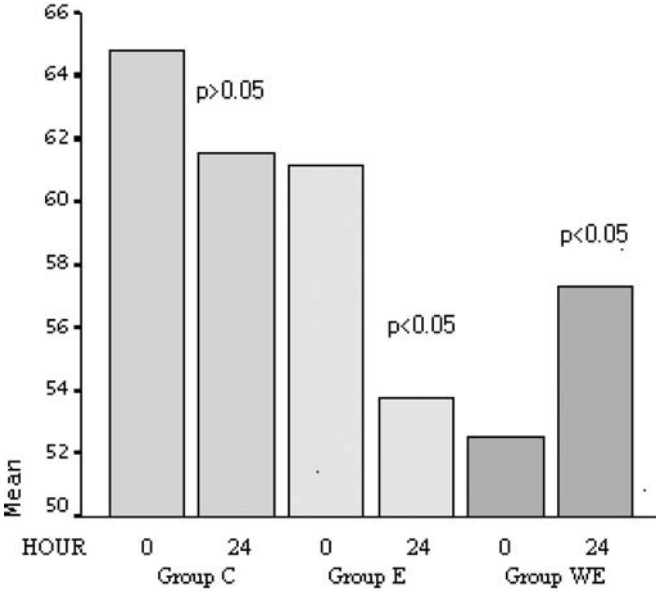

Fig. 1. Alkaline phosphatase values (U/l). *Group C*, control group; *Group E*, brisk walking on a treadmill for 30 min at submaximal intensity; *Group WE*, brisk walking on a treadmill for 30 min at submaximal intensity carrying 5 kg of weight in a backpack

Discussion

Variances in ALP values within subjects after exercise were statistically significant (AVRM, $P = 0.000$). E caused a significant decrease, while WE caused a significant increase in ALP values at the 24th h (BPC *t*-test; $P = 0.028$, $P = 0.000$, respectively) (Table 3 and Fig. 1). Variances in PTH values within subjects after exercise were statistically significant (AVRM, $P = 0.029$), while diurnal variation was not significant ($P = 0.981$). E caused significant alterations in PTH levels (an increase at the 30th min, turned towards baseline at the 45th min) (BPC *t*-test, $P = 0.007$). WE also caused alterations in PTH levels, though insignificant (BPC *t*-test, $P = 1.00$) (Table 4 and Fig. 2).

Physical activity is important in maintaining skeletal mass. Determination of the acute response of bone to exercise may help estimating its long-term benefits. Several studies investigated the acute effects of exercises on bone metabolism.[9,10,15–23] Reported results are conflicting. Some studies observed anabolic effects[10,16,19] while some others reported negative effects of exercises on bone metabolism.[9,23] There are also studies in which both resorption and formation markers were found as altered, reflecting an increase in Type 1 collagen turnover.[18,20–22] In one study, a transient decrease both in bone formation and resorption was observed with intense resistance exercise.[15] Walking has been

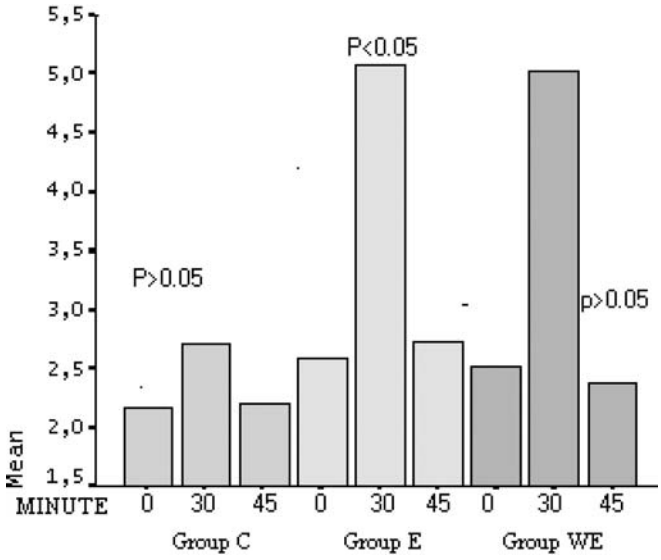

Fig. 2. Parathyroid hormone values (pmol/l). *Group C*, control group; *Group E*, brisk walking on a treadmill for 30 min at submaximal intensity; *Group WE*, brisk walking on a treadmill for 30 min at submaximal intensity carrying 5 kg of weight in a backpack

reported to have beneficial effects on skeletal mass.[11–13] This study aimed to investigate the acute effects of a single session of brisk walking on bone turnover and to determine whether added weight-lifting would cause observable differences on bone metabolism. ALP, OC, PICP, PINP, ICTP, D-Pyr, PTH, and CT levels were studied in evaluating the acute response of bone tissue to a single-session of aerobic exercise.

In our study, ALP was found to be significantly decreased at the 24th h after E ($P = 0.028$). Contrary to this, a significant increase was observed at the 24th h after WE ($P = 0.000$). Alkaline phosphatase is a marker of bone formation and our results may be interpreted as that brisk walking with added weight-lifting has anabolic effects on bone metabolism, while the same exercise without weight-lifting seems to cause a decrease in bone formation. However, total ALP was used to evaluate bone turnover in our study. Total ALP is composed of bone, liver, intestine, kidney, and placental alkaline phosphatases. Although both serum total alkaline phosphatase and serum bone-specific alkaline phosphatase are considered as bone turnover markers, bone-specific ALP is a more specific diagnostic tool.[8,24,25] In adults, bone and liver isoenzymes contribute approximately equally to the total ALP with the intestinal fraction <10%. So, measurement of total ALP may reflect bone turnover when the concentrations of other sources are stable.[26] Because of the ease of use and availability in our laboratory and considering the other sources as stable (fasting blood samples were collected in order to eliminate alterations in intestinal levels), total ALP was used in the study and this was a limitation of our study. In addition to this, having considered the rather longer half-life (1–2 days) of ALP, ALP was only studied initially and at the 24th h of the exercise. Therefore, short-lasting and also delayed alterations in ALP values reported in some studies may have been overlooked in our study. Rudberg et al. reported an increase in bone ALP, which turned towards baseline within 20 min after a moderate jogging exercise of 30–40 min.[19] Wallace et al. reported a significant increase in bone ALP in response to cycling exercise 80% VO_{2max}, which turned towards baseline in 2 h[22] Ashizawa et al. reported a decrease in bone ALP 2 and 3 days after a single bout of resistance exercise.[15] Welsh et al. did not find any alteration in ALP in response to brisk walking of 30 min at 60 HR_{max}%[23] and similarly Brahm et al. did not find any alteration in ALP levels in response to a long-distance run.[9] Therefore, more measurements during the exercise day and the days following are needed for more accurate interpretations.

Another finding in this study was a rapid increase in PTH values, which turned towards baseline in 15 min in both E and WE groups. This was not observed in the control group. This increase was statistically significant in the E group ($P = 0.007$) but not significant in the WE group ($P = 1.00$). Parathyroid hormone has both anabolic and catabolic effects on bone metabolism. Chronic hypersecretion of PTH causes a bone loss whereas intermittent PTH secretion has anabolic properties and enhances bone formation.[16] The transient significant increase of PTH observed in the E group suggests an anabolic effect of exercise on bone metabolism. WE also caused similar transient increase in PTH values, though statistically not significant. Similar to our results, Rudberg et al. reported a PTH increase besides bone ALP which turned towards baseline in 20 min in response to a moderate jogging exercise.[19] There are also differing results in the literature. Rong et al. reported an increase in PTH immediately after a strength exercise,[10] Thorsen et al. reported an increase in PTH in response to running at 50% VO_{2max} at the 2nd and 3rd days,[21] and Bouassida et al. reported an increase in PTH values in response to exercises at 70% and 85% of VO_{2max}.[16] Brahm et al. did not find any alteration in PTH values after a long-distance run[9] and Thorsen et al. did not find any alteration in PTH values in response to brisk walking at 50% HR_{max} in postmenopausal women.[20]

In this study, no significant alterations were observed in OC, CT, PINP, PICP, and ICTP levels. Similar results were also reported in the literature for OC,[15,19,20,22,23] CT,[10,20] and ICTP.[19] This might be due to the short follow-up of these parameters in our study.

In our experimental study, healthy young women were included. The results would probably differ in postmenopausal osteoporotic women since there are studies stating that weight-bearing exercises do not have positive effects on skeletal mass in postmenopausal women; which recalls that estrogen receptors might have a role on the response of osteoblasts to exercise.[27–29]

In conclusion, total ALP and PTH levels were affected by a single bout of aerobic exercise. E had more effect on PTH levels, but ALP was found to be decreased after E while it increased with WE at the 24th h. We conclude that brisk walking for 30 min has stimulating effects on bone turnover by various mechanisms, without any significant additive effect of weight bearing. Further studies, which

monitor bone metabolism in the short term and for even longer periods after exercise, are needed to clarify the acute effects of exercise on bone metabolism.

References

1. Delaney MF, LeBoff MS. Metabolic bone disease. In: Ruddy S, Harris ED, Sledge CB, editors. Kelley's textbook of rheumatology. 6th ed. Philadelphia: Saunders; 2001. p. 1635–52.
2. Kleerekoper M, Avioli LV. Evaluation and treatment of postmenopausal osteoporosis. In: Favus MJ, editor. Primer on the metabolic bone diseases and disorders of mineral metabolism. 2nd ed. New York: Raven Press; 1993. p. 223–9.
3. Shaw JM, Witzke KA. Exercise for skeletal health and osteoporosis prevention. In: Roitman JL, editor. ACSM's resource manual for guidelines for exercise testing and prescription. Baltimore: Williams and Wilkins; 1998. p. 288–93.
4. Haapasalo H, Kannus P, Sievanen H, Heinonen A, Oja P, Vuori I. Long-term unilateral loading and bone mineral density and content in female squash players. Calcif Tissue Int 1994;54(4):249–55.
5. Lanyon LE. Using functional loading to influence bone mass and architecture: objectives, mechanisms, and relationship with estrogen of the mechanically adaptive process in bone. Bone 1996;18 (1 Suppl):37S–43S.
6. Tommerup LJ, Raab DM, Crenshaw TD, Smith EL. Does weight-bearing exercise affect non-weight-bearing bone? J Bone Miner Res 1993;8(9):1053–8.
7. Layne JE, Nelson ME. The effects of progressive resistance training on bone density: a review. Med Sci Sports Exerc 1999;31(1):25–30.
8. Looker AC, Bauer DC, Chestnut CH III. Clinical use of biochemical markers of bone remodeling: current status and future directions. Osteoporosis Int 2000;11:467–80.
9. Brahm H, Piehl-Aulin K, Ljunghall S. Biochemical markers of bone metabolism during distance running in healthy, regularly exercising men and women. Scand J Med Sci Sports 1996;6:26–30.
10. Rong H, Berg U, Torring O, Sundberg CJ, Granberg B, Bucht E. Effect of acute endurance and strength exercise on circulating calcium-regulating hormones and bone markers in young healthy males. Scand J Med Sci Sports 1997;7:152–159.
11. Hatori M, Hasegawa A, Adachi H, Shinozaki A, Hayashi R, Okano H et al. The effects of walking at the anaerobic threshold level on vertebral bone loss in postmenopausal women. Calcif Tissue Int 1993;52(6):411–4.
12. Krall EA, Dawson-Hughes B. Walking is relates to bone density and rates of bone loss. Am J Med 1994;96(1):20–6.
13. Prince R, Devine A, Dick I, Criddle A, Kerr D, Kent N, et al. The effects of calcium supplementation (milk powder or tablets) and exercise on bone density in postmenopausal women. J Bone Miner Res 1995;10:1068–75.
14. Moldover JR, Bartels MN. Cardiac rehabilitation. In: Braddom RL, editor. Physical medicine and rehabilitation. 2nd ed. Philadelphia: Saunders; 2000. p. 665–86.
15. Ashizawa N, Ouchi G, Fujimura R, Yoshida Y, Tokuyama K, Suzuki M. Effect of a single bout of exercise on calcium and bone metabolism in untrained young males. Calcif Tissue Int 1998;62:104–8.
16. Bouassida A, Zalleg D, Ajina MZ, Gharbi N, Duclos M, Richalet JP, et al. Parathyroid hormone concentrations during and after two periods of high intensity exercise with and without an intervening recovery period. Eur J Appl Physiol 2003;88:339–44.
17. Kristoffersson A, Hultdin J, Holmlund I, Thorsen K, Lorentzon R. Effects of short-term maximal work on plasma calcium, parathyroid hormone, osteocalcin and biochemical markers of collagen metabolism. Int J Sports Med 1995;16:145–9.
18. Lanberg H, Skovgaard D, Asp S, Kjaer M. Time pattern of exercise-induced changes in type 1 collagen turnover after prolonged endurance exercise in humans. Calcif Tissue Int 2000;67:41–4.
19. Rudberg A, Magnusson P, Larsson L, Joborn H. Serum Isoforms of bone alkaline phosphatase increase during physical exercise in women. Calcif Tissue Int 2000;66:342–7.
20. Thorsen K, Kristoffersson A, Lorenzton R. The effects of brisk walking on markers of bone and calcium metabolism in postmenopausal women. Calcif Tissue Int 1996;58:221–5.
21. Thorsen K, Kristoffersson A, Hultdin J, Lorentzon R. Effects of moderate endurance exercise on calcium, parathyroid hormone, and markers of bone metabolism in young women. Calcif Tissue Int 1997;60:16–20.
22. Wallace JD, Cuneo RC, Lundberg PA, Rosen T, Jorgensen JOLL, Longobardi S, et al. Responses of markers of bone and collagen turnover to exercise, growth hormone (GH) administration, and GH withdrawal in trained adult males. J Clin Endocrinol Metab 2000;85:124–33.
23. Welsh L, Rutherford OM, James I, Crowley C, Corner M, Wolman R. The acute effects of exercise on bone turnover. Int J Sports Med 1997;18:247–51.
24. Rosen CJ, Tenenhouse A. Biochemical markers of bone turnover. A look at laboratory tests that reflect bone status. Postgrad Med 1998;104:101–2, 107–10, 113–4.
25. Woitge HW, Scheidt-Nave C, Kissling C, Leidig-Bruckner G, Meyer K, Grauger A, et al. Seasonal variation of biochemical indexes of bone turnover: results of a population-based study. J Clin Endocrinol Metab 1998;83:68–75.
26. Watts NB. Clinical utility of biochemical markers of bone remodelling. Clin Chem 1999;45:1359–68.
27. Bassey EJ, Rothwell MC, Littlewood JJ, Pye DW. Pre- and postmenopausal women have different bone mineral density responses to the same high-impact exercise. J Bone Miner Res 1998;13:1805–13.
28. Bravo G, Gauthier P, Roy PM, Payette H, Gaulin P. A weight-bearing, water-based exercise program for osteopenic women: its impact on bone, functional fitness and well-being. Arch Phys Med Rehabil 1997;78:1375–80.
29. Sugiyama T, Yamaguchi A, Kawai S. Effects of skeletal loading on bone mass and compensation mechanism in bone: a new insight into the "mechanostat" theory. J Bone Miner Metab 2002;20:196–200.