Original Article

Commencing, Continuing and Stopping Brisk Walking: Effects on Bone Mineral Density, Quantitative Ultrasound of Bone and Markers of Bone Metabolism in Postmenopausal Women

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Abstract. Regular walking is associated with reduced risk of fracture and, in our recent randomized trial, reduced calcaneal bone loss relative to controls. The present follow-up study compared the effects on dualenergy X-ray absorptiometry, ultrasound and biochemical indices of bone density and metabolism of (i) taking up (ii) continuing with and (iii) ceasing brisk walking for exercise. Subjects were 68 postmenopausal women aged 60-70 years. Twenty previously sedentary women remained sedentary (Sed/Sed) whilst 17 took up brisk walking (Sed/Walk). Fifteen women who had been walking regularly for 1 year returned to their former sedentary lifestyle (Walk/Sed), whilst 16 continued brisk walking over a second year (Walk/ Walk). Bone mineral density (BMD), broadband ultrasonic attenuation (BUA), and biochemical markers of bone formation (serum osteocalcin, C-terminal propeptide of type I collagen and bone alkaline phosphatase) and resorption (urinary deoxypyridinoline) were assessed at baseline and 12 months. Women in the Sed/Walk and Walk/Walk groups completed a mean (SEM) of 16.9 (0.7) and 20.8 (1.2) min of brisk walking per day, respectively. Changes in BMD did not differ significantly between groups. Calcaneal BMD decreased significantly in Walk/Sed women [by 2.7 (1.4)%; p =0.01] whilst changes in other groups were not significant. Calcaneal BUA increased significantly (p = 0.02) in Sed/Walk women [by 7.4 (3.3)%] relative to

Correspondence and offprint requests to: Dr Katherine Brooke-Wavell, Department of Human Sciences, Loughborough University, Loughborough, Leics LE11 3TU, UK. Tel: +44 (0)1509 222749. Fax: +44 (0)1509 223940. e-mail: K.S.F.Brooke-wavell@lboro.ac.uk other groups. Urinary deoxypyridinoline increased over the year in the Sed/Sed group but there were no significant changes in biochemical markers in other groups. Women taking up brisk walking for exercise showed no change in BMD but a significant increase in calcaneal BUA. There was no significant effect on BMD or BUA of continuing brisk walking but calcaneal BMD declined on ceasing brisk walking. Bone resorption increased in sedentary women but not exercisers, suggesting the effect on exercise on bone in postmenopausal women could be through amelioration of this increased turnover.

Keywords: Bone mineral density; Exercise; Postmenopausal women; Quantitative ultrasound; Walking

Introduction

Walking for exercise is associated with reduced risk of osteoporotic fracture [1], with individuals who report walking briskly having the lowest risk [2]. Brisk walking is a cheap and attainable form of exercise, popular with older adults, which carries a low risk of injury and confers other health benefits [3]. It is therefore an ideal intervention which could have a role in prevention of osteoporosis.

The association of walking with fracture risk could be due in part to effects on bone mineral density (BMD). It has also been hypothesized that mechanical forces might influence trabecular microarchitecture [4]. Furthermore, exercise might influence other BMD-independent factors such as muscular strength and coordination which could reduce the risk of falling – although in one study the relationship of exercise with fracture risk persisted even after adjustment for tests of neuromuscular function [1]. Despite the association between walking and fracture risk, longitudinal studies of the influence of walking on BMD in postmenopausal women have yielded conflicting findings [5–11]. Most studies had a duration of 1 year or less [5–10]. Recent findings in premenopausal women suggest that the response of BMD to exercise may not be evident until the second year of intervention [12], so it is possible that walking might exert continued effects on bone after 12 months. The influence of walking on BMD thus requires further elucidation.

In a recent randomized, controlled intervention study, we observed that a 1 year program of brisk walking reduced the loss in calcaneal BMD and broadband ultrasound attenuation (BUA) in postmenopausal women [10]. Quantitative ultrasound measures of bone are related to bone density but also to structural parameters such as trabecular thickness and orientation [13]. Calcaneal BUA has been found to be as good a predictor of fracture as BMD in elderly women [14] and remained a significant predictor of fracture even after adjustment for BMD [14].

The present study followed up the subjects of our 1 year randomized controlled trial [10] for a further 12 months, during which time half the walkers continued walking for exercise whilst the other half stopped; half the original sedentary controls continued their sedentary lifestyle, whilst the other half took up walking. The aims of the follow-up study were to address the following questions: (i) Is there a further effect on BMD or BUA when brisk walking is continued beyond 1 year? (ii) Are changes reversed on ceasing brisk walking for exercise? and (iii) Are there any changes in biochemical markers of bone formation or resorption which provide an indication of the mechanism of exercise-induced changes in bone quality in postmenopausal women?

Subjects and Methods

Subjects

Subjects were recruited from the 38 walkers and 40 controls who had completed our year-long randomized brisk walking intervention study. Subjects were all at least 5 years postmenopausal and not taking hormone replacement therapy. To maintain compliance, subjects were allowed to choose which of the four groups to join for the present follow-up study. Of the women allocated to the walking group during the randomized study, 17 chose to continue walking (Walk/Walk) and 15 chose to return to their previous sedentary lifestyles (Walk/Sed). Of the women allocated to the sedentary control group during the randomized study, 13 opted to take up brisk walking (Sed/Walk), whilst 21 elected to continue their sedentary lifestyles (Sed/Sed). A further 5 sedentary women, who were all at least 5 years postmenopausal, in good health and not using any medication thought to influence bone metabolism (users of menopausal hormone replacement therapy, oral corticosteroids, bisphosphonates or calcium supplements were excluded), were recruited to increase sample size in the Sed/Walk group. This sample size was calculated to provide a power of 80% to detect differences in response between groups of approximately 3% for BMD or 5% for BUA at the 95% confidence level. Some characteristics of subjects are detailed in Table 1. Subjects gave their written informed consent for all procedures, which were approved by the University Ethics Committee.

Brisk Walking

The brisk walking was self-governed but subjects were given a clear target amount to complete each fortnight. For subjects from the Sed/Walk group, targets increased from 120 min, in the first fortnight, to 280 min after 3 months and remained at this level thereafter. Continuing walkers (Walk/Walk group) were instructed to continue

Table 1. Baseline characteristics of postmenopausal women remaining sedentary (Sed/Sed), or commencing (Sed/Walk), ceasing (Walk/Sed) or continuing with (Walk/Walk) brisk walking for exercise

Variable	Walk/Sed $(n = 15)$	$\frac{\text{Sed/Sed}}{(n=20)}$	Walk/Walk $(n = 16)$	Sed/Walk $(n = 17)$
Age (years)	66.5 ± 3.2	65.0 ± 3.1	65.5 ± 2.7	65.0 ± 3.2
Stature (m)	1.63 ± 0.06	1.64 ± 0.07	1.61 ± 0.07	1.63 ± 0.07
Body mass (kg)	67.4 ± 9.9	71.4 ± 12.1	65.6 ± 9.5	68.5 ± 8.9
BUA (dB/MHz)	58.1 ± 9.6	58.3 ± 12.1	65.2 ± 14.8	65.4 ±12.6
Aerobic capacity (ml/kg min ⁻¹)	$27.7 \pm 6.1*$	23.4 ± 3.5	$28.5 \pm 6.8*$	24.4 ± 5.1
Calcium intake (mg/day)	896 ± 215	802 ± 265	939 ± 206	911 ± 251
Lumbar spine BMD (g/cm^2)	1.017 ± 0.156	0.967 ± 0.111	1.082 ± 0.196	1.099 ± 0.171
Femoral neck BMD (g/cm ²)	0.825 ± 0.098	0.831 ± 0.070	0.851 ± 0.133	0.856 ± 0.116
Calcaneal BMD (g/cm ²)	0.500 ± 0.090	0.504 ± 0.075	0.510 ± 0.119	0.523 ± 0.108

Values are mean \pm SD.

*Significantly different from Sed/Sed, p < 0.05.

completing 280 min each fortnight throughout the year. Subjects were allowed some flexibility in the number and duration of walks required to meet these targets, although each walk had to be at least 20 min long and at least 3 brisk walks for exercise were to be undertaken each week. Each exercise walk had to be in addition to any habitual slower walking for shopping, etc. Subjects completed a training diary which listed the duration of each walk completed and which was returned to the University monthly. Subjects were advised that footwear should be low-heeled, comfortable and well-fitting, and that clothing should not restrict movement, but were not required to wear any special clothing or footwear whilst walking.

Functional Capacity

Functional capacity was estimated in order to provide evidence of physiologic adaptation and, by inference, compliance with the walking prescription. Maximal oxygen uptake was predicted from the relationship between heart rate (ECG) and oxygen uptake (Douglas bag techniques), during a submaximal, incremental treadmill walking test, the final stage of which elicited approximately 70% of each woman's maximal oxygen uptake as determined at baseline.

Dietary Intakes

Dietary intakes of energy and calcium were evaluated by means of 7 day weighed food inventories with subsequent analysis using a computerized version of food composition tables (Compeat 4.1, Nutrition Systems, London).

Bone Mineral Density

BMD was assessed using dual-energy X-ray absorptiometry (DXA) at the lumbar spine (L2–L4), hip (femoral neck, trochanter and Ward's triangle) and calcaneus using a DPX-L bone densitometer (Lunar, Madison, WI). Standard errors of measurement (with subjects being repositioned between measurements) at lumbar spine, femoral neck and calcaneus were 0.014, 0.017 and 0.016 g/cm^2 respectively; coefficients of variation were 1.4%, 1.9% and 2.7%. Staff making DXA measurements were masked to subjects' group assignment. A phantom was scanned monthly to allow detection of any drift in DXA measurement. Some drift in phantom measurements over time was observed (maximum magnitude 3%). A curve was fitted to summarize change in phantom measurement with time. The regression equation was then used to adjust BMD data for the estimated drift at that date.

Broadband Ultrasonic Attenuation

BUA was measured at the calcaneus using a CUBA research system (McCue Ultrasonics, Winchester, UK). The standard error of measurement in our hands in women of this age was 3.2 dB/MHz (coefficient of variation 5.4%). Measurements were made in triplicate with repositioning of the foot between each measurement. The mean of the three measurements was calculated.

Biochemical Markers of Bone Metabolism

Sixty-two women agreed to provide blood and urine samples for assessment of biochemical markers of bone turnover (distribution between groups shown in Table 4). Blood samples from venepuncture and second-void urine samples were collected between 08:00 and 10:00 hours (to minimize effects of diurnal variation), after a 12 h fast. Subjects were instructed to avoid exercise in the 24 h preceding these samples. Serum was separated 1 h after sampling and stored at -70 °C. Urine samples were stored at -20 °C. Serum osteocalcin and urinary deoxypyridinoline (DPD) were measured by ELISA kits (Metra Biosystems, US) at baseline and after 3, 6 and 12 months. DPD concentration was adjusted by creatinine excretion (measured photometrically). The Cterminal propeptide of type I procollagen (PICP) and bone alkaline phosphatase activity in serum were measured at baseline and after 1 year by ELISA kits (Metra Biosystems, US). All measurements were performed in duplicate and all samples from the same subject were analyzed on the same assay plate. Interassay CVs for osteocalcin, PICP, bone alkaline phosphatase and DPD were 2.4%, 5.4%, 8.0% and 9.8%, respectively.

Statistical Analysis

Analysis of variance was used to compare baseline measures between groups. Repeated measures analysis of variance was used to detect significant changes within groups and differences in response over time between groups.

Results

Sixty-eight women completed the study, i.e., 15 Walk/ Sed, 16 Walk/Walk, 20 Sed/Sed and 17 Sed/Walk. Three subjects dropped out: one (Sed/Sed) was unable to continue due to accidental injury, one (Sed/Walk) developed health problems unrelated to walking and one (Walk/Walk) lost interest. Some characteristics of subjects are shown in Table 1. Predicted maximal oxygen uptake at baseline differed significantly between groups, being significantly higher in the Walk/Sed and Walk/Walk groups than in the Sed/Sed group. There

Variable	Walk/Sed $(n = 15)$	$\frac{\text{Sed/Sed}}{(n=20)}$	Walk/Walk $(n = 16)$	Sed/Walk $(n = 17)$
Lumbar spine BMD (g/cm ²) Femoral neck BMD (g/cm ²) Calcaneal BMD (g/cm ²) BUA (dB/MHz)	$\begin{array}{c} +0.007\;(0.005)\\ +0.011\;(0.009)\\ -0.000\;(0.006)\\ -1.5\;(1.4)\end{array}$	$\begin{array}{c} -0.001 \ (0.006) \\ + \ 0.012 \ (0.007) \\ - \ 0.006 \ (0.005) \\ - \ 3.8 \ (1.1)^{a,b} \end{array}$	+0.004 (0.007) +0.025 (0.009) -0.001 (0.006) -0.2 (1.3)	$\begin{array}{r} -0.001 \ (0.008) \\ +0.022 \ (0.015) \\ -0.015 \ (0.007) \\ -3.6 \ (1.6) \end{array}$

Table 2. Changes in bone during the year prior to the present study, i.e., during our previous randomized intervention trial [10], in those women participating in the present follow-up study

Values are mean (SE).

Sed/Sed, women remaining sedentary; Sed/Walk, women beginning brisk walking; Walk/Sed, women ceasing brisk walking; Walk/Walk, women continuing brisk walking.

^a Significantly different from 0: p < 0.05

^b Significant difference in response over time between groups

were no significant differences between groups in other variables at baseline.

Table 2 shows changes in bone variables for each of the groups during the randomized controlled study, i.e., during the year prior to the present follow-up study. In the randomized study, BMD changes did not differ significantly between groups, but BUA differed significantly between the Walk/Walk group (in whom bone was maintained) and the Sed/Sed group (in whom a significant reduction in BUA occurred).

Subjects in the Sed/Walk group averaged (mean \pm SD) 232 ± 48 min brisk walking per fortnight during the first 3 months of the follow-up study and 243 \pm 46 min per fortnight thereafter. Walk/Walk subjects averaged 302 \pm 69 min per fortnight over the year. The amount of walking completed each fortnight is shown in Fig. 1. Changes in predicted maximal oxygen uptake differed significantly between groups (p = 0.015; Table 3). Predicted maximal oxygen uptake decreased in women who stopped walking. There was also a statistically significant decrease in the Walk/Walk group. A nonsignificant decrease occurred in the Sed/Sed group whilst a mean increase was observed the Sed/Walk group (p = 0.10). Mean changes in body mass were small (<1) kg) and did not differ significantly between groups (Table 3).



Fig. 1. Minutes of brisk walking reported each fortnight by women continuing (Walk/Walk) and commencing (Sed/Walk) brisk walking for exercise. Values are mean (SE).

The response over time in BMD did not differ significantly between groups at any site (Table 3). There were however some within-group changes which were significant (p < 0.05), i.e., an increase of 2.0 % in lumbar spine BMD in Sed/Sed women, and a decrease (3.2%) in calcaneal BMD in Walk/Sed women (Table 3,

Table 3. Changes in body mass, predicted maximal oxygen uptake, bone mineral density (BMD) and broadband ultrasonic attenuation (BUA) of postmenopausal women remaining sedentary (Sed/Sed), or commencing (Sed/Walk), ceasing (Walk/Sed) or continuing with (Walk/Walk) brisk walking for exercise

Variable	Walk/Sed $(n = 15)$	Sed/Sed $(n = 20)$	Walk/Walk $(n = 16)$	Sed/Walk $(n = 17)$
Body mass (kg)	+0.7(0.5)	-0.3 (0.8)	-0.5 (0.5)	-1.0 (0.5)
Predicted maximal oxygen uptake (ml/kg min ⁻¹)	-2.9 (1.3) ^{a,b}	-0.2 (0.9)	$-3.7 (1.3)^{a,b}$	+1.3 (0.8)
BMD lumbar spine (g/cm ²)	-0.001 (0.007)	$+0.017 (0.006)^{a}$	+0.004 (0.008)	-0.003 (0.008)
BMD femoral neck (g/cm ²)	+0.003(0.007)	-0.002 (0.006)	-0.007 (0.008)	-0.008(0.008)
BMD calcaneus (g/cm ²)	$-0.016 (0.006)^{a}$	-0.006 (0.008)	-0.009 (0.007)	-0.000 (0.007)
BUA calcaneus (dB/MHz)	-0.5 (1.1)	+1.2(1.1)	-1.3 (0.8)	$+3.8(1.7)^{a,b}$
(ml/kg min ⁻¹) BMD lumbar spine (g/cm ²) BMD femoral neck (g/cm ²) BMD calcaneus (g/cm ²) BUA calcaneus (dB/MHz)	$\begin{array}{c} -0.001 \ (0.007) \\ +0.003 \ (0.007) \\ -0.016 \ (0.006)^{a} \\ -0.5 \ (1.1) \end{array}$	$+0.017 (0.006)^{a}$ -0.002 (0.006) -0.006 (0.008) +1.2 (1.1)	+0.004 (0.008) -0.007 (0.008) -0.009 (0.007) -1.3 (0.8)	$\begin{array}{c} -0.003 \ (0.008) \\ -0.008 \ (0.008) \\ -0.000 \ (0.007) \\ +3.8 \ (1.7)^{a,b} \end{array}$

Values are mean (SE).

^a Significantly different from 0: p < 0.05.

^b Significant difference in response over time between groups.



Fig. 2. Annual changes in calcaneal BMD and BUA in postmenopausal women remaining sedentary (Sed/Sed) or commencing (Sed/ Walk), ceasing (Walk/Sed) or continuing (Walk/Walk) brisk walking for exercise. Values are mean (SE). *Significant change: p < 0.05.

Fig. 2). BUA of the calcaneus, however, did show significant differences in response between groups (p = 0.02; Table 3, Fig. 2), with an increase of 7.4 (3.3)% in the Sed/Walk group.

Urinary DPD showed significant differences in response between groups (p = 0.03) with a significant increase occurring in the Sed/Sed group (Table 4). There were no significant changes in other biochemical markers.

Discussion

Brisk walking is clearly an acceptable form of exercise for women in this age group. In our previous randomized controlled trial [10], only 5 of the 84 subjects dropped out, with 91% of women assigned to take up brisk walking adhering to the program. In the current study 94% of women who chose to participate in the brisk walking program were adherent. Those who continued with walking for exercise exceeded their target duration on average, whilst those who took up walking for the first time completed over 85% of the required amount.

The reduction in predicted maximal oxygen uptake in the Walk/Sed group confirms that their physical activity levels had fallen sufficiently to lose the cardiovascular adaptations evident on taking up walking. The increase in the Sed/Walk group was not significant but this group included one poor complier (analysis was performed on an intention-to-treat basis). After exclusion of this subject who had been unable to walk for exercise for 14 weeks due to illness, the mean increase in maximal oxygen uptake in this group [7.4 (3.6) %] approached significance (p = 0.05). Hence it appeared that the majority of the group had complied and undertaken exercise of sufficient duration and intensity to improve endurance fitness. The decline in maximal oxygen uptake in the Walk/Walk group was unexpected, as these subjects had completed as much walking in this study as in the previous year, according to their diary records. This decline could be due to either reduced duration (not detected due to imprecision of diary recording) or reduced intensity of walking. As maximal oxygen uptake can be maintained in young adults even when training frequency is cut to one-third, but not when training intensity declines below 70% of maximal oxygen uptake [15], perhaps reduced intensity (i.e., slower walking speed) is the more likely explanation, although we have no evidence for this.

The increase in lumbar spine BMD in the Sed/Sed group is surprising as women of this age would be expected to lose bone. Changes in spinal BMD are, however, difficult to interpret in women of this age as artifactual increases could be produced by vertebral fracture, osteophytosis or osteoarthritic change.

At the calcaneus (where walking was found to reduce bone loss in our previous study) women who stopped exercising lost bone, confirming the findings of Dalsky et al. [16] that the effects of physical activity are reversed on cessation. There was no evidence of further benefit in

Table 4. Biochemical markers of bone turnover in postmenopausal women remaining sedentary (Sed/Sed), or commencing (Sed/Walk), ceasing (Walk/Sed) or continuing with (Walk/Walk) brisk walking for exercise

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Variable	Months	Sed/Sed $(n = 14)$	Sed/Walk $(n = 17)$	Walk/Sed $(n = 14)$	Walk/Walk $(n = 15)$
Osteocalcin (ng/ml)	0	11.2 (0.7)	10.6 (0.7)	11.8 (0.8)	10.4 (0.6)
	3	11.5 (0.7)	10.9 (0.7)	12.4 (0.9)	10.6 (0.6)
	6	11.7 (0.9)	11.0 (0.7)	12.5 (1.0)	11.2 (0.5)
	12	11.9 (0.7)	10.7 (0.8)	11.8 (1.1)	10.8 (0.5)
Deoxypyridinoline	0	6.9 (0.5)	5.8 (0.3)	6.2 (0.4)	6.3 (0.5)
(nM/mM creatinine)	3	7.4 (0.6)	6.1 (0.3)	7.1 (1.0)	5.8 (0.4)
	6	$7.6 (0.5)^{a,b}$	6.4 (0.2)	6.7 (0.9)	6.2 (0.5)
	12	$7.5(0.6)^{a,b}$	6.3 (0.4)	6.1 (0.4)	6.0 (0.4)
C-terminal propeptide of	0	113 (7)	94 (5)	110 (7)	87 (5)
type I collagen (ng/ml)	12	115 (6)	101 (8)	111 (11)	92 (5)
Bone alkaline phosphatase (U/l)	0	19.9 (1.2)	19.9 (1.1)	19.2 (1.3)	16.9 (1.0)
	12	20.0 (1.2)	19.7 (1.1)	19.9 (2.0)	17.3 (1.0)

Values are mean (SE)

^a Significantly different from 0 months: p < 0.05.

^bSignificant difference in response over time between groups.

women continuing brisk walking for exercise for a second year, suggesting that after 1 year the skeleton had adapted to the increased level of loading. These findings conflict with earlier reports of further improvement in the second year of intervention in postmenopausal [16] and premenopausal women [12]. However, these studies employed progressive exercise programs, so the increase in the second year of exercise may be due to the continued increase in loading in this period – although no corresponding increase in muscular strength was reported [12]. Furthermore if the decline in maximal oxygen uptake in our Walk/Walk group was indicative of a decrease in walking speed – as speculated above – then the magnitude of bone loading may not have been maintained. The greatest improvements in bone are produced with the largest strain magnitudes, both in animals [17] and in humans [18]. A slower walking speed would engender smaller strains, thus providing less of an adaptive stimulus to bone.

BUA of the calcaneus increased in subjects who started walking, relative to other groups. This shows a different trend from the changes in calcaneal BMD, where the only significant change was a decrease in the Walk/Sed group (Fig. 2). BUA is reported to be dependent on structural characteristics such as trabecular thickness and orientation [13]. Our findings thus suggest the possibility that loading through walking may produce structural reorganization of trabecular bone even in the absence of changes in BMD. This finding is also subtly different from those from our previous study, where rather than increasing BUA, walkers avoided the loss observed in controls [10]. In the absence of a reliable phantom for ultrasound measurement, it is impossible to exclude electronic drift having occurred which might have contributed to this disparity in findings. However, subjects from each group were always measured at the same time, so that any drift would influence measurements in all groups to an equal extent. In both this study and our previous study, the difference in response between groups was consistent, with women taking up brisk walking showing more favourable changes in BUA relative to controls.

The study was designed to detect a difference in response between groups of 3% for BMD or 5% for BUA. The decrease in BMD in the Walk/Sed group and the increase in BUA in the Sed/Walk group were of this magnitude. However, any more modest effect of continuing brisk walking would not have been detected.

There was a significant increase in urinary DPD (a sensitive marker of bone resorption) in subjects who had acted as controls for both years, whilst similar changes in serum osteocalcin were not statistically significant. An increase in osteocalcin in controls but not exercisers has been reported previously [8,16]. The increase in DPD confirms that this was related to an increase in bone turnover, rather than just bone formation. These changes in biochemical markers are consistent with findings from a cross-sectional study [19]. Increases in markers of both formation and resorption are associated with greater bone loss after menopause [20], suggesting that the

exercise-induced changes in bone in this population could be mediated through prevention of an age-related increase in bone turnover.

Women who stopped walking did not show any significant changes in biochemical markers between baseline and 12 months, despite a significant reduction in BMD. There is evidence that brisk walking can produce some acute changes in bone metabolism in postmenopausal women: a bout of brisk walking has been reported to provoke changes in markers of collagen formation and degradation within several days [21]. Chronic changes in bone metabolism might thus be concealed by acute effects of exercise conducted in the days preceding measurement (although subjects avoided exercising for 24 h prior to sampling). Furthermore, biochemical markers are subject to circadian variation [22]. Although sampling time was carefully standardized, this factor could still have contributed to intra-subject variation and thus type II error.

Dietary calcium appears to potentiate the effect of physical activity on bone [23], with beneficial effects being reported at intakes above 1000 mg/day. The dietary calcium intake of women in the study, although typical for the wider population, was below this amount and might thus have limited their BMD response to physical activity.

Our findings showed that women commencing brisk walking for exercise experienced an increase in calcaneal BUA but not changes in BMD large enough to be detected. Subjects who continued walking for a second year did not show any significant further improvement in BMD or BUA, although their response might have been limited by their moderate dietary calcium intake or a reduction in habitual walking speed. A significant decrease in calcaneal BMD (but not BUA) occurred in women who ceased walking, consistent with previous findings that the benefits of exercise are not maintained when exercise ceases. Deoxypyridinoline increased in controls but not exercisers, suggesting that the influence of exercise on bone could be through alleviation of this increased bone resorption. The contribution of factors independent of bone density to the reduction in fracture risk associated with walking requires further research.

Acknowledgements. This research was funded by the National Osteoporosis Society, UK and the Arthritis and Rheumatism Council, UK, grant J0048. I. T. was supported by a visiting fellowship from the Department of Hygiene, Kanazawa Medical University. We are grateful to Dr David Pye, Sheila Black, Mandy Blaze, Catherine Flint and Rachel Vincent (Department of Medical Physics, Queen's Medical Centre, Nottingham, UK) for carrying out bone mineral density measurements by dual-energy X-ray absorptiometry.

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Received for publication 12 September 2000 Accepted in revised form 13 February 2001