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# Participation in road cycling vs running is associated with lower bone mineral density in men

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#### Abstract

The effects of regular non-weight-bearing (NWB) exercise on bone health are largely unknown. The objective of the study was to determine the effects of participation in NWB sports on bone health in adult male recreational athletes. Male cyclists (NWB; n = 27) and runners (weight-bearing [WB]; n = 16) aged 20 to 59 years were recruited from the community. Whole-body and regional bone mineral content and bone mineral density (BMD), and body composition were assessed using dual x-ray absorptiometry. Bone formation and resorption markers, and hormones were measured in serum. Bone-loading history was estimated from a sports participation history questionnaire. Nutrient intake and current physical activity were estimated from 7-day written logs. The NWB athletes had significantly lower BMD of the whole body and spine than the WB athletes, despite having similar age, weight, body mass index, body composition, hormonal status, current activity level, and nutrient intakes. Sixty-three percent of NWB athletes had osteopenia of the spine or hip, compared with 19% of WB athletes. Cyclists were 7 times more likely to have osteopenia of the spine than runners, controlling for age, body weight, and bone-loading history. There were no group differences in serum markers of bone turnover. Based on the results of this study, current bone loading is an important determinant of whole-body and lumbar spine BMD. Therefore, bone-loading activity should be sustained during adulthood to maintain bone mass.

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#### 1. Introduction

Osteoporosis affects more than 2 million men in the United States, and nearly 12 million more have osteopenia [1]. In addition, 85% to 90% of all hip and vertebral fractures in men occur in osteoporotic individuals. Furthermore, the number of fractures associated with osteopenia is nearly double that of osteoporotic fractures [2]. Nevertheless, osteopenia and osteoporosis in men often remain undiagnosed and inadequately treated. Unlike women who are often identified as osteopenic or osteoporotic via routine dual x-ray absorptiometry scans, men usually present with fragility fractures, back pain, or diminishing stature [3]. In addition, even after having a fracture, men are less likely to receive follow-up care and to be prescribed antiresorptive pharmacotherapy than women [4].

Risk factors for osteoporosis in men are similar to those identified in women: family history, age, low body weight, smoking, excessive alcohol consumption, inadequate calcium or vitamin D intake, low reproductive hormone levels, physical inactivity, and disease or medication affecting bone metabolism [5,6]. One might expect that men who participate in endurance sports, such as running and road cycling, would be at reduced risk for osteopenia and osteoporosis because of their healthful lifestyle and high levels of physical activity [7,8]. Surprisingly, the prevalence rates of osteopenia and osteoporosis are alarmingly high in adult male road cyclists [9-12], but not in distance runners [11].

Low body weight and weight loss have been associated with reduced bone mineral density (BMD) [13,14] due to reduced mechanical loading on the skeleton and hormonal changes associated with inadequate energy intake [15,16]. However, because runners and cyclists both have relatively low body weight compared with sedentary men [11], the discrepant BMD has been attributed to lack of ground reaction forces (GRF) on the skeleton during cycling [9,11,12].

The importance of mechanical stress in maintaining the balance between bone formation and resorption is evident

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in the rapid loss of bone that occurs during weightlessness and prolonged bed rest [17,18]. Road cycling has little osteogenic effect on bone because of the type of forces it exerts on the skeleton [18]. Based on longitudinal studies of bed rest [19,20] and space flight [20], one might expect bone resorption to be elevated in cyclists, at least transiently, and bone formation to be unchanged. However, the effects of cycling on rates of bone formation and resorption and, therefore, on the remodeling imbalance that leads to bone loss in cyclists remain to be identified. Therefore, the objectives of this study were to (1) determine the effects of participation in non-weight-bearing (NWB) vs weight-bearing (WB) sports on bone mineral content (BMC) and BMD of the whole body, hip, spine, and appendicular skeleton and (2) compare rates of bone turnover between male athletes in NWB (cycling) and WB (running) sports.

#### 2. Materials and methods

#### 2.1. Experimental subjects

The effects of participation in a WB vs NWB sport on BMC and BMD and on serum markers of bone turnover in adult male athletes were compared using a cross-sectional study design.

Forty-three male athletes in WB (running, n = 16) and NWB sports (cycling, n = 27) aged 20 to 59 years were recruited from the University of Missouri and Columbia community via flyers posted on campus, at local bicycle and sporting goods stores, and on Web sites of local cycling and running clubs. To be eligible for the study, participants had to perform a minimum of 6 hours per week of sport-specific training for at least the past 2 years. Exclusion criteria included current or previous medical condition or use of medication affecting bone health, implanted metal that would interfere with determination of BMD, and cigarette smoking. Before initial screening, all participants were informed of any risks associated with this study, they read a consent form, and they gave written consent. This study was conducted in accordance with the guidelines in the Declaration of Helsinki and was approved by the University of Missouri Health Sciences Institutional Review Board.

## 2.2. Anthropometric data

Participant weight was determined to the nearest 0.05 kg, height was determined to the nearest 0.5 cm, and the results were used to calculate body mass index (BMI) (in kilograms per square meter).

#### 2.3. Serum hormones and bone turnover markers

To control for diurnal variation in serum hormones and bone turnover markers, blood was drawn in the early morning (6:00 AM to 9:00 AM) after an overnight fast. Participants were asked to refrain from exercise during the 24 hours before the blood collection (15 mL) via an antecubital vein by a trained phlebotomist. Blood was dispensed into serum separator tubes and allowed to coagulate at room temperature or on ice according to assay protocols. The coagulated blood was centrifuged at 2000g for 15 minutes, and the serum was removed and frozen at -80°C. All hormone and bone turnover marker assessments were done in duplicate, and all assays were performed in a single run to eliminate interassay variability. The concentrations of total testosterone, sex hormone-binding globulin (SHBG), dehydroepiandrosterone, cortisol, and free triiodothyronine (fT3) in serum were determined using commercially available chemiluminescent immunoassays (Immulite 1000; Diagnostic Products, Los Angeles, CA; intraassay coefficient of variation [CV] <5%). The free androgen index (FAI) was calculated as (testosterone/SHBG) × 100. The concentration of total insulin-like growth factor I (IGF-I) was measured after IGF-I binding proteins were removed by acid precipitation using a commercially available enzyme-linked immunosorbent assay (ELISA) (Diagnostic Systems Laboratories, Webster, TX; intraassay CV was 2.7%). Serum estradiol also was measured using ELISA (Bio-Quant, San Diego, CA; intraassay CV was 10.5%).

Serum markers of bone formation and resorption can be used as indirect measures of bone remodeling. Markers of bone formation include bone–alkaline phosphatase (bone-AP) and osteocalcin (OC), which are secreted by osteoblasts during bone formation. C terminal telopeptide of type I collagen (CTX) is released when bone collagen is broken down during bone resorption. Serum OC, bone-AP, and CTX were measured by ELISA (Nordic Bioscience Diagnostics, Herlev, Denmark). Cross-reactivity of the anti–human bone-AP antibody is 3% to 8% with liver AP and 0.4% with intestinal bone-AP.

#### 2.4. Bone mineral content and density

Dual x-ray absorptiometry (Hologic Delphi A, Waltham, MA) was used to measure BMC and areal BMD at the lumbar spine, total hip, appendicular skeleton, and whole body. Areal BMD (in grams per square centimeter) was calculated from bone area (in square centimeters) and BMC (in grams). All dual x-ray absorptiometry scans were performed and analyzed by one investigator (PSH). The CVs for BMC and BMD were <1%. The World Health Organization definitions were used to categorize participants as having normal BMD (>-1.0 SD), osteopenia ( $\leq$ -1.0 SD, >-2.5 SD), or osteoporosis ( $\leq$ -2.5 SD) of the spine and hip [21].

#### 2.5. Questionnaires

Current physical activity was quantified using a 7-day written training log of activity type, duration, intensity, and frequency. The Compendium of Physical Activities was used to estimate daily energy expended during purposeful exercise [22]. Nutrient intake was assessed using 7-day written diet record. Food diaries, not including multivitamin supplements, were analyzed for energy and macroand micronutrient content (Food Processor 8.0; ESHA, Salem, OR).

Subjects completed a medical history questionnaire and the Historical Leisure Activity Questionnaire (HLAQ) [23]. The HLAQ was developed to measure historical leisure time physical activity across the life span and to relate prior activity to bone density in postmenopausal women. The original interviewer-administrated version of the HLAQ has been modified for self-administration with good reliability, that is, intraclass correlation coefficients of approximately 0.86 for lifetime vigorous-intensity activities [24]. The HLAO has been used to examine the relationship between lifetime WB activity and current bone density in adult men [9,25] and women [26]. In the present study, the HLAQ was used to assess participation in leisure time physical activity during 3 periods of the life span: adolescence (13-18 years), young adulthood (19-29 years), and adulthood (30-59 years). To enhance recall of past physical activity, participants were provided standardized verbal prompting by study personnel (PSH). Study personnel reviewed each subject's responses on the medical history and HLAQ to verify completeness and accuracy of the written history.

#### 2.6. Bone-loading history

Questionnaires that assess the effect of physical activity history on BMD must include information regarding activity type, frequency, duration, loading on bone, and developmental period during which the physical activity occurred [27]. Thus, bone-loading impact scores were calculated for adolescence, young adulthood, and adulthood (>30 years) using the responses provided in the HLAQ and biomechanical GRF for each activity, as described by Groothausen et al [28]. Based on the GRF, all reported activities were classified into 4 categories (0-3): 0 (GRF <1× body weight; eg, cycling, swimming), 1 (GRF between 1× and 2× body weight; eg, rowing, aquarobics), 2 (GRF between 2× and 4× body weight; eg, jogging), and 3 (GRF >4× body weight; eg, basketball, soccer, volleyball).

A bone-loading exposure (LOAD EXPOSURE) score was then calculated for each developmental period as the product of the frequency, duration, and classification score, that is, 0 to 3, for each activity. A lifetime cumulative boneloading exposure score was calculated as the sum of the LOAD EXPOSURE scores for adolescence, young adulthood, and adulthood. This method of quantifying bone loading is similar to those described by Dolan et al [29] and by Daly and Bass [25] in that GRF, frequency, and duration of each activity determine the score. Bone-load history quantified in this manner was positively associated with BMD in adult women [29] and cortical BMC in adult men [25]. An annualized bone-loading exposure score also was calculated for each developmental period and for lifetime cumulative exposure by dividing the LOAD EXPOSURE score for each period by the number of years in that period. The purpose of the annualized score was to allow

comparison of bone-loading exposure during adolescence, young adulthood, and adulthood. A bone-loading score for the prior 12 months also was calculated.

#### 2.7. Statistics

Descriptive statistics (means  $\pm$  SEM) were performed on demographic, anthropometric, nutrient intake, serum hormones, physical activity, and sports history variables. Outcome variables were normally distributed. Bone-loading scores were not normally distributed and, therefore, were transformed by taking the square root; means ± SEM of untransformed data are presented in the results. Paired t tests were used to test for significant differences between the WB and NWB groups. Multiple linear regression also was used to test for group differences (group, 0 = NWB and 1 = WB) in whole-body and regional BMC and BMD, controlling for age, body weight, and bone-loading history (LOAD EXPOSURE). In addition, multiple linear regression was used to test for group differences in serum markers of bone turnover, adjusting for whole-body BMC. Logistic regression was used to determine the effects of participation in cycling on likelihood of having osteopenia of the spine; body weight, age, and bone-loading history (LOAD EXPOSURE) were included as covariates in the model. Group means and least squared means were considered statistically different at P < .05, as determined by the protected least significant difference technique. Bivariate relationships between BMD, BMC, bone turnover markers, and serum hormones were evaluated using Pearson correlations and multiple linear regression to control for potential covariates (age, body weight) (P < .05).

# 3. Results

The WB and NWB athletes were, on average, the same age, height, weight, BMI, and body composition (Table 1). The age ranges for the cyclists (20-57 years) and runners (23-59 years) also were comparable. In addition, the WB and

Table 1
Participant characteristics

Turdelpuit characteristics			
	WB (n = 16)	NWB (n = 27)	
Age (y)	$39.8 \pm 2.4$	38.1 ± 2.5	
Anthropometry			
Weight (kg)	$74.0 \pm 1.4$	$76.6 \pm 1.4$	
Height (m)	$1.80\pm0.01$	$1.82\pm0.01$	
BMI $(kg/m^2)$	$22.9 \pm 0.4$	$23.1 \pm 0.3$	
Body fat (%)	$13.8 \pm 0.6$	$14.2 \pm 0.7$	
Lean body mass (kg)	$61.0 \pm 1.1$	$62.7\pm0.9$	
PA			
PA (h/wk)	$11.4 \pm 1.5$	$13.0 \pm 1.2$	
PA energy expenditure (kcal/d)	$1220\pm191$	$1481 \pm 119$	

Data are means  $\pm$  SEM. Physical activity energy expenditure was calculated using activity type, duration, intensity, frequency, and body weight. PA indicates physical activity.

Table 4

Table 5

 Table 2

 Bone-loading history of adult male athletes in WB and NWB sports

<b>C</b> ,		*
Life stage	WB	NWB
Adolescence (13-18 y)	n = 16	n = 27
LOAD EXPOSURE	$6502\pm2204$	$3516\pm795$
LOAD PER YEAR	$1060\pm370$	$600 \pm 131$
Young adulthood (19-29 y)	n = 16	n = 25
LOAD EXPOSURE	$5020 \pm 1431$	$4326\pm1989$
LOAD PER YEAR	$520 \pm 181$	$397 \pm 181$
Adulthood (29-60 y)	n = 14	n = 18
LOAD EXPOSURE	$6216 \pm 1432$	$5414\pm3369$
LOAD PER YEAR	$550 \pm 117$	$330\pm143$
Lifetime cumulative	n = 16	n = 27
LOAD EXPOSURE	$14400\pm164$	$6561 \pm 169$
LOAD PER YEAR	$592 \pm 11^{a}$	$270 \pm 5^{b}$
Current (prior 12 mo)	n = 16	n = 27
LOAD EXPOSURE	$547\pm94^{a}$	$105 \pm 27^{b}$

Data are means  $\pm$  SEM. Means with different letter superscripts are significantly different; P < .05. LOAD EXPOSURE: bone-loading exposure is the sum of the products of GRF classification scores × duration × frequency for each activity [29] (see Materials and methods for details).

NWB groups had equivalent physical activity levels when quantified as hours per week or as daily energy expenditure during training (Table 1). By design, the groups differed in their current bone-loading scores (Table 2). Lifetime cumulative bone-loading exposure and loading exposure scores during adolescence and young adulthood did not differ between groups (Table 2). However, annualized lifetime cumulative bone-loading exposure was significantly greater in the runners compared with cyclists (P = .05, Table 2). The nutrient intakes of the 2 groups were similar; however, the NWB athletes had greater intake of fat (Table 3). It is worth noting that neither group consumed the adequate intake for vitamin D (>5  $\mu$ g/d; Institute of Medicine).

Despite their similar stature, weight, and body composition, the WB athletes had significantly greater whole-body and lumbar spine BMD compared with the NWB athletes; BMC of the lumbar spine also was greater in the WB athletes (Table 4). The group differences in whole-body and lumbar spine BMD remained significant (P < .01) after controlling for age, body weight, and lifetime cumulative load exposure, as well as bone-loading history during adolescence and young adulthood (data not shown). Based on the WHO definition, 60% of NWB had osteopenia of the spine compared with 19% for WB athletes. Cyclists were 7.4

Table 3 Nutrient intakes of adult male athletes in WB and NWB sports

Daily nutrient intakes	WB (n = 14)	NWB (n = 22)
Energy (kcal)	$2520\pm205$	$3038 \pm 175$
Fat (g)	$77 \pm 7^{\mathrm{b}}$	$99\pm 6^{a}$
Carbohydrates (g)	$348 \pm 35$	$406 \pm 25$
Protein (g)	$104 \pm 7$	$117 \pm 8$
Vitamin D ( $\mu$ g)	$3.2 \pm 0.8$	$3.3 \pm 0.6$
Calcium (mg)	$1017\pm79$	$1255\pm102$

Data are means  $\pm$  SEM. Means with different letter superscripts are significantly different; P < .05.

Bone mineral content and areal BMD of adult male athletes in WB and NWB sports

	WB (n = 16)	NWB $(n = 27)$	P
BMC (g)			
Whole body	$2918 \pm 91$	$2795 \pm 50$	.21
Lumbar spine	$81 \pm 3^{a}$	$70 \pm 2^{b}$	.01
Hip	$45 \pm 1$	$42 \pm 1$	.26
Leg	$567 \pm 18$	$549 \pm 13$	.40
Arm	$216 \pm 7$	$214 \pm 5$	.72
Femoral neck	$5.2 \pm 0.2$	$5.0 \pm 0.2$	.36
Ward triangle	$0.83\pm0.05$	$0.82\pm0.04$	.88
BMD $(g/cm^2)$			
Whole body	$1.26\pm0.03^{a}$	$1.20\pm0.01^{\rm b}$	.04
Lumbar spine	$1.10 \pm 0.04^{\rm a}$	$0.99\pm0.02^{\rm b}$	.007
Hip	$1.07\pm0.03$	$1.01\pm0.02$	.09
Leg	$1.37\pm0.03$	$1.30\pm0.02$	.08
Arm	$0.89\pm0.02$	$0.86\pm0.01$	.10
Femoral neck	$0.90\pm0.03$	$0.87\pm0.02$	.52
Ward triangle	$0.74\pm0.04$	$0.71\pm0.03$	.56

Data are means  $\pm$  SEM. Means with different letter superscripts are significantly different; P < .05.

times more likely to have osteopenia of the spine than runners after adjusting for age, body weight, and boneloading history (regression equation not shown).

To characterize the nature of the bone loss, that is, excessive resorption vs suppressed formation, we measured bone turnover markers in serum. There were no differences in markers of formation (bone-AP: WB =  $5.5 \pm 1.1$ , NWB =  $7.0 \pm 1.0$  U/L; OC: WB =  $16.6 \pm 3.6$ , NWB =  $14.7 \pm 1.2 \mu g/$ L) or resorption (CTX: WB =  $0.67 \pm 0.08$ , NWB =  $0.63 \pm 0.07 \mu g/$ L). Correcting for whole-body BMC did not alter this finding (data not shown). There were no significant correlations between bone turnover markers and BMD at any site, controlling for age and body weight (data not shown).

There were no group differences in serum hormone concentrations except in fT3, which was lower in the NWB than the WB athletes (Table 5). Free T3 was positively

Hormone concentrations in serum of adult male athletes in WB and NWB sports

ITTE Sports			
Hormone	WB (n = 16)	NWB (n = 27)	Normal range
Testosterone (ng/dL)	$570 \pm 36$	$539 \pm 31$	260-1500
SHBG (µg/mL)	$13.2 \pm 0.9$	$12.4 \pm 0.9$	5-25
FAI	$55.4 \pm 5.4$	$59.3 \pm 5.2$	14-128
DHEA (µg/dL)	$182 \pm 12$	$222 \pm 21$	80-560
Estradiol (pg/mL)	$27.0\pm3.3$	$24.1 \pm 3.2$	<60
Cortisol (µg/dL)	$18.8\pm1.2$	$17.0\pm1.0$	5-25
fT <sub>3</sub> (pg/mL)	$2.9\pm0.1^{a}$	$2.6\pm0.1^{\mathrm{b}}$	2.0-6.0
IGF-I (ng/mL)	$161 \pm 10$	$153 \pm 15$	100-400
PTH (pg/mL)	$49.0\pm3.9$	$46.2\pm4.1$	10-65

Data are means  $\pm$  SEM. Means with different letter superscripts are significantly different; P < .05. Free androgen index = (testosterone/SHBG) \*100. Conversion factors for metric to SI units: testosterone, 0.0347; SHBG, 2.87; dehydroepiandrosterone, 3.47; cortisol, 27.59; T<sub>3</sub>, 0.0154; IGF-I, 0.131; parathyroid hormone, 1.0. DHEA indicates dehydroepiandrosterone; PTH, parathyroid hormone.

correlated with bone-AP (r = 0.449, P = .0025), whereas estradiol was negatively correlated with bone-AP (r = -0.302, P = .04). Testosterone (r = 0.422, P = .004), FAI (r = 0.386, P = .01), and cortisol (r = 0.443, P = .003) all were positively correlated with serum CTX. These significant relationships held after adjusting for age (data not shown). Serum hormone concentrations were not significantly associated with BMD at any site, adjusting for age and weight (data not shown).

#### 4. Discussion

The results of the present study are consistent with earlier studies documenting increased prevalence of osteopenia in adult male road cyclists [9-12]. By evaluating bone-loading history, we were able to control for the effects of prior activity on current BMD. We found that the cyclists had lower BMD compared with the runners, independent of lifetime cumulative bone loading and of bone loading during adolescence and young adulthood.

## 4.1. Bone turnover markers: WB vs NWB athletes

In the present study, NWB athletes had significantly reduced whole-body and lumbar spine BMD (-4% and -10%, respectively; P < .05) and marginal reductions at other sites (ie, leg, -5%; hip, -5%; arm, -4%; all P < .1). These differences do not appear to be due to differences in markers of bone turnover, as no differences were detected between runners and cyclists. Because serum markers reflect turnover of the entire skeleton, they may not be sensitive enough to detect differences that mostly are attributable to the lumbar spine, a site that accounted for approximately 2.5% of total BMC in our study participants. In addition, markers of bone formation and resorption respond to shortterm bouts of cycling [30,31] and running [32-34]. However, our study design allowed us to examine long-term, but not short-term, effects of participating in a WB vs NWB sport on bone turnover. Thus, it may be necessary to measure the short-term effects of exercise on bone turnover markers to detect differences associated with different types of activity. It also is possible that the magnitude of the bone turnover uncoupling is large initially and then decreases over time, as is seen during bed rest [19,35] and space flight [20]. Thus, it is plausible that a similar phenomenon may occur in men who spend a significant amount of time cycling in the unloaded condition.

#### 4.2. Hormones and bone turnover markers

Exercise-associated changes in bone turnover may be mediated by hormones in 2 ways. First, exercise per se may induce hormone synthesis and/or secretion [36,37]. Second, exercise may create an energy deficit, which, in turn, alters hormone secretion [13-16]. It has been suggested that bone loss associated with endurance exercise results from hormonally mediated suppression of bone turnover and acceleration of bone resorption. Thus, hormones known to affect bone turnover and that are induced by exercise and/or are sensitive to energy balance were measured to explore mechanisms of reduced BMD in cyclists compared with runners. We observed expected relationships between hormones and bone turnover markers: fT3 was positively correlated with bone-AP, whereas estradiol was negatively correlated with bone-AP; cortisol and total testosterone were positively associated with CTX. However, there were no significant differences between WB and NWB athletes, suggesting that, in this cross-sectional study, hormones and bone turnover markers were unrelated to differences in BMD.

#### 4.3. BMD and past and present bone loading

Bone loading during physical development significantly affects peak bone mass and vulnerability to osteoporosis [38] because the skeleton is more responsive to mechanical loading during periods of growth, that is, adolescence [7]. Because there may be residual effects of prior skeletal loading [28,39,40], especially during adolescence [7,29,40-42], on adult BMD, in the present study, bone-loading histories during adolescence and young adulthood were included as control variables when testing for group differences in BMD. Although group differences in developmental stage-specific and lifetime cumulative bone loading were not significant, the runners had tended to have higher scores than the cyclists across the life span (Table 2). Thus, it was important to control for bone-loading history when examining group differences in BMD. We found that WB athletes had significantly greater whole-body and lumbar spine BMD than NWB athletes, controlling for bone-loading history. Consistent with the present findings, Nichols et al [9] reported that WB exercise during adolescence and young adulthood had no apparent effect on BMD in adult male cyclists approximately 50 years of age. These results and those of the present study confirm the necessity of sustained skeletal loading to maintain gains in bone mass, as was demonstrated in a prospective study of adolescent male athletes and nonathlete controls [41]. Similarly, Van Langendonck et al [43], in a longitudinal study following male subjects from age 13 to 40 years, found that participation in high-impact sports during adolescence (13-18 years) had a beneficial effect on BMD of the lumbar spine, with additional benefit of continued participation in high-impact sports throughout adulthood (ages 19-39 years).

The primary limitation of this study is the cross-sectional design. Although we attempted to control for differences in bone-loading history between groups, WB and NWB athletes may have differed in BMC and BMD before taking up running or cycling. Furthermore, because serum markers of bone formation and hormones were measured at a single time point, we were unable to assess changes in bone turnover during the dynamic phase of bone loss that occurs before stabilization at a new steady state. A strength of this study is the thorough characterization of the participants, which demonstrated that cyclists and runners were very similar to each other regarding current physical activity level, nutrient intake, and body composition. Another strength of this study is the assessment of bone turnover markers and hormones, which demonstrated that bone turnover and hormonal status are not chronically altered by participation in a NWB sport.

#### 4.4. Summary

In summary, we found that adult male cyclists had significantly lower BMD of the whole body, especially of the lumbar spine, compared with runners. Moreover, more than 60% of the cyclists had osteopenia of the spine and were 7 times more likely to have osteopenia of the spine than the runners. This striking difference in bone health between the cyclists and runners could not be attributed to differences in age, body weight, body composition, diet, hormonal status, overall activity level, or bone-loading history. Low bone density affects millions of men in the United States today, causing significant morbidity and mortality. Based on the results of this study, current bone loading is an important determinant of whole-body and lumbar spine BMD; and bone-loading activities such as running or jogging should be sustained throughout life to maintain bone mass.

#### References

- NOF 2002 American's bone health: the state of osteoporosis and low bone mass in our nation. National Osteoporosis Foundation, Washington, DC.
- [2] Pasco JA, Seeman E, Henry MJ, Merriman EN, Nicholson GC, Kotowicz MA. The population burden of fractures originates in women with osteopenia, not osteoporosis. Osteoporos Int 2006;17:1404-9.
- [3] Wright VJ. Osteoporosis in men. J Am Acad Orthop Surg 2006;14: 347-53.
- [4] Kiebzak GM, Beinart GA, Perser K, Ambrose CG, Siff SJ, Heggeness MH. Undertreatment of osteoporosis in men with hip fracture. Arch Intern Med 2002;162:2217-22.
- [5] Bakhireva LN, Barrett-Connor E, Kritz-Silverstein D, Morton DJ. Modifiable predictors of bone loss in older men: a prospective study. Am J Prev Med 2004;26:436-42.
- [6] Olszynski WP, Shawn Davison K, Adachi JD, et al. Osteoporosis in men: epidemiology, diagnosis, prevention, and treatment. Clin Ther 2004;26:15-28.
- [7] Lorentzon M, Mellstrom D, Ohlsson C. Age of attainment of peak bone mass is site specific in Swedish men—the GOOD study. J Bone Miner Res 2005;20:1223-7.
- [8] Michaelsson K, Olofsson H, Jensevik K, et al. Leisure physical activity and the risk of fracture in men. PLoS Med 2007;4:e199.
- [9] Nichols JF, Palmer JE, Levy SS. Low bone mineral density in highly trained male master cyclists. Osteoporos Int 2003;14:644-9.
- [10] Sabo D, Bernd L, Pfeil J, Reiter A. Bone quality in the lumbar spine in high-performance athletes. Eur Spine J 1996;5:258-63.
- [11] Stewart AD, Hannan J. Total and regional bone density in male runners, cyclists, and controls. Med Sci Sports Exerc 2000;32:1373-7.
- [12] Warner SE, Shaw JM, Dalsky GP. Bone mineral density of competitive male mountain and road cyclists. Bone 2002;30:281-6.
- [13] Prouteau S, Pelle A, Collomp K, Benhamou L, Courteix D. Bone density in elite judoists and effects of weight cycling on bone metabolic balance. Med Sci Sports Exerc 2006;38:694-700.

- [14] Prouteau S, Benhamou L, Courteix D. Relationships between serum leptin and bone markers during stable weight, weight reduction and weight regain in male and female judoists. Eur J Endocrinol 2006;154: 389-95.
- [15] Zanker CL, Swaine IL. Responses of bone turnover markers to repeated endurance running in humans under conditions of energy balance or energy restriction. Eur J Appl Physiol 2000;83:434-40.
- [16] Ihle R, Loucks AB. Dose-response relationships between energy availability and bone turnover in young exercising women. J Bone Miner Res 2004;19:1231-40.
- [17] Bikle DD, Halloran BP. The response of bone to unloading. J Bone Miner Metab 1999;17:233-44.
- [18] Woodward MI, Cunningham JL. Skeletal accelerations measured during different exercises. Proc Inst Mech Eng [H] 1993;207:79-85.
- [19] Smith SM, Nillen JL, Leblanc A, et al. Collagen cross-link excretion during space flight and bed rest. J Clin Endocrinol Metab 1998;83: 3584-91.
- [20] Smith SM, Zwart SR, Block G, Rice BL, Davis-Street JE. The nutritional status of astronauts is altered after long-term space flight aboard the International Space Station. J Nutr 2005;135:437-43.
- [21] Kanis JA, Melton III LJ, Christiansen C, Johnston CC, Khaltaev N. The diagnosis of osteoporosis. J Bone Miner Res 1994;9:1137-41.
- [22] Ainsworth BE, Haskell WL, Whitt MC, et al. Compendium of physical activities: an update of activity codes and MET intensities. Med Sci Sports Exerc 2000;32:S498-504.
- [23] Kriska AM, Sandler RB, Cauley JA, LaPorte RE, Hom DL, Pambianco G. The assessment of historical physical activity and its relation to adult bone parameters. Am J Epidemiol 1988;127:1053-63.
- [24] Chasan-Taber L, Erickson JB, McBride JW, Nasca PC, Chasan-Taber S, Freedson PS. Reproducibility of a self-administered lifetime physical activity questionnaire among female college alumnae. Am J Epidemiol 2002;155:282-9.
- [25] Daly RM, Bass SL. Lifetime sport and leisure activity participation is associated with greater bone size, quality and strength in older men. Osteoporos Int 2006;17:1258-67.
- [26] Rideout CA, McKay HA, Barr SI. Self-reported lifetime physical activity and areal bone mineral density in healthy postmenopausal women: the importance of teenage activity. Calcif Tissue Int 2006;79: 214-22.
- [27] Ainsworth BE, Shaw JM, Hueglin S. Methodology of activity surveys to estimate mechanical loading on bones in humans. Bone 2002;30: 787-91.
- [28] Groothausen JSH, Kemper HCG, Twisk JWR, Welten DC. Influence of peak strain on lumbar bone mineral density: an analysis of 15-year physical activity in young males and females. Pediatr Exerc Sci 1997; 9:159-73.
- [29] Dolan SH, Williams DP, Ainsworth BE, Shaw JM. Development and reproducibility of the bone loading history questionnaire. Med Sci Sports Exerc 2006;38:1121-31.
- [30] Maimoun L, Manetta J, Couret I, et al. The intensity level of physical exercise and the bone metabolism response. Int J Sports Med 2006;27: 105-11.
- [31] Guillemant J, Accarie C, Peres G, Guillemant S. Acute effects of an oral calcium load on markers of bone metabolism during endurance cycling exercise in male athletes. Calcif Tissue Int 2004;74:407-14.
- [32] 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.
- [33] Brahm H, Piehl-Aulin K, Ljunghall S. Bone metabolism during exercise and recovery: the influence of plasma volume and physical fitness. Calcif Tissue Int 1997;61:192-8.
- [34] Crespo R, Revilla M, Villa LF, Usabiaga J, Leibar X, Rico H. Transient dissociation of bone metabolism induced by high performance exercise: a study in elite marathon runners. Calcif Tissue Int 1999; 64:287-90.
- [35] Zerwekh JE, Ruml LA, Gottschalk F, Pak CY. The effects of twelve weeks of bed rest on bone histology, biochemical markers of bone

turnover, and calcium homeostasis in eleven normal subjects. J Bone Miner Res 1998;13:1594-601.

- [36] Ehrnborg C, Lange KH, Dall R, et al. The growth hormone/insulin-like growth factor–I axis hormones and bone markers in elite athletes in response to a maximum exercise test. J Clin Endocrinol Metab 2003; 88:394-401.
- [37] Kraemer WJ, Ratamess NA. Hormonal responses and adaptations to resistance exercise and training. Sports Med 2005;35:339-61.
- [38] Rizzoli R, Bonjour JP. Determinants of peak bone mass and mechanisms of bone loss. Osteoporos Int 1999;9(Suppl 2):S17-23.
- [39] Kontulainen S, Heinonen A, Kannus P, Pasanen M, Sievanen H, Vuori I. Former exercisers of an 18-month intervention display residual aBMD benefits compared with control women 3.5 years postintervention: a follow-up of a randomized controlled high-impact trial. Osteoporos Int 2004;15:248-51.
- [40] Zanker CL, Osborne C, Cooke CB, Oldroyd B, Truscott JG. Bone density, body composition and menstrual history of sedentary female former gymnasts, aged 20-32 years. Osteoporos Int 2004;15: 145-54.
- [41] Nordstrom A, Olsson T, Nordstrom P. Sustained benefits from previous physical activity on bone mineral density in males. J Clin Endocrinol Metab 2006;91:2600-4.
- [42] Kemper HC, Bakker I, Twisk JW, van Mechelen W. Validation of a physical activity questionnaire to measure the effect of mechanical strain on bone mass. Bone 2002;30:799-804.
- [43] Van Langendonck LLJ, Claessens AL, Thomis M, Philippaerts R, Delvaux K, Lysens R, et al. Influence of participation in high-impact sports during adolescence and adulthood on bone mineral density in middle-aged men: a 27-year follow-up study. Am J Epidemiol 2003; 158:525-33.