Published in final edited form as:
Clin J Sport Med. 2014 July ; 24(4): 331–336. doi:10.1097/JSM.0000000000000050.

Bone loss over one year of training and competition in female cyclists

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Abstract

Objective—To observe changes in hip, spine, and tibia bone characteristics in female cyclists over the course of 1 year of training.

Design—Prospective observational study

Setting—Laboratory

Participants—Female cyclists (n=14) aged 26-41 years with at least 1 year of competition history and intent to compete in 10 or more races in the coming year.

Assessment of Risk Factors—Women who train and compete in road cycling as their primary sport.

Main Outcome Measures—Total body fat-free and fat mass, and lumbar spine and proximal femur areal bone mineral density (aBMD) and bone mineral content (BMC) assessments by DXA. Volumetric BMD (vBMD) and BMC of the tibia were measured by pQCT at sites corresponding to 4%, 38%, 66%, and 96% of tibia length. Time points were baseline and after 12 months of training and competition.

Results—Weight and body composition did not change significantly over 12 months. Total hip aBMD and BMC decreased by −1.4±1.9% and −2.1±2.3% ($p<0.02$), subtrochanter aBMD and BMC decreased by −2.1±2.0% and −3.3±3.7% ($p<0.01$). There was a significant decrease in lumbar spine BMC (−1.1±1.9%; $p=0.03$). There were no significant bone changes in the tibia ($p>0.11$).

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The authors declare no conflicts of interest.

None of the authors have any conflicts of interest to disclose.

Conclusions—Bone loss in female cyclists was site-specific and similar in magnitude to losses previously reported in male cyclists. Research is needed to understand the mechanisms for bone loss in cyclists.

Keywords

Cycling; BMD; pQCT

Introduction

Bone-loading exercise interventions typically result in an increase (or attenuation of loss) in bone mineral density (BMD)[1-6]. Although cycling is a weight-supported activity, it can generate high muscle forces that could have favorable effects on bone. However, male cyclists from adolescence through 60 years of age have been reported to have low hip and lumbar spine areal BMD (aBMD) values when compared with runners or nonathletic controls[7-13] and prospective studies of adult male (27-44 y) and female (>35 y) cyclists have found annual decreases in aBMD that are comparable to the accelerated rates of loss that occur in postmenopausal women (~1.5%/year)[14,15]. The decrease in aBMD in cyclists may have important implications for long-term bone health, particularly if it persists over many years of training. Trabecular and cortical bone tissue have different relative contributions to whole bone strength[16], but the tissue-specificity of bone loss in cyclists is currently unknown.

Moderate- and high-intensity cycling bouts result in a decrease in serum calcium and increases in parathyroid hormone (PTH) and bone resorption[17,18]. These metabolic factors may influence bone metabolism in cyclists. To our knowledge, there has been only one prospective study of changes in aBMD in female cyclists[15], who may be particularly at risk for developing osteoporosis based on previous reports of reduced aBMD in young female athletes in weight-sensitive sports[19]. The purpose of this study was to measure changes in proximal femur, lumbar spine, and tibia bone characteristics in female cyclists over 1 year of training and competition. We hypothesized that total hip and lumbar spine aBMD would decrease at least 1% over 12 months of training and competition, as observed in men[14]. Exploratory aims to generate hypotheses for future studies were to evaluate 1) changes in trabecular and cortical bone, and 2) whether hormonal contraceptive use helps to prevent a decline in bone mass.

Methods

Participants

Premenopausal female cyclists aged 18-45 years with at least 1 year of competition history and intent to compete in 10 or more races in the coming year participated in the study. They were recruited from the greater Denver metro area through fliers and postings on cycling racing websites from November 2009 through February 2010. Women who were triathletes or did not consider road cycling to be their primary sport were excluded. Exclusion criteria included pregnancy or plans to become pregnant, hysterectomy, thyroid stimulating hormone level of < 0.5 or > 5.0 mU/L, calculated creatinine clearance < 50 mL/min, alkaline

phosphatase level > 1.5 times the upper limit of normal, PTH > 69 pg/mL, 25-hydroxyvitamin D < 20 ng/mL, hypercalcuria determined by spot urine calcium-to-creatinine ratio > 0.30, and use of drugs known to influence bone metabolism (e.g., oral steroids, bisphosphonates, teriparatide, calcitonin). Use of hormonal contraceptives was allowed. All participants provided written informed consent to participate and the study was approved by the Colorado Multiple Institutional Review Board.

Participants provided information about their menstrual cycle history, hormonal contraceptive use, cycling history (road and off-road), and participation in other forms of exercise at the beginning of the study. Participants were asked to record their racing and training activity (cycling and other exercise) over the 12 months of observation. A food frequency questionnaire was used to estimate daily calcium intake from dairy sources, calcium-fortified foods and juices, and supplements.

Musculoskeletal Assessments

Dual-energy X-ray Absorptiometry (DXA)

aBMD and BMC of the lumbar spine (L1-L4), total hip, femoral neck, trochanter, and subtrochanter (also known as intertrochanter) regions of the hip were measured at baseline (January-February) and month 12 (December-January) on a Discovery W DXA instrument (Hologic Inc, Waltham, Massachusetts). T-scores are reported for the lumbar spine, total hip, trochanter, and femoral neck. Fat-free mass (FFM; kg), fat mass (FM; kg), and relative adiposity (%) were obtained from the total body scan. The *in vivo* precision (coefficient of variation; CV) of aBMD ranges from 0.7% to 1.6% for sites of interest sites (lumbar spine, 0.9%; total hip, 0.7%; femoral neck, 1.6%; trochanter, 0.8%; subtrochanter, 1.2%). The same experienced technician reviewed all scans.

Peripheral Quantitative Computed Tomography (pQCT)

At baseline and month 12, participants had their nondominant tibia scanned at 4%, 38%, 66%, and 96% of the tibia length from distal to proximal using pQCT (XCT 3000 with software version 6.00; Stratec Medizintechnik GmbH, Pforzheim, Germany). The 4, 38, 66 series is a commonly used series, and the 96% site served as a trabecular-rich proximal tibia site[20-23]. Scans were performed at 20 mm/sec with a 0.4 mm voxel, and a 2.4 mm slice thickness. Locations for measurement were determined by measuring the length from the tibial plateau to the medial malleolus. A reference line was placed at the distal end of the tibia using a scout view. The same tibia length was used for baseline and follow-up testing; scout views were compared for consistent placement of reference lines. Parameters assessed included: trabecular BMC and volumetric BMD (vBMD) at 4%; cortical BMC and vBMD at 38% and 66%; total BMC and vBMD at 96%.

Scans were analyzed with the Stratec software. The threshold to define the outer bone contour was 169 mg/cm^3 at the 4% and 96% sites and 710 mg/cm^3 at the diaphyseal sites. The threshold to separate cortical from trabecular bone was 650 mg/cm^3 at the 4% site and 710 mg/cm^3 at the diaphyseal sites. CVs range from 0.2-1.3% for total bone parameters, 0.2-0.6% for cortical bone parameters, and 0.4-1.6% for trabecular bone variables. A quality control cone phantom was scanned daily. An experienced technician reviewed all scans.

Statistical Analysis

Statistical analyses were performed using SAS version 9.3 (SAS Institute Inc., Cary, North Carolina). In male cyclists[14], total hip and lumbar spine BMD declined by 1.5 ± 2.1% and 1.0 ± 1.2% over 12 months. The estimated power to detect changes of these magnitudes for a sample size of 14 (alpha = 0.05) is 70% for the hip and 82% for the spine. Paired t-tests were used to detect significant changes from baseline to 12 months in body composition and bone characteristics. Pearson's correlation coefficients (r) were used to determine the association of cycling-specific and total training volumes with baseline bone values and changes over 12 months. Because the interactive effects of exercise and hormonal contraceptives have not been well studied, descriptive statistics showing changes in bone values in hormonal contraceptive users (n=9) and nonusers (n=5) were generated to guide future studies. The level of statistical significance was $p<0.05$. Data are presented as mean ± SD unless otherwise specified.

Results

Seventeen women were enrolled in the study, but 3 were lost to follow-up. All participants self-identified as Non-Hispanic Caucasian.

Participant characteristics

Years of road cycling experience ranged from 1.5 to 18 (8.9±4.7) and years of road racing ranged from 1.5 to 13 (5.3±3.9). Age of menarche ranged from 11 to 15 years. Nine women were using hormonal contraceptives. Duration of use was > 1 year and the hormone regimen was IUD (n=1), NuvaRing (n=2), or oral contraceptives (OC) (n=6). Two additional women reported stopping OC use in the 6 months before the baseline assessments. At baseline, two women reported an absence of menses > 90 days (n=1 IUD, n=1 OC user) and one OC user on a regimen to intentionally reduce the number of cycles had an average cycle length of 90 days. One woman had a BMI less than 18.5 kg/m^2 and was eumenorrheic. Estimated calcium intake ranged from 350 to 3653 mg/d at baseline, but was not assessed during the observation period. Over the 12 months of observation, changes in body mass, FM, and FFM were 0.2±2.1 kg (p=0.68), −0.2±1.9 kg (p=0.76), and 0.4±0.8 kg (p=0.10).

At baseline, the prevalence of low bone mass (i.e., T-score ≤ −1.0) was 35% at the lumbar spine (T-score range: −1.1, −2.2), 14% at the femoral neck (T-scores: −1.3, −1.4), and 7% total hip (T-score = −1.1). No participants had BMD levels at any region that met the criterion for osteoporosis (i.e., T-score ≤ −2.5). Participants without regular menses had normal bone mass (i.e., T-score > −1.0) at all sites. Participants with low bone mass at the lumbar spine were eumenorrheic; one was a current OC user. BMI for participants with low bone mass at any site ranged from 17.0 to 22.1 kg/m^2 and relative body fat content ranged from 17.2% to 26.2% of body weight.

Changes over the period of observation

Cycling was the primary mode of training and averaged 9.6 ± 4.8 hours per week. Most also reported running (11/14) and weight lifting (12/14) (Table 2). Road cycling was the primary form of racing, but 6 women also completed at least 1 mountain biking race and 9 completed

at least one cyclocross race. The volume of training and the number of races during the year of observation were similar between OC users and nonusers (Table 2).

Total hip and subtrochanteric aBMD (% change: −1.4±1.9% and −2.1±2.0%) and BMC (% change: −2.1±2.3% and −3.3±3.7%) decreased (all $p<0.02$) during the observation period (Table 3). Use of hormonal contraception may have influenced the changes in total hip (Users: −2.3±1.2%; Nonusers: 0.2±2.0%) and subtrochanteric (Users: −3.0±0.4%; Nonusers: −0.4±2.0%) aBMD. There was a significant decrease in lumbar spine BMC (−1.1±1.9%, $p=0.03$) but not aBMD (0.09±1.7%, $p=0.65$). The decline in BMC but not aBMD reflected a decrease in bone area (data not shown). Use of hormonal contraception may have influenced the changes in aBMD (Users: −0.7±1.2%; Nonusers: 1.1±1.9%) and BMC (Users: −2.0±1.0%; Nonusers: 0.4±2.1%). Neither baseline values nor changes in aBMD were correlated with training volumes or number of races (all $p>0.17$). Changes in hip aBMD were not related to changes in body weight, FFM, or FM (all $p>0.13$).

There was wide variability in change in vBMD and BMC at the trabecular rich tibial sites and changes were not significant (Table 4). There were also no significant changes in vBMD or BMC at the 38% and 66% sites. Training volume from non-cycling aerobic activities (e.g. running, cross-country skiing) was correlated with baseline trabecular vBMD (0.78, $p<0.001$) at the 4% site. Changes in pQCT-derived bone values were not significantly correlated with cycling-specific or total training volumes or number of races (all $p>0.40$).

Discussion

The primary finding of this prospective observational study of female cyclists was that bone loss occurred at the lumbar spine, total hip, and subtrochanteric region of the hip, but there were no significant trabecular or cortical bone changes in the tibia over 12 months of training. There was considerable variability in the magnitudes of bone changes. The changes were not related to training volume, but may have been influenced by use of hormonal contraception. The average decline in total hip aBMD of −1.4% over a year was similar to the decline of −1.5% over 1 year in male road cyclists[14]. The maintenance of lumbar spine aBMD in the current study was not consistent with the trend for a decline (−1%) in male cyclists[14] or the significant decline (−2.3%) in master female cyclists over an 18-month interval[15]. The decrease in the latter study may have been related to the longer period of observation or the older age of the cyclists. The decrease in spine aBMD in cyclists was similar to that of sedentary controls, but runners had an attenuated rate of decline[15].

Assessing bone changes in female cyclists is important because bone loss during premenopausal years, coupled with accelerated bone loss during and after menopause, may increase fracture risk. Endurance athletes and women participating in sports where leanness is emphasized for performance tend to be at greater risk for bone quality impairments than other athletes[24]. Although leanness is a common characteristic in competitive cyclists,[25-29] the bone status of female cyclists has not been well characterized. Studies have been predominantly cross-sectional comparisons of male cyclists with other groups of athletes or non-athletes. In such studies, proximal femur and lumbar spine aBMD have been 3.3% to 17.7% lower in road cyclists than in age-matched runners or untrained controls[7,8,10,11,13],

despite cyclists having greater lean mass in some studies[7,10,11]. Differences persisted when adjusted for lower body mass[8]. Differences in aBMD and BMC between cyclists and controls aged 17-21 years were larger than the differences between cyclists and controls under 17 years of age[12]. Master cyclists have also been found to have lower hip and spine aBMD values than young adult cyclists or age-matched controls[11]. Studies with longer follow-up are needed to determine if these observations reflect a progressive decline in aBMD in cyclists that exceeds the expected age-related decline[30].

The site differences in bone changes in the current study may reflect differences in loading forces that act on the femur, spine and tibia during cycling. Although hip moments and activation of hip extensors have a high relative contribution to power output during cycling[31,32], some of the muscle actions that load the proximal femur (hip abduction, lateral rotation) are limited during cycling[33]. During seated cycling, trunk muscle activity may not be of sufficient intensity to generate increases in BMD at the lumbar spine and proximal femur, but should be sufficient to maintain BMD[34]. Further, when cycling off the seat, trunk muscle activity would be expected to increase considerably[35]. Strains experienced by the anterior tibia during cycling have been measured directly and range from 271-628 με, which are lower than activities that generate ground-reaction forces[36]. Power output and pedaling rates may affect site-specific responses to riding because of differing rates of muscle force production related to cadence[37-39].

Some of the cyclists in the current study also engaged in regular off-road cycling, resistance training, and running. The variety of bone-loading modalities makes it difficult to isolate the effects of road cycling on bone. Inherent differences in terrain between onand off-road cycling, coupled with the previous observation that mountain bikers have higher hip and spine aBMD than road cyclists and controls, suggest that off-road cycling may provide a different stimulus to bone than road cycling[9,40]. Male master cyclists who also participated in resistance training or high-impact exercises had a smaller decrease in hip aBMD and a larger increase in spine aBMD over 7 years than those who did not[41]. Similarly, sprint cyclists who engaged in resistance training had a greater tibia section modulus than those who did not[42]. Resistance training is a recommended means of increasing BMD and bone strength because of the large joint-reaction forces that can be generated[3]. Skeletal adaptations to running appear to depend on the intensity of the resulting ground-reaction forces, which increase directly with running speed[39]. Participation in both of these activities may have helped to preserve bone in the spine, femur, and tibia by increasing mechanical loads at those sites.

The effects of cycle training on the coupling or intensity of resorption and formation are not known. The loss of BMD in cyclists in the current study and others[14,15,41] may have been related to increased rates of bone turnover, which can be influenced by both the mechanical and metabolic characteristics of the activity. Although cycling can generate high muscle forces to stimulate bone modeling[31,35], as a weight-supported activity it may not be as effective in doing so as weight-bearing activities. In this context, the ‘anabolic stimulus’ to bone during cycling may be only modest. However, this would not be expected to trigger bone loss. It seems plausible that bone loss in cyclists is the result of an increase in bone resorption that is not effectively coupled with an increase in formation.

One possible mechanism for the activation of bone resorption during cycling is the disruption of serum calcium homeostasis. The loss of calcium through dermal and other sources during exercise can cause a decrease in serum ionized calcium and an increase in PTH, which defends against a decline in serum calcium by increasing calcium absorption, reducing calcium excretion, and stimulating bone resorption to mobilize skeletal calcium. It has been demonstrated that 2 hours of cycling at a moderate intensity and 1 hour of cycling at a vigorous intensity both caused a decline in serum calcium, a marked increase (71-138%) in PTH, and an increase (16-38%) in C-terminal telopeptide of type 1 collagen (CTX), a serum marker of bone resorption[17,18]. Shorter, higher intensity bouts of exercise also elicit an increase in PTH[17,43-45]. The ingestion of calcium before and during cycling blunts the increases in PTH[17,43] and CTX[43]. Further research is needed to determine whether these responses to acute cycling exercise are important determinants of skeletal adaptations to training.

The impact of hormonal contraceptive use on skeletal adaptations to exercise has not been well studied. If female cyclists practice energy restriction to maintain a low body weight, this can result in reproductive dysfunction and a decline in BMD[46-48]. In this context, if the cause of bone loss in cyclists is a decline in sex hormones, it might be expected that use of hormonal contraceptives would protect against loss. That did not appear to be the case in the current study, but the small size prevented an evaluation of whether contraceptive use influenced changes over time. The mechanisms by which hormonal contraceptives would exaggerate bone loss in female cyclists are not clear, but may be related to suppression of endogenous estradiol by ethinyl estradiol, which is the type of estrogen in most formulations of contraceptives. Further studies of the effects of hormonal contraceptives on skeletal adaptations to exercise are warranted.

The current study had limitations that should be acknowledged. The study was not powered to detect changes in pQCT variables or to evaluate differences between hormonal contraceptive users and non-users. These exploratory analyses were carried out to help generate hypotheses for future studies. As a prospective observational study, there was no control over factors that may have influenced bone metabolism, such as exercise intensity, macro- or micronutrient intake, or energy expenditure. Low energy availability has been associated with decreased LH pulsatility and increased bone resorption[47,48]. The women in our sample did not have a high prevalence of overt consequences of low energy availability (i.e., oligomenorrhea, amenorrhea), but the possibility that subtle disruptions in reproductive function influenced the findings cannot be ruled out. Although mean body weight did not change over the year of observation, there may have been intervals when energy availability was insufficient or when short-term decreases in body weight occurred. Such events could have contributed to the observed bone loss.

Conclusions

This prospective observational study of competitive female cyclists corroborated the growing evidence from studies of competitive male cyclists[14,41] that hip aBMD declines by 1% to 2% over a year of training and competition. Further research will be necessary to determine the mechanisms of bone loss in cyclists.

Acknowledgments

This study was supported by NIH Colorado CTSI award UL1 RR025780, NIH Nutrition and Obesity Research Center award P30 DK048520, NIH R01 AG018857, NIH F32 AG035460, NIH T32 DK007658. Contents are the authors' sole responsibility and do not necessarily represent official NIH views.

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Table 1

Baseline characteristics.

	Mean ± SD	Range
Age, y	34.9 ± 4.8	26.0, 41.0
Height, m	1.69 ± 0.07	1.56, 1.83
Weight, kg	59.1 ± 7.1	47.7, 70.3
BMI, kg/m^2	20.8 ± 1.9	17.0, 24.0
Bone-free fat-free mass, kg	45.1 ± 5.7	36.5, 55.3
Fat mass, kg	11.8 ± 3.1	7.3, 16.5
Body fat %	19.8 ± 4.3	13.4, 26.2
Cycling experience, y	8.9 ± 4.7	1.5, 18.0
Racing experience, y	5.3 ± 3.9	1.5, 13.0

Table 2

Training and competition characteristics of all participants and by hormonal contraception use during the year of observation.

	All		Users		Nonusers	
Training	**N, Mean ± SD**	**Range**	**N, Mean ± SD**	**Range**	**N, Mean ± SD**	**Range**
Months Cycling	14, 11.3 ± 1.2	8, 12	9, 11.0 ± 1.4	8, 12	5, 11.8 ± 0.4	11, 12
Hours/Week Cycling	14,9.6 ± 4.8	2, 20	9, 9.6 ± 4.1	2, 15	5, 9.8 ± 6.4	4, 20
Months Running	11,7.9 ± 3.9	1, 12	7, 6.6 ± 4.2	1, 12	4, 10.3 ± 2.1	8, 12
Hours/Week Running	11, 1.4 ± 0.6	0.5, 2.5	7, 1.4 ± 0.5	0.8, 2	4, 1.4 ± 0.9	0.5, 2.5
Months Lifting	12,8.8 ± 3.6	4, 12	7, 9.1 ± 3.8	4, 12	5, 8.2 ± 3.8	4, 12
Sessions/Week Lifting	12, 1.5 ± 0.7	1, 3	7, 1.3 ± 0.5	1, 2	5, 1.8 ± 0.8	1, 3
Road Races	14, 26.1 ± 20.7	1, 70	9, 26.8 ± 22.3	1, 70	5, 25.0 ± 9.8	12, 60
Mountain Bike Races	6, 2.2 ± 1.6	1, 5	5, 2.4 ± 1.7	1, 5	1, 1	1
Cyclocross Races	9, 10.3 ± 7.9	2, 21	6, 10.0 ± 7.9	3, 20	3, 11.0 ± 9.5	2, 21
Total Races	14, 33.7 ± 23.1	3, 91	9, 34.8 ± 24.8	7, 91	5, 31.8 ± 22.3	16, 70

N values refer to the number of cyclists that reported running, lifting, or competing in mountain bike or cyclocross races. Range includes only participants who reported participating in the activity.

Table 3

Baseline, 12-month, and change in lumbar spine and proximal femur aBMD (g/cm^2), and BMC (mg). (Mean ± SD)

Variable	Baseline	12 Months	Mean Change (95% Confidence Interval)	P value
Lumbar Spine				
aBMD	1.024 ± 0.176	1.021 ± 0.169	−0.002 (−0.012, 0.008)	0.65
BMC	62.88 ± 14.16	62.12 ± 13.69	−0.76 (−1.44, 0.08)	0.03
Total Hip				
aBMD	0.975 ± 0.115	0.961 ± 0.109	−0.014 (−0.026, −0.003)	0.02
BMC	34.35 ± 6.25	33.66 ± 6.33	−0.69 (−1.13, −0.25)	0.005
Femoral Neck				
aBMD	0.829 ± 0.099	0.827 ± 0.101	−0.002 (−0.018, 0.014)	0.81
BMC	4.22 ± 0.72	4.27 ± 0.77	0.05 (−0.06, 0.17)	0.35
Trochanter				
aBMD	0.734 ± 0.098	0.736 ± 0.098	0.002 (−0.011, 0.014)	0.79
BMC	8.22 ± 1.61	8.18 ± 1.82	0.03 (−0.37, 0.30)	0.83
Subtrochanter				
aBMD	1.158 ± 0.133	1.133 ± 0.124	−0.025 (−0.039, −0.011)	0.002
BMC	21.92 ± 4.16	21.21 ± 4.10	−0.71 (−1.15, −0.27)	0.004

aBMD: Areal Bone Mineral Density; BMC: Bone Mineral Content.

Table 4

Baseline, 12-month, and change in tibia vBMD and BMC. (Mean ± SD)

Variable	Baseline	12 Months	Change (95% Confidence Interval)	P value
Tibia 4%				
Trab vBMD	237.3 ± 28.1	234.0 ± 27.9	−3.3 (−7.5, 0.9)	0.11
Trab BMC	194.6 ± 29.2	196.6 ± 39.0	−1.0 (−9.7, 7.7)	0.81
Tibia 38%				
Cort vBMD	1196.9 ± 20.1	1199.0 ± 18.5	2.0 (−3.7, 7.7)	0.45
Cort BMC	360.6 ± 47.4	360.8 ± 45.4	0.2 (−2.7, 3.2)	0.87
Tibia 66%				
Cort vBMD	1154.5 ± 23.2	1155.8 ± 23.3	1.3 (−4.4, 7.0)	0.63
Cort BMC	365.4 ± 45.5	365.8 ± 47.6	0.5 (−4.5, 5.5)	0.84
Tibia 96%				
Total vBMD	206.7 ± 22.4	203.9 ± 18.7	−2.8 (−6.8, 1.1)	0.14
Total BMC	542.4 ± 61.7	533.0 ± 58.8	−9.5 (−23.8, 4.9)	0.18

vBMD: Volumetric Bone Mineral Density (mg/cm^3); BMC: Bone Mineral Content (mg/mm); Trab: Trabecular; Cort: Cortical.