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Original Research

Two-year Skeletal Adaptations in Development and National Level Female Figure Skaters

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Abstract

International Journal of Exercise Science 18(3): 1297-1309, 2025. https://doi.org/10.70252/ZSKN7069 Figure skaters typically have higher bone mineral density (BMD) than the general population. However, the current literature is limited to cross-sectional studies. The objective of this study was to determine two-year changes in volumetric BMD, bone microarchitecture, and estimated bone strength in elite national and development level Canadian figure skaters. Eleven female figure skaters aged 14+ years were recruited for this longitudinal study. Measurements occurred annually. High-resolution peripheral quantitative computed tomography (HR-pQCT) scans of the radius and tibia underwent three-dimensional image registration. Total (Tt), cortical (Ct) and trabecular (Tb) volumetric BMD (mg HA/cm^3), trabecular thickness (TbTh, mm) and cortical thickness (CtTh, mm) were determined. Finite element analysis estimated bone strength. Linear mixed effects models with subject random intercept and time×level interaction evaluated the influence of TtBMD over two-years. Eleven figure skaters completed baseline and one-year data collection, and nine completed the two-year study. All skaters were included in our models and were either national (n=5, 18.6-28.1 years) or development (n=6; 14.4-17.7 years) athletes. Significant time-by-level status (development or national) interactions indicated increases over time in development level athletes only for TtBMD, CtBMD, CtTh and bone strength at the radius and TtBMD, TbBMD, TbTh and bone strength at the tibia ($p<0.01$ for all). Bone outcomes did not change significantly over the two years in national level figure skaters. Changes in bone density, microarchitecture and strength were only observed in the younger, development level athletes. Given the differences in age between development and national level figure skaters, these results are understandable.

Keywords: Sport, athlete, bone density, microarchitecture, high resolution peripheral quantitative computed tomography

Introduction

Figure skating is an aesthetic-based sport consisting of five disciplines: singles skating for men and women, pairs, ice dance, and synchronized skating.[1] Today, it is common to see figure

skaters landing multi-revolution jumps that were unimaginable only a few decades ago[1] to achieve competitive success. Both singles and pair skaters experience high magnitude impacts because of frequent jumping, whereas ice dance and synchronized skating perform more footwork and lifts[2]. As a result, the landing forces and mechanical loading are expected to be lower in ice dancers and synchronized skaters than in single or pair skaters,[2] coinciding with differences in bone mineral density (BMD) between figure skating disciplines.[3]

Compared to low-impact sports like swimming, non-aquatic female athletes who experience higher impact activities associated with high ground reaction forces (GRF) tend to have higher BMD.[4] Mechanical loading is associated with greater BMD among many high-impact athletic populations, including skiers and soccer players,[5] and depending on the sport-specific loading, bilateral BMD asymmetry has been reported.[3,6] While differences in BMD are reported in figure skaters compared with controls, the same trends in higher BMD are observed irrespective of the imaging modality used (i.e., dual X-ray absorptiometry (DXA)[7] or high-resolution peripheral quantitative computed tomography (HR-pQCT)).[3]

Bone accrues rapidly during adolescence, and BMD peaks by the end of the second or early in the third decade of life.[8] However, changes due to normal growth and maturation can mask those associated with exercise.[9] Osteogenic sports that cause BMD to increase are weight-bearing, impact or strength activities where BMD tends to increase more than expected due to growth and development.[4] Young males and females who participate in sports that involve high-impact or odd-impact loading exhibit the greatest gains in bone health.[10] Participating in impact sports during early puberty may enhance bone mass,[11] while continued sport participation appears to maintain the full benefits of increased peak bone mass.[10] It is important to understand and optimize bone health from the circumpubertal years for accruing bone mineral, as there is strong consensus that early-life experiences are essential for reducing the risk of osteoporosis in later life.[12] Understanding the age at which peak bone mass is attained is also crucial for assessing its impact on future fracture risk.[8] Furthermore, peak sport-specific skill development may coincide with the onset of puberty,[13] and it is possible that designing training programs around an athlete's biological growth and maturity status could maximize both sport-specific skill development and BMD.

Although longitudinal studies explore bone changes in athletes over a sporting season,[14] few studies explore BMD adaptations in athletes for longer than 12 months, and there are no longitudinal studies in figure skaters. In addition, HR-pQCT provides 3D imaging of compartment-specific (i.e., cortical and trabecular) bone microarchitecture, something DXA is unable to provide. Therefore, the objective of this study was to determine two-year changes in volumetric bone mineral density (BMD), bone microarchitecture and estimated bone strength in national and development level elite Canadian figure skaters. Given the age of the development level skaters corresponds to the timing when loading may have the greatest effect on bone mass accrual, we hypothesized volumetric BMD and estimated bone strength would increase, whereas older national level skaters would remain constant over the two-year period.

Methods

Participants

This is an exploratory analysis based on a convenient sample of elite figure skaters, representing all eligible athletes who were accessible during the recruitment period. However, we also performed an a *priori* power analyses based on data from Gabel et al.[11] With an estimated large effect size of 0.6 with 90% power for two groups with three measurement time points a total of eight participants were required. Eleven elite female figure skaters aged 14+ years from across Canada participated in this study. Recruitment took place over eleven months, and all data collection occurred at the University of Calgary between August 2016 and December 2019. Data collection occurred three times over two years: baseline, year one and year two. To be included in this study, participants had to be affiliated with Skate Canada training at a tier three level or higher[15] and could have been training and competing in a variety of figure skating disciplines, including singles, pairs or ice dance. Participants were excluded if they were under 14 years of age or had a known medical condition affecting bone metabolism. All females in this study had achieved menses, and at the time of recruitment, none were on hormonal contraceptives at baseline. Approval for all procedures was obtained from the university's Conjoint Health Research Ethics Board (REB16-0758). Participants over the age of 18 years gave written consent prior to involvement in the study. For those participants under 18 years, either a parent gave written consent on behalf of their child, or the child was assessed and treated as a mature minor, able to sign their own consent form. This research was carried out fully in accordance with the ethical standards of the *International Journal of Exercise Science.*[16]

Protocol

Height (Charder model HM200P, Charder Electronic Co Ltd, Taiwan) and weight (Seca model 876, Seca, Germany) were measured using standard protocols to the nearest 0.1 cm and 0.1 kg, respectively. Body mass index (BMI) was calculated as weight/height^2 (kg/m^2). Participants' skinfolds were measured at baseline only by ISAK level-2 accredited anthropometrists. The following calculations have been reported at each study visit: muscle mass, fat free mass, fat mass and percent body fat, and were calculated using the Matiegka and Parizkova methods for muscle and fat calculations respectively.[17,18]

Participants completed a series of questionnaires to assess their overall health. Questionnaires included health, injury and sport-specific history, capturing information on known health concerns, current medication, previous injuries, fractures and specific figure skating training requirements.

To assess measurements of volumetric bone mineral density (BMD, mg HA/cm^3), and microarchitecture of the peripheral skeleton, participants received HR-pQCT (XtremeCT II, Scanco Medical, Brüttisellen, Switzerland) scans of their non-dominant radius and dominant tibia with a nominal isotropic resolution of 61 µm. Limb dominance was defined as writing hand for the radius and figure skating landing leg for the tibia. The dominant tibia was selected as we have previously shown side-to-side differences between figure skaters' landing and takeoff tibia.[3] If prior fractures were reported at the scan region (radius or tibia), the opposite limb was scanned. Scans were captured using the fixed offset method[19] and were located 9.5mm and 22.5

mm proximal to the reference line for the radius and tibia, respectively. The reference line was placed at the mid-inclination radial tuberosity and at the plateau portion of the tibial endplate. Total and trabecular volumetric BMD (mg HA/cm^3), trabecular number (mm^{-1}), trabecular separation (mm), thickness (mm), total and trabecular area (mm^2) were obtained by the standard morphologic analysis.[19] Cortical parameters, including total cross-sectional area (mm^2), cortical volumetric BMD (mg HA/cm^3), cortical thickness (mm) and cortical porosity (%) were determined using an automated segmentation method.[19] Estimates of bone strength were based on custom finite element analysis (FEA) software (FAIM, version 8.0, Numerics88 Solutions, Calgary, Canada) applied to each HR-pQCT scan. An axial compression test was simulated using a 1% compressive strain, Young's modulus of 8748 MPa and a Poisson's ratio of 0.3. Our primary estimate of bone strength was failure load (N) based on 2% of the elements exceeding 7,000 microstrain.[20]

To ensure the same region of bone was assessed at each study visit, three-dimensional image registration was performed for all variables except failure load, due to non-parallel surfaces[21] and bone area measurements. While image registration is the preferred method for longitudinal studies investigating adults,[19] it is not recommended during periods of growth.[22] However, at the time data collection began (2016) these recommendations were not widely adopted in the HR-pQCT community. Due to the younger age of the development skaters, image data were analyzed using both 3D image registration and non-registered (no match) methods. Scans were graded for motion artifacts where a scan scoring one had no motion, and a scan scoring five had severe blurring and discontinuities.[23] Scans with motion scores of four or higher and scans where the percent overlap of registered images was below 75% were removed from the analysis. The Root-Mean-Square Coefficient of Variation (RMSCV) in our laboratory is <1% for density, <3% for microarchitecture except CtPo (<12%).[24]

Statistical Analysis

Participant characteristics were described using mean or median and 95% confidence intervals (continuous data) or total number and percentage (categorical data). Individuals who completed at least one follow-up visit were included in this analysis. We analyzed outcome variables using linear mixed effects models with fixed effects for team status (development or national) and time. Random intercepts allowed individuals their own intercept for the effect of time. Missing data were accounted for within the mixed effects model. Specifically, we explored time-by-level status, which, if significant, indicated a difference between the development and national level team figure skaters. The primary outcome variable of this study was TtBMD and we did not adjust for multiple comparisons. Model assumptions were assessed graphically using plots of residuals and significance was set at $p < 0.05$. Analyses were performed in Stata, Version 16 (StataCorp, College Station, TX, USA).

Results

Eleven figure skaters completed baseline and one-year data collection, and nine completed the two-year study. One skater withdrew from the study because they did not wish to undergo additional imaging, and the other changed coaches and was unavailable at the time of data

collection. All eleven skaters were included in our models and were either national (n=5, 18.6-28.1 years) or development (n=6; 14.4-17.7 years) Skate Canada athletes. Skaters were predominantly singles skaters (n=8) with one pair skater and two ice dancers.

The descriptive characteristics of the figure skaters at baseline are presented in Table 1. None of the athletes in this study have been or were currently pregnant, and none reported amenorrhea. One athlete reported starting oral contraceptive use during the study. Over the two years, three athletes (27%) reported a fracture, with no individuals reporting multiple fractures. Two of the three fractures were skating-related: a stress fracture of the foot and a facial fracture (cheek bone).

Table 1. Descriptive characteristics of the figure skaters at baseline.

Variable	Development (n = 6) Median (95% CI)	National(n = 5) Median (95% CI)
Age (years)	15.6 (15.0-16.3)	25.5 (20.8-25.6)
Height (m)	1.61 (1.59-1.64)	1.61 (1.56-1.66)
Weight (kg)	56.7 (52.0-61.4)	52.5 (51.6-56.1)
Body Mass Index (kg/m^2)	21.9 (20.5-22.7)	19.8 (19.6-21.5)
Training age (years)	6.5 (6.0-10.0)	18.0 (16.0-20.0)
Training volume (h/week) [a]	20.8 (17.8-23.7)	34.4 (30.0-36.0)
Age of menarche (years)	12.5 (12.0-13.0)	13.0 (12.0-13.0)
Previous fracture (%) [b]	3.0 (50.0%)	2.0 (40.0%)
Muscle Mass (kg) [c]	25.1 (21.9-25.4)	22.5 (22.5-23.8)
Fat Free Mass (kg) [c]	47.8 (45.8-49.7)	47.5 (46.4-48.7)
Fat Mass (kg) [c]	8.3 (5.9-8.9)	5.2 (3.8-6.6)
Percent fat (%)[c]	14.0 (10.9-15.8)	10.1 (7.3-11.0)

[a] Includes all types of training (strength, cross-training etc.) in addition to figure skating. [b] Number of participants with a previous fracture at baseline, data presented as n and percentage rather than median and 95% CI. [c] Calculated from skinfold measurements, not DXA.

No scans were removed from the analyses due to motion (score of four or higher) or inadequate (<75%) percent overlap. HR-pQCT results for the radius and tibia at each time point are presented in Table 2.

Changes in volumetric BMD and bone strength throughout the study for the development and national level athletes are reported in Figure 1 for the tibia and Figure 2 for the radius. Over two years, significant time-by-level status (development or national) interactions showed increases over time in development level athletes only for TtBMD (6.45%, $p<0.001$), CtBMD (4.25%, $p<0.001$), TbBMD (4.87%, $p<0.001$), failure load (5.33%, $p<0.001$) and TbTh (4.06%, $p<0.001$) at the tibia. Time-by-level status increases over two years were also observed in development level skaters at the radius for TtBMD (11.73%, $p<0.001$), CtBMD (7.83%, $p<0.001$), failure load (13.71%, $p<0.001$), CtTh (11.51%, $p=0.010$) and CtAr (15.05%, $p=0.012$). Finally, over the duration of the study there was a time-by-level status decrease (-6.27%, $p=0.004$) in TbAr at the radius for development level skaters. No additional time-by-level status interactions were observed.

There were significant time only interactions for TbN and TbSp at both the radius and tibia. TbN at the radius (2.13%, $p=0.006$) and tibia (2.07%, $p=0.040$) increased for the development level athletes at year one relative to baseline, but were not different to baseline values at study end.

Similarly, TbSp decreased at the radius (-1.52%,p=0.001) and tibia (-1.56%, p=0.012) for the development level athletes at year one relative to baseline, but not at study end. The only interaction reported for the national level skaters was TbSp at the tibia, which increased (1.88%, p=0.021) at study end relative to baseline.

Table 2. Mixed-model analysis results for the radius and tibia at each time point.

	Baseline **Mean (95% CI)**	**Year 1** **Mean (95% CI)**	**Year 2** **Mean (95% CI)**
Radius	n=6	n=6	n=4
Development			
TtBMD	299.7 (258.6-340.8)	325.8 (287.6-364.0)[a]	334.8 (274.7-354.9)[a]
TbBMD	156.8 (135.0-178.7)	159.1 (136.9-181.4)	161.8 (138.7-184.9)
CtBMD	816.8 (771.6-862.0)	864.5 (829.2-899.8)[a]	880.8 (853.8-907.7)[a]
TbN	1.41 (1.34-1.48)	1.44 (1.37-1.50)[b]	1.41 (1.34-1.47)
TbTh	0.220 (0.208-0.232)	0.221 (0.208-0.233)	0.220 (0.207-0.234)
TbSp	0.66 (0.62-0.70)	0.65 (0.61-0.69)[a]	0.66 (0.62-0.69)
CtTh	1.01 (0.87-1.14)	1.11 (0.99-1.22)[a]	1.12 (1.02-1.22)[b]
CtPo	0.37 (0.01-0.77)	0.28 (0.02-0.53)	0.24 (0.04-0.44)
TtAr	272.9 (246.5-299.3)	267.9 (243.5-292.3)	266.6 (243.8-289.3)
TbAr	219.3 (189.6-249.0)	207.4 (180.8-234.1)[b]	205.6 (181.6-229.6)[b]
CtAr	57.1 (50.1-64.1)	63.9 (59.0-68.9)[a]	65.7 (61.6-69.8)[b]
Failure Load	3141.2 (2696.3-3586.1)	3424.4 (3028.3-3820.6)[a]	3571.8 (2649.8-3411.7)[a]
Radius National	n=5	n=5	n=5
TtBMD	312.7 (267.6-357.7)	312.4 (270.5-354.3)	314.8 (274.7-354.9)
TbBMD	144.6 (120.7-168.5)	142.4 (118.0-166.7)	144.9 (119.8-170.1)
CtBMD	927.7 (878.1-977.2)	932.5 (893.8-971.1)	932.9 (904.1-961.8)
TbN	1.37 (1.29-1.44)	1.36 (1.28-1.43)	1.38 (1.30-1.45)
TbTh	0.217 (0.204-0.229)	0.217 (0.203-0.230)	0.219 (0.204-0.233)
TbSp	0.69 (0.65-0.74)	0.70 (0.65-0.74)	0.69 (0.65-0.74)
CtTh	1.03 (0.88-1.18)	1.03 (0.91-1.16)	1.03 (0.92-1.14)
CtPo	0.47 (0.05-0.89)	0.51 (0.23-0.78)	0.23 (0.05-0.42)
TtAr	271.4 (242.5-300.3)	268.7 (241.9-295.4)	265.5 (240.9-290.0)
TbAr	216.6 (184.0-249.1)	213.4 (184.2-242.6)	209.4 (183.6-235.3)
CtAr	58.4 (50.7-65.9)	58.8 (53.4-64.2)	59.5 (55.2-63.8)
Failure Load	3062.8 (2575.4-3550.2)	3042.5 (2608.5-3476.5)	3030.8 (2649.8-3411.7)
Tibia	n=6	n=6	n=4
Development			
TtBMD	368.2 (321.5-414.8)	386.6 (340.5-432.6)[a]	391.9 (345.9-437.9)[a]
TbBMD	222.9 (194.9-250.9)	230.6 (201.8-259.3)[a]	233.8 (204.1-263.4)[a]
CtBMD	884.4 (859.5-909.2)	913.0 (892.2-933.8)[a]	921.9 (902.8-941.2)[a]
TbN	1.45 (1.35-1.54)	1.48(1.39-1.58)[c]	1.42 (1.32-1.52)
TbTh	0.275 (0.256-0.294)	0.281 (0.262-0.300)[a]	0.286 (0.266-0.306)[a]
TbSp	0.64 (0.59-0.69)	0.63 (0.58-0.67)[c]	0.64 (0.59-0.69)
CtTh	1.71 (1.47-1.95)	1.78 (1.54-2.01)	1.82 (1.58-2.06)
CtPo	1.04 (0.56-1.52)	1.14 (0.66-1.62)	1.00 (0.47-1.52)
TtAr	650.3 (580.1-720.6)	649.9 (581.7-718.1)	648.8 (582.4-715.2)
TbAr	512.7 (436.5-588.9)	506.9 (432.8-581.1)	504.6 (432.0-577.2)
CtAr	142.6 (125.7-159.6)	148.0 (133.0-163.0)	150.2 (136.2-164.2)
Failure Load	10504.5 (9247.7-11761.3)	10841.0 (9587.9-12094.1)[a]	11064.2 (9811.2-12317.3)[a]
Tibia National	n=5	n=5	n=5
TtBMD	361.2 (310.1-412.3)	362.5 (312.1-412.9)	361.19 (310.9-411.5)
TbBMD	190.7 (160.0-221.3)	190.5 (158.9-221.9)	189.5 (157.1-221.9)

CtBMD	967.7 (940.5-994.9)	970.2 (947.4-993.0)	968.5 (948.5-988.6)
TbN	1.39 (1.29-1.49)	1.39 (1.28-1.49)	1.37 (1.26-1.47)
TbTh	0.262 (0.241-0.282)	0.262 (0.241-0.283)	0.260 (0.239-0.282)
TbSp	0.69 (0.63-0.74)	0.69 (0.64-0.74)	0.70 (0.65-0.75)[c]
CtTh	1.74 (1.48-2.00)	1.75 (1.49-2.00)	1.75 (1.49-2.01)
CtPo	1.18 (0.65-1.71)	1.24 (0.72-1.76)	1.21 (0.64-1.78)
TtAr	656.9 (579.9-733.8)	654.9 (580.2-729.7)	659.7 (587.1-732.3)
TbAr	514.4 (430.8-597.9)	511.1 (429.9-592.4)	516.4 (437.0-595.7)
CtAr	147.6 (129.1-166.1)	148.9 (132.5-165.4)	148.4 (133.1-163.7)
Failure Load	9863.1 (8486.3-11239.9)	9922.9 (8550.2-11295.7)	9899.8 (8529.9-11269.8)

Data are presented as adjusted mean (95% CI) from our mixed-effect models and represent three-dimensional registered data with the exception of failure load and bone area measurements where a "no match" analysis was performed. Significant difference from baseline for time-by-level status interactions are represented by [a] $p<0.001$, [b] $p<0.010$ or [c] $p<0.050$.

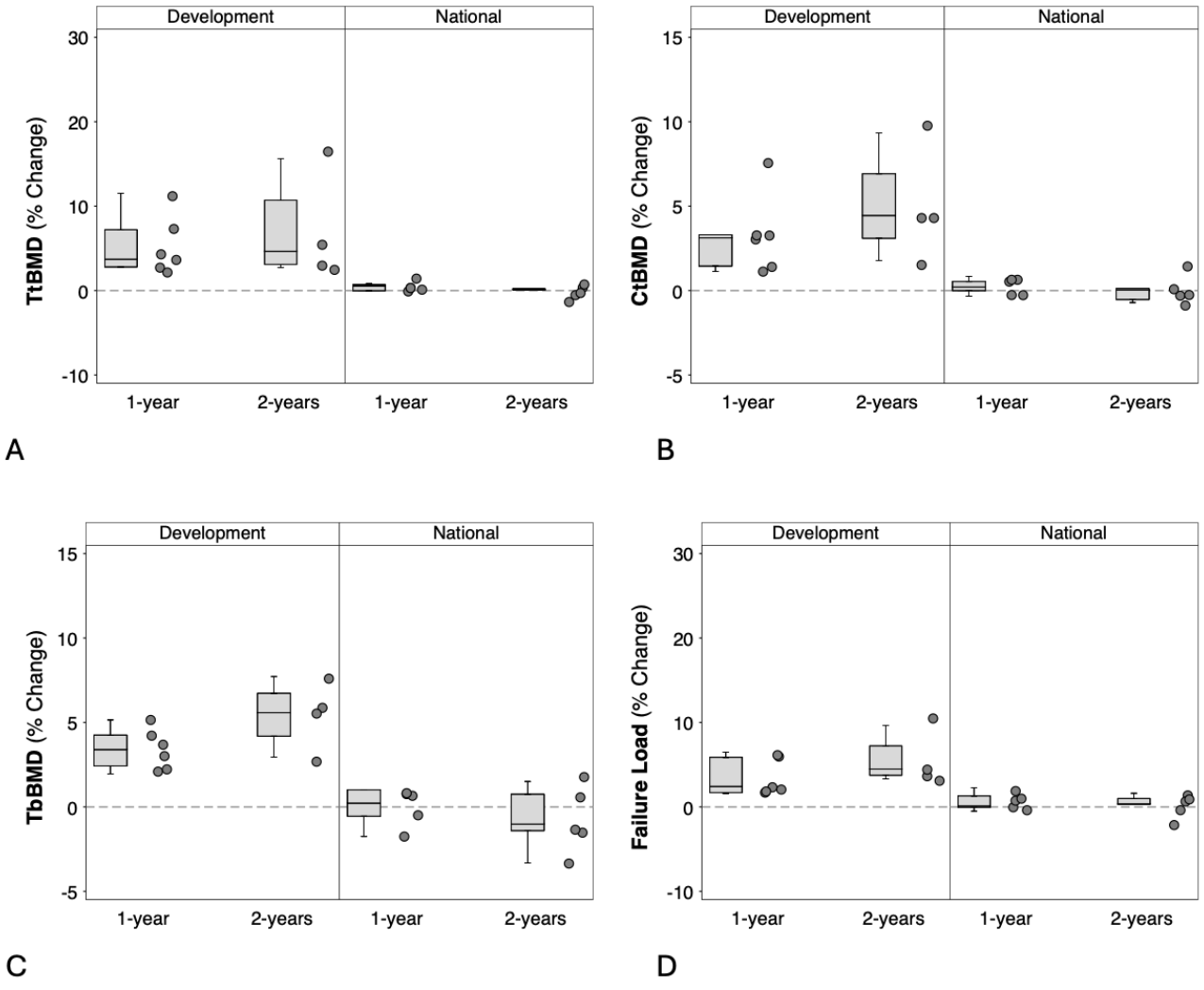

Figure 1. Changes in A total volumetric bone mineral density (TtBMD), B cortical volumetric bone mineral density (CtBMD), C trabecular volumetric bone mineral density (TbBMD) and D failure load throughout the study for the development and national athletes at the tibia.

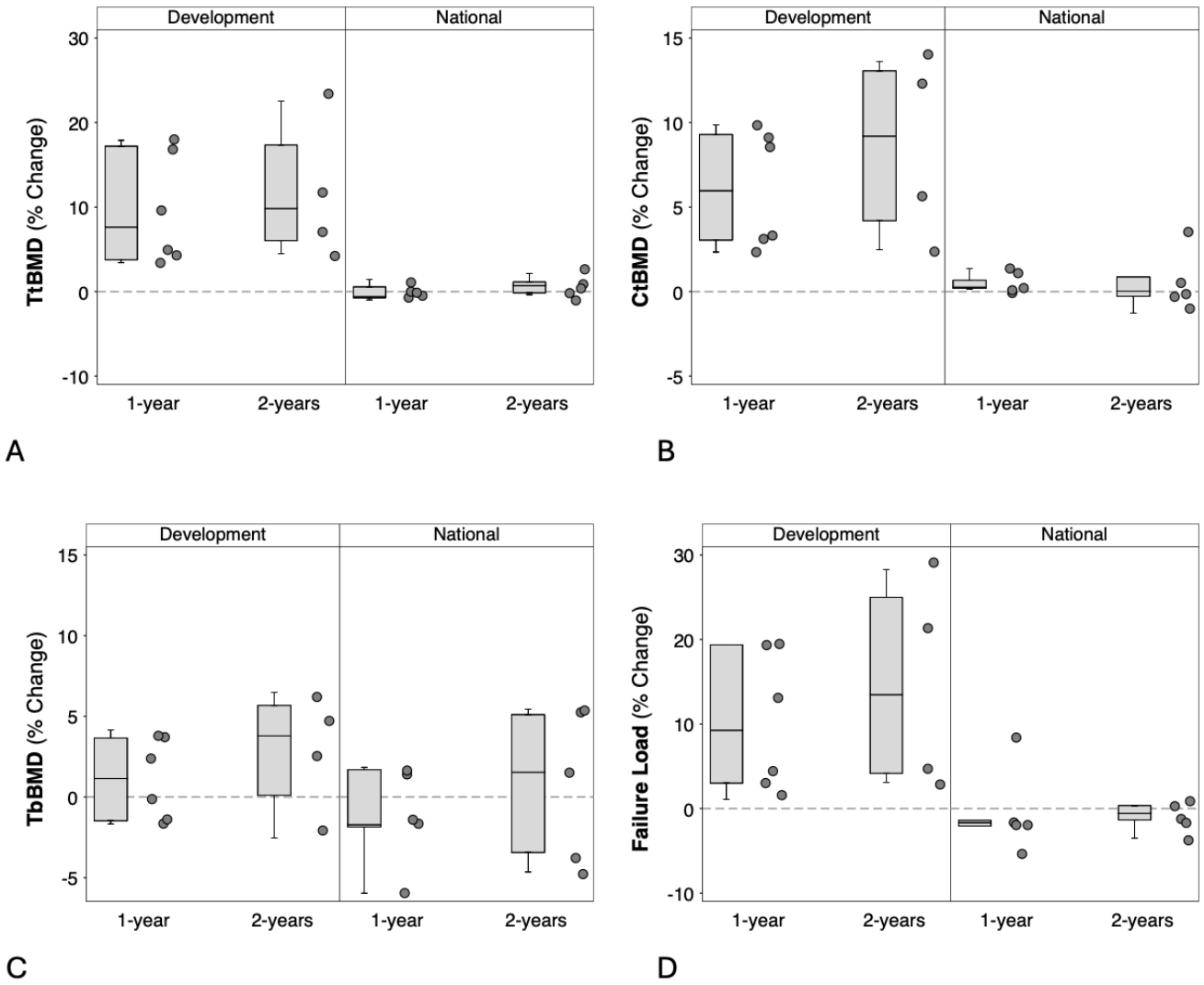

Figure 2. Changes in A total volumetric bone mineral density (TtBMD), B cortical volumetric bone mineral density (CtBMD), C trabecular volumetric bone mineral density (TbBMD) and D failure load throughout the study for the development and national athletes at the radius.

TtBMD: total bone mineral density (mg HA cm^3); TbBMD: trabecular bone mineral density (mg HA cm^3); CtBMD: cortical bone mineral density (mg HA cm^3); TbN: trabecular number (1/mm); TbTh: trabecular thickness (mm); TbSp: trabecular separation (mm); CtTh: cortical thickness (mm); CtPo: cortical porosity (%); TtAr: total area (mm^2); TbAr: trabecular area (mm^2); CtAr: cortical area (mm^2); failure load (N).

No significant interactions were observed for TtAr or CtPo at either the radius or tibia for development or national level skaters.

Discussion

The objective of this study was to determine two-year changes in volumetric BMD, bone microarchitecture, and estimated bone strength in national and development level elite

Canadian figure skaters. We confirmed our hypothesis that development level skaters' bone density and strength increased over the two-year period, whereas national skaters' remain constant. Specifically, the changes we observed in the development level skaters align with other HR-pQCT studies reporting bone density and strength accrual and adaptation in bone microarchitecture from childhood to early adulthood.[25] Over two years, our study revealed increases in volumetric BMD at the tibia between 4.3% and 6.5% and increases in bone strength of 5.3% for development level figure skaters. These values are larger than the reproducibility of the scanner and the least significant change.[24] Females undergoing normal growth have reported increases in volumetric BMD at the tibia between 6.6% and 7.2%, with bone strength increasing 6.5%. Similar to our results, increases were higher at the radius than the tibia.[25] In the absence of a control group, our findings should not be interpreted as evidence of a sport- specific effect. To observe increases in volumetric BMD and bone strength beyond normal growth, the entire adolescent growth spurt, capturing peak height velocity, should be monitored.

While studies in athletes using HR-pQCT are common, longitudinal studies are not,[26] making comparisons in bone adaptation difficult. Within the athlete realm, HR-pQCT has shown decreases in volumetric BMD following bone stress injury[27] and anterior cruciate ligament injury.[28] Changes in BMD and bone strength in young, healthy athletic populations participating in impact loading sports typically reveal positive adaptations aligning with ours, irrespective of the imaging modality used (i.e., DXA, pQCT, HR-pQCT).[29-31]

In our study, changes in bone density and strength appear to be driven by trabecular microarchitecture (i.e., trabecular thickness) at the tibia. Growth-related changes to the trabecular compartment are typically the result of thickening trabeculae rather than changes in trabecular number or separation.[25,32] Following remodeling cycles, bone is added to the trabeculae resulting in increases in thickness over time;[32] however, during growth, females tend to have stable trabecular number and separation and it is thought that these trabecular parameters are programed early in life.[33] While, it is possible participation in figure skating during growth enhanced the increase in trabecular thickness reported in the current study (4% compared with 2%),[25] additional research is needed to confirm.

At the radius, the increased cortical BMD we observed is likely the result of a thickening and expanding cortex. We found no interaction effect with cortical porosity; although there was a decreasing trend. Others have reported cortical thickness at the distal radius remains thin and porous during growth, and might even decrease;[34] however, once growth stops, cortical thickness changes in parallel with the chronological age of the adolescent and cortical thickness increases.[35] Although pubertal stage was not measured in this study, we assume the development level figure skaters to be peri to post pubertal (Tanner stage four or five).[36] Differences in cortical thickness between peri and post pubertal females have previously been reported,[33,35] and cortical thickness is known to sharply increases at the end of puberty.[33] This is supported by an increase of 19.5% reported for girls between four and six years post peak height velocity.[25] The 11.5% increase in cortical thickness we observed at the radius does not appear to be greater than adaptations associated with normal growth.

Within the confines of a cross-sectional study design, we have previously found that most microstructural and strength parameters among females peaked by 16 to 19 years at the tibia

and appeared to decline thereafter, whereas these same parameters at the distal radius remained relatively stable between 16 and 29 years.[37] Another cross-sectional study reported significant differences in bone microarchitecture at the distal radius between late- (age 15-17 years) and post-pubertal individuals (age 18-21 years) that would have been missed if individuals between 15 and 21 years were grouped together.[33] The current study supports splitting individuals in late adolescence and early adulthood groupings or adjusting individuals for age or maturity status as we report increases in volumetric BMD, bone microarchitecture and strength in younger female figure skaters aged 14 to 17 years at both the radius and tibia, and no significant changes in older female figure skaters 18 to 28 years.

This is the first study to explore skeletal changes in figure skaters using HR-pQCT. While our results are novel, they should be taken within the context of several limitations. We did not adjust our analyses for multiple comparisons. The data are limited to a small sample size, with adaptive skeletal responses differing for both development and national level skaters. This is evident by the wide confidence intervals we report and our results should be interpreted with some caution. Nevertheless, our results align with other HR-pQCT studies with a larger sample size.[25] Several components fell outside the scope of this two-year study, including change in bone turnover markers, nutrition and hormone assessment. Furthermore, we did not capture maturation or skeletal age and did not have a control group. Due to our female only cohort, we were unable to compare sex differences, and the results presented here should not be generalised to male figure skaters. The training load was consistent across time for the national level skaters in our study. It is possible that skaters the same age, whose training load increases over two years, may result in favourable skeletal changes over time.

We used the fixed offset adult scanning protocol during this study.[19] Current recommendations for HR-pQCT imaging in children, published after the data collection period of this study, recommend relative offset methods and advise against image registration due to changes in bone cross-section.[22] However, total bone area did not significantly change over the study duration for development skaters (radius: -2.2%; tibia: 0.3%), where changes reported are within the error of the scanner,[24] and support the findings that for females total bone area does not change between 14 years and adults.[38] Our bone density and microarchitecture results were the same using both 3D registered and non-matched techniques (data not shown). However, we reported non-matched data for bone size and strength variables as the 3D registration process applies whole bone masks at follow-up visits to the baseline image, and creates non-parallel surfaces, making comparisons in bone area and strength over time more challenging.[21,24]

Additional longitudinal studies, with a larger sample size, including non-athlete controls, are needed to accurately track bone changes over time in athletic cohorts, highlighting a sport-specific understanding of bone adaptation using HR-pQCT. Future studies should consider capturing bone turnover markers and performing nutrition assessments. Following athletes from the development level through to retirement from elite sport would provide valuable information on the importance of early and sustained participation in high-impact sports for optimizing bone health and potentially reducing fracture risk later in life.

This two-year longitudinal study revealed that skeletal adaptations in elite female figure skaters are dependent on developmental stage. Adolescent, development level skaters demonstrated

significant increases in volumetric bone mineral density, improved microarchitecture, and greater estimated strength at the distal radius and tibia. In contrast, skeletally mature, national level skaters showed maintenance of these skeletal parameters, with no significant changes observed over the two-year period. These findings suggest a critical period for sport-related bone accrual during adolescence, with sustained participation preserving these skeletal advantages into young adulthood.

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