

**BONE DENSITY IN
COMPETITIVE PRE-MENARCHIAL GYMNASTS**

BY

D. RENNIE BENEDICT

**A Thesis
Submitted to the Faculty of Graduate Studies
in Partial Fulfillment of the Requirements
for the Degree of**

MASTER OF SCIENCE

**Faculty of Physical Education and Recreation Studies
University of Manitoba
Winnipeg, Manitoba**

(c) September, 1996



National Library
of Canada

Acquisitions and
Bibliographic Services Branch

395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale
du Canada

Direction des acquisitions et
des services bibliographiques

395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Your file Votre référence

Our file Notre référence

The author has granted an irrevocable non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of his/her thesis by any means and in any form or format, making this thesis available to interested persons.

L'auteur a accordé une licence irrévocable et non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de sa thèse de quelque manière et sous quelque forme que ce soit pour mettre des exemplaires de cette thèse à la disposition des personnes intéressées.

The author retains ownership of the copyright in his/her thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without his/her permission.

L'auteur conserve la propriété du droit d'auteur qui protège sa thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

ISBN 0-612-16090-4

Canada

Name _____

Dissertation Abstracts International and *Masters Abstracts International* are arranged by broad, general subject categories. Please select the one subject which most nearly describes the content of your dissertation or thesis. Enter the corresponding four-digit code in the spaces provided.

SUBJECT TERM

HUMAN DEVELOPMENT.

0	7	5	8
---	---	---	---

U·M·I

SUBJECT CODE

Subject Categories

THE HUMANITIES AND SOCIAL SCIENCES**COMMUNICATIONS AND THE ARTS**

Architecture	0729
Art History	0377
Cinema	0900
Dance	0378
Design and Decorative Arts	0389
Fine Arts	0357
Information Science	0723
Journalism	0391
Landscape Architecture	0390
Library Science	0399
Mass Communications	0708
Music	0413
Speech Communication	0459
Theater	0465

EDUCATION

General	0515
Administration	0514
Adult and Continuing	0516
Agricultural	0517
Art	0273
Bilingual and Multicultural	0282
Business	0688
Community College	0275
Curriculum and Instruction	0727
Early Childhood	0518
Elementary	0524
Educational Psychology	0525
Finance	0277
Guidance and Counseling	0519
Health	0680
Higher	0745
History of	0520
Home Economics	0278
Industrial	0521
Language and Literature	0279
Mathematics	0280
Music	0522
Philosophy of	0998

Physical	0523
Reading	0535
Religious	0527
Sciences	0714
Secondary	0533
Social Sciences	0534
Sociology of	0340
Special	0529
Teacher Training	0530
Technology	0710
Tests and Measurements	0288
Vocational	0747

LANGUAGE, LITERATURE AND LINGUISTICS

Language	
General	0679
Ancient	0289
Linguistics	0290
Modern	0291
Rhetoric and Composition	0681
Literature	
General	0401
Classical	0294
Comparative	0295
Medieval	0297
Modern	0298
African	0316
American	0591
Asian	0305
Canadian (English)	0352
Canadian (French)	0355
Caribbean	0360
English	0593
Germanic	0311
Latin American	0312
Middle Eastern	0315
Romance	0313
Slavic and East European	0314

PHILOSOPHY, RELIGION AND THEOLOGY

Philosophy	0422
Religion	
General	0318
Biblical Studies	0321
Clergy	0319
History of	0320
Philosophy of	0322
Theology	0469

SOCIAL SCIENCES

American Studies	0323
Anthropology	
Archaeology	0324
Cultural	0326
Physical	0327
Business Administration	
General	0310
Accounting	0272
Banking	0770
Management	0454
Marketing	0338
Canadian Studies	0385
Economics	
General	0501
Agricultural	0503
Commerce-Business	0505
Finance	0508
History	0509
Labor	0510
Theory	0511
Folklore	0358
Geography	0366
Gerontology	0351
History	
General	0578
Ancient	0579

Medieval	0581
Modern	0582
Church	0330
Black	0328
African	0331
Asia, Australia and Oceania	0332
Canadian	0334
European	0335
Latin American	0336
Middle Eastern	0333
United States	0337
History of Science	0585
Law	0398
Political Science	
General	0615
International Law and Relations	0616
Public Administration	0617
Recreation	0814
Social Work	0452
Sociology	
General	0626
Criminology and Penology	0627
Demography	0938
Ethnic and Racial Studies	0631
Individual and Family Studies	0628
Industrial and Labor Relations	0629
Public and Social Welfare	0630
Social Structure and Development	0700
Theory and Methods	0344
Transportation	0709
Urban and Regional Planning	0999
Women's Studies	0453

THE SCIENCES AND ENGINEERING**BIOLOGICAL SCIENCES**

Agriculture	
General	0473
Agronomy	0285
Animal Culture and Nutrition	0475
Animal Pathology	0476
Fisheries and Aquaculture	0792
Food Science and Technology	0359
Forestry and Wildlife	0478
Plant Culture	0479
Plant Pathology	0480
Range Management	0777
Soil Science	0481
Wood Technology	0746
Biology	
General	0306
Anatomy	0287
Animal Physiology	0433
Biostatistics	0308
Botany	0309
Cell	0379
Ecology	0329
Entomology	0353
Genetics	0369
Limnology	0793
Microbiology	0410
Molecular	0307
Neuroscience	0317
Oceanography	0416
Plant Physiology	0817
Veterinary Science	0778
Zoology	0472
Biophysics	
General	0786
Medical	0760

Geodesy	0370
Geology	0372
Geophysics	0373
Hydrology	0388
Mineralogy	0411
Paleobotany	0345
Paleoecology	0426
Paleontology	0418
Paleozoology	0985
Palynology	0427
Physical Geography	0368
Physical Oceanography	0415

HEALTH AND ENVIRONMENTAL SCIENCES

Environmental Sciences	0768
Health Sciences	
General	0566
Audiology	0300
Dentistry	0567
Education	0350
Administration, Health Care	0769
Human Development	0758
Immunology	0982
Medicine and Surgery	0564
Mental Health	0347
Nursing	0569
Nutrition	0570
Obstetrics and Gynecology	0380
Occupational Health and Safety	0354
Oncology	0992
Ophthalmology	0381
Pathology	0571
Pharmacology	0419
Pharmacy	0572
Public Health	0573
Radiology	0574
Recreation	0575
Rehabilitation and Therapy	0382

Speech Pathology	0460
Toxicology	0383
Home Economics	0386

PHYSICAL SCIENCES

Pure Sciences	
Chemistry	
General	0485
Agricultural	0749
Analytical	0486
Biochemistry	0487
Inorganic	0488
Nuclear	0738
Organic	0490
Pharmaceutical	0491
Physical	0494
Polymer	0495
Radiation	0754
Mathematics	0405
Physics	
General	0605
Acoustics	0986
Astronomy and Astrophysics	0606
Atmospheric Science	0608
Atomic	0748
Condensed Matter	0611
Electricity and Magnetism	0607
Elementary Particles and High Energy	0798
Fluid and Plasma	0759
Molecular	0609
Nuclear	0610
Optics	0752
Radiation	0756
Statistics	0463
Applied Sciences	
Applied Mechanics	0346
Computer Science	0984

Engineering	
General	0537
Aerospace	0538
Agricultural	0539
Automotive	0540
Biomedical	0541
Chemical	0542
Civil	0543
Electronics and Electrical	0544
Environmental	0775
Industrial	0546
Marine and Ocean	0547
Materials Science	0794
Mechanical	0548
Metallurgy	0743
Mining	0551
Nuclear	0552
Packaging	0549
Petroleum	0765
Sanitary and Municipal	0554
System Science	0790
Geotechnology	0428
Operations Research	0796
Plastics Technology	0795
Textile Technology	0994

PSYCHOLOGY

General	0621
Behavioral	0384
Clinical	0622
Cognitive	0633
Developmental	0620
Experimental	0623
Industrial	0624
Personality	0625
Physiological	0989
Psychobiology	0349
Psychometrics	0632
Social	0451

**THE UNIVERSITY OF MANITOBA
FACULTY OF GRADUATE STUDIES
COPYRIGHT PERMISSION**

BONE DENSITY IN COMPETITIVE PRE-MENARCHIAL GYMNASTS

BY

D. RENNIE BENEDICT

**A Thesis/Practicum submitted to the Faculty of Graduate Studies of the University of Manitoba in partial
fulfillment of the requirements for the degree of**

MASTER OF SCIENCE

D. Rennie Benedict

© 1996

**Permission has been granted to the LIBRARY OF THE UNIVERSITY OF MANITOBA to lend or sell copies
of this thesis/practicum, to the NATIONAL LIBRARY OF CANADA to microfilm this thesis/practicum and
to lend or sell copies of the film, and to UNIVERSITY MICROFILMS INC. to publish an abstract of this
thesis/practicum..**

**This reproduction or copy of this thesis has been made available by authority of the copyright owner solely
for the purpose of private study and research, and may only be reproduced and copied as permitted by
copyright laws or with express written authorization from the copyright owner.**

TABLE OF CONTENTS

	Page
ABSTRACT.....	v
ACKNOWLEDGEMENTS.....	vi
LIST OF TABLES.....	vii
LIST OF FIGURES.....	viii
 1. INTRODUCTION.....	 1
1.0 Structure, Function and Metabolism of Bone.....	2
1.1 Forms of Bone.....	4
1.2 Methods to Measure Bone Mass.....	5
1.2.0 Body Composition Assessment by DXA.....	6
1.3 Skeletal Growth and Development During Childhood and Adolescence.....	7
1.4 Determinants of Peak Bone Mass in Girls and Young Women.....	8
1.4.0 Environmental Factors.....	9
1.4.1 Physical Activity.....	10
1.4.2 Nutritional Factors.....	11
1.4.3 Hormonal Factors.....	14
1.4.4 Genetic Factors.....	16
1.5 Summary.....	17
1.6 Rationale for the Study.....	17
1.7 Statement of the Problem.....	19
1.8 Delimitations.....	20
1.9 Limitations.....	21
1.10 Definition of Terms.....	22
 2. REVIEW OF THE LITERATURE.....	 23
2.0 Introduction.....	23
2.1 Techniques Used to Measure Bone Mineral Status.....	23
2.2 Assessment of Body Composition by DXA.....	28
2.3 Factors Affecting Peak Bone Mass in Girls and Young Women.....	29
2.3.0 Physical Activity and Hormonal Factors.....	29
2.3.1 Site Specific Effects of Physical Activity on Bone Mineral Density.....	35
2.3.2 Maturation of the Reproductive System in Prepubertal and Pubertal Girls.....	39
2.3.3 Delayed Menarche, Physical Training and Bone Health.....	41
2.3.4 Delayed Menarche and Peak Bone Mass.....	45
2.3.5 Calcium Intake.....	48
2.3.6 Genetic Factors.....	54

2.4 The Effect of Developmental Factors on Bone Mineral Density in Children and Adolescents.....	57
2.5 Variability of Bone Mineral Density in Children.....	64
3. METHODS AND PROCEDURES.....	67
3.0 Sample Size Estimation.....	67
3.1 Subject Recruitment and Selection.....	68
3.2 Experimental Design.....	71
3.3 Data Collection.....	71
3.3.0 Questionnaires.....	71
3.3.1 Bone Densitometry and Body Composition Measurements.....	73
3.3.2 Anthropometric Measurements.....	75
3.3.3 Dietary Intake.....	76
3.4 Ethics.....	78
3.5 Statistical Analyses.....	78
4. RESULTS.....	80
4.0 Description of Subjects.....	80
4.1 Body Composition Measurements.....	81
4.2 Anthropometric Measurements.....	82
4.2.0 Skinfold Thickness Measurements.....	82
4.2.1 Body Circumference Measurements.....	86
4.2.2 Bone Length Measurements.....	86
4.2.3 Bone Breadth Measurements.....	88
4.3 Bone Mineral Measurements.....	88
4.3.0 L ₂ -L ₄ and Proximal Femur.....	88
4.3.1 Regional and Total Body.....	89
4.4 Results of Dietary Analysis.....	89
4.5 Relationships between Variables.....	92
4.5.0 Age and Weight.....	92
4.5.1 Height and Weight.....	93
4.5.2 Bone-Free Lean Tissue and Weight.....	93
4.5.3 Age and BMD.....	93
4.5.4 Height and BMD.....	93
4.5.5 Weight and BMD.....	96
4.5.6 Bone-Free Lean Tissue and BMD.....	97
4.5.7 Physical Activity and BMD.....	100
4.5.8 Calcium Intake and BMD.....	100

5. DISCUSSION.....	101
5.0 Summary of Study Design and Results.....	101
5.1 General Subject Characteristics.....	101
5.2 Body Composition and Anthropometric Measurements.....	102
5.2.0 Estimates of Percent Body Fat in Subjects.....	104
5.3 Nutrient Intake.....	105
5.4 Bone Mineral Measurements.....	107
5.4.0 Lumbar Spine and Proximal Femur Bone Mass.....	107
5.4.1 Regional and Total Body Bone Mass.....	111
5.5 Relationships between Independent Variables and BMD.....	113
5.5.0 Age, Height, Weight, Bone-Free Lean Tissue and BMD.....	113
5.5.1 Physical Activity, Calcium Intake and BMD.....	118
5.6 Summary.....	120
6. CONCLUSIONS AND RECOMMENDATIONS.....	121
REFERENCES.....	124
APPENDICES.....	138
APPENDIX A Description of the Study and Informed Consent Form.....	139
APPENDIX B Medical Questionnaire, Physical Activity Questionnaire, Food Intake Questionnaire, and Calcium Intake Questionnaire.....	144
APPENDIX C Eating Disorder Inventory.....	161
APPENDIX D Equations Used for Estimating Body Density and Percent Body Fat from Skinfold Thicknesses.....	165
APPENDIX E Instructions for Keeping a 3-Day Food Record and Food Record Sheet.....	167
APPENDIX F Participant Information Sheet.....	173
APPENDIX G Data Collection Sheet for Anthropometric Measurements.....	175
APPENDIX H List of Abbreviations.....	179

ABSTRACT

Objective: (i) To determine if intensive physical training exerts an effect on bone mineral density (BMD) in pre-menarchial girls. (ii) To clarify the relationship between BMD and age, height, weight, bone-free lean tissue (BFLT) and calcium intake in pre-menarchial girls.

Design: Cross-sectional study: (i) comparing BMD in competitive pre-menarchial gymnasts to BMD in normally active girls (controls) of the same age and pubertal status and (ii) examining the relationship between BMD and each independent variable (i.e., age, height, weight, BFLT and calcium intake) in pre-menarchial gymnasts and controls.

Subjects: 11 competitive pre-menarchial rhythmic and artistic gymnasts aged 9-13 years (mean physical activity level 11.3 ± 0.6 h/wk) and 15 pre-menarchial normally active girls aged 9-12 years (mean physical activity level 3.0 ± 0.3 h/wk).

Measurements: BMD (g/cm^2) of the lumbar spine (L_2-L_4), femoral neck (FN), Ward's Triangle (WT), trochanter and total body, and percent body fat as measured by dual energy x-ray absorptiometry (DXA). Anthropometry: height, weight, skinfold thicknesses, body girths, bone lengths and bone widths. Nutrient intake: energy, protein, fat, carbohydrate, calcium, phosphorus and vitamin D as determined from 3-day food records.

Main Results: Gymnasts and controls did not differ with respect to age, height, pubertal status or calcium intake. Gymnasts were characterized by significantly lower body weight ($p < 0.05$), less body fat ($p < 0.01$) and a lower energy intake ($p < 0.01$) compared to controls. Gymnasts exhibited significantly greater BMD than controls at L_2-L_4 (0.919 ± 0.04 vs. 0.809 ± 0.024 , $p < 0.01$), FN (0.973 ± 0.051 vs. 0.813 ± 0.017 , $p < 0.01$), WT (0.994 ± 0.048 vs. 0.826 ± 0.024 , $p < 0.01$), the trochanter (0.814 ± 0.033 vs. 0.688 ± 0.024 , $p < 0.01$) and the arms (0.675 ± 0.015 vs. 0.638 ± 0.008 , $p < 0.05$). Total body BMD did not differ between the two groups. In gymnasts, age, height, weight and BFLT were significantly and positively related to BMD at L_2-L_4 , FN and the total body, while in controls, the only significant positive associations seen were between weight and BMD at L_2-L_4 and the total body, and BFLT and L_2-L_4 BMD. Calcium intake was not significantly related to BMD at any skeletal site in either gymnasts or controls.

Conclusions: Competitive pre-menarchial gymnasts exhibit higher BMD of the lumbar spine, proximal femur and the arms than their normally active counterparts. Gymnastics appears to provide the appropriate type of training to enhance BMD in pre-menarchial girls, particularly at predominantly trabecular skeletal sites. Homogeneity in terms of calcium intake by subjects and mean calcium consumption levels which were close to the Recommended Nutrient Intake may have been responsible for the lack of association between calcium and BMD in this sample of pre-menarchial girls.

ACKNOWLEDGEMENTS

I am deeply grateful to my husband, my parents and my sister for their loving support, encouragement and patience throughout this endeavour. In particular, I wish to extend my sincerest thanks to the following people for assisting me with this thesis and helping me to further my academic career. Their contributions are truly appreciated.

Roman, Jack, June and Deirdre

Dr. A.E. Ready

Dr. L. Smith and Dr. G. Sevenhuysen

Donna McDonald

Brian, Barb and Mira Lecker

NSERC

LIST OF TABLES

Table 4.1 General characteristics of gymnasts and controls (mean \pm SE).....	80
Table 4.2 Body composition data of gymnasts and controls (mean \pm SE).....	81
Table 4.3 Skinfold thicknesses of gymnasts and controls (mean \pm SE).....	83
Table 4.4 Comparison of methods predicting percent body fat of gymnasts and controls (mean \pm SE).....	84
Table 4.5 Body circumferences of gymnasts and controls (mean \pm SE).....	87
Table 4.6 Bone lengths of gymnasts and controls (mean \pm SE).....	87
Table 4.7 Bone breadths of gymnasts and controls (mean \pm SE).....	88
Table 4.8 Bone mineral data (L ₂ -L ₄ and proximal femur) of gymnasts and controls (mean \pm SE).....	89
Table 4.9 Bone mineral data (regional and total body) of gymnasts and controls (mean \pm SE).....	90
Table 4.10 Nutritional data of gymnasts and controls (mean \pm SE).....	91
Table 4.11 Regression line equations (slope and intercept) of the relationship between height (cm) and BMD (g/cm ²) in gymnasts and controls.....	96
Table 4.12 Regression line equations (slope and intercept) of the relationship between bone-free lean tissue weight (kg) and BMD (g/cm ²) in gymnasts and controls.....	99

LIST OF FIGURES

Figure 4.1 Body compartments (fat tissue weight, bone-free lean tissue weight and bone weight) of gymnasts and controls, expressed as percentage of total body tissue weight.....	82
Figure 4.2 Relationship between percent body fat estimated by Jackson et al. (1980) and Siri (1961) and percent body fat determined by dual energy x-ray absorptiometry (DXA) in 21 pre-menarchial girls aged 9-13 years ($p<0.01$).....	85
Figure 4.3 Relationship between percent body fat estimated by Slaughter et al. (1988) using triceps and subscapular skinfolds and percent body fat determined by dual energy x-ray absorptiometry (DXA) in 21 pre-menarchial girls aged 9-13 years ($p<0.01$).....	85
Figure 4.4 Relationship between percent body fat estimated by Slaughter et al. (1988) using triceps and calf skinfolds and percent body fat determined by dual energy x-ray absorptiometry (DXA) in 21 pre-menarchial girls aged 9-13 years ($p<0.01$).....	86
Figure 4.5 The relationship between age and total body bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p<0.01$), and (B) 15 pre-menarchial normally active girls (controls) (ns).....	94
Figure 4.6 The relationship between age and L_2-L_4 bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p<0.01$), and (B) 15 pre-menarchial normally active girls (controls) (ns).....	94
Figure 4.7 The relationship between age and femoral neck bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p<0.01$), and (B) 15 pre-menarchial normally active girls (controls) ($p<0.05$).....	95
Figure 4.8 The relationship between age and Ward's Triangle bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts (ns), and (B) 15 pre-menarchial normally active girls (controls) ($p<0.01$).....	95
Figure 4.9 The relationship between weight and total body bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p<0.01$), and (B) 15 pre-menarchial normally active girls (controls) ($p<0.05$).....	97

Figure 4.10 The relationship between weight and L₂-L₄ bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p < 0.01$), and (B) 15 pre-menarchial normally active girls (controls) ($p < 0.05$).....98

Figure 4.11 The relationship between weight and femoral neck bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p < 0.01$), and (B) 15 pre-menarchial normally active girls (controls) (ns).....98

Figure 4.12 The relationship between weight and Ward's Triangle bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts (ns), and (B) 15 pre-menarchial normally active girls (controls) (ns).....99

1. INTRODUCTION

The amount of bone acquired during growth, and the subsequent rate at which it is lost throughout adulthood, will determine a woman's bone mass at any given point in her lifetime. Until recently, the main approach to the issue of bone demineralization and osteoporosis has been a therapeutic one, in that it has focussed on methods of slowing, halting or attempting to reverse the bone loss process in post-menopausal women. However, over the past few years, increasing attention has been given to ways of preventing this debilitating disease. The basic premise behind the preventive approach is that attainment and maintenance of a high peak bone mass (PBM) in young adulthood will serve as a buffer against the gradual loss of bone mass which occurs with age (Riggs et al., 1982). It is becoming ever more apparent that skeletal health is not just a concern for post-menopausal women. Rather, strategies to protect against osteoporosis need to be directed at girls and women of all ages.

In an effort to define ways of maximizing PBM in girls and young women, an accumulating body of research has examined factors which are thought to influence bone accretion during growth and development. Lifestyle habits, nutrient intake, hormonal status, physical activity levels and genetics have been identified as major determinants of PBM, although the relative contribution of each remains to be clarified (Chestnut III, 1991; Ott, 1990; Pollitzer & Anderson, 1989). However, prior to examining these factors in more detail, it is important to have a general

understanding of bone structure, function and metabolism as well as bone growth and development during childhood and adolescence. In addition, a brief description of the forms of bone and methods of measuring bone mass is warranted.

1.0 Structure, Function and Metabolism of Bone

The skeletal system plays two fundamental roles, which Parfitt and Chir (1987) have described as follows: (1) the rigidity and hardness of bone provide a structural framework for maintenance of body shape, protect soft tissues, and allow for mechanical leverage, and (2) since bone is approximately two-thirds mineral by weight, it serves as a large mineral reservoir, thus contributing to the regulation of extra-cellular fluid composition (e.g., maintenance of stable calcium concentrations). Serum calcium levels are mediated by the secretion of parathyroid hormone (PTH) and synthesis of the active form of vitamin D ($1,25[\text{OH}]_2\text{D}_3$). Chronically low serum calcium concentrations stimulate an increase in PTH, which in turn reduces renal excretion of calcium and promotes formation of $1,25[\text{OH}]_2\text{D}_3$ by the kidney. This sequence of events represents an attempt to maintain serum calcium levels within narrow limits of physiological tolerance (Genuth, 1988).

At the cellular level of bone, osteoclasts are large, multi-nucleated cells responsible for extracting calcium from the skeleton, destroying bone matrix and reducing bone mass (Genuth, 1988). In contrast, osteoblasts synthesize collagen fibrils which, in turn, produce an organic matrix termed osteoid. Calcium phosphate is then deposited into osteoid along with hydroxide and bicarbonate ions. Within

10 days of osteoid formation, hydroxyapatite crystals are formed and the mineralization process is complete (Genuth, 1988).

In terms of metabolism, bone is a dynamic tissue which undergoes continuous cycles of resorption (erosion of bone surface by osteoclasts) and formation (deposition of new bone by osteoblasts). The goal of this process is two-fold: (1) to maintain mineral homeostasis (e.g., calcium) and (2) to enhance skeletal strength such that bone can withstand mechanical stress (Dalsky, 1990). The degree to which resorption and formation are coupled determines whether bone is in a positive or negative balance situation, or if homeostasis is maintained. In positive balance, formation of new bone exceeds erosion of old. This process is referred to as modelling and is characterized by net bone accretion (Charles et al., 1991). Ultimately, it leads to the creation of the mature skeleton. Negative balance, on the other hand, occurs when bone is lost more rapidly than it is formed (i.e., net bone resorption results). Bone homeostasis refers to a balance between resorption and formation, such that PBM is preserved.

According to Dalsky (1990), positive balance is seen throughout childhood, adolescence and early adulthood, until PBM is realized, while negative balance is accompanied by a slow, progressive bone loss, usually in association with the aging process. During menopause, bone demineralization is especially dramatic as there is an accelerated rate of loss related to estrogen withdrawal. This rapid demineralization occurs in addition to the age-associated loss of bone (Dalsky, 1990). Thus, bone status throughout the life cycle is a function of both PBM

achieved during growth and rate of loss during adult years.

1.1 Forms of Bone

The skeletal system is made up of both cortical (compact) and trabecular (cancellous) bone. Cortical bone is comprised of a regular and consistent network of tissue, and constitutes approximately 80% of the total skeletal mass (Wells, 1991). It is of low porosity and surface to volume ratio. Thus, it is more solid than trabecular bone. Cortical bone is found in the outer walls of all bones, but the bulk of it resides in the shafts of the long bones of the appendicular skeleton (Parfitt & Chir, 1987). It undergoes a slower turnover rate than trabecular bone and is therefore less prone to demineralization. Only about 5% of its surface is subjected to active remodelling at any given time, compared to 20% of trabecular bone (Dalsky, 1990).

Trabecular bone consists of an irregular lattice network which is sponge-like in appearance (Wells, 1991). It is of high porosity and surface to volume ratio (Parfitt & Chir, 1987). Trabecular bone comprises the remaining 20% of the skeletal system and predominates in the vertebral bodies (axial skeleton) and the ends of the long bones (e.g., proximal femur and ultra-distal radius) (Dalsky, 1990; Wells, 1991). In view of its high turnover rate, trabecular bone is more susceptible than cortical bone to hormonal stimuli that can lead to demineralization.

Because trabecular bone is more metabolically active than cortical bone, measuring bone mineral status at sites of primarily trabecular bone is a better

indicator of bone mass than measuring at cortical sites (Buchanan et al., 1988; Riggs & Melton, 1986).

1.2 Methods to Measure Bone Mass

Four modalities are commonly used to measure bone mineral content (BMC) and bone mineral density (BMD): radiogrammetry (RG), quantitative computed tomography (QCT), photon absorptiometry, and dual energy x-ray absorptiometry (DXA).

RG employs standardized x-rays to measure the degree of ossification at the hand or wrist. Thus, it is a method of assessing skeletal maturity (Wells, 1991).

In QCT, an image density of bone is obtained by making repeated CT scans over an area, such as the lumbar vertebrae. BMD is then determined by comparing the image with a calibration phantom (Sinning & Little, 1987). The advantage of this technique is that it allows for exclusive measurement of trabecular bone in the centre of the vertebral body (Riggs & Melton, 1986).

The principle behind photon absorptiometry is as follows: when a photon beam is passed through bone, the amount of energy absorbed is directly proportional to the amount of bone mineral present (Health and Public Policy Committee, 1987). Soft tissue also absorbs photons, but to a lesser extent than bone.

Two methods of photon absorptiometry are available: single photon absorptiometry (SPA) and dual photon absorptiometry (DPA). SPA is typically used

for peripheral bone densitometry (measuring sites such as the forearm and os calcis, which feature little soft tissue) and therefore reflects primarily cortical bone content (Mazess & Barden, 1989; Riggs & Melton, 1986). DPA is a suitable modality for determining bone mass of the spine and proximal femur (sites which contain predominantly trabecular bone) and the total body. However, DPA cannot distinguish between the two types of bone (Wahner, 1989).

DXA is a relatively new densitometric technique which utilizes an x-ray tube as the energy source. As with DPA, it can be used to measure bone mineral of the spine, hip and total body (Wahner, 1989). However, it provides better spatial resolution, improved imaging, faster scanning speeds and less radiation exposure compared to conventional DPA (Mazess & Barden, 1988; Wahner, 1989). Thus, DXA is considered an ideal technique for determining BMD in children (Glastre et al., 1990; Sartoris & Resnick, 1988).

1.2.0 Body Composition Assessment by DXA

In addition to assessing BMD, DXA is capable of providing precise regional and total body soft tissue composition analysis (Mazess et al., 1990). Lohman (1992) has commented on the lack of methodologies to accurately evaluate body composition (i.e., muscle, fat and bone) in growing children. Densitometry (i.e., determination of total body density) is generally labour-intensive and may be difficult to perform with children (Weststrate & Deurenberg, 1989). Furthermore, it has limitations related to the assumption of a constant density for fat-free mass

which may not be true in children (Houtkooper, 1996; Jensen, 1992). While skinfold thicknesses offer an estimate of the subcutaneous fat layer, attempts to relate this value to a true estimate of total percent body fat are not always accurate in children (Weststrate & Deurenberg, 1989). Given its ability to assess both regional and total body composition, DXA may provide a better alternative than densitometry or anthropometry for evaluating body composition in children, although further validation studies are required before it can be accepted as a criterion method for soft tissue measurements (Lohman, 1992; Ogle et al., 1995).

1.3 Skeletal Growth and Development During Childhood and Adolescence

From birth to approximately age 16, bones are in a phase of accelerated growth and bone modelling (Matkovic, 1992). Skeletal growth increases seven-fold from birth to puberty, and is followed by a further three-fold increase during adolescence (Peacock, 1991).

In infants, peak height velocity is approximately 18 cm/year (Matkovic, 1992). This is followed by a deceleration in the rate of growth between 2 and 8 years of age, during which time height velocity declines from 9 to 5.5 cm/year in both males and females (Matkovic, 1992). However, with the onset of puberty, height velocity increases dramatically from 5.5 to 8.5 cm/year (Matkovic, 1992). In girls, this peak height velocity (or growth spurt) occurs between 10 and 12 years of age and coincides with an increase in total bone mineral and bone mineral density (Marcus, 1987; Wells, 1991). With menarche, there is usually a decline in the rate of linear

growth, and by age 15, mean height velocity approximates zero, due to closure of the epiphyses (growth plates) in the long bones. Following cessation of puberty and linear growth, girls continue to gain bone mass due to consolidation of the skeleton. This period of bone mass accumulation allows for ossification of bone that was not fully mineralized during adolescence, and is thought to continue into the third decade (Peacock, 1991).

1.4 Determinants of Peak Bone Mass in Girls and Young Women

Peak bone mass (PBM) may be defined as the highest value an individual achieves during his or her lifetime (Ott, 1990). Although there is no consensus on the age at which PBM is reached in women, recent information suggests that cortical bone mass peaks by the beginning of the third decade while trabecular bone mass of the vertebral column and hip reaches a maximum by late adolescence (Lu et al., 1994; Matkovic, 1992; Matkovic et al., 1994; Theintz et al., 1992). There is speculation that the age at which a woman achieves PBM, and the number of years she maintains the peak mass, will vary depending upon the individual (Gordon et al., 1991).

There is a significant increase in total bone mineral and BMD during the adolescent growth spurt, which commences at approximately age 10 for girls (Gilsanz et al., 1988; Glastre et al., 1990; Marcus, 1987). Thus, factors which influence bone accretion during growth and development are important determinants of future resistance to skeletal fracture. As Riggs and Melton (1986)

have pointed out, "...insufficient accumulation of skeletal mass by young adulthood predisposes a person to fractures later in life as age-related bone loss ensues" (p. 1678).

As alluded to earlier, environmental, physical activity, nutritional and genetic factors have been identified as major determinants of PBM (Chestnut III, 1991; Ott, 1990; Pollitzer & Anderson, 1989). However, it is difficult to designate any one factor as the primary physiological signal to bone, since they all appear to operate synergistically in influencing bone mass. Halioua and Anderson (1989) illustrate this point well in their statement "...as far as peak bone mass is concerned, nutritional factors are permissive in their effects whereas mechanical loading, i.e., physical activity, exerts a modifying effect on the expression of an individual's genetic potential" (p. 539). Although each factor will be discussed separately, it is important to recognize that they are interdependent in terms of influencing PBM.

1.4.0 Environmental Factors

Briefly, environmental factors include lifestyle variables such as smoking, drug use, caffeine intake and alcohol consumption. Smoking has been found to be negatively correlated to bone density of the spine and radius in healthy premenopausal women (Aloia et al., 1988). A negative correlation has also been reported between caffeine consumption and bone mass, although this may be explained in part by the negative relationship between calcium intake and caffeine use (Toss, 1992). In healthy subjects given large doses of alcohol, as well as in

alcoholics, reductions in serum levels of osteocalcin (a marker of bone formation) have been observed. The implication is that alcohol may exert a direct inhibitory effect on the osteoblasts (Toss, 1992).

Although the preceding results were obtained in studies using adult subjects, it is nevertheless likely that smoking and excessive consumption of caffeine and alcohol may have an unfavourable influence on attainment of PBM in growing girls. Therefore, if the goal is to optimize PBM, then young girls should be advised against indulging in these habits.

1.4.1 Physical Activity

The literature contains a number of reports which identify exercise as a factor in skeletal growth and development (Chestnut III, 1991; Fehily et al., 1992; Halioua & Anderson, 1989; McCulloch et al., 1990; Ott, 1990; Ruiz et al., 1995; Slemenda et al., 1991; Slemenda et al., 1994; Toss, 1992). In general, physical activity is considered to have a beneficial influence on bone in that regular weight-bearing exercise induces an increase in PBM (Dalsky, 1990). During growth, more pronounced increases in BMD have been observed at weight-bearing skeletal sites, such as the legs (Geusens et al., 1991). Athletic children and adolescents demonstrate significantly greater BMD of the hip than their non-athletic counterparts (Kroger et al., 1992; Slemenda et al., 1991). Bone tissue hypertrophies in response to mechanical loading (above and beyond what it is normally subjected to) and reorganizes to better manage the internal stresses placed upon it (Martin &

McCulloch, 1987). Therefore, sufficient mechanical loading (i.e., exercise) appears to be a necessary ingredient in maximizing PBM in growing girls by exerting an osteogenic effect on bone. Good exercise habits are important throughout the early part of the life cycle and during young adulthood, in order to optimize genetic potential for bone development of the individual (Halioua & Anderson, 1989).

1.4.2 Nutritional Factors

There is convincing evidence to support the view that dietary calcium exerts a positive influence on accretion of bone mass in growing girls (Halioua & Anderson, 1989; Johnston et al., 1992; Matkovic et al., 1990; Matkovic et al., 1979; Rubin et al., 1989; Ruiz et al., 1995; Sandler et al., 1985; Sentipal et al., 1991) despite the fact that some researchers have been unable to establish an association between BMD and calcium intake in children (Grimston et al., 1992; Glastre et al., 1990; Katzman et al., 1991; Kroger et al., 1992; Kroger et al., 1993). There are also reports that high intakes of dietary phosphorus and protein are negatively associated with bone mass in young women (Calvo, 1993; Metz et al., 1993).

Calcium constitutes the single largest component of hydroxyapatite, the inorganic complex which imparts rigidity to bone (Dalsky, 1990). The average girl acquires 1000 g of skeletal calcium over the first twenty years of life, which translates into approximately 150 mg/day net positive balance when divided equally over this time frame (Marcus, 1987). Under conditions of suboptimal calcium

intake, PTH-stimulated remodelling activity is increased, which leads to a less positive, or possibly even negative balance situation (Matkovic, 1991; Matkovic et al., 1990). Consequently, marginal calcium intakes during the growth and development years may preclude the attainment of optimal PBM and increase the subsequent risk of fracture (Marcus, 1987).

Peacock (1991) has pointed out that in order to realize maximal PBM, calcium intake and intestinal absorption must be adequate to support skeletal growth and consolidation as well as to offset obligatory losses (i.e., in the urine, feces and sweat). The only source of calcium available to the body is that provided by the diet. Therefore, throughout childhood and adolescence it is essential that calcium be continuously supplied to the body in amounts corresponding to changing needs for growth. This will encourage healthy bone development by ensuring that the mineralization process is achieved as bone matrix is laid down.

The question arises: what are optimal dietary calcium levels to support skeletal growth and consolidation in childhood and adolescence? According to the Canadian Recommended Nutrient Intakes (RNI's) (Health and Welfare Canada, 1990), suggested daily intakes for girls are as follows: 700 mg for ages 7-9 years, 1100 mg for ages 10-12 years, 1000 mg for ages 13-15 years and 700 mg for ages 16-24 years. These levels are slightly below the American Recommended Dietary Allowances (RDA's) for calcium of 800 mg/day for girls 10 years and under and 1200 mg/day for adolescent girls (aged 11-19 years) (Schaafsma, 1992). Both the Canadian RNI's and American RDA's for calcium are considered to meet the

requirements of almost all individuals in a particular segment of the population (based on age and/or sex) and include a margin of safety to compensate for inter-individual variations (Health and Welfare Canada, 1990; Schaafsma, 1992). However, many of the reports published in the literature advocate daily calcium consumption of 1200-1800 mg/day for adolescent girls, to support a more positive calcium balance and encourage attainment of maximal PBM (Halioua & Anderson, 1989; Heaney, 1991; Marcus, 1987; Matkovic et al., 1990).

Unfortunately, there is evidence to indicate that girls habitually underconsume calcium, and the phenomenon becomes more prevalent with increasing age (Sentipal et al., 1991). The observation that calcium intake declines as young girls approach and reach puberty is disturbing in that this decrease in calcium consumption coincides with a critical time in skeletal development, when calcium requirements are at their highest. Although opinions vary as to the level of calcium which is most beneficial for bone growth and consolidation, it is generally agreed that inadequate calcium intake during childhood and particularly adolescence will impose limitations on the attainment of PBM in young women, and may predispose them to osteoporosis and fractures in later years.

Furthermore, there is some evidence suggesting that a prolonged high intake of phosphorus in combination with limited dietary calcium may result in persistent changes in calcium regulating hormones and create a hormonal environment which is not conducive to optimization of PBM (Calvo, 1993). Also, protein consumption in excess of recommended amounts may have an adverse effect on bone mass in

young women (Metz et al., 1993).

1.4.3 Hormonal Factors

Bone metabolism is affected by a number of different hormones, including PTH, calcitonin (CT), vitamin D, growth hormone (GH), insulin-like growth factor-I (IGF-I) and estrogen. PTH and CT are physiological antagonists and have opposing effects on bone. PTH is released in response to hypocalcemia and acts to increase calcium influx into the plasma. It stimulates the resorption of mineralized bone by osteoclasts and thus has a catabolic effect on bone tissue. In contrast, CT is secreted in response to a rise in plasma calcium levels. It functions to lower the plasma calcium concentration and inhibit bone resorption (Genuth, 1988). The active form of vitamin D is essential for bone health: it increases calcium absorption efficiency at the gut level (Heaney, 1987) and is also a stimulator of osteocalcin (Nielsen et al., 1990). The normal mineralization of newly formed osteoid is highly dependent on vitamin D (Genuth, 1988). GH is another important modulator of skeletal mass and is believed to exert an effect by increasing the production of IGF-I (Raisz, 1988). IGF-I is, itself, a powerful stimulator of linear bone growth (Canalis, 1993). A significant correlation has been established between osteocalcin and serum IGF-I concentrations in children (Johansen et al., 1988). GH may also regulate binding proteins for IGF-I which, in turn, mediate its activity (Raisz, 1988).

Estrogen is thought to modulate bone metabolism both directly and indirectly.

There is evidence that specific estrogen receptors exist in osteoblasts, suggesting a direct effect of estrogen on these cells (Eriksen et al., 1988). Indirectly, estrogen serves to prevent bone resorption and slow the rate of remodelling, although the precise mechanism has yet to be elucidated (Prior, 1991).

Estrogen also plays a fundamental role in pubertal progression. During the childhood years, circulating estrogen is maintained at low levels (Genuth, 1988; Wells, 1991). However, at the onset of puberty, estrogen concentration rises, as is manifested by an increase in linear growth (i.e., the adolescent growth spurt) and breast budding. Menarche (onset of menses) occurs approximately two years later (Genuth, 1988). With advancing pubertal development, estrogen stimulates closure of the epiphyseal growth plates at the ends of the long bones and thus causes a slowing of linear growth in height (Warren et al., 1986; Wells, 1991). Girls who are late to mature have a more extended skeletal growth period compared to early maturers; thus late maturing girls tend to be taller than those who mature earlier (Wells, 1991).

In some instances, strenuous exercise may disrupt the female neuroendocrine and reproductive systems which, in turn, have an impact on bone mass (Highet, 1987). For example, there is evidence that skeletal integrity may be adversely affected (predominantly at trabecular bone sites) if the exercise program or training regimen results in menstrual cycle disturbances such as amenorrhoea/oligomenorrhoea (absent or infrequent menstrual cycles) or delay of menarche (Dhuper et al., 1990; Drinkwater et al., 1984; Fisher et al., 1986; Lloyd

et al., 1988). In adolescent girls, menstrual dysfunction may have a detrimental effect on PBM attainment, thereby predisposing these active young women to premature bone demineralization, stress fractures and development of osteoporosis in later years (Chestnut III, 1991). Of particular interest is the observation that athletic girls and women with menstrual disturbances generally exhibit chronically low circulating levels of estrogen compared to their regularly menstruating counterparts (Dhuper et al., 1990; Drinkwater et al., 1984; Fisher et al., 1986; Lloyd et al., 1988). This reported hypoestrogenemia is of concern, in that it may influence bone turnover rates and lead to accelerated bone loss at a time when adolescent and young adult women should, in fact, be establishing a high level of PBM (Barr, 1987).

1.4.4 Genetic Factors

Genetic (or hereditary) factors control the basic skeletal program, including bone size and density, and are therefore thought to be the primary determinants of PBM during bone growth and consolidation (Heaney, 1987). Heredity may also influence bone status indirectly, as bone density is correlated with body size, muscle mass and hormonal levels, all of which are under genetic control (Ott, 1990; Pollitzer & Anderson, 1989). In terms of racial factors, blacks exhibit greater bone densities than whites, even after adjusting for body mass, whereas Orientals, whether American or foreign-born, have significantly less cortical bone mass than whites (McCormick et al., 1991; Ortiz et al., 1992; Pollitzer & Anderson, 1989).

Studies of monozygotic and dizygotic twins provide evidence for a genetic link in the attainment of PBM (Dequeker et al., 1987; Pocock et al., 1987). In addition, comparisons between parent-offspring pairs indicate strong associations of BMC and bone density at commonly measured sites (Lutz, 1986; Lutz & Tesar, 1990; Matkovic et al., 1990), although environmental, nutritional and physical activity factors may also serve to enhance family resemblances.

1.5 Summary

Optimizing PBM should be a major goal for all girls and young women, in order to lessen the impact of age-related bone loss and reduce the risk of osteoporosis in later years. The key determinants of PBM include environmental, mechanical loading (i.e., physical activity), nutritional, hormonal and genetic factors; although the relative contribution of each has yet to be clarified. It is likely that genetics impose a maximum limit on PBM while the remaining factors operate in concert to define the extent to which the genetic potential is expressed.

Evidence suggests that exercise and calcium consumption early in life are prerequisites for high PBM attainment in young women. Since these are two factors over which girls can exert a substantial degree of control, sufficient calcium intake and adequate exercise during pre-adolescence and adolescence represent a voluntary means of optimizing PBM.

1.6 Rationale for the Study

There is a definite need to further evaluate the issue of skeletal health in

growing girls, since attainment and maintenance of a high PBM in young adulthood is a primary strategy for protecting against osteoporosis in subsequent years (Riggs et al., 1982). Although there exists a fairly large body of research on bone density, physical activity and nutritional patterns in adolescent girls and pre- and post-menopausal women, there is less information available on these parameters in pre-menarchial girls. Thus, the effects of physical activity and calcium intake on bone mass require additional investigation in this age group.

Research examining bone density in physically active girls and young women seems to focus on specific groups of athletes such as runners, ballet dancers, swimmers, tennis players and rowers (Drinkwater et al., 1984; Fisher et al., 1986; Frusztajer et al., 1990; Jacobson et al., 1984; Kannus et al., 1995). However, there is a paucity of data available on bone density in young rhythmic and artistic gymnasts, particularly during the pre-menarchial years.

Initiation of girls into the sport of gymnastics (both rhythmic and artistic) begins at an early age (usually prepubertally). Even at the novice levels of competition (which typically include girls aged 9-11 years), training involves fairly intensive regimens of three to four sessions per week for approximately 3 hours per session. The sport of gymnastics combines athleticism (which requires a great deal of strength for execution of leaps and tumbles), artistic components and aesthetic ideals, with emphasis on technical proficiency and visual appeal (Alexander, 1991). Evaluation of physique is an integral part of judging performance, which raises the concern of gymnasts restricting food intake in an effort to achieve or maintain an

ultra-lean, lithe appearance (Benardot, 1996; O'Connor et al., 1995; Sundgot-Borgen, 1996).

Nutritional inadequacy, especially in terms of bone minerals such as calcium, may influence accretion of bone and subsequent attainment of PBM in this group of young girls. Suboptimal energy and nutrient intake could also affect BMD indirectly, by contributing to low body weight and percent body fat. Conversely, the weight-bearing exercise which gymnasts regularly engage in may serve to promote bone modelling and therefore enhance accumulation of bone mass in these active girls. Thus, it is important to examine the effects of physical activity, calcium intake and anthropometric parameters on bone mass in young gymnasts.

1.7 Statement of the Problem

The main purpose of this study was to compare BMD in competitive pre-menarchial rhythmic and artistic gymnasts aged 9-13 years with BMD in normally active female controls of the same age and pubertal status, to determine if intensive physical training exerts an effect on bone mass in pre-menarchial girls. It was hypothesized that girls who engaged in less physical activity (i.e., normally active control subjects) would exhibit lower BMD, while those who took part in higher levels of physical activity (i.e., gymnasts) would demonstrate greater BMD.

Of further interest was to clarify the relationship between BMD and age, height, weight, bone-free lean tissue, exercise and calcium intake in pre-menarchial gymnasts and their normally active counterparts.

1.8 Delimitations

Subjects were delimited to pre-menarchial Caucasian girls, aged 9-13 years, whose breast development corresponded to Stage 1, 2 or 3 of Tanner's (1962) breast development scale. The gymnasts were delimited to national and provincial stream athletes training a minimum of 9 hours/week at a novice or junior level with a local Winnipeg rhythmic or artistic gymnastics club. Control subjects were delimited to normally active girls who engaged in a maximum of 5 hours/week of structured physical activity (i.e., participation in school physical education classes, school and club teams, and lessons).

Exclusion criteria were as follows: achievement of menarche; breast development corresponding to Stage 4 or 5 of Tanner's (1962) breast development scale; presence of a medical condition(s) which may have affected bone metabolism (such as diabetes, renal disease, arthritis, celiac disease, etc.); the chronic use of any medication which may have influenced bone metabolism (e.g., glucocorticoids for asthma); history of restricted food intake; the presence of eating disorder symptoms; smoking; caffeine use and alcohol consumption. Given the young age of the subjects, these last three criteria were not expected to pose a concern.

BMD measurements and determination of body composition were delimited to DXA of the lumbar spine, proximal femur and total body. Nutritional information was delimited to collection of one 3-day food record (documenting intake for two

weekdays and one weekend day), which was analyzed for intake of energy, protein, fat, carbohydrate, calcium, phosphorus and vitamin D. Anthropometric measurements were delimited to height, weight, sum of 7 skinfold thicknesses (triceps, biceps, subscapular, suprailiac, abdominal anterior mid-thigh and medial calf), body circumferences, bone lengths and bone breadths.

1.9 Limitations

The main limitation with this study was that it employed a cross-sectional design whereby subjects were recruited on the basis of two discrete levels of physical activity (i.e., participation in either intensive gymnastics training or low to moderate involvement in structured physical activity). Since subjects were self-selected, they were not necessarily representative of the population from which they were drawn. Consequently, the study's external validity is limited, and caution must be used when interpreting the data and generalizing the findings to other groups.

A second limitation was that the study employed questionnaires as a means of gathering data on medical history, past and present physical activity patterns, dietary habits and consumption of calcium-rich foods. Given the retrospective nature of some of the questions, the collection of accurate data depended on the ability of each subject and her parent(s) to recall and report past events. Furthermore, the validity of the self-report tool used to screen for the presence of eating disorder symptoms depended on the degree to which subjects answered honestly.

1.10 Definition of Terms

Bone Mineral Content (BMC):

Bone mineral content is defined as the total quantity of bone mineral measured per unit length of bone, and is expressed as g/cm of hydroxyapatite.

Bone Mineral Density (BMD):

Bone mineral density is defined as the total amount of bone mineral in a projected area of bone (areal density), and is calculated by dividing the quantity of bone mineral (BMC) by the projected area within the region of interest. BMD is expressed as g/cm² of hydroxyapatite.

Menarche:

Menarche is defined as the initiation of menstrual function as indicated by the first menstrual cycle. Menarche occurs at a mean age of 12.75 years (Tanner, 1962), and usually coincides with stage 4 of the Tanner sexual maturation scale.

Pre-menarche:

Pre-menarche is defined as the state in which initiation of menses has not yet occurred.

Structured Physical Activity:

Structured physical activity is defined as participation in school physical education classes, on school and club teams, and in lessons and practice sessions. For gymnasts, structured physical activity includes gymnastics training.

2. REVIEW OF THE LITERATURE

2.0 Introduction

The purpose of this review of the literature is as follows: (1) to provide a detailed discussion of the key factors involved in the determination of PBM in young girls and women with an emphasis on physical activity, hormonal status, calcium intake and genetics, (2) to describe the association between BMD and developmental parameters in children and adolescents, (3) to consider variability of BMD in children, and (4) to critically evaluate the recent research pertaining to (1), (2) and (3) above, in terms of methodologies and interpretation of results. In addition, techniques routinely used to measure bone mass will be reviewed in depth.

2.1 Techniques Used to Measure Bone Mineral Status

As mentioned earlier, radiogrammetry (RG), quantitative computed tomography (QCT), single photon absorptiometry (SPA), dual photon absorptiometry (DPA), and dual energy x-ray absorptiometry (DXA) are methods frequently employed to measure BMC and BMD.

RG utilizes standardized x-rays to determine the extent of bone formation at the hand or wrist. Although it provides a gross indication of cortical bone status, it cannot significantly predict bone mass at the spine or hip (i.e., trabecular bone) nor can it forecast future risk for fracture (Chestnut III, 1987; Firooznia et al., 1989).

QCT uses radiographs to generate cross-sectional images of a specific region of interest (e.g., vertebral bodies of the lumbar spine) (Health and Public Policy Committee, 1987). The average density of a region is then referenced to that of a bone mineral calibration phantom. The most important advantage of this technique is that it can isolate purely trabecular bone of the vertebrae (Firooznia et al., 1989; Mazess, 1990). Furthermore, QCT is the only non-invasive bone mineral densitometric method to measure true three dimensional density rather than areal density (Genant et al., 1991). However, precision with QCT ranges from approximately 3% in young normal subjects to 7-15% in osteoporotic patients. Precision refers to the ability of the measuring instrument to reproduce the same value when measurements are repeatedly performed. Accuracy with QCT is poor (12-30%), mainly due to the confounding effect of bone marrow fat which can falsely lower vertebral bone density. Accuracy is the extent to which the measured BMD value reflects the actual (or true) BMD value in the bones assessed (Mazess & Barden, 1988). Radiation exposure with QCT is high, ranging between 200 and 1000 mrem per scan (Wahner, 1989). Given the substantial radiation dose, QCT may not be practical for evaluating vertebral BMD in children or for repeatedly measuring BMD in single subjects.

SPA employs an iodine-125 radionuclide source which emits at a single energy. The radiation beam is capable of distinguishing between bone and soft tissue, providing that the thickness and composition of the soft tissue around the bone are uniform (Wahner, 1989). To ensure a constant soft tissue thickness,

water or tissue-equivalent gel is used to surround the appendage (Wahner, 1989). The radiation dose with SPA is low (5-10 mrem), routine precision ranges between 1 and 3%, and the accuracy error has been reported as 5-10% (Mazess & Barden, 1989; Riggs & Melton, 1986; Wahner, 1989). However, SPA is only accurate at sites with very little soft tissue (e.g., the radius, ulna and os calcis) and is therefore limited to measuring primarily cortical bone (Health and Public Policy Committee, 1987; Mazess & Barden, 1989; Riggs & Melton, 1986). Although the ultra-distal radius contains the same proportion of trabecular bone as the spine, trabecular bone at this appendicular site is not metabolically active (Wahner, 1989). Measurements at regions in the appendicular skeleton cannot accurately predict spinal bone mineral content, as correlation between the two is low (Kelly et al., 1988; Riggs & Melton, 1988).

DPA measures both cortical and trabecular bone (although it cannot differentiate between the two), and is an appropriate modality for determination of bone mineral status of the spine, hip and entire skeleton. This method utilizes a rectilinear scanner with an isotope source (gadolinium-153) that emits at two photoelectric peaks (44 keV and 100 keV) (Riggs & Melton, 1986; Wahner, 1989). The use of pairs of energies adjusts for variable amounts of soft tissue in the scan path (Mazess, 1990). DPA offers good reproducibility (2%) and accuracy (5-10%), and radiation exposure is low (5-10 mrem) (Genant et al., 1991; Riggs & Melton, 1986; Wahner, 1989). However, DPA measurements can be affected by the gadolinium-153 photon source, which constantly decreases in intensity over time

and must periodically be replaced (Kelly et al., 1988; Wahner et al., 1988). In addition, it is a relatively time-consuming method, requiring 20-40 minutes for a hip or lumbar spine scan and 60 minutes for a total body scan (Wahner, 1989). Also, DPA values depict bone mineral areal density (i.e., g/cm^2) rather than true volumetric density (i.e., g/cm^3), although the two are closely correlated (Fogelman & Ryan, 1992).

DXA represents the most recent advance in absorptiometry and employs an x-ray tube rather than a radionuclide source to produce the photon beam (Fogelman & Ryan, 1992). This provides photon flux which is 500-1000 times higher than that seen with DPA (Wahner et al., 1988). Unlike the isotope source used in DPA, output from the x-ray tube is stable and does not diminish in strength with time. The higher photon flux with DXA allows for enhanced spatial resolution (2 vs. 4 mm), better imaging, faster scanning speeds and less radiation exposure (<5 mrem) in comparison with DPA (Mazess & Barden, 1988; Wahner, 1989). DXA also offers a smaller precision error (1%) than DPA, while accuracy is reported to range from 4-8% (Genant et al., 1991; Mazess & Barden, 1988; Wahner, 1989). DXA is a suitable modality for measuring bone mass of the spine, hip and total body, although it cannot distinguish between trabecular and cortical bone (Wahner, 1989). Tissue thickness and composition have a negligible influence on the results (Sartoris & Resnick, 1988). Approximately 6 minutes is required to complete a DXA scan of the lumbar spine or hip, while a total body scan can be done in about 20 minutes (Wahner, 1989). However, as with DPA, DXA measures bone mineral per

surface area scanned (i.e., g/cm²) instead of true volumetric density (i.e., g/cm³) (Wahner, 1988).

Three different brands of DXA systems are currently available, but vary in terms of the method used to generate x-rays, the resultant effective beam energies, and the hardware and software arrangements used for data collection and analyses (Kellie, 1992; Mazess & Barden, 1988; Roubenoff et al., 1993; Sartoris & Resnick, 1988). Quantitative digital radiography (QDR), manufactured by Hologic, Inc., generates dual photon flux by alternating kilovoltages between 70 and 140 kVp. The beam gives effective energies of 43 and 110 keV (Mazess & Barden, 1988; Sartoris & Resnick, 1988). QDR features an automatic internal reference system which compensates for drifts in measurement (Glastre et al., 1990). The DPX device (manufactured by Lunar Radiation Corp.) and the XR-26 system (produced by Norland Corp.) utilize selective constant-potential K-edge filters at a fixed kilovoltage to generate photons at two energies (Kellie, 1992). For the DPX instrument, the resultant effective beam energies are 40 and 70 keV (Mazess & Barden, 1988; Sartoris & Resnick, 1988). Spinal BMD values obtained with the DXA system from Lunar (i.e., DPX) are reported to be directly comparable to those measured by DPA (Sartoris & Resnick, 1988). However, spinal BMD results with the Hologic device (i.e., QDR) have been shown to be 6-12% lower than DPA measurements, apparently due to differences in instrument calibration, bone edge detection and bone area (Mazess & Barden, 1988; Wahner, 1989). Wahner et al. (1988) have stated that conversion of data is necessary when comparing

measurements obtained from QDR and DPA instruments. DXA BMD values are not reliable predictors of bone densities measured by QCT (Sartoris & Resnick, 1988).

2.2 Assessment of Body Composition by DXA

In addition to assessing bone mineral status, DXA is an accurate and precise method of measuring soft tissue body composition (Fuller et al., 1992; Svendsen et al., 1993). The ratio of absorbance of photons at two different energy levels is linearly correlated to percent fat in the soft tissues of the body (Mazess et al., 1990). DXA has been validated for measurement of three principle body components (i.e., bone, bone-free lean soft tissue and fat). This is in contrast to many other body composition assessment techniques which measure one component and then extrapolate the findings to other compartments (Haarbo et al., 1991). The precision error for DXA measurements of lean tissue mass has been reported as 1.5% and 2% for the total body and sub-regions (i.e., arms, legs and trunk), respectively (Mazess et al., 1990). In terms of accuracy, Haarbo et al. (1991) have shown agreement between fat percentage and lean body mass by DXA and three other methods (total body potassium, underwater weighing and DPA). The accuracy error of predicting true percent fat from DXA values has been found to be 4.9%, which is considered satisfactory for measurements of single subjects (Haarbo et al., 1991). Agreement between percent body fat values determined by DXA and skinfold anthropometry has also been observed in children (Ogle et al., 1995).

2.3 Factors Affecting Bone Mass in Girls and Young Women

2.3.0 Physical Activity and Hormonal Factors

In the following section, physical activity will be discussed in conjunction with hormonal status (specifically, estrogen levels) as these two factors appear to be closely linked in girls and young women. Exercise may exert both a positive and a negative effect on bone mass in growing girls, depending upon the hormonal milieu.

The geometry and architectural arrangement of bone reflects a functional adaptation to normal stresses. However, through molecular, cellular and metabolic changes, bone has the capability of adapting to new physiological loading conditions (Einhorn, 1992). Exercise represents a situation of weight-loading above and beyond that which bone is typically subjected to. Chronically stressing bone through physical activity and increased loading acts as an osteogenic stimulus for bone tissue hypertrophy and re-arrangement (Martin & McCulloch, 1987). Through these adaptive processes, bone can adjust its internal structure to better manage the new strains placed upon it.

Several reports in the literature have supported a role for exercise in maximizing PBM in growing girls and young women (Dalsky, 1990; Fehily et al., 1992; Halioua & Anderson, 1989; Kannus et al., 1995; Kroger et al., 1992; McCulloch et al., 1990; Ott, 1990). Conversely, a lack of mechanical loading or physical inactivity is thought to inhibit the bone formation process, and may result in a suboptimal bone mass (Dalsky, 1990).

McCulloch et al. (1990) measured calcaneal bone density of 101 healthy female subjects (aged 20-35 years) to evaluate possible effects of physical activity on bone density. Data were gathered on childhood physical activity levels via questionnaire, while a two week recall was used to assess current exercise levels. The researchers were careful to establish reliability and validity of the data collection instruments they employed. QCT was utilized for determination of trabecular BMD of the os calcis. Due to its high content of trabecular bone, the os calcis provided a more sensitive location for detecting early bone loss than sites composed primarily of cortical bone. Results indicated significantly higher bone densities for subjects who had been involved in organized sports or fitness programs as children versus those who had not. Furthermore, subjects who were very active as children demonstrated significantly higher bone densities compared to subjects who had been less active. Childhood physical activity seemed to have a more pronounced influence on bone density than current exercise levels in these subjects. Based on their findings, this group concluded that mechanical stress (i.e., physical activity) during the growing years is a significant determinant of calcaneal bone density in young women.

Results of a study by Kannus et al. (1995) lend further credence to the concept that exercise during the early part of the life cycle exerts a positive influence on bone mass accretion. This investigation analyzed the effect of biological age at which training was initiated (relative to age at menarche) on differences in bone mass between the playing and non-playing arms of female

racquet sport competitors. Subjects were 105 nationally-ranked female tennis and squash players (with a minimum of 5 years of training) and 50 non-athletic female controls aged 16-50 years. Players were divided into sub-groups according to the number of years before or after menarche at which their playing careers commenced. DXA was used to measure BMC of the proximal humerus, humeral shaft, radial shaft and distal radius in both arms. Results indicated that BMC of the dominant arm was significantly greater in players than controls, although non-dominant arm BMC values were similar for the two groups. The difference between BMC of the dominant and non-dominant arm was significantly greater at every measured site in players compared to controls. When sub-groups of players were examined, side to side BMC differences were highly significant regardless of age at which training was initiated. Interestingly, the mean BMC difference (between arms) of player sub-groups was observed to decrease with increasing biological age at which playing commenced. The difference was two to four times greater in players who began their playing careers before or at menarche than those who started more than 15 years post-menarche. Given their findings, these researchers concluded that physical activity during the pubescent years is critical for maximizing bone mass.

Halioua and Anderson (1989) examined physical activity habits and lifetime calcium consumption in 181 healthy, pre-menopausal Caucasian women (aged 20-50 years) to determine the independent and synergistic effects of these variables on BMC and BMD of the forearm. Current and past activity levels were determined

via questionnaire and combined to provide a lifetime physical activity rating. Levels were classified as sedentary, moderate or active. Current and past calcium intakes (estimated from food frequency questionnaires) were also combined to yield a lifetime calcium intake variable, which was categorized as being low (<500 mg/day), intermediate (≥ 500 mg and <800 mg/day) or high (≥ 800 mg/day). BMC and BMD of the distal and mid radius were measured by SPA. Results of regression analysis showed that lifetime physical activity habits and lifetime calcium consumption were both significant positive predictors of BMC and BMD at the forearm, whether the independent variables were analyzed together or separately. Maximal values for both the distal and mid radius were seen at high physical activity levels and intermediate or high calcium intakes. In view of their results, the authors deduced that good exercise habits and adequate calcium consumption are important during the early portion of the life cycle, in order to optimize PBM. However, one criticism with this study pertains to the fact that only sites of the appendicular skeleton were chosen for determination of bone mass. Consequently, the researchers were measuring primarily cortical bone, which is less susceptible to demineralization than the more sensitive trabecular bone of the axial skeleton (Chestnut III, 1987; Dalsky, 1990; Wahner, 1989).

Fehily et al. (1992) conducted a 14 year follow-up study of low socio-economic status children (males and females) who had participated in a randomized control trial to test for the effect of a milk supplement on childhood growth. The main focus of the follow-up investigation was to determine if childhood

calcium consumption had an impact on adult bone mass. Interestingly, after collecting and analyzing data on subjects' past and present involvement in sports activities, they reported a significant positive correlation between radial BMD and the amount of time devoted to playing sports at age 12 years. BMD was 4% greater in female subjects who had engaged in more than 7 hours of sports/week than those who played sports only one hour/week. The association was independent of body weight, and was stronger than the relationship between BMD and current sports participation. In terms of calcium's influence on bone mass, Fehily's group was unable to detect a significant difference in BMC and BMD values between subjects who had received the calcium supplement (i.e., 228 mg of calcium in the form of 190 mL of milk) for two years during childhood, and those who had not. However, they suggested that more than two years of supplementation may be necessary to induce a significant effect in bone. They also speculated that the magnitude of the effect may have been larger had the supplement been administered during adolescence (when the rate of bone mineralization is greatest). Again, it is worth pointing out that differences in bone mass between the calcium supplemented and control groups may have been evident had BMD been measured at trabecular skeletal sites (such as the lumbar spine) instead of cortical regions (i.e., the radius).

Results of a cross-sectional study by Kroger et al. (1992) lend additional support to the premise that adequate mechanical loading helps to promote bone mass accretion during growth. These researchers used DXA to measure BMD of

the lumbar spine and femoral neck in 84 healthy children and adolescents (40 males and 44 females), aged 6-19 years. Subjects were assigned to one of three exercise groups based on their level of physical activity: little or no physical activity outside of school (class I); sports/physical activity at least 3 hours/week (class II); and a minimum of 5 hours of sports/week in athletic clubs (class III). After adjusting BMD measurements for age, height and weight, a comparison of mean BMD between the three exercise groups showed significantly greater femoral neck values in the class III (i.e., highly physically active) exercise group compared to classes I and II. Class III subjects also exhibited higher spinal BMD values than the less active children in classes I and II, although differences between the groups were not significant. Based on their results, the authors concluded that "...physical activity may be an important determinant of bone density and peak bone mass in children and adolescents" (p. 84).

In examining BMD in a sample of healthy white children and young adults, Geusens et al. (1992) reported that increases in bone density from 3-9 years to 20-25 years were more dramatic at weight-bearing skeletal sites (such as the legs and lumbar spine) compared to the arms and radial regions. This provides further evidence that mechanical loading of the skeleton during the growth and development years seems to stimulate bone modelling and enhance acquisition of BMD.

Grimston et al. (1993) set out to establish if high impact weight-bearing activities had a greater impact on BMD in children than activities which actively

load the skeleton through muscle contraction. They compared BMD in 17 children (8 boys and 9 girls) who competed in impact loading sports (impact load group) and 17 children whose sport specialty was swimming (active load group). Groups were matched for gender, pubertal stage and body weight, and all subjects engaged in a minimum of 3 training sessions (60 minutes/session) per week. BMD of the lumbar spine and femoral neck was measured by DPA. Results of *t*-tests revealed that femoral neck BMD was significantly greater for the impact load versus active load group, although no difference was detected for lumbar spine BMD between groups. The authors attributed the lack of statistical difference at the lumbar site to the low number of subjects in their study. In terms of the higher femoral neck BMD observed in the impact load subjects, Grimston interpreted this finding to mean that there may be a site specific influence of mechanical loading on bone. In addition to the small sample size employed in this investigation, another limitation is the failure to include a non-athletic control group in the study design. Such a group would have permitted conclusions to be drawn regarding BMD of both impact load and active load athletes relative to more sedentary children.

2.3.1 Site Specific Effects of Physical Activity on Bone Mineral Density

A number of research teams have observed significantly greater BMD's at specific sites in female athletes who compete in weight-bearing sports compared to non-athletic girls and women, thus providing further evidence that loading of the skeleton through exercise confers a benefit to bone mass (Fehling et al, 1995;

Kirchner et al., 1995; Slemenda & Johnston, 1993). Slemenda and Johnston (1993) measured regional and total body BMD with DXA in 22 competitive female figure skaters aged 11-23 years, and 22 non-athletic controls of the same age and body weight. Although no significant effects of skating were seen for BMD's of upper body sites, leg and pelvis BMD's were significantly greater in skaters versus control subjects. In view of their findings, they speculated that the mechanism responsible for the site specific increase in BMD in skaters likely involves intensive training consisting of repetitious movements (such as jumping) which place considerable impact on the body.

Kirchner et al. (1995) compared BMD's of 26 competitive female collegiate gymnasts, aged 18-22 years, with those of 26 age-, height- and weight-matched non-athletic controls. DXA was employed to quantify BMD of the lumbar spine, proximal femur and total body. The key finding in this cross-sectional analysis was that gymnasts demonstrated significantly higher BMD's than controls at all sites measured. Since the gymnasts had been competing for an average of 11.1 years, the authors surmised that long-term gymnastics training, initiated prior to and continued throughout the time of rapid bone mineral accretion, contributed to the high BMD's observed in this group. They suggested that the impact and resistance created by landing, swinging and tumbling during gymnastics participation likely induces an osteogenic effect on bone mass of the spine and hip.

Further confirmation that weight-bearing activity is advantageous to bone mass at specific skeletal locations is provided by Fehling et al. (1995). This

research team compared lumbar spine, proximal femur and total body BMD in four groups of young college women: volleyball players (n=8), gymnasts (n=13), swimmers (n=7) and non-athletic controls (n=17). Bone densitometry was done with DXA instrumentation. When BMD values were adjusted for differences in height and weight between groups, results of analysis of variance revealed significantly greater BMD's of the lumbar spine, proximal femur, total body, legs and pelvis in volleyball players and gymnasts compared to swimmers and controls. Gymnasts exhibited significantly higher adjusted BMD values for the arms, in comparison with all other groups, a finding which the authors attributed to the unique way in which gymnasts mechanically load their arms (i.e., by using the arms to support the body or sustain repeated movements of high impact). The authors pointed out that although swimmers utilize their arms to propel their bodies through water, this kind of active loading does not appear to confer any benefits in terms of bone mass. In fact, it is interesting to note that swimmers did not differ from control subjects in any measure of BMD. Based on their findings, Fehling's group concluded that loading of the skeleton through impact activities results in site specific increases in BMD.

Despite proof to support a favourable influence of physical activity on bone mass in growing girls and young women, reports in the literature suggest that rigorous exercise may somehow interfere with the female neuroendocrine and reproductive systems and thus have a negative impact on bone density (Dueck et al., 1996; Highet, 1987). Girls and women who participate in intensive exercise

programs, whether as high-performance competitors or recreational athletes, experience an increased incidence of delayed menarche and menstrual abnormalities in comparison with sedentary females (Barr, 1987; Dueck et al., 1996; Nattiv & Mandelbaum, 1993; Warren et al., 1986). Reports indicate that the occurrence of menstrual abnormalities rises from 2-5% in the sedentary population to 44% or more in dancers and runners (Barr, 1987; Dueck et al., 1996; Loucks & Horvath, 1985). Menstrual dysfunction is not a discrete entity, but rather, encompasses several disorders which occur on a continuum (from a shortened luteal phase initially, to anovulation, to oligo- and amenorrhoea) (Dueck et al., 1996; Prior et al., 1990). Although luteal phase deficiency and anovulation may not manifest themselves overtly (i.e., they are masked by seemingly normal menstrual cycles at regular intervals), Barr (1987) has reported that both conditions are associated with a decreased exposure to high circulating levels of estrogen and progesterone which are normally present in the luteal phase of the menstrual cycle. Estrogen serves to prevent bone resorption and slow the rate of remodelling while progesterone is thought to facilitate bone formation and accelerate remodelling (Prior et al., 1990). Consequently, the presence of asymptomatic menstrual disturbances could be a potential risk for excess bone loss in female athletes (Dueck et al., 1996; Prior et al., 1990). The concern regarding the possible deleterious effect of menstrual dysfunction on skeletal integrity in women athletes is no longer limited to oligomenorrhoea and amenorrhoea; it now includes the more subtle conditions of luteal inadequacy and anovulation.

Prior to a more in-depth analysis of the effects of menstrual status on bone density in physically active girls and young women, a brief description of hormonal events during the prepubertal and pubertal years is in order.

2.3.2 Maturation of the Reproductive System in Prepubertal and Pubertal Girls

The gonadotropins follicle stimulating hormone (FSH) and luteinizing hormone (LH) are secreted by the anterior pituitary and are involved in menstrual cycle and gonadal regulation. In the mature female, FSH stimulates the growth and development of the primary follicles in the ovary, while LH promotes estrogen production and secretion, ovulation and formation of the corpus luteum. Gonadotropin releasing hormone (GnRH) is a peptide hormone synthesized in the hypothalamic neurons and released in pulsatile fashion to stimulate secretion of FSH and LH (Wells, 1991). During the childhood years, a very sensitive negative feedback system is in operation. Small levels of circulating gonadal steroid hormones (i.e., estrogen) ensure that the intermittent release of GnRH occurs at a slow rate. Consequently, gonadotropins are maintained at very low levels (Genuth, 1988; Wells, 1991).

The initiation of reproductive function is characterized by a reduced sensitivity to the negative feedback mechanism at the level of the hypothalamus. In other words, hypothalamic neurons that produce GnRH become gradually desensitized to suppression by gonadal steroids. Release of LH dramatically rises in response to increased pulsatile discharges of GnRH. This, in turn, stimulates

increased gonadal steroid production (Wells, 1991). The rise in estrogen concentrations coincides with the first observable physical signs of puberty (i.e., initiation of the growth spurt and breast budding). Menarche occurs approximately two years later, after a further substantial increase in LH levels is evident (Genuth, 1988). Maturation of the hypothalamic-pituitary-ovarian axis is considered to be complete when a positive feedback system of estrogen on gonadotropins is in place, and ovulation occurs (Genuth, 1988; Wells, 1991).

There is a substantial degree of variation in the age at which the adolescent growth spurt begins in girls (Tanner, 1962). Early-maturing girls may demonstrate complete adolescent development before late maturers, of the same age, exhibit any sign of development whatsoever. Taking into account the wide age range for normal sexual maturation in girls, Tanner devised a sequencing scheme for the inter-related events of puberty (i.e., breast development and enlargement, appearance of pubic hair, growth in height and achievement of menarche). According to this model, breast development can be categorized into five stages, ranging from prepubertal (stage 1) to mature (stage 5). Appearance of the breast bud (stage 2) is typically seen between the ages of 8 and 13 years (average age 11 years). Menarche is one of the last events in pubertal development, occurring at an average age of 12.75 years (Tanner, 1962). Tanner has noted that complete reproductive function is usually not achieved until approximately one year after menarche, as the first few menstrual cycles a girl experiences are often anovulatory. Several groups of researchers investigating the issue of bone

mineralization in children have employed Tanner's sexual maturation staging in order to rate the degree of pubertal development in their young subjects (DeSchepper et al., 1991; Glastre et al., 1990; Grimston et al., 1992; Katzman et al., 1991; Sentipal et al., 1991; Southard et al., 1991).

2.3.3 Delayed Menarche, Physical Training and Bone Health

Several studies have documented that menarche occurs at a significantly later age in young athletes as compared to their non-athletic counterparts (Frisch et al., 1981; Malina et al., 1978; Vandenbroucke et al., 1982; Warren, 1980). However, it is unclear whether the later menarchial age observed in athletes results from prepubertal physical training or if it is associated with factors that confer enhanced performance.

According to results of a study by Malina et al. (1978), the more competitive and highly skilled the athlete, the greater the delay in menarche seems to be. When these researchers compared age at menarche in non-athletes, high school athletes, college athletes and Olympic volleyball candidates, they discovered that the non-athletes achieved menarche significantly earlier than the three groups of athletes. A further comparison of just the athletic groups revealed that the Olympic athletes had a significantly later menarchial age than both the high school and college athletes. To explain the later age at menarche seen in athletic girls, the authors developed a plausible two part hypothesis. First, they pointed out that the late maturing girl generally has longer legs and narrower hips than her early

maturing counterpart, and this type of physique is usually better suited to athletic success. Second, they postulated that the early maturer may be socialized away from sport participation, whereas the late maturing girl may not experience the same degree of pressure to conform to socially acceptable ideals. As a result, the late maturer may be more inclined to continue on in athletics to increasingly advanced levels of competition.

Frisch et al. (1981) contend that athletes experience menarche at an older age due to prepubertal athletic participation, with the magnitude of the delay being proportional to the number of years of training prior to menarche. Stager et al. (1984) disagree with this viewpoint, arguing that the older an athlete is at menarche, the greater the likelihood that training will be implicated as the causal factor. Conversely, a girl who experiences menarche at an early age will have had fewer years of pre-menarchial training.

Stager et al. (1984) were interested in determining if the later menarche in athletes is associated with prepubertal training, or if it is due to a performance related selection phenomenon in which those who mature later have a competitive advantage over early maturers. They rationalized that if menarche is delayed by early training, then athletes who commence training at a young age should exhibit the effect, regardless of athletic ability. On the other hand, if performance and menarchial age are related, then the best performers should have a later age of menarche. Competitive swimmers, ranging in age from 12-22 years, were selected as the athletic group (n=287). Because swimming tends to be an early entrance

sport, the authors reasoned that swimmers should demonstrate pronounced effects of prepubertal training. Although a non-athletic, age-matched control group ($n=495$) was also included in the study, many of the control subjects indicated sports participation in the past, which could have confounded the results or clouded interpretation of study findings. Results of a *t*-test indicated that the mean age at menarche of swimmers as a group was significantly later than the controls (13.4 years vs. 13.0 years, respectively). When swimmers were assigned to groups on the basis of performance in specific swimming events, the better competitors were found to have a significantly later age at menarche than the less talented performers. Thus, Stager's group concluded that athletic ability in swimming and age at menarche are related.

Frisch et al. (1981) examined the age at menarche and menstrual cycle regularity in 21 college long- and middle-distance runners, in relation to the age at which training was initiated. Athletes were classified into two groups (those who began training before menarche [$n=18$] and those who commenced training following menarche [$n=20$]), and were studied over the course of the training season. Ten non-athletic college women were recruited to act as controls, although their activity level prior to menarche was not established. Subjects were categorized as having primary amenorrhoea (no menarche to-date), secondary amenorrhoea (6 month interval between cycles), irregular cycles (differing in length by 9 or more days) or regular cycles. Results indicated that the mean menarchial age of the athletes who commenced training before menarche was significantly

delayed in comparison to post-menarchially trained athletes and controls (15.1 years, 12.8 years and 12.7 years, respectively). Furthermore, for the pre-menarchially trained athletes, menarche was postponed 0.4 years for each year of training prior to menarche. At the beginning of the training season, 61% of the pre-menarchially trained athletes had irregular cycles and 22% were amenorrhoeic. Sixty percent of the post-menarchially trained athletes had regular cycles, 40% had irregular cycles and none were amenorrhoeic. However, at the end of the season, the incidence of oligomenorrhoea and amenorrhoea rose for both groups of athletes. The authors attributed this increase to the effect of intensive training. Unfortunately, changes in body weight or body fat over the season were not reported, although it is possible that either or both may have been involved in disruption of menstrual cycles in some of the athletes. Frisch concluded that athletic training is a potential cause of menstrual dysfunction in athletes, especially if training was initiated prior to menarche.

The research studies described above illustrate that intensive training pre-menarchially may impede normal pubertal progression, although the theories put forth in an effort to explain this phenomenon are speculative and controversial. It is clear, however, that delayed menarche represents the inability of the hypothalamic-pituitary-ovarian axis to cyclically produce sufficient quantities of the hormones necessary for menstrual function (Kustin & Rebar, 1987), which raises an interesting question: is delayed menarche harmful in terms of achieving optimal PBM?

2.3.4 Delayed Menarche and Peak Bone Mass

In an attempt to answer this question, Warren et al. (1986) studied the effect of delayed menarche on bone status in 75 professional ballet dancers, aged 18-36 years. Twenty-four percent of the dancers were found to have scoliosis (i.e., curvature of the spine). Linear regression analysis demonstrated a significant positive correlation between age at menarche and prevalence of scoliosis. In a more detailed examination of a sub-group of 40 dancers, it was noted that those who had sustained stress fractures were significantly older at menarche than those who had not experienced such injury. Based on their findings, these researchers deduced that delayed menarche in young dancers constitutes a risk factor for scoliosis and fractures. They speculated that chronic hypoestrogenism in adolescent girls may postpone "...maturation of the osseous centres in the spine and predispose [the] person to vertebral instability and curvature" (p. 1352). In addition, estrogen deficiency may cause a decrease in bone apposition so that bone density is lower than normal at the time of maturation. Consequently, the skeleton may be more susceptible to fractures.

Dhuper et al. (1990) conducted a two-part study which examined factors affecting peak bone density in females. The first part of the research was a cross-sectional investigation to determine the effects of estrogen exposure and pubertal progression on BMD of the spine, wrist and foot in 43 white girls, aged 13-20 years. Subjects were selected to represent a wide range of pubertal development and exercise levels. Twenty-eight of the girls were dancers while the remaining 15 were

non-dancers. As well as examining hormonal status of their subjects, the researchers evaluated nutritional intake and the existence of eating disorders such as anorexia nervosa and bulimia. BMD of the radius was measured by SPA, while DPA was used to quantify bone density of the lumbar spine and first metatarsal. To assess integrated estrogen exposure, they employed a scoring system which was based on several variables that purportedly reflected circulating estrogen levels during adolescence. Girls who were pre-menarchial, over the age of 14 years at menarche or amenorrhoeic received a lower estrogen exposure score than those who had achieved menarche prior to age 14 years and experienced normal menstrual cycles. In addition, girls with low serum estrogen levels were assigned low scores, while those with higher estrogen concentrations were given correspondingly higher scores. The scoring system was intended to evaluate how pubertal progression interfaces with bone mass. Results of regression analysis demonstrated that both weight and estrogen exposure were significantly and positively correlated to bone density of the spine and wrist. Further statistical tests revealed that the effects of weight and estrogen exposure on bone density were interdependent. The researchers speculated that since puberty is highly associated with weight gain, lower weights may be indicative of a delay in pubertal progression.

The second part of Dhuper's study was undertaken to identify variables which played an important role in determining bone density at maturity. It involved a longitudinal analysis in which a subset of subjects ($n=24$) aged 18-20 years was observed for a two year period. The girls were divided into low, medium and high

estrogen exposure groups, depending upon their scores. Results showed that the groups did not differ with respect to number of eating disorders; intake of calcium, vitamin D and calories; and dietary composition (i.e., percent of total calories derived from protein, carbohydrate and fat). However, girls in the low estrogen exposure group exhibited significantly lower bone densities at the spine and wrist sites than those in the medium or high groups. When subjects were separated into low and high bone density groups, the researchers were surprised to find that girls with a high degree of activity had the lowest bone densities as well as lower estrogen scores than less active girls. They therefore surmised that "...activity alone is insufficient in maintaining bone density of stressed bones, and estrogen deficiency may be a contributing factor" (p. 1087). In reaching their conclusions, the authors asserted that lower bone density in late adolescence is primarily due to a low estrogen status during puberty, although other factors such as thinness and a high level of physical training may also exert a negative effect on bone mass. Unfortunately, one potentially serious problem with this study relates to the scoring system used to measure integrated estrogen exposure in the subjects. Even though the researchers provided a relatively detailed description of the system, they failed to establish reliability and validity of this instrument. Consequently, the ability of the estrogen exposure scores to accurately assess the relationship between pubertal progression and bone mass is questionable.

To summarize the findings of the preceding studies, it would appear that bone health in adolescent girls is compromised by the existence of a

hypoestrogenic state. While sufficient mechanical loading (i.e., exercise) appears to be a prerequisite in maximizing PBM in growing girls, it apparently cannot substitute for a lack of estrogen. Thus, there is little doubt that estrogen is inexorably linked to bone status in young women.

2.3.5 Calcium Intake

The process of bone modelling involves a net accumulation of calcium by the skeleton. There is general consensus in the literature that adequate dietary calcium intake during the growth and development years is a necessary ingredient for accretion of PBM. However, the importance of calcium in relation to other factors which affect acquisition of bone mass has yet to be defined. Furthermore, the issue of establishing optimal calcium intake levels for children and adolescents remains controversial.

In their classic, population-based study, Matkovic et al. (1979) provided evidence to support a critical role for calcium in the accumulation of skeletal mass early in life. These researchers examined cortical bone density in residents of two districts of rural Yugoslavia. The regions differed significantly in terms of mean calcium intake (500 mg vs. 1000 mg/day), but other variables were similar. Both regional populations were characterized by high levels of physical activity, as dictated by their agrarian lifestyles. As well, age, weight and other anthropometric indices were the same between populations. Results indicated that in the high calcium region, bone densities were significantly greater by age 20 than in the low

calcium district, but these differences did not diverge further as age progressed. In addition, the incidence of femoral neck fractures was significantly lower among people from the high versus low calcium district. The authors speculated that the differences between communities with respect to adult bone status and fracture risk were established by late adolescence. Thus, they suggested that the major effect of calcium was on the acquisition of peak bone density.

Sandler et al. (1985) supplied further evidence to support a role for calcium in bone accretion during the growth and development years. This group of researchers reported a significant positive relationship between cortical bone density in a large group of post-menopausal women and milk consumption during childhood and adolescence. However, the retrospective nature of this study made it difficult to control for several confounding variables which may have influenced bone density in these subjects (for e.g., lifestyle factors, physical activity patterns, menstrual history, etc.).

As described in the previous section on mechanical stimuli and physical activity, Halioua and Anderson (1989) found that both lifetime calcium intake and lifetime physical activity were significant positive predictors of bone mass in 181 healthy, pre-menopausal Caucasian women. This beneficial effect on BMC and BMD was seen when lifetime calcium intake was considered independently, as well as when it was combined with lifetime physical activity in the same regression model. The authors stated that their findings emphasize the importance of adequate calcium consumption and physical activity throughout adolescence and

early adulthood, in order to maximize genetic potential for bone development of the individual.

Results of a study by Sentipal et al. (1991) also confirm the hypothesis that dietary calcium has a major impact on achievement of PBM. This group of researchers assessed current calcium intake and vertebral BMD in 49 healthy Caucasian pre-adolescent and adolescent females (aged 8-18 years). The subjects were categorized according to Tanner's stages of pubertal development. Data collected from personal interviews with each subject and her mother, as well as from retrospective food frequency forms and 4-day diet records, were used to calculate past and present calcium intake. Multiple regression analysis of the data revealed that sexual maturity rating, age and calcium intake were significant contributors to vertebral BMD, and accounted for 81% of the variance in the dependent variable. As values for sexual maturity rating, age and calcium intake increased, so too, did vertebral BMD. Unfortunately, it is not clear whether subjects' past, present or lifetime calcium intakes were used in the regression analysis. Although the authors assessed current activity patterns in their subjects, they failed to consider past involvement in exercise. They pointed out that of the three variables which represented the most significant predictors of vertebral BMD in their subjects, chronological age and pubertal progression occur naturally, whereas calcium intake can be controlled. Thus, this research group advocated adequate dietary calcium consumption during pre-adolescence and adolescence, as a voluntary means of optimizing PBM.

Johnston et al. (1992) conducted a double-blind, 3 year longitudinal trial to ascertain whether calcium alone is effective in enhancing the rate of change in BMD in 45 pairs of monozygotic twins aged 6-14 years. Twenty-two pairs of twins were prepubertal throughout the entire investigation, 4 pairs were postpubertal at baseline and 19 pairs underwent puberty during the trial. Over the course of the study, one twin received a daily calcium supplement of 1000 mg (in addition to calcium intake from usual dietary sources) while the other twin served as a control and was given a placebo. Radial BMD was quantified with SPA and lumbar spine and hip BMD determinations were made with DPA. No significant differences in height, weight, nutrient intake (including calcium) or level of physical activity were detected between groups at baseline. During the study, the calcium supplemented group ingested an average of 1612 mg calcium/day (supplement plus dietary calcium intake), compared to 908 mg/day consumed by the placebo group. Three year results indicated that, in twin pairs who remained prepubertal throughout the study, the calcium supplemented twins demonstrated significantly greater increases in BMD of the radius and lumbar spine than twins who received the placebo. However, in the twin pairs that experienced puberty during the study or were postpubertal at baseline, no difference in the rate of increase in BMD was evident at any skeletal site for the calcium supplemented or placebo groups. Based on their findings, Johnston's group concluded that dietary supplementation with calcium alone is associated with an enhanced rate of bone mineral acquisition in prepubertal children. They postulated that if such a gain were to persist, then peak

bone mass would be expected to increase, thus reducing the risk of osteoporotic fractures later in life.

Ruiz et al. (1995) provided further confirmation that calcium is a strong independent determinant of BMD in children, especially prior to puberty. They employed DXA to measure lumbar spine and femoral BMD in 70 boys and 81 girls, aged 7-15 years. Subjects were classified according to pubertal status as follows: n=49 prepubertal (Tanner stage 1), n=65 pubertal (Tanner stages 2-4) and n=37 postpubertal (Tanner stage 5). A semiquantitative food frequency questionnaire was used to estimate each subject's daily calcium intake. Mean calcium consumption was found to be 810 mg/day, but ranged from 157 to 2033 mg/day. Results of multiple regression analysis revealed a significant positive relationship between calcium consumption and lumbar spine BMD in prepubertal children. This association was independent of age, body weight or sexual maturation. Furthermore, 93% of children with the lowest vertebral BMD and 84% of those with the lowest femoral BMD had calcium intakes of less than 1000 mg/day. Consequently, these researchers advised that recommendations for dietary calcium intake be at least 1000 mg/day for prepubertal and pubertal children.

In an attempt to define the amount of calcium needed for optimal skeletal growth and maturation, Matkovic (1991) analyzed 487 calcium balances from previously published studies according to subjects' developmental phase and calcium intake. Subjects were categorized into one of the following stages: infants, children (aged 2-8 years), adolescents (aged 9-17 years) and young adults (aged

18-30 years). Analysis of the data indicated that infancy and adolescence were the two stages with the highest requirement for calcium. As well, a significant positive correlation between calcium intake and body retention was found to exist for the entire group of subjects, although the effect varied depending upon the age group. In children, the estimated daily skeletal demand for calcium was approximately 100 mg/day. Given that the urinary calcium excretion rate in this age group was relatively low, Matkovic concluded that most children could probably meet their daily calcium requirements on an intake corresponding to the RDA (i.e., 800 mg/day). However, calcium balance differed for adolescents (particularly girls). Calcium requirements during this period are high, in order to support an increase in bone modelling and skeletal consolidation, yet Matkovic found the ratio between calcium intake and maximal skeletal calcium retention was lowest for adolescent females. Thus, he expressed concern that many adolescent girls may be jeopardizing skeletal health by consuming marginal levels of calcium.

Sentipal et al. (1991) reported similar findings with respect to calcium intake in 49 prepubertal and pubertal female subjects. Their results suggested that girls chronically underconsume calcium, and the phenomenon becomes more pronounced with increasing age. For example, 67% of subjects aged 8-10 years had current calcium intakes which met the RDA (800 mg/day) whereas only 16% of subjects aged 11-18 years had intakes conforming to the RDA (1200 mg/day). Furthermore, when retrospective food frequencies were analyzed, results indicated that the percentage of subjects who met recommended daily servings from the dairy food

group declined from 73% between the ages of 2-5 years to 52% between kindergarten and grade 6. None of the subjects met the recommended serving numbers during adolescence. Thus, calcium intake appears to drop as young girls approach and reach puberty. Unfortunately, this decrease in calcium consumption occurs at a crucial time in skeletal development, when the need for dietary calcium is greatest.

To summarize, there is considerable evidence to support the concept that dietary calcium exerts a positive influence on the accretion of bone mass in growing girls.

2.3.6 Genetic Factors

Genetic factors are thought to be the most important determinants of PBM, accounting for 60-80% of the variance (Chestnut III, 1991; Ott, 1990; Slemenda et al., 1994). It has been proposed that genetic factors dictate the degree to which bone is influenced by physical activity, dietary calcium intake, alcohol consumption and cigarette smoking (Eisman et al., 1991; Kelly et al., 1993).

Racial differences in bone mass have been shown to exist between black, white and Asian people. In both weight-bearing and non-weight-bearing regions of the skeleton, blacks exhibit a higher bone density than whites (McCormick et al., 1991; Ortiz et al., 1992). This difference persists even after adjusting for level of physical activity, calcium intake and BMI (Ortiz et al., 1992). Conversely, cortical bone mass is significantly greater in whites compared to people of Asian descent

(Pollitzer & Anderson, 1989).

Dequeker et al. (1987) examined 16 pairs of monozygotic (MZ) and 14 pairs of dizygotic (DZ) twins to assess the magnitude of genetic effects on bone mass of the distal radius and spine. They found that the genetic influence on BMC in the spine (which is predominantly trabecular bone) was substantial in the younger individuals (those less than 24 years of age), although the same trend was not evident in cortical bone. They therefore postulated that genetic factors exert differing effects at axial and peripheral sites, depending upon the age of the individual.

Pocock et al. (1987) also investigated heritability of bone mass in twin pairs. They evaluated BMD of the forearm, lumbar spine and proximal femur in 19 pre-menopausal MZ twin pairs and 22 pre-menopausal DZ twin pairs, and observed a significant genetic contribution to bone mass at all sites measured.

Comparisons between parents and their children have also shown strong correlations for BMC and bone density at different skeletal sites. For example, Lutz (1986) studied 26 Caucasian mother-daughter pairs (mean age 55 and 26 years, respectively) to estimate the heritability of BMC and BMC to bone width ratio (BMC:W) of the mid radius. The purpose of the BMC:W measurement was to normalize for bone size. Analysis of the data indicated a significant positive association between BMC and BMC:W of mothers and daughters, which led the author to conclude that heritability of bone mass at the radial site is high. However, she was careful to point out that this estimate of heritability does not distinguish

between genetic components and degree of familial resemblance (i.e., the extent to which a mother's lifestyle is emulated by her daughter). Furthermore, since the study only assessed cortical BMC, one cannot generalize findings to bone mass at trabecular sites (such as the hip and spine).

In a subsequent investigation, Lutz and Tesar (1990) examined the degree to which familial resemblances could be demonstrated for BMD of the lumbar spine and proximal femur in 37 healthy, white mother-daughter pairs. Daughters ranged in age from 20-35 years, while the mothers' ages were between 41 and 68 years. Twenty of the mothers were pre-menopausal while 17 were post-menopausal. A significant positive correlation was found to exist between mothers and daughters for BMD at both sites measured, and this relationship was stronger in the pre-menopausal versus post-menopausal mother-daughter pairs. The authors stated that their data support the existence of familial resemblances for BMD of trabecular bone sites (i.e., areas of the skeleton most prone to osteoporotic fracture), although they acknowledged that lifestyle similarities between mothers and daughters may have increased the correlation of BMD in mother-daughter pairs.

Matkovic et al. (1990) also evaluated possible hereditary influences on skeletal status by comparing bone mass in a group of 14 year old post-menarchial girls and their parents. Bone size and bone mass at the distal radius and axial skeleton were measured in 24 mother-father-daughter trios. Analysis of the data indicated a significant correlation between mean values for parents (i.e., mother + father/2) and daughters. Thus, these researchers deduced that bone size, mass

and density in daughters can be attributed, to a reasonable degree, to the parents. They pointed out, however, that their inability to control completely for potentially confounding environmental, nutritional and physical activity factors may have enhanced family resemblances.

The research studies described above illustrate that genetic factors play a significant role in the attainment of PBM. It may well be that heredity dictates maximum achievable PBM in a woman, while mechanical loading (i.e., exercise), nutrition, hormonal status and environmental factors determine the degree to which she reaches her genetic potential. Unfortunately, it is difficult to design a well-controlled study which would enable this hypothesis to be tested in girls and young women.

2.4 The Effect of Developmental Factors on Bone Mineral Density in Children and Adolescents

There are several reports in the literature suggesting that BMD in children is positively linked to a number of developmental factors, including chronological age, height, weight, body surface area, bone-free lean tissue, bone age, body mass index (BMI) and pubertal stage (DeSchepper et al., 1991; Faulkner et al., 1993; Geusens et al., 1991; Gilsanz et al., 1988; Gordon et al., 1991; Grimston et al., 1992; Kroger et al., 1992; Ponder et al., 1990; Rubin et al., 1989; Sentipal et al., 1991). Although no one investigation has demonstrated an effect of all these variables on BMD during growth, the majority of research studies implicate weight, height, age and/or puberty as developmental factors which are most strongly

associated with accretion of bone mass in children.

In a cross-sectional investigation, Ponder et al. (1990) collected data on lumbar spine BMD in 184 healthy black, white and Hispanic children (101 males and 83 females) aged 5.00-11.99 years, in order to establish reference values for future research. DPA was employed for the purpose of bone densitometry. Several other parameters were also assessed in each subject, including height, weight, triceps skinfold thickness and percent body fat. Regression analysis of the data revealed that body weight, height and age were highly correlated with L₂-L₄ BMD in this sample of children, with body weight exhibiting the strongest association. The results of this study led Ponder's group to conclude that the spinal mineralization process in children is more responsive to load-bearing (represented by body weight) than to a child's chronological age. They pointed out that this may confound interpretation of spinal BMD values in children who are underweight for their age.

Kroger et al. (1992) also reported a close relationship between BMD of the lumbar spine and femoral neck and age, height and weight in a group of 84 healthy children (40 males and 44 females) aged 6-19 years. BMD values of the two skeletal sites were obtained using DXA instrumentation. No significant differences were detected between boys and girls under the age of 12 years in terms of lumbar spine BMD. A steeper increase in BMD of the spine was observed after 10 and 12 years of age in girls and boys, respectively, presumably due to the effect of puberty on bone mass. Unfortunately, as subjects were not categorized on the basis of

sexual development in this study, an assessment of the effect of puberty on BMD was precluded.

In a study by Geusens et al. (1991), cross-sectional data were gathered on bone mass in 202 healthy, white male and female subjects, aged 3-25 years, in order to determine bone growth patterns in this age group. SPA was utilized to quantify bone mass at the proximal and distal radius, while DPA was employed to measure BMC and BMD of the lumbar spine and total body. Regression analysis of the data revealed strong positive correlations between total body BMC and BMD and age, height and weight, although weight was found to be a significantly better predictor of both bone mineral parameters than age. These researchers also observed heterogeneity of bone growth in terms of magnitude, skeletal site measured, expression of the measurement (i.e., BMC or BMD) and gender. For example, a more pronounced increase in BMD was evident at weight-bearing sites, such as the legs. Prepubertally, no bone mass differences were evident between the sexes, which led the authors to conclude that growth factors which influence bone prior to puberty are probably not dependent upon sex hormones. However, a problem with this study relates to the fact that subjects were grouped according to chronological age rather than stage of pubertal development. Consequently, one cannot assume that the age categories subjects were assigned to accurately reflected their pubertal status.

Rubin et al. (1989) examined the impact of several different parameters, including age, height, weight, pubertal stage and grip strength, on axial and

appendicular bone density in 183 healthy Caucasian children (aged 6-18 years) of both sexes. Bone density of the mid radius and lumbar spine was determined by SPA and DPA, respectively. Multiple regression analysis of the data revealed that 83% of the variation observed in bone density in the subjects could be explained by the developmental factors under investigation (i.e., age, height, weight, pubertal stage and grip strength).

Gordon et al. (1991) conducted a cross-sectional study of lumbar spine BMC and BMD in 236 subjects aged 3-30 years, to determine the rate of increase of bone mass during childhood, adolescence and early adulthood. BMC and BMD of the L₂-L₄ region of the spine was measured with DPA. Results showed that while BMD in male subjects increased at a fairly steady rate from 3 to 30 years of age, the only increase in BMD for females older than 10 years of age occurred during puberty. Puberty was found to contribute 39% of BMD and 55% of BMC to PBM in the female subjects examined. Gordon commented that this finding is not surprising given the association between bone growth and body weight during the developmental years, and the fact that a significant portion of adult body weight is acquired by girls during puberty. Thus, the marked increases in BMC and BMD seen in girls during puberty may be partly mediated by substantial increases in body weight which occur at this time.

DeSchepper et al. (1991) also examined the effects of pubertal development, in addition to age, height and weight, on BMC and BMD of the lumbar spine in 136 children aged 1-18 years (58 males and 78 females). Subjects were categorized

according to chronological age, as well as on the basis of Tanner's pubertal staging (i.e., stages 1-5). DPA was utilized for determination of BMD of the L₂-L₄ vertebral region. Results of multiple regression analysis indicated that BMD of subjects was significantly correlated to age, body weight and height. However, the association between these variables was markedly lower in the pubertal versus prepubertal children. DeSchepper's group interpreted this finding to mean that factors other than growth influence bone mineralization in pubertal children to a greater extent than in prepubertal children. Although BMD values rose with each successive stage of puberty, the only significant increase was demonstrated in stage 4 subjects, for both boys and girls. The authors attributed this observation to a slowing of the growth spurt and attainment of adult levels of sex hormones during the fourth pubertal stage.

In contrast to results of the studies described above, Grimston et al. (1992) reported that neither chronological age nor height were significant predictors of spinal BMD in a sample of healthy children and adolescents. These researchers used a cross-sectional design to examine the effect of puberty (and its associated anthropometric changes) on accretion of bone mass in physically active children. In an attempt to control for the potentially confounding effects of differing levels of exercise on bone mineral status, the sample was limited to male and female competitive swimmers (n=74) aged 9-16 years. Tanner staging was employed to assess pubertal development of each subject while DPA was utilized to quantify BMD of the lumbar vertebrae and femoral neck. Stepwise linear regression

analysis revealed that 77% of the variability in spinal BMD could be accounted for by pubertal stage, while 68% was attributable to body weight. Thus, the authors concluded that incremental increases in spinal BMD seen in children aged 9-16 years occur primarily as a function of pubertal development and concomitant gains in body weight. They also emphasized the importance of factoring in the effect of puberty when studying BMD in healthy children, to control for wide biological variability in chronological age at each pubertal stage.

Gilsanz et al. (1988) and Glastre et al. (1990) also provide confirmation that adolescence represents a particularly critical period for skeletal growth and maturation. Gilsanz and colleagues examined 101 prepubertal and pubertal children, aged 2-18 years, to determine the effect of puberty on bone density during skeletal development. QCT was used to measure trabecular bone density of the lumbar spine and provide an index of cortical bone in the vertebral body. Results of regression analysis showed a significant increase in bone density during puberty, both in terms of trabecular vertebral density and cortical index density. However, height, weight, surface area and BMI were not found to be significant predictors of trabecular bone density in either the prepubertal or pubertal children. The authors surmised that puberty has a positive impact on bone development by stimulating the accumulation of skeletal mass through linear growth and increasing bone density.

In a similar study, Glastre et al. (1990) used DXA to investigate BMD of the lumbar spine in 135 Caucasian children (70 males and 65 females) aged 1-15 years. All subjects demonstrated normal growth velocities. Children were

categorized into one of four stages (prepuberty, puberty, advanced puberty and achieved puberty) according to Tanner's sexual maturation ratings. Results of regression analysis indicated a high correlation between BMD and height, weight, body surface and bone age in the subjects. A significant increase in spinal BMD was evident with age, and this rise was steepest at the time of puberty.

Contrary to findings reported above, Faulkner et al. (1993) identified bone-free lean tissue as the most important predictor of total body BMD in young girls. In this cross-sectional investigation, they utilized DXA to measure total body BMC, BMD and soft tissue (i.e., fat tissue and bone-free lean tissue) in 124 female subjects aged 8-16 years. The relative contribution of age, height, weight, bone-free lean tissue and fat tissue on total body BMD was determined by stepwise multiple regression analysis. Results showed a significant positive correlation between bone-free lean tissue and total body BMD ($r^2=0.80$). Age explained an additional 2% of the variance and height accounted for an additional 1%, but neither body weight nor fat tissue were found to be significant predictors of total BMD. To put their findings into perspective, Faulkner's group pointed out that a substantial proportion of bone-free lean tissue is muscle, and since the skeletal and muscular systems are dynamic and closely aligned, it is likely that muscle tissue would be a stronger predictor of total body BMD than body mass alone.

To summarize the preceding studies, increases in BMD in children have been related to several different developmental parameters. However, in reviewing the literature, two factors seem to emerge as having the strongest association with

bone mass accretion in growing children: puberty and body weight. Considering that pubertal development is accompanied by weight gain, it appears likely that increases in skeletal mass at this phase of the life cycle are mediated, to a large extent, by hormonal events and associated increases in body weight.

2.5 Variability of Bone Mineral Density in Children

Although several researchers have attempted to establish normative values for BMD in children, the complicating factor in interpreting the data is that different studies have utilized different methodologies for determination of bone mass. Thus, a direct comparison of findings reported in the literature is often precluded. A further difficulty arises when studies employ a heterogeneous sample of subjects in terms of age, sex, pubertal status and racial background. Consequently, it may not be possible to identify mean BMD values and variability for a selected group of children.

For DPA measurements of the lumbar spine, variability has been reported to range between 0.02 and 0.05 g/cm² in competitive female swimmers whose pubertal development corresponded to Tanner stages 1 to 3 (Grimston et al., 1992). Variability of femoral neck BMD was 0.02-0.03 g/cm² in the same three groups of subjects (i.e., Tanner stages 1-3). However, the number of subjects per group was small (5, 3 and 8 for Tanner stages 1, 2 and 3, respectively) which may have undermined the power of the statistical tests used.

In 25 healthy white girls between the ages of 10 and 15 years, variability of

lumbar spine BMD (measured by DPA) was reported as 0.04 g/cm^2 (Geusens et al., 1991).

DeSchepper et al. (1991) used DPA to determine BMD of the lumbar spine in 16 males and females categorized as Tanner stage 2 and 13 males and females classified as Tanner stage 3. Variability was reported as 0.08 g/cm^2 for the stage 2 subjects and 0.06 g/cm^2 for the stage 3 subjects. Although BMD results were pooled for boys and girls in this study, McCormick et al. (1989) have stated that males and females less than 12 years of age can be grouped together in terms of bone density. Geusens et al. (1991) were also unable to detect any BMD differences between prepubertal males and females at the proximal radius, lumbar spine, arms, legs and total body. In a study by Glastre et al. (1990), BMD of the spine did not differ significantly between boys and girls aged 1-15 years, with the exception of age 12 when girls exhibited a higher BMD than boys.

In a group of 44 prepubertal (Tanner stage 1) boys and girls measured by DPA, BMD variability of the lumbar spine, femoral neck, trochanter and Ward's Triangle was 0.06 , 0.08 , 0.07 and 0.09 g/cm^2 , respectively (Slemenda et al., 1994). Higher BMD variabilities were observed in 38 peripubertal (Tanner stages 2-4) boys and girls: 0.12 g/cm^2 at the lumbar spine, 0.12 g/cm^2 at the femoral neck, 0.11 g/cm^2 at the trochanter and 0.14 g/cm^2 at Ward's Triangle (Slemenda et al., 1994). The wider range of pubertal stages represented by the peripubertal children may have been somewhat responsible for the higher variabilities seen in this group.

Kroger et al. (1992) reported BMD variability at the lumbar spine as 0.069 ,

0.031 and 0.112 g/cm² in girls aged 8-9, 10-11 and 12-13 years, respectively. Variability of femoral neck BMD ranged between 0.063 and 0.138 g/cm² for the same three groups of subjects. BMD in this study was assessed with the DXA instrumentation (Lunar DPX system). Unfortunately, the number of subjects in each of the age categories was not provided. It is likely that the sample size for these age groups was small, given that 44 girls participated in the study and there were 7 age categories in total (ranging from 6-7 years to 18-19 years).

According to unpublished data from the University of Saskatchewan, variability of lumbar spine BMD was between 0.067 and 0.097 g/cm² in 67 girls aged 8-12 years (D. Drinkwater, personal communication, Dec. 21, 1992). The DXA Hologic QDR system was used to measure BMD in these subjects. However, reports in the literature indicate that values obtained with the QDR instrument are not directly comparable to DPA measurements (Mazess & Barden, 1988; Wahner et al., 1988).

Given the wide range of variability published for BMD in children, and the fact that these values have been obtained using different measurement devices, it is obvious that further research needs to be directed at establishing norms for BMD during the childhood years. To ensure that direct comparisons of data are possible, it is important that standardized instrumentation be used and specific groups of children be evaluated.

3. METHODS AND PROCEDURES

3.0 Sample Size Estimation

Sample size was estimated according to the formula for studies involving two sample means ($n = 2[PI \times \delta / \mu_1 - \mu_2]^2$), where PI is the power index, δ is the assumed variability and $\mu_1 - \mu_2$ is the difference between means (Hassard, 1991). A power index of 2.80 was chosen, which represents an α value of 0.05 and a power level of $1 - \beta = 0.80$ (Hassard, 1991).

Published data for young girls were consulted to determine variability in BMD of the lumbar spine. Although other sites have been measured in children, information on lumbar spine BMD and variability is more widely available in the literature. A figure of 0.04 g/cm^2 was selected, based on lumbar spine BMD variability reported for 25 white girls, aged 10-15 years (Geusens et al., 1991). To account for overlaps in standard deviation between two groups, 0.04 g/cm^2 was multiplied by 2, which generated a variability value (δ) of 0.08 g/cm^2 .

It was difficult to establish a value for $\mu_1 - \mu_2$ *a priori* given that published data on BMD's in athletic and non-athletic children are scarce. Although information is available on BMD's in healthy children, activity levels of the subjects have not usually been identified (DeSchepper et al., 1991; Geusens et al., 1990; Gilsanz et al., 1988; Glastre et al., 1990; Ponder et al., 1990; Slemenda et al., 1994). Thus, it is likely that these studies have documented mean BMD's for children whose activity levels ranged from sedentary to highly active.

However, Kroger et al. (1992) have offered some insight into the issue of BMD in athletic and non-athletic children. These researchers measured BMD of the lumbar spine and femoral neck by DXA (Lunar DPX) in 84 healthy males and females, aged 6-19 years. When they stratified their subjects according to physical activity level, they discovered that those who engaged in more than 5 hours of structured sports/week demonstrated a mean spinal BMD which was 0.053 g/cm^2 greater (not significant) than those who participated in little or no physical activity outside of school. Similarly, mean femoral neck BMD was 0.070 g/cm^2 higher ($p < 0.05$) in the athletic versus non-athletic subjects.

Given the findings of Kroger et al. (1992) and the fact that the gymnasts participating in our investigation would be training a minimum of 9 hours/week (a physical activity level almost twice that of the highly active subjects in the study by Kroger et al. [1992]), a value of 0.08 g/cm^2 was assigned to $\mu_1 - \mu_2$. In other words, the maximum mean difference to be detected between BMD's of pre-menarchial competitive gymnasts and normally active girls was 0.08 g/cm^2 .

Therefore, based on the above estimates, sample size was calculated to be 32 (16 in each of the gymnast and normally active control groups).

3.1 Subject Recruitment and Selection

In advance of the study, the Manitoba Rhythmic Sportive Gymnastics Association (MRSGA) and the Manitoba Gymnastics Association (MGA) were contacted and received an information package explaining the intent of the study

and the scope of the research. Permission to approach local Winnipeg rhythmic and artistic gymnastics clubs for the purpose of recruiting subjects was requested from the MRSGA and MGA. Following approval, subjects were recruited by letter and personal contact to gymnastics coaches and parents of girls (aged 9-13 years) training as national or provincial stream athletes (at the novice or junior level with a local rhythmic or artistic gymnastics club). Control subjects were approached in the same manner by making contact with parents of girls (aged 9-13 years) who participated in the 1992 Mini U Camps (University of Manitoba).

The purpose of the study, degree of subject/parental participation, eligibility criteria, testing locales and procedures, inconveniences to subjects/parents, and potential risks to subjects were explained upon initial contact with prospective subjects/parents. Eligibility criteria for participation in the study were as follows: pre-menarchial, Caucasian girl, aged 9-13 years; Stage 1, 2 or 3 breast development according to Tanner's (1962) breast development scale; for gymnasts, affiliation with the novice or junior level of a local Winnipeg rhythmic or artistic gymnastics club and training a minimum of 9 hours/week; for control subjects, participation in a maximum of 5 hours of structured physical activity per week (i.e., school physical education classes, school and club teams, and lessons); no chronic use of medications which may have influenced bone metabolism (e.g., glucocorticoids for asthma); absence of a medical condition(s) which may have affected bone metabolism (e.g., diabetes, renal disease, arthritis, celiac disease, etc.); no history of restricted food intake or presence of eating disorder symptoms;

non-smoker; no coffee consumption; and no alcohol intake.

Subjects/parents were informed of their right to deny consent or to withdraw from the study at any time, without prejudice. Subjects/parents signed a voluntary consent form (Appendix A) and completed brief screening questionnaires (medical history, physical activity, calcium intake and Eating Disorder Inventory [Garner & Olmstead, 1984]) to ensure that subjects met inclusion criteria and rule out the presence of any potentially confounding variable[s] (Appendix B and C). Given the young age of the girls recruited for this study, the majority of subjects received assistance from their parent(s) in completing the screening questionnaires. Individual results along with general findings of the investigation were promised to all participants upon completion of the study. Payment was not offered for taking part in the study, but parking costs were reimbursed for bone densitometry appointments. Upon receipt of signed consent forms and preliminary screening questionnaires, subjects were enrolled in the study and contacted regarding further participation.

Twelve gymnasts ($n = 8$ rhythmic gymnasts and 4 artistic gymnasts) and 15 normally active girls (controls) met eligibility criteria and were initially registered for the study. However, one gymnast was subsequently diagnosed with Type I diabetes mellitus and withdrew from the investigation. Attempts were made to recruit additional subjects (particularly gymnasts) through recommendation by those currently enrolled in the study, but efforts were unsuccessful. The final sample therefore consisted of 26 participants (11 gymnasts and 15 controls) and fell short

of the estimated number of subjects required to attain a power level of 0.80 (i.e., 32 subjects). The power level with a sample of 26 was determined to be 0.70 (Neter & Wasserman, 1990).

3.2 Experimental Design

The study design was cross-sectional and descriptive in nature. The key focus was twofold: (1) to determine if pre-menarchial competitive gymnasts and normally active girls of the same age and pubertal status differed with respect to bone mineral density, and (2) to define the relationship between the dependent variable (bone mineral density) and each of the independent variables (age, height, weight, bone-free lean tissue, physical activity and calcium intake) in pre-menarchial gymnasts and normally active girls aged 9-13 years.

3.3 Data Collection

3.3.0 Questionnaires

At the outset of the study, screening questionnaires were used to collect information regarding subjects' medical histories, past and present physical activity patterns and training regimens, dietary habits, calcium intake levels and symptoms related to eating disorders. With the exception of the calcium intake questionnaire (adapted from the Calcium Calculator, B.C. Dairy Foundation, 1987) and Eating Disorder Inventory (EDI) (Garner & Olmstead, 1984), questionnaires were pilot tested in a group of 4 adults. In accordance with comments received from those in the pilot test group, minor alterations were made to the text and format of the

physical activity questionnaire to improve clarity and eliminate ambiguities in the final tool.

The medical history survey (Appendix B) consisted of questions regarding general medical history, use of medications, fracture information and menstrual status (i.e., pre-menarchial vs. post-menarchial). Subjects were also asked to complete a self-assessment of breast development (stages 1-5) using Tanner's (1962) standard photographs. Self-rating according to this method is considered to provide an accurate assessment of developmental stage in children aged 9-18 years (Duke et al., 1980).

The physical activity questionnaire (Appendix B) was used to obtain information on past and present exercise patterns, including the frequency, length and type of activities subjects participated in. As well, gymnasts responded to questions regarding their current training regimen. Information collected from this questionnaire was used to tabulate the hours/week of structured physical activity for each subject.

The food intake survey (Appendix B) elicited general information on adherence to special diets (if any), presence of food allergies/intolerances, caffeine consumption and use of vitamin/mineral supplements, while the calcium intake questionnaire (Appendix B) provided a 24 hour estimate of calcium consumption.

The EDI (Garner & Olmstead, 1984) (Appendix C) is a 64-item self-report questionnaire and was used to measure 8 constructs that have theoretical relevance to eating disorders (i.e., Drive for Thinness, Bulimia, Body

Dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal Distrust, Interoceptive Awareness and Maturity Fears). The EDI has been validated as an objective screening instrument for detection of attitudinal and behavioural symptoms related to eating disorders, although it is not considered to be a diagnostic tool in and of itself (Garner et al., 1983; O'Connor et al., 1995; Phelps & Bajorek, 1991).

3.3.1 Bone Densitometry and Body Composition Measurements

Bone mineral content (BMC) and bone mineral density (BMD) of the lumbar spine (L_2 - L_4), proximal femur (i.e., femoral neck, trochanter and Ward's Triangle) and total body, as well as percent body fat and total and regional lean and fat tissue mass were measured by dual energy x-ray absorptiometry (Lunar DPX, Lunar Radiation Corp., Madison, WI) with software version 3.4, in the Department of Nuclear Medicine, St. Boniface General Hospital, by a research technologist. The rationale for measuring total body bone mineral was that it would provide an assessment of bone mineral status throughout the entire skeleton as opposed to only specific skeletal sites. Since bone growth is not homogeneous throughout the skeleton and bone mass may differ significantly depending upon the site measured, bone mineral status at one location cannot be extrapolated to other sites with any degree of accuracy (Chestnut III, 1987; Fogelman & Ryan, 1992; Geusens et al., 1991). Furthermore, it was expected that total body measurements would be more likely to show regional differences in bone mass between subjects, compared to measurements at just one or two sites.

Dual energy x-ray absorptiometry (DXA) is a non-invasive technique which utilizes a highly stable x-ray tube as its energy source (Genant et al., 1991). Very low radiation doses, fast scanning times and high precision and accuracy make DXA a method which is well-suited to studying BMD in children (Glastre et al., 1990; Sartoris & Resnick, 1988). Radiation exposure with DXA is 1-3 mrem, which is approximately one-tenth of a standard chest x-ray and negligible for practical purposes (Glastre et al., 1990; Haarbo et al., 1991; Mazess & Barden, 1989). DXA presents a low risk of harm to subjects and is considered to be a safe modality for assessing bone mineral status (Kellie, 1992). Precision of BMD measurements with DXA is 1%, while accuracy has been reported at 4-8% (Genant et al., 1991, Mazess & Barden, 1988; Wahner, 1989). DXA also provides a three-compartment model of body composition (i.e., fat, lean tissue mass and BMC) and has been validated for measurement of such (Haarbo et al., 1991, Ogle et al., 1995). Total body and regional lean tissue mass can be determined with a small precision error (1.5-2%) (Mazess et al., 1990) and the accuracy error of estimating actual percent body fat from DXA measurements is 4.9% (Haarbo et al., 1991).

In order to assess bone mineral and body composition with DXA, each subject was positioned supine on the scanning table. A series of rectilinear scans was then made at 1 cm intervals from the subject's head to her toes for determination of total and regional body BMC, BMD and body composition. The proximal femur and L₂-L₄ region of the lumbar spine were also scanned for assessment of bone mineral status at these sites. Subjects were all scanned in the

medium scan mode (8 cm/sec.) with a sample size of 4.8 x 9.6 mm for the total body and 1.2 x 1.2 mm for the proximal femur and lumbar spine, and source collimation of 1.68 mm. There was no discomfort to the subjects, and they remained fully clothed throughout the procedure. Combined scan time for the total body, proximal femur and lumbar spine was approximately 45 minutes. Bone-free lean tissue mass (BFLT) was calculated for each subject by subtracting total body BMC from total body lean tissue mass.

3.3.2 Anthropometric Measurements

Anthropometric measurements consisted of height, weight, skinfold thicknesses, body girths, and bone lengths and breadths. Each subject's height and weight was determined in the Department of Nuclear Medicine (St. Boniface General Hospital), at the time of bone densitometry. A stadiometer and balance scale were used to measure height and weight, respectively. The remaining anthropometric measurements were made within two weeks of the bone densitometry appointment, and were taken at one of the following locations (depending upon which was most convenient for the subject and her parent[s]): the Department of Nuclear Medicine (St. Boniface General Hospital); the Health, Leisure and Human Performance Research Institute (Max Bell Centre, University of Manitoba); or the subject's home. Skinfold thicknesses, body girths, and bone lengths and breadths were measured in accordance with standardized techniques described by Lohman et al. (1988). Skinfolds were obtained using Lange calipers

on the right side triceps, biceps, subscapular, suprailiac, abdomen, anterior mid-thigh and medial calf sites. Circumferences were measured with a steel measuring tape on the right side upper arm, forearm, wrist, chest, waist, gluteal, proximal thigh, calf and ankle sites. Bone lengths of the right upper arm, forearm, hand, thigh and tibia were obtained with a steel measuring tape. Bone breadths of the right humerus, knee, ankle and both wrists were determined with sliding calipers. Each measurement was made in triplicate by the same observer. If the third measurement differed substantially from the first two, it was subsequently disregarded. Means of either two or three measurements were used for all analyses. Body circumferences and bone lengths and breadths were obtained for the entire sample of 26 subjects. However, skinfold measurements could not be done on 5 control subjects as these girls complained of discomfort from the calipers. Therefore, skinfold values were collected for a total of 21 subjects.

The sum of 7 skinfolds was used to assess body fatness. Skinfold measurements were also utilized to estimate percent body fat according to equations developed by Jackson et al. (1980) and Slaughter et al. (1988) (Appendix D). The percent body fat values derived from skinfold equations were then compared to those obtained with DXA.

3.3.3 Dietary Intake

Subjects and their parents received written and verbal instruction from a registered dietitian on how to complete a 3-day diet record (Appendix E). Three-

or 4-day food records are considered an acceptable compromise between reflecting a subject's typical intake and waning subject enthusiasm and compliance as the period of record-keeping increases beyond this time frame (Barr, 1987). Each subject was asked to select 3 consecutive days (i.e., 2 weekdays and 1 weekend day) that were representative of her usual eating habits for which to record dietary intake. During this 3 day period, she was instructed to document all foods, beverages and vitamin and/or mineral supplements consumed, including an estimate of portion sizes (using common household measures), a list of brand names used, and a description of food preparation methods. Diet records were reviewed with the subject and her parent(s) at the time of completion, to verify accuracy. Given the relatively young age of subjects, the majority of these girls received parental assistance in documenting their food intake.

Dietary records (including any vitamin and/or mineral supplements) were analyzed by computer for energy, protein, fat, carbohydrate, calcium, phosphorus and vitamin D using Demeter version 1.00 software (Northern Technical Data Inc.) and the 1991 Canadian Nutrient File. The Canadian Nutrient File is a database containing approximately 3500 food items, each with 100 different nutrients and food components. An average intake value for energy and the nutrients listed above was calculated for each subject from her 3-day food record. Mean values were then compared to Canadian Recommended Nutrient Intakes (Health and Welfare Canada, 1990). Further analysis was performed to determine percentages of total energy derived from protein, fat and carbohydrate. Also, protein

consumption on a gram/kg body weight basis as well as the calcium to phosphorus ratio were calculated for each subject.

3.4 Ethics

This study received ethics approval from the Faculty of Physical Education and Recreation Studies Committee on Research Involving Human Subjects (University of Manitoba). Written consent for participation in this study was obtained from all subjects and their parent(s).

3.5 Statistical Analyses

Statistical analyses were performed using StatView SE + Graphics software version 1.04 (Abacus Concepts, Inc., Berkeley, CA). Probability values were considered significant if they were below 0.05. The Statistical Advisory Service (University of Manitoba) was consulted to provide advice regarding data analysis.

Means and standard errors were calculated for bone mineral and body composition data, anthropometric measurements, and nutrient intake levels for the gymnasts and normally active control subjects. Unpaired 1- and 2-tailed Student's *t*-tests were used to test for significant differences between mean values for the gymnasts and controls.

Simple linear regression analysis was employed to clarify the relationship between each independent variable (i.e., age, height, weight, bone-free lean tissue, hours of structured physical activity and calcium intake) and BMD in the gymnast and control groups separately. In addition, simple linear regression analysis was

used to determine the association between two independent variables (such as age and weight, and height and weight) in each group of subjects.

4. RESULTS

4.0 Description of Subjects

Subjects were 11 pre-menarchial competitive gymnasts aged 9 to 13 years and 15 pre-menarchial normally active girls (controls) aged 9 to 12 years. In the gymnast group, 7 subjects trained and competed as rhythmic gymnasts and 4 were artistic gymnasts. Gymnasts had been training for an average of 5.5 years (range 3 to 8 years) prior to the study. All subjects were at stage 1, 2 or 3 of the Tanner (1962) breast development scale, with a mean of stage 2 for each group. General characteristics of the gymnast and control groups are exhibited in Table 4.1.

Table 4.1 General characteristics of gymnasts and controls (mean \pm SE)

Variable	Gymnasts (n=11)	Controls (n=15)	p Value
Age (y)	11.8 \pm 0.4	11.1 \pm 0.2	ns ^a
Height (cm)	147.6 \pm 2.8	147.7 \pm 1.6	ns ^a
Weight (kg)	37.0 \pm 2.2	42.4 \pm 1.8	p<0.05 ^b
Gymnastics training (h/wk)	10.0 \pm 0.7	-	
Total structured physical activity (h/wk)*	11.3 \pm 0.6	3.0 \pm 0.3	p<0.01 ^b

ns = not significant

^a2-tailed *t*-test

^b1-tailed *t*-test

*Refers to organized physical activities and includes participation in school physical education classes, school and club teams, lessons, practice sessions and gymnastics training

4.1 Body Composition Measurements

Body composition values (as determined by DXA) of the gymnast and control groups are shown in Table 4.2. Total body tissue weight and fat tissue weight were significantly lower in gymnasts than controls, while lean tissue weight and bone-free lean tissue weight did not differ between the two groups. In terms of percent body fat, gymnasts were significantly leaner than controls.

Table 4.2 Body composition data of gymnasts and controls (mean \pm SE)

Variable	Gymnasts (n=11)	Controls (n=15)	p Value ^a
Total body tissue weight (g)	34648 \pm 2178	40444 \pm 1804	p<0.05
Fat tissue weight (g)	2941 \pm 680	7770 \pm 1137	p<0.01
Lean tissue weight (g)	31708 \pm 1745	32674 \pm 1132	ns
Bone-free lean tissue weight (g)*	30073 \pm 1639	31099 \pm 1075	ns
Body fat (%)	7.9 \pm 1.4	18.4 \pm 2.0	p<0.01

ns = not significant

^a1-tailed *t*-test

*Bone-free lean tissue weight = Lean tissue weight - Total body bone mineral content

Figure 4.1 depicts the relative proportion of fat, bone free lean and bone tissue of gymnasts and controls, with each tissue compartment expressed as a percentage of total body tissue weight.

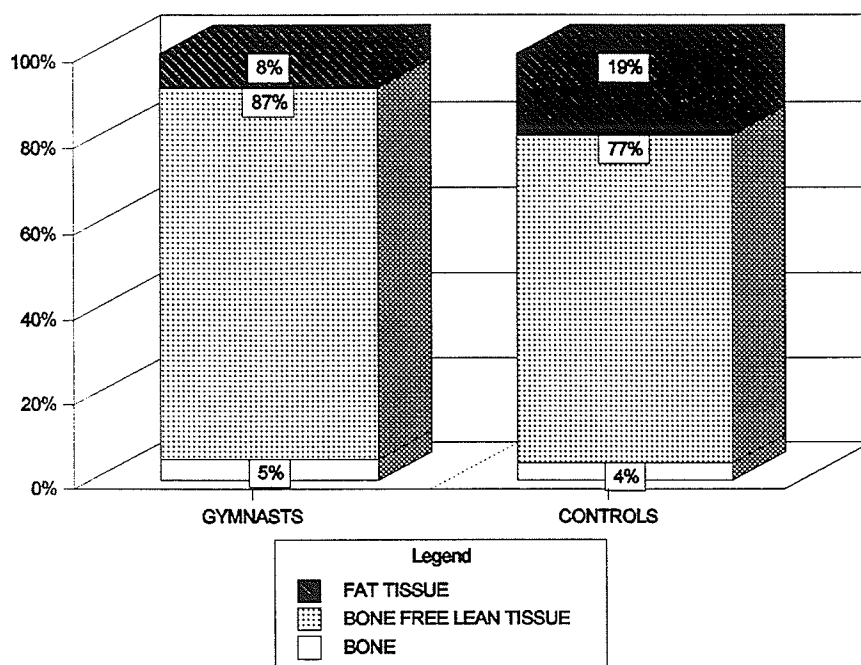


Figure 4.1 Body compartments (fat tissue weight, bone-free lean tissue weight and bone weight) of gymnasts and controls, expressed as percentage of total body tissue weight

4.2 Anthropometric Measurements

4.2.0 Skinfold Thickness Measurements

Skinfold thicknesses for 11 gymnasts and 10 control subjects are exhibited in Table 4.3. Five girls in the control group indicated that the skinfold calipers pinched or created discomfort when measurements were attempted at certain sites and consequently, some skinfold thicknesses could not be obtained for these particular subjects. As a result, skinfold data for these 5 subjects were not included in the aggregate score for the control group.

Gymnasts exhibited significantly lower skinfold thicknesses than controls at all 7 sites measured. As well, the sum of 7 skinfolds was significantly less for

gymnasts than controls.

Table 4.3 Skinfold thicknesses of gymnasts and controls (mean \pm SE)

Site	Gymnasts (n=11)	Controls (n=10)	p Value ^a
Triceps (mm)	9.70 \pm 0.73	13.62 \pm 0.99	p<0.01
Biceps (mm)	5.80 \pm 0.55	8.50 \pm 0.64	p<0.01
Subscapular (mm)	6.39 \pm 0.63	9.91 \pm 1.00	p<0.01
Suprailiac (mm)	3.98 \pm 0.25	7.80 \pm 1.11	p<0.01
Abdomen (mm)	5.13 \pm 0.35	7.99 \pm 0.84	p<0.01
Anterior mid-thigh (mm)	10.88 \pm 0.64	15.83 \pm 1.05	p<0.01
Medial calf (mm)	8.37 \pm 0.56	12.46 \pm 0.79	p<0.01
Sum of 7 skinfolds (mm)	50.24 \pm 2.77	76.11 \pm 4.93	p<0.01

^a1-tailed *t*-test

Skinfold measurements were entered into prediction equations by Jackson et al. (1980) and Slaughter et al. (1988) in order to calculate percent body fat of gymnasts and controls. Estimates of percent body fat (determined by three different equations as well as DXA) for 11 gymnasts and 10 controls are shown in Table 4.4. Regardless of the method used to predict body fatness, gymnasts demonstrated significantly lower percent fat values than control subjects (p<0.01). However, as is evident in Table 4.4, the different methods used to estimate percent body fat yielded discrepant values for each group of subjects.

Table 4.4 Comparison of methods predicting percent body fat of gymnasts and controls (mean \pm SE)

Method	Gymnasts % Body Fat (n=11)	Controls % Body Fat (n=10)	p Value ^a
Jackson et al. (1980) & Siri (1961)*	10.2 \pm 0.4	14.3 \pm 0.8	p<0.01
Slaughter et al. (1988) (triceps + subscapular skinfolds)*	15.3 \pm 1.1	21.3 \pm 1.3	p<0.01
Slaughter et al. (1988) (triceps + calf skinfolds)*	16.2 \pm 0.7	21.0 \pm 0.9	p<0.01
DXA	7.9 \pm 1.4	15.4 \pm 2.0	p<0.01

DXA = dual energy x-ray absorptiometry

^a1-tailed *t*-test

*Refer to Appendix C for equations.

Least squares regression analysis was utilized to examine the relationship between percent body fat values calculated by prediction equations and DXA measurements. Percent body fat data of the two groups were pooled for the purpose of this analysis. A significant positive association was apparent when percent body fat levels estimated by the Jackson et al. (1980) and Siri (1961) equations were plotted against DXA values (Figure 4.2). However, this relationship appeared to be stronger in the leanest subjects and weaker in the fattest subjects. Percent body fat predicted by the Slaughter et al. (1988) equation using triceps and subscapular skinfolds was significantly correlated to values determined by DXA (Figure 4.3). There was also a significant positive relationship between percent

body fat estimated by the Slaughter et al. (1988) equation using triceps and calf skinfolds and DXA values (Figure 4.4). The relationship between percent body fat values predicted by either of the Slaughter et al. (1988) equations and those measured by DXA appeared to be consistent throughout the entire spectrum of body fatness (i.e., from the leanest to the fattest subjects) (Figures 4.3 and 4.4).

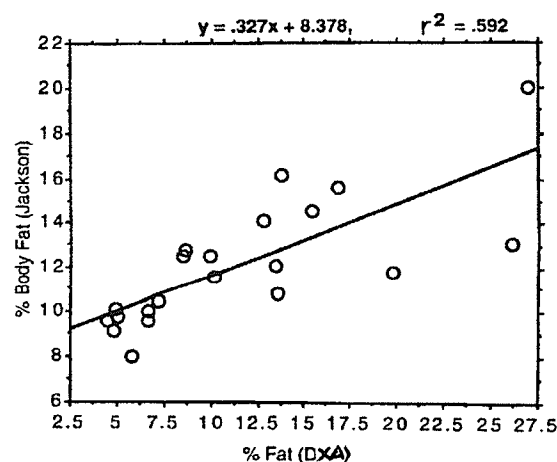


Figure 4.2 Relationship between percent body fat estimated by Jackson et al. (1980) and Siri (1961) and percent body fat determined by dual energy x-ray absorptiometry (DXA) in 21 pre-menarchial girls aged 9 - 13 years ($p < 0.01$)

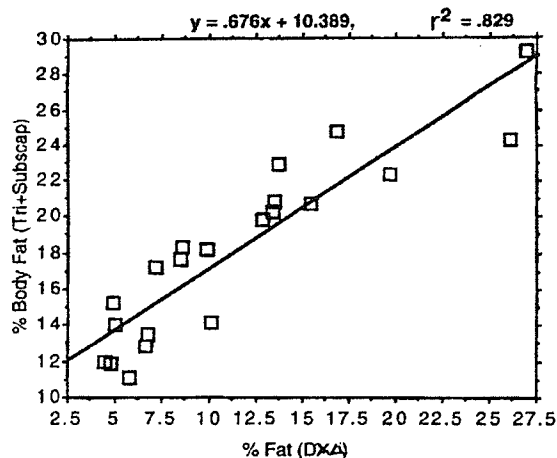


Figure 4.3 Relationship between percent body fat estimated by Slaughter et al. (1988) using triceps and subscapular skinfolds and percent body fat determined by dual energy x-ray absorptiometry (DXA) in 21 pre-menarchial girls aged 9 - 13 years ($p < 0.01$)

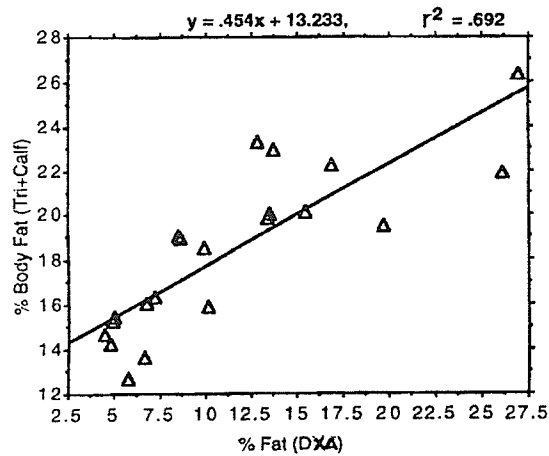


Figure 4.4 Relationship between percent body fat estimated by Slaughter et al. (1988) using triceps and calf skinfolds and percent body fat determined by dual energy x-ray absorptiometry (DXA) in 21 pre-menarchial girls aged 9 - 13 years ($p < 0.01$)

4.2.1 Body Circumference Measurements

Body circumference values for the gymnast and control groups are presented in Table 4.5. Gymnasts exhibited significantly lower circumferences than control subjects at all sites except the calf. When calf girths of gymnasts and controls were corrected for the medial calf skinfold thickness (corrected calf girth), no difference was detected between the two groups.

4.2.2 Bone Length Measurements

Bone length values of gymnast and control subjects are presented in Table 4.6. The two groups did not differ with respect to bone lengths at any site measured.

Table 4.5 Body circumferences of gymnasts and controls (mean \pm SE)

Site	Gymnasts (n=11)	Controls (n=15)	p Value ^a
Relaxed arm (cm)	20.9 \pm 0.6	23.1 \pm 0.7	p<0.05
Flexed/relaxed arm (cm)	21.8 \pm 0.6	23.9 \pm 0.7	p<0.05
Forearm (cm)	19.3 \pm 0.5	20.4 \pm 0.4	p<0.05
Wrist (cm)	13.6 \pm 0.2	14.3 \pm 0.2	p<0.05
Chest (cm)	70.9 \pm 2.1	76.4 \pm 1.4	p<0.05
Waist (cm)	59.2 \pm 1.4	64.9 \pm 1.4	p<0.01
Gluteal (cm)	74.7 \pm 2.0	82.0 \pm 1.7	p<0.01
Proximal thigh (cm)	42.8 \pm 1.2	46.7 \pm 1.0	p<0.01
Ankle (cm)	18.8 \pm 0.6	20.2 \pm 0.3	p<0.05
Calf (cm)	29.2 \pm 0.9	30.0 \pm 0.6	ns
Corrected calf girth (cm)*	26.6 \pm 0.9	25.8 \pm 0.8 (n=10)	ns

ns = not significant

^a1-tailed *t*-test*Corrected calf girth = (calf girth) - [(3.1416) \times (medial calf skinfold)/10]**Table 4.6 Bone lengths of gymnasts and controls (mean \pm SE)**

Site	Gymnasts (n=11)	Controls (n=15)	p Value ^a
Upper arm length (cm)	31.0 \pm 0.7	30.9 \pm 0.4	ns
Forearm length (cm)	22.5 \pm 0.5	22.4 \pm 0.3	ns
Hand length (cm)	17.6 \pm 0.4	17.3 \pm 0.2	ns
Thigh length (cm)	39.9 \pm 0.9	40.1 \pm 0.9	ns
Tibia length (cm)	36.4 \pm 1.1	35.7 \pm 0.5	ns

ns = not significant

^a2-tailed *t*-test

4.2.3 Bone Breadth Measurements

Table 4.7 depicts bone breadth values of the gymnast and control groups. With the exception of wrist breadth of the non-dominant hand, bone breadths of the gymnasts were significantly lower than those of controls at all sites evaluated.

Table 4.7 Bone breadths of gymnasts and controls (mean \pm SE)

Site	Gymnasts (n=11)	Controls (n=15)	p Value ^a
Humerus breadth (cm)	5.60 \pm 0.09	5.87 \pm 0.09	p<0.05
Wrist breadth: dominant hand (cm)	4.77 \pm 0.08	4.97 \pm 0.06	p<0.05
Wrist breadth: non-dominant hand (cm)	4.75 \pm 0.09	4.91 \pm 0.06	ns
Knee breadth (cm)	7.70 \pm 0.21	8.55 \pm 0.14	p<0.01
Ankle breadth (cm)	5.92 \pm 0.11	6.13 \pm 0.06	p<0.05

ns = not significant

^a1-tailed t-test

4.3 Bone Mineral Measurements

4.3.0 L₂-L₄ and Proximal Femur

Bone mineral content (BMC) and bone mineral density (BMD) values for gymnasts and controls at L₂-L₄, the femoral neck (FN), Ward's Triangle (WT) and the trochanter are presented in Table 4.8. Gymnasts demonstrated significantly greater BMC and BMD than control subjects at all sites, with the exception of trochanter BMC which did not differ between the two groups.

Table 4.8 Bone mineral data (L₂-L₄ and proximal femur) of gymnasts and controls (mean \pm SE)

Site	Gymnasts (n=11)	Controls (n=15)	p Value ^a
L ₂ -L ₄ BMC (g)	27.9 \pm 2.2	23.6 \pm 1.4	p<0.05
L ₂ -L ₄ BMD (g/cm ²)	0.919 \pm 0.04	0.809 \pm 0.024	p<0.01
Femoral neck BMC (g)	3.9 \pm 0.2	3.2 \pm 0.1	p<0.01
Femoral neck BMD (g/cm ²)	0.973 \pm 0.051	0.813 \pm 0.017	p<0.01
Ward's Triangle BMC (g)	1.7 \pm 0.1	1.5 \pm 0.1	p<0.05
Ward's Triangle BMD (g/cm ²)	0.994 \pm 0.048	0.826 \pm 0.024	p<0.01
Trochanter BMC (g)	7.3 \pm 0.7	6.0 \pm 0.4	ns
Trochanter BMD (g/cm ²)	0.814 \pm 0.033	0.688 \pm 0.024	p<0.01

BMC = bone mineral content; BMD = bone mineral density; ns = not significant.

^a1-tailed *t*-test

4.3.1 Regional and Total Body

Table 4.9 illustrates regional and total body BMC and BMD measurements of gymnast and control groups. BMC and BMD values did not differ between the two groups at any site measured, with the exception of the arms (BMD significantly greater in gymnasts) and lumbar spine (both BMC and BMD significantly greater in gymnasts).

4.4 Results of Dietary Analysis

None of the subjects in either the gymnast or control group exhibited symptoms suggestive of eating disorders, as assessed by the Eating Disorder Inventory (Garner & Olmstead, 1984). Daily energy and nutrient intake data of the gymnast and control groups are presented in Table 4.10. Gymnasts consumed

Table 4.9 Bone mineral data (regional and total body) of gymnasts and controls (mean \pm SE)

Site	Gymnasts (n=11)	Controls (n=15)	p Value ^a
Arms BMC (g)	262.3 \pm 14.6	234.3 \pm 10.0	ns
Arms BMD (g/cm ²)	0.675 \pm 0.015	0.638 \pm 0.008	p<0.05
Legs BMC (g)	602.6 \pm 49.1	581.8 \pm 32.2	ns
Legs BMD (g/cm ²)	0.914 \pm 0.04	0.900 \pm 0.023	ns
Trunk BMC (g)	425.5 \pm 39.6	407.1 \pm 21.2	ns
Trunk BMD (g/cm ²)	0.737 \pm 0.027	0.698 \pm 0.013	ns
Ribs BMC (g)	150.4 \pm 13.8	142.0 \pm 7.5	ns
Ribs BMD (g/cm ²)	0.601 \pm 0.021	0.567 \pm 0.008	ns
Pelvis BMC (g)	135.7 \pm 12.9	127.5 \pm 8.2	ns
Pelvis BMD (g/cm ²)	0.900 \pm 0.04	0.855 \pm 0.022	ns
Spine BMC (g)	138.7 \pm 13.5	137.7 \pm 6.4	ns
Spine BMD (g/cm ²)	0.793 \pm 0.033	0.751 \pm 0.017	ns
Thoracic BMC (g)	85.3 \pm 9.5	91.9 \pm 4.6	ns
Thoracic BMD (g/cm ²)	0.724 \pm 0.031	0.720 \pm 0.016	ns
Lumbar spine BMC (g)	54.2 \pm 4.6	45.8 \pm 2.4	p<0.05
Lumbar spine BMD (g/cm ²)	0.931 \pm 0.044	0.820 \pm 0.024	p<0.05
Total body BMC (g)	1634.6 \pm 111.2	1568.0 \pm 61.4	ns
Total body BMD (g/cm ²)	0.887 \pm 0.027	0.865 \pm 0.013	ns

BMC = bone mineral content; BMD = bone mineral density; ns = not significant

^a1-tailed *t*-test

significantly less energy, fat and carbohydrate than controls, but protein intake did not differ between the two groups. Compared to control subjects, gymnasts ingested a significantly higher percentage of total daily energy as protein. However, the groups showed no difference with respect to percentage of energy derived from fat and carbohydrate. Protein intake (expressed on a g/kg body weight basis) of gymnasts and controls was 186% and 185%, respectively, of the Canadian Nutrition Recommendations (Health and Welfare Canada, 1990).

Table 4.10 Nutritional data of gymnasts and controls (mean \pm SE)

Variable	Gymnasts (n=11)	Controls (n=15)	p Value ^a
Energy (kJ/d)	7176.0 \pm 360.8	9455.4 \pm 556.6	p<0.01
Protein (g/d)	67.3 \pm 3.2	77.6 \pm 6.7	ns
Protein (g/kg/d)	1.88 \pm 0.14	1.87 \pm 0.16	ns
Fat (g/d)	59.5 \pm 5.2	83.1 \pm 7.7	p<0.05
Carbohydrate (g/d)	233.5 \pm 12.2	316.6 \pm 16.7	p<0.01
Protein (%kJ)	16.1 \pm 0.7	13.9 \pm 0.8	p<0.05
Fat (%kJ)	30.3 \pm 1.7	31.8 \pm 1.5	ns
Carbohydrate (%kJ)	55.5 \pm 1.7	56.4 \pm 1.8	ns
Calcium (mg/d)	956.8 \pm 78.1	1070.1 \pm 88.6	ns
Phosphorus (mg/d)	1213.4 \pm 69.4	1508.4 \pm 143.9	ns
Calcium:phosphorus ratio	0.78 \pm 0.03	0.74 \pm 0.04	ns
Vitamin D (μ /d)	6.95 \pm 1.59	6.74 \pm 1.30	ns

ns = not significant

^a2-tailed *t*-test

There were no differences between the gymnast and control groups in terms of calcium, phosphorus or vitamin D consumption, or calcium to phosphorus ratio. Mean daily calcium intake for the gymnasts and controls represented 87% (range 50% - 128%) and 97% (range 55% - 161%), respectively, of the RNI (1100 mg of calcium per day for girls aged 10 to 12 years). Eighteen percent of gymnasts (n=2) and 20% of controls (n=3) consumed calcium levels which were less than two-thirds of the RNI. Phosphorus intake of gymnast and control subjects was 152% and 189%, respectively, of the RNI. In terms of vitamin D, gymnasts consumed 139% and controls ingested 135% of the RNI.

4.5 Relationships between Variables

Least squares linear regression analysis was performed to define the relationship between the following variables in the gymnast and control groups: age and weight, age and height, age and BMD, height and BMD, weight and BMD, bone-free lean tissue and BMD, hours of structured physical activity and BMD, and calcium intake and BMD. Results of regression analysis are reported below.

4.5.0 Age and Weight

A significant positive correlation was observed between age and weight in gymnasts ($r^2=0.60$, $p<0.01$), while no relationship was detected between these two variables in control subjects.

4.5.1 Height and Weight

In the gymnast group, height and weight showed a significant positive association ($r^2=0.81$, $p<0.01$). However, these two variables failed to demonstrate a significant relationship in controls.

4.5.2 Bone-Free Lean Tissue and Weight

Bone-free lean tissue (BFLT) was significantly and positively related to weight in both gymnasts ($r^2=0.92$, $p<0.01$) and controls ($r^2=0.60$, $p<0.01$).

4.5.3 Age and BMD

A significant positive correlation between age and BMD was evident for the total body (Figure 4.5A), L_2-L_4 (Figure 4.6A) and FN (Figure 4.7A) in gymnasts, while age and WT BMD were not significantly related in this group (Figure 4.8A).

In the control group, the slopes of the regression lines between age and BMD were not significantly different from zero for the total body (Figure 4.5B) and L_2-L_4 sites (Figure 4.6B). However, a significant negative association was observed between age and BMD of FN (Figure 4.7B) and WT (Figure 4.8B) for control subjects.

4.5.4 Height and BMD

Table 4.11 depicts the relationship between height and BMD of L_2-L_4 , FN, WT and the total body in both gymnast and control groups. For gymnasts, height and BMD were significantly and positively correlated at L_2-L_4 , FN and the total body,

but no association was evident at WT. In control subjects, height and BMD were not significantly related at any of the sites analyzed.

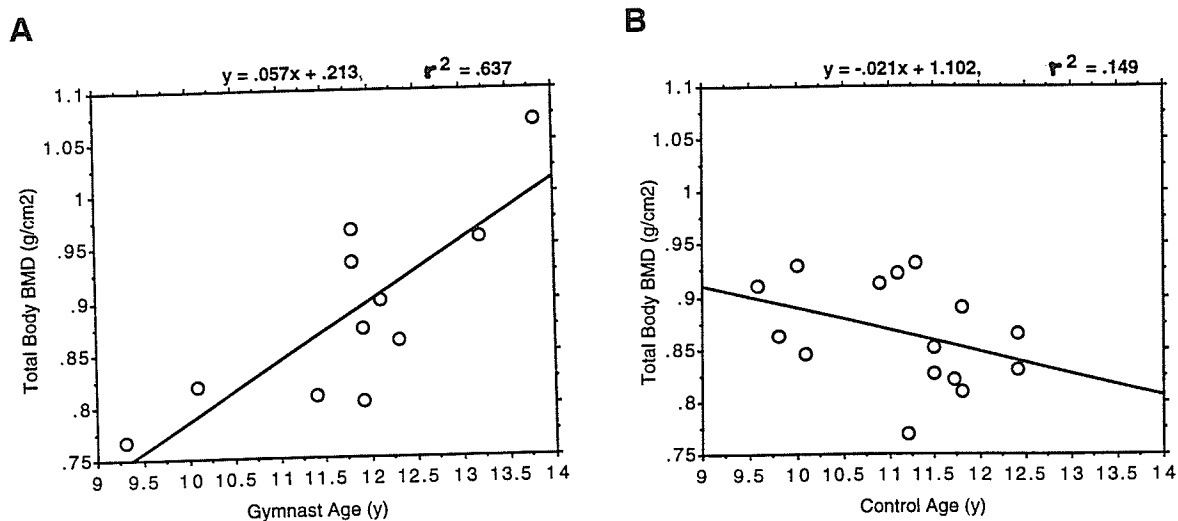


Figure 4.5 The relationship between age and total body bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p < 0.01$), and (B) 15 pre-menarchial normally active girls (controls) (ns)

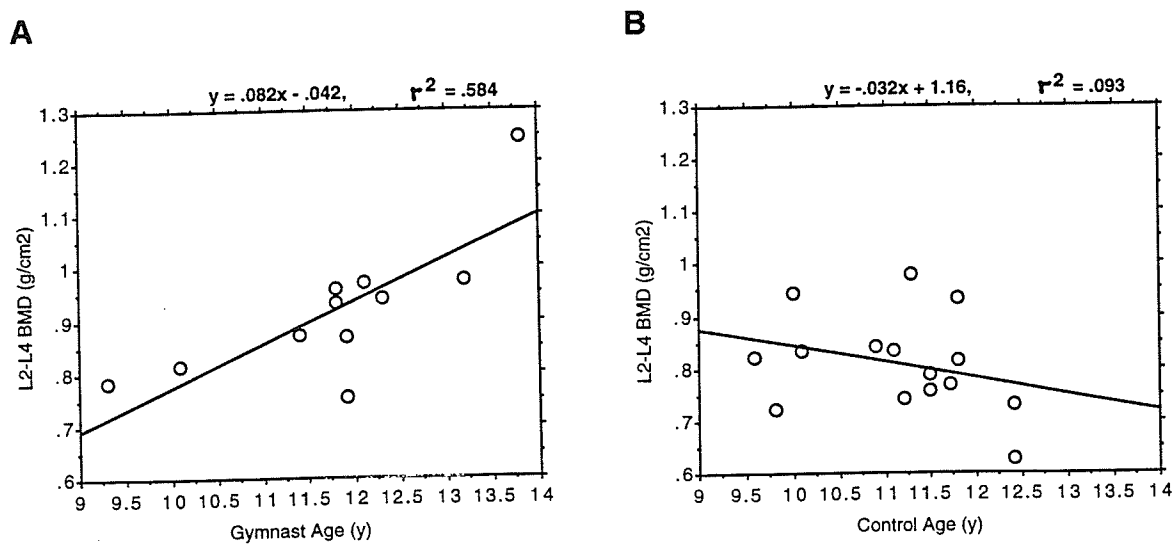


Figure 4.6 The relationship between age and L₂-L₄ bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p < 0.01$), and (B) 15 pre-menarchial normally active girls (controls) (ns)

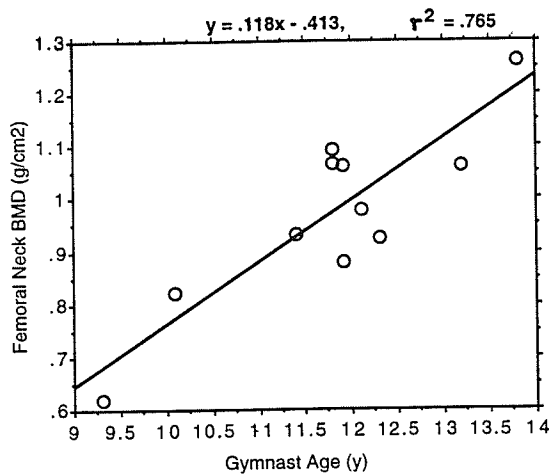
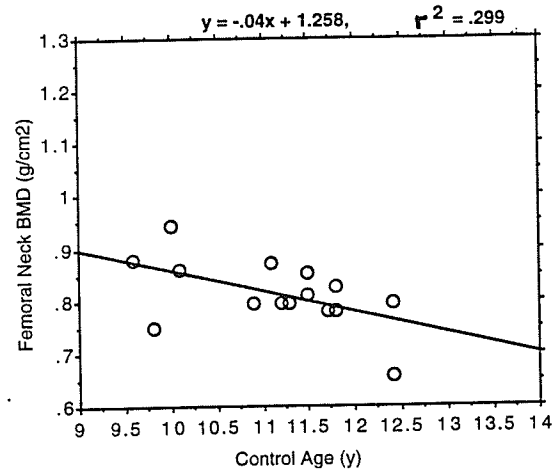
A**B**

Figure 4.7 The relationship between age and femoral neck bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p < 0.01$), and (B) 15 pre-menarchial normally active girls (controls) ($p < 0.05$)

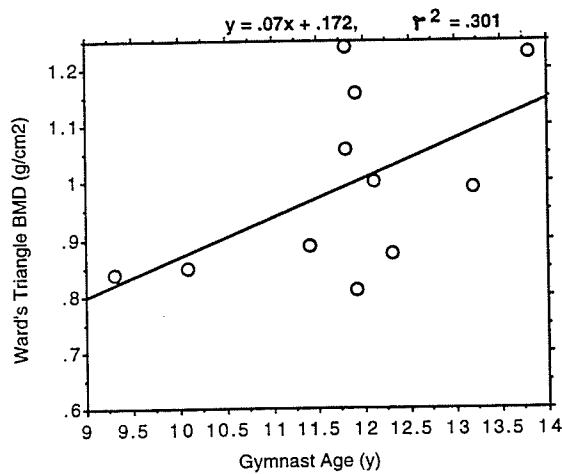
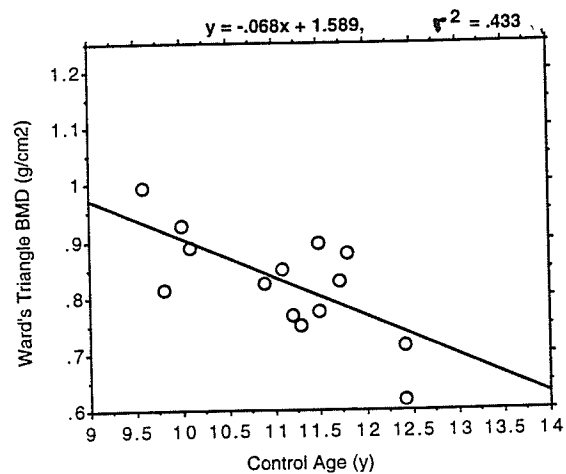
A**B**

Figure 4.8 The relationship between age and Ward's Triangle bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts (ns), and (B) 15 pre-menarchial normally active girls (controls) ($p < 0.01$)

4.5.5 Weight and BMD

In the gymnast group, a significant positive correlation was apparent between weight and BMD of the total body (Figure 4.9A), L₂-L₄ (Figure 4.10A) and FN (Figure 4.11A), but not WT (Figure 4.12A). For control subjects, weight and BMD were significantly and positively related for the total body (Figure 4.9B) and L₂-L₄ sites (Figure 4.10B). However, no association was demonstrated between these two variables at FN (Figure 4.11B) or WT (Figure 4.12B) in the control group.

Table 4.11 Regression line equations (slope and intercept) of the relationship between height (cm) and BMD (g/cm²) in gymnasts and controls

BMD	Regression Equation (slope and intercept)	r ²	p Value
Gymnasts (n = 11)			
L ₂ -L ₄	= 0.01(Ht) - 0.614	0.52	p<0.05
FN	= 0.014(Ht) - 1.036	0.57	p<0.01
WT	= 0.007(Ht) + 0.034	0.14	ns
Total Body	= 0.007(Ht) - 0.213	0.60	p<0.01
Controls (n = 15)			
L ₂ -L ₄	= 0.006(Ht) - 0.09	0.17	ns
FN	= 0.004(Ht) + 0.231	0.14	ns
WT	= 0.003(Ht) + 0.371	0.04	ns
Total Body	= 0.002(Ht) + 0.543	0.08	ns

BMD = bone mineral density; FN = femoral neck; Ht = height; ns = not significant; WT = Ward's Triangle.

4.5.6 Bone-Free Lean Tissue and BMD

Table 4.12 shows the relationship between bone-free lean tissue (BFLT) and BMD of L₂-L₄, FN, WT and the total body in gymnasts and controls. In the gymnast group, BFLT was significantly and positively associated with BMD at all sites. In control subjects, a significant positive correlation between these two variables was evident at L₂-L₄, but not at FN, WT or the total body.

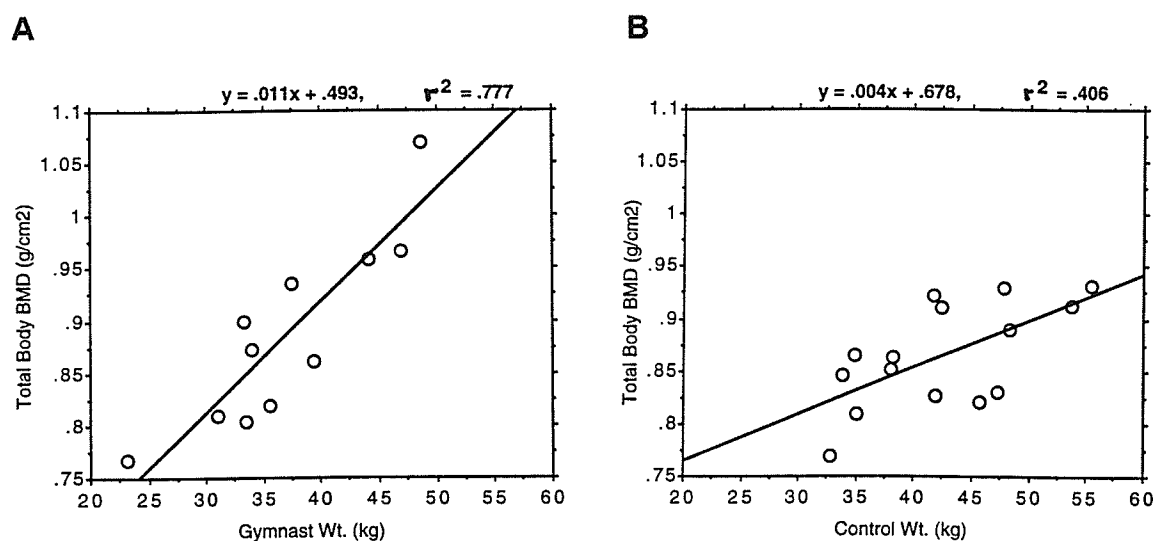


Figure 4.9 The relationship between weight and total body bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p < 0.01$), and (B) 15 pre-menarchial normally active girls (controls) ($p < 0.05$)

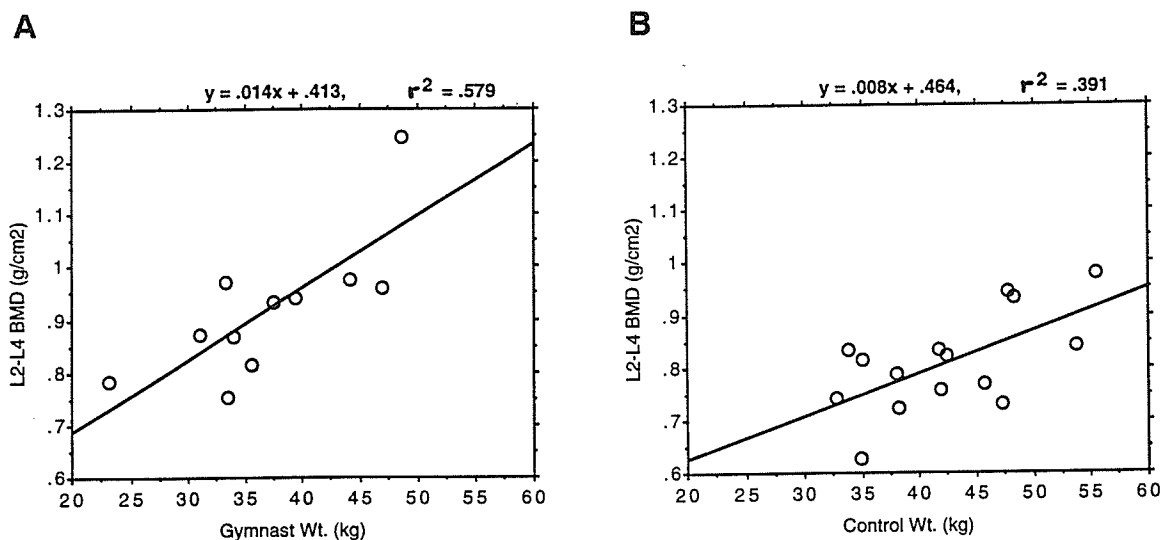


Figure 4.10 The relationship between weight and L₂-L₄ bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p < 0.01$), and (B) 15 pre-menarchial normally active girls (controls) ($p < 0.05$)

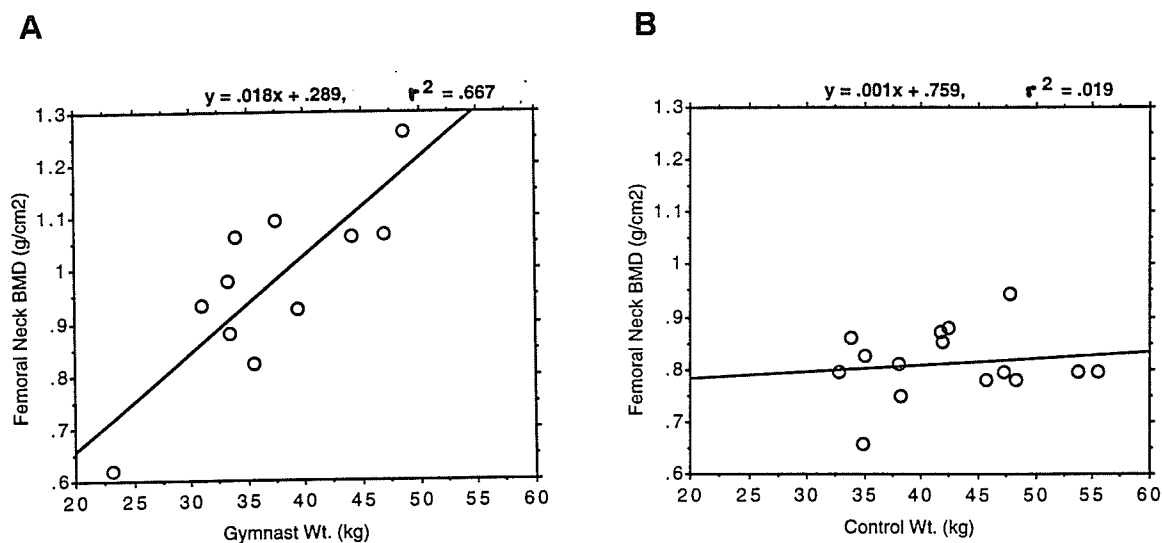


Figure 4.11 The relationship between weight and femoral neck bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts ($p < 0.01$), and (B) 15 pre-menarchial normally active girls (controls) (ns)

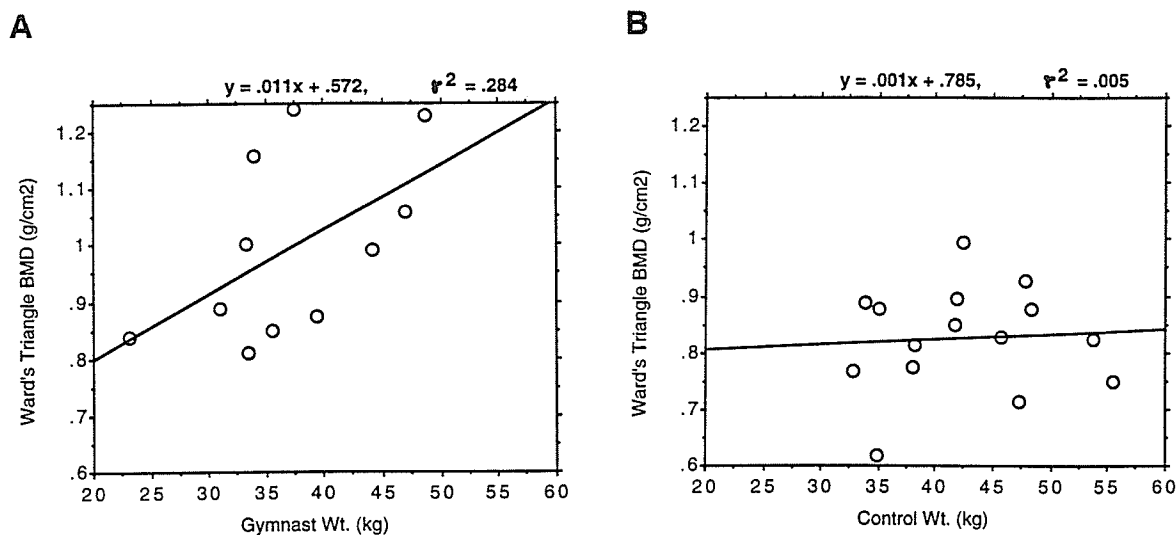


Figure 4.12 The relationship between weight and Ward's Triangle bone mineral density (BMD) in: (A) 11 pre-menarchial gymnasts (ns), and (B) 15 pre-menarchial normally active girls (controls) (ns)

Table 4.12 Regression line equations (slope and intercept) of the relationship between bone-free lean tissue weight (kg) and BMD (g/cm²) in gymnasts and controls

BMD	Regression Equation (slope and intercept)	r ²	p Value
Gymnasts (n = 11)			
L ₂ -L ₄	= 0.02(BFL) + 0.317	0.66	p<0.01
FN	= 0.027(BFL) + 0.154	0.78	p<0.01
WT	= 0.018(BFL) + 0.445	0.39	p<0.05
Total Body	= 0.015(BFL) + 0.431	0.85	p<0.01
Controls (n = 15)			
L ₂ -L ₄	= 0.013(BFL) + 0.407	0.34	p<0.05
FN	= 0.006(BFL) + 0.632	0.14	ns
WT	= 0.007(BFL) + 0.593	0.11	ns
Total Body	= 0.006(BFL) + 0.691	0.22	ns

BFL = bone free lean tissue; BMD = bone mineral density; FN = femoral neck; ns = not significant; WT = Ward's Triangle.

4.5.7 Physical Activity and BMD

No significant relationship between hours/week of structured physical activity and BMD of the total body, L₂-L₄, FN, or WT was detected in either the gymnast or control group.

4.5.8 Calcium Intake and BMD

Calcium intake was not found to be significantly associated with BMD of the total body, L₂-L₄, FN or WT in either gymnast or control subjects.

5. DISCUSSION

5.0 Summary of Study Design and Results

The present study was a cross-sectional investigation which compared bone mineral density (BMD) in pre-menarchial competitive gymnasts aged 9 to 13 years to that of normally active girls (controls) of the same age and pubertal status. In addition, an attempt was made to identify factors which most strongly predict BMD in this cohort of young females. The key findings of this study were that pre-menarchial girls who participate in competitive gymnastics are characterized by significantly lower body weight, less body fat, and higher BMD of the lumbar spine, femoral neck (FN), Ward's Triangle (WT), trochanter and arms than their normally active counterparts.

5.1 General Subject Characteristics

Subjects had to meet specific age and Tanner stage criteria in order to participate in the study; therefore, it was not surprising that age and level of pubertal development were similar for gymnasts and controls. Members of the two groups were also comparable in terms of height. Gymnasts weighed significantly less than control subjects, an observation which was expected based on reports in the literature (Fehling et al., 1995; Lindholm et al., 1994; Nichols et al., 1994). Control subjects of the same height and weight as gymnasts may have provided a more useful comparison group in this investigation; however, few such individuals

who met these criteria could be located.

5.2 Body Composition and Anthropometric Measurements

Gymnasts and controls exhibited similar lean tissue and bone-free lean tissue (BFLT) mass when values were expressed in absolute terms (i.e., g or kg lean tissue weight). However, when values were stated in relative terms (i.e., percent of total body weight), lean tissue and BFLT comprised significantly greater portions of body mass in gymnasts compared to controls. This observation can be explained by the fact that gymnasts weighed less than controls, yet demonstrated comparable absolute values for lean tissue and BFLT mass.

Mean sum of 7 skinfold scores for both the gymnast and control groups were less than that reported for 86 healthy girls aged 5-14 years (mean age 9.53 years) (Miller et al., 1991). Regardless of the method used to determine percent body fat in our study (i.e., DXA or skinfold prediction equations), values for both gymnasts and controls were lower than those featured in the literature for girls of a similar age and pubertal status (Frerichs et al., 1979; Janz et al., 1993; Slaughter et al., 1988). Thus, it would appear that our sample of gymnasts and controls was leaner than average.

Not surprisingly, gymnasts were significantly leaner than control subjects, as was evidenced by less fat tissue mass, smaller skinfold thicknesses, and lower body fat percentages. Reports in the literature corroborate this finding; Fehling et al. (1995) and Kirchner et al. (1995) observed significantly lower body fat

percentages in female collegiate gymnasts compared to sedentary college-aged women, despite similarities in mean group weight. Given that the gymnasts in our study were healthy young girls, their leanness (and lower body weights) was likely related to their intensive physical training and/or an energy intake insufficient to support the demands of high levels of training. It is reasonable to assume that the gymnasts expended more energy than control subjects, considering that their involvement in structured physical activity was over three times that of controls. Thus, a high energy output combined with a lower energy intake (relative to controls) provides a reasonable explanation for the difference in body fat (and body weight) seen in the groups. However, one cannot rule out the possibility that young girls who are genetically predisposed to leanness gravitate towards gymnastics and demonstrate talent in the sport, while other girls with a genetic inclination to possess higher body fat levels fail to perform well at gymnastics and drop out or never participate to begin with (Kirchner et al., 1995).

With the exception of calf girth, body circumferences of gymnasts were significantly smaller than those of controls. Since lean body tissue mass was comparable for both groups of subjects, the difference in body circumferences was presumably a function of lower body fat levels in the gymnasts. Similarly, the lower bone breadths observed at a majority of sites in gymnasts relative to controls was likely due to gymnasts' leanness. Bone lengths of gymnasts and control subjects were similar, which was not surprising given that stature of the two groups was comparable.

5.2.0 Estimates of Percent Body Fat in Subjects

Percent body fat values determined by DXA ($\% \text{fat}_{\text{DXA}}$) were markedly lower in gymnasts and controls when compared to estimates obtained from prediction equations using skinfold thicknesses. The equation by Jackson et al. (1980) yielded percent body fat values which were closest to DXA measurements for both the gymnast and control groups, while the two equations by Slaughter et al. (1988) generated substantially higher percent body fat estimates. This observation is contrary to findings of Ogle et al. (1995), who reported that DXA overestimated percent body fat in girls and young women compared to values predicted by the Slaughter equations. Measurement errors, including variation in skinfold compressibility, skinfold site location and measurement technique, may explain the contradictory nature of our results and those of Ogle's group (Lohman, 1988).

The Jackson et al. (1980) equation was developed for predicting body density in women. Equations by Slaughter et al. (1988) have been validated for use with children and youths aged 8-18 years, and thus would presumably be more appropriate for predicting body fatness in our subjects. At present, the Slaughter skinfold equations are considered the best anthropometric method available for estimating percent body fat in children and adolescents (Janz et al., 1993).

Percent fat_{DXA} and percent fat values calculated by each of the three prediction equations were significantly and positively correlated when data from the gymnast and control groups were pooled. The Slaughter et al. (1988) equation

using triceps and subscapular skinfold thicknesses showed the strongest relationship with % fat_{DXA} throughout the entire spectrum of body fat levels in subjects, while the Jackson et al. (1980) equation demonstrated the weakest association with % fat_{DXA}. Although the correlation with % fat_{DXA} was quite close in the leanest subjects, the association appeared to weaken as subjects' body fat values increased. Thus, the Jackson equation may not be an appropriate method for predicting percent body fat in pre-menarchial girls

Based on findings from this study, it is not possible to conclude which method yielded the most accurate estimate of fat content for our subjects. However, it should be noted that subject compliance was much higher with DXA than skinfold anthropometry. Values for percent body fat were obtained on all 26 subjects when DXA was used to assess body composition. However, when skinfold thicknesses were determined, 5 subjects complained of discomfort, and consequently, were not measured. Thus, in terms of subject cooperation, DXA may be the preferred method for determination of body fatness in children in clinical research settings.

5.3 Nutrient Intake

In terms of overall nutriture, gymnasts exhibited suboptimal levels of energy and calcium intake, while control subjects fell marginally below the Canadian Nutrition Recommendations (1990) for calcium. Although it is tempting to attribute the gymnasts' low energy intake to under-eating during the diet record period or

under-reporting of food consumption, the observation of low energy intakes among competitive gymnasts has been well documented in the literature (Benardot, 1996; Benardot et al., 1989; Kirchner et al., 1995; Loosli et al., 1986; Nattiv & Mandelbaum, 1993, Sundgot-Borgen, 1996). Thus, it was not surprising to observe the same trend in our gymnastics subjects. None of the gymnasts exhibited signs of eating disorders, but given that aesthetic appeal is an integral component in judging gymnastics performance, these young competitors were likely cognizant of physique. Thus, it is possible that some of the gymnasts in this study restricted energy intake in an effort to achieve or maintain their perceived ideal body image. Another explanation for the lower consumption of energy by the gymnasts could have been simply a lack of time to eat. Given that their busy schedules accommodated gymnastics training and competition, school, homework and other recreational pursuits, finding time in their day to consume adequate energy may have posed a challenge.

It stands to reason that the reduced fat and CHO intake by gymnasts compared to controls was a reflection of the gymnasts' lower energy consumption. While protein intake did not differ between the groups when expressed in absolute terms (i.e., g protein/day) or as relative consumption (i.e., g protein/kg body weight/day), protein did comprise a significantly greater percentage of total energy intake for gymnasts. Thus it would appear that gymnasts consumed a more protein dense diet than controls.

The gymnasts and controls ingested comparable levels of calcium, although

neither group met the Canadian RNI of 1100 mg/day. The observation that both groups failed to meet the RNI is substantiated by reports in the literature which indicate an habitual underconsumption of calcium by girls and young women (Kirchner et al., 1995; Matkovic, 1991; Nichols et al., 1994; Sentipal et al., 1991). However, it is important to acknowledge that RNI's are intended to meet the needs of almost all individuals and therefore exceed the actual requirements of the majority of people. Thus, a nutrient intake which is less than the RNI is not necessarily inadequate, although the further an intake falls below the recommended level, the greater the probability of insufficiency (Health and Welfare Canada, 1990).

It is interesting to compare nutrient intakes of the gymnasts in this investigation with those of elite U.S. female artistic gymnasts aged 7-14 years documented by Benardot et al. (1989). Gymnasts in our study ingested amounts of energy, fat, CHO and protein which were almost identical to levels consumed by the U.S. gymnasts. Intake of calcium and phosphorus was approximately 100 mg/day lower in the U.S. gymnasts compared to that of our gymnasts. Benardot et al. (1989) did not report vitamin D intake in their nutritional assessment.

5.4 Bone Mineral Measurements

5.4.0 Lumbar Spine and Proximal Femur Bone Mass

As hypothesized, gymnasts demonstrated significantly greater bone mineral content (BMC) and BMD at L₂-L₄, FN, WT, and the trochanter compared with

controls. Other studies examining BMD in children and young women who engage in regular, weight-bearing exercise have reported comparable results. Grimston et al. (1993) observed significantly higher FN BMD in children (aged 10 - 16 years) who competed in impact loading sports versus children of the same age who participated in an active loading sport (i.e., swimming). Nichols et al. (1994) found that in female collegiate gymnasts, L₂-L₄ and FN BMD's were 112% and 121%, respectively, of normative values for women of the same age group. Similarly, Kirchner et al. (1995) reported that BMD's of the lumbar spine and proximal femur were significantly higher in female college gymnasts than age-matched non-athletic controls.

A key factor distinguishing the gymnasts from controls in this study was the amount of physical activity performed by each group. As mentioned earlier, gymnasts devoted more than three times as many hours per week to structured physical activity as controls. Furthermore, the bulk of this time was allocated to gymnastics training which consists of dynamic, weight-bearing exercise. Unfortunately, it was not possible to evaluate the proportion of weekly structured weight-bearing activity (i.e., impact loading through gravitational forces acting on bone) versus non-weight-bearing exercise (i.e., active loading through muscular contraction pulling on bone) for control subjects. It would be useful to quantify time devoted to each of these activity categories, since weight-bearing exercise appears to benefit bone mass while non-weight-bearing exercise does not seem to confer any such advantage (Grimston et al., 1993; Fehling et al., 1995).

Despite the fact that gymnasts weighed less than control subjects, they demonstrated significantly greater BMC and BMD at lumbar spine and proximal femur sites. Since height, lean tissue mass and calcium consumption were similar for the two groups, it is possible that the difference in quantity of bone mineral at these highly trabecular sites was partially a function of dynamic skeletal loading produced through rigorous gymnastics training. In support of this theory, it has been reported that female collegiate athletes who participated in sports which loaded the skeleton with high magnitude, short duration stimuli (i.e., gymnastics and volleyball) exhibited significantly greater lumbar spine and proximal femur BMD than swimmers and non-athletic young women (Fehling et al., 1995). Others have observed site specific elevations of BMD in female college gymnasts (Kirchner et al., 1995; Nichols et al., 1994), elite figure skaters (Slemenda & Johnston, 1993), and children who engaged in high impact, weight-bearing sports (Grimston et al., 1993).

One potentially confounding factor which may have influenced lumbar spine bone mass in the gymnasts relates to spondylolysis, a spinal injury which is fairly common in young female gymnasts (Lindholm et al., 1994; Nattiv & Mandelbaum, 1993). It is thought that repetitive flexion, rotation and extension of the spine during gymnastics training may cause a stress fracture in the pars interarticularis, resulting in spondylolysis (Nattiv & Mandelbaum, 1993). This condition most often occurs at L₄ or L₅, and results in a shift in the weight distribution pattern such that the superior lumbar vertebrae assume a greater share of the load. Although none of the

gymnasts indicated a history of back pain or back injury, spondylolysis is not necessarily manifested by overt symptoms and may therefore go undetected (N. Craton, personal communication, July 18, 1996). Spondylolysis can be diagnosed by x-ray or bone scan. However, since neither procedure was conducted in this investigation, the presence of spondylolysis in one or more of the gymnasts cannot be ruled out. If spondylolysis had existed in any of the gymnasts, calcification at the fracture site may have contributed to the higher lumbar BMC and BMD seen in this group of subjects. Since gymnasts also exhibited greater bone mass at FN, WT and the trochanter (skeletal sites comprised of predominantly trabecular bone, as are the vertebral bodies), it is reasonable to surmise that intensive physical training rather than calcification arising from spondylolysis was responsible for the higher lumbar bone mass exhibited by the gymnasts.

In terms of other variables which may have accounted for the higher lumbar spine and hip BMD's observed in gymnasts, it seems safe to rule out smoking, caffeine intake and alcohol consumption, as these lifestyle factors were not an issue for our subjects. Also, given that all girls were Caucasian, the potential effect of racial differences on bone mass is negated. However, it is feasible that genetics and/or the hormonal milieu may have contributed to the difference in lumbar spine and hip BMD's between gymnasts and controls. Genetic factors are thought to play a major role in determination of bone mass, accounting for 60 to 80% of the variance (Slemenda et al., 1994). Furthermore, there is speculation that an individual's sensitivity to specific environmental or lifestyle factors may be mediated

by genetics (Kelly et al., 1993). It is certainly possible that girls who inherit the potential for higher BMD are also gifted gymnasts, while others who are genetically programmed to have lower BMD avoid participation in the sport (Kirchner et al., 1995).

In terms of hormonal effects, we attempted to control for the influence of estrogen on bone mass by examining only pre-menarchial gymnasts and controls at the same stage of puberty. However, since blood sampling was not performed on our subjects, differences in levels of hormones which have an impact on bone metabolism (such as growth hormone, insulin-like growth factor, calcitonin, parathyroid hormone and vitamin D) cannot be ruled out.

5.4.1 Regional and Total Body Bone Mass

When BMC and BMD of regional sites and the total body were compared in the two groups, BMD of the arms was found to be significantly greater in the gymnasts versus controls. This finding is corroborated by Fehling et al. (1995), who observed significantly higher arm BMD in collegiate female gymnasts compared to competitive swimmers and non-athletic women of the same age). The greater arm BMD seen in gymnasts is likely related to the high mechanical strain rates placed on the arms during gymnastics training and competition (Fehling et al., 1995).

In this investigation, no significant differences in BMC or BMD were detected between the gymnast and control groups at the remaining regional sites or for the total body. The activities involved in gymnastics training are thought to produce

forces up to 10 times the body weight at the tissue level (Fehling et al., 1995), which would lead one to expect a marked difference in bone mass between the gymnasts and controls, particularly at loading sites such as the legs and the pelvis. Fehling et al. (1995) reported significantly greater total body, leg and pelvis BMD in their gymnastics subjects compared to swimmers and non-athletes. Kirchner et al. (1995) also discovered significantly higher total body BMD in female college gymnasts than age-matched non-athletic women. Conversely, Slemenda and Johnston (1993) observed no difference in BMD of the legs or pelvis between competitive female figure skaters aged 11-14 years and non-athletic girls, although significant differences between the groups did emerge in subjects 15 years and older.

In our study, it is possible that the higher body weight demonstrated by the control group exerted enough of a loading effect on the skeleton to produce BMD's comparable to those of gymnasts. As for the greater arm BMD in gymnasts, training for gymnastics demands that the arms be routinely stressed through repetitive mechanical loading, while many other sports and activities of daily living do not. Thus, a heavier body weight would not be expected to confer an advantage at upper body skeletal sites.

Gymnasts exhibited significantly greater bone mass at the lumbar spine and proximal femur, while no difference was apparent in total body, leg and pelvis BMD between the two groups. It is conceivable that the type of bone which predominates at these sites (i.e., trabecular or cortical) may determine the extent to which

mechanical loading can exert an effect. Although the positive impact of intensive weight-bearing exercise on bone mass at highly trabecular sites (i.e., the lumbar spine and proximal femur) is well documented in the literature (Fehling et al., 1995; Grimston et al., 1993; Kirchner et al., 1995; Nichols et al., 1994; Ruiz et al., 1995; Slemenda et al., 1991), only a few studies have reported regional and total body BMD values (i.e., sites at which cortical bone prevails) (Fehling et al., 1995; Kirchner et al., 1995; Nichols et al., 1994; Slemenda & Johnston, 1993). Trabecular bone is more metabolically active than cortical bone, and therefore is considered a better indicator of bone mass (Buchanan et al., 1988; Riggs & Melton, 1986). It also predominates at clinically important sites which are prone to fracture in osteoporotic women (Dalsky, 1990). In our pre-menarchial subjects, it would appear that rigorous gymnastics training had a dramatic impact on bone mass at trabecular sites, but less influence at cortical locations.

5.5 Relationships Between Independent Variables and BMD

5.5.0 Age, Height, Weight, Bone-Free Lean Tissue and BMD

Simple regression analysis revealed that, in gymnasts, age and BMD were significantly and positively related at the lumbar spine, FN, and total body, although no association was evident at WT. Conversely, a very different pattern emerged in control subjects. A strong negative relationship was detected between age and BMD at FN and WT in controls, suggesting that bone density at these sites decreased with increasing age, while no correlation was found at the lumbar spine

or total body in the control group. Several reports in the literature have described a strong positive relationship between age and BMD in children and adolescents (Kroger et al., 1992; Kroger et al., 1993; Lu et al., 1994; Matkovic et al., 1994; Slemenda et al., 1991; Teegarden et al., 1995). However, others have argued that any effect of age on bone mass in children is probably mediated by associated changes in height, weight or pubertal status (Grimston et al., 1992; Katzman et al., 1991). Since our subjects were homogeneous with respect to height and pubertal stage, the contrasting relationships between age and BMD seen in gymnasts and controls cannot be attributed to differences in these two developmental factors. In the gymnasts, it is conceivable that the positive association between age and BMD was also a reflection of the number of years devoted to training (i.e., the older gymnasts would have trained for a greater length of time than the younger gymnasts, which may have confounded the age-BMD relationship). In the control subjects, the possibility that weight clouded the relationship between age and BMD warrants consideration. A number of studies have demonstrated that increased body weight is associated with higher bone mass during childhood and adolescence (DeSchepper et al., 1991; Glastre et al., 1990; Katzman et al., 1991; Kroger et al., 1992; Ponder et al., 1990; Rubin et al., 1989; Slemenda et al., 1994; Teegarden et al., 1995). In our investigation, age and weight were significantly and positively correlated in gymnasts, but no such relationship was apparent in controls. Thus, in the control group, the youngest subjects were not necessarily the lightest, and vice versa. As a result, the association between age and BMD of the hip was

likely obscured by weight.

A significant positive relationship was evident between height and BMD of the lumbar spine, FN and total body in gymnasts, but not at WT. On the other hand, height was not found to be an independent predictor of BMD at any skeletal site in the control group. Associations between height and bone mass have frequently been observed in children (DeSchepper et al., 1991; Glastre et al., 1990; Katzman et al., 1991; Ponder et al., 1990; Rubin et al., 1989; Slemenda et al., 1994). In our group of gymnasts, height and weight showed a significant positive relationship, but no such correlation was detected in control subjects. Consequently, it would appear that weight also played a confounding role in the height-BMD association in control subjects.

In gymnasts, a strong positive relationship was seen between weight and BMD at all sites, with the exception, once again, of WT. In controls, a significant positive association between weight and BMD was noted only for the lumbar spine and total body, while no relationship was seen at either hip site. As mentioned earlier, weight has often been cited as having a positive association with bone mass in growing children and youths (DeSchepper et al., 1991; Glastre et al., 1990; Katzman et al., 1991; Kroger et al., 1992; Ponder et al., 1990; Rubin et al., 1989; Slemenda et al., 1994; Teegarden et al., 1995). In our study, this relationship held true for gymnasts at three of the four sites examined. However, in controls, weight did not independently predict BMD at either location in the proximal femur. One explanation for the lack of association is that weight operated in concert with other

independent variables (such as age, height, physical activity and/or calcium intake) to exert an effect on proximal femur BMD. Unfortunately, due to the small number of subjects enrolled in this investigation, we were unable to perform multiple regression analysis on each group separately in order to clarify the relative contribution of the independent variables on BMD of gymnasts and controls.

Bone-free lean tissue represents the weight of muscle, the vital organs and visceral tissue. Although it is not synonymous with muscle mass, BFLT is considered a good substitute as muscle comprises a considerable portion of its composition (Faulkner et al., 1993). In gymnasts, a significant positive relationship between BFLT and BMD of the lumbar spine, proximal femur and total body was observed. However, only lumbar spine BMD was positively associated with BFLT in control subjects, while the remaining sites showed no correlation between these two variables. The literature is equivocal regarding the effect of lean body mass on bone mineral measures in children. Faulkner et al. (1993) have asserted that the amount of BFLT appears to be more important than body weight in predicting total body BMD during childhood and adolescence. Conversely, Teegarden et al. (1995) found that lean mass alone did not provide the best model to estimate bone mineral in children and young adults. In our study, BFLT was significantly and positively correlated to body weight in both the gymnast and controls groups. Thus, one would expect that in each group of subjects, the BFLT-BMD relationship would reflect that of the weight-BMD relationship. With the exception of WT BMD in gymnasts (which was related to BFLT but not weight) and total body in controls

(which showed an association with weight but not BFLT), this was in fact the pattern that emerged.

To summarize the preceding section, age, height, weight and BFLT were all strong, independent predictors of lumbar spine, FN and total body BMD in gymnasts when simple linear regression analyses were performed, although WT BMD was associated with only BFLT. The strong relationship between age and weight, height and weight, and BFLT and weight in gymnasts provides an explanation as to why each of these variables was significantly associated with BMD in these subjects. As for the lack of correlation between age, height or weight and WT BMD in members of the gymnast group, this site may have been more responsive to a combination of factors working synergistically than a single factor exerting its effect independently.

In control subjects, age was negatively related to BMD of the proximal femur, and showed no association with lumbar spine or total body BMD. Height was not correlated with BMD at any site, while weight was positively related to BMD of the lumbar spine and total body, but not the proximal femur. BFLT showed a positive association with BMD only at the lumbar spine. These relationships (and absence thereof) are very different from those observed in the gymnasts, and likely reflect a lack of correlation between age, height, and weight in members of the control group. Consequently, the relationships resulting from simple regression analyses may have been spurious ones for control subjects. As alluded to previously, had our sample size been larger, it would have been useful to perform multiple

regression analysis on each group separately, in order to determine the ability of the independent variables to predict BMD of gymnasts and controls.

5.5.1 Physical Activity, Calcium Intake and BMD

Weekly structured physical activity was not significantly related to BMD of the lumbar spine, proximal femur or total body in either gymnasts or control subjects. The majority of reports in the literature have indicated that exercise plays an important role in maximizing peak bone mass in children and adolescents (Fehily et al., 1992; Geusens et al., 1991; Halioua & Anderson, 1989; Kroger et al., 1992; McCulloch et al., 1990; Slemenda et al., 1991; Slemenda et al., 1994) although the definition of exercise is inconsistent from one investigation to another. Nevertheless, studies by Katzman et al. (1991) and Kroger et al. (1993) have failed to detect a correlation between physical activity and BMD during childhood and adolescence. In our study, the most plausible explanation for the lack of association between physical activity and BMD in gymnasts and controls relates to the homogeneity of each group in terms of the amount of structured exercise performed. Little variation in hours of structured physical activity per week was evident within each group, and as a result, the lack of association between physical activity and BMD for gymnasts and controls was not surprising.

Calcium intake failed to independently predict BMD at any skeletal site in gymnasts or controls. This finding is in agreement with several reports in the literature, which have indicated no significant association between calcium

consumption and bone mass during growth and maturation (Glastre et al., 1990; Grimston et al., 1992; Katzman et al., 1991; Kroger et al., 1993; Slemenda and Johnston, 1993). Nevertheless, a number of other studies have demonstrated that calcium is a strong determinant of bone mass in children and adolescents (Johnston et al., 1992; Matkovic et al., 1990; Ruiz et al., 1995; Sentipal et al., 1991). These researchers have typically employed large samples comprised of subjects with widespread levels of calcium consumption. In our investigation, the range of calcium intake in each of the subject groups may not have been broad enough to elicit any detectable impact of calcium on bone. Furthermore, a large percentage of the gymnasts and controls ingested calcium levels which were at least two-thirds of the RNI, suggesting that calcium consumption of the majority of subjects was substantial enough to preclude a negative effect on bone mass.

The inability of calcium to predict BMD should not be construed to mean that this nutrient was unimportant to bone mass in our pre-menarchial subjects. Heaney (1991) has commented that calcium does not ensure optimal bone status, but is a necessary prerequisite for it to occur. There is speculation that a trade-off situation between calcium and physical activity exists, such that highly active females can optimize bone mass even if their calcium intake is low (Anderson & Metz, 1993).

5.6 Summary

In spite of their light body weight, gymnasts demonstrated significantly greater bone mass than controls at sites of the lumbar spine and proximal femur,

while total body BMD was similar for the two groups. Thus, it is possible that trabecular bone was more responsive to mechanical loading through physical activity than cortical bone in our study participants. Since the lumbar spine and hip are particularly susceptible to fractures in post-menopausal women, maximizing bone mass at these sites early in life cannot be overemphasized.

The results of this study lend support to the theory that regular physical activity is critical for the optimization of peak bone mass in young girls. Maximizing peak bone mass in the early years could translate into higher bone mineral reserves at menopause and a reduced risk of osteoporosis in later years.

6. CONCLUSIONS AND RECOMMENDATIONS

The results of the present study support the following conclusions:

1. In a sample of competitive pre-menarchial gymnasts, aged 9-13 years, bone mineral density of the lumbar spine, proximal femur and arms is higher than that in a sample of normally active girls of the same age and pubertal status.
2. Gymnastics appears to provide the appropriate type of training to increase bone mineral density in pre-menarchial girls.
3. In a sample of pre-menarchial girls, physical activity appears to have a positive effect on bone mineral density at predominantly trabecular skeletal sites of the lumbar spine and proximal femur, but less influence at primarily cortical regions of the total body.
4. The lack of association between calcium consumption and bone mineral density in a sample of pre-menarchial gymnasts and normally active girls is likely due to relatively homogeneous calcium intakes which are, on average, marginally below the Recommended Nutrient Intake.
5. Percent body fat determinations by dual energy x-ray absorptiometry are highly correlated with those estimated by skinfold prediction equations in a sample of pre-menarchial girls. However, contrary to reports in the literature, dual energy x-ray absorptiometry yields lower estimates of percent body fat than skinfold equations in samples of highly active and normally active pre-menarchial girls.

This investigation has identified physical activity as an important determinant of skeletal health in pre-menarchial girls. Specifically, high performance gymnastics training pre-menarchially seems to be advantageous in terms of enhancing bone mineral density. However, it is not known if ongoing training throughout adolescence and into the adult years will continue to benefit bone mass, or if other factors commonly reported in gymnasts (such as delayed menarche and menstrual disturbances, low calcium intake and eating disorders) will negatively alter the bone health profile seen pre-menarchially. Therefore, research which prospectively examines gymnasts early on in their training careers and follows them through puberty and post-puberty is needed.

The results of this study underline the importance of girls establishing a regular pattern of physical activity early in life to augment bone mineral density. Although the sport of gymnastics seems ideal for increasing bone mass in pre-menarchial girls, it would not be reasonable to advocate that all young girls train intensively as gymnasts. Thus, unresolved issues which require further investigation include identifying the minimum volume of exercise, the type of exercise and the minimum number of years of training necessary to achieve maximum benefit to bone. At the present time, it is not clear if more exercise leads to further increases in bone mass, or if there is an activity threshold beyond which no further accumulation of bone mass can be expected in girls. From a practical perspective, there likely comes a point where the time demands of exercising and/or the risk of injury exceed any positive effects of physical activity on bone mass.

Thus, exercise prescriptions geared towards optimizing bone health in young girls must be realistic.

Although calcium intake did not demonstrate an effect on bone in this study, young girls should nevertheless be advised to consume a diet which meets current nutrition recommendations for calcium, in order to provide the substrate needed for bone mineralization. Obviously, the issues of optimal calcium intake level and its impact on bone mass in girls are unresolved and warrant additional clarification.

Based on the results of this investigation, young girls are strongly encouraged to participate in regular, weight-bearing physical activity in order to enhance bone mineral density and lessen their risk of osteoporotic fracture later in life.

REFERENCES

REFERENCES

- Alexander, M.J.L. 1991. A comparison of physiological characteristics of elite and subelite rhythmic gymnasts. Journal of Human Movement Studies. 20:49-69.
- Aloia, J.F., Vaswani, A.N., Yeh, J.K. and Cohn, S.H. 1988. Premenopausal bone mass is related to physical activity. Archives of Internal Medicine. 148:121-123.
- Anderson, J.J.B. and Metz, J.A. 1993. Contributions of dietary calcium and physical activity to primary prevention of osteoporosis in females. Journal of the American College of Nutrition. 12:378-383.
- Barr, S.I. 1987. Women, nutrition and exercise: a review of athletes' intakes and a discussion of energy balance in active women. Progress in Food and Nutrition Science. 11:307-361.
- Benardot, D. 1996. Working with young athletes: views of a nutritionist on the sports medicine team. International Journal of Sport Nutrition. 6:110-120.
- Benardot, D., Schwarz, M. and Weitzenfeld-Heller, D. 1989. Nutrient intake in young, highly competitive gymnasts. Journal of the American Dietetic Association. 89:401-403.
- Buchanan, J.R., Meyers, C., Lloyd, R., Leuenberger, P. and Demers, L. 1988. Determinants of peak trabecular bone density in women: the role of androgens, estrogens and exercise. Journal of Bone and Mineral Research. 8:673-680.
- Calvo, M.S. 1993. Dietary phosphorus, calcium metabolism and bone. Journal of Nutrition. 123:1627-1633.
- Canalis, E. 1993. Insulin like growth factors and the local regulation of bone formation. Bone. 14:273-276.
- Charles, P., Eriksen, E.F., Hasling, C., Sondergard, K. and Mosekilde, L. 1991. Dermal, intestinal, and renal obligatory losses of calcium: relation to skeletal calcium loss. American Journal of Clinical Nutrition. 54:266S-273S.
- Chestnut III, C.H. 1987. Noninvasive techniques for measuring bone mass: a comparative review. Clinical Obstetrics and Gynecology. 30:812-819.

Chestnut III, C.H. 1991. Theoretical overview: bone development, peak bone mass, bone loss, and fracture risk. The American Journal of Medicine. 91(suppl. 5B):2S-4S.

Dalsky, G.P. 1990. Effect of exercise on bone: permissive influence of estrogen and calcium. Medicine and Science in Sports and Exercise. 22:281-285.

Dequeker, J., Nijas, J., Verstraeten, A., Geusens, P. and Gevers, G. 1987. Genetic determinants of bone mineral content at the spine and radius: a twin study. Bone. 8:207-209.

DeSchepper, J., Derde, M.P., Van den Broeck, M., Piepsz, A. and Jonckheer, M.H. 1991. Normative data for lumbar spine bone mineral content in children: influence of age, height, weight, and pubertal status. The Journal of Nuclear Medicine. 32:216-220.

Dhuper, S., Warren, M.P., Brooks-Gunn, J. and Fox, R. 1990. Effects of hormonal status on bone density in adolescent girls. Journal of Clinical Endocrinology and Metabolism. 71:1083-1088.

Drinkwater, B.L., Nilson, K., Chestnut, C.H., Bremner, W.J., Shainholtz, S. and Southworth, M.B. 1984. Bone mineral content of amenorrhoeic and eumenorrhoeic athletes. New England Journal of Medicine. 811:277-281.

Dueck, C.A., Manore, M.M. and Matt, K.S. 1996. Role of energy balance in athletic menstrual dysfunction. International Journal of Sport Nutrition. 6:165-190.

Duke, P.M., Litt, I.F. and Gross, R.T. 1980. Adolescents' self-assessment of sexual maturation. Pediatrics. 66:918-920.

Einhorn, T.A. 1992. Bone strength: the bottom line. Calcified Tissue International. 51:333-339.

Eisman, J.A., Sambrook, P.N., Kelly, P.J. and Pocock, N.A. 1991. Exercise and its interaction with genetic influences in the determination of bone mineral density. The American Journal of Medicine. 91(suppl. 5B):5S-9S.

Eriksen, E.F., Colvard, D.S., Berg, N.J., Graham, M.L., Mann, K.G., Spelsberg, T.C. and Riggs, B.L. 1988. Evidence of estrogen receptors in normal human osteoblast-like cells. Science. 241:84-86.

Faulkner, R.A., Bailey, D.A., Drinkwater, D.T., Wilkinson, A.A., Houston, C.S. and McKay, H.A. 1993. Regional and total body bone mineral content, bone mineral density, and total body tissue composition in children 8-16 years of age. Calcified Tissue International. 53:7-12.

Fehily, A.M., Coles, R.J., Evans, W.D. and Elwood, P.C. 1992. Factors affecting bone density in young adults. American Journal of Clinical Nutrition. 56:579-586.

Fehling, P.C., Alekel, L., Clasey, J., Rector, A. and Stillman, R.J. 1995. A comparison of bone mineral densities among female athletes in impact loading and active loading sports. Bone. 17:205-210.

Firooznia, H., Golimbu, C., Rafii, M. and Schwartz, M.S. 1989. Osteoporosis: comparative value of currently available diagnostic radiographic modalities. Resident and Staff Physician. 35(12):53-60.

Fisher, E.C., Nelson, M.E., Frontera, W.R., Turksoy, R.N. and Evans, W.J. 1986. Bone mineral content and levels of gonadotropins and estrogens in amenorrhoeic running women. Journal of Clinical Endocrinology and Metabolism. 62:1232-1236.

Fogelman, I. and Ryan, P. 1992. Measurement of bone mass. Bone. 13:S23-S28.

Frerichs, R.R., Harsha, D.W. and Berenson, G.S. 1979. Equations for estimating percentage of body fat in children 10-14 years old. Pediatric Research. 13:170-174.

Frisch, R.E., Gotz-Welbergen, A.V., McArthur, J.W., Albright, R., Witschi, J., Bullen, B., Birnholz, J., Reed, R.B. and Hermann, H. 1981. Delayed menarche and amenorrhoea of college athletes in relation to age of onset of training. Journal of the American Medical Association. 246(14):1559-1563.

Frusztajer, N.T., Dhuper, S., Warren, M.P., Brooks-Gunn, J. and Fox, R.P. 1990. Nutrition and incidence of stress fractures in ballet dancers. American Journal of Clinical Nutrition. 51:779-783.

Fuller, N.J., Laskey, M.A. and Elia, M. 1992. Assessment of the composition of major body regions by dual-energy x-ray absorptiometry (DEXA), with special reference to limb muscle mass. Clinical Physiology. 12:253-266.

Garner, D.M. and Olmstead, M.P. 1984. Eating Disorder Inventory. Odessa, FL: Psychological Assessment Resources, Inc.

Garner, D.M., Olmstead, M.P. and Polivy, J. 1983. Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. International Journal of Eating Disorders. 2:15-34.

Genant, H.K., Faulkner, K.G. and Gluer, C.C. 1991. Measurement of bone mineral density: current status. The American Journal of Medicine. 91(suppl. 5B):49S-53S.

Genuth, S.M. 1988. The endocrine system. In: Berne, R.M. and Levy, M.N. (Eds.), Physiology (2nd ed.). St. Louis, MO: The C.V. Mosby Company.

Geusens, P., Cantatore, F., Nijs, J., Proesmans, W., Emma, F. and Dequeker, J. 1991. Heterogeneity of growth of bone in children at the spine, radius and total skeleton. Growth, Development and Aging. 55:249-256.

Gilsanz, V., Gibbons, D.T., Roe, T.F., Carlson, M., Senac, M.O., Boechat, M.I., Huang, H.K., Schulz, E.E., Libanati, C.R. and Cann, C.C. 1988. Vertebral bone density in children: effect of puberty. Radiology. 166:847-850.

Glastre, C., Braillon, P., David, L., Cochat, P., Meunier, P.J. and Delmas, P.D. 1990. Measurement of bone mineral content of the lumbar spine by dual x-ray absorptiometry in normal children: correlations with growth parameters. Journal of Clinical Endocrinology and Metabolism. 70:1330-1333.

Gordon, C.L., Halton, J.M., Atkinson, S.A. and Webber, C.E. 1991. The contributions of growth and puberty to peak bone mass. Growth, Development and Aging. 55:257-262.

Grimston, S.K., Morrison, K., Harder, J.A. and Hanley, D.A. 1992. Bone mineral density during puberty in Western Canadian children. Bone and Mineral. 19:85-96.

Grimston, S.K., Willows, N.D. and Hanley, D.A. 1993. Mechanical loading regime and its relationship to bone mineral density in children. Medicine and Science in Sports and Exercise. 25:1203-1210.

Haarbo, J., Gottfredsen, A., Hassager, C. and Christiansen, C. 1991. Validation of body composition by dual energy x-ray absorptiometry (DEXA). Clinical Physiology. 11:331-341.

Halioua, L. and Anderson, J.J.B. 1989. Lifetime calcium intake and physical activity habits: independent and combined effects on the radial bone of healthy premenopausal Caucasian women. American Journal of Clinical Nutrition. 49:534-541.

Hassard, T.H. 1991. Understanding Biostatistics. St. Louis, MO: Mosby - Year Book, Inc.

Health and Public Policy Committee, American College of Physicians. 1987. Bone mineral densitometry. Annals of Internal Medicine. 107:932-936.

Health and Welfare Canada, 1990. Nutrition Recommendations. Ottawa, ON: Supply and Services Canada.

Heaney, R.P. 1991. Effect of calcium on skeletal development, bone loss, and risk of fractures. The American Journal of Medicine. 91(suppl. 5B):23S-28S.

Heaney, R.P. 1987. The role of nutrition in prevention and management of osteoporosis. Clinical Obstetrics and Gynecology. 70:833-846.

Hight, R. 1987. Athletic amenorrhoea: an update on aetiology, complications and management. Sports Medicine. 7:82-108.

Houtkooper, L.B. 1996. Assessment of body composition in youths and relationship to sport. International Journal of Sport Nutrition. 6:146-164.

Jackson, A.S., Pollock, M.L. and Ward., A. 1980. Generalized equations for predicting body density of women. Medicine and Science in Sports and Exercise. 12:175-182.

Jacobson, P.C., Beaver, W., Grubb, S.A., Taft, T.N. and Talmage, R.V. 1984. Bone density in women: college athletes and older athletic women. Journal of Orthopaedic Research. 2:328-332.

Janz, K.F., Nielsen, D.H., Cassady, S.L., Cook, J.S., Wu, Y.-T. and Hansen, J.R. 1993. Cross-validation of the Slaughter skinfold equations for children and adolescents. Medicine and Science in Sports and Exercise. 25:1070-1076.

Jensen, M.D. 1992. Research techniques for body composition assessment. Journal of the American Dietetic Association. 92:454-460.

Johansen, J.S., Giwercman, A., Hartwell, D. et al. 1988. Serum bone Gla-protein as a marker of bone growth in children and adolescents: correlation with age, height, serum insulin-like growth factor I, and serum testosterone. Journal of Clinical Endocrinology and Metabolism. 76:273-278.

Johnston, C.C., Miller, J.Z., Slemenda, C.W., Reister, T.K., Hui, S., Christian, J.C. and Peacock, M. 1992. Calcium supplementation and increases in bone mineral density in children. The New England Journal of Medicine. 327(2):82-87.

Kannus, P., Haapasalo, H., Sankelo, M., Sivanen, H., Pasanen, M., Heinonen, A., Oja, P. and Vuori, I. 1995. Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. Annals of Internal Medicine. 123:27-31.

Katzman, D.K., Bachrach, L.K., Carter, D.R. and Marcus, R. 1991. Clinical and anthropometric correlates of bone mineral acquisition in healthy adolescent girls. Journal of Clinical Endocrinology and Metabolism. 73:1332-1339.

Kellie, S.E. 1992. Diagnostic and Therapeutic Technology Assessment (DATTA). Journal of the American Medical Association. 267(2):286-294.

Kelly, P.J., Nguyen, T., Hopper, J., Pocock, N., Sambrook, P. and Eisman, J. 1993. Changes in axial bone density with age: a twin study. Journal of Bone and Mineral Research. 8:11-17.

Kelly, T.L., Slovick, D.M., Schoenfeld, D.A. and Neer, R.M. 1988. Quantitative digital radiography versus dual photon absorptiometry of the lumbar spine. Journal of Clinical Endocrinology and Metabolism. 67:839-844.

Kirchner, E.M., Lewis, R.D. and O'Connor, P.J. 1995. Bone mineral density and dietary intake of female college gymnasts. Medicine and Science in Sports and Exercise. 27:543-549.

Kroger, H., Kotaniemi, A., Kroger, L. and Alhava, E. 1993. Development of bone mass and bone density of the spine and femoral neck -- a prospective study of 65 children and adolescents. Bone and Mineral. 23:171-182.

Kroger, H., Kotaniemi, A., Vainio, P. and Alhava, E. 1992. Bone densitometry of the spine and femur in children by dual-energy x-ray absorptiometry. Bone and Mineral. 17:75-85.

Kustin, J. and Rebar, R.W. 1987. Menstrual disorders in the adolescent age group. Primary Care. 14:139-166.

Lindholm, C., Hagenfeldt, K. and Ringertz, B. 1994. Pubertal development in elite juvenile gymnasts: effects of physical training. Acta Obstetrica et Gynecologica Scandinavica. 73:269-273.

Lloyd, T., Myers, C., Buchanan, J.R. and Demers, L.M. 1988. Collegiate women athletes with irregular menses during adolescence have decreased bone density. Obstetrics and Gynecology. 72:639-642.

Lohman, T.G. 1992. Exercise training and body composition in childhood. Canadian Journal of Sport Sciences. 17:284-287.

Lohman, T.G. 1988. Anthropometry and body composition. In: Lohman, T.G., Roche, A.F. and Martorell, R. (Eds.), Anthropometric Standardization Reference Manual. Champaign, IL: Human Kinetics Books.

Lohman, T.G., Roche, A.F. and Martorell, R. (Eds.). 1988. Anthropometric Standardization Reference Manual. Champaign, IL: Human Kinetics Books.

Loosli, A.R., Benson, J., Gillien, D.M. and Bourdet, K. 1986. Nutrition habits and knowledge in competitive adolescent female gymnasts. The Physician and Sportsmedicine. 14:118-130.

Loucks, A.B. and Horvath, S.M. 1985. Athletic amenorrhoea: a review. Medicine and Science in Sports and Exercise. 17:56-72.

Lu, P.W., Briody, J.N., Ogle, G.D., Morley, K., Humphries, I.R.J., Allen, J., Howman-Giles, R., Sillence, D. and Cowell, C.T. 1994. Bone mineral density of total body, spine, and femoral neck in children and young adults: a cross-sectional and longitudinal study. Journal of Bone and Mineral Research. 9:1451-1458.

Lutz, J. 1986. Bone mineral, serum calcium, and dietary intakes of mother/daughter pairs. American Journal of Clinical Nutrition. 44:99-106.

Lutz, J. and Tesar, R. 1990. Mother-daughter pairs: spinal and femoral bone densities and dietary intakes. American Journal of Clinical Nutrition. 52:872-877.

Malina, R.M., Wyrick, W., Tate, C. and Baylor, A.M. 1978. Age of menarche and selected menstrual characteristics in athletes at different competitive levels and in different sports. Medicine and Science in Sports and Exercise. 10:218-222.

Marcus, R. 1987. Calcium intake and skeletal integrity: is there a critical relationship? Journal of Nutrition. 117:631-635.

Martin, A.D. and McCulloch, R.G. 1987. Bone dynamics: stress, strain and fractures. Journal of Sports Medicine. 5:144-163.

Matkovic, V. 1992. Calcium and peak bone mass. Journal of Internal Medicine. 231:151-160.

Matkovic, V. 1991. Calcium metabolism and calcium requirements during skeletal modelling and consolidation of bone mass. American Journal of Clinical Nutrition. 52:540-549.

Matkovic, V., Fontana, D., Tominac, C., Goel, P. and Chestnut III, C.H. 1990. Factors that influence peak bone mass formation: a study of calcium balance and the inheritance of bone mass in adolescent females. American Journal of Clinical Nutrition. 52:878-888.

Matkovic, V., Jelic, T., Wardlaw, G.M., Ilich, J. Z., Goel, P.K., Wright, J.K., Andon, M.B., Smith, K.T. and Heaney, R.P. 1994. Timing of peak bone mass in Caucasian females and its implication for the prevention of osteoporosis. The Journal of Clinical Investigation. 93:799-808.

Matkovic, V. Kostial, K., Simonovic, I., Buzina, R., Brodarec, A. and Nordin, B.E.C. 1979. Bone status and fracture rates in two regions of Yugoslavia. American Journal of Clinical Nutrition. 32:540-549.

Mazess, R.B. 1990. Bone density of the axial skeleton. The Orthopedic Clinics of North America. 21(1):51-63.

Mazess, R.B. and Barden, H.S. 1988. Measurement of bone by dual-photon absorptiometry (DPA) and dual-energy x-ray absorptiometry (DEXA). Annales Chirurgiae et Gynaecologiae. 77:197-203.

Mazess, R.B. and Barden, H.S. 1989. Bone densitometry for diagnosis and monitoring osteoporosis. Proceedings of the Society for Experimental Biology and Medicine. 191:261-271.

Mazess, R.B., Barden, H.S., Bisek, J.P. and Hanson, J. 1990. Dual-energy x-ray absorptiometry for total-body and regional bone-mineral and soft-tissue composition. American Journal of Clinical Nutrition. 51:1106-1112.

McCormick, D.P., Fawcett, H.D. and Ponder, S.W. 1989. Spinal bone mineral density in healthy children. [abstract]. Journal of Bone and Mineral Research. 4(S1):S308.

McCormick, D.P., Ponder, S.W., Fawcett, H.D. and Palmer, J.L. 1991. Spinal bone mineral density in 335 normal and obese children and adolescents: evidence for ethnic and sex differences. Journal of Bone and Mineral Research. 6:507-513.

McCulloch, R.G., Bailey, D.A., Houston, C.S. and Dodd, B.L. 1990. Effects of physical activity, dietary calcium intake and selected lifestyle factors on bone density in young women. Canadian Medical Association Journal. 142:221-227.

McCulloch, R.G., Whiting, S.J., Bailey, D.A. and Houston, C.S. 1991. The effect of cigarette smoking on trabecular bone density in premenopausal women, aged 20-35 years. Canadian Journal of Public Health. 82:434-435.

Metz, J.A., Anderson, J.J. and Gallagher Jr., P.N. 1993. Intakes of calcium, phosphorus, and protein, and physical-activity level are related to radial bone mass in young adult women. American Journal of Clinical Nutrition. 58:537-542.

Miller, J.Z., Slemenda, C.W., Meaney, F.J., Reister, T.K., Hui S. and Johnston, C.C. 1991. The relationship of bone mineral density and anthropometric variables in healthy male and female children. Bone and Mineral. 14:137-152.

Nattiv, A. and Mandelbaum, B.R. 1993. Injuries and special concerns in female gymnasts. The Physician and Sportsmedicine. 21(7):66-82.

Neter, J. and Wasserman, W. In: Kutner, M.H. 1990. Applied Linear Statistical Models (3rd ed.). Homewood, IL: Irwin.

Nichols, D.L., Sanborn, C.F., Bonnick, S.L., Ben-Ezra, V., Gench, B. and DiMarco, N.M. 1994. The effects of gymnastics training on bone mineral density. Medicine and Science in Sports and Exercise. 26:1220-1225.

Nielsen, H.K., Brixen, K., Bouillon, R. and Mosekilde, L. 1990. Changes in biochemical markers of osteoblastic activity during the menstrual cycle. Journal of Clinical Endocrinology and Metabolism. 70:1431-1437.

O'Connor, P.J., Lewis, R.D. and Kirchner, E.M. 1995. Eating disorder symptoms in female college gymnasts. Medicine and Science in Sports and Exercise. 27:550-555.

Ogle, G.D., Allen, J.R., Humphries, I.R.J., Lu, P.W., Briody, J.N., Morley, K., Howman-Giles, R. and Cowell, C.T. 1995. Body-composition assessment by dual-energy x-ray absorptiometry in subjects aged 4-26 y. American Journal of Clinical Nutrition. 61:746-753.

Oritz, O., Russell, M., Daley, T.L., Baumgartner, R.N., Waki, M., Lichtman, S., Wang, J., Pierson Jr., R.N. and Heymsfield, S.B. 1992. Differences in skeletal muscle and bone mineral mass between black and white females and their relevance to estimates of body composition. American Journal of Clinical Nutrition. 55:8-13.

Ott, S.M. 1990. Editorial: attainment of peak bone mass. Journal of Clinical Endocrinology and Metabolism. 71:1082A-1082C.

Parfitt, A.M. and Chir, B. 1987. Bone remodelling and bone loss: understanding the pathophysiology of osteoporosis. Clinical Obstetrics and Gynecology. 30:789-811.

Peacock, M. 1991. Calcium absorption efficiency and calcium requirements in children and adolescents. American Journal of Clinical Nutrition. 54:261S-265S.

Phelps, L. and Bajorek, E. 1991. Eating disorders of the adolescent: current issues in etiology, assessment, and treatment. School Psychology Review. 20:9-22.

Pocock, N.A., Eisman, J.A., Hopper, J.L., Yeates, M.G., Sambrook, P.N. and Eberl, S. 1987. Genetic determinants of bone mass in adults. Journal of Clinical Investigation. 80:706-710.

Pollitzer, W.S. and Anderson, J.J.B. 1989. Ethnic and genetic differences in bone mass: a review with a hereditary vs. environmental perspective. American Journal of Clinical Nutrition. 50:1244-1259.

Ponder, S.W., McCormick, D.P., Fawcett, D., Palmer, J.L., McKernan, M.G. and Brouhard, B.H. 1990. Spinal bone mineral density in children aged 5.00 through 11.99 years. American Journal of Disease in Children. 144:1346-1348.

Prior, J.C. Vigna, Y.M., Schechter, M.T. and Burgess, A.E. 1990. Spinal bone loss and ovulatory disturbances. The New England Journal of Medicine. 323(18):1221-1227.

Raisz, L.G. 1988. Local and systemic factors in the pathogenesis of osteoporosis. The New England Journal of Medicine. 318:818-828.

Riggs, B.L. and Melton, L.J. 1986. Involutional osteoporosis. The New England Journal of Medicine. 314:1676-1686.

Riggs, B.L., Wahner, H.W., Seeman, E., Offord, K.P., Dunn, W.L., Mazess, R.B., Johnson, K.A. and Melton III, L.J. 1982. Differences between the post-menopausal and senile osteoporosis syndromes. Journal of Clinical Investigation. 70:716-723.

Roubenoff, R., Kehayias, J.J., Dawson-Hughes, B. and Heymsfield, S.B. 1993. Use of dual-energy x-ray absorptiometry in body-composition studies: not yet a "gold standard". American Journal of Clinical Nutrition. 58:589-591.

Rubin, K.R., Schirduan, V.M., Gendreau, P. and Dalsky, G.P. 1989. Determinants of bone density in healthy children and adolescents. [abstract]. Journal of Bone and Mineral Research. 4(S1):S373.

Ruiz, J.C., Mandel, C. and Garabedian, M. 1995. Influence of spontaneous calcium intake and physical exercise on the vertebral and femoral bone mineral density of children and adolescents. Journal of Bone and Mineral Research. 10:675-682.

Sandler, R.B., Slemenda, C.W., LaPorte, R.E., Cauley, J.A., Schramm, M.M., Varresi, M.L. and Kriska, A.M. 1985. Postmenopausal bone density and milk consumption in childhood and adolescence. American Journal of Clinical Nutrition. 42:270-274.

Sartoris, D.J. and Resnick, D. 1988. Editorial: new radiographic technique may renew credibility of bone densitometry. Mayo Clinic Proceedings. 63:1147-1150.

Schaafsma, G. 1992. The scientific basis of recommended dietary allowances for calcium. Journal of Internal Medicine. 231:187-194.

Schweiger, U., Laessle, R., Schweiger, M., Hermann, F., Riedel, W. and Pirke, K.M. 1988. Calorie intake, stress, and menstrual function in athletes. Fertility and Sterility. 49:447-450.

Sentipal, J.M., Wardlaw, G.M., Mahan, J. and Matkovic, V. 1991. Influence of calcium intake and growth indexes on vertebral bone mineral density in young females. American Journal of Clinical Nutrition. 54:425-428.

Sinning, W.E. and Little, K.D. 1987. Body composition and menstrual function in athletes. Sports Medicine. 4:34-45.

Siri, W.E. 1961. Body composition from fluid spaces and density: analysis of methods. In: Techniques for Measuring Body Composition. Washington, DC: National Academy of Science and National Research Council.

Slaughter, M.H., Lohman, T.G., Boileau, R.A., Horswill, C.A., Stillman, R.J., Van Loan, M.D. and Bembien, D.A. 1988. Skinfold equations for estimation of body fatness in children and youth. Human Biology. 60:709-723.

- Slemenda, C.W. and Johnston, C.C. 1993. High intensity activities in young women: site specific bone mass effects among female figure skaters. Bone and Mineral. 20:125-132.
- Slemenda, C.W., Miller, J.Z., Hui, S.L., Reister, T.K. and Johnston, C.C. 1991. Role of physical activity in the development of skeletal mass in children. Journal of Bone and Mineral Research. 6:1227-1233.
- Slemenda, C.W., Reister, T.K., Hui, S.L., Miller, J.Z. Christian, J.C. and Johnston, C.C. 1994. Influences on skeletal mineralization in children and adolescents: evidence for varying effects of sexual maturation and physical activity. The Journal of Pediatrics. 125:201-207.
- Southard, R.N., Morris, J.D., Mahan, J.D., Hayes, J.R., Torch, M.A., Sommer, A. and Zipf, W.B. 1991. Bone mass in healthy children: measurement with quantitative DXA. Radiology. 179:735-738.
- Stager, J.M., Robertshaw, D. and Miescher, E. 1984. Delayed menarche in swimmers in relation to age at onset of training and athletic performance. Medicine and Science in Sports and Exercise. 16:550-555.
- Sundgot-Borgen, J. 1996. Eating disorders, energy intake, training volume, and menstrual function in high-level modern rhythmic gymnasts. International Journal of Sport Nutrition. 6:100-109.
- Svendson, O.L., Haarbo, J., Hassager, C. and Christiansen, C. 1993. Accuracy of measurements of body composition by dual-energy x-ray absorptiometry in vivo. American Journal of Clinical Nutrition. 57:605-608.
- Tanner, J.M. 1962. Growth in Adolescence (2nd ed.). Oxford: Blackwell Scientific Publications.
- Teegarden, D., Proulx, W.R., Martin, B.R., Zhao, J., McCabe, G.P., Lyle, R.M., Peacock, M., Slemenda, C., Johnston, C.C. and Weaver, C.M. 1995. Peak bone mass in young women. Journal of Bone and Mineral Research. 10:711-715.
- Theintz, G., Buchs, B., Rizzoli, R., Slosman, D., Clavien, H., Sizonenko, P.C. and Bonjour, J.-PH. 1992. Longitudinal monitoring of bone mass accumulation in healthy adolescents: evidence for a marked reduction after 16 years of age at the levels of lumbar spine and femoral neck in female subjects. Journal of Clinical Endocrinology and Metabolism. 75:1060-1065.

Toss, G. 1992. Effect of calcium intake vs. other lifestyle factors on bone mass. Journal of Internal medicine. 321:181-186.

Vandenbroucke, J.P., van Lar, A. and Valkenburg, H.A. 1982. Synergy between thinness and intensive sports activity in delaying menarche. British Medical Journal. 284:1907-1908.

Wahner, H.W. 1989. Measurements of bone mass and bone density. Endocrinology and Metabolism Clinics of North America. 18(4):995-1005.

Wahner, H.W., Dunn, W.L., Brown, M.L., Morin, R.L. and Riggs, B.L. 1988. Comparison of dual-energy x-ray absorptiometry and dual photon absorptiometry for bone mineral measurements of the lumbar spine. Mayo Clinic Proceedings. 63:1075-1084.

Warren, M. 1980. The effects of exercise in pubertal progression and reproductive function of girls. Journal of Clinical Endocrinology and Metabolism. 51:1150-1157.

Warren, M.P., Brooks-Gunn, J., Hamilton, L.H., Warren, L.F. and Hamilton, W.G. 1986. Scoliosis and fractures in young ballet dancers. The New England Journal of Medicine. 314(21):1348-1353.

Wells, C.L. 1991. Women, Sports and Performance (2nd ed.). Champagne, IL: Human Kinetics Books.

Weststrate, J.A. and Deurenberg, P. 1989. Body composition in children: proposal for a method for calculating body fat percentage from total body density of skinfold-thickness measurements. American Journal of Clinical Nutrition. 50:1104-1115.

APPENDICES

APPENDIX A

Description of the Study and Informed Consent Form

BONE DENSITY IN COMPETITIVE PRE-MENARCHIAL GYMNASTS

Description of the Study

Reasons for the Study

It is important that girls and young women acquire a high level of bone mass by the time they reach maturity, to lessen their risk of developing osteoporosis and sustaining bone fractures later in life. Although there is scientific evidence to indicate that exercise and calcium intake during adolescence are linked to the amount of bone mass a girl accumulates, it is not clear if exercise and calcium are as critical during the pre-adolescent years. In addition, there is a lack of information on bone density in competitive young artistic and rhythmic gymnasts. Through this research, we wish to gain further insight into the issue of skeletal health and the factors which influence it, in artistic and rhythmic gymnasts and non-athletic girls who have yet to experience their first menstrual cycle (i.e., pre-menarchial girls).

Purpose of the Study

This study seeks to relate physical training and calcium intake to bone density in pre-menarchial artistic and rhythmic gymnasts and non-athletic girls, aged 9 to 13 years.

Eligibility Criteria

In order to take part in the study, subjects must meet eligibility criteria as follows: pre-menarchial, Caucasian girl in the early stages of breast development; for artistic and rhythmic gymnasts, affiliation with the novice or junior level of a local Winnipeg artistic or rhythmic gymnastics club and training a minimum of 9 hours per week; for non-athletic girls, involvement in no more than 5 hours per week of structured physical activity (i.e., school physical education classes, school and club teams, and lessons); no chronic use of medications which may influence bone density; no history of extreme dieting or an eating disorder; non-smoker; and no alcohol consumption.

Measurements and Procedures

The study will involve an assessment of each participant's bone mineral density, percent body fat, calcium intake and anthropometry (i.e., height, weight, skinfold thicknesses, girths, bone lengths and bone breadths).

Bone mineral density and percent body fat will be measured by dual energy x-ray absorptiometry (DXA) at the St. Boniface General Hospital. Three DXA scans will be done per subject: one of the lumbar spine, one of the hip region, and one of the total body. DXA is a safe, non-invasive method of determining bone mineral status and body composition. Radiation exposure with DXA is negligible (approximately 1/10th of a standard chest x-ray and 1/100th of a dental radiographic examination).

Each participant will be asked to keep a 3-day food record, for determination of her usual nutrient intake through a computerized analysis of her diet.

Height, weight, skinfold thicknesses, girths, bone lengths and bone breadths will be measured at the Health, Leisure and Human performance Research Institute (Max Bell Centre, University of Manitoba).

All measurements to be carried out in this study involve safe, non-invasive techniques which

will not create any discomfort for the subjects. Since participants will not be subjected to any invasive or potentially harmful procedures, foreseeable risks to the subjects are minimal. Subjects will not be requested to alter their physical activity patterns or diet in any way.

Confidentiality

Information collected in this study will be kept confidential. A subject's identity will not be released without prior written permission from the subject and her parent(s). Data gathered on all participants will be treated in aggregate form (i.e., individual values will not be reported). To protect the identity of subjects, a code number will be assigned to each participant and used when analyzing the data.

Retention of Data for Use in Future Studies

Data will be retained for future use should a follow-up study be conducted. Confidentiality terms (as described above) with respect to use of data from this study will be honoured.

Inconveniences and Anticipated Time Commitment for Subjects and Their Parents

The only inconvenience to subjects and their parents is the time factor involved in participating in the study. Every effort will be made to schedule meetings and testing sessions at a time which best suits each subject and her parents. Subjects and their parents will need to allot time for: completion of screening questionnaires (medical history, physical activity patterns, eating disorder inventory, and dietary intake); completion of the 3-day food record; meetings (as necessary) for clarification of information; travel time to and from testing locations (St. Boniface General Hospital and the Health, Leisure and Human performance Research Institute); and bone mineral density/percent body fat and anthropometric measurements.

Rules for Terminating the Study and for Withdrawing a Subject

Conditions under which we may have to terminate the study or withdraw a subject are as follows: (a) an illness or personal/family crisis which prevents the researcher from completing the study, (b) the subject experiences her first menstrual cycle, (c) the subject develops a medical condition and/or is required to take medication which may affect bone development, interfere with her normal training regimen or require she modify her typical diet, and (d) the subject fails to comply with the rules of the study (e.g., repeatedly missing testing sessions, failure to complete the 3-day food record, etc.).

Subject's Right to Withdraw from the Study

Each subject and her parents reserve the right to deny consent or withdraw from the study at any time, without prejudice.

Benefits for Subjects and Parents

The benefits of participating in the study are as follows: (a) it represents an educational opportunity for subjects, (b) the analysis of food intake will provide each subject with a computerized

nutritional assessment of her diet, at no financial cost to her parents (this is normally a \$45.00 service), (c) each subject will have access to the services of a registered dietitian at no financial cost to her parents, (d) bone density measurements can help to identify subjects who may be at increased risk of injury due to low bone mass, thus allowing for early intervention, and (e) upon completion of the study, each subject and her parents will receive a copy of her individual results, along with general research findings.

*Health, Leisure and Human Performance Research Institute
The University of Manitoba, Winnipeg
Canada R3T 2N2*

CONSENT FORM

I have read the description of the study and understand the measurement procedures involved.

I understand that I may withdraw from the study at any time without prejudice.

All information will be kept confidential.

Date

Participant

Parent/Guardian's Signature

Witness

APPENDIX B

Medical Questionnaire, Physical Activity Questionnaire,
Food intake Questionnaire, and Calcium Intake Questionnaire

Name: _____

Date: _____

Date of Birth: _____

Age: _____

MEDICAL QUESTIONNAIRE

1. In the last 6 months, have you seen a doctor? ____yes ____no

2. If yes, why did you go to the doctor? _____

3. In the last 6 months, have you had any injuries that caused you to go to the doctor? ____yes ____no

4. If yes, what was the nature of the injury? _____

5. In the last 6 months, have you been hospitalized? ____yes ____no

6. If yes, why? _____

7. In the last 6 months, have you been sick with fever? ____yes ____no

8. Are you taking any medications? ____yes ____no

9. If yes, what are they? _____

10. Please indicate what you are taking these medications for.

11. (a) Please indicate if you have any of the following medical conditions:

_____ diabetes

_____ kidney disease

_____ cystic fibrosis

_____ celiac disease

_____ anemia

_____ arthritis

_____ cancer

_____ any other medical condition for which you are being
treated or monitored by a doctor (please specify type
of medical condition) _____

(b) If you have a medical condition, please indicate what age you were
when it was first diagnosed _____

12. Have you had any problems with your bones such as a fracture, a
stress fracture or shin splints? ____yes ____no

13. If any, how many stress fractures have you had? _____

14. If any, how many other fractures have you had? _____

15. Please fill in (if applicable):

Type of fracture	Location of fracture	Age at which fracture occurred
_____	_____	_____
_____	_____	_____
_____	_____	_____

16. Did you have a bone scan to diagnose any of these fractures?

___yes ___no

If yes, when? _____

17. Have you ever been told that you have scoliosis? ___yes ___no

18. If yes, when was it diagnosed? _____

19. What degree of curvature of the spine was diagnosed? _____

20. Have you had your first menstrual period? ___yes ___no

21. Based on the attached diagram, please indicate which stage best describes your level of breast development at the present time (check one).

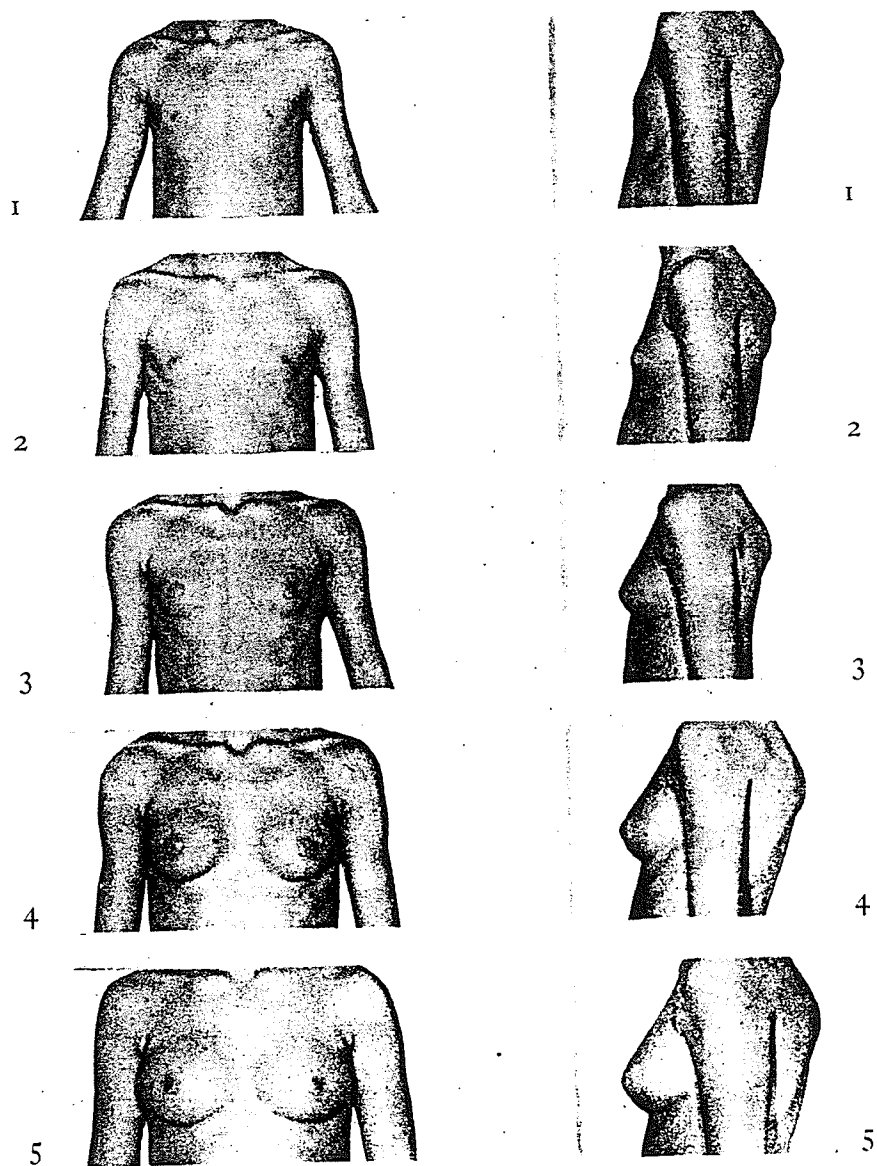
_____ Stage 1

_____ Stage 2

_____ Stage 3

_____ Stage 4

_____ Stage 5



Standards for breast development ratings during adolescence.

Name: _____

Date: _____

Date of Birth: _____

Age: _____

PHYSICAL ACTIVITY QUESTIONNAIREPART A

1. In this question, "physical activity" includes both structured (organized) and unstructured activities.

Examples of structured physical activities are: school physical education classes; school teams; clubs (e.g., swim clubs, track & field clubs, community club teams, etc.); lessons (e.g., swimming, dance, tennis, gymnastics, etc.).

Examples of unstructured physical activities are: playing games which involve physical activity (e.g., tag, skipping, hopscotch, etc.); outings with family/friends (e.g., cycling, skiing, swimming for fun, hiking, etc.).

Please rate your overall level of physical activity for each of the following age categories (starting from ages 5 and 6 years, up to and including your present age). (circle one)

<u>Physical Activity Level</u>	<u>5 & 6 yrs</u>	<u>7 & 8 yrs</u>	<u>9 & 10 yrs</u>	<u>11 & 12 yrs</u>
Highly active (9 or more hours/week)	1	1	1	1
Moderately active (6 - 9 hours/week)	2	2	2	2
Sometimes active (3 - <6 hours/week)	3	3	3	3
Seldom active (0 - <3 hours/week)	4	4	4	4

2. How would you describe the games you played most often, for each of the following age categories? (circle one)

<u>Type of game</u>	<u>5 & 6 yrs</u>	<u>7 & 8 yrs</u>	<u>9 & 10 yrs</u>	<u>11 & 12 yrs</u>
Mostly running, jumping, climbing, throwing games	1	1	1	1
Games requiring some running, jumping, climbing, etc.	2	2	2	2
Games requiring little running, jumping, climbing, etc. (mostly start & stop games)	3	3	3	3
Sedentary games (e.g., board games, drawing, puzzles, etc.)	4	4	4	4

3. How much television did you watch during each of the following age categories? (circle one)

<u>Hours of television</u>	<u>5 & 6 yrs</u>	<u>7 & 8 yrs</u>	<u>9 & 10 yrs</u>	<u>11 & 12 yrs</u>
0 - 1 hour/day	1	1	1	1
1 - 2 hours/day	2	2	2	2
2 - 3 hours/day	3	3	3	3
3 or more hours/day	4	4	4	4

4. How much reading/studying did you do during each of the following age categories? (circle one)

<u>Hours of reading/ Studying</u>	<u>5 & 6 yrs</u>	<u>7 & 8 yrs</u>	<u>9 & 10 yrs</u>	<u>11 & 12 yrs</u>
0 - 1 hour/day	1	1	1	1
1 - 2 hours/day	2	2	2	2
2 - 3 hours/day	3	3	3	3
3 or more hours/day	4	4	4	4

5. For each of the following age categories, please answer question (a), and questions (b) to (e) if applicable.

- | | <u>5 &
6 yrs</u> | <u>7 &
8 yrs</u> | <u>9 &
10 yrs</u> | <u>11 &
12 yrs</u> |
|--|--------------------------|--------------------------|---------------------------|----------------------------|
| (a) Did you participate in organized sports? (yes or no) | | | | |
| (b) If yes, list the sports in which you participated. | | | | |
| (c) If yes, how many days/week did you practice? | | | | |
| (d) If yes, how many weeks/year did you practice? | | | | |
| (e) If yes, approximately how long did each practice last? (hours & minutes) | | | | |

6. For each of the following age categories, please answer question (a), and questions (b) to (e) if applicable.

	<u>5 & 6 yrs</u>	<u>7 & 8 yrs</u>	<u>9 & 10 yrs</u>	<u>11 & 12 yrs</u>
(a)	In addition to organized sports, did you participate in any other form of regular physical activity? (yes or no)			
(b)	If yes, list the kind(s) of physical activity in which you participated.			
(c)	If yes, how many days/ week did you participate in the physical activity?			
(d)	If yes, how many weeks/ year did you participate in the physical activity?			
(e)	If yes, approximately how long did each physical activity session last (hours & minutes)			

7. During which age category were you most active? (circle one)

<u>5 &</u> <u>6 yrs</u>	<u>7 &</u> <u>8 yrs</u>	<u>9 &</u> <u>10 yrs</u>	<u>11 &</u> <u>12 yrs</u>
1	2	3	4

8. How would you rate your level of physical fitness right now? (circle one)

<u>Very High</u>	<u>High</u>	<u>Average</u>	<u>Low</u>	<u>Very Low</u>
1	2	3	4	5

9. How many hours per week do you participate in school physical education classes right now? (check one)

☐ 0 hours/week
☐ More than 0, but <1 hour/week
☐ 1 - <2 hours/week
☐ 2 - <3 hours/week
☐ 3 - <4 hours/week
☐ 4 - <5 hours/week
☐ 5 or more hours/week

10. How many hours per week of structured (organized) physical activity do you participate in right now, not including school physical education classes? (check one)

☐ 0 hours/week
☐ More than 0 but <2 hours/week
☐ 2 - <4 hours/week
☐ 4 - <6 hours/week
☐ 6 - <8 hours/week
☐ 8 - <10 hours/week
☐ 10 - <12 hours/week
☐ 12 or more hours/week

PART B

If you are a gymnast, please complete the following section.

11. How many years have you been training as a gymnast? _____
12. What is the name of the gymnastics club with which you are currently training? _____
13. At which level are you presently training with this club? _____
14. Training schedule:
 - Number of hours per training session _____
 - Number of days per week _____
 - Number of weeks per month _____
 - Number of months per year _____

Season(s) of maximal training (circle)

Winter Spring Summer Fall

15. Competitive schedule: (circle the appropriate season or seasons)

Provincial competition	Winter	Spring	Summer	Fall
---------------------------	--------	--------	--------	------

National competition	Winter	Spring	Summer	Fall
-------------------------	--------	--------	--------	------

Season of maximal competition	Winter	Spring	Summer	Fall
-------------------------------------	--------	--------	--------	------

16. Rest schedule:

Are rest days included in your weekly schedule? ____yes ____no

If yes, how many days do you rest per week (i.e., number of days per week in which you do not engage in gymnastics training)? _____

Are periods of fairly low (or easy) training intensities included in your yearly schedule? ____yes ____no

Name: _____

Date: _____

Date of Birth: _____

Age: _____

FOOD INTAKE QUESTIONNAIRE

1.(a) In the past, have you ever been on a special diet? ____yes ____no

If yes, what age(s) were you when you followed this diet? _____

If yes, please indicate the type of diet you were on (check):

____vegetarian
____hypoallergenic
____weight loss
____other (please specify) _____

(b) Do you presently eat a special diet? ____yes ____no

If yes, please indicate the type of diet you eat (check):

____vegetarian
____hypoallergenic
____weight loss
____other (please specify) _____

2. Do you have any food allergies or food intolerances (i.e., adverse reactions to a specific food or foods)? ____yes ____no

If yes, please specify which food(s) you are allergic to or intolerant of (check):

<input type="checkbox"/> milk	<input type="checkbox"/> cheese
<input type="checkbox"/> citrus fruits/juices	<input type="checkbox"/> tomatoes/tomato products
<input type="checkbox"/> berries	<input type="checkbox"/> corn/corn products
<input type="checkbox"/> wheat/wheat products	<input type="checkbox"/> oats, rye & barley
<input type="checkbox"/> fish	<input type="checkbox"/> shellfish
<input type="checkbox"/> pork	<input type="checkbox"/> nuts
<input type="checkbox"/> peanuts/peanut butter	<input type="checkbox"/> eggs
<input type="checkbox"/> chocolate/cocoa	<input type="checkbox"/> other (please specify food[s])

3. Do you drink pop (diet or regular)? ☐ yes ☐ no

If yes, how many 12 oz. (355 mL) cans of pop do you drink each day?

4. Do you drink coffee? ☐ yes ☐ no

If yes, is it decaffeinated coffee? ☐ yes ☐ no

If you drink coffee, how many cups of coffee do you drink each week?

5. Do you drink tea? ☐ yes ☐ no

If yes, is it decaffeinated or herbal tea? ☐ yes ☐ no

If you drink tea, how many cups of tea do you drink each week?

6. Do you take a multi-vitamin/mineral supplement? ____yes ____no

If yes:

(a) What is the supplement brand and name? _____

(b) How many milligrams (mg) of calcium does it contain? _____

(c) How many international units (IU) of vitamin D does it contain?

(d) How many times a week do you take the supplement? _____

7. Do you take a calcium supplement? ____yes ____no

If yes:

(a) What is the supplement brand and name? _____

(b) How many milligrams (mg) of calcium does it contain? _____

(c) How many international units (IU) of vitamin D does it contain (if any)? _____

(d) How many times a week do you take the supplement? _____

Name: _____

Date: _____

Date of Birth: _____

Age: _____

CALCIUM INTAKE QUESTIONNAIRE*Instructions:

1. Check the calcium-rich foods you ate yesterday.
2. Write the number of servings you ate for each food checked.

<u>Calcium-Rich Foods</u>	<u>Usual Serving Size</u>	<u>No. of Servings Eaten Yesterday</u>
____ Canned sardines with bones	7 medium	_____
____ Macaroni & cheese (home-baked recipe)	1 cup	_____
____ Milkshake (restaurant type)	10 oz.	_____
____ Plain yogurt	3/4 cup	_____
____ Cheese (cheddar, edam, gouda)	1 1/4" cube	_____
____ Dried skim milk powder	1/3 cup	_____
____ Milk (skim, 1%, 2% whole)	1 cup	_____
____ Milk (buttermilk, chocolate)	1 cup	_____
____ Fruit-flavoured yogurt	3/4 cup	_____

*Adapted from the B.C. Dairy Foundation "Calcium Calculator" (1987)

<u>Calcium-Rich Foods</u>	<u>Usual Serving Size</u>	<u>No. of Servings Eaten Yesterday</u>
___ Cheese (all other hard types)	1¼" cube	_____
___ Processed cheese slices	2 slices	_____
___ Canned salmon with bones	3 oz.	_____
___ Soup made with milk	1 cup	_____
___ Pancake/waffle (from mix with milk)	Two - 4" diameter	_____
___ Pudding made with milk	½ cup	_____
___ Cheese pizza	1/8 of 12" diameter	_____
___ Bok choy, kale (cooked)	½ cup	_____
___ Tofu (made with calcium -- read the label)	4 oz.	_____
___ Beans (kidney, garbanzo, lima, navy, soy)	1 cup	_____
___ Ice milk, ice cream	½ cup	_____
___ Chili con carne (made with beans)	1 cup	_____
___ Cottage cheese	½ cup	_____
___ Broccoli (cooked & chopped)	½ cup	_____
___ Whole wheat bread	2 slices	_____
___ Parmesan cheese	1 Tbsp.	_____
___ Orange (whole)	1 medium	_____

APPENDIX C

Eating Disorder Inventory

EDI

Name _____ Date _____

Age _____

Present weight _____ Height _____

Highest past weight _____ (lbs)

How long ago? _____ (months)

How long did you weigh this weight? _____ (months)

What do you consider your ideal weight? _____ (lbs)

Age at which weight problems began (if any) _____

Father's occupation _____ Mother's occupation _____

INSTRUCTIONS

This is a scale which measures a variety of attitudes, feelings and behaviors. Some of the items relate to food and eating. Others ask you about your feelings about yourself. THERE ARE NO RIGHT OR WRONG ANSWERS SO TRY VERY HARD TO BE COMPLETELY HONEST IN YOUR ANSWERS. RESULTS ARE COMPLETELY CONFIDENTIAL. Read each question and fill in the circle under the column which applies best to you. Please answer each question very carefully. Thank you.

	ALWAYS	USUALLY	OFTEN	SOMETIMES	RARELY	NEVER
1. I eat sweets and carbohydrates without feeling nervous.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
2. I think that my stomach is too big.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
4. I eat when I am upset.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
5. I stuff myself with food.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
6. I wish that I could be younger.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
7. I think about dieting.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
8. I get frightened when my feelings are too strong.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
9. I think that my thighs are too large.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
10. I feel ineffective as a person.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
11. I feel extremely guilty after overeating.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
12. I think that my stomach is just the right size.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
13. Only outstanding performance is good enough in my family.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
14. The happiest time in life is when you are a child.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
15. I am open about my feelings.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
16. I am terrified of gaining weight.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
17. I trust others.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
18. I feel alone in the world.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
19. I feel satisfied with the shape of my body.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
20. I feel generally in control of things in my life.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
21. I get confused about what emotion I am feeling.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
22. I would rather be an adult than a child.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
23. I can communicate with others easily.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
24. I wish I were someone else.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
25. I exaggerate or magnify the importance of weight.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
26. I can clearly identify what emotion I am feeling.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
27. I feel inadequate.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
28. I have gone on eating binges where I have felt that I could not stop.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
29. I try very hard to avoid disappointing my parents and teachers.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
30. I have close relationships.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

	ALW	USU	OFT	SOM	RAR	NEV
31. I like the shape of my buttocks.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
32. I am preoccupied with the desire to be thinner.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
33. I don't know what's going on inside me.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
34. I have trouble expressing my emotions to others.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
36. I hate being less than best at things.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
37. I feel secure about myself.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
38. I think about bingeing (over-eating).	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
40. I get confused as to whether or not I am hungry.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
41. I have a low opinion of myself.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
42. I feel that I can achieve my standards.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
43. My parents have expected excellence of me.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
44. I worry that my feelings will get out of control.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
45. I think that my hips are too big.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
46. I eat moderately in front of others and stuff myself when they're gone	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
47. I feel bloated after eating a normal meal.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
48. I feel that people are happiest when they are children.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
49. If I gain a pound, I worry that I will keep gaining.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
50. I feel that I am a worthwhile person.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
51. When I am upset, I don't know if I am sad, frightened, or angry. .	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
52. I feel that I must do things perfectly, or not do them at all.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
53. I have the thought of trying to vomit in order to lose weight.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
54. I need to keep people at a certain distance (feel uncomfortable if someone tries to get too close).	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
55. I think that my thighs are just the right size.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
56. I feel empty inside (emotionally).	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
57. I can talk about personal thoughts or feelings.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
58. The best years of your life are when you become an adult.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
59. I think that my buttocks are too large.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
60. I have feelings that I can't quite identify.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
61. I eat or drink in secrecy.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
62. I think that my hips are just the right size.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
63. I have extremely high goals.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
64. When I am upset, I worry that I will start eating.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

FOR OFFICE USE ONLY

DT	B	SD	I	P	ID	IA	MF

APPENDIX D

Equations Used for Estimating Body Density and Percent Body Fat from Skinfold Thicknesses

EQUATIONS USED FOR ESTIMATING BODY DENSITY AND PERCENT BODY FAT FROM SKINFOLD THICKNESSES

Prediction equation for estimating body density from skinfold thicknesses in females (Jackson et al., 1980):

Sum of 4 skinfolds (Sum4SF) = triceps + suprailiac + abdomen + anterior
mid-thigh

$$\text{Body Density (D}_b\text{)} = 1.0960950 - 0.0006952(\text{Sum4SF}) + \\ 0.0000011(\text{Sum4SF})^2 - 0.0000714(\text{Age})$$

Prediction equation for estimating percent body fat from body density (Siri, 1961):

$$\% \text{ Body Fat (Siri Equation)} = 495/D_b - 450$$

Prediction equations for estimating percent body fat from skinfold thicknesses in females 8-18 years of age (Slaughter et al., 1988):

For triceps and calf skinfolds:

$$\% \text{ Body Fat} = 0.610(\text{triceps} + \text{calf SF}) + 5.1$$

For triceps and subscapular skinfolds*:

$$\% \text{ Body Fat} = 1.33(\text{triceps} + \text{subscapular SF}) - 0.013 (\text{triceps} + \\ \text{subscapular SF})^2 - 2.5$$

*For a sum of triceps and subscapular skinfolds greater than 35 mm, the following equation should be applied:

$$\% \text{ Body Fat} = 0.546 (\text{triceps} + \text{subscapular SF}) + 9.7$$

APPENDIX E

Instructions for Keeping a 3-Day Food Record and Food Record Sheet

HOW TO KEEP A 3-DAY FOOD RECORD

1. Keep a complete record of all the foods you eat and beverages you drink (e.g., soft drinks, diet drinks, coffee, tea, juices, etc.), including snacks. (Don't forget to record candies, chocolate bars, gum, etc.). Please use the sheet you have been provided with to write down your intake.
2. Keep the food record for 3 consecutive days (2 weekdays and 1 weekend day -- i.e., Thursday, Friday and Saturday, OR Sunday, Monday and Tuesday).
3. Please do not alter the way you typically eat just because you are keeping the record. Try to eat as you normally would if you weren't recording your intake.
4. Record the time of day that you eat each meal or snack.
5. Record everything immediately after each meal or snack, so that you don't forget what you've eaten.
6. Estimate the amount of each food/beverage eaten using tablespoons (T or Tbsp), teaspoons (t or tsp), cups (c), ounces (oz), inches ("), millilitres (mL), grams (g) or centimetres (cm). Use Imperial or metric measures -- whichever you feel most comfortable with.
7. For meat, poultry and fish, record cooked boneless portion size, either by weight (g or oz) or by dimension (cm or inches).

e.g. • 4 oz. broiled sirloin steak, trimmed of visible fat, OR
 • 3" x 2" x 1/2" thick serving of broiled sirloin steak, trimmed of visible fat.
8. For cheese, record by weight (g or oz) or by dimension (cm or inches). Indicate brand name and percent milk fat (% M.F.) or butter fat (% B.F.) as indicated on the package label.

e.g. • 1½ oz Lucerne cheddar cheese (33% M.F.), OR
 • one 1¼" cube Lucerne cheddar cheese (33% M.F.).

9. For dairy products (i.e., milk, chocolate milk, buttermilk, yogurt, cheese, cottage cheese, ice cream, ice milk, frozen yogurt, sour cream, cereal/whipping cream), indicate the amount eaten and percent milk fat (% M.F.) or butter fat (% B.F.) as indicated on the label.

e.g. • 175 mL Beatrice diet raspberry yogurt (0.01% M.F.)
 • 1¼ c 1% chocolate milk
 • 125 mL 2% cottage cheese
 • ½ c cereal cream (10% B.F.)

10. Be as detailed as possible in describing foods and beverages (if in doubt, it's better to be overly specific than too vague). Include food labels, brand names and recipes (for home-made items).

e.g. • 1 c Post Fruit 'n Fibre blueberry almond cereal with ½ c 2% milk
 • 2 Dad's chocolate chip cookies
 • 1 piece grape Hubba Bubba bubble gum
 • 175 mL Campbell's chunky mediterranean vegetable soup
 • 1 complete Lean Cuisine oriental beef, vegetables and rice frozen dinner
 • 1 serving of home-made tuna noodle casserole*
 *Note: include the recipe and indicate how much 1 serving is (e.g., ¼ of the recipe).

11. Be sure to include how the food was prepared (e.g., baked, broiled, boiled, roasted, fried, deep-fried, steamed, microwaved, barbecued, etc.).

12. Record only the amount you ate or drank, not the amount put on your plate or in your glass (unless you consumed all of it).

e.g. • If you had ½ c steamed broccoli on your dinner plate but only ate half of it, then record it as ¼ c steamed broccoli
 • If you drank ¾ of a 12 oz (355 mL) can of Coke, then record it as 9 oz (or 266 mL) of Coke

13. Remember to record any condiments that were added to food, and the amounts in which they were added.

e.g. • Butter or margarine
 • Mayonnaise (light or regular)

- Salad dressing (specify brand name and whether fat-free, diet, light or regular -- e.g., Kraft fat-free ranch dressing)
- Sour cream (specify % B.F.), gravy (home-made or commercial) and sauces (home-made or commercial)
- Ketchup (light or regular), mustard, relish
- Croutons, pickles, bacon bits, grated parmesan cheese
- Sugar, honey
- Jam or jelly (diet, sugar-free or regular)
- Syrup (diet, light or regular)

14. For whole pieces of fresh fruit and vegetables, try to approximate the size eaten.

- e.g.
- 1 large Granny Smith apple
 - 1 medium baked potato (with skin)
 - ½ small banana
 - 1 large carrot, raw

15. Dining out:

Dining out may require some "eye-balling" of portion sizes in order to estimate how much you ate (serving personnel should be able to tell you the actual portion sizes on your plate or in your glass if you ask). Specify the name of the restaurant, what you ordered (please be as detailed as possible in describing the food/beverage) and how much of it you ate.

- e.g. McDonald's
- 1 Big Mac
 - 1 small serving French fries
 - 1 packet of ketchup (approx. 1 Tbsp)
 - 1 garden salad (approx. 1 cup)
 - 1 container of thousand island dressing (approx. 2 Tbsp)
 - 1 small diet Coke (8 oz)

Pizza Hut

- ¼ of 13" (medium) pepperoni and mushroom thick crust pizza with double cheese
- 1 large 7-Up (16 oz)

The Olive Garden Restaurant

- approx. 1 c of spinach fettucine with alfredo sauce
- 1 Tbsp parmesan cheese
- 1 slice garlic toast (½" thick)
- 8 oz 2% milk
- 1 small Caesar salad with croutons (approx. 1 cup)

The Keg Restaurant

- Salad bar:
 - 1 c iceberg lettuce
 - ½ c broccoli flowerettes
 - ½ c baby carrots
 - 4 cherry tomatoes
 - ½ c chick peas
 - ¼ c creamed cottage cheese
 - 4 Tbsp Italian dressing
 - 1 Tbsp bacon bits
 - 1 slice French bread (1" thick)

Grant Park Cinema

- 1 small popcorn with butter topping
- 1 medium Coke (16 oz)
- 4 Twizzler licorice sticks

16. Vitamin and Mineral Supplements

Record all vitamin and/or mineral supplements taken, and specify: (a) the brand name and (b) the dosage taken.

e.g. • Centrum Forte multivitamin/mineral with beta-carotene: 1 tablet taken

In the case of supplements containing single vitamins or minerals, specify: (a) the brand name, (b) the quantity of the vitamin or mineral contained in the supplement and (c) the dosage taken.

e.g. • No-name brand chewable vitamin C (250 mg/tablet): 1 tablet taken
 • Caltrate calcium supplement with vitamin D (600 mg/tablet): 1 tablet taken

Name: _____

Page ____ of ____

Date: _____

FOOD RECORD

Did you take any vitamin and/or mineral supplements today? ____yes ____no
If yes, specify the brand name and dosage taken for each supplement (e.g.,
One-A-Day multivitamin with iron: 1 tablet).

<u>Meal/Snack</u>	<u>Description of Food/Beverage</u>	<u>Amount</u>
-------------------	-------------------------------------	---------------

Time:

APPENDIX F

Participant Information Sheet

PARTICIPANT INFORMATION SHEET

Participant's name: _____

Date of birth: _____

Age: _____

Participant's address: _____
(Street)

(City)

(Postal Code)

Participant's home phone no.: _____

PARENT/GUARDIAN INFORMATION

Parent/guardian name: _____

Address: _____
(Street)

(City)

(Postal Code)

Home phone no.: _____

Work phone no.: _____

Parent/guardian name: _____

Address: _____
(Street)

(City)

(Postal Code)

Home phone no.: _____

Work phone no.: _____

APPENDIX G

Data Collection Sheet for Anthropometric Measurements

Participant's Name: _____

Date of Birth: _____

Age: _____

Name of Tester: _____

Date: _____

DATA COLLECTION SHEET
ANTHROPOMETRIC MEASUREMENTS

Height (cm): _____

Weight (kg): _____

Skinfold Thicknesses (mm) (right side)

SITE	TRIAL 1	TRIAL 2	TRIAL 3	MEAN
Triceps				
Biceps				
Sub-scapular				
Suprailiac				
Abdomen (Left)				
Anterior mid-thigh				
Medial calf				

Sum of 7 skinfold thicknesses (mm): _____

DATA COLLECTION SHEET
ANTHROPOMETRIC MEASUREMENTS

Girths (cm) (right side)

SITE	TRIAL 1	TRIAL 2	TRIAL 3	MEAN
Relaxed-arm				
Flexed-and-tensed arm				
Forearm				
Wrist				
Chest				
Waist				
Gluteal (hip)				
Thigh				
Calf				
Ankle				

Bone Breadths (cm) (right side)

SITE	TRIAL 1	TRIAL 2	TRIAL 3	MEAN
Humerus width (elbow 90 degrees)				
Wrist				
Femur width (knee 90 degrees)				
Ankle				

Participant's Name: _____

Date of Birth: _____

Age: _____

Name of Tester: _____

Date: _____

DATA COLLECTION SHEET
ANTHROPOMETRIC MEASUREMENTS

Lengths (cm) (right side)

PROJECTED LENGTH	TRIAL 1	TRIAL 2	TRIAL 3	MEAN
Upper arm length				
Forearm length				
Hand length				
Thigh length				
Tibial length				

APPENDIX H

List of Abbreviations

LIST OF ABBREVIATIONS

BMC	Bone mineral content
BMD	Bone mineral density
DXA	Dual energy x-ray absorptiometry
DPA	Dual photon absorptiometry
DPX	DXA system manufactured by Lunar Radiation Corp.
PBM	Peak bone mass
QCT	Quantitative computed tomography
QDR	Quantitative digital radiography (DXA system manufactured by Hologic, Inc.)
SPA	Single photon absorptiometry