ABSTRACT

SMITH, D.M. The effects of exercise on bone mineral density in postmenopausal women: A meta-analysis. MS in Adult Fitness/Cardiac Rehabilitation, December 1999, 68pp. (N. Butts)

A meta-analysis was done to determine what effect exercise has on the bone mineral density (BMD) of the lumbar spine, proximal femur, and distal radius in postmenopausal women, utilizing data from 18 studies. Treatment effects (TE) were calculated for each of the skeletal sites, aerobic and strength training, as well as methods of BMD measurement (dual energy x-ray absorptiometry and dual photon absorptiometry). The resulting TEs found exercise, in general, to significantly increase ($p < 0.05$) the BMD of the lumbar spine (0.73%), and the proximal femur (0.35%); however, the distal radius showed a decrease (-0.91%) in BMD. When looking for the effect of aerobic versus strength training, significant ($p < 0.05$) increases in BMD were found in the spine and femur. Since positive changes were not seen in all sites, the benefits of exercise may be specific to the skeletal sites which the working muscles are attached. When the % change for exercise and control groups were analyzed separately, the exercise group increased 2.31% at the lumbar spine and 0.61% at the proximal femur. However, the exercise group decreased -2.21% at the radius. The control group decreased at the lumbar spine -1.09%, and at the femur -1.11%, however, increased at the radius 0.36%. The BMD % changes were significantly larger for the spine and femur, therefore, vigorous exercise along with resistance training may help slow the decrease in BMD as one ages.

THE EFFECTS OF EXERCISE ON BONE MINERAL DENSITY IN POSTMENOPAUSAL WOMEN: A META-ANALYSIS

A MANUSCRIPT STYLE THESIS PRESENTED

TO

THE GRADUATE FACULTY

UNIVERSITY OF WISCONSIN-LA CROSSE

IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE

MASTER OF SCIENCE DEGREE

BY

DANA M. SMITH

DECEMBER 1999

COLLEGE OF HEALTH, PHYSICAL EDUCATION, AND RECREATION

UNIVERSITY OF WISCONSIN-LA CROSSE

THESIS FINAL ORAL DEFENSE FORM

Candidate: Dana M. Smith

We recommend acceptance of this thesis in partial fulfillment of this candidate's requirements for the degree:

Master of Science - Adult Fitness and Cardiac Rehabilitation

The candidate has successfully completed the thesis final oral defense.

Thesis Committee Chairperson Signature	Date 4-13-99
Thesis Committee Member Signature	Date 4-12-99
Thesis Committee Member Signature	Date 4/12/99

This thesis is approved by the College of Health, Physical Education, and Recreation.

Associate Dean, College of Health, Physical Education, and Recreation	Date 4-27-99
Director of University Graduate Studies	Date 4-27-99

ACKNOWLEDGEMENTS

I would like to dedicate this study to my Mom, Dad, Danielle, and Jonah. Without them, I would not have had the strength and perseverance to attain all of my goals. I would also like to thank Dr. Elfessi for his help with the statistics, Dr. George Kelley for his guidance and knowledge of the meta-analytic process, Dr. John Zappala for proofreading my thesis, and my committee for their time and patience.

TABLE OF CONTENTS

PAGE

ACKNOWLEDGEMENTS iii

LIST OF TABLES v

LIST OF APPENDICES vi

INTRODUCTION 1

METHODS 2

Data Sources 2

Study Selection 3

Data Synthesis 5

Subgroup Analysis 6

Other Variables 6

RESULTS AND DISCUSSION 7

Study Characteristics 7

Physical Characteristics of the Subjects 7

Main Effects 7

Subgroup Analysis Effects 11

CONCLUSION 15

REFERENCES 16

APPENDICES 20

LIST OF TABLES

TABLE PAGE

1. Subjects' physical characteristics during pretest (mean and standard deviation)..... 8

2. Correlations between subjects' physical characteristics from the exercise and control groups of included studies at baseline..... 9

3. Changes observed in the subjects' BMDs following the completion of an exercise intervention..... 9

4. Subgroup analysis comparing BMD changes in response to training (aerobic vs. strength) at the lumbar spine and proximal femur..... 11

5. Lumbar spine BMD measurements (gm/cm^2) for included studies (mean and standard deviation)..... 27

6. Proximal femur BMD measurements (gm/cm^2) for included studies (mean and standard deviation)..... 30

7. Distal radius BMD measurements (gm/cm^2) for included studies (mean and standard deviation)..... 33

8. Subject's physical characteristics at baseline measurement (mean and standard deviation)..... 35

9. Descrpitions of the exercise intervention used by each study..... 40

LIST OF APPENDICES

APPENDIX PAGE

A. Sources Not Meeting Inclusion Criteria..... 20

B. Pre and Post BMD Data..... 26

C. Physical Characteristics of Subjects..... 34

D. Descriptions of the Interventions..... 39

E. Formulas..... 45

F. Review of Related Literature..... 48

INTRODUCTION

The onset of menopause introduces many new changes to a woman's body which could lead to a decrease in skeletal bone mineral density (BMD). Decreases in the density of bone have been found with the absence of estrogen, exercise, or other dietary factors such as calcium and vitamin D which put postmenopausal women at risk for bone fractures, breaks, and other bone diseases such as osteoporosis (Salamone et al.,1996).

Osteoporosis, or porous bones, is "a disease characterized by low bone mass and microarchitectural deterioration of bone tissue leading to enhanced bone fragility and a consequent increase in fracture risk " (Drinkwater, 1994, p. 197). In women, peak bone mass usually occurs between the ages of 20-25 and deterioration of bone mass is generally seen thereafter (Anderson & Metz, 1993). The majority of fractures reported in women occur late in life as a result of low BMD. Therefore, the purpose of this meta-analysis was to determine if exercise maintained and/or increased BMD of postmenopausal women.

While the major focus of this report centered on the lumbar spine, the proximal femur and distal radius were also evaluated to determine if site specific responses occurred due to exercise. According to Swezey (1996), aerobic exercises such as walking and jogging have a modest benefit in the prevention and treatment of osteoporosis, however, site-specific training, which involves muscles attached to the bones, appears to have a more consistent effect on bone mineralization and muscular strength.

METHODS

Data Sources

The first step for this meta-analysis involved a literature review of all articles which included studies, and/or information pertaining to, the effects of physical activity on bone density, in postmenopausal women. The review was limited to published articles from 1965 to present due to the limits of the MEDLINE database at the Murphy Library on the University of Wisconsin-La Crosse campus. In order to focus the search, key words included "postmenopausal," "women," "bone density," physical activity," "resistance training," "osteoporosis," "dual photon absorptiometry," "dual energy x-ray absorptiometry," and "exercise."

The MEDLINE search displayed a list of published articles. The abstracts were read and then obtained in hard copy to determine if they could be included in this meta-analysis as a study or could be used as a reference. All abstracts or summaries which were read through MEDLINE are recorded in Appendix A. Periodicals were obtained at the Murphy Library on the campus of the University of Wisconsin-La Crosse, at Gundersen Lutheran Hospital, La Crosse, WI, or through interlibrary loan.

As articles were collected and read, the data were extracted and put into several spreadsheets. Appendix B contains BMD measurement device and mean pre and post BMD measurements for each skeletal site. Appendix C contains data such as the length of each study, number of subjects for each group, subjects' ages, years since the onset of menopause, weight, and body mass index (BMI). Descriptions of the interventions used, year of study, as well as frequency and duration of the interventions are recorded in Appendix D. A

particular study may not have been included in this review due to the age of a study's subjects, a medical history of osteoporosis, use of hormone replacement therapy, or measurement device used. A study was also excluded if it did not have a control group, if the baseline BMDs were not reported, or if the method of regulating exercise by the treatment group was entirely self-reported rather than supervised by a staff member.

Study Selection

For a study to be included in this meta-analysis, the following criteria had to be met: (1) the subjects had to be postmenopausal women not on hormone replacement therapy (HRT); (2) the intervention (independent variable) had to be exercise of any form, such as aerobic or strength training; (3) the length of the study was a minimum of 6 months; (4) the method used to measure BMD had to be either dual energy x-ray absorptiometry (DEXA) or dual photon absorptiometry (DPA); (5) the pre- and post-BMD measurements along with the standard deviation of either the lumbar spine, proximal femur, or distal radius had to be included; and, (6) a control group had to be included. The studies meeting the above criteria are marked with an asterisk in the reference section.

The study was limited to postmenopausal women in order to determine what effect exercise plays in maintaining BMD without the presence of estrogen. All subjects were a minimum of 12 months menopausal, termed "healthy" by the authors, and were not on hormone replacement therapy or other medications which may have affected bone mineralization. It was assumed that prior to their inclusion in the research study, the subjects were not participating in a regular exercise program.

Since the purpose of this study was to evaluate the changes in BMD independent of the type of exercise performed, all forms of exercise were included. Several authors (Bravo et al., 1996; Etherington et al., 1996; Krall & Dawson-Hughes, 1994; Revel, Mayoux-Benhamou, Rabourdin, Bagheri, & Roux, 1993) have demonstrated the effectiveness of exercise in preserving BMD by using interventions ranging from walking or jogging to psoas strengthening or resistance training. Bone mineral density could potentially be affected by any activity, aerobic or resistance training, which produces a stress on the bones.

Since bone turnover rates are normally 3 to 4 months, the length of the studies was also a consideration. Only studies with a minimum length of 6 months were included in order to provide sufficient time for changes in BMD to be manifested. However, the maximum length of each study was the duration of the exercise intervention.

Due to inconsistent results between different BMD measurement devices, the DEXA and DPA were chosen since they appear to provide consistent, reliable, and valid results (Goodwin, 1987). The DEXA methods which are available can measure several different sites, such as the lumbar spine, femur, forearm, heel, and the whole body, by using a X-Y pattern of light beams to determine BMD ("Bone density," 1997). Since the DEXA provides short scanning times, high resolution, and precision ranging from 1% at the lumbar spine to 3% at the femur, it is used quite frequently and is cost effective. According to Grove and Londeree (1992), DPA also uses two energy sources to perform BMD measurements of several skeletal sites, and the precision error is less than 3%. Instruments which used dual energy sources offer greater

accuracy and can compensate for the presence of fat (Goodwin, 1987). According to Wahner, Dunn, Brown, Morin, and Riggs (1988), bone mineral content was found to be accurate in both DEXA and DPA.

Three sites, the lumbar spine, proximal femur, and distal radius, were evaluated. However, emphasis was placed on the lumbar spine since all of the studies included these BMD measurements but not necessarily the latter two. Each study had to include pre- and post-BMD measurements (mean and standard deviations) in order to be used in this project. The BMD of two studies (Kohrt, Ehsani, & Birge, 1997; Kohrt, Snead, Slatopolsky, & Birge, 1995) was interpreted from a graph, while the remaining studies listed the BMD values either in a chart or in the text.

In order to calculate the effectiveness of exercise on BMD, a control group was necessary to provide a means of comparison. Although several authors were listed in more than one study, careful attention was given to ensure that the subjects were not duplicated between studies.

Data Synthesis

The main unit of evaluation for this meta-analysis was the determination of treatment effect (TE). TE is a measure of change in BMD comparing the exercise and control groups for each study. First, the percent (%) change for both the exercise group and the control group were found using the following equation:

$$\% \text{ change in BMD} = [(\text{post} - \text{pre}) / \text{pre}] \times 100$$

Then, to determine TE, the % change of the control group was subtracted from the % change of the exercise group. In order to allow the study with the largest subject number to have the greatest TE, all of the TEs were then weighted by

the inverse of the variance with equations by Hedges and Olkin (1985) in Appendix E. With the remaining equations in Appendix E, a group TE, its variance, and confidence intervals of 95% were calculated.

The test for homogeneity among TEs was found by using the *Q* statistic, formula four in Appendix E, which is an approximate Chi square distribution with the degrees of freedom equal to the number of studies minus one. Therefore, a statistically significant *Q* value represents heterogeneity among all TEs.

Subgroup Analysis

Subgroup analysis of treatment effect changes was completed using the same method as above and by dividing the data into the following categories: (1) mode of training (aerobic vs. strength training); (2) site of BMD assessment, (lumbar spine, proximal femur, and distal radius); and, (3) method of BMD assessment (DEXA vs. DPA). A separate analysis comparing the % change of the exercise versus control group was also calculated for each of the skeletal sites.

Other Variables

Correlations between the exercise and control group's baseline BMD and physical characteristics such as age, onset of menopause, body mass index (BMI), and weight were calculated. Correlations were also calculated between the TE changes in BMD and all variables included in this study. All other comparisons (i.e., age, weight, BMI, subjects number, and initial BMD assessment) between the exercise and control groups were calculated between by using independent *t* tests (Microsoft Excel, 1995).

RESULTS AND DISCUSSION

Study Characteristics

A total of 18 studies met the criteria for this meta-analysis with 10 using DEXA and the remaining 8 using DPA as the method of BMD assessment. Each study contained exercise and control groups with exercise being the main intervention. All studies reported BMD measurements for the lumbar spine, 15 studies reported the proximal femur measurements, and 6 studies reported distal radius measurements. There were a total of 320 subjects in the exercise groups and 325 in the control groups.

A description of the exercise protocol used in each study can be found in Appendix D. All of the interventions were at least six months long and met three to four times per week for an average of 51.3 minutes per session.

Physical Characteristics of the Subjects

The physical characteristics for the exercise and control groups are presented in Table 1, with individual data for each of the studies in Appendix B. No significant ($p < 0.05$) differences in baseline measurements were found between the groups for any variable, or between the TE changes and the physical characteristic variables. As seen in Table 2, the variables for the control and exercise groups were significantly correlated with each other. Overall, subjects in both groups were not significantly different from each other at baseline.

Main Effects

Treatment effect changes, along with established confidence intervals, and Q values for the lumbar spine, proximal femur, and distal radius, can be seen in Table 3.

Table 1. Subjects' physical characteristics during pretest (mean and standard deviation)

Variable	Exercise group	Control group
Age (yrs)	61.9 1.23	62.6 1.37
Menopause (yrs)	9.90 1.59	10.0 1.56
BMI (kg/m^2)	25.6 0.45	25.5 0.45
Weight (kg)	67.5 1.28	66.6 1.30
BMD lumbar (gm/cm^2)	1.01484 0.03436	0.98106 0.03507
BMD femur (gm/cm^2)	0.76886 0.02617	0.75227 0.02172
BMD radial (gm/cm^2)	0.57633 0.10214	0.54250 0.09388

Positive changes in BMD, and confidence intervals which were significantly different from zero, were found in the lumbar spine and proximal femur. A positive TE represents increases in the exercise group's BMD compared to the control group. Since negative and positive TEs were found, the effects of exercise may be limited to the skeletal site which is attached to the working

Table 2. Correlations between subjects' physical characteristics from the exercise and control groups of included studies at baseline

Variable	Correlation
Age (yrs)	0.94
Onset of menopause (yrs)	0.85
BMI (kg/m^2)	0.86
weight (kg)	0.76
BMD lumbar spine (gm/cm^2)	0.94
BMD proximal femur (gm/cm^2)	0.95
BMD distal radius (gm/cm^2)	1.00

Table 3. Changes observed in the subjects' BMDs following the completion of an exercise intervention

Site	Treatment Effect (% change)	Confidence Interval (95%)	*Q*
Lumbar spine	0.7341	0.5298 - 0.9389*	539.45
Proximal femur	0.3522	0.1594 - 0.5451*	338.94
Distal radius	-0.9104	-1.3168 - -1.5041*	131.31

* 95% confidence interval significantly different from zero.

muscles. In other words, BMD changes may be a result of site specific responses to exercise.

Swezey (1996) stated that "site specific resistive exercises appear to have a more consistent effect on bone mineralization and muscle strength" (p. 2,812). The spine and femur are two major weight-bearing skeletal sites, and are under constant resistance from the body's own weight. The radius is not always involved in executing many forms of exercise, such as walking, jogging, or other activities which focus on the cardiovascular system, therefore, may only increase in density when the forearm muscles are included in other activities.

When the exercise and control groups were analyzed separately by the % changes rather than TE, which combines the exercise and control groups, the exercise group's BMD increased in the lumbar spine (2.31%), and the proximal femur (0.61%), but decreased at the distal radius (- 2.21%). The control group's BMD decreased at the lumbar spine (- 1.09%), and the proximal femur (-1.11%), however, increased at the distal radius (0.36%). Significant increases in BMD were seen in the exercise groups for both the lumbar spine ($p = 0.01$), and proximal femur ($p = 0.02$), while no significant difference was found between groups for the distal radius.

In contrast to the increase in BMD for the proximal femur and lumbar spine, the distal radius demonstrated a decrease in BMD. This finding was somewhat surprising in that the exercise group actually decreased more than the control group. Upon further review of the specific studies which included the radial measurements, three of the six studies were from the same laboratory and had extremely low radial BMD measurements compared to the other three. So although statistically exercise apears to decrease the BMD of the distal

radius, this finding may be due to several factors: (1) few studies provided radial BMD measurements; (2) three of the six studies were from the same laboratory; and, (3) the radial BMD measurements had larger variances as compared to the spine and femur.

Subgroup Analysis Effects

As seen in Table 4, when using subgroup analysis for the lumbar spine and proximal femur, both the strength training and aerobic groups showed an increase in BMD. There was no significant difference between the mode of exercise which may suggest that if exercise is going to be used as a tool for the

Table 4. Subgroup analysis comparing BMD changes in response to training (aerobic vs. strength) at the lumbar spine and proximal femur

Group	Treatment Effect (% change)	Confidence Interval (95%)	*Q*
Lumbar spine			
aerobic exercise	0.7718	0.6287 - 0.9148*	390.28
strength training	1.3273	0.9462 - 1.7085*	120.31
Proximal femur			
aerobic exercise	0.1801	-0.0446 - 0.4048	202.75
strength training	0.8329	0.7992 - 0.8696*	120.20

* 95% confidence interval significantly different from zero.

preservation and/or maintenance of BMD after menopause, the perfect exercise prescription would include both aerobic and weight training. Not enough data were reported for the distal radius to determine differences in BMD due to strength training or aerobic exercises, therefore, future studies may consider assessing BMD at this site in order to provide a more complete picture of BMD changes as one ages. In the meantime, incorporating an upper body resistance training program with forearm exercises may guard against low BMD at the wrist.

Swezey (1996) attributes much of the positive changes in BMD to resistance or strength training. She notes that although walking provides several realms of improvement physically and psychologically, it does not provide constant stimulation to the hip and spine areas to the degree of jogging or resistance training. The results of a cross-sectional study by Layne and Nelson (1999) found female athletes to have positive changes in BMD, as well as significantly greater BMD values in weight lifters compared to other exercisers. Assuming this holds true for women without the presence of estrogen, perhaps all females, regardless of age, should incorporate a resistance training program into their exercise routine.

Positive TEs resulted when comparing BMD assessment of the lumbar spine with the DEXA (0.68%) and DPA (0.87%). Limiting the types of BMD assessment was important while making comparisons between different studies due to the inconsistencies found between machines. Although methods for determining BMD have been available for many years, the recent and future development of techniques has been focused on perfecting precision and accuracy (Goodwin, 1987).

Although the *Q* statistic yielded heterogeneity and p values < 0.05 between studies for all analyses, outliers were not figured since all subjects in both groups were not significantly different from each other initially for all variables (i.e., age, onset of menopause, weight, or BMI). Some studies reported subject mortality within their design, but only evaluated the data from those who were compliant for the entire study. This allows for greater positive effects and perhaps publication biasness. Future studies may consider entering a zero in place of the missing data in order to better represent true figures, since health professionals generally want to know how a variable affects everyone. Furthermore, those who are compliant in a study may represent the part of the population which are proactive in preserving their health, where as those who were not compliant, may represent the sedentary population who, in general, are at increased risk for many diseases due to their inactivity. Although more negative results may arise, the treatment effect will be based on effectiveness of the treatment instead of efficacy of the treatment, or what is expected to occur.

A meta-analysis by Kelley (1998) which used studies from 1987 to 1995, also found positive TEs for the lumbar spine and proximal femur, however, BMD decreased for both sites when analyzed by % change. He also found that BMD increased for both aerobic and strength training exercises, and that the effect of exercise may be specific to the skeletal site which the working muscles are attached. Another meta-analysis by Berard, Bravo, and Gauthier (1997) found that when analyzing studies from 1966 to 1996, "physical activity was not found to have a beneficial effect on bone loss after menopause" (p. 336), however, they did find that after 1996, physical activity was effective in preventing bone loss at the lumbar spine.

Increases in the BMD of the lumbar spine and proximal femur after taking part in an exercise program were found to be significant in this meta-analysis. This may emphasize the fact that vigorous aerobic exercise along with resistance training may help slow the decrease of bone density as one ages (Swezey, 1996). The American College of Sports Medicine position stand on osteoporosis and exercise (1995) makes the following statements:

> 1. Weight-bearing physical activity is essential for the normal development and maintenance of a healthy skeleton. Activities that focus on increasing muscle strength may also be beneficial, particularly for nonweight-bearing bones. 2. Sedentary women may increase bone mass slightly by becoming more active but the primary benefit of the increased activity may be in avoiding the further loss of bone that occurs with inactivity. 3. Exercise cannot be recommended as a substitute for hormone replacement therapy at the time of menopause. 4. The optimal program for older women would include activities that improve strength, flexibility, and coordination that may indirectly, but effectively, decrease the incidence of osteoporotic fractures by lessening the likelihood of falling. (p. i)

One of the largest risks for postmenopausal women with low BMD is falling and fracturing a bone. Due to the fact that women have a lower total skeletal mass at maturity, have larger BMD losses, have a longer life expectancy, and fall more frequently than men for many undetermined reasons, the lifetime risk factor for an osteoporotic fracture in women over the age of 50 is three times higher than in men (Kanis, 1996). If a postmenopausal woman can continue daily living activities while also including additional aerobic and weight training activities, she may increase or maintain her strength, balance, coordination, and also decrease her risk of fracture.

CONCLUSION

In this meta-analysis, BMD was found to significantly increase in the lumbar spine and proximal femur with exercise. It appears beneficial to live an active lifestyle in place of a sedentary one in order to prevent and/or decrease the development of low bone mass. Future research involving the BMD of the radius could lead to more complete data, and therefore, conclusions as to whether or not exercise results in overall improvements of BMD, or just changes specific to the bone attached to the working muscles.

REFERENCES

American College of Sports Medicine. (1995). Position stand on osteoporosis and exercise. Medicine and Science in Sports and Exercise, 27, i-vii.

Anderson, J.J.B., & Metz, J.A. (1993). Contributions of dietary calcium and physical activity to prevention of osteoporosis in females. Journal of the American College of Nutrition, 12, 378-383.

*Bassey, E.J., & Ramsdale, S.J. (1995). Weight-bearing exercise and ground reaction forces: A 12 month randomized controlled trial of effects on bone mineral density in healthy postmenopausal women. Bone, 16, 469-476.

Berard, A., Bravo, G., & Gauthier, P. (1997). Meta-analysis of the effectiveness of physical activity for the prevention of bone loss in postmenopausal women. Osteoporosis International, 7, 331-337.

*Bloomfield, S.A., Williams, N.I., Lamb, D.R., & Jackson, R.D. (1993). Non-weightbearing exercise may increase lumbar spine bone mineral density in healthy postmenopausal women. American Journal of Physical Medicine and Rehabilitation, 72, 204-209

Bone density measurement - a systematic review. A report from SBU, the Swedish Council on Technology Assessment in health care. (1997). Journal of Internal Medicine, 739, (Suppl.), 1-60.

Bravo, G., Gauthier, P., Roy, P.M., Payette, H., Gaulin, P., Harvey, M., Peloquin, L., & Dubois, M.F. (1996). Impact of a 12 month exercise program on the physical and psychological health of osteopenic women. Journal of the American Geriatric Society, 44, 756-762.

*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.

Drinkwater, B.L. (1994). Does physical activity play a role in preventing osteoporosis? Research Quarterly for Exercise and Sport, 65, 197-206.

*Ebrahim, S., Thompson, P.W., Baskaran, R., & Evans, K. (1997). Randomized placebo-controlled trial of brisk walking in the prevention of postmenopausal osteoporosis. Age and Ageing, 26, 253-260.

Etherington, J., Harris, P.A., Nandra, D., Hart, D.J., Wolman, R.L., Doyle, D.V., & Spector, T.D. (1996). The effect of weight-bearing exercise on bone mineral density: A study of female ex-elite athletes and the general population. Journal of Bone Mineral Research, 11, 1333-1338.

Goodwin, P.N. (1987). Methodologies for the measurement of bone density and their precision and accuracy [Abstract]. Seminars in Nuclear Medicine, 4, 293.

*Grove, K.A., & Londeree, B.R. (1992). Bone density in postmenopausal women: High impact vs. low impact exercise. Medicine and Science in Sports and Exercise, 24, 1190-1161.

*Hartard, M., Haber, P., Ilieva, D., Preisinger, E., Seidl, G., & Huber, J. Systematic strength training as a model of therapeutic intervention. A controlled trial in postmenopausal women with osteopenia. American Journal of Physical Medicine and Rehabilitation, 75, 21-29.

Hedges, L.V., & Olkin, I. (1985). Statistical methods for meta-analysis. Orlando: Academic Press.

Kanis, J.A. (1996). Estrogens, the menopause, and osteoporosis. Bone, 19, (Suppl. 5), 185-190.

Kelley, G. (1998). Exercise and regional bone mineral density in postmenopausal women: A meta-analytic review of randomized trials. American Journal of Physical Medicine and Rehabilitation, 77, 76-87.

*Kohrt, W.M., Ehsani, A.A., & Birge, S.J., Jr. (1997). Effects of exercise involving predominantly either joint-reaction or ground-reaction forces on bone mineral density in older women. Journal of Bone and Mineral Research, 12, 1253-1261.

*Kohrt, W.M., Snead, D.B., Slatopolsky, E., & Birge, S.J., Jr. (1995). Additive effects of weight-bearing exercise and estrogen on bone mineral density in older women. Journal of Bone and Mineral Research, 10, 1303-1311.

Krall, E.A., & Dawson-Hughes, B. (1994). Walking is related to bone density and rates of bone loss. The American Journal of Medicine, 96, 20-25.

Layne, J.E., & Nelson, M.E. (1999). The effects of progressive resistance training on bone density: A review. Journal of Medicine and Science in Sports and Exercise, 31, 25-30.

*Lord, S.R., Ward, J.A., Williams, P., & Zivanovic, E. (1996). The effects of a community exercise program on fracture risk factors in older women. Osteoporosis International, 6, 361-367.

Microsoft Excel for Windows 7.0 [Computer Software]. (1995). Santa Rosa, CA: Microsoft Corporation.

*Nelson, M.E., Fiatarone, M.A., Morganti, C.M., Trice, I., Greenberg, R.A., & Evans, W.J. (1994). Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures: A randomized controlled trial. Journal of the American Medical Association, 272, 1909-1914.

*Nelson, M.E., Fisher, E.C., Dilmanian, F.A., Dallal, G.E., & Evans, W.J. (1991). A one year walking program and increased dietary calcium in postmenopausal women: Effect on bone. American Journal of Clinical Nutrition, 53, 1304-1311.

*Pruitt, L.A., Taaffe, D.R., & Marcus, R. (1995). Effects of a one-year high-intensity versus low-intensity resistance training program on bone mineral density in older women. Journal of Bone and Mineral Research, 10, 1788-1795.

Revel, M., Mayoux-Benhamou, M.A., Rabourdin, J.P., Bagheri, F., & Roux, C. (1993). One-year psoas training can prevent lumbar bone loss in postmenopausal women: A randomized controlled trial. Calcified Tissue International, 53, 307-311.

*Ryan, A.S., Nicklas, B.J., & Dennis, K.E. (1998). Aerobic exercise maintains regional bone mineral density during weight loss in post menopausal women. Journal of Applied Physiology, 84, 1305-1310.

Salamone, I.M., Glynn, N.W., Black, D.M., Ferrell, R.E., Palermo, L., Epstein, R.S., Kuller, L.H., & Cauley, J.A. (1996). Determinants of premenopausal bone mineral density: The interplay of genetic and lifestyle factors. Journal of Bone Mineral Density, 11, 1557-1565.

*Sinaki, M., Wahner, H.W., Offord, K.P., & Hodgson, S.F. (1989). Efficacy of nonloading exercises in prevention of vertebral bone loss in postmenopausal women: A controlled trial. Mayo Clinical Proceedings, 64, 762-769.

*Smidt, G.L., Lin, S., O'Dwyer, K.D., & Blanpied, P.R. (1992). The effect of high-intensity trunk exercise on bone mineral density of postmenopausal women. Spine, 17, 280-285.

Swezey, R.L. (1996). Exercise for osteoporosis--is walking enough? The case for site specificity and resistive exercise. Spine, 1, 2809-2813.

Wahner, H.W., Dunn, W.L., Brown, M.L., Morin, R.L., & Riggs, B.L. (1988). Comparison of dual energy x-ray absorptiometry and dual photon absorptiometry for bone mineral measurements of the lumbar spine [Abstract]. Mayo Clinical Procedures, 11, 1075.

APPENDIX A

SOURCES NOT MEETING INCLUSION CRITERIA

Alekel, L., Clasey, J.L., Fehling, P.C., Weigel, R.M., Boileau, R.A., Erdman, J.W., & Stillman, R. (1995). Contributions of exercise, body composition, and age to bone mineral density in premenopausal women. Medicine and Science in Sports and Exercise, 27, 1477-1485.

Aloia, J.F., Cohn, S.H., Ostuni, J.A., Cane, R., & Ellis, K. (1978). Prevention of involutional bone loss by exercise. Annals of Internal Medicine, 89, 356-358.

Ballard, J.E., McKeown, B.C., Graham, H.M., & Zinkgraf, S.A. (1990). The effect of high level physical activity (8.5 METs or greater) and estrogen replacement therapy upon bone mass in postmenopausal females, aged 50 - 68 years. Internal Journal of Sports Medicine, 11, 208-214.

Beverly, M.C., Rider, T.A., Evans, M.J., & Smith, R. (1984). Local bone mineral response to brief exercise that stressed the skeleton. British Medical Journal, 299, 233-235.

Bidoli, E., Schinella, D., & Franseschi, S. (1998). Physical activity and bone mineral density in Italian middle-aged women. European Journal of Epidemiology, 14, 153-157.

Blumenthal, J.A., Emery, C.F., Madden, D.J., Schniebolk, S., Riddle, M.W., Cobb, F.R., Higginbotham, M., & Coleman, R.E. (1991). Effects of exercise training on bone density in older men and women. Journal of the American Geriatric Society, 39, 1065-1070.

Brahm, H., Mallmin, H., Michaelsson, K., Strom, H., & Ljunghall, S. (1998). Relationship between bone mass measurements and lifetime physical activity in a Swedish population. Calcified Tissue International, 62, 400-412.

Cheng, S., Suominen, H., Rantanen, R., Parkatti, T., & Heikkinen, E. (1997). Bone mineral density and physical activity in 50 - 60 year old women. Bone and Mineral, 12, 123-132.

Cohen, M., Millett, P.J., Mist, B., Laskey, M.A., & Rushton, N. (1995). Effect of exercise training programme on bone mineral density in novice college rowers. British Journal of Sports Medicine, 29, 85-88.

Dornemann, T.M., McMurray, R.G., Renner, J.B., & Anderson, J.J. (1997). Effects of high-intensity resistance exercise on bone mineral density and muscle strength of 40 - 50 year old women. Journal of Sports Medicine and Physical Fitness, 37, 246-251.

Drinkwater, B.L., Buremner, B., & Chestnut, C.H. III. (1990). Menstrual history as a determinant of current bone density in young athletes. Journal of American Medical Association, 263, 545-548.

Fessenich, C.R., Guyatt, G.H., & Rosen, C.J. (1998). Health related quality of life and participation in osteoporosis clinical trials. Calcified Tissue International, 62, 189-192.

Fielding, R.A. (1995). The role of progressive resistance training and nutrition in the preservation of lean body mass in the elderly. Journal of American College of Nutrition, 14, 587-594.

Gregg, E.W., Cauley, J.A., Seeley, D.G., Ensrud, K.E., & Bauer, D.C. (1998). Physical activity and osteoporotic fracture risk in older women. Study of Osteoporotic Fractures Research Group. Annals of Internal Medicine, 129, 81-88.

Greendale, G.A., Barrett-Connor, E., Edelstein, S., Ingles, S., & Haile, R. (1995). Lifetime leisure exercise and osteoporosis. The Rancho Bernardo study. American Journal of Epidemiology, 141, 951-959.

Hartard, M., Bottermann, P., Bartenstein, P., Jeschke, D., & Schwaiger, M. (1997). Effects on bone mineral density of low-dosed oral contraceptives compared to and combined with physical activity. Contraception, 55, 87-90.

Heinonen, A., Kannus, P., Sievanen, H., Oja, P., Pasanen, M., Rinne, M., Uusi-Rasi, K., & Vuori, I. (1996). Randomized controlled trial of effect of high-impact exercise on selected risk factors for osteoporotic fractures . Lancet, 348, 1343-1347.

Hopper, J.L., Green, R.M., Nowson, D.A., Young, D., Sherwin, A.J., Kaymakci, B., Larkins, R.G., & Wark, J.D. Genetic, common environment, and individual specific components of variance for bone mineral density in 10 to 26 year old females: A twin study. American Journal of Epidemiology, 147, 17-29.

Jonsson, B., Ringsberg, K., Josefsson, P.O., Johnell, O., & Birch-Jensen, M. (1992). Effects of physical activity on bone mineral content and muscle strength in women: A cross-sectional study. Bone, 13, 191-195.

Judex, S., Gross, T.S., & Zernicke, R.F. (1997). Strain gradients correlate with sites of exercise-induced bone-forming surfaces in the adult skeleton. Journal of Bone and Mineral Research, 12, 1737-1745.

Kano, K. (1998). Relationship between exercise and bone mineral density among over 5,000 women aged 40 years and above. Journal of Epidemiology, 8, 28-32.

Kerschan, K., Alacamlioglu, Y., Kollmitzer, J., Wober, C., Kaider, A., Hartard, M., Ghanem, A.H., & Preisinger, E. (1998). Functional impact of unvarying exercise program in women after menopause. American Journal of Physical and Medical Rehabilitation, 77, 326-332.

Kriska, A.M., Bayles, C., Cauley, J.A., LaPorte, R.E., Sandler, R.B., & Pambianco, G. (1986). A randomized exercise trial in older women: Increased activity over two years and the factors associated with compliance. Medicine and Science in Sports and Exercise, 18, 557-562.

Kreighbaum, E., & Barthels, K. (1996). Glossary. In S. Spivey (Eds.), Biomechanics: A qualitative approach for studying human movement (pp. 567-571). Needham Heights, MA: Simon & Schuster.

Kritz-Silverstein, D., & Barrett-Connor, E. (1994). Grip strength and bone mineral density in older women. Journal of Bone and Mineral Research, 9, 45-51.

Kudlacek, S., Pietschmann, F., Bernecker, P., Resch, H., & Willvonseder, R. (1997). The impact of a senior dancing program on spinal and peripheral bone mass. American Journal of Physical Medical Rehabilitation, 76, (6) 477-81.

Little, K.D., & Clapp, J.F. III. (1998). Self-selected recreational exercise has no impact on early postpartum lactation-induced bone loss. Medicine and Science in Sports and Exercise, 30, 831-836.

McCartney, N., Hicks, A.L., Martin, J., & Webber, C.E. (1995). Long-term resistance training in the elderly: effects on dynamic strength, exercise capacity, muscle, and bone. Journal of Gerontology A: Biological Science and Medicine, 50, 97-104.

Nordstrom, P., Nordstrom, G., & Lorentzon, R. (1998). Massive increase in bone density by high impact loading exercise in a 26-year-old osteoporotic woman on high doses of glucocorticoids. Osteoporosis International, 8, 196.

Pocock, N.A., Eisman, J.A., Yeates, M.G., Sambrook, P.N., & Eberl, S. (1986). Physical fitness is a major determinant of femoral neck and lumbar spine bone mineral density. Journal of Clinical Investigation, 78, 618-621.

Preisinger, E., Alacamlioglu, Y., Pils, K., Saradeth, T., & Schneider, B. (1995). Therapeutic exercise in the prevention of bone loss. A controlled trial with women after menopause. American Journal of Physical and Medical Rehabilitation, 74, 120-123.

Preisinger, E., Alacamlioglu, Y., Pils, K., Bisubam E., Metka, M., Schneider, B., & Ernst, E. (1996). Exercise therapy for osteoporosis: Results of a randomized controlled trial. British Journal of Sports Medicine, 30, 209-212.

Riggio, O., Andreoli, A., Diana, F., Fiore, P., Meddi, P., Lionetti, R., Montagnese, F., Merli, M., Capocacci, L., & DeLorenzo, A. (1997). Whole body and regional body composition analysis by dual-energy x-ray absorptiometry in cirrhotic patients. European Journal of Clinical Nutrition, 51, 810-814.

Rikli, R.E., & McManis, B.G. (1990). Effects of exercise on bone mineral content in postmenopausal women. Research Quarterly for Exercise and Sport, 61, 243-249.

Robinson, R.J., Krzywicki, T., Almond, L., Azzawi, F., Abrams, K., Iqbal, S.J., & Mayberry, J.F. (1998). Effect of a low-impact exercise program on bone mineral density in Crohn's disease: A randomized controlled trial. Gastroenterology, 115, 36-41.

Rong, H., Berg, U., Tirring, O., Sundberg, C.J., Granberg, B., & Bucht, E. (1997). Effect of acute endurance and strength exercise on circulating calcium-regulating hormones and bone markers in young healthy males. Scandinavian Journal of Medicine and Science in Sports, 7, 152-159.

Rudgren, A., Niansson, A., Ljungber, P., & Wetterqvist, H. (1984). Effects of a training programme for elderly people on mineral content of the heel bone. Archives of Gerontological Geriatrics, 3, 243-248.

Ryan, A.S., Treuth, M.S., Hunter, G.R., & Elahi, D. (1998). Resistive training maintains bone mineral density in postmenopausal women. Calcified Tissue International, 62, 295-299.

Sinaki, M., & Offord, K.P. (1988). Physical activity in postmenopausal women: Effect on back muscle strength and bone mineral density of the spine. Archives in Physical and Medical Rehabilitation, 69, 277-280.

Snow-Harter, C., Bouxsein, M.L., Lewis, B.T., Carter, D.R., & Marcus, R. (1992). Effects of resistance and endurance exercise on bone mineral status of young women: A randomized exercise intervention trial. Journal of Bone and Mineral Research, 7, 761-769.

Stanley, S.N., Marshall, R.N., Tilyard, M.W., & Taylor, N.A. (1994) Skeletal muscle mechanics in osteoporotic and nonosteoporotic postmenopausal women. European Journal of Applies Physiology, 69, 450-455.

Worley, R.J. (1981). Age, estrogen, and bone density. Clinical Obstetrics and Gynecology, 24, 203-218.

Trevisan, C., Gandolini, G.G., Sibilla, P., Penotti, M., Caraceni, M.P., & Ortolani, S. (1992). Bone mass measurement by DXA: Influence of analysis procedures and interunit variation. Jounal of Bone and Mineral Research, 7, 1373-1382.

Willing, M., Sower, M., Aron, D., Clark, M.K., Burns, T., Bunten, C., Crutchfield, M., D'Agostino, D., & Jannausch, M. (1998). Bone mineral density and its change in white women: estrogen and vitamin D receptor genotypes and their interaction. Journal of Bone and Mineral Research, 13, 695-705.

APPENDIX B

PRE AND POST BMD VALUES

Table 5. Lumbar spine BMD measurements ($gm/cm^{2)}$ for included studies (mean and standard deviation)

Authors article no.	Device	Exercise pre	Exercise post	Control pre	Control post
1) Ryan et al.	DEXA	1.127 0.132	1.143 0.133	1.133 0.154	1.123 0.166
2) Nelson et al.	DEXA	1.020 0.164	1.029 0.033	0.986 0.152	0.967 0.035
3) Kohrt et al.	DEXA	0.943 0.043	0.959 0.007	0.926 0.047	0.928 0.005
4) Kohrt et al.	DEXA	0.907 0.037	0.921 0.005	0.926 0.047	0.928 0.006
5) Grove & Londeree	DPA	1.170 0.100	1.190 0.120	1.150 0.120	1.080 0.150
6) Grove & Londeree	DPA	1.180 0.100	1.180 0.110	1.150 0.120	1.080 0.150

Table 5. (cont'd)

Authors article no.	Device	Exercise pre	Exercise post	Control pre	Control post
7) Khort et al.	DEXA	0.885 0.148	0.909 0.016	0.828 0.104	0.826 0.002
8) Pruitt et al.	DEXA	1.128 0.027	1.140 0.022	1.117 0.045	1.094 0.050
9) Dalsky et al.	DPA	1.003 0.003	1.062 0.035	1.016 0.036	1.003 0.038
10) Smidt	DPA	1.117 0.031	1.097 0.027	1.149 0.029	1.122 0.027
11) Bloomfield et al.	DPA	1.100 0.060	1.140 0.016	1.040 0.040	1.010 0.008
12) Nelson et al.	DPA	1.181 0.069	1.170 0.069	1.065 0.051	1.082 0.042
13) Pruitt et al.	DPA	1.079 0.176	1.084 0.178	0.901 0.158	0.901 0.158

Table 5. (cont'd)

Authors article no.	Device	Exercise pre	Exercise post	Control pre	Control post
14) Pruitt et al.	DPA	0.940 0.152	0.947 0.159	0.901 0.158	0.901 0.158
15) Bassey & Ramsdale	DEXA	0.597 0.027	0.582 0.027	0.555 0.018	0.549 0.022
16) Harfard et al.	DEXA	0.871 0.207	0.874 0.210	0.876 0.141	0.822 0.125
17) Ebrahim et al.	DEXA	0.997 0.194	1.002 0.093	0.938 0.093	1.019 0.041
18) Lord et al.	DEXA	1.024 0.198	1.036 0.209	1.004 0.175	1.008 0.189

Table 6. Proximal femur BMD measurements (gm/cm^2) for included studies (mean & standard deviation)

Authors article no.	Device	Exercise pre	Exercise post	Control pre	Control post
1) Ryan et al.	DEXA	0.908 0.136	0.917 0.127	0.888 0.084	0.865 0.083
2) Nelson et al.	DEXA	0.853 0.134	0.858 0.039	0.828 0.109	0.806 0.035
3) Kohrt et al.	DEXA	0.668 0.031	0.691 0.005	0.679 0.026	0.673 0.006
4) Kohrt et al.	DEXA	0.662 0.023	0.649 0.004	0.679 0.026	0.673 0.006
7) Khort et al.	DEXA	0.612 0.043	0.646 0.019	0.643 0.050	0.631 0.019
8) Pruitt et al.	DEXA	0.834 0.029	0.814 0.028	0.799 0.032	0.797 0.036

Table 6. (cont'd)

Authors article no.	Device	Exercise pre	Exercise post	Control pre	Control post
10) Smidt	DPA	0.757 0.022	0.765 0.021	0.792 0.021	0.790 0.022
11) Bloomfield et al.	DPA	0.840 0.040	0.861 0.018	0.760 0.030	0.754 0.005
12) Nelson et al.	DPA	0.837 0.054	0.827 0.053	0.804 0.025	0.796 0.026
13) Pruitt et al.	DPA	0.692 0.162	0.700 0.142	0.636 0.075	0.641 0.070
14) Pruitt et al.	DPA	0.613 0.100	0.610 0.093	0.636 0.075	0.641 0.070
15) Bassey & Ramsdale	DEXA	0.923 0.026	0.924 0.024	0.887 0.027	0.880 0.027
16) Harfard et al.	DEXA	0.747 0.133	0.736 0.137	0.732 0.092	0.670 0.080

Table 6. (cont'd)

Authors article no.	Device	Exercise pre	Exercise post	Control pre	Control post
17) Ebrahim et al.	DEXA	0.806 0.122	0.807 0.047	0.765 0.145	0.758 0.045
18) Lord et al.	DEXA	0.781 0.125	0.791 0.122	0.756 0.118	0.776 0.110

Table 7. Distal radius BMD measurements (gm/cm^2) for included studies (mean and standard deviation)

Authors article no.	Device	Exercise pre	Exercise post	Control pre	Control post
3) Kohrt et al.	DEXA	0.372 0.020	0.369 0.003	0.354 0.017	0.350 0.004
4) Kohrt et al.	DEXA	0.347 0.016	0.349 0.004	0.354 0.017	0.350 0.004
7) Khort et al.	DEXA	0.371 0.034	0.333 0.023	0.328 0.035	0.321 0.030
8) Pruitt et al.	DEXA	0.929 0.034	0.925 0.030	0.854 0.052	0.898 0.059
12) Nelson et al.	DPA	0.645 0.042	0.637 0.039	0.608 0.040	0.612 0.038
13) Bassey et al.	DEXA	0.794 0.018	0.785 0.018	0.757 0.019	0.763 0.016

APPENDIX C

PHYSICAL CHARACTERISTICS OF SUBJECTS

Table 8. Subject's physical characteristics at baseline measurement (mean & standard deviation)

	Study Number									
	1		2		3		4		5	
	ex*	c**	ex	c	ex	c	ex	c	ex	c
Age (yrs)	62.0 6.0	63.0 6.0	61.6 3.7	57.3 6.3	66.0 1.0	68.0 1.0	65.0 1.9	68.0 4.5	54.0 1.9	56.0 4.5
Onset of Menopause (yrs)	>2.0	>2.0	>5.0	>5.0	16.0 1.0	11.0 2.0	16.0 1.0	11.0 2.0	4.8 2.9	9.2 1.6
Weight (kg)	79.3	83.1	64.7	62.2	70.9	71.6	72.6	71.6	72.3	69.3
BMI (kg/m^2)	30.5	30.9	24.4	23.1	26.9	26.9	26.7	26.9	N/A	N/A

*ex = exercise group
**c = control group

Table 8. (cont'd)

	Study Number									
	6		7		8		9		10	
	ex*	c**	ex	c	ex	c	ex	c	ex	c
Age (yrs)	56.6 4.3	56.0 4.5	65.0 3.0	66.0 3.0	53.6 1.0	55.6 0.9	61.6 1.0	62.6 1.2	56.6 6.6	55.4 8.0
Onset of Menopause (yrs)	15.0 2.0	9.2 1.6	20.0 4.0	18.0 4.0	3.1 0.5	4.0 0.6	11.6 1.7	13.5 1.9	9.1 6.8	11.3 6.0
Weight (kg)	69.0	69.3	63.4	63.4	64.2	65.5	61.9	59.5	65.3	66.8
BMI (kg/m^2)	N/A	N/A	24.5	24.5	24.3	24.6	23.7	23.0	24.7	25.2

*ex = exercise group
**c = control group

Table 8. (cont'd)

	Study Number									
	11		12		13		14		15	
	ex*	c**	ex	c	ex	c	ex	c	ex	c
Age (yrs)	62.0	58.8	>60.2	>60.2	68.0	69.0	67.0	69.0	54.4	55.0
	0.8	2.0	6.5	6.5	N/A	N/A	N/A	N/A	4.0	3.0
Onset of Menopause (yrs)	11.4	15.4	N/A	N/A	N/A	N/A	N/A	N/A	7.0	7.0
	2.6	2.0							4.0	5.0
Weight (kg)	77.4	64.4	64.0	64.0	61.5	63.8	64.5	63.8	63.3	64.7
BMI (kg/m^2)	28.0	25.0	24.4	24.4	23.9	25.1	24.5	25.1	24.6	24.9

*ex = exercise group
**c = control group

Table 8. (cont'd)

	Study Number					
	16		17		18	
Group	ex*	c**	ex	c	ex	c
Age (yrs)	63.6 6.2	67.4 9.7	66.4 7.8	68.1 7.8	71.7 5.4	71.5 5.3
Onset of Menopause (yrs)	>2.0 N/A	>2.0 N/A	17.7 N/A	20.0 N/A	N/A	N/A
Weight (kg)	67.0	63.8	N/A	N/A	66.0	64.7
BMI (kg/m^2)	25.6	25.4	26.6	26.3	26.8	26.2

*ex = exercise group
**c = control group

APPENDIX D

DESCRIPTIONS OF THE INTERVENTIONS

Table 9. Descriptions of the exercise intervention used by each study.

Study	Year	Duration (months)	Frequency (per week)	Minutes	Program Description
1	1998	6	3	60	Exercise session included walking or jogging on treadmills along with warm up and cool down on cycle ergometers.
2	1994	12	2	60	High intensity strength training for each of the major muscle groups was performed. At least one day of rest was required between exercise sessions.
3	1997	11	3	45-60	The ground reaction force group (GRF) performed activities such as walking, jogging or stair climbing for at least 30 minutes plus a warm up and cool down.
4	1997	11	3	45-60	The joint reaction force group (JRF) performed activities such as rowing and weight lifting for at least 30 minutes plus a warm up and cool down.

Table 9. (cont'd)

Study	Year	Duration (months)	Frequency (per week)	Minutes	Program Description
5	1992	12	3	60	This exercise group performed high impact aerobics for 25 minutes, strengthening and toning exercises, and a warm up and cool down.
6	1992	12	3	60	This exercise group performed low impact aerobics for 25 minutes, stretching and toning exercises, and a warm up and cool down.
7	1995	11	3	30	This intervention included flexibility training (2 months) and weight bearing exercise training (9 months).
8	1992	9	3	60	This exercise group began with a 10 minute warm up, weight trained for 40 minutes, and cooled down for the remaining 10 minutes.

Table 9. (cont'd)

Study	Year	Duration (months)	Frequency (per week)	Minutes	Program Description
9	1988	9	3	60	Weight bearing activities, such as walking or jogging, for 50-60 minutes per session were used. Stair climbing was added later in the study along with nonweightbearing exercises such as cycling, rowing, and weight training.
10	1991	12	3/4		This study used resistance training, which included 10 repetitions each of sit ups, double leg raises, and prone trunk extension
11	1993	8	3	50	A 15 minute warm up period with flexibility and calisthenics, 30 minutes of stationary cycling and a 5 minute cool down was used as the exercise intervention.

Table 9. (cont'd)

Study	Year	Duration (months)	Frequency (per week)	Minutes	Program Description
12	1991	12	4	50	Supervised walking sessions were used as the exercise protocol.
13	1995	12	3	60	The exercise group used Nautilus and Universal Gym Equipment to perform low intensity resistance exercises for all of the major muscle groups. Stretching was done for 5-10 minutes and resistance training following for approximately 50 minutes.
14	1995	12	3	60	This group used the same protocol as the previously mentioned group with the exception of high intensity training vs. low intensity.
15	1995	12	5		This exercise regimen included daily home exercise and heel drops along with a weekly exercise to music supervised class.

Table 9. (cont'd)

Study	Year	Duration (months)	Frequency (per week)	Minutes	Program Description
16	1996	12	2		Strength training was performed on eleven of the major muscle groups (8-12 reps) along with a warm up and cool down session.
17	1997	12	3	40	This exercise group performed brisk walking as the intervention.
18	1996	12	2	60	This study used a 5 minute warm up, a 35 minute aerobic session, a 15 minute stretching period, and a 5-10 minute cool down as the exercise group's treatment.

APPENDIX E

FORMULAS

Formula 1. Use this equation to figure the variance of the individual TEs.

$$\text{variance TE} = \frac{N_e + N_c}{N_e * N_c} + \frac{(TE_i)^2}{2(N_e + N_c)}$$

where N_e = the number of subjects in the exercise group, N_c = the number of subjects in the control group, and TE_i = the unweighted individual TE. Then, to find the inverse of the variance, divide one by the answer found above for each study.

Formula 2. The weighted mean for a group TE can be calulated by:

group TE = the sum of X divided by the sum of Y

$$X = \frac{TE_1}{\text{var } TE_1} + \frac{TE_2}{\text{var } TE_2} + \ldots\ldots\ldots\ldots + \frac{TE_p}{\text{var } TE_p}$$

$$Y = \frac{1}{\text{var } TE_1} + \frac{1}{\text{var } TE_2} + \ldots\ldots\ldots\ldots + \frac{1}{\text{var } TE_p}$$

TE = the unweighted individual TE, and var of TE = the variance of each individual TE in the group.

Formula 3. In order to find the variance of the group TE:

$$\text{variance of group TE} = \frac{1}{Y}, \text{ where Y is defined above.}$$

The 95% confidence intervals are then figure by taking the group TE + or - 1.96 multiplied by the square root of the variance of the group TE.

Formula 4. Testing for homogeneity was performed by:

$$Q = A_1 + A_2 + \ldots\ldots + A_p$$

$$\text{where } A_i = \frac{(\text{group TE} - \text{TE}_i)}{\text{variance of TE}_i},$$

$$\text{and } i = 1, 2, \ldots\ldots, p.$$

APPENDIX F

REVIEW OF RELATED LITERATURE

REVIEW OF RELATED LITERATURE

Introduction

Since a woman can expect to live at least one third of her life after menopause, it is important to prevent the development of osteoporosis (Gregory, 1982). Many variables such as physical activity, diet, hormones, calcium, and genetics influence the progression of osteoporosis. Identifying behaviors which may lead to this disease, both before and after menopause, is extremely important in order to eliminate such risk factors and provide a means of prevention (Gregory, 1982). While much has been learned about bone mineral density (BMD) from the diagnosis and treatment of osteoporosis, "prevention is an essential, and often overlooked aspect of this disease" (Tsukahara, Toda, Goto, & Ezawa, 1993, p. 45).

According to Gregory (1982), risk factors for bone fractures as a result of low bone density are as high as 50% for women and 25% for men. As time passes, more and more studies are being done to investigate changes in bone density in postmenopausal women. For example, Duppe, Gardsell, Johnell, Nilsson, and Ringsberg (1997) showed the benefits of increased of physical activity on muscle strength and bone density in children. Others, such as Sinaki et al. (1996), compared changes in the BMD of active middle age females to their age matched controls. Furthermore, Goto, Shigeta, Hayakutake, and Yamagata (1996) studied the BMD of former athletes for several years as they entered menopause, and demonstrated that BMD in active females is comparatively higher than sedentary females. Although men are sometimes

diagnosed with osteoporosis, it is primarily a disease which affects women, especially those who are postmenopausal. Therefore, finding a means to reduce the incidence of osteoporosis in postmenopausal females is very important.

An overview of these factors which are known to influence bone density, and theories concerning the impact of each factor and its relation to osteoporosis, are reviewed below as well as different techniques used to measure bone density.

Factors Influencing Bone Density

Weight Loss and Body Composition

In 1997, Kohrt, Ehsani, and Birge experimented with the effects of weight loss and physical activity on BMD. They found that the positive effects of exercise on the skeleton were counteracted with the negative effects of a weight loss diet. A heavier body weight places increased stress on bones and promotes maintenance (Whitney & Rolfes, 1996). When body weight is below normal, such as in amenorrheic athletes, BMD decreases and fracture risk increases (Drinkwater, 1996). Two reports (Cann, Cavaraugh, & Schnurpfiel, 1988; Drinkwater, 1994) found that even when menses returned, the athletes did not regain BMD. Drinkwater (1996) states "body weight can be a confounding factor in studies of bone mineral density because the correlation between weight and bone mineral density is usually significant" (p. 34).

Calcium

During the first two decades of life, the skeleton continues to grow stronger and denser as it accumulates more minerals including calcium. Peak bone mass in white women is thought to be in its final stage between the ages

of 20 - 25 years (Anderson & Metz, 1993). Not only does the older adult seem to intake lower levels of calcium, but the absorption process of calcium after age 65 significantly declines (Whitney & Rolfes, 1996). In order for calcium to be absorbed, vitamin D must also be present. Once again, during the aging process, the kidneys do not activate vitamin D as effectively. Another factor influencing the absorption of calcium, related to vitamin D, is sunlight. Older people also tend to remain indoors more often and therefore, vitamin D cannot be formed naturally within the body.

Many scientists agree that calcium recommendations set for adults in the United States and Canada exceed what is actually needed since BMD is greatly influenced by how well the bones developed and were maintained during youth and early adulthood (Whitney & Rolfes, 1996). For adult women, the Recommended Daily Allowance (RDA) suggests 800 to 1,200 mg from food sources. However, postmenopausal women with osteoporosis should accumulate 1,500 mg if they are not receiving estrogen therapy (Whitney & Rolfes). Anderson and Metz (1993) concluded that premenopausal adult women must ingest calcium at the RDA level in order to maintain peak bone mass throughout menopause if their activity level is moderate.

Another dietary factor related to calcium intake and osteoporosis is the effect of caffeine on bone loss. Harris and Dawson-Hughes (1994) found that consumption of caffeine in healthy postmenopausal women with calcium intakes at or above the RDA had no effect on BMD. However, they did find that women with low intakes of calcium had a higher risk for bone loss if they did consume caffeine. This is an important detail since many adults drink coffee, tea, or soda frequently. The combination of high caffeine intake and low

calcium intake may put more women at a higher risk for osteoporosis after menopause. Therefore, enforcing healthy habits during childhood may increase lifetime calcium intake and decrease caffeine intake.

National Health Promotion and Disease Prevention Objectives (U.S. Department of Health and Human Services, 1996) included a goal for the entire nation as a strategy in preventing osteoporosis. The goal is aimed primarily at children and the prevention of risk factors of bone deterioration. By the year 2000, all school age children over 10 years of age should be involved in activities which increase physical activity, calcium intake, and education on nutritional issues. This is only one step which is being taken in order to prevent the incidence of bone loss in adults.

For many adults, getting enough calcium in their normal diet is difficult since it often means increasing total caloric intake, therefore, they are more likely to take supplements to compensate for this deficiency. Whitney and Rolfes (1996) list several problems with calcium supplements: "impaired iron status; accelerated calcium loss; urinary tract stones or kidney damage in susceptible individuals; exposure to contaminants; vitamin D toxicity; excess blood calcium; milk alkali syndrome; other nutrient interactions; drug interactions; and constipation, intestinal bloating, and excess gas" (p. 469). As with any medication, the positive and negative effects of taking a calcium supplement should be thoroughly discussed with a health care provider before beginning a supplementation program.

Age

Suleimen, Nelson, Ji, Buxton-Thomas, and Moniz (1997) concluded that calcium intake and physical activity levels are most important earlier in life

allowing for a high peak bone mass to be reached. As years pass, the cells which build bone become gradually less active, while the destruction of bone is continuous (Whitney & Rolfes, 1996). The median age at which women begin to lose BMD is 34 years of age (Stacey, 1989). Therefore, if a young athlete experiences exercise-induced amenorrhea, not only does she put herself in a situation where she is lacking calcium, but she is also estrogen deficient (Crowley,1997).

Both dietary calcium and physical activity levels should be high during the premenopausal years in order to maintain and create bone mass (Suleiman et al., 1997). Bone development in young people responds differently to mechanical loading as compared to older people. According to Drinkwater (1996), "young bone is more responsive to loading than old bone and the response is an increase in bone size rather than bone density" (p. 33). Since a woman's body cannot sufficiently stockpile calcium for her postmenopausal years, physical activity and calcium intake is very important during childhood, adolescence, and all the way through life.

Hormones

After menopause, hormone replacement therapy (HRT) is sometimes used to treat osteoporosis. Gonadal steroids such as estrogens and progestogens along with pharmacological doses of calcium, biphosphonates, and even vitamin D supplements are prescribed in order to replace the estrogen no longer produced in sufficient amounts in postmenopausal women (Kanis, 1996). Kanis also suggested that HRT is responsible for a decrease in cardiovascular morbidity and mortality along with controlling many of the symptoms of the menopausal change. In some cases, the advantages of HRT

outweigh the slightly increased risk of endometrial carcinoma and breast cancer which is sometimes associated with the administration of HRT (Crowley, 1997; "Drug prevents," 1998).

As estrogen is removed from the body, BMD decreases rapidly (Whitney & Rolfes, 1996).

> "....accelerated losses last for six to eight years following menopause, then taper off, so that women again lose bone at the same rate as men their age. Losses of bone minerals continue throughout the remainder of a woman's lifetime, but not at the free-fall pace of the menopause years" (Whitney & Rolfes, 1996, p. 465).

A similar effect is seen in amenorrehic females placing them at increased risk for low BMD in the future.

Tamoxifen is a relatively new drug therapy out on the market. Love, Barden, Mazess, Epstein, and Chappell (1994) conducted a five year study on the effects of tamoxifen on BMD. They found that during the first two years of the study, the control group which was taking a placebo drug loss spinal BMD where as the experiment group taking tamoxifen gained BMD. However, neither group continued to gain or lose spinal BMD in the next three years, thus they concluded: "studies in vitro and in animals have demonstrated that the effects of tamoxifen on bone resemble those of estrogen. The effects in humans have been less clear" (Love et al., 1994, p. 2586).

Alendronate (Fosamax), a nonhormone drug which is nearly as effective as HRT in preventing osteoporosis has been recently introduced on the market ("Drug prevents," 1998). Alendronate has been shown to increase BMD of the spine, hip and total body by about 1 to 4% when compared to a placebo group which lost BMD at approximately 2 to 5%. The long term effects of this new drug are still unknown and side effects include nausea and difficulty swallowing. The

same article also refers to the importance of physical activity, adequate calcium, and vitamin D intake in the management of osteoporosis.

Genetics and Race

In 1989, Pollitzer and Anderson stated that 70% to 80% of peak bone mass development was related to genetic factors. Anderson and Metz (1993) report a similar figure stating that up to approximately the age of 20 years, 50 - 70% of bone mass development is related strongly to heredity. After age 20, environmental factors may take on a larger effect for BMD.

A vitamin D receptor gene seems to be the mediator in determining bone mass and peak bone mass which is linked to bone turnover (Drinkwater, 1994). Heredity is likely to not only influence peak bone mass, but also the extent of bone loss after menopause (Whitney & Rolfes, 1996). However, "one's genetic predisposition for BMD may be modified by environmental factors" (Salamone et al., 1996, p. 1558). Exercise, along with other factors, may increase BMD above the level determined genetically.

Race also tends to be a factor in osteoporosis and low bone density. According to Whitney and Rolfes (1996) "significant differences in bone metabolism divide elderly African Americans from Caucasians. Black people have denser bones than do white people, and these differences are evident even before birth" (p. 466). Hip fractures are three times greater in Caucasian people over 80 years of age when compared to African-Americans. However, Asians from China and Japan, Mexican Americans, Hispanics from Central and South America, and Inuit people from St. Lawrence Island all have less dense bones than do people from the European heritage. This too, lays the

groundwork for environmental factors to override the genetic disposition for osteoporosis (Whitney & Rolfes, 1996).

Physical Activity

Physical inactivity is one of the most commonly recognized risk factors for osteoporosis and according to Ernst (1994), the occurrence of hip fractures has doubled in the past 30 years due to this lack of activity. Bed rest has been shown to result in severe and rapid decreases in bone mass reported up to 1% per week according to Ernst. Physically active adults have a higher BMD when compared to inactive adults of the same age and weight (Dalen & Olsson, 1974). Athletes are reported to have higher BMDs compared to sedentary control groups (Drinkwater, 1996). Increased activity is considered beneficial therapy for osteoporosis; however, the type of activity and the load which is placed on the bones is a concern.

A load or stress must be placed on the bones in order for them to maintain bone density levels. Also, only the bones which are being stressed will change in density. Evidence of this was found in Wolff's Law which states that "a change in the form or function of bone leads to change in internal structure" (Drinkwater, 1996, p. 33). In other words, "bone adapts to the functional forces placed upon it" (Stacey, 1989, p. 211). Ernst (1994) stated that the response of bone when a stress is applied to it is "to generate an electric signal creating alkaline conditions locally, which in turn favor new bone generation" (p. 5). Thus, exercise modalities which apply stress, shear, tension, or strain to the skeletal bones will enhance the piezoelectric field and therefore provide an effective environment to increase the density of bones (Stacey, 1989). Swimming, a nonweightbearing activity, apparently does not have the

same effect on BMD as weight bearing exercises since BMDs of swimmers were found to remain below the level of the sedentary control group (Drinkwater, 1996).

Aerobic exercise. Aerobic exercise is a "method of conditioning the cardiorespiratory system by using a variety of activities that create an increased demand for oxygen over an extended period of time" (Beam, 1995, p. 450) and is a popular form of physical activity, especially among females (Alfredson, Nordstrom, & Lorentzon, 1997). Although it is an activity which targets the cardiopulmonary system, positive effects have been shown on muscles, tendons, and BMD (Alfredson et al., 1997). Hoover, Webber, Beaumont, and Blake (1996) reported that cardiovascular fitness has a direct relationship to BMD; however, the intensity of the exercise is important to regulate when attempting to counteract the effects of osteoporosis.

Ernst (1994) reported that mild activities, such as those done in daily living, will not increase bone mineral content in a substantial form. Heikkinen et al. (1991) supported the same theory as Ernst after their research, which included bone loading exercises for the entire body, did not produce an increase in lumbar BMD, but did however, increase radial BMD. This also suggests that changes in BMD are site specific depending on the activity involved.

On the opposite end of the spectrum, vigorous exercise (i.e., jogging, rowing, or stair climbing), allowed a 5.2% increase in lumbar bone mineral content after 9 months (Ayalon, Cohn, Ostuni, Canc, & Ellisk, 1987). Four years of a vigorous exercise program, according to Krolner, Toft, Nielsen, and Tondevold (1983), showed complete reversal of bone loss after menopause.

Alfredson et al. (1997) also found a higher bone mass in sites such as the spine and hip after three years of aerobic training in women 21.4 to 26.8 years of age. Bassett (1995) suggests that the difference in skeletal response to various modes and intensities of exercise is "almost exclusively from specific details of the activity, largely outside controlled by endogenous hormone production" (p. 515).

Drinkwater (1994) notes that walking, an aerobic exercise, is the most frequently prescribed exercise for bone health in women. However, she found that even though the exercise provides improvement for overall health, it does not provide enough stress or overload on the bones to increase their density. For the exercise to be helpful in preventing osteoporosis, a greater stress than normal must be applied.

Weight training. Since many aerobic activities do not require stress to be placed on the bones of the upper body, weight training can be useful to help prevent deterioration of the upper body bones. Weight training also provides many of the same benefits as aerobic activities, such as increased muscle endurance and strength, and decreased body fat. Peterson et al. (1991) found that weight training had a greater effect on bone in nondominant arms compared to dominant arms. Although the applied loads were equal in both arms, the nondominant arm experienced more of an overload and therefore gained more strength and increased BMD (Peterson et al., 1991).

Furthermore, including weight training with an aerobic workout seemed to increase compliance according to Ernst (1994). Therefore, if compliance can be increased, the resulting bone density may be increased due to both aerobic exercise and weight training. Pruitt, Jackson, Bartels, and Lehnhard (1992)

found that retention of bone mineral content in the radius and ulna coincided with the inclusion of weight training in workouts.

Devices to Measure Bone Density

Noninvasive techniques for measuring bone density have evolved over the past 50 years. The goal is precise timing, accuracy, and consistency. According to Markel and Chao (1993) noninvasive techniques have several applicable uses when fractures are involved: (1) determining appropriate time to remove fixation devices, (2) to allow recommendations for patients to accelerate from nonweightbearing activities to weight bearing activities, and (3) to detect abnormal healing. Noninvasive techniques are also used to diagnosis diseases of the skeletal bones, such as osteoporosis.

As the different methods of measuring BMD are introduced, several key terms will be necessary to address. Precision and accuracy are commonly questioned and sometimes used interchangeably when comparing the results of various techniques. "Good precision means few random errors, and good accuracy means that the measured value corresponds well to the true value" ("Bone density," 1997, p.12).

Measurement devices are divided into two categories, nonionizing and ionizing radiation ("Bone density," 1997). Nonionizing radiation is a technique which does not involve ionized atoms or molecules in tissues. Devices which use this technique include ultrasound, laser, or ultraviolet radiation. Ionizing radiation is classified into those which use isotopes, such as dual energy x-ray absortiometry and quantitative computed tomography, and those which use x-ray tubes, such as single photon absorptiometry and dual photon absorptiomety, as the main radiation source ("Bone density,").

Until the advance of technology, methods for BMD measurement included manual examination, radiographic evidence, passage of time, and symptomatic pain reported by patient (Markel & Chao, 1993). They suggest that noninvasive techniques also allow the technician to detect for bone problems without disruption, to evaluate the quality of tissues, and to establish the state of bone healing. The following are some of the commonly used noninvasive techniques available to technicians and scientists today.

Resonant Frequency and Impedance

This method measures BMD by monitoring the response of bone as an accelerated force is applied to the skin. This type of test is used to establish the mechanical properties of the bone (Markel & Chao, 1993). Resonance and impedance has been helpful in determining bone healing diagnosis, however, it is not widely accepted for the following reasons: (1) it can only be used on bones which are accessible for excitation and measurement, (2) the electrical impulse must pass through tissue layers and does not come into direct contact with the bone, (3) interpretation is affected by muscles, tendons, and ligaments, and (4) stabilized fractures would be difficult to assess (Markel & Chao).

Ultrasound

Ultrasound uses the speed at which sound travels through bones to determine bone density. This is a rather new technique when used as a BMD measuring device. One skeletal site, the calcaneus, is measured to assess BMD. Furthermore, ultrasound can also measure the elasticity of bone along with the amount of bone mass. Precision has been reported to be between 2 and 4% while accuracy in determining BMD is approximately 20% ("Bone density," 1997).

Radiographic Photodensitometry

Bone mineral content is determined by identifying photon absorption of bone on a standard roentgenographic film through radiographic photodensitometry (Markel & Chao, 1993). The degree to which the film absorbs the photons is measured on a photodensitometer. These authors stated that the film is calibrated by simultaneously exposing a reference aluminum alloy wedge with a similar rate of x-ray absorption of the bone. This technique is used in most of the new methods to measure BMD such as quantitative computed tomography (QCT), single photon absorptiometry (SPA), and dual energy x-ray absorptiometry (DEXA).

Neutron Activation Analysis (NAA)

Since 98% of all calcium in the body is found in the skeleton, the total mass of calcium in bones can be identified through NAA ("Bone density," 1997). The NAA irradiates the whole body with neutrons and then measures the induced gamma radiation from bone calcium. The accuracy is 3 to 5% while the precision is between 1 and 5% depending on measurement site. Unfortunately, this method is not readily available.

Compton Scattering

This measurement device also generally uses the calcaneus as the site for measurement, however,

> ".....skeletal density in a measurement site can be determined by utilizing the relationship between the number of photons that scatter with and without energy loss. Because the energy difference between coherently and incoherently scattered photons is small, attenuation in the surrounding tissue will not influence the result. The method has a precision in vivo of approximately 2%. Accuracy remains to be determined" ("Bone density," 1997, p.17).

Quantitative Computed Tomography (QCT)

The QCT technique has been used to assess skeletal status and detect age and disease related changes in BMD (Grampp et al., 1997). Also, "QCT can be used to measure trabecular bone within vertebral body" (Goodwin, 1987, p. 295). By using the previously mentioned scanning system of radiographic photodensitometry, QCT has been proven reliable and accurate in measuring bone density according to Markel and Chao (1993).

The QCT has an advantage over other methods in that it can measure porous and compact areas of the skeleton separately. Precision is approximately 1.5 to 4% and accuracy is between 10 and 20% ("Bone density,").

Dual-energy X-ray Absorptiometry (DEXA)

This procedure uses two energy x-ray sources to measure BMD. A X-Y pattern results from crossing a light beam with a calibration disk containing various x-ray absorbing material (Markel & Chao 1993). According to Bravo, Gauthier, Roy, Payette, and Gaulin (1997), DEXA offers "improved precision (1 %), a shorter scanning time, and reduced radiation doses to study subjects" (p. 1376). Variations do occur with varying bone distance from the imaging table. However, the BMD decreases in a linear fashion as the height of the subject increases off the table (Markel & Chao, 1993).

The DEXA methods which are available can measure several different sites such as the lumbar spine, femur, forearm, heel, and the whole body as well providing lateral and anterior views ("Bone density," 1997). Furthermore, the DEXA is increasingly being used not only for the whole body measurements, but for body composition studies. Precision of the anterior

vertebrae is 1%, for the lateral measurement of the vertebral body approximately 2 to 3% and at the femoral neck approximately 1.5% ("Bone density").

In a comparison of DEXA conducted by Wahner, Dunn, Brown, Morin, and Riggs (1988), DEXA had a short scanning time and high resolution. It also had better precision in BMD measurements of the spine. The DEXA also allows BMD to be measured independently of fat and tissues surrounding the bone (Stacey, 1989). According to Wahner and associates, this method is used quite frequently since it is also one of the most cost effective.

Single-photon absorptiometry (SPA)

This technique, having been around the longest ("Bone density," 1997), has been used frequently in patients with osteoporosis due to a decrease in errors and increase in accuracy and precision (Markel & Chao,1993). Accuracy in vivo is around 9% and precision about 1 to 2% ("Bone density"). It is commonly used to measure BMD in the radius with accuracy of 3 to 5% (Goodwin, 1987).

The process of SPA is performed by submerging the body into water and determining a constant BMD in comparison with the water and soft tissue ("Bone density," 1997). However, this is only used to measure BMD of appendicular bones where there is uniform soft tissue and because the gamma ray is of low intensity (Markel & Chao, 1993).

Dual photon absorptiometry (DPA)

Like SPA, DPA is also used to measure BMD, but does not require submerging a body in water ("Bone density," 1997). It generally uses two gadolinium sources (Gd 153) to perform the measurements (Markel & Chao,

1993). As reported by Grove and Londeree (1992) it has a precision error less than 3% and prediction error of 4%. This method requires the radiation source to be replaced annually at a significant cost, however, DPA is being replaced by DEXA world-wide.

Summary

There are various factors that influence BMD. The availability of research studies in this area is increasing over time. The results seem to show that genetic and hormonal factors can be strongly influenced by environment, physical activity, and diet. The right combination of all risk factors could predispose a woman for low bone density and the possibility of osteoporosis after menopause. However, there seems to be enough evidence available to assume that lifestyle changes made at a young age may decrease the likelihood of bone fragility and disease.

It is important for young females to include physical activity in their daily routines along with adequate calcium supplies through their diets. As Whitney and Rolfes (1996) suggest, calcium supplementation should be used cautiously and only if diet modification is not feasible. Exercise prescription must also be detailed to the individual. The design of an exercise program must take into consideration the interests, current levels of activity, objectives, and compliance of the individual in order for positive results to prevail (Birge & Dalsky, 1989). Although there is not much control over genetic predisposition for osteoporosis, controlling the modifiable risk factors is a step in preventing disease after menopause.

REFERENCES

Alfredson, H., Nordstrom, P., & Lorentzon, R. (1997). Aerobic workout and bone mass in females. Scandinavian Journal of Medicine, Science and Sport, 7, 336-41.

Anderson, J.J.B., & Metz, J.A. (1993). Contributions of dietary calcium and physical activity to prevention of osteoporosis in females. Journal of the American College of Nutrition, 12, 378-383.

Ayalon, J.F., Cohn, S.H., Ostuni, J.A. Canc, R., & Ellisk. (1987). Dynamic bone loading exercises for postmenopausal women. Archives in Physical and Medical Rehabilitation, 68, 280-283.

Bassett, C.A.L. (1995). Why are the principals of physics and anatomy important in treating osteoporosis? Calcified Tissue International, 56, 515-516.

Beam, W.D. (1995). Developing and maintaining aerobic fitness. In Jordan, P. (Ed.), Fitness: Theory and Practice (pp. 450-456). Sherman Oaks, CA: Aerobics and Fitness Association of America & Reebok University Press.

Birge, S.J., & Dalsky, G. (1989). The role of exercise in preventing osteoporosis. Public Health Report, 104, 54-58.

Bone density measurement-- a systematic review. A report from SBU, the Swedish Council on Technology Assessment in health care. (1997). Journal of Internal Medicine, 739, (Suppl.), 1-60.

Bravo, G., Gauthier, P., Roy, P.M., Payette, H., & Gaulin, P. (1997). A weight-bearing, water based exercise program for osteopenic women: Its impact on bone, functional fitness, and well-being. Archives in Physical and Medical Rehabilitation, 78, 1375-1380.

Cann, C.E., Cavanaugh, D.J., & Schnurpfiel, K. (1988). Menstrual history is the primary determinant of trabecular bone density in women. Medicine in Science Sports and Exercise, 20, 59.

Crowley, L.V. (1997). The musculoskeletal system. In J.E. Burns (Eds.), Introduction tc human disease (pp.803-835). London: Jones and Bartlett.

Dalen, N., & Olsson, K.E. (1974). Bone mineral content and physical activity. Acta Orthopedics Scandanavian, 45, 170-174.

Drinkwater, B.L. (1996). Exercise and bones: Lessons learned from female athletes. The American Journal of Sports Medicine, 24, (Suppl. 6) 33-35.

Drinkwater, B.L. (1994). Does physical activity play a role in preventing osteoporosis? Research Quarterly for Exercise and Sport, 65, 197-206.

Drug prevents bone loss in healthy women. (1998, April). The Harvard Health Letter, 23, 5.

Duppe, H., Gardsell, P., Johnell, O., Nilsson, B.E., & Ringsberg, K. (1997). Bone mineral density, muscle strength, and physical activity. A population-based study of 332 subjects aged 15-42 years. Acta Orthopedic Scandinavian, 68, 97-103.

Ernst, E. (1994). Can exercise prevent postmenopausal osteoporosis? British Journal of Sports Medicine, 28 (1) 5-6.

Goodwin, P.N. (1987). Methodologies for the measurement of bone density and their precision and accuracy [Abstract]. Seminars in Nuclear Medicine, 4, 293.

Goto, S., Shigeta, H., Hyakutake, S., & Yamagata M. (1996). Comparison between menopause-related changes in bone mineral density of the lumbar spine and the proximal femur in Japanese female athletes: A long-term longitudinal study using dual-energy x-ray absorptiometry. Calcified Tissue International, 59, 461-465.

Grampp, S., Genant, H.K., Mathur, A., Lang, P., Jergas, M., Takada, M., Gluer, C.C., Lu, Y., & Chavez, M. (1997). Comparisons of noninvasive bone mineral measurements in assessing age-related loss, fracture discrimination, and diagnostic classification. Jounal of Bone and Mineral Research, 12, 697-711.

Gregory, C.A. (1982). Possible influence of physical activity on musculoskeletal symptoms of menopausal and postmenopausal women. Journal of Geriatric Nursing, (March/April), 103-107.

Grove, K.A., & Londeree, B.R. (1992). Bone density in postmenopausal women: High impact vs low impact exercise. Medicine and Science in Sports and Exercise, 24, 1190-1194.

Harris, S.S., & Dawson-Hughes, B. (1994). Caffeine and bone loss in healthy postmenopausal women. American Journal of Clinical Nutrition, 60, 573-578.

Heikkinen, J., Kurttila-Matero, E., Kyllonen, E., Vuori, J., Takala, T., & Vaananen, H.K. (1991). Moderate exercise does not enhance the positive effect of estrogen on bone mineral density in postmenopausal women. Calcified Tissue International, 49, (Suppl.), 83-S84.

Hoover, P.A., Webber, C.E., Beaumont, L.F., & Blake, J.M. (1996). Postmenopausal bone mineral density: Relationship to calcium intake, calcium absorption, residual estrogen, body composition, and physical activity. Canadian Journal of Physiological Pharmacology, 74, 911-917.

Kanis, J.A. (1996). Estrogens, the menopause, and osteoporosis. Bone, 19, (Suppl.), 185S-190S.

Kohrt, W.M., Ehsani, A.A., & Birge, S.J., Jr. (1997). Effects of exercise involving predominantly either joint-reaction or ground-reaction forces on bone mineral density in older women. Journal of Bone Mineral Research, 12, 1253-61.

Krolner, B., Toft, B., Nielsen, S.P., & Tondevold, E. (1983). Physical exercise as porphylaxis against involutional vertebral bone loss: A controlled trial. Clinical Science, 64, 541-546.

Love, R.R., Barden, H.S., Mazess, R.B., Epstein, S., & Chappell, R.J. (1994). Effect of tamoxifen on lumbar spine bone mineral density in postmenopausal women after five years. Archives in Internal Medicine, 154, 2585-2588.

Markel, M.D., & Chao, E.Y. (1993). Noninvasive monitoring techniques for quantitative description of callus mineral content and mechanical properties. Clinical Orthopaedics and Related Research, 293, 37-45.

Peterson, S.E., Peterson, M.D., Raymond, G., Gilligan, C., Checovich, M.M., & Smith, E.L. (1991). Muscular strength and bone density with weight training in middle-aged women. Medicine and Science in Sports and Exercise, 23, 499-504.

Pollitzer, W.S., & Anderson, 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.

Pruitt, L.A., Jackson, R.D., Bartels, R.L., & Lehnhard, H.J. (1992). Muscular strength and bone density with weight training in middle-aged women. Jounal of Bone and Mineral Research, 7, 179-185.

Salamone, I.M., Glynn, N.W., Black, D.M., Ferrell, R.E., Palermo, L., Epstein, R.S., Kuller, L.H., & Cauley, J.A. (1996). Determinants of premenopausal bone mineral density: The interplay of genetic and lifestyle factors. Journal of Bone Mineral Density, 11, 1557-1565.

Sinaki, M., Wahner, H.W., Bergstralh, E.J., Hodgson, S.F., Offord, K.P., Squires, R.W., Swee, R.G. & Kao, P.C. (1996). Three-year controlled, randomized trial of the effect of dose specified loading and strengthening exercises on bone mineral density of spine and femur in nonathletic, physically active women. Bone, 19, 233-244.

Stacey, T.A. (1989). Osteoporosis: Exercise therapy, pre and postdiagnosis. Journal of Manipulative Physiological Therapy, 12, 211-219.

Suleimen, S., Nelson, M., Ji, F., Buxton-Thomas, M., & Moniz, C. (1997). Effect of calcium intake and physical activity level on bone mass and turnover in healthy, white, postmenopausal women. American Journal of Clinical Nutrition, 66, 937-943.

Tsukahara, N., Toda, A., Goto, J., & Ezawa, I. (1993). Cross-sectional and longitudinal studies on the effect of water exercise in controlling bone loss in Japanese postmenopausal women. Journal of Nutritional Science and Vitaminology, 40, 37-47.

United States Department of Health and Human Services-Public Health Service. (1996). Healthy People 2000: National Health Promotion and Disease Prevention. Washington, DC: Author

Wahner, H.W., Dunn, W.L., Brown, M.L., Morin, R.L., & Riggs, B.L. (1988). Comparison of dual energy x-ray absorptiometry and dual photon absorptiometry for bone mineral measurements of the lumbar spine [Abstract]. Mayo Clinical Procedures, 11, 1075.

Whitney, E.N., & Rolfes, S.R. (1996). Osteoporosis and calcium. Understanding nutrition, (pp.462-471). St. Paul, MN: West.