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Original article

Risk factors and Prevalence of Osteoporosis amidst Postmenopausal females turning up the Diabetes and Endocrinology clinic at Azadi Teaching Hospital at Kirkuk /Iraq

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Abstract:

- ***Background*:** Osteoporosis is a significant health concern in postmenopausal women, leading to increased morbidity. This study aimed to estimate the prevalence of osteoporosis and osteopenia and to identify associated risk factors among Iraqi postmenopausal females attending a specialized clinic.
- ***Methods:*** A descriptive cross-sectional study was conducted at the Diabetes and Endocrinology Clinic of Azadi Teaching Hospital in Kirkuk, Iraq, from April 2019 to April 2021. A total of 1085 postmenopausal women aged 45 to 84 years were included. Bone mineral density (BMD) was measured using dual-energy X-ray absorptiometry (DEXA), and results were interpreted using WHO criteria for T scores.
- ***Result:*** The prevalence of osteoporosis and osteopenia was 37.5% and 44.6%, respectively. Osteoporosis was most commonly observed in the lumbar spine (32.4%) and left femoral neck (14.4%). Osteopenia was most prevalent in the left femoral neck (56.1%) and lumbar spine (41.3%). Significant risk factors associated with osteoporosis included extended duration of menopause, high parity, physical inactivity, overweight or normal BMI, positive family history, poor sun exposure, high daily caffeine intake, low calcium intake, and late menarche. Interestingly, women with type 2 diabetes had a lower prevalence of osteoporosis.
- ***Conclusions:*** Osteoporosis and osteopenia are highly prevalent among Iraqi postmenopausal women. Increased public education and preventive strategies are essential to reduce the burden of this disease.

- **Keywords:** Prevalence, osteoporosis, osteopenia, type 2 diabetes mellitus

INTRODUCTION

Osteoporosis is a significant global health concern, responsible for more than 8.9 million fractures annually equivalent to one osteoporotic fracture every three seconds (1). Women are at a particularly increased risk, with an estimated 200 million affected worldwide (2). Shilbayeh reported that the general prevalence of osteoporosis among women in Jordan was 30%, increasing to 43.3% in postmenopausal women (3). Moreover, factors such as advancing age, hypertension, diabetes mellitus, renal disease, and age at menarche have been associated with an elevated risk of developing osteoporosis (4).

During their lifetime, approximately half of all postmenopausal women will experience an osteoporosis-related fracture. Among these, 25% will develop vertebral deformities and 15% will suffer a hip fracture (5). Notably, women represent about 75% of all hip fractures, which are frequently accompanied by long-term consequences such as chronic pain, reduced mobility, dependency, and the risk of subsequent fractures. Furthermore, hip fractures due to osteoporosis are linked with increased mortality, with up to 20–25% of affected individuals dying within the first year post-fracture.

Several risk factors contribute to the development of osteoporosis, including female sex, age, ethnicity, smoking, family history of osteoporosis, vitamin D deficiency, low calcium intake, high caffeine consumption, physical inactivity, sedentary lifestyle, late menarche, early menopause, and low body mass index (BMI) (7,8).

The relationship between osteoporosis and diabetes mellitus is complex. Type 1 diabetes mellitus (T1DM) is associated with reduced bone mineral density (BMD) due to low levels of insulin and insulin-like growth factor 1 (IGF-1), particularly before peak bone mass is reached. On the other hand, type 2 diabetes mellitus (T2DM), more prevalent in older adults who have typically achieved peak bone mass, may have a variable impact on BMD—ranging from reduced, unchanged, to even increased density in some cases (9,10). This study aims to estimate the prevalence of osteoporosis and osteopenia among postmenopausal women attending a Diabetes and Endocrinology clinic, and to identify the associated risk factors.

PATIENT and METHOD

A cross-sectional study was conducted at the Diabetes and Endocrinology Clinic of Azadi Teaching Hospital in Kirkuk, Iraq, from April 2020 to April 2021. The study enrolled postmenopausal women aged over 45 years who had been menopausal for more than one year and underwent bone mineral density (BMD) evaluation using dual-energy X-ray absorptiometry (DEXA) at the clinic during the study period. Women were excluded if they had type 1 diabetes mellitus (T1DM), malignancy, uncontrolled hypothyroidism or hyperthyroidism, primary or secondary hyperparathyroidism, rheumatoid arthritis, systemic lupus erythematosus (SLE), epilepsy, renal failure, Cushing's syndrome,

cirrhosis, premature menopause (menopause before the age of 45), or were on medications known to affect bone metabolism such as warfarin, heparin, or thiazolidinediones.

Data collection included information from both patient medical records and structured interviews conducted face-to-face using a validated questionnaire. Variables collected included current age, marital status, educational level, duration of menopause, age at menopause and menarche, parity, number of pregnancies and abortions, history of breastfeeding, total years of menstruation, presence and duration of diabetes and hypertension, use of vitamin D3 supplements or statins, and anthropometric measurements (weight and height).

Ethical approval was granted by the Ethics Committee of the Diabetic and Endocrinology Clinic. Verbal informed consent was obtained from all participants following assurances of confidentiality and that data would be used strictly for scientific purposes.

Body mass index (BMI) was calculated by dividing weight in kilograms by height in meters squared (kg/m^2) and classified based on the World Health Organization (WHO) recommendations adopted by the American Diabetes Association (ADA) (11). Smoking status was defined according to WHO guidelines (12), categorizing participants as current smokers, past smokers, or nonsmokers.

Physical activity was defined as participation in moderate-intensity aerobic exercise for at least 30 minutes on five days per week, while immobilization referred to individuals bedridden for more than two months or those with restricted movement (14). Diabetes and prediabetes were diagnosed according to ADA 2014 criteria. Glycemic control was classified as good if HbA1c was below 7.0%, moderate if between 7.0–7.9%, and poor if ≥8.0%.

Sun exposure was defined as at least 10–15 minutes of daily sunlight exposure to the face, arms, or hands without sunscreen. Caffeine intake was calculated by summing daily consumption of coffee (instant, brewed, or Turkish), tea, cola, iced coffee, and chocolate, with caffeine content approximated using data from the International Food Information Council Foundation. Based on these estimates, individuals were categorized as having low (≤300 mg/day) or high (>300 mg/day) lifetime daily caffeine intake (15–18).

Menopause duration was determined by subtracting the age at menopause from the age at BMD assessment. Years of menstruation were calculated as the difference between age at menopause and age at menarche.

Bone mineral density was assessed using the Hologic Discovery A DEXA scanner at the lumbar spine (L1–L4) and left femoral neck. BMD was expressed in g/cm^2, and T-scores were used for classification per WHO criteria (19): normal (T-score ≥ -1.0), osteopenia (T-score between -1.0 and -2.5), and osteoporosis (T-score ≤ -2.5). Vitamin D3 levels were measured using a radioimmunoassay method (BIOSOURCE Europe S.A., Nivelles, Belgium) and categorized as deficient (<20 ng/mL), insufficient (20–29 ng/mL), or sufficient (≥30 ng/mL).

Statistical analysis was performed using SPSS version 20.0. All data were screened for entry errors and outliers. Descriptive statistics were calculated to estimate the prevalence of osteoporosis and osteopenia. The Chi-square and t-tests were applied to explore associations between osteoporosis and potential risk factors. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 1,085 postmenopausal women aged between 45 and 85 years participated in the study. The mean age of participants was 61.1 years with a standard deviation of 7.2 years. The study collected comprehensive data on family history of osteoporosis and fragility fractures, personal history of fragility fractures, lifestyle patterns, and various sociodemographic, reproductive, and clinical characteristics.

Table 1. Frequency Distribution of Osteoporosis-Related Variables

Variable	Number (%)
Running age at BMD (years)	
<60	491 (45.20)
≥60	594 (54.80)
Matrimonial state	
Celibate	88 (8.00)
Wedded	976 (90.30)
Divorced or widowed	21 (1.80)
Instructional level	
Basic instruction	299 (27.50)
Intermediate instruction	223 (20.50)
Advanced instruction	563 (52.00)
BMI (kg/m²)	
Normal	98 (8.70)
Overweight	325 (29.70)
Obese	662 (61.60)
Fumigation	
Not fumigate	925 (85.40)
Running fumigate	160 (14.60)
Corporal activity	
Yes	436 (40.10)
No	649 (59.90)
Exposition to sun	
Yes	515 (47.50)
No	570 (52.50)
Lifetime diurnal caffeine (mg/day)	
≤300	518 (47.70)
>300	567 (52.30)
Ingestion of calcium	
<600	335 (30.90)
600-1000	478 (44.10)

>1000	272 (25.00)
Presence of diabetes mellitus	
No DM	147 (14.40)
Pre-DM	406 (37.70)
DM	532 (49.10)
Duration of diabetes mellitus	
<5 years	176 (34.70)
5-9 years	131 (25.70)
≥10 years	201 (39.60)
Statin utilization	
Yes	736 (67.90)
No	349 (32.10)
Presence of hypertension	
Yes	696 (64.10)
No	389 (35.80)
Vitamin D3 usage	
Yes	967 (89.30)
No	118 (10.70)
Solidification	
Yes	31 (2.60)
No	1054 (97.40)
HbA1c	
Good Controlled (<7)	262 (49.30)
Fairly controlled (7-7.9)	142 (26.60)
Poor controlled (≥8)	129 (24.10)
Parathyroid hormone (pg/ml)	
≤55	515 (66.30)
>55	263 (33.70)
Vitamin D3 level (ng/ml)	
Normal (≥30)	695 (61.80)
Insufficiency (20-29)	185 (18.10)
Deficiency (<20)	205 (20.10)
Age at menopause (years)	
≤50	670 (61.80)
>50	415 (38.20)
Menopausal duration when DXA carried out	
≤5 years	285 (26.20)
6-10 years	270 (24.80)
≥11 years	530 (48.90)
Age at menarche (year) (Mean±SD)	13.71+/-1.58
Years of menstruation (Mean±SD)	36.12+/-3.58
Number of parities	
≤2	212 (19.50)
3-5	466 (43.00)
≥6	407 (37.50)
Number of abortions	

None	540 (49.90)
1-2	420 (38.70)
≥3	125 (11.40)
Breast feeding	
Yes	857 (79.10)
No	228 (20.90)
Family history of osteoporosis	
Negative	742 (68.50)
Positive	343 (31.50)
Family history of fragility fracture	
Negative	820 (75.70)
Positive	265 (24.30)
Individual history previous fragility fracture	
Negative	877 (81.00)
Positive	208 (19.00)

As shown in Table 2, the prevalence of osteoporosis was highest at the lumbar spine, where 32.4% of participants were diagnosed, followed by the left femoral neck at 14.4%. In contrast, osteopenia was more prevalent at the left femoral neck (56.1%), followed by the lumbar spine (41.3%).

Table 2. Prevalence of Osteopenia and Osteoporosis Amidst Postmenopausal Females

(No. of cases 1085)	Normal (%)	Osteopenia (%)	Osteoporosis (%)
Total	17.89	44.58	37.53
Lumbar spine	26.2	41.3	32.4
Femoral neck	29.6	56.1	14.4

To assess the distribution of osteoporosis in relation to specific study variables, Chi-square statistical analysis was applied. The associations between osteoporosis and different sociodemographic, lifestyle, and reproductive factors are summarized in Table 3.

Table 3. Chi-Square Distribution and Degree of Prominence of Osteoporosis via Sociodemographic and Health Variables

Variable	Normal BMD (n, %)	Osteoporosis (n, %)	P-Value
Running age at BMD (years)			**< 0.001**
<60	343 (69.9)	149 (30.1)	
≥ 60	334 (56.3)	259 (43.7)	
Marital status			**0.002**

Single	42 (47.7)	46 (52.3)	
Married	626 (64.2)	350 (35.8)	
Divorced or widowed	9 (42.1)	12 (57.9)	
Education level			0.089
<secondary school	171 (57.2)	128 (42.8)	
Secondary school	142 (63.8)	81 (36.2)	
>secondary school	364 (64.6)	199 (35.3)	
BMI (kg/m²)			**< 0.001**
Normal	43 (44.7)	53 (55.3)	
Overweight	167 (51.7)	156 (48.3)	
Obese	462 (70.1)	197 (29.9)	
Smoking			0.261
Never smoke	582 (62.9)	344 (37.1)	
Current smoke	95 (59.9)	64 (40.1)	
Physical activity			**< 0.001**
No	369 (56.8)	281 (43.2)	
Yes	309 (70.9)	128 (29.1)	
Sun exposure			**< 0.001**
No	323 (56.6)	248 (43.4)	
Yes	354 (68.9)	160 (31.1)	
Life-time diurnal caffeine (mg/day)			**0.015**
≤3000	433 (66.2)	175 (33.8)	
>3000	334 (59)	232 (41)	
Ingestion of calcium			**0.013**
<600	188 (56.2)	147 (43.8)	
600-1000	307 (64.3)	171 (35.7)	
>1000	182 (67.0)	90 (33.0)	
Diabetic status			**0.003**
No DM	76 (51.7)	61 (48.3)	
Pre - DM	247 (60.9)	159 (39.1)	
DM	354 (66.6)	178 (33.4)	
Duration of diabetes (years)			**0.045**
≤5	130 (74.1)	46 (25.9)	
5-9	85 (65.1)	46 (34.9)	
≥10	125 (62.3)	76(37.7)	
Use of statin			0.337
No	211 (60.4)	139 (39.6)	
Yes	466 (63.4)	269 (36.6)	
Hypertension			0.295
No	235 (60.4)	155 (39.6)	
Yes	466 (63.4)	253 (36.6)	
Use of vitamin D3			0.325
No	69 (58.3)	50 (41.7)	

Yes	608 (63.0)	358 (37.0)	
Immunosibilization			0.556
No	660 (62.6)	395 (37.4)	
Yes	17 (57.1)	13 (42.9)	
HbA1C			0.403
Good Control (<7)	131 (66.5)	88 (33.5)	
Fairly control (7-7.9)	91 (70.0)	43 (30.0)	
Poorly controlled (≥8)	80 (62.2)	49 (37.8)	
Parathyroid hormone (ng/ml)			**0.038**
≤55	327 (63.5)	189 (36.5)	
>55	146 (55.8)	116 (44.2)	
Vitamin D3 level (ng/ml)			0.279
Normal (≥30)	378 (60.4)	248 (39.6)	
Insufficiency (20–29)	117 (63.4)	68 (36.6)	
Deficiency (<20)	136 (66.5)	69 (33.5)	
Age at menopause (years)			0.390
≤50	412 (61.5)	259 (38.5)	
>50	265 (64.1)	149 (35.9)	
Menopausal duration at time of DXA performing			< **0.001**
≤5	212 (74.6)	73 (25.4)	
6–10	167 (61.9)	103 (38.1)	
≥11	280 (56.2)	232 (43.8)	
Parity			0.052
≤2	126 (59.5)	86 (40.5)	
3–5	310 (66.6)	156 (33.4)	
≥6	241 (59.3)	166 (40.7)	
Abortion			0.752
None	343 (63.6)	197 (36.4)	
1–2	257 (61.2)	163 (38.8)	
≥3	77 (61.8)	48 (38.2)	
Breast feeding			0.186
No	134 (58.7)	95 (41.3)	
Yes	543 (63.5)	313 (36.5)	
Family history of osteoporosis			< **0.001**
No	494 (66.6)	249 (33.4)	
Yes	183 (53.5)	159 (46.5)	
Family history of fragility fracture			< **0.001**

No	540 (65.9)	281 (34.1)	
Yes	137 (51.9)	127 (48.1)	
Personal history of prior fragility fracture			**0.001**
No	569 (64.9)	309 (35.1)	
Yes	108 (52.2)	99 (47.8)	

DISCUSSION

The prevalence of osteoporosis among postmenopausal women attending the Diabetes and Endocrinology Clinic was found to be 37.5% in this study. This rate is higher than the average prevalence reported in Turkey (16.2%) (21), similar to the rate observed in India (37.8%) (22), and lower than that reported in the Kingdom of Saudi Arabia (44.1%) (23). Such differences may be attributed to variations in study design, diagnostic methods, bone scan sites, lifestyle habits, and participant selection.

Our results revealed that non-diabetic and pre-diabetic women were more likely to have osteoporosis than those with type 2 diabetes. This finding aligns with other studies (24–26) but contrasts with some that found no such association (27,28). Even after adjusting for other variables, the number of years since menopause remained a significant predictor of osteoporosis, a conclusion supported by prior literature (21,29,30).

This study also identified an association between early menarche and a reduced risk of osteoporosis. Earlier menarche is linked to prolonged exposure to estrogen, which may protect against bone loss and is correlated with higher BMD (31,32). However, other studies have not found a significant relationship between menarche age and BMD or fracture risk (33–35).

Our findings indicate that higher parity is a risk factor for osteoporosis, which agrees with several studies (36,37). Conversely, Sadat-Ali et al. reported a protective effect of increased parity against osteoporosis (23). We also found that obesity had a protective effect on bone health. A higher BMI may counteract the negative effects of hypoestrogenism post-menopause. This is supported by other research suggesting that increased body weight enhances bone density and reduces fracture risk (38,39). The proposed mechanisms include the mechanical loading effect of higher body mass and the estrogen production by adipose tissue. However, some reports suggest that fat mass does not positively affect bone health, contradicting our findings (40).

In this study, postmenopausal women with caffeine intake exceeding 300 mg/day were more likely to have osteoporosis. This supports the findings by Rapuri et al., who noted increased spinal bone loss with high caffeine consumption in elderly postmenopausal women (18). However, some research has shown no significant or even protective effects of coffee on bone health (41). Experimental studies have demonstrated that caffeine may negatively affect osteoblast and osteocyte function and survival (42–

45), while promoting osteoclast activity and increasing urinary calcium loss (46), contributing to reduced BMD.

Adequate calcium intake is crucial for bone health. Low calcium intake (<600 mg/day) was significantly associated with osteoporosis in our study. In agreement, Ensrud et al. reported that poor calcium absorption and low intake elevate hip fracture risk in older women (53). Although many patients in our study took vitamin D supplements, low sun exposure was still associated with osteoporosis. Due to widespread supplementation, we could not adequately assess the impact of vitamin D deficiency. Other studies have shown low vitamin D levels as a major risk factor for osteoporosis in postmenopausal women (54,55).

Our results demonstrated a significant association between physical inactivity and osteoporosis, underlining the importance of exercise in maintaining bone mass and preventing falls. Regular physical activity improves muscle strength, delays sarcopenia, and supports neuromuscular coordination, all of which are essential for balance and fall prevention. Moreover, mechanical loading from exercise directly stimulates bone formation and improves BMD (56).

No significant association was found between smoking and osteoporosis in our study, a finding echoed by Young et al. (57). However, other studies have reported a significant association between cigarette smoking and increased osteoporosis risk (58,59).

Finally, our findings showed a clear correlation between a family history of osteoporosis and increased risk of the condition. This concurs with previous studies identifying family history as an independent risk factor for osteoporosis (60–62).

The study has a few limitations. Firstly, the sample was drawn from a population of postmenopausal women referred for DEXA screening, which may introduce selection bias and potentially affect the generalizability of the findings. However, this was partially mitigated by strict exclusion criteria, which ruled out individuals with chronic diseases and those on medications known to influence bone density. Secondly, much of the data was obtained from the center's medical and radiological records, which may limit the completeness of certain variables.

CONCLUSION

significant prevalence of both osteoporosis and osteopenia among postmenopausal women in Kirkuk. These findings emphasize the need for broader public health education and awareness campaigns to enhance knowledge about osteoporosis, its associated risk factors, and effective prevention strategies.

Ethical Clearance:

All ethical aspects of this study were approved. Before enrolling the participants, an informed oral consent was obtained from their families as an ethical agreement. Additionally, approval from the hospital administrator was obtained.

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References

1. Johnell O, Kanis JA. An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. *Osteoporos Int*. 2006;17:1726–33.
2. Kanis JA. WHO Technical Report. University of Sheffield, United Kingdom; 2007.
3. Shilbayeh S. Prevalence of osteoporosis and its reproductive risk factors among Jordanian women: a cross-sectional study. *Osteoporos Int*. 2003;14:929–40.
4. El-Heis MA, Al-Kamil EA, Kheirallah KA, et al. Factors associated with osteoporosis among a sample of Jordanian women referred for investigation. *East Mediterr Health J*. 2013;19:459.
5. Chon KS, Sartoris DJ, Brown SA, et al. Alcoholism-associated spinal and femoral bone loss in abstinent male alcoholics. *Skeletal Radiol*. 1992;21:431–6.
6. Jordan KM, Cooper C. Epidemiology of osteoporosis. *Best Pract Res Clin Rheumatol*. 2002;16:795.
7. Leibson CL, Tosteson AN, Gabriel SE, et al. Mortality, disability, and nursing home use with and without hip fracture. *J Am Geriatr Soc*. 2002;50:1644–50.
8. Abushaikha L, Omran S. A survey of osteoporosis risk factors and practices among Jordanian women. *J Int Womens Stud*. 2013;11:153–61.
9. Wongdee K, Charoenphandhu N. Osteoporosis in diabetes mellitus: possible cellular and molecular mechanisms. *World J Diabetes*. 2011;2:41–8.
10. Adil C, Aydın T, Taşpınar Ö, et al. Bone mineral density evaluation in type 2 diabetic patients. *J Phys Ther Sci*. 2015;27:179.
11. American Diabetes Association. Standards of medical care in diabetes 2011. *Diabetes Care*. 2011;30(Suppl 1):S4–40.
12. World Health Organization. Guidelines for Controlling and Monitoring the Tobacco Epidemic. Geneva: WHO; 1998.
13. World Health Organization. Global Recommendations on Physical Activity for Health. Geneva: WHO; 2010.
14. World Health Organization. Physical Status: The Use and Interpretation of Anthropometry. Geneva: WHO; 1995.
15. International Osteoporosis Foundation. Sources of Vitamin D. 2008. Available from: http://iofbonehealth.org
16. International Food Information Council. IFIC Review: Caffeine and Health. 2014. Available from: www.foodinsight.org
17. Afifi S, Rahahleh WA, Hadidi KA. Caffeine content in Turkish coffee. *Dirasat: Educ Sci*. 2008;35:730–8.
18. Rapuri PB, Gallagher JC, Kinyamu HK, et al. Caffeine intake increases bone loss in elderly women. *Am J Clin Nutr*. 2001;74:694–700.

19. World Health Organization. Research on the Menopause in the 1990s. Geneva: WHO; 1996.
20. Holick MF. Vitamin D deficiency. *N Engl J Med*. 2007;357:266–81.
21. Demir B, Haberal A, Geyik P, et al. Risk factors for osteoporosis among postmenopausal women. *Maturitas*. 2008;60:253–6.
22. Waliullah S, Sharma V, Srivastava R, et al. Prevalence of postmenopausal osteoporosis in Indian females. *Int J Health Sci Res*. 2014;4:113–7.
23. Sadat-Ali M, Al-Habdan IM, Al-Mulhim AA, et al. Bone mineral density in postmenopausal Saudi women. *Saudi Med J*. 2004;25:1623–5.
24. Harifi F, Ahmadimoghadam N, Mousavinasab N. T2DM and bone density in postmenopausal women. *Int J Endocrinol Metab*. 2006;4:117–22.
25. Anaforoğlu I, Nar-Demirer A, Başçıl-Tütüncü N, et al. Osteoporosis in Turkish postmenopausal diabetics. *J Diabetes Complications*. 2009;23:12–7.
26. Van Daele PL, Stolk RP, Burger H, et al. Bone density in non-insulin-dependent diabetes mellitus. *Ann Intern Med*. 1995;122:409–14.
27. Al-Maatouq MA, El-Desouki MI, Othman SA, et al. Osteoporosis in postmenopausal diabetics. *Saudi Med J*. 2004;25:1423–6.
28. Moghimi N, Rahimi E, Derakhshan S, et al. Osteoporosis in diabetic postmenopausal women. *Iran J Nucl Med*. 2008;16:28–33.
29. Sharami SH, Milani F, Alizadeh A, et al. Risk factors for osteoporosis in women over 50. *J Turk Ger Gynecol Assoc*. 2008;9:38–44.
30. D'Amelio P, Spertino E, Martino F, et al. Postmenopausal osteoporosis in Italy. *Calcif Tissue Int*. 2013;92:437–43.
31. Ito M, Yamada M, Hayashi K, et al. Early menarche and high BMD. *Calcif Tissue Int*. 1995;57:11–4.
32. Parker SE, Troisi R, Wise LA, et al. Menstrual and menopausal history and osteoporosis. *J Clin Endocrinol Metab*. 2013;99:594–601.
33. Gerdhem P, Obrant KJ. Menarche, menopause and BMD in old age. *J Bone Miner Metab*. 2004;22:372–5.
34. Sioka C, Fotopoulos A, Georgiou A, et al. Reproductive factors and osteoporosis risk. *Climacteric*. 2010;13:63–71.
35. Paganini-Hill A, Atchison KA, Gornbein JA, et al. Reproductive history and fracture risk. *J Womens Health*. 2005;14:808–19.
36. Allali F, Maaroufi H, El Aichaoui S, et al. Parity and BMD in Moroccan women. *Maturitas*. 2007;57:392–8.
37. Gur A, Nas K, Cevik R, et al. Number of pregnancies and BMD in postmenopausal women. *J Bone Miner Metab*. 2003;21:234–41.
38. Harris SS, Dawson-Hughes B. Body composition and BMD in postmenopausal women. *Calcif Tissue Int*. 1996;59:428–32.
39. Pruzansky ME, Turano M, Luckey M, et al. Low body weight and hip fracture risk. *J Orthop Res*. 1989;7:192–7.

40. Zhao LJ, Liu YJ, Liu PY, et al. Obesity and osteoporosis. *J Clin Endocrinol Metab*. 2007;92:1640–6.
41. Ng N, Kaye EK, Garcia RI. Coffee and periodontal disease. *J Periodontol*. 2014;85:1042–9.
42. Su SJ, Chang KL, Su SH, et al. Caffeine and osteogenic differentiation. *Int J Food Sci Nutr*. 2013;64:429–36.
43. Macedo RM, et al. Bone graft and caffeine in osteointegration. *Implant Dent*. 2011;20:369–73.
44. Lu PZ, Lai CY, Chan WH. Caffeine induces apoptosis in osteoblasts. *Int J Mol Sci*. 2008;9:698–718.
45. Liu SH, et al. Caffeine enhances osteoclast differentiation. *J Orthop Res*. 2011;29:954–60.
46. Lacerda SA, Matuoka RI, Macedo RM, et al. Daily coffee intake and bone quality. *Braz Dent J*. 2010;21:199–204.
47. Krall EA, Dawson-Hughes B. Determinants of BMD. *J Bone Miner Res*. 1993;8:1–9.
48. Ross AC, Taylor CL, Yaktine AL, et al. Dietary Reference Intakes: Calcium, Vitamin D. Washington, DC: National Academies Press; 2011.
49. National Institutes of Health. Optimal Calcium Intake. Bethesda: NIH; 1994.
50. Shea BJ, Adachi JD, Cranney A, et al. Calcium supplements for postmenopausal women. *Cochrane Database Syst Rev*. 2004;CD004526.
51. Allain TJ, Dhesi JK. Hypovitaminosis D in older adults. *Gerontology*. 2003;49:273–8.
52. Parfitt AM, Gallagher JC, Heaney RP, et al. Vitamin D and bone health in the elderly. *Am J Clin Nutr*. 1982;36:1014–31.
53. Ensrud KE, Duong TU, Cauley JA, et al. Low calcium absorption and hip fracture risk. *Ann Intern Med*. 2000;132:345–53.
54. Gaugris S, Heaney R, Boonen S, et al. Vitamin D inadequacy in postmenopausal women. *QJM*. 2005;98:667–76.
55. Mezquita-Raya P, Muñoz-Torres M, Luna JD, et al. Vitamin D insufficiency and bone density. *J Bone Miner Res*. 2001;16:1408–15.
56. Omland LM, Tell GS, Ofjord S, et al. Risk factors for low BMD in Norwegian women. *Eur J Epidemiol*. 2000;16:223–9.
57. Young D, Hopper JL, Nowson CA, et al. Determinants of bone mass in females. *J Bone Miner Res*. 1995;10:558–67.
58. Benson BW, Shulman JD. Tobacco exposure and bone content. *Nicotine Tob Res*. 2005;7:719–24.
59. Krall EA, Dawson-Hughes B. Smoking and calcium absorption. *J Bone Miner Res*. 1999;14:215–20.
60. Seeman E, Hopper JL, Bach LA, et al. Bone mass in daughters of osteoporotic women. *N Engl J Med*. 1989;320:554–8.
61. Soroko SB, Barrett-Connor E, Edelstein SL, et al. Family history and BMD. *J Bone Miner Res*. 1994;9:761–9.
62. Robitaille J, Yoon PW, Moore CA, et al. Osteoporosis prevalence and family history. *Am J Prev Med*. 2008;35:47–54.