The Effect of Behavioural Risk Factors on Osteoporosis in Irish Women

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Abstract

Background Osteoporosis constitutes a major public health concern and its underlying pathogenesis is complex and multifactorial. Although hereditary factors strongly contribute to bone health, behavioural factors can modulate the genetically determined pattern of skeletal modelling and remodelling.

Aims The aim of this study was to investigate the effect(s) of behavioural risk factors on osteoporosis in Irish women.

Methods Pre- and post-menopausal adult women (n = 189; 44 ± 15y) participated in this cross-sectional study. Demographic, anthropometric and lifestyle data were collected during a single clinic visit. Dietary calcium intake and lifetime physical activity (PA) was assessed for each subject. Lumbar and femoral bone mineral density (BMD) was measured by dual energy X-ray absorptiometry (DXA). Multivariate analysis was used to determine the independent predictors of low BMD.

Results Low BMD was present in 59% of subjects (42% pre- and 77% post-menopausal). Smoking was the strongest behavioural predictor of lumbar and femoral BMD. Age, height, family history, smoking, metabolic (MET) and mechanical (MECH) PA (lifetime) and weight (body mass) accounted for 39% of the variance in lumbar BMD. Age, height, family history, alcohol consumption, MET and MECH PA (lifetime) and weight accounted for 41% of the variance in femoral BMD.

Conclusions Prevalence of osteopenia and osteoporosis is high in Irish women and is associated with modifiable risk factors. A clearer focus should be paid to educating Irish women on preventative health behaviours for osteoporosis in order to curb the prevalence of this disease and the human and fiscal costs associated with it.

Keywords

Behavioural risk factors. Osteoporosis, Prevalence, Prevention, Ireland

Introduction

Osteoporosis constitutes a major public health concern and, with the demographic trend towards an older population, it represents a significant challenge for health care systems in the future. Osteoporotic fractures, particularly of the hip, are associated with high mortality rates, loss of independence [1] and a considerable financial burden on economies. Throughout the European Union the burden of osteoporotic fractures on healthcare budgets is €36 billion and is greater than for breast cancer, prostate cancer or myocardial infarction [2]. Since 1990 there has been an almost four-fold increase in hip fractures alone [3] and it is predicted that the current incidence rate of hip fractures in Ireland will double by 2026 [4]. This rise cannot solely be attributed to demographics; it is probable that modification in behavioural risk factors is partly culpable. The underlying pathogenesis of osteoporosis is complex and multifactorial; however, if we are to address this *silent epidemic* at a public health level, it is critically important to identify the key determinants of fragility fractures.

Osteoporosis is a direct consequence of insufficient bone mineral accrual in youth (unattained Peak Bone Mass (PBM)) and/or excessive bone loss in later years. It is worth considering that while osteoporosis is clinically considered an age related condition, the antecedents of low bone mineral and skeletal fragility in the elderly may begin during the first two decades of life. Although genetic factors contribute strongly to PBM [5] and the rate of bone loss [6], environmental factors modulate the genetically determined pattern of skeletal modelling and remodelling.

Body weight (mass) impacts on bone turnover and bone density and low body mass index (BMI) is a significant risk factor for fracture even after adjustment of fracture risk by bone mineral density (BMD). When compared with a healthy BMI of 25 kg/m^2 a BMI of 20 kg/m^2 is associated with a nearly 2-fold increase in risk ratio (RR=1.95, 95% CI 1.71-1.22) for hip fracture [7]. An inverse relationship between smoking and BMD has been reported and many factors are believed to contribute to this including reduced body weight, an earlier menopause, and increased metabolic breakdown of exogenous oestrogen in women [8]. Excessive intake of alcohol can

also have adverse effects on skeletal health; the hip fracture risk ratio for an alcohol intake of >2units daily is 1.68 (95% CI 1.19-2.36) [9].

Calcium intake is one of the main determinants of the development of PBM during adolescence and also slows subsequent age-related bone loss [10]. There may also be a permissive action of calcium enhancing the effect of physical activity (PA) on BMD. PA is reported to increase PBM and may delay or prevent age-related bone loss [11, 12]. Bone remodelling is governed at a local level by mechanical stimuli and at a systemic level by metabolic stimuli; PA has the unique ability to activate the basic multicellular unit (BMU) via mechanical and metabolic stimuli. In this respect the mode, intensity, frequency and duration of activity is relevant, with dynamic weight-bearing activity being the most effective form of exercise for the skeleton [13]. For example, in a 15year follow-up study of physical activity in young males and females weight-bearing ("peak strain") physical activity during adolescence and adulthood was a significant predictor of lumbar BMD at age 27 [14].

The most convincing evidence for a causal relationship between PA and BMD comes from intervention studies. However, because a chronic disease such as osteoporosis tends to have a long-term developmental period, in order to determine the relationship between PA and BMD, it is important to assess *lifetime* physical activity patterns. PA may play different roles throughout the lifespan by maximising bone mass during childhood and adolescence, maintaining bone mass during adulthood, attenuating bone loss with aging and reducing falls and fractures in the elderly. To date, however, few studies have evaluated total lifetime occupational and leisure activity in cohorts of differing ages. Occupational activity should be considered in relation to skeletal health as sedentary work has been reported to be a risk factor for low BMD [15]. Therefore, the relationship between *lifetime* PA and BMD has yet to be fully determined.

Although it is now known that behavioural factors such as physical activity and calcium intake play a significant role in the development of BMD and therefore in the prevention of osteoporosis, the magnitude of the influence has not yet been fully established. Also, these factors have not previously been examined in a large-scale study of different cohorts in Ireland. Therefore the aim of this study is to investigate

the effect(s) of behavioural and unmodifiable factors on skeletal health as measured by BMD in Irish pre- and post-menopausal females. A further aim of the study was to determine the prevalence of low bone mass among this cohort.

Methods

Study Design

This cross-sectional, multicentre study was reviewed and approved by the Research Ethics Committees at the University of Limerick and Waterford Institute of Technology, Ireland and therefore was conducted in accordance with the 1964 Declaration of Helsinki. Female subjects were recruited by posters, email and word of mouth at both campuses and surrounding areas. Written informed consent was obtained from all subjects prior to study entry. Post-menopausal status was defined by duration of greater than 18 months post menopause.

Medical History

All participants were administered detailed questionnaires to obtain information on medical history which were completed with the assistance of trained personnel. The questionnaire was given to ascertain whether there were any histories of diseases relating to skeletal health and in pre-menopausal females abnormalities in menstruation that would affect BMD. Information was also collected on medications, fracture history, family history of osteoporosis, smoking status and alcohol consumption (units/wk).

Anthropometric Measures

Height and body weight measurements were taken by a trained laboratory technician. Standing height was measured to the nearest centimetre using a portable stadiometer. Body weight was measured to the nearest 0.1kg using an electronic balance. Prior to testing, the scale was checked for accuracy using known weights. For both of these measurements subjects wore light indoor clothing and no shoes. Body mass index (BMI) was calculated as weight divided by the square of height (kg/m^2).

Bone Densitometry

Bone mineral density measurements were performed by means of Dual-energy X-ray Absorptiometry (DXA) at the lumbar spine (L2-L4) and femoral neck. Quality assurance on the Excell™ DXA scanner was carried out by daily calibration against the manufacturer's standard phantoms *(Norland Medical Systems, NY, USA).* In-house precision tests have shown that the coefficient of variation is <2% for both sites which is in keeping with the degree of precision of the Norland device.

Calcium Intake

Current dietary calcium intake was measured by a food frequency questionnaire. This was an adaptation of the questionnaire used by the University of Cambridge in the European Prospective Investigation of Cancer (EPIC) study [16]. The study group were asked the frequency of consumption and the average portion size of common foods in the Irish diet with high calcium content. A photographic atlas and common household measures (e.g. cups, glasses, bowls) were used to assist in portion size estimations. The questionnaire was designed to estimate intakes of dairy foods, breads, cereals, fish, eggs, vegetables and snacks. Calcium intake was adjusted accordingly for those subjects on dietary supplements. Calcium intake from foods and beverages was calculated using a computerised dietary analysis program based on UK food composition tables (Comp-Eat, Nutrition Systems, Grantham, UK).

Physical Activity Assessment

The physical activity (PA) questionnaire was adapted from that of Kriska et al [17]. The PA questionnaire was self-administered and completed under the guidance of trained personnel. The test-retest reliability of this modified questionnaire was found to be acceptable (r=0.95). Historical information was obtained on leisure and occupational activity for 4 time-periods (epochs) Age 6 – 18y, 19 – 30y, 31 – 45y and 45y+. A list of activities was provided and subjects recalled the total number of years in each epoch that they engaged in each activity as well as months per year and hours per week. Occupational and leisure activities were then analysed for their Metabolic (MET) and Mechanical (MECH) components. Each activity was assigned a MET level according to the Compendium of Physical Activities [18]. The frequency and duration of each activity was multiplied by the MET level for that activity in order to calculate MET.hr. Peak strain scores were assigned to each activity according to the

method developed by Groothausen et al [14] to estimate the effects of mechanical impacts on bone. Activities were classified according to their ground reaction force (GRF).

0 = GRF <1 (Non weight-bearing)

1 = GRF 1-2 times bodyweight (Weight-bearing)

2 = GRF 2-4 times bodyweight (Weight-bearing with explosive actions)

3 = GRF >4 times bodyweight (Weight-bearing involving jumping actions)

The frequency and duration of each activity was multiplied by the peak strain score for that activity in order to calculate MECH.hr. For both MET and MECH activity the occupational and leisure values were summed to give activity totals for each epoch and also lifetime totals. In order to standardise for comparative purposes between cohorts these totals were then age-adjusted by simply dividing by the subjects' age so that final values are displayed as MET.h/y and MECH.h/y. In this study the focus is on activity levels in the age 6-18y epoch and lifetime totals.

Statistical Analyses

Statistical analysis was performed using STATA software (Version 10.1, StataCorp, Texas, USA). Descriptive statistics are presented as mean values and standard deviations (SDs). Variables were tested for normality using the Kolmogorov-Smirnov test. Depending on the normality of the distribution, Pearsons or Spearmans correlation coefficients were calculated to examine the relationship between variables. The Students t-test or the Mann-Whitney test (MWU) was used, as appropriate, for comparisons of two groups. Univariate and multivariate regression analysis were performed on the study sample. Initially a single model was applied to all females and separate models were also constructed for pre-menopausal and post-menopausal women. All continuous variables have been divided by standard deviations (SD) to allow for fair comparability of these regression coefficients.

Results

Table 1 Characteristics of the study sample (n=189), expressed as Mean ± SD or number (n)

Variable	Group (n=189)	Premeno (n=98)	Postmeno (n=91)	Premeno vs. Postmeno
Age, y	44 ± 15	32 ± 11	57 ± 5	p≤0.0001
Height, m	1.63 ± 0.06	1.65 ± 0.06	1.62 ± 0.05	p≤0.0001
Weight, kg	67.1 ± 11.2	66.8 ± 12.0	67.5 ± 10.4	p≤0.354
BMI, kg/m²	25.1 ± 4.1	24.6 ± 4.5	25.7 ± 3.5	p≤0.01
Lumbar BMD, g/cm²	1.023 ± 0.173	1.096 ± 0.143	0.944 ± 0.169	p≤0.0001
Femoral BMD, g/cm²	0.870 ± 0.135	0.931 ± 0.115	0.805 ± 0.124	p≤0.0001
Calcium intake, mg/d	851 ± 382	842 ± 412	861 ± 349	p≤0.579
MET PA (Age 6-18), met.h/y	5175 ± 1952	5045 ± 1980	5315 ± 1922	p≤0.309
MET PA (Lifetime), met.h/y	6796 ± 1713	5937 ± 1630	7720 ± 1264	p≤0.0001
MECH PA (Age 6-18), mech.h/y	655 ± 373	656 ± 380	654 ± 368	p≤0.896
MECH PA (Lifetime), mech.h/y	1584 ± 675	1171 ± 552	2028 ± 489	p≤0.0001
Alcohol intake, unit/w	5 ± 6	6 ± 7	4 ± 5	p≤0.05
Smokers, n	n=28	n=14	n=14	N/A
Family History Osteoporosis, n	n=50	n=25	n=25	N/A
Fractures, n	n=39	n=19	n=20	N/A

A total of 189 females participated in this study (mean age 44±15, range 18-67y) and comprised two cohorts; pre-menopausal females (n = 98) and post-menopausal females (n = 91). Characteristics of the study sample are presented in Table 1. As expected, there was a significant difference in age (p≤0.0001), lumbar and femoral BMD (p≤0.0001) between pre- and post-menopausal females. The incidence of low BMD at either site in this study was 59% (n=111). In pre-menopausal females 42% (n=41) had low BMD (41% osteopenic and 1% osteoporotic) and in post-menopausal females 77% (n=70) had low BMD (56% osteopenic and 21% osteoporotic). Height (p≤0.0001) and BMI (p≤0.01) differed between pre- and post-menopausal females. BMI ranged from 18.5-38.2 kg/m^2 with 52% of the study sample being of normal weight (n=99), 35% overweight (n=66) and 13% categorised as being obese (n=24).

Current calcium intake ranged from 143-2430 mg/d and there was no significant difference between the cohorts. However, a total of 128 females (68%) did not meet

their daily calcium requirements (53% of pre- and 84% of post-menopausal females). MET and MECH PA age 6-18y did not differ between pre- and post-menopausal females. However, a significant difference was observed in lifetime MET and MECH PA levels ($p \leq 0.0001$). A similar number of smokers (n=14) was observed in both cohorts (14% pre- and 15% post-menopausal women). Alcohol intake ranged from 0-32 unit/w with 9% of subjects (n=16) consuming ≥14unit/w. There was a significant difference in consumption between pre- and post-menopausal groups ($p \leq 0.05$). A family history of osteoporosis was reported in 27% of the study sample (n=50) and previous fractures were recorded in 21% of subjects (n=39).

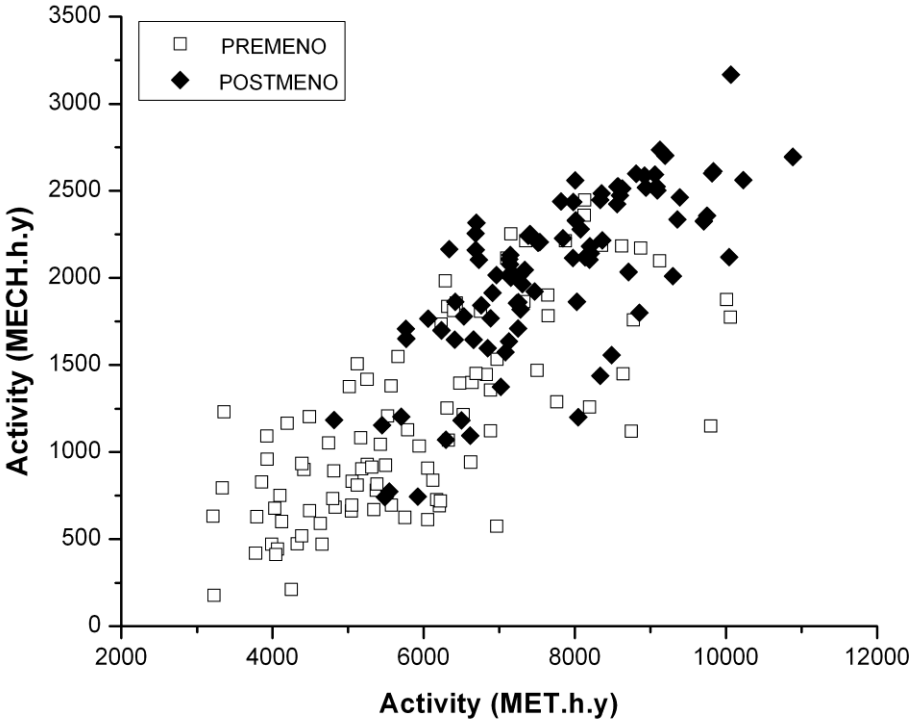

Fig. 1 Relationship between lifetime metabolic and mechanical activity (R=0.66) for pre- and postmenopausal subjects (n = 189)

The relationship between lifetime metabolic and mechanical activity was investigated and a strong positive correlation (R=0.66) was observed between the different components of physical activity. It's evident that as total (metabolic) activity increases, the proportion of that activity which is mechanical in nature will also increase. A similar relationship was also observed between MET and MECH PA aged 6-18y (R=0.55; data not shown). It can be seen in Figure 1 that lifetime PA levels

were significantly higher ($p \leq 0.0001$) in post- than in pre-menopausal women which was somewhat unexpected. The effect of behavioural and unmodifiable risk factors on BMD was investigated using univariate and multivariate regression analysis (Tables 2-5).

Table 2. Univariate analysis (regression coefficients) for lumbar BMD (coefficients for continuous variables are standardised).

	Group (n=189)	Premenopausal (n=98)	Postmenopausal (n=91)
Age	-0.07267[c]	-0.21419	-0.11425[a]
Height	0.05800[c]	0.04105[b]	0.03384
Weight	0.05351[c]	0.04945[c]	0.06553[c]
Calcium intake	0.03213	0.00743	0.00156
MET PA (6-18y)	-0.01989	-0.01455	-0.01484
MET PA (Lifetime)	-0.05728[c]	-0.00973	-0.05003[a]
MECH PA (6-18y)	-0.00457	0.00169	-0.01210
MECH PA (Lifetime)	-0.05926[c]	-0.00622	-0.03472
Alcohol intake	0.00224	-0.00010	-0.00015
Smoking	-0.08058[a]	-0.04630	-0.10870[a]
Family history	-0.08848[b]	-0.08818[b]	-0.08112[a]

[a] $p \leq 0.05$; [b] $p \leq 0.01$; [c] $p \leq 0.001$

In the univariate analysis for lumbar BMD, height was the strongest positive predictor and family history was the strongest negative predictor (Table 2). There were some differences in predictive factors for lumbar BMD between pre- and post-menopausal women. For example, age had a greater influence on BMD in pre- than in post-menopausal subjects (ß -0.21419 vs. -0.11425). Of the behavioural factors, smoking was the strongest predictor, however it appeared to exert a lesser influence on lumbar BMD in pre- than in post-menopausal women (ß -0.04630 vs. -0.10870). PA seemed to have a greater effect in post- than in pre-menopausal women, however, these differences were minor and in general PA was not a strong predictor of lumbar BMD.

Table 3. Multivariate analysis (regression coefficients) for lumbar BMD (coefficients for continuous variables are standardised).

	Group (n=189)	Premenopausal (n=98)	Postmenopausal (n=91)
Age	-0.07585[c]		-0.11165[a]
Height	0.00947	0.02949[a]	
Weight	0.05781[c]	0.04551[c]	0.05312[b]
MET PA (Lifetime)	-0.01742		-0.01043
MECH PA (Lifetime)	0.01404		
Smoking	-0.09047[b]		-0.11113[a]
Family history	-0.06403[b]	-0.08526[b]	-0.08671[a]

[a] $p \leq 0.05$; [b] $p \leq 0.01$; [c] $p \leq 0.001$

Significant univariate predictors for lumbar BMD were then entered into a multivariate model (Table 3). In the total study sample; age, height, weight, MET and MECH PA (lifetime), smoking and family history accounted for 39% of the variance in lumbar BMD (R^2=0.39). For pre-menopausal females; height, weight and family history explained 25% of the variance (R^2=0.25) and for post-menopausal females; age, weight, MET PA (lifetime), smoking and family history accounted for 28% of the variance in lumbar BMD (R^2=0.28).

Table 4. Univariate analysis (regression coefficients) for femoral BMD (coefficients for continuous variables are standardised).

	Group (n=189)	Premenopausal (n=98)	Postmenopausal (n=91)
Age	-0.06644[c]	-0.03704[a]	-0.10888[b]
Height	0.03819[c]	0.02677[a]	0.01254
Weight	0.03794[c]	0.03274[b]	0.05051[c]
Calcium intake	-0.01080	0.01033	-0.03878[b]
MET PA (6-18y)	-0.00417	0.00146	-0.00128
MET PA (Lifetime)	-0.03769[c]	0.0000078	-0.01866
MECH PA (6-18y)	0.00034	0.00823	-0.00898
MECH PA (Lifetime)	-0.04984[c]	-0.014704	-0.01887
Alcohol intake	0.00349[c]	0.00169	0.00138
Smoking	-0.04223	0.00375	-0.08334[a]
Family history	-0.07750[c]	-0.10358[c]	-0.04438

[a] $p \leq 0.05$; [b] $p \leq 0.01$; [c] $p \leq 0.001$

Similarly, in the univariate analysis for femoral BMD, height was the strongest positive predictor and family history was the strongest negative predictor (Table 4). Differences in predictive factors between pre- and post-menopausal women were also observed for femoral BMD. Height appeared to have a more significant effect in pre- than in post-menopausal subjects (ß 0.02677 vs. 0.01254), as did a family history of osteoporosis (ß -0.10358 vs. -0.04438). However, age had a lesser influence on femoral BMD in pre- than in post-menopausal women (ß -0.03704 vs. -0.10888). Of the behavioural factors, smoking was again the strongest predictor, however it appeared to exert a lesser influence on femoral BMD in pre- than in post-menopausal women (ß 0.00375 vs. -0.08334). MET and MECH PA was not a strong predictor of femoral BMD which was similar to the results obtained for lumbar BMD.

Significant univariate predictors for femoral BMD were also entered into a multivariate model (Table 5). In the combined analysis for all females; age, height, weight, MET and MECH PA (lifetime), alcohol intake and family history accounted for 41% of the variance in femoral BMD (R^2=0.41). For pre-menopausal females;

age, height, weight and family history explained 30% of the variance (R^2=0.30) and for post-menopausal females; age, weight, calcium intake and smoking accounted for 32% of the variance in femoral BMD (R^2=0.32).

Table 5. Multivariate analysis (regression coefficients) for femoral BMD (coefficients for continuous variables are standardised).

	Group (n=189)	Premenopausal (n=98)	Postmenopausal (n=91)
Age	-0.07116[c]	-0.03142[a]	-0.08872[b]
Height	-0.00653	0.00918	
Weight	0.04791[c]	0.03758[c]	0.04179[c]
Calcium intake			-0.02964[a]
MET PA (Lifetime)	0.005184		
MECH PA (Lifetime)	-0.00380		
Alcohol intake	0.00011		
Smoking			-0.08532[b]
Family history	-0.05454[b]	-0.08656[c]	

[a] $p \leq 0.05$; [b] $p \leq 0.01$; [c] $p \leq 0.001$

Discussion

The aim of this study was to investigate the effect(s) of behavioural (calcium intake, physical activity, weight, smoking status and alcohol consumption) and unmodifiable (age, height, family history of osteoporosis and fracture history) risk factors on bone health as measured by BMD in Irish pre- and post-menopausal women. A further aim was to determine the prevalence of osteoporosis among these cohorts.

A high prevalence of osteopenia (48%) and osteoporosis (11%) was found in the sample population. Low bone density profiles have previously been reported in Irish women [19]. However, of particular concern was the incidence of low BMD in pre-menopausal females (41% osteopenic; 1% osteoporotic) which is considerably higher than that reported elsewhere (Brazil 26% [20] and Canada 20% [21]). Pre-menopausal low bone mass may be indicative of insufficient bone accrual during growth (low PBM) and coupled with accelerated post-menopausal bone loss greatly increases the probability of these women developing osteoporosis and experiencing a fragility fracture in the future. Some 77% of the post-menopausal cohort in this study had low BMD (56% osteopenic and 21% osteoporotic). It is estimated that by 2031 there will be an additional 500,000 older people living in Ireland [22] and therefore the proportion of elderly and post-menopausal women is on the rise. Health promotion and prevention strategies are now required to offset the implications for this demographic shift on osteoporosis healthcare in the future.

The multivariate analysis undertaken in this study confirmed once again the importance of unmodifiable factors such as age, height and family history for bone health. It has been reported that 60-80% of bone mass is hereditary [5], however factors such as these are effectively beyond ones control and therefore preventative efforts against osteoporosis must be directed at the variation in bone mass that is due to behavioural factors. This analysis demonstrated that in addition to the unmodifiable risk factors; smoking, MET and MECH PA (lifetime) and weight accounted for 39% of the variance in lumbar BMD. Age, height, family history, alcohol consumption, MET and MECH PA (lifetime) and weight contributed to 41% of the variance in

femoral BMD. The univariate analysis also identified smoking as the strongest behavioural predictor of lumbar and femoral BMD.

Smoking has been reported to increase bone resorption [23] therefore increasing the risk of osteoporosis. In this study a similar number of smokers (n=14) was observed in both cohorts (14% pre- and 15% post-menopausal women). This figure is lower than has been previously reported by the Survey of Lifestyle, Attitudes and Nutrition (SLÁN) study in 2007 which recorded a smoking incidence of 27% in Irish females [24] which may suggest an increased awareness of health in this subset of the population. Of course it's possible that this subset may be biased in this respect, as choosing to participate in this study may already reflect an increased awareness of health and/or interest in health issues. Although the link between smoking and lung cancer has long been established [25] and widely publicised, anecdotal evidence suggests that women in Ireland are unaware of the risks associated with smoking and osteoporosis.

Physical activity may play different roles throughout the lifespan and PA age 6-18 was assessed in this study because rapid bone accrual occurs during puberty with approximately 90-95% of peak bone density being accumulated by the late teenage years [26]. MET and MECH PA age 6-18y did not differ between pre- and post-menopausal females, which may be due to the fact that most women at this age would be enrolled in full-time education and therefore have very similar levels of occupational activity. A significant difference was observed in lifetime MET and MECH PA ($p \leq 0.0001$) with higher levels of activity being recorded in post-menopausal women, which may be attributed to higher levels of occupational activity in this cohort. Many of the pre-menopausal women recruited in this study were university students and as such had relatively sedentary occupations. As expected a strong relationship was observed between lifetime metabolic and mechanical activity ($R=0.66$). It's clear that as total (metabolic) activity increases, the proportion of that activity which is mechanical in nature will also increase concurrently.

Physical activity may play an important role in preventing the development of a number of chronic diseases, including osteoporosis. Despite this, it has previously been reported that 45% of Irish adults do not regularly participate in physical activity

[24]. It is potentially the long-term, chronic exposure to physical inactivity that increases risk of disease, however, the assessment of lifetime physical activity has not yet been well developed. To the best of the authors knowledge very few questionnaires have previously been used to assess the duration, intensity and frequency of lifetime physical activity [17, 27-29]. The lifetime physical activity questionnaire used in the current study is unique in that it is self-administered and assesses leisure, occupational and household activities.

The univariate analysis in this study found no significant relationship between total lifetime metabolic or mechanical activity and BMD. This is in keeping with the findings of other studies using similar questionnaires [29-31]. In a study sample from Northern Ireland, an association was found between sports activity and BMD in men but not in women and the authors concluded this reflected the women's lower participation in activities with a high mechanical component [30]. The present study also found no relationship between BMD and activity from age 6-18y. In contrast Micklesfield et al [29] did report an association between activity during adolescence and current BMD. It's possible that this was not observed in the current study due to recall bias which is one of the main difficulties associated with the assessment of lifetime physical activity [32]. A further limitation of this study is its relatively small sample size. However, it is feasible that the relationship between physical activity and bone mass is non-linear which would explain why no association was found between lifetime PA and BMD using linear regression. It is also possible that PA may increase bone strength by influencing bone quality which is a change that may not be reflected in a BMD value. Considering the magnitude of genetic influence on bone health it's possible that certain genotypes influencing BMD may be up-regulated by physical activity and therefore further investigation is warranted in this area. Whether calcium and physical activity interact synergistically is also yet to be established. It is possible that low levels of calcium intake may mask the effects of high physical activity. Indirect evidence suggests that the beneficial effect of high physical activity on bone may only be evident at high calcium intakes (>1000mg/d) with little or no effect at mean calcium intakes of <1000mg/d [33].

It is important to note that there are other benefits besides bone mass to be gained from physical activity. Risk of falling and therefore fracture risk may be reduced by

increasing muscle strength, balance and postural stability [34]. Specific physical activity guidelines for osteoporosis should be developed similar to those that are currently in place for cardiovascular health. Weight-bearing exercise of 30 – 40min duration undertaken 3 – 4 times per week has been shown to have an osteogenic effect [35]. The importance of physical activity at every phase of the lifecycle must be stressed with particular emphasis on children, teenagers and the elderly.

In this study the range of BMI was 18.5-38.2 kg/m^2 and although 48% of subjects were categorised as being overweight (35%) or obese (13%), this is in line with the increasing prevalence of obesity in Western countries [36]. Fracture risk is markedly higher at lower values of BMI, particularly with a BMI of less than $20kg/m^2$; however, the relationship between BMI and fracture risk is non-linear and from 25-$35kg/m^2$ the differences in fracture risk are quite small [7]. Therefore, obesity should not be regarded as a protective factor against fracture but it is a significant risk factor for type 2 diabetes, cardiovascular disease and hypertension [36].

There was a wide range of alcohol intake (0-32 unit/w) in this study with a significant difference in consumption between pre- and post-menopausal women (6 vs. 4 unit/w; $p \leq 0.05$). This age-related trend was also observed in the SLÁN data [24]. In this study 9% of subjects reported consuming more than the recommended amount of alcohol per week (14unit/w), which is higher than the figure of 5% which was also self-reported in the SLÁN study. However, it should be noted that the validity of self-reported alcohol consumption in general is notoriously unreliable [37] which may account for why, according to the univariate analysis, current alcohol intake was not a strong predictor of BMD in this study. Excessive alcohol intake is accepted as a risk factor for low BMD and osteoporotic fracture, however, low to moderate intake has been reported to have beneficial effects on bone [38].

In this study current calcium intake was a poor predictor of lumbar and femoral BMD. Current calcium intake was evaluated in this study as there is some evidence that dietary behaviours such as milk and calcium intake developed in childhood persist into adulthood (R=0.38) [39]. However it's possible that current calcium intake may not reflect lifetime patterns in this study sample. It is notable however, that 68% of women did not meet their daily calcium requirements, which is substantially higher

than the previously reported figure of 36% [40]. Dairy products account for 48% of calcium in the Irish diet [41]. Current dietary guidelines such as the Food Pyramid recommend 3 servings per day of dairy products [42]. However, Irish women have been reported to only consume 1.5 servings of dairy products daily [43]. Avoidance of dairy products may be due to fears of weight gain and the misperception that milk and other dairy products are fattening [44]; however calcium insufficiency may increase the risk of osteoporosis in these subjects [10] and particularly in the post-menopausal women of whom 84% were calcium deficient.

The use of clinical risk factor (CRF) algorithms such as FRAX to estimate fracture risk is a significant advance in the management of osteoporosis [45]. FRAX has been designed as a platform technology which can be upgraded as new validated risk indicators become available. CRFs that should be considered for incorporation into FRAX in the future include behavioural risk factors with dose response effects such as alcohol consumption, diet (in particular calcium and vitamin D) and physical activity.

Conclusions

Low bone mass is prevalent among Irish adult women and there is a need for a greater focus on preventative behaviours to tackle this silent epidemic in Irish society. Education should specifically focus on the association between smoking, alcohol consumption, PA and calcium intake on bone health. Given the demographic trend towards an older population in Ireland, action is required now to offset the catastrophic human and economic costs associated with the predicted fragility fractures in Ireland in the future.

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Conflict of Interest None

References

1. Cornwall R, Gilbert MS, Koval KJ, Strauss E,Siu AL (2004) Functional outcomes and mortality vary among different types of hip fractures: a function of patient characteristics. Clin Orthop Relat Res 425(64-71
2. Kanis JA,Johnell O (2005) Requirements for DXA for the management of osteoporosis in Europe. Osteoporos Int 16(229-238
3. Kai MC, Anderson M,Lau EM (2003) Exercise interventions: defusing the world's osteoporosis time bomb. Bulletin of the World Health Organisation 81(11): 827-30
4. Dodds MK, Codd MB, Looney A,Mulhall KJ (2009) Incidence of hip fracture in the Republic of Ireland and future projections; a population based study. Osteoporos Int 20(12): 2105-2110
5. Ferrari S, Rizzoli R, Slosman D,Bonjour JP (1998) Familial Resemblance for bone mineral mass is expressed before puberty. J Clin Endocrinol Metab 83(2): 358-361
6. Lau HH, Ho AY, Luk KD,Kung AW (2004) Transforming growth factor-beta1 gene polymorphisms and bone turnover, bone mineral density and fracture risk in southern Chinese women. Calcif Tissue Int 74(6): 516-521
7. De Laet C, Kanis JA, Odén A et al; (2005) Body mass index as a predictor of fracture risk: a meta-analysis. Osteoporos Int 16(11): 1330-1338
8. Christodoulou C,Cooper C (2002) What is osteoporosis? Postgrad Med J 79(133-138
9. Kanis JA, Johansson H, Johnell O et al; (2005) Alcohol intake as a risk factor for fracture. Osteoporos Int 16(737-742
10. Heaney RP (2000) Calcium, Dairy Products and Osteoporosis. J Am Coll Nutr 19(83S-99S
11. Välimäki MJ, Kärkkäinen M, Lamberg-Allardt C et al; (1994) Exercise, smoking, and calcium intake during adolescence and early adulthood as determinants of peak bone mass. Br Med J 309(230-235
12. Vincent KR,Braith RW (2002) Resistance exercise and bone turnover in elderly men and women. Med Sci Sports Exerc 34(17-23
13. Lanyon LE, Goodship AE, Pye CJ,Macfie JH (1982) Mechanically adaptive bone remodelling. J Biomech 15(141-154
14. Groothausen J, Siemer H, Kemper HCG, Twisk J,Welten DC (1997) Influence of peak strain on lumbar bone mineral density: an analysis of 15 year physical activity in young males and females. Pediatr Exerc Sci 9(159-173
15. Weiss M, Yogev R,Dolev E (1998) Occupational sitting and low hip mineral density. Calcif Tissue Int 62(47-50
16. Bingham SA, Welch AA, McTaggart A et al; (2001) Nutritional methods in the European Prospective Investigation of Cancer in Norfolk. Public Health Nutr 4(847-858
17. Kriska AM, Sandler RB, Cauley JA et al; (1988) The assessment of historical physical activity and its relation to adult bone parameters. Am J Epidemiol 127(1053-1063
18. Ainsworth BE, Haskell WL, Whitt MC et al; (2000) Compendium of physical activities: an update of activity codes and MET intensities. Med Sci Sports Exerc 32(S498-504

19. Donne B, Kelly M, Kelly A et al; (1996) Bone Density Profiles and Osteoporosis Incidence in Irish Females. Ir Med J 89(3): 92-94
20. de Azevedo MN, Nunes JV, Pasqualette HA, Tuma Mde F,Nunes LC (2009) Bone mineral density at 45 years of age, before menopause. J Natl Med Assoc 101(8): 788-792
21. Jamal SA, Ridout R, Chase C et al; (1999) Bone mineral density testing and osteoporosis education improve lifestyle behaviours in premenopausal women; A prospective study. J Bone Miner Res 14(12): 2143-2149
22. Department of Health and Children and National Council on Ageing and Older People, *Strategy to Prevent Falls and Fractures in Ireland's Ageing Population*, 2008, Health Services Executive: Dublin, Ireland.
23. Szulc P, Garnero P, Claustrat B et al; (2002) Increased bone resorption in moderate smokers with low body weight: the Minos study. J Clin Endocrinol Metab 87(666-674
24. Morgan K, McGee H, Watson D et al;, *SLÁN 2007: Survey of Lifestyle, Attitudes & Nutrition in Ireland*, 2008, Department of Health and Children: Dublin.
25. Levin ML, Goldstein H,Gerhardt PR (1950) Cancer and tobacco smoking; a preliminary report. J Am Med Assoc 143(4): 336-338
26. Heaney RP, Abrams S, Dawson-Hughes B et al; (2000) Peak Bone Mass. Osteoporos Int 11(12): 985-1009
27. Friedenreich CM, Courneya KS,Bryant HE (1998) The lifetime total activity questionnaire: development and reliability. Med Sci Sports Exerc 30(266-274
28. Chasan-Taber L, Erickson JB, McBride JW et al; (2002) Reproducibility of a self-administered lifetime physical activity questionnaire among female college alumnae. Am J Epidemiol 155(282-289
29. Micklesfield L, Rosenberg L, Cooper D et al; (2003) Bone Mineral Density and Lifetime Physical Activity in South African Women. Calcif Tissue Int 73(5): 463-469
30. Neville CE, Murray LJ, Boreham CA et al; (2002) Relationship between physical activity and bone mineral status in young adults: the Northern Ireland Young Hearts Project. Bone 30(5): 792-798
31. Brahms H, Mallmin K, Michaelsson K, Strom H,Ljunghall S (1998) Relationships between bone mass measurements and lifetime physical activity in a Swedish population. Calcif Tissue Int 62(400-412
32. Gammon MD, John EM,Britton JA (1998) Recreational and occupational physical activities and risk of breast cancer. J Natl Cancer Inst 90(2): 100-117
33. Specker BL (1996) Evidence for an interaction between calcium intake and physical activity on changes in bone mineral density. J Bone Miner Res 11(1539-1544
34. Nelson ME, Fiatarone MA, Morganti CM et al; (1994) Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures. A randomized controlled trial. J Am Med Assoc 272(24): 1909-1914
35. Kohrt WM, Bloomfield SA, Little KD, Nelson ME,Yingling VR (2004) American College of Sports Medicine Position Stand: physical activity and bone health. Med Sci Sports Exerc. 36(11): 1985-1996
36. World Health Organization, *Obesity: Preventing and Managing the Global Epidemic*, in *WHO Technical Report Series 894*2000: Geneva.
37. Midanik LT (1988) Validity of self-reported alcohol use: a literature review and assessment. Br J Addict 83(9): 1019-1030

38. Berg KM, Kunins HV, Jackson JL et al; (2008) Association between alcohol consumption and both osteoporotic fracture and bone density. Am J Med 121(5): 406-418
39. Welten DC, Kemper HC, Post GB, Van Staveren WA,Twisk JW (1997) Longitudinal development and tracking of calcium and dairy intake from teenager to adult. Eur J Clin Nutr 51(612-618
40. Hannon EM, Kiely M, Harrington KE et al; (2001) The North/South Ireland Food Consumption Survey: mineral intakes in 18-64-year-old adults. Public Health Nutr 4(1081-1088
41. Burke SJ, Gibney MJ, O'Dwyer NA,McCarthy SN (2005) The influence of cereal and dairy consumption on the Irish diet: implications for developing food-based dietary guidelines. Public Health Nutr 8(227-237
42. National Institute of Health, *Optimal Calcium Intake. Consensus Statement.*, 1994. p. 1-31.
43. Irish Universities Nutrition Alliance, *North/South Ireland Food Consumption Survey*, 2001. p. 1027-1127.
44. Horwath CC, Govan CH, Campbell J, Busby W,Scott V (1995) Factors influencing milk and milk product consumption in young and elderly women with low calcium intakes. Nutr Res 15(12): 1735-1745
45. Kanis JA, Johnell O, Oden A, Johansson H,McCloskey E (2008) FRAX and the assessment of fracture probability in men and women from the UK. Osteoporos Int 19(4): 385-397