VIA MEDICA

ORIGINAL ARTICLE

Anna Kopiczko[1], Michał Czapla[2, 3, 4], Patrycja Widłak[5]

[1]Section of Anthropology, Department of Human Biology, Józef Piłsudski University of Physical Education in Warsaw, Poland
[2]Department of Emergency Medical Service, Wroclaw Medical University, Wroclaw, Poland
[3]Institute of Heart Diseases, University Hospital, Wroclaw, Poland
[4]Group of Research in Care (GRUPAC), Faculty of Health Science, University of La Rioja, Logrono, Spain
[5]Section of Sports Medicine, Department of Human Biology, Józef Piłsudski University of Physical Education in Warsaw, Poland

Bone health in young women: the effect of tobacco smoking, environmental tobacco smoke exposure and physical activity on bone mineral density

Corresponding author:

Michał Czapla PhD,
Department of Emergency Medical Service, Wrocław Medical University, Wrocław, Poland;
e-mail: michal.czapla@umw.edu.pl

Medical Research Journal 2023; Volume 8, Number 2, 116–127
10.5603/MRJ.a2023.0022

ISSN 2451-2591
e-ISSN 2451-4101

ABSTRACT

Health behaviours are a key component of bone health. In 657 young women examined the relationship between bone mineral density (BMD), bone mineral content (BMC) in the distal and proximal part of the forearm, and physical activity (PA), smoking (AS), environmental tobacco smoke exposure (ETS) and body mass index (BMI). The densitometry method, Global Adult Tobacco Survey Questionnaire and International Physical Activity Questionnaire were used. Weekly physical activity was calculated by adding up the Metabolic Equivalent of Task (MET). Smoking women had significantly lower bone parameters. The predictor of interactions of three variables: PA, BMI (positive direction), and years of ETS exposure (negative direction) was significant for distal BMD and BMC. The predictor of interactions of PA, BMI (positive direction), and AS in numbers of cigarettes per day and years of ETS exposure (negative direction) was significant for BMD prox. MET min/week (positive direction), years of AS, and ETS exposure (negative direction) were significant predictors for T-score dis. The predictor of interactions of five variables: PA (MET), BMI, age of starting smoking (positive direction), AS in numbers of cigarettes per day, and years of ETS exposure (negative direction) was significant for T-score prox. Cigarette smoking and ETS are modifiable determinants of low bone mineral density in young Polish women. Despite the women's good socio-economic status, a high, alarming incidence of low BMD was reported. The current study may be important in understanding the relationship between BMD, BMC, and smoking in young women as risk determinants of osteoporosis in future.

Key words: active smoking, environmental tobacco smoke, physical activity, Metabolic Equivalent of Task, bone mineral density, bone mineral content

Med Res J 2023; 8 (2): 116–127

Introduction

Health behaviour research is important in assessing the risk of several diseases, including bone disease. Bone mineral density (BMD) is a complex parameter that is influenced by both genetic and environmental factors, and possibly also by interactions between them. It is known that body weight or body mass index (BMI) are positively associated with BMD in older men and women [1–3]. However, in young women, this relationship has not been fully established. Physical activity is considered an important factor positively influencing BMD. However, it is unclear whether this beneficial effect on BMD could be reduced by modifiable risk behaviours associated with lifestyle. Socioeconomic factors have been recognized as associated factors of many chronic diseases such as diabetes and cardiovascular disease [4–6]. Scientists concluded that there is limited evidence of good quality for impact assessments of socio-economic status in mineral density and bone fractures [7].

Cigarette smoking is widely believed to contribute to premature death [3], cancer [8], and many chronic diseases [9]. Smoking may be one of the most important modifiable risk behaviours related to lifestyle, also in the development of osteoporosis [10–12]. Many studies have identified smoking as a risk factor for osteopenia and osteoporosis, with its effects greatest in men and older adults, and dose-dependent [10, 13]. Studies on the effect of tobacco smoking on BMD in young women are much less numerous and their results are inconclusive [10, 12, 14]. Passive smoking (PS) is the effect of environmental tobacco smoke (ETS) exposure on the human body [15, 16]. Environmental smoke and tobacco smoke exposure (TSE) cause the same serious diseases as active smoking [3, 8, 17–19]. Long-term active smoking was a strong risk factor for postmenopausal osteoporosis [20]. Meta-analyzes of the effects of smoking on bone health showed a low bone mass (BMC) and reduced BMD in current smokers compared to non-smokers [10]. In particular, exposure to cadmium from tobacco smoke plays an important role in the development of osteoporosis [21]. Smokers experience deterioration of bone microarchitecture, especially in the trabecular compartment [13]. Quitting smoking may at least partially reverse the adverse effects of smoking on the skeletal system [22]. The effect appears to be dose-dependent [23]. Few studies have looked at the effects of both ETS and AS on young adults [19, 24]. It is especially important and necessary to study the effects of smoking on bone health before developing osteoporosis among the young because it is a modifiable factor.A secular trend confirming positive changes in the standard of living was observed in the population of Polish women. The improvement of living conditions allows for the full use of the genetically determined growth potential [25].

This study examined the relationship between forearm BMD, BMC, and health behaviour such as physical activity (PA), smoking (AS), ETS and BMI in young Polish women.

Material and methods

Study design and procedure

This study involved 657 healthy young Polish women (Caucasians of European origin) between 25 and 35 years of age with good socio-economic status (financial income per person in the family in the self-assessment good, high, satisfactory; higher education, employment on a permanent contract of employment — professional status in the self-assessment of female respondents stable; type of residence — the metropolitan areas, the capital of Poland, Warsaw). The study sample was selected by a non-random sampling method (snowball sampling) of recruiting participants by other participants. The exclusion criteria included: bone disease, pregnancy, hormone therapy, hormonal and menstrual disorders (amenorrhea, oligomenorrhea, premenstrual syndrome, premenstrual dysphoric disorder), kidney disease, thyroid and parathyroid diseases, cancers, rheumatoid arthritis, and long-term steroid treatment, use of restrictive diets that eliminate or limit protein intake.

All participants provided informed consent according to the Declaration of Helsinki. Participants were not compensated for their participation but received the results of their tests with the specialist's interpretation. The study was carried out in 2020–2022 in the Department of Human Biology, Anthropology Section, the Józef Piłsudski University of Physical Education in Warsaw, Poland, in the laboratory of densitometry and anthropometric tests. The team with the necessary qualifications and experience in research performed the measurements on the entire study group.

Assessment of somatic and bone parameters

The anthropometric measurement protocol was used as described by Hall et al. [26]. Body mass was measured using a JAWON MEDICAL X-SCAN PLUS II analyzer (Certificate No. EC0197 for medical devices), with subjects standing barefoot and wearing light clothing. Body height was measured to the nearest 0.1 cm using a Martin anthropometer (GMP, Renens, Switzerland). Body mass index (BMI) was calculated using the standard formula and classified according to the World Health Organization (WHO) cut-off points [27]. For the assessment of BMD (in g/cm^2) and BMC (in grams), the densitometry method of the forearm bone was used at two measurement points, proximal (prox) and distal (dis), using the NORLAND company apparatus (Swissray, Edison, NJ, USA; Norland Medical Systems, Fort Atkinson, WI, USA). A bone examination was performed once. The effective dose (μSv) for this densitometer is 0.05. A T-score was used in the analysis (ratio of BMD of the person to mean BMD in a young healthy reference group expressed in standard deviations). The Norland DXA has a global distal site, a global ⅓ proximal site, and a ⅓ proximal radius site [28]. The scanner was calibrated daily against the standard calibration block supplied by the manufacturer to control for possible baseline drift. Absorptiometry measurements of BMC are very accurate (error of 1% to 3%). All the data were collected according to the recommendations of the International Society for Clinical Densitometry [29]. The authors used the WHO Classification of BMD based on the normative data of young adults aged 20–29 years, with modifications using information from the Third National Health

and Nutrition Examination Survey (NHANES): T-score normal for –1.0 or greater, low bone mineral density (osteopenia) between –1.0 and –2.5, and osteoporosis for –2.5 and below [30].

Assessment of physical activity and smoking

The International Physical Activity Questionnaire (IPAQ) short version was used to assess the present level of physical activity in a direct interview recommended in such international studies as the European Health Interview Survey (EUROHIS) and the European Physical Activity Surveyance System (EUPASS). Weekly physical activity was calculated by adding up the Metabolic Equivalent of Task (MET) values obtained during vigorous activity, moderate activity and walking performed during the entire week [31].

In the interview was used the Global Adult Tobacco Survey questionnaire (GATS), section B on active smoking (AS). A direct interview was conducted by a trained interviewer with extensive experience in collecting data using this method and the GATS questionnaire. Data were collected on the number of years of smoking and the number of cigarettes smoked per day. Childhood exposure to ETS was assessed. Exposure to ETS was measured by self-reported indicators of exposure using questionnaires and interviews. Data were collected on the number of years of childhood exposure to environmental tobacco smoke. The method of conducting the survey followed the guidelines of WHO experts [16] and the GATS methodology used in Poland, including a standard protocol for the interview questionnaires, sample weights, data management, analysis, reporting, and release of information. In Poland, the Ministry of Health revised and approved the questionnaires and also appointed two committees, the GATS Poland Scientific Committee, and the GATS Poland Steering Committee, which handle the scientific and technical coordination of the nationally-representative GATS survey [32].

Ethical approval

The study was conducted according to the guidelines of the Declaration of Helsinki, and approved by the independent Bioethics Committee of the Józef Piłsudski University of Physical Education in Warsaw (protocol number 01-10/2011). Informed consent was obtained from all subjects.

Statistical analysis

The research results were analysed with the use of Statistica software (v.11, Stat. Soft. USA). Means and standard deviations were calculated for each somatic, bone and smoking parameter. In order to determine the significance of differences between the values of particular variables for smoking and non-smoking women, the Student's *t*-test for independent variables was applied. The effect size was calculated using Cohen's d = 2t/ /(df ^ 1/2), (small effect: < 0.5; medium effect: 0.5–0.8; large effect: > 0.8). Chi-square was used to test the significance of the incidence of Low BMD/Osteopenia and Osteoporosis. In turn, the one-way ANOVA and two-way analysis of variance (ANOVA) were used to evaluate the significance of differences in T-score (prox and dis) in the context of smoking. In order to determine the relationships between bone parameters (BMD, BMC and T-score) in the distal and proximal segments and particular predictor variables, the multiple forward stepwise regression model was applied. The following levels of significance were used in the analyses: *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$ (p — p-value).

Results

The basic characteristics of biometric, somatic, and bone parameters

The basic characteristics of the two women groups (smokers and nonsmokers) of biometric, somatic, and bone parameters and the significance of differences and effect sizes calculated using Cohen's d are presented in Table 1. The groups differed significantly in 12 of the 14 analysed parameters. The smoker women were slightly lighter (small effect $d = 0.176$), had smaller BMI (small effect $d = 0.254$), and significantly lower (< 0.001) all bone parameters in two parts of the forearm (large effect $d > 0.8$) compared to nonsmokers women. Smoker women had significantly lower MET (min/week), (large effect d > 0.8). Smoker women had indeed longer significantly longer exposure to ETS about 4 years (large effect $d = 0.991$). Table 1 also shows an assessment of the incidence of low BMD (osteopenia) and osteoporosis. The highest frequency of reduced BMD in the distal and proximal part of the forearm occurs in smokers women (Tab. 1).

Relationships between bone parameters and selected variables in smokers and nonsmokers

Relationships between bone parameters separately for dis and prox segments and somatic, lifestyle factors in smokers and nonsmokers women (results of ANCOVA analyses, age-continuous variable) are presented in Table 2 (ANCOVA). Of all the variables analysed, the strongest relationships with bone parameters were consistently found for MET both in the smoking and non-smoking group. Covariance analysis indicated that, in smoking women, the main parameters significantly affecting BMD in the dis. part of the forearm were three

Table 1. Comparison of the biometric, somatic, and bones parameters in smokers and nonsmokers women

Variables	Smokers (n = 275)		Nonsmokers (n = 382)		*t*	Cohen's *d*
	M	SD	M	SD		
Somatic parameters						
Age (year)	28.7	2,9	28.5	3.2	–5,77	0.066
Body Height (cm)	166.1	4.5	166.2	3.9	0.23	0.023
Body Weight (kg)	62.5	9.1	64.0	7.9	2.24*	0.176
BMI [kg/m^2]	22.6	2.1	23.2	2.6	2.50**	0.254
Bone parameters						
BMD dis [g/cm^2]	0.352	0.073	0.419	0.082	10.79***	0.863
BMD prox [g/cm^2]	0.715	0.089	0.822	0.107	13.49***	1.087
BMC dis (g)	1.442	0.342	1.775	0.410	10.99***	0.882
BMC prox (g)	1.839	0.376	2.238	0.478	11.52***	0.928
T-score dis	–0.500	0.870	0.350	0.942	11.78***	0.937
T-score prox	–1.638	0.760	–0.796	0.810	13.49***	1.072
% young ref. dis	79.7	10.4	94.8	48.2	2.98***	0.433
% young ref. prox	94.1	49.5	106.4	53.8	5.11***	0.238
Lifestyle factors						
MET (min/week)	875.5	149.7	1297.9	191.4	30.51***	2.459
Age when starting AS	18.1	3.3	–	–	–	–
AS (∑years)	6.4	3.4	–	–	–	–
AS (cigarette/day)	8.8	3.8	–	–	–	–
ETS (∑years)	4.4	4.2	0.9	2.7	–13.05***	0.991
			n (%)		Chi-square test, p-value	
T-score dis						
Normal	184 (66.9)		349 (91.4)		***p < 0.001	
Low BMD/Osteopenia	90 (32.7)		33 (8.6)			
Osteoporosis	1 (0.4)		0 (0)			
T-score prox						
Normal	52 (18.9)		227 (59.4)		***p < 0.001	
Low BMD/Osteopenia	194 (70.5)		150 (39.3)			
Osteoporosis	29 (10.6)		5 (1.3)			
ETS						
Yes	170 (61.8)		47 (12.3)		***p < 0.001	
No	105 (38.2)		335 (87.7)			

AS — active smoking in adults; BMC — bone mineral content; BMD — bone mineral density; *d* — effect sizes calculated using Cohen's formula; dis — in the distal part of the forearm; ETS — environmental tobacco smoke; prox — in the proximal part of forearm; *t* — Student's *t*-test

*p < 0.05; **p < 0.01; ***p < 0.001

variables MET (min/week), age when starting AS and ETS (∑years) (adj. R2 = 0.14). In turn, BMD in the prox. part the main parameters significantly affecting were two variables MET (min/week) and ETS (∑years) (adj. R2 = 0.16). Similar analyses in smokers indicated that the parameters significantly affecting BMC in the dis. part of the forearm were four variables MET (min/week), age when starting AS, AS (∑years) and ETS (∑years) (adj. R2 = 0.21). In turn, BMC in the prox. part the main parameters significantly affecting were three variables MET (min/week), age when starting AS and ETS (∑years) (adj. R2 = 0.20).

In the nonsmoking women covariance analysis indicated that the main parameter significantly affecting BMD in the dis. part of the forearm was only MET (min/week), (adj. R2 = 0.11). In turn, BMD in the prox. part the main parameters significantly affecting were two variables MET (min/week) and ETS (∑years) (adj. R2 = 0.15). In nonsmokers, it was indicated that the parameters significantly affecting BMC in the

Table 2. Relationships between bone parameters and somatic, lifestyle factors in smokers and nonsmokers women (results of ANCOVA analyses, age-continuous variable)

	BMD dis. [g/cm^2]	BMD prox. [g/cm^2]	BMC dis. (g)	BMC prox. (g)
	F (p)	*F (p)*	*F (p)*	*F (p)*
		Smokers		
Age (year)	0.554 (0.457)	2.697 (0.102)	2.401 (0.122)	0.666 (0.415)
Body Height (cm)	1.638 (0.202)	1.918 (0.167)	0.763 (0.383)	1.139 (0.287)
Body Weight (kg)	1.324 (0.251)	1.358 (0.245)	0.292 (0.589)	0.445 (0.505)
MET (min/week)	6.565 (0.011)**	8.200 (0.005)**	9.320 (0.002)**	15.74 (0.000)***
Age when starting AS	5.652 (0.018)*	0.452 (0.502)	10.30 (0.001)***	6.735 (0.010)**
AS (∑years)	2.770 (0.097)	1.952 (0.164)	5.300 (0.022)*	0.190 (0.664)
AS (cigarette/day)	0.639 (0.425)	3.491 (0.063)	0.697 (0.404)	3.248 (0.073)
ETS (∑years)	8.689 (0.003)**	11.05 (0.001)*	12.84 (0.000)***	7.377 (0.007)**
F (p)	6.05 (0.000)	6.65 (0.000)	9.001 (0.000)	8.88 (0.000)
R ^ 2 adj.	0.14	0.16	0.21	0.20
		Nonsmokers		
Age (year)	3.666 (0.056)	1.230 (0.268)	6.478 (0.011)**	2.552 (0.111)
Body Height (cm)	0.119 (0.730)	0.162 (0.668)	0.554 (0.457)	0.300 (0.584)
Body Weight (kg)	0.406 (0.525)	0.029 (0.864)	0.227 (0.634)	0.119 (0.731)
MET (min/week)	12.08 (0.001)***	14.63 (0.000)***	7.293 (0.007)**	4.484 (0.035)*
ETS (∑years)	2.214 (0.138)	6.504 (0.011)**	0.096 (0.757)	0.353 (0.553)
F (p)	8.15 (0.000)	11.86 (0.000)	6.72 (0.000)	5.66 (0.000)
R ^ 2 adj.	0.11	0.15	0.10	0.12

AS — active smoking in adults; BMC — bone mineral content; BMD — bone mineral density; ETS — environmental tobacco smoke; F — Ronald A. Fisher's test; p — p-value; R ^ 2 adj. — the adjusted R-squared values of determination. Level of statistical significance: *$p \leq 0.05$, **$p \leq 0.01$ and ***$p \leq 0.001$

dis. part of the forearm were two variables age and MET (min/week), (adj. R2 = 0.10). In turn, BMC in the prox. part the main parameter significantly affecting was only MET (min/week), (adj. R2 = 0.12) (Tab. 2).

Relationships between BMD, BMC and T-score in the distal and proximal part of the forearm and BMI, MET and smoking parameters were evaluated using the multiple forward stepwise regression (Tab. 3). The presented model explained 23–41% (adjusted R2 = 0.23–0.41; $p < 0.001$) of the variance in bone parameters. The predictor of interactions of three variables: physical activity (MET) and BMI (positive direction), as well as years of ETS (negative direction), was significant for BMD dis and BMC dis (adjusted R2 0.23–0.41; $p < 0.001$). It was also found that the predictor of interactions of four variables: physical activity (MET) and BMI (positive direction), as well as active smoking numbers of cigarettes per day and years of ETS (negative direction), was significant for BMD prox (adjusted R2 = 0.33; $p < 0.001$). Furthermore, the predictor of interactions MET and BMI (additive direction), as well as age when starting active smoking and years of ETS (negative direction), was significant for BMC prox (adjusted R2 = 0.25; $p < 0.001$). Significant predictors for T-score in both measurement points were also noted. Physical activity in MET (positive value of the standardized β coefficient) and years of active smoking and ETS (negative direction) was significant predictor for T-score dis (adjusted R2 = 0.29; $p < 0.001$). The predictor of interactions of five variables: physical activity (MET), BMI and age when starting active smoking (positive direction), as well as active smoking numbers of cigarettes per day and years of ETS (negative direction), was significant for T-score prox (adjusted R2 = 0.41; $p < 0.001$), (Tab. 3).

Figures 1 and 2 present a graphical representation of the results of the analysis of variance. Regardless of the active smoking category, women without childhood ETS exposure had the most advantageous values of T-score dis (Fig. 1).

In the proximal part of the forearm, only non-active smoking and non-childhood ETS exposure seems to guarantee a high T-score prox (Fig. 2).

Figure 3 presents a graphical representation of the relationships of years of the active smoking category

Table 3. Relationships between bone mineral density (BMD), bone mineral content (BMC) and T-score in the distal and proximal part of the forearm and BMI, MET and smoking parameters (multiple forward stepwise regression)

Bone parameters	Predictor	Standardized β	Adjusted R^2	F (p)
BMD dis	BMI	0.194	0.24	34.99 (< 0.001)
	MET	0.203		
	AS ∑years	–0.070		
	AS cigarette/day	–0.015		
	Age when starting AS	–0.091		
	ETS ∑years	–0.145		
BMD prox	BMI	0.226	0.33	54.43 (< 0.001)
	MET	0.253		
	AS ∑years	–0.034		
	AS cigarette/day	–0.143		
	Age when starting AS	–0.024		
	ETS ∑years	–0.159		
BMC dis	BMI	0.205	0.23	34.87 (< 0.001)
	MET	0.153		
	AS ∑years	–0.101		
	AS cigarette/day	–0.003		
	Age when starting AS	–0.129		
	ETS ∑years	–0.129		
BMC prox	BMI	0.223	0.25	36.86 (< 0.001)
	MET	0.169		
	AS ∑years	–0.013		
	AS cigarette/day	–0.073		
	Age when starting AS	–0.152		
	ETS ∑years	–0.101		
T-score dis	BMI	0.065	0.29	45.23 (< 0.001)
	MET	0.383		
	AS ∑years	–0.144		
	AS cigarette/day	–0.070		
	Age when starting AS	0.096		
	ETS ∑years	–0.113		
T-score prox	BMI	0.096	0.41	76.10 (< 0.001)
	MET	0.421		
	AS ∑years	–0.061		
	AS cigarette/day	–0.385		
	Age when starting AS	0.395		
	ETS ∑years	–0.233		

AS — active smoking; BMC — bone mineral content; BMD — bone mineral density; BMI — body mass index; dis — in the distal part of forearm; prox — in the proximal part of forearm; ETS — environmental tobacco smoke; MET — metabolic equivalent of task; p — p-value

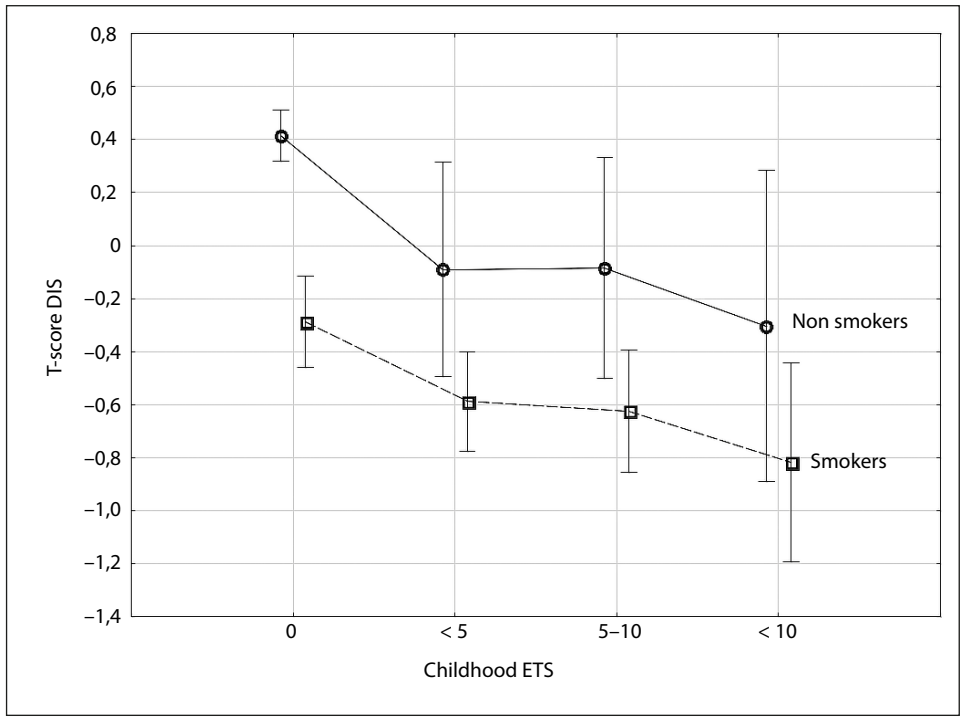

Figure 1. Relationships of childhood ETS years category and active smoking category with T-score dis (two-way ANOVA results, F(3, 649) = 0.357; p = 0.784), vertical lines –0.95 CI — confidence intervals

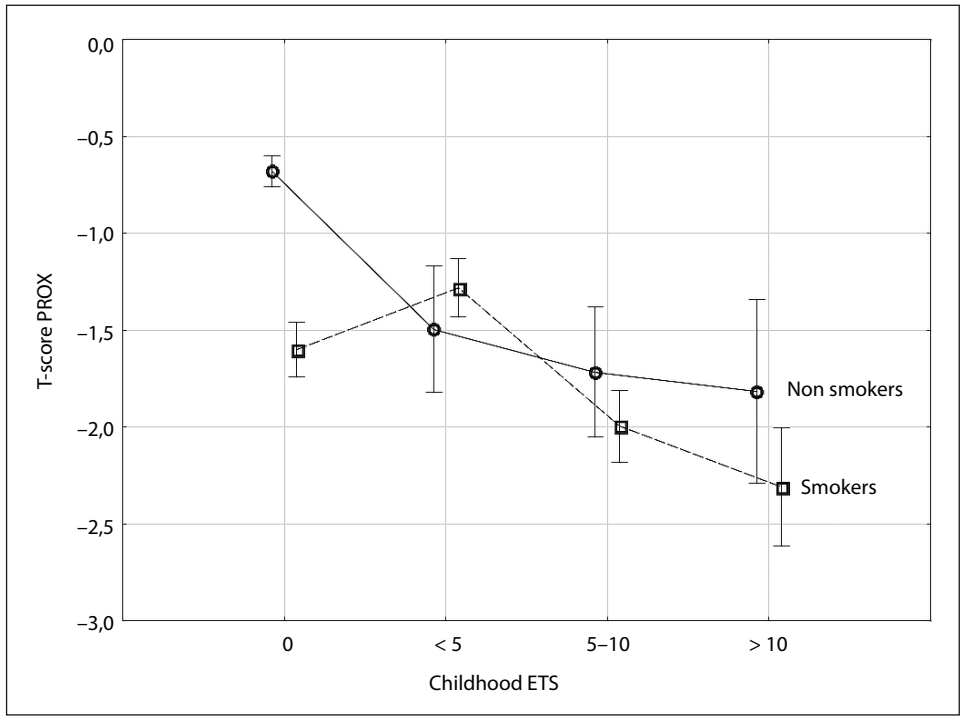

Figure 2. Relationships of childhood ETS years category and active smoking category with **T-score prox** (two-way ANOVA results, F(3, 649) =12.451; p = 0.000), vertical lines –0.95 CI — confidence intervals

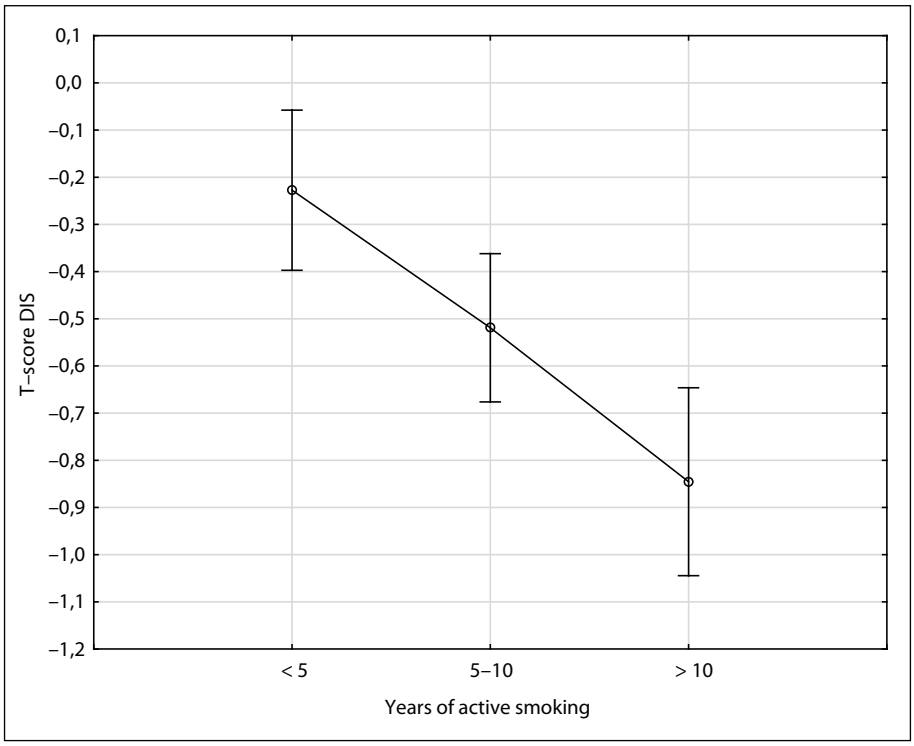

Figure 3 a. Relationships of Years of the active smoking category with **T-score dis** (one-way ANOVA results, F(2, 272) = 10,849; p < 0.001), vertical lines –0.95 CI — confidence intervals

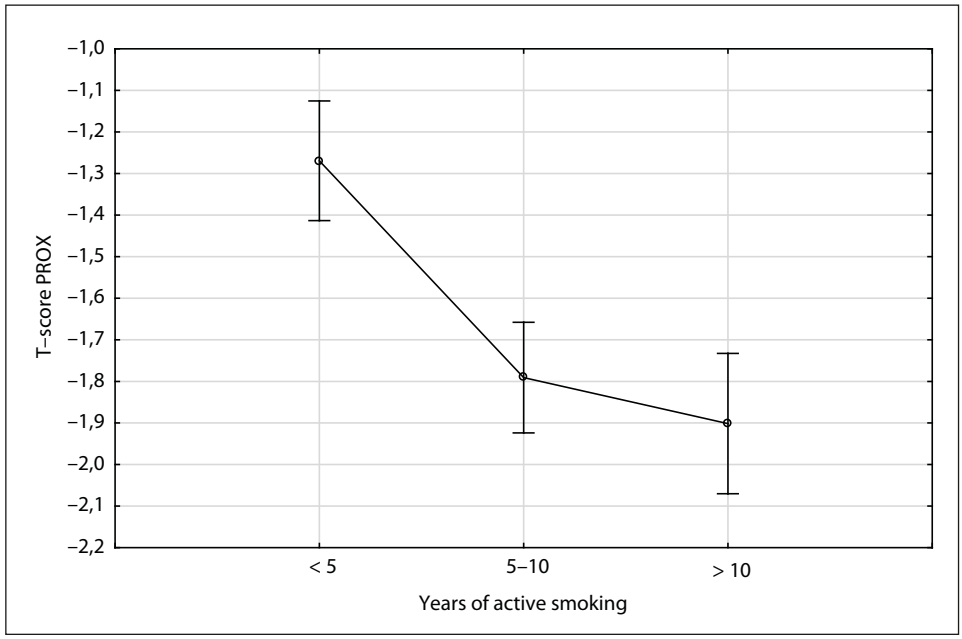

Figure 3 b. Relationships of Years of the active smoking category with **T-score prox** (one-way ANOVA results, F(2, 272) = 19.986; p < 0.001), vertical lines –0.95 CI — confidence intervals

with T-score in the distal (a) and proximal part (b) of the forearm. Women with less than 5 years of active smoking had the most advantageous values of T-score dis and prox. Significantly statistically the lowest T-score values were in women who had been active smokers for more than 10 years (Figs. 3a, b).

Discussion

The present cross-sectional observational study assessed the impact of both modifiable lifestyle risk behaviours such as childhood ETS exposure and active smoking (AS) on BMD in the forearm in young women. Smoking women had significantly smaller bone parameters in two parts of the forearm compared to non-smoking women. Significantly longer exposure to ETS (by 4 years) was found in smoking women. The highest prevalence of reduced BMD occurred in smoking women. In the proximal part of the forearm, low BMD (osteopenia) and osteoporosis risk were found in more than 80% of individuals from the group of smokers.

Some of the first studies documented inverse relationships between smoking and low bone mass, low bone mineral density, and fracture risk [10, 33, 34]. Contrary to these results, several studies have found no evidence of a relationship between smoking and BMC and BMD [10, 35]. Difficulties in comparing the results of studies by different authors may be due to different ways to measure exposure to smoking, in different populations. Studies have mainly focused on evaluating the effect of active smoking on BMC, BMD, and fracture risk [13, 23]. Studies of the effects of smoking on bone parameters in women are often divided into studies of premenopausal [14, 35] and postmenopausal women [13, 20, 36]. Studies of postmenopausal women are the most numerous and evaluate the risk of bone fractures in various skeletal locations in smokers [33, 36]. A review of studies on the effects of smoking on the bones of young women showed conflicting results. Most previous studies of premenopausal and early postmenopausal women have found no association between smoking and low BMC and BMD (osteopenia) [37]. A meta-analysis including 29 cross-sectional studies and 19 cohort or case-control studies confirmed that smoking has no major effect on premenopausal bone density [34]. Data from a large Danish national cohort study showed significant negative associations of current cigarette smoking ex-, or never smoking with bone mass in the lumbar spine, femoral neck, and total body ($p < 0.001$). However, differences between current smokers and never smokers were limited to 3%, which is significantly less than in the present study of young Polish women. In a study by Hermann et al. [38], the authors found no effect of smoking on forearm BMD.

In the current study of young Polish women, there was a significant effect of smoking on the prevalence of low BMD in the forearm, especially in the proximal segment. In smoking women, low BMD was found in the distal segment in 30%. There was a significant difference in the prevalence of osteopenia and osteoporosis risk in both forearm segments between smoking and non-smoking women (24.5% in the distal segment and 40.5% in the proximal segment). Similarly, in a study of women aged 35 years and older, BMD in smokers was 8% lower than in non-smokers [39]. A study of healthy young women found that at 2 years of follow-up, smokers aged 20–39 years had lower spinal BMD than non-smokers [37]. Compared to previous studies of a similar age group and the same ethnic group of women [28] the prevalence of low BMD in this study was high. In the present study, the interactions of three variables such as physical activity (MET), BMI (positive direction), and years of exposure to ETS (negative direction) were significant for BMD dis and BMC dis. The predictor of interactions of physical activity (MET), BMI (positive direction), and active smoking in numbers of cigarettes per day and years of exposure to ETS (negative direction) was significant for BMD prox. Interactions of MET, BMI (additive direction), age of starting active smoking, and years of exposure to ETS (negative direction) were found to be significant for BMC prox. Physical activity in MET (positive direction) and years of active smoking and exposure to ETS (negative direction) was significant predictor for T-score dis. The predictor of interactions of five variables: physical activity (MET), BMI, age of starting active smoking (positive direction), active smoking in numbers of cigarettes per day, and years of exposure to ETS (negative direction) was significant for T-score prox. As argued by Wong [23], smoking is a major lifestyle risk factor for osteoporosis. However, this effect appears dose-dependent and maybe, at least partially, reversible [23]. The research shows that the negative impact of smoking is already visible in young women who are still in the phase of building peak bone mass.

The present study of young Polish women found significantly lowest T-score dis and prox of the forearm, depending on the number of years of active smoking and years of ETS exposure. A significantly worse bone status occurred in women who actively smoked for more than 10 years. The worst results of bone parameters were found in women with ETS exposure of over 10 years. The dose of active smoking (AS in years) also led to low bone status. A prospective cohort study involving 121,701 female subjects aged 30–55 years found a dose-dependent increase in hip fracture rates in current smokers compared to never-smokers [40]. Cornuz et al. [40] concluded that smokers are at increased risk of hip fracture while the risk rises with

greater cigarette consumption. The risk declines among former smokers, but the benefits are not observed until 10 years after cessation. These studies indicated a potential protective effect of physical activity and BMI on bone parameters. Adequate body weight and MET showed significant positive effects on almost all bone parameters.

The effect of PS, including that of exposure to ETS, on bone status in young women has been the subject of some studies [41–43]. The effect of passive smoking on alveolar bone density and bone resorption was examined in 60 females aged 30 to 45 years. In this study, exposure to passive smoking was destructive and led to decreased bone density and height of the supporting structure of teeth [42]. Passive smoke exposure was linked to the risk of osteoporosis in adults in a study cohort including 1,422 individuals aged from 3 to 18 years. Parental smoking in childhood was associated with a lower bone sum index in adulthood [43]. One retrospective study of premenopausal women found that exposure to household tobacco smoke during adolescence and young adulthood is negatively associated with BMD at the total hip and femoral neck, whereas the duration of exposure was negatively associated with BMD in premenopausal women [41]. Far fewer studies describe the effect of ETS on forearm BMD. The condition of the bone tissue of the forearm has a significant impact on the quality of manual activities important in daily life and the level of functional efficiency.

In this study, the factor of good socio-economic status did not protect the studied women from the high frequency of low BMD. In studies by other authors, it is most often stated that low education and income levels were more highly associated with osteoporosis prevalence in women than in men [44]. Evaluation of the relationship between socio-economic status and bone mineral density (BMD) in 4446 men aged ≥ 20 years from the NHANES showed that individuals with the highest degree (college degree or above) had significantly greater lumbar BMD than that of the lowest degree [45]. Other studies have highlighted that childhood socio-economic advantage and adult education level were associated with higher adult lumbar spine BMD and financial advantage was not associated with BMD. The authors showed that childhood socio-economic factors may influence the acquisition of lumbar BMD [46]. Studies evaluating the impact of socio-economic factors on BMD of the forearm, a skeletal location important for functional performance, are still lacking.

In young women, early detection of the risk of low BMD allows for taking effective prophylactic measures and reducing the risk of osteopenia. The major strength of the study is a multifactorial analysis of smoking determinants (active and ETS) of key forearm bone parameters that offers the opportunity to assess the strength and direction of the effect of several important and diverse determinants rather than a single determinant on BMD and BMC. Another strength of the present study is that a reliable and accurate research methodology was used. The research was conducted by a highly-qualified team with many years of research experience in the field. All data were collected using well-selected and internationally recommended research tools.

The study has some limitations. One of the study limitations is the relatively small yet sufficient size of the study group. It cannot provide a full representation of the population of Polish young women at this age, although it is satisfactory for drawing conclusions concerning the effect of smoking on bone parameters. Screening in all regions of the skeleton would be of great value. This project did not determine biochemical blood indicators and did not assess nutrition and calcium, protein, and vitamin D intake in diets. This would provide detailed information on the determinants of BMD. Despite these limitations, this study may be an important contribution to the implications results of scientific study for Practice and/or Policy. These findings can potentially contribute to the development of more effective public health strategies for health promotion and osteoporosis prevention in this population. Education about the negative effects of environmental tobacco smoke on bone health and an indication of the ETS-associated increased risk of low BMD in adulthood can help in the effective prevention of osteopenia and osteoporosis in women.

In conclusion, cigarette smoking and exposure to environmental tobacco smoke are modifiable determinants of low bone mineral density in young Caucasian women of European origin. Despite the women's good socio-economic status, a high, alarming incidence of low BMD was reported. The results expand the knowledge of the multifactorial determinants of forearm bone mineral density. Active smoking and exposure to ETS should be considered in screening the bone status of the young population.

Acknowledgements: *The Authors would like to thank all Participants for their time and participation in the scientific project.*

Conflict of interest: *None.*

Funding: *This work was supported by the Ministry of Science and Higher Education in 2020/2022 as part of the Scientific School of the University of Physical Education in Warsaw — SN No. 5 "Biomedical determinants of physical fitness and sports training in adult population".*

References

1. Nguyen TV, Center JR, Eisman JA. Osteoporosis in elderly men and women: effects of dietary calcium, physical activity, and body mass index. J Bone Miner Res. 2000; 15(2): 322–331, doi: 10.1359/jbmr.2000.15.2.322, indexed in Pubmed: 10703935.
2. World Health Organization. Global status report on noncommunicable diseases. Geneva: WHO, 2010. https://apps.who.int/iris/bitstream/handle/10665/44579/9789240686458_eng.pdf?sequence=1 (19.03.2023).
3. Li K, Yao C, Di X, et al. Smoking and risk of all-cause deaths in younger and older adults: a population-based prospective cohort study among beijing adults in china. Medicine (Baltimore). 2016; 95(3): e2438, doi: 10.1097/MD.0000000000002438, indexed in Pubmed: 26817876.
4. Kyrou I, Tsigos C, Mavrogianni C, et al. Feel4Diabetes-study Group. Sociodemographic and lifestyle-related risk factors for identifying vulnerable groups for type 2 diabetes: a narrative review with emphasis on data from Europe. BMC Endocr Disord. 2020; 20(Suppl 1): 134, doi: 10.1186/s12902-019-0463-3, indexed in Pubmed: 32164656.
5. Hao Z, Wang M, Zhu Q, et al. Association between socioeconomic status and prevalence of cardio-metabolic risk factors: a cross-sectional study on residents in north china. Front Cardiovasc Med. 2022; 9: 698895, doi: 10.3389/fcvm.2022.698895, indexed in Pubmed: 35330947.
6. Lopuszanska-Dawid M. Trends in health behavior of Polish women in 1986-2021: the importance of socioeconomic status. Int J Environ Res Public Health. 2023; 20(5), doi: 10.3390/ijerph20053964, indexed in Pubmed: 36900975.
7. Brennan SL, Pasco JA, Urquhart DM, et al. Association between socioeconomic status and bone mineral density in adults: a systematic review. Osteoporos Int. 2011; 22(2): 517–527, doi: 10.1007/s00198-010-1261-y, indexed in Pubmed: 20449573.
8. O'Keeffe LM, Taylor G, Huxley RR, et al. Smoking as a risk factor for lung cancer in women and men: a systematic review and meta-analysis. BMJ Open. 2018; 8(10): e021611, doi: 10.1136/bmjopen-2018-021611, indexed in Pubmed: 30287668.
9. Hudson NL, Mannino DM. Tobacco use: a chronic illness? J Community Health. 2010; 35(5): 549–553, doi: 10.1007/s10900-010-9241-x, indexed in Pubmed: 20177752.
10. Ward KD, Klesges RC. A meta-analysis of the effects of cigarette smoking on bone mineral density. Calcif Tissue Int. 2001; 68(5): 259–270, doi: 10.1007/BF02390832, indexed in Pubmed: 11683532.
11. Al-Bashaireh AM, Haddad LG, Weaver M, et al. The effect of tobacco smoking on bone mass: an overview of pathophysiologic mechanisms. J Osteoporos. 2018; 2018: 1206235, doi: 10.1155/2018/1206235, indexed in Pubmed: 30631414.
12. Ratajczak AE, Szymczak-Tomczak A, Rychter AM, et al. Impact of cigarette smoking on the risk of osteoporosis in inflammatory bowel diseases. J Clin Med. 2021; 10(7), doi: 10.3390/jcm10071515, indexed in Pubmed: 33916465.
13. Kanis JA, Johnell O, Oden A, et al. Smoking and fracture risk: a meta-analysis. Osteoporos Int. 2005; 16(2): 155–162, doi: 10.1007/s00198-004-1640-3, indexed in Pubmed: 15175845.
14. Jones G, Scott FS. A cross-sectional study of smoking and bone mineral density in premenopausal parous women: effect of body mass index, breastfeeding, and sports participation. J Bone Miner Res. 1999; 14(9): 1628–1633, doi: 10.1359/jbmr.1999.14.9.1628, indexed in Pubmed: 10469293.
15. Florescu A, Ferrence R, Einarson T, et al. Methods for quantification of exposure to cigarette smoking and environmental tobacco smoke: focus on developmental toxicology. Ther Drug Monit. 2009; 31(1): 14–30, doi: 10.1097/FTD.0b013e3181957a3b, indexed in Pubmed: 19125149.
16. WHO Report on the Global Tobacco Epidemic, 2008: the MPOWER package. http://apps.who.int/iris/bitstream/handle/10665/43818/9789241596282_eng.pdf?sequence=1 (19.03.2023).
17. Merianos AL, Odar Stough C, Nabors LA, et al. Tobacco smoke exposure and health-care utilization among children in the united states. Am J Health Promot. 2018; 32(1): 123–130, doi: 10.1177/0890117116686885, indexed in Pubmed: 29214835.
18. McEvoy CT, Spindel ER. Pulmonary effects of maternal smoking on the fetus and child: effects on lung development, respiratory morbidities, and life long lung health. Paediatr Respir Rev. 2017; 21: 27–33, doi: 10.1016/j.prrv.2016.08.005, indexed in Pubmed: 27639458.
19. Peterson LA, Hecht SS. Tobacco, e-cigarettes, and child health. Curr Opin Pediatr. 2017; 29(2): 225–230, doi: 10.1097/MOP.0000000000000456, indexed in Pubmed: 28059903.
20. Giampietro PF, McCarty C, Mukesh B, et al. The role of cigarette smoking and statins in the development of postmenopausal osteoporosis: a pilot study utilizing the Marshfield Clinic Personalized Medicine Cohort. Osteoporos Int. 2010; 21(3): 467–477, doi: 10.1007/s00198-009-0981-3, indexed in Pubmed: 19506792.
21. Li H, Wallin M, Barregard L, et al. Smoking-induced risk of osteoporosis is partly mediated by cadmium from tobacco smoke: the mros sweden study. J Bone Miner Res. 2020; 35(8): 1424–1429, doi: 10.1002/jbmr.4014, indexed in Pubmed: 32191351.
22. Cusano NE. Skeletal effects of smoking. Curr Osteoporos Rep. 2015; 13(5): 302–309, doi: 10.1007/s11914-015-0278-8, indexed in Pubmed: 26205852.
23. Wong PKK, Christie JJ, Wark JD. The effects of smoking on bone health. Clin Sci (Lond). 2007; 113(5): 233–241, doi: 10.1042/CS20060173, indexed in Pubmed: 17663660.
24. Jacobs-van der Bruggen MAM, Wijga AH, Brunekreef B, et al. Do parents who smoke underutilize health care services for their children? A cross sectional study within the longitudinal PIAMA study. BMC Health Serv Res. 2007; 7: 83, doi: 10.1186/1472-6963-7-83, indexed in Pubmed: 17565678.
25. Łopuszańska-Dawid M, Szklarska A. Growth change in Polish women: Reduction of the secular trends? PLoS One. 2020; 15(11): e0242074, doi: 10.1371/journal.pone.0242074, indexed in Pubmed: 33253200.
26. Hall JG, Allanson JE, Gripp KW, Slavotinek AM. Handbook of physical measurements. Oxford University Press, Oxford 2007.
27. World Health Organization. Obesity: Preventing and managing the global epidemic Report of a WHO consultation. https://apps.who.int/iris/handle/10665/42330 (19.03.2023).
28. Kopiczko A, Łopuszańska-Dawid M, Gryko K. Bone mineral density in young adults: the influence of vitamin D status, biochemical indicators, physical activity and body composition. Arch Osteoporos. 2020; 15(1): 45, doi: 10.1007/s11657-020-0684-0, indexed in Pubmed: 32166587.
29. Hans D, Downs RW, Duboeuf F, et al. International Society for Clinical Densitometry. Skeletal sites for osteoporosis diagnosis: the 2005 ISCD Official Positions. J Clin Densitom. 2006; 9(1): 15–21, doi: 10.1016/j.jocd.2006.05.003, indexed in Pubmed: 16731427.
30. Lu Y, Genant HK, Shepherd J, et al. Classification of osteoporosis based on bone mineral densities. J Bone Miner Res. 2001; 16(5): 901–910, doi: 10.1359/jbmr.2001.16.5.901, indexed in Pubmed: 11341335.
31. Craig CL, Marshall AL, Sjöström M, et al. International physical activity questionnaire: 12-country reliability and validity. Med Sci Sports Exerc. 2003; 35(8): 1381–1395, doi: 10.1249/01.MSS.0000078924.61453.FB, indexed in Pubmed: 12900694.
32. Kaleta D, Kozieł A, Miśkiewicz P. Global Adult Tobacco Survey in Poland - the aim and current experiences. Med Pr. 2009; 60(3): 197–200, indexed in Pubmed: 19746887.
33. Cooper C, Wickham C. Cigarette smoking and the risk of age-related fractures. In: Wald N, Baron J. ed. Smoking and hormone-related disorders. Oxford University Press, Oxford 1990: 93–100.
34. Law MR, Hackshaw AK. A meta-analysis of cigarette smoking, bone mineral density and risk of hip fracture: recognition of a major effect. BMJ. 1997; 315(7112): 841–846, doi: 10.1136/bmj.315.7112.841, indexed in Pubmed: 9353503.
35. Daniel M, Martin AD, Drinkwater DT. Cigarette smoking, steroid hormones, and bone mineral density in young women. Calcif Tissue Int. 1992; 50(4): 300–305, doi: 10.1007/BF00301626, indexed in Pubmed: 1571840.
36. Vestergaard P, Mosekilde L. Fracture risk associated with smoking: a meta-analysis. J Intern Med. 2003; 254(6): 572–583, doi: 10.1111/j.1365-2796.2003.01232.x, indexed in Pubmed: 14641798.
37. Mazess RB, Barden HS. Bone density in premenopausal women: effects of age, dietary intake, physical activity, smoking, and birth-control pills. Am J Clin Nutr. 1991; 53(1): 132–142, doi: 10.1093/ajcn/53.1.132, indexed in Pubmed: 1984338.
38. Hermann AP, Brot C, Gram J, et al. Premenopausal smoking and bone density in 2015 perimenopausal women. J Bone Miner Res. 2000; 15(4): 780–787, doi: 10.1359/jbmr.2000.15.4.780, indexed in Pubmed: 10780870.
39. Baheiraei A, Pocock NA, Eisman JA, et al. Bone mineral density, body mass index and cigarette smoking among Iranian women: implications for prevention. BMC Musculoskelet Disord. 2005; 6: 34, doi: 10.1186/1471-2474-6-34, indexed in Pubmed: 15975151.
40. Cornuz J, Feskanich D, Willett WC, et al. Smoking, smoking cessation, and risk of hip fracture in women. Am J Med. 1999; 106(3): 311–314, doi: 10.1016/s0002-9343(99)00022-4, indexed in Pubmed: 10190380.
41. Blum M, Harris SS, Must A, et al. Household tobacco smoke exposure is negatively associated with premenopausal bone mass. Osteoporos Int. 2002; 13(8): 663–668, doi: 10.1007/s001980200090, indexed in Pubmed: 12181626.
42. El-Batran MM, Soliman NL, Mikhael FF. Passive smoking and alveolar bone density. Aust J Basic Appl Sci. 2009; 3(2): 713–719.

43. Juonala M, Pitkänen N, Tolonen S, et al. Childhood exposure to passive smoking and bone health in adulthood: the cardiovascular risk in young finns study. J Clin Endocrinol Metab. 2019; 104(6): 2403–2411, doi: 10.1210/jc.2018-02501, indexed in Pubmed: 30715377.
44. Noh JW, Park H, Kim M, et al. Gender differences and socioeconomic factors related to osteoporosis: a cross-sectional analysis of nationally representative data. J Womens Health (Larchmt). 2018; 27(2): 196–202, doi: 10.1089/jwh.2016.6244, indexed in Pubmed: 28832241.
45. Xiao PL, Fuerwa C, Hsu CJ, et al. Socioeconomic status influences on bone mineral density in American men: findings from NHANES 2011--2020. Osteoporos Int. 2022; 33(11): 2347–2355, doi: 10.1007/s00198-022-06498-5, indexed in Pubmed: 35833955.
46. Crandall CJ, Merkin SS, Seeman TE, et al. Socioeconomic status over the life-course and adult bone mineral density: the Midlife in the U.S. Study. Bone. 2012; 51(1): 107–113, doi: 10.1016/j.bone.2012.04.009, indexed in Pubmed: 22543227.