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Socioeconomic and biochemical profiles of smokers in Poland

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**WHAT’S NEW**

Smoking may exaggerate an unfavourable cardiovascular profile through various pathways, including increasing inflammation, cholesterol and potassium, and decreasing bilirubin and creatinine level. This is the first cross-sectional study that confirmed the association between adverse biochemical profile and smoking in Poland. However, due to the lack of a longitudinal assessment, it would be difficult to infer the temporal association between a risk factor and an outcome. The most important finding in this study was the existence of a social gradient in tobacco use in the Polish population, which identified unmarried people with lower socioeconomic status who were from small towns to be more likely to use tobacco, and have an unfavourable biochemical profile. It is not evident what the temporal relations are in mechanisms involved in the gradient observed between biochemical markers and smoking. Therefore, we still need measures targeted at the most disadvantaged to prevent health risks caused by smoking.
ABSTRACT

Introduction: Epidemiological studies show a fairly constant association between socioeconomic status and smoking. However, associations between smoking and biological indicators of health status have not been well described.

Objectives: To determine the relationship between smoking, biochemical risk factors, and sociodemographic characteristics in the Polish population.

Patients and methods: This survey was carried out on a representative sample of Polish residents aged 18–79 years. A total of 2,413 randomly selected subjects participated in the survey. Logistic regression analysis and parametrical and non-parametrical tests were performed.

Results: Statistically significantly higher cholesterol, apolipoprotein B, and potassium levels were observed both in smoking women and in smoking men compared to the non-smoking population. Statistically significantly lower bilirubin levels were noted in smoking individuals. Higher C-reactive protein and lower creatinine levels were reported only in the male smoking population compared to non-smokers. There was a statistically significant inverse gradient observed in the relationship between income and smoking. Single women and men were at statistically significant higher odds of being smokers (odds ratio [OR] 1.9 and OR 2.39, respectively). Individuals from small towns (less than 50,000 inhabitants) were at statistically significant higher odds of smoking compared to those living in rural areas (OR 1.45 and OR 1.64 in women and men, respectively).

Conclusions: We found differences regarding socioeconomic characteristics and major biochemical parameters between smokers and non-smokers in Poland; however, it is difficult to establish which associations are causal for cardiovascular risk due to the cross-sectional design of this study.
**INTRODUCTION**

Smoking is widely believed to be the strongest single adverse health factor [1-4]. Epidemiological studies show a fairly constant association between socioeconomic status and smoking, with a higher prevalence of smoking among those who are poorer and less educated [5,6]. Although a negative effect of low socioeconomic status and smoking on mortality risk and health status has been clearly established, associations between smoking and other biological indicators of health status have not been well described. Associations between smoking and single clinical parameters and socioeconomic status have been indicated in the literature [7-9]. Lower body mass has also been related to smoking [10]. A relation has been shown between smoking and loneliness and mental health problems [11]. In the search for a more precise model of adverse effects of nicotine, attempts have been made to characterise in more detail how smoking and other health behaviours are related to biochemical and even metabolomic correlates of the effects of smoking [12-14]. Elucidating the associations between smoking and selected biological determinants of smoking-related disease may help understand their pathomechanism and identify possible ways to decrease their risk. Additionally, the role of the new non-traditional cardiovascular risk factors, like serum bilirubin and creatinine levels, in the prediction of cardiovascular events need to be better understood also in the context of smoking [15,16]. So far, there was no attempt to compare both biochemical and socioeconomic multiple cardiovascular risk profiles between smokers and non-smokers in Central-Eastern Europe.
The aim of this study is to assess the relationship between smoking and pattern of biochemical risk factors, and sociodemographic characteristic in a Central-Eastern European population, based on data collected in a representative sample of the Polish general population in the NATPOL 2011 (Arterial hypertension and other cardiovascular disease risk factors in Poland; full Polish title: Naciskienie tętnicze oraz inne czynniki ryzyka chorób serca i naczyń w Polsce) study.

PATIENTS AND METHODS

The NATPOL 2011 survey was designed as a cross-sectional representative observational study. It was carried out in a representative sample of Polish residents aged 18–79 years. The participants were randomly selected in bundles using a stratified, proportional draw performed in three stages. Overall, 2,413 subjects (1,245 females and 1,168 males) participated in the survey. The response rate among respondents who were invited and eligible for the study was 66.4%. The survey fieldwork was carried out by 234 well-trained nurses who lived in or close to the randomly selected geographical bundles. The participants were examined during two home visits. Evaluation of an individual subject included the following components: completion of a questionnaire, blood pressure readings and anthropometric measurements, and blood and urine sample collection. The questionnaire was completed during the first visit. Blood pressure readings were taken three times during the first and the second visit using a fully automatic oscillometric blood pressure measuring device (A&D UA 767). Arterial hypertension was diagnosed according to the 2007 ESH/ESC Guidelines for the Management of Arterial Hypertension if during both visits the systolic blood pressure was ≥140 mmHg or diastolic blood pressure was ≥90 mmHg, or the patient was taking antihypertensive drugs over the previous two weeks due to previously diagnosed
hypertension. Overweight was defined as body mass index (BMI) 25.0-29.9 kg/m\(^2\), and obesity as BMI ≥30 kg/m\(^2\). Smoking was defined as active regular smoking of at least one cigarette per day. Education levels were divided into the following categories: primary (includes vocational), secondary (includes incomplete higher education i.e. without a master's degree), and higher education. Blood and urine samples were taken from the subjects during the second visit, after 10 to 12 hours of fasting. However, participants were allowed to drink water while fasting. Frozen samples were transported to a central laboratory for blood and urine analyses.

The study protocol was approved by the Institutional Ethics Committee at the Medical University in Gdansk, and all participants provided written informed consent. Details on the questionnaire, sample selection, and laboratory parameters have been published previously [17].

**Statistical analysis**

The sample size calculation was based on the assumption that the amount of acceptable (or allowable) margin of error in the estimation for prevalence of smoking or hypertension within different gender groups was not greater than 3%. The calculated sample size was 2400 participants. Data are presented as the number and percentage of patients, mean values with standard deviation (SD) or median and quartile 1 and quartile 3 for not-normally distributed data. For normally distributed continuous variables, the Student’s t-test for independent samples was applied. For variables that did not follow a normal distribution, the Mann-Whitney U test was used for comparison of independent measurements. The Kruskal-Wallis test or one-way ANOVA was used for comparison of multiple groups depending on whether the data fit the assumptions of normality. Differences between categorical variables were tested using the chi-square test. Data on income was grouped into quartiles. Logistic
regression analysis was performed to identify characteristics associated with smoking status. The following variables, for which \( P \leq 0.2 \) in the univariable analysis, were included in the multivariable logistic regression analysis: BMI, education, income, place of residence, marital status, hypercholesterolaemia, and hypertension. Former smokers were classified as non-smokers in all analyses. \( P < 0.05 \) was considered statistically significant. Smoking was a dependent variable for models with sociodemographic and some clinical characteristic and in other health outcomes analyses it was an independent variable. All statistical analyses were performed using STATA software (version 12.1, STATA Corp.).

**Patient and Public Involvement**

This research was done without patient involvement. Patients were not invited to comment on the study design and were not consulted to develop patient relevant outcomes or interpret the results. Patients were not invited to contribute to the writing or editing of this document for readability or accuracy.

**RESULTS**

Overall, the study included 2,413 respondents (1,245 women and 1,168 men). The average age (SD) was 47 (17) years in women and 45 (16) years in men. The study group included 331 (26.6%) female smokers and 402 (34.4%) male smokers. Former smokers accounted for 21.2% of women and 31.6% of men. More than half of the surveyed women (52.2%) and 34% of men declared that they had never smoked tobacco. The percentage of smokers differed significantly between men and women (\( P < 0.001 \)).

*Table 1 here*
Table 1 shows the mean or median values of the parameters analysed in the study in men and women. Men were found to have statistically significantly higher values of BMI, apolipoprotein B (apoB), bilirubin, creatinine, and potassium levels, fasting blood glucose, and systolic and diastolic blood pressure.

Table 2 here

The prevalence of hypertension was higher in smoking men than in smoking women (Table 2). We found no statistically significant increase in the odds of hypertension among smoking men and women compared to non-smokers. Both smoking women and men had a 40% lower odds of excess body weight in comparison to non-smokers. The proportion of women who believed that they were overweight or obese was about 40%, and similarly among smokers and non-smokers. Smoking men had a significantly lower chance of perceiving their body weight as excessive (odds ratio [OR] 0.64, 95% confidence interval [CI] 0.49-0.83).

Table 3 here

Smokers were younger and thinner than non-smokers. We found statistically significant differences in the mean age and BMI between smoking and non-smoking women and men (Table 3). Significantly higher fasting blood glucose levels were observed in the oldest group of female non-smokers (P= 0.02) and in male smokers aged 18-39 years (P=0.02) in comparison to their peers with the opposite smoking status. Significantly higher cholesterol levels were observed in smoking women, and this difference between smokers and non-smokers increased with the age of the respondents. In smoking men, significantly higher cholesterol levels were observed in all respondents compared to non-smokers (P=0.01). In smoking women, significantly higher apoB levels were observed in all respondents (P=0.002), in those aged 40-59 years (P=0.001), and in the oldest age group (P=0.046). In smoking men, significantly higher apoB levels were observed in all respondents (P=0.007) and in the oldest
age group (P=0.046). Average C-reactive protein (CRP) levels were not significantly different between smoking and non-smoking women. However, significant differences in CRP levels were noted between smoking and non-smoking men, and these differences increased with the age of the respondents (2.29 and 1.94 respectively, P=0.01 for those aged 40-59 years and 2.85 and 2.05 respectively, P<0.001 for those aged 60-79 years). No significant differences in creatinine levels were found between smoking and non-smoking women (P>0.05). In men, creatinine levels were significantly higher in non-smokers except for the oldest age group. Bilirubin levels were significantly lower in smoking women and men, except for the oldest age group of men (P=0.88). Potassium levels were significantly higher (P<0.05) in smoking women and men, except for the oldest group of women (P=0.09).

Table 4 here

Although smoking rates declined with age, the relation between smoking and age was of borderline significance (Table 4). Women with secondary education had an approximately 40% lower odds of smoking as demonstrated in the univariable and multivariable analyses in comparison to women with primary education. The effect of education was stronger among men: the likelihood of smoking was about 70% lower in men with secondary and incomplete higher education, and about 35% lower in men with higher education compared to primary education (OR 0.33, 95% CI 0.21-0.52 and OR 0.66, 95% CI 0.49-0.9, respectively). We found a strong relationship between the smoking status and the financial situation of the respondents. Univariable and multivariable analyses showed that the lower the income, the higher likelihood of smoking. Men and women in the upper quartile of income had a 50% lower likelihood of smoking compared to those in the lower quartile (OR 0.51, 95% CI 0.35-0.76 and OR 0.55, 95% CI 0.36-0.85, respectively). In both genders, highest smoking rates were observed in the group with the lowest income. In the univariable and multivariable analyses, the likelihood of smoking was also related to the marital status and was statistically
significantly higher in single subjects compared to married ones (adjusted OR 2.39, 95% CI 1.30-4.40 and OR 1.90, 95% CI 1.20-3.00 in men and women, respectively).

*Table 5 here*

Results of the univariable logistic regression analyses in women and men showed a statistically significant relationship between BMI and smoking (Table 5). However, the 95% confidence interval only reached a borderline value in the multivariable analysis for women. There was no association in logistic regression analysis between smoking status and hypertension. We found a 60% increased odds of hypercholesterolaemia in smoking women but no association between smoking and hypercholesterolaemia in men (OR 1.59, 95% CI 1.15-2.21 and OR 1.21, 95% CI 0.89-1.64, respectively).

**DISCUSSION**

Smoking is an established risk factor for malignancies, metabolic disease, and cardiovascular disease. A number of observations indicate that smoking is related to metabolic syndrome and changes in lipid levels [18]. A well-known and documented association exists between smoking and lower body mass, although this may lead to insulin resistance and an elevated risk of type 2 diabetes in heavy smokers [19]. Our findings confirm the observations of a lower rate of excess body weight in smokers compared to non-smokers [10,20]. The odds of obesity or overweight were significantly lower in smokers, by about 40% in both women and men (Table 2). In our study, smoking was associated with a significantly lower likelihood of self-perceived overweight or obesity in men but not in women (Table 2). The reason for a lower self-awareness of excess body weight among smoking men compared to women may be related to their ignorance regarding positive health behaviours, probably due to their lower
socioeconomic status and lower level of education [21]. Smoking is more common among those socially and economically disadvantaged, which was also found in our study [5,22]. Studies indicate that smokers consume less fruit and vegetables, drink more alcohol, and are less physically active compared to non-smokers [23,24]. Hypercholesterolaemia is a major risk factor for cardiovascular disease. In our study, we found a significantly higher cholesterol level in smokers compared to non-smokers (Table 3). Similar observations were made in other studies that also showed significantly higher cholesterol levels in smokers compared to non-smokers [25,26]. Our findings regarding apoB are also consistent with other reports of a significantly higher apoB level in smokers compared to non-smokers (Table 3) [25]. Insulin plays a key role in the regulation of apoB and the insulin resistant-state is associated with increased secretion and decreased clearance of apoB. This is the likely mechanism of observed increased blood glucose level in some age groups of smokers. Overall, fasting blood glucose level was not significantly different between smokers and non-smokers in our study. However, detailed subgroup analysis showed that among women in the oldest age group (60-79 years), the mean fasting blood glucose level was significantly higher in non-smokers compared to smokers, while among men in the youngest age group (18-39 years), the mean fasting blood glucose level was significantly higher in smokers compared to non-smokers (Table 3). The effect of smoking on blood glucose control may vary depending on the history of smoking and the presence of type 2 diabetes in a smoking subject. It is known that the risk of type 2 diabetes and glucose intolerance depends on the nicotine dose in smoked cigarettes [27]. Longitudinal studies showed that in smokers with type 2 diabetes, quitting was paradoxically associated with worse glycaemic control for up to three years [28]. As we did not include ex-smokers as a separate group in the analysis, we were not informed about the possible reversibility of these changes; nonetheless, this survey is not able to verify a causal relationship. In the short-term, smoking is usually associated with an increase in blood
glucose levels, and hormonal effects of smoking combined with a reduction of visceral fat which also results in higher long-term blood glucose levels despite a reduction in BMI [29,30]. Based on our cross-sectional study, it is difficult to draw conclusions about causal relationships, but the observed differences in blood glucose levels between age groups and genders may have been related to differences in behaviours and smoking history in these groups. Similarly, it is difficult to clearly interpret the specific patterns found in our study in terms of the relationship between smoking and CRP and creatinine levels (Table 3). The CRP levels were higher, and creatinine levels were lower in smokers compared to non-smokers, which is consistent with literature data [31]. Studies indicate a potential role of CRP in smokers as a mediator of glomerular hyperfiltration, an increase in proteinuria, and kidney dysfunction in a healthy population [32]. Smoking is known to have varying effects on vascular endothelial physiology and biomarkers of inflammation depending on gender, which may to some extent explain the different patterns of the levels of these markers observed in men and women [33,34].

Epidemiological studies show an inverse relation between bilirubin levels and the risk of cardiovascular disease, stroke, and metabolic syndrome [35-37]. Our study showed significantly lower bilirubin levels in smokers compared to non-smokers, both overall and in all age- and gender-specific groups except for men aged 60-79 years (Table 3). A role of bilirubin in biological pathways that lead to smoking-related disease has been suggested by reports of a higher risk of lung cancer in subjects with low bilirubin levels [14]. In our study, potassium levels were significantly lower in non-smokers, which is also associated with a lower risk of cardiovascular mortality [38]. The increase in potassium levels may be attributed to cigarette smoking induced skeletal muscle damage which may leak cellular contents along with potassium into the serum [39].
Rates of smoking are known to be inversely proportional to socioeconomic status [5,6,40]. In the present study, we found clear associations between socioeconomic factors and smoking. In the multivariable analysis, the odds of smoking in women with secondary or incomplete higher education were nearly 40% lower compared to women with primary or incomplete secondary education (Table 4). The proportion of smokers among women with higher education was similar to that among women with primary education, which probably reflects the increase in the rate of smokers among women with higher education that has been observed in recent years. A simpler relation was seen in men: the higher the level of education, the lower the odds of being a smoker (Table 4). Another trend clearly seen in our data was that the higher the income level, the lower the rate of smoking. However, there was no statistically significant correlation between income and education in our data. These findings are consistent with other reports showing that subjects with the lowest income have the highest odds of initiating smoking and are the least likely to quit smoking [41]. The reasons for the highest rates of smoking among those with the lowest income level are believed to include stress, family problems, daily life struggles (including financial problems), boredom, and social influences in a community with a large proportion of smokers [22]. These factors are most likely to characterise small-town communities where we found the highest odds of smoking in our study (Table 4). In addition, we found that being alone was associated with higher odds of smoking. These findings are consistent with literature data which show that the rate of smoking was more than twofold higher among single people compared to non-single people [42,43]. Data from a Polish study showed that being a single woman increased the risk of smoking [44]. These patterns of relations between smoking and socioeconomic status and loneliness correspond to the reported association of smoking with depressed mood, stress, and depression among smokers [11]. Longitudinal studies even show an increased suicidal risk
among smokers [45]. Although we did not evaluate mood changes in the present study, the role of inflammation markers as mediators of mood disorders suggests that smoking and an increased CRP level may be two components of a larger spectrum encompassing stress and depression. Loneliness is also associated with an increased CRP level, which was reported in the context of atherogenesis [46,47]. These associations are consistent with the postulated pathomechanism underlying the effect of smoking on the cardiovascular risk, and they are supported by the results of the present study. The inability to disentangle the effect of smoking, via insulin resistance, from different fasting blood glucose levels in different age and gender groups due to the lack of smoking history and type 2 diabetes data is a significant limitation in the interpretation of the findings in this study. Another major limitation of the present study is its cross-sectional design, precluding conclusions regarding temporality and potential causation. The practical application of potential risk prediction algorithms that incorporate different biochemical profiles of non-traditional risk factors requires further research.

The observed differences regarding major biochemical and clinical parameters between smokers and non-smokers in Poland indicate an adverse cardiovascular risk profile in smokers. We also found clear socioeconomic differences between smokers and non-smokers, which are probably major determinants of the increased cardiovascular risk associated with smoking in the Polish population. We observed that smoking behaviour differs substantially between lower and higher socioeconomic status, single and married individuals, and place of residence.

In order to revisit the role of socioeconomic factors in the development of the unfavourable biochemical profile among smokers, future studies would need to employ designs based on follow-up data collected in controlled settings.


**CONTRIBUTORSHIP:**

PZ conceived of the presented idea. TZ and ZG designed the study. PB was responsible for drawing the sample. MP was responsible for data analysis. MZ-B contributed to part of data-analysis and bibliographic searching. PJ was responsible for data collection and study management. AW as responsible for data collection and study management. TJ as responsible for data collection and study management. PP as responsible for data collection and study management. PZ wrote the manuscript with input from all authors.

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**CONFLICT OF INTEREST:** The authors declare that they have no conflict of interest.

**DATA SHARING STATEMENT:** Extra data is available by emailing corresponding author
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Table 1. Characteristics of the study population by gender.

|                          | Women         | Men           | P  |
|--------------------------|---------------|---------------|----|
| Overall [n, %]           | 1245 (51.6)   | 1168 (48.4)   |    |
| Smoking [n, %]           | 331 (26.6)    | 402 (34.4)    | <0.001 |
| BMI, kg/m² [median (Q1, Q3)] | 25.1 (22, 29.2) | 26.8 (24.1, 30) | <0.001 |
| Fasting blood glucose, mg/dL [median (Q1, Q3)] | 88 (83, 96)  | 92 (86, 101)  | <0.001 |
| Total cholesterol, mg/dL [median (Q1, Q3)] | 196 (171, 226) | 196 (168, 227) | 0.53 |
| apoB, g/L [median (Q1, Q3)] | 0.86 (0.7, 1.04) | 0.92 (0.74, 1.1) | <0.001 |
| CRP, mg/dL [median (Q1, Q3)] | 1.4 (0.6, 3.1) | 1.3 (0.6, 2.7) | 0.22 |
| Creatinine, mg/dL [median (Q1, Q3)] | 0.74 (0.69, 0.79) | 0.88 (0.8, 0.97) | <0.001 |
| Bilirubin, mg/dL [median (Q1, Q3)] | 0.57 (0.43, 0.77) | 0.71 (0.51, 0.96) | <0.001 |
| Potassium, mmol/L [median (Q1, Q3)] | 4.3 (4.1, 4.6) | 4.4 (4.2, 4.7) | <0.001 |
| SBP, mmHg                | 127.2 (21)    | 134.3 (18.6)  | <0.001 |
| DBP, mmHg                | 80.2 (10.6)   | 81.6 (11.5)   | 0.002 |

Data are reported as numbers and percentages, median, quartile 1 and quartile 3 or mean and SD.

apoB- apolipoprotein B
BMI- Body Mass Index
CRP- C-reactive Protein
DBP- Diastolic blood pressure
SBP- Systolic blood pressure
Table 2. Prevalence and univariable odds ratios for hypertension, overweight, and obesity in relation to smoking.

|                          | Women                          |               | Men                          |               |
|--------------------------|--------------------------------|---------------|-----------------------------|---------------|
|                          | Smokers                        | Non-smokers   | OR 95% CI                   | Smokers       | Non-smokers   | OR 95% CI       |
| Arterial hypertension    | 26.06% (86)                    | 33.63% (304)  | 0.78 (0.58-1.06)            | 34.84% (139)  | 37.48% (286)  | 0.90 (0.69-1.17) |
| Overweight or obesity    | 14.50% (48)                    | 23.52% (215)  | 0.61 (0.42-0.88)            | 18.66% (75)   | 27.94% (214)  | 0.59 (0.43-0.81) |
| Self-perceived overweight| 38.07% (126)                   | 40.37% (369)  | 0.91 (0.69-1.19)            | 31.59% (127)  | 42.43% (325)  | 0.64 (0.49-0.83) |

Data are reported as percentages and numbers.

95% CI – 95% confidence intervals

OR- Odds Ratio
Table 3. Body Mass Index and laboratory parameters in women and men in relation to age and smoking status.

|                | Women                          | Men                          |
|----------------|--------------------------------|------------------------------|
|                | Smokers (Age, BMI)              | Non-smokers (Age, BMI)       | Smokers (Age, BMI)              | Non-smokers (Age, BMI)       | P    |
| Age            | 43.55 (14.22)                   | 47.82 (18.04)                | <0.000                         | 43.22 (14.36)                | 45.75 (16.78)                | <0.001 |
| BMI, kg/m²     |                                |                              |                                |                              |                               |       |
| Overall        | 26.67 (4.58)                   | 27.67 (4.53)                | 0.03                           | 25.42 (4.90)                | 26.00 (4.18)                | 0.30   |
| 18–39 years    | 25.67 (4.46)                   | 26.64 (4.05)                | 0.14                           | 25.42 (4.90)                | 26.00 (4.18)                | 0.30   |
| 40–59 years    | 27.01 (5.29)                   | 29.03 (4.47)                | 0.41                           | 26.64 (4.05)                | 29.03 (4.47)                | <0.001 |
| 60–79 years    | 29.68 (5.27)                   | 28.57 (4.28)                | <0.001                         | 27.30 (4.88)                | 28.57 (4.28)                | 0.08   |
| Fasting blood glucose, mg/dL |                     |                              |                                |                              |                               |       |
| Overall        | 97.32 (29.85)                  | 97.09 (23.22)                | 0.2                            | 97.32 (29.85)                | 97.09 (23.22)                | 0.2    |
| 18–39 years    | 90.14 (10.78)                  | 88.70 (9.70)                | 0.53                           | 90.14 (10.78)                | 88.70 (9.70)                | 0.02   |
| 40–59 years    | 101.84 (40.57)                 | 100.79 (27.06)               | 0.30                           | 101.84 (40.57)               | 100.79 (27.06)               | 0.32   |
| 60–79 years    | 104.28 (22.84)                 | 105.64 (28.21)               | 0.02                           | 104.28 (22.84)               | 105.64 (28.21)               | 0.78   |
| Total cholesterol, mg/dL |                     |                              |                                |                              |                               |       |
| Overall        | 202.98 (45.06)                 | 196.12 (43.10)               | 0.01                           | 202.98 (45.06)               | 196.12 (43.10)               | 0.01   |
| 18–39 years    | 188.14 (40.83)                 | 185.85 (38.35)               | 0.34                           | 188.14 (40.83)               | 185.85 (38.35)               | 0.65   |
| 40–59 years    | 218.54 (42.35)                 | 210.61 (43.51)               | 0.01                           | 218.54 (42.35)               | 210.61 (43.51)               | 0.28   |
| 60–79 years    | 195.25 (49.62)                 | 192.82 (44.97)               | 0.008                          | 195.25 (49.62)               | 192.82 (44.97)               | 0.29   |
| apoB, g/L      |                                |                              |                                |                              |                               |       |
| Overall        | 0.92 (0.27)                    | 0.87 (0.25)                  | 0.002                          | 0.92 (0.27)                  | 0.87 (0.25)                  | 0.007  |
| 18–39 years    | 0.78 (0.22)                    | 0.78 (0.21)                  | 0.76                           | 0.78 (0.22)                  | 0.78 (0.21)                  | 0.35   |
| 40–59 years    | 1.02 (0.25)                    | 0.93 (0.24)                  | 0.001                          | 1.02 (0.25)                  | 0.93 (0.24)                  | 0.27   |
| 60–79 years    | 1.02 (0.29)                    | 0.93 (0.26)                  | 0.005                          | 1.02 (0.29)                  | 0.93 (0.26)                  | 0.046  |
| CRP, mg/L      |                                |                              |                                |                              |                               |       |
| Overall        | 2.06 (1.95)                    | 1.76 (1.82)                  | 0.73                           | 2.06 (1.95)                  | 1.76 (1.82)                  | <0.001 |
| 18–39 years    | 1.56 (1.71)                    | 1.45 (1.62)                  | 0.59                           | 1.56 (1.71)                  | 1.45 (1.62)                  | 0.28   |
| 40–59 years    | 2.29 (2.07)                    | 1.94 (1.96)                  | 0.91                           | 2.29 (2.07)                  | 1.94 (1.96)                  | 0.01   |
| 60–79 years    | 2.85 (1.83)                    | 2.05 (1.86)                  | 0.48                           | 2.85 (1.83)                  | 2.05 (1.86)                  | <0.001 |
| Creatinine, mg/dL |                        |                              |                                |                              |                               |       |
### Bilirubin, mg/dL

|                | Women   | Men     |    | Women   | Men     |
|----------------|---------|---------|----|---------|---------|
| Overall        | 0.74 (0.10) | 0.76 (0.20) | 0.12 | 0.88 (0.17) | 0.91 (0.17) | <0.001 |
| 18–39 years    | 0.72 (0.07) | 0.72 (0.07) | 0.52 | 0.88 (0.14) | 0.90 (0.11) | 0.008 |
| 40–59 years    | 0.74 (0.10) | 0.74 (0.10) | 0.97 | 0.86 (0.12) | 0.90 (0.14) | <0.001 |
| 60–79 years    | 0.78 (0.13) | 0.80 (0.25) | 0.41 | 0.97 (0.31) | 0.97 (0.26) | 0.23  |

### Potassium, mmol/L

|                | Women   | Men     |    | Women   | Men     |
|----------------|---------|---------|----|---------|---------|
| Overall        | 4.45 (0.45) | 4.34 (0.39) | <0.001 | 4.51 (0.42) | 4.39 (0.38) | <0.001 |
| 18–39 years    | 4.37 (0.39) | 4.24 (0.31) | 0.001 | 4.46 (0.39) | 4.38 (0.34) | 0.04  |
| 40–59 years    | 4.50 (0.43) | 4.38 (0.43) | 0.01 | 4.50 (0.40) | 4.40 (0.36) | 0.006 |
| 60–79 years    | 4.57 (0.59) | 4.42 (0.41) | 0.09 | 4.70 (0.54) | 4.42 (0.44) | 0.001 |

|      | Women | Men     |
|------|-------|---------|
| apoB- apolipoprotein B |
| BMI- Body Mass Index |
| CRP- C- reactive Protein |

Table 4. Odds ratios with 95% confidence intervals for smoking in relation to socioeconomic and demographic factors.
| Age (per 1 year) | 0.99 (0.98-1.00) | 0.98 (0.97-1.00) | 0.99 (0.99-1.00) | 0.99 (0.98-1.00) |
|------------------|------------------|------------------|------------------|------------------|
| Education level  |                  |                  |                  |                  |
| primary          | 1                | 1                | 1                | 1                |
| secondary        | 0.61 (0.42-0.91) | 0.62 (0.40-0.97) | 0.30 (0.20-0.47) | 0.33 (0.21-0.52) |
| high             | 1.15 (0.41-1.56) | 1.17 (0.83-1.64) | 0.63 (0.48-0.83) | 0.66 (0.49-0.90) |
| Place of residence|                  |                  |                  |                  |
| Rural area       | 1                | 1                | 1                | 1                |
| Town < 50.000    | 1.32 (0.93-1.89) | 1.45 (1.00-2.11) | 1.37 (0.99-1.90) | 1.64 (1.15-2.33) |
| Town 50.000 – 200.000 | 1.01 (0.66-1.54) | 1.14 (0.73-1.78) | 1.12 (0.78-1.60) | 1.38 (0.94-2.03) |
| City > 200.000   | 1.18 (0.82-1.70) | 1.42 (0.96-2.09) | 0.82 (0.57-1.19) | 1.17 (0.78-1.75) |
| Income quartiles |                  |                  |                  |                  |
| [1] lowest       | 1                | 1                | 1                | 1                |
| [2]              | 0.58 (0.40-0.83) | 0.63 (0.43-0.94) | 0.59 (0.42-0.83) | 0.67 (0.47-0.97) |
| [3]              | 0.50 (0.33-0.74) | 0.57 (0.37-0.89) | 0.39 (0.27-0.57) | 0.48 (0.32-0.73) |
| [4] highest      | 0.47 (0.31-0.69) | 0.55 (0.36-0.85) | 0.41 (0.29-0.59) | 0.51 (0.35-0.76) |
| Marital status   |                  |                  |                  |                  |
| Married          | 1                | 1                | 1                | 1                |
| Widowed          | 0.93 (0.66-1.30) | 0.75 (0.50-1.12) | 1.04 (0.77-1.39) | 0.82 (0.55-1.21) |
| Single           | 1.83 (1.18-2.85) | 1.90 (1.20-3.00) | 2.14 (1.21-3.74) | 2.39 (1.30-4.40) |
| Divorced         | 0.77 (0.51-1.16) | 0.98 (0.61-1.58) | 1.25 (0.59-2.64) | 1.17 (0.52-2.62) |

* Multivariable Analysis

95% CI – 95% confidence intervals

OR- Odds Ratio
Table 5. Odds ratios with 95% confidence intervals for selected clinical outcomes in relation to smoking.

|                      | Women |                     | Men    |                     |
|----------------------|-------|---------------------|--------|---------------------|
|                      | OR    | 95% CI              | OR     | 95% CI              |
| BMI (per 1 kg/m²)    | 0.97  | (0.94-0.99)         | 0.97   | (0.94-1.00)         |
|                      | 0.93  | (0.91-0.96)         | 0.93   | (0.90-0.97)         |
| Hypertension         |       |                     |        |                     |
| No                   | 1     | 1                   | 1      | 1                   |
| Yes                  | 0.78  | (0.58-1.06)         | 0.95   | (0.65-1.4)          |
|                      | 0.9   | (0.69-1.17)         | 1.07   | (0.78-1.46)         |
| Hypercholesterolemia |       |                     |        |                     |
| No                   | 1     | 1                   | 1      | 1                   |
| Yes                  | 1.21  | (0.91-1.60)         | 1.59   | (1.15-2.21)         |
|                      | 1.04  | (0.80-1.35)         | 1.21   | (0.89-1.64)         |

* Multivariable Analysis

95% CI – 95% confidence intervals

BMI- Body Mass Index

OR- Odds Ratio