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Prevalence of cardiovascular disease risk factors in Tallinn, Estonia

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ABSTRACT

Background and objective: Cardiovascular diseases are still a major public health concern in Estonia despite the decline in the mortality rate during the past decade. For better preventive strategies we aimed to investigate the prevalence of cardiovascular disease risk factors and their relations with age, gender and ethnicity.

Materials and methods: The cross-sectional study was carried out in Tallinn, Estonia. Two hundred individuals from each of the sex and 10-year age group (range 20–65 years of age) were randomly selected and invited to participate. Final study sample consisted of 511 men and 600 women (mean age of 46 years). Physiological measurements were taken and blood samples were drawn for standard measurements of the following markers: total cholesterol, high- and low-density lipoprotein cholesterol, apolipoproteins, triglycerides, glucose and inflammatory markers.

Results: Overall, 31% of the study subjects had high blood pressure, 23% had metabolic syndrome, and 55% were overweight/obese. The prevalence of all risk factors increased with age amongst both genders. The proportion of individuals having increased cholesterol, apolipoprotein B-100, and homocysteine levels was very high amongst both genders (60–80%). More Russians and other ethnic minorities compared to ethnic Estonians had calculated 10-year CHD risk ≥ 10%.

Conclusions: The study established a high prevalence of cardiovascular disease risk factors in Estonian adults (20–65 years of age). Younger portion of the population and some extent ethnic considerations should be taken into account when designing future studies, health prevention activities and interventions.

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1. Introduction

According to European cardiovascular disease statistics [1] in 2010, Estonia ranked third amongst men and fourth amongst women when considering the proportion of years lost due to cardiovascular diseases (CVD), outperformed only by the Russian Federation and Hungary (and additionally Slovakia amongst women). CVD is thus a major public health concern in Estonia. This disease makes a significant contribution to potential years of life lost (25%), kills approximately 10,000 people per year and has one of the highest surgical CVD treatment rates when compared to other European countries [1,2].

There is a need for studies that could describe, evaluate, and provide a broader understanding of the situation in Estonia in terms of CVD risk factors. For example, the importance of cholesterol level in CVD prevention is widely known, but there is no data available for the Estonian population regarding mean high- and low-density lipoprotein cholesterol levels. The Framingham Risk Score algorithm has been available for more than 20 years, but no estimates amongst Estonians have been reported so far. The data is also scarce or lacking for CVD risk factors such as apolipoprotein B and A-1, homocysteine, high-sensitive C-reactive protein, fibrinogen and lipoprotein (a), which have been linked to the development of coronary heart disease (CHD) [3–7] and can provide important clinical information, when conventional markers are already taken into account [8].

Additionally, due to genetic, environmental, and cultural factors, people of certain ethnic groups experience a greater burden of CVD [9], a fact that should be also considered in heterogeneous populations such as Estonia. The last study published analyzing connections between CVD risk factors and ethnicity is from 1995 [10]. A new assessment is greatly needed due to the rapid societal changes in Eastern Europe since the beginning of the 1990s. The influences of these changes upon CVD risk are largely unknown.

This is the first comprehensive study about the prevalence of CVD risk factors in Estonia undertaken with the purpose of designing novel future interventions. Our aim was to investigate the prevalence of CVD risk factors and their associations with age, sex and ethnic origin.

2. Materials and methods

2.1. Subjects

The survey sample for the cross-sectional study was drawn in 2007 from the total population in Tallinn (Ministry of the Interior, Government of Estonia). The total population of Estonia is approximately 1.3 million, with ~400,000 living in the capital city of Tallinn. The sample size calculation was based on the protocol of the Countrywide Integrated Non-communicable Disease Intervention (CINDI) program [11]. Stratified random sampling was used: 200 individuals from each of the sex and age group (20–29, 30–39, 40–49, 50–59, and 60–65 years of age) were randomly selected and invited to participate by mail with up to two follow-up letters sent to non-respondents.

The response rate was 55% for men and 64% for women being the lowest at 20–29 years of age. Initially the sample consisted of a total of 1184 participants, including 545 men and 639 women. Thirty-four men and thirty-nine women were excluded because their laboratory results were not available. Thus the final study consisted of 1111 participants: 511 men and 600 women with an average age of 45.8 ± 12.2 years (range 20–65 years). Participants were questioned about smoking status (daily, ex-smoker, how many cigarettes per day), pregnancy status, physician-diagnosed type 2 diabetes mellitus, hypertension and hypercholesterolemia. Additionally awareness about high blood pressure or elevated cholesterol was identified.

The data and blood samples were collected and analyzed during 2007–2009. All the participants were examined by one trained cardiologist. Blood was collected by one qualified nurse and analyzed by one qualified technician. The procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1983.

2.2. Physiological measurements

Blood pressure was measured two times using a mercury sphygmomanometer on the right upper arm while the participant was seated and resting for a minimum of 5 min. Results were recorded to the nearest 2 mmHg. The mean values of both readings were used for the analysis.

The body mass (weight) of participants was measured without shoes and heavy outer garments and then recorded to the nearest 100 g. Height was recorded to the nearest 0.5 cm. Body mass index (BMI) was calculated as the weight in kilograms divided by the height in meters squared [BMI = body mass (kg)/height (m^2)].

Waist circumference (WC) was determined with a tape measure and recorded to the nearest 0.5 cm at a point midway between the costal margin and iliac crest along the midaxillary line.

2.3. Blood sample collection and analysis

Blood sample collection description and laboratory methods are specifically described elsewhere [12]. Shortly all blood samples were obtained after an overnight fast, while the participant was seated. Sample collection from the *vena cubitalis* was carried out using a standard method with Vacutainer tubes (BD Vacutainer, Belliver Industrial Estate, Plymouth; Becton, Dickinson and Co., UK). Serum and plasma were separated by centrifugation and kept at 4 °C until analysis.

All measurements were determined using the standard procedures with Roche reagents (Roche Diagnostics, Mannheim, Germany) on a Cobas analyzer (Roche Diagnostics, Indianapolis, USA or Mannheim Germany). Fibrinogen was determined with an STA Compact auto analyzer (Diagnostica Stago, S.A.S. France) using the Clauss method.

2.4. Diagnostic criteria

A Framingham risk score (10-year CHD risk) was calculated for each participant based on Wilson et al. [13]. Participants were

stratified into two risk score groups: <10% and ≥10% group, defined as low-risk and moderate-to-high risk groups respectively. The International Diabetes Federation (IDF) definition was applied for metabolic syndrome [14]. A subject was considered to have: type-2 diabetes, when fasting plasma glucose was ≥7 mmol/L or they had previously been diagnosed with type-2 diabetes; overweight, when BMI ≥ 25; obesity, when BMI ≥ 30; central obesity, when waist circumference ≥ 94 cm for men and ≥80 cm for women; high blood pressure, when systolic blood pressure (SBP) ≥ 140 and/or diastolic blood pressure (DBP) ≥ 90 mmHg and/or had been using anti-hypertension medication during the last two weeks.

The cut-off values for biochemical CHD risk factors were as follows: total cholesterol (TC), ≥5 mmol/L; low-density lipoprotein cholesterol (LDL-C), ≥3 mmol/L; high-density lipoprotein cholesterol (HDL-C), <1.03 mmol/L for men and <1.29 mmol/L for women; triglycerides (TG), ≥1.7 mmol/L; glucose, 5.60–6.99 mmol/L; apolipoprotein A-1 (ApoA-1), <1.04 g/L (men) and <1.08 g/L (women); apolipoprotein B-100 (ApoB-100), >1.33 g/L (men) and >1.17 g/L women; lipoprotein (a) Lp(a), >0.3 g/L; fibrinogen, >4 g/L; high sensitive C-reactive protein (hsCRP), >3 g/L; and homocysteine (Hcy), ≥12 μmol/L [14,15]. TC and LDL-C levels were also considered abnormal when subject had been using cholesterol lowering medication during the last two weeks.

2.5. *Statistical analysis*

The Shapiro–Wilk test was applied to determine the normal distribution of collected variables (cholesterol, triglyceride s, glucose etc. values) by gender (grouping factor). The data was not normally distributed and a nonparametric, Mann–Whitney *U* test, was utilized to compare the distributions.

Categorical variables were tested by the Pearson chi-square test. The data were weighted to match the age distribution of Estonian men and women aged 20–65 years using the 2011 Population and Housing census data for Tallinn obtained from Statistics Estonia (Ministry of Finance, Government of Estonia).

Considering ethnicity, Bonferroni correction was applied for multiple comparisons (observed *P* value multiplied by three). Gender based odds ratio (OR) and 95% confidence interval (CI) was calculated based on Szumilas [16]. For all percentages, 95% CI calculations were based on Vollset [17].

P values of less than .05 were considered statistically significant. Statistical analyses were performed using SPSS software version 21.0 (SPSS Inc., Chicago, IL, USA).

3. Results

The baseline characteristics of participants are presented in Table 1.

The average 10-year CHD risk was 8.2% ± 7.6%. The odds for moderate-to-high 10-year CHD risk (≥10%) were 4.0 times higher for men compared to women (95% CI 3.0–5.4, $P < 0.001$).

The gender-stratified weighted prevalence of cardiovascular disease risk markers are presented in Table 2. Almost a quarter (24%) of the study subjects were classified in the moderate-to-high 10-year group with a significantly male predominance. The weighted prevalence of high blood pressure was 31%, also, with a strong male predominance. Reported awareness of the condition was around 80% (74% and 84% for men and women, respectively). Approximately half of the subjects, with identified high blood pressure, reported taking anti-hypertension medication(s) upon recruitment for the study but only 32% had normal blood pressure.

The prevalence of metabolic syndrome was significantly higher amongst male compared to female participants. High blood pressure (according to IDF criteria) was the main abnormality (after abdominal obesity) amongst both genders (91% and 81% for men and women, respectively) followed by raised triglycerides (71% and 64%), glucose (52% and 59%), and reduced HDL-C levels (34% and 51%). Overall, 31% of the men and 35% of the women with metabolic disorder had ≥3 defined syndrome components.

Of the study subjects, according to BMI, 33% were found to be overweight (42% of men and 25% of women), 22% were obese (21% and 23%) and 46% had central obesity (49% and 45% respectively).

Increased TC, LDL-C and ApoB-100 levels were very high amongst both genders. Roughly 60–80% of male and female participants had abnormal TC, LDL-C and ApoB-100 levels. The reported awareness of high cholesterol was 18% for men and 35% for women respectively and approximately 9% of those who were aware taking cholesterol-lowering medication.

The prevalence of high hsCRP and fibrinogen was low compared to the other previously described risk factors. However one exception was homocysteine: approximately half of the study subjects had elevated homocysteine levels with a strong male predominance.

The prevalence of risk factors tends to increase with age in both genders (Figs. 1 and 2). Amongst men aged 20–29, high homocysteine levels showed the highest prevalence followed by high ApoB-100, LDL-C, TC levels and daily smoking. For ages 30–65, high ApoB-100 level showed the highest prevalence followed by high LDL-C, high TC, high homocysteine levels, being overweight (including obesity), and having high blood pressure. Amongst women the tendency was the same: for ages 20–65, high ApoB-100 was the most prevalent risk factor followed by high homocysteine, high TC, high LDL-C levels, and being overweight.

Amongst men aged 20–29 and 30–39, the awareness of high blood pressure levels (50% and 56% respectively) was the lowest compared to other age groups (75–90%). Amongst women, the awareness was 70–95%, being lowest in the 30–49 year-old group. The awareness of high cholesterol levels was extremely low: 3–25% amongst men and 7–53% amongst women and lowest in those aged 20–39.

Table 3 shows the prevalence for different ethnic groups amongst men. Russian men and male subjects from ethnic groups other than Estonian had a higher prevalence of moderate-to-high 10-year CHD risk and high blood pressure. Furthermore total cholesterol and LDL-C were significantly higher amongst subjects from other ethnic groups compared to Russians and Estonians.

Amongst women (Table 4), the risk factor spectrum was wider and different. For example, instead of high TC and LDL-C, which were highlighted amongst men, low HDL-C and high

Table 1 – Age-stratified baseline characteristics for participants by gender.

Risk factor	Total n = 511/600	20–29 years n = 65/71	30–39 years n = 101/131	40–49 years n = 125/140	50–59 years n = 134/160	60–65 years n = 86/98
Age, years						
Men	46.0 ± 12.3	25.1 ± 2.8	35.3 ± 2.6	44.8 ± 3.0	54.7 ± 2.6	62.2 ± 1.6
Women	45.7 ± 12.2	25.5 ± 2.1	34.4 ± 2.9	45.0 ± 2.8	54.6 ± 2.9	61.9 ± 1.5
10-Year risk for CHD event, %						
Men	11.1 ± 8.0	NA	4.6 ± 2.4	7.5 ± 3.5	13.4 ± 6.6	20.5 ± 8.9
Women	5.8 ± 6.2***	NA	1.2 ± 1.0	3.3 ± 3.8	8.0 ± 5.8	11.8 ± 7.2
Smoking, cigarettes per day						
Men	5.8 ± 9.2	4.6 ± 7.1	5.8 ± 9.8	7.4 ± 9.1	6.1 ± 9.6	4.0 ± 9.4
Women	2.0 ± 5.1***	2.3 ± 4.5	1.8 ± 4.5	2.2 ± 5.3	2.6 ± 6.3	1.0 ± 3.2
SBP, mmHg						
Men	133.2 ± 22.5	119.9 ± 12.9	123.1 ± 15.1	130.5 ± 19.7	138.4 ± 21.4	151.3 ± 26.3
Women	125.8 ± 22.1***	108.0 ± 10.6	114.7 ± 15.3	125.7 ± 20.9	132.5 ± 20.1	142.9 ± 24.0
DBP, mmHg						
Men	84.8 ± 13.1	74.1 ± 11.4	81.9 ± 12.6	85.9 ± 12.4	88.7 ± 11.8	89.6 ± 13.0
Women	78.5 ± 13.0***	67.5 ± 8.7	73.5 ± 12.9	80.1 ± 11.6	82.2 ± 11.1	84.7 ± 13.7
BMI, kg/m^2						
Men	27.5 ± 4.5	24.8 ± 3.5	27.1 ± 4.3	27.6 ± 4.2	28.1 ± 4.6	28.7 ± 5.0
Women	26.6 ± 5.7**	22.7 ± 4.7	25.3 ± 5.5	27.1 ± 5.7	27.7 ± 4.9	28.8 ± 6.0
Waist circumference, cm						
Men	95.3 ± 12.3	85.6 ± 10.3	92.7 ± 11.0	95.5 ± 10.2	98.3 ± 11.9	101.5 ± 13.1
Women	81.2 ± 13.5***	71.0 ± 2.7	76.5 ± 11.8	82.5 ± 14.1	83.8 ± 11.8	87.4 ± 13.0
TC, mmol/L						
Men	5.5 ± 1.1	4.7 ± 0.9	5.5 ± 1.0	5.7 ± 1.0	5.7 ± 1.1	5.7 ± 1.1
Women	5.5 ± 1.1	4.8 ± 0.8	4.9 ± 0.9	5.5 ± 0.9	5.9 ± 1.0	6.1 ± 1.1
HDL-C, mmol/L						
Men	1.4 ± 0.4	1.4 ± 0.5	1.3 ± 0.3	1.5 ± 0.4	1.4 ± 0.3	1.3 ± 0.4
Women	1.7 ± 0.5***	1.7 ± 0.3	1.6 ± 0.5	1.7 ± 0.5	1.8 ± 0.5	1.8 ± 0.5
LDL-C, mmol/L						
Men	3.7 ± 1.0	2.9 ± 0.9	3.7 ± 0.9	3.7 ± 0.9	4.0 ± 1.1	3.9 ± 0.9
Women	3.5 ± 1.0***	2.9 ± 0.7	3.1 ± 0.9	3.6 ± 0.9	3.7 ± 1.0	3.9 ± 0.9
TG, mmol/L						
Men	1.6 ± 1.2	1.1 ± 0.7	1.8 ± 1.2	1.5 ± 0.8	1.7 ± 1.5	1.8 ± 1.3
Women	1.3 ± 0.7***	1.0 ± 0.6	1.1 ± 0.7	1.2 ± 0.7	1.4 ± 0.6	1.5 ± 0.8
Glucose, mmol/L						
Men	5.4 ± 1.2	5.0 ± 0.6	5.1 ± 0.6	5.3 ± 0.7	5.7 ± 1.4	6.0 ± 1.7
Women	5.2 ± 1.0**	4.8 ± 0.8	5.0 ± 0.7	5.4 ± 1.8	5.3 ± 0.8	5.4 ± 0.7
ApoA-1 (g/L)						
Men	1.5 ± 0.3	1.4 ± 2.5	1.5 ± 0.2	1.5 ± 0.2	1.6 ± 0.2	1.5 ± 0.3
Women	1.8 ± 0.3***	1.8 ± 0.3	1.7 ± 0.3	1.8 ± 0.3	1.8 ± 0.3	1.8 ± 0.2
ApoB-100, g/L						
Men	1.8 ± 0.5	1.4 ± 0.4	1.8 ± 0.4	1.8 ± 0.4	1.9 ± 0.5	1.9 ± 0.5
Women	1.7 ± 0.5***	1.4 ± 0.3	1.4 ± 0.4	1.7 ± 0.5	1.8 ± 0.6	1.8 ± 0.4
Lp(a), g/L						
Men	0.3 ± 0.4	0.2 ± 0.2	0.3 ± 0.3	0.2 ± 0.2	0.3 ± 0.4	0.4 ± 0.5
Women	0.3 ± 0.3	0.3 ± 0.3	0.2 ± 0.3	0.3 ± 0.3	0.3 ± 0.3	0.3 ± 0.4
Fibrinogen, g/L						
Men	3.1 ± 0.8	2.6 ± 0.6	2.9 ± 0.7	3.0 ± 0.6	3.3 ± 0.7	3.5 ± 0.9
Women	3.3 ± 0.9***	3.2 ± 1.1	3.1 ± 0.7	3.2 ± 0.9	3.5 ± 0.7	3.8 ± 0.8
hsCRP, mg/L						
Men	2.3 ± 3.4	1.6 ± 1.6	1.6 ± 1.7	1.9 ± 2.9	2.8 ± 3.9	3.6 ± 5.1
Women	2.3 ± 3.2	1.8 ± 3.0	2.0 ± 3.2	2.1 ± 2.9	2.0 ± 2.0	3.0 ± 4.6
Hcy, μmol/L						
Men	14.3 ± 5.6	13.6 ± 3.5	13.1 ± 3.5	14.0 ± 7.4	14.4 ± 5.9	15.9 ± 5.4
Women	12.5 ± 4.4***	11.7 ± 3.1	10.7 ± 2.5	11.9 ± 4.2	13.4 ± 5.3	14.4 ± 4.1

Values are mean ± standard deviation. P values calculated using Mann–Whitney U test.

** $P < 0.01$.

*** $P < 0.001$ comparing men and women.

CHD, coronary heart disease; NA, not applicable; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; ApoB-100, apolipoprotein B-100; ApoA-1, apolipoprotein A-1; Lp(a), lipoprotein(a); hsCRP, high-sensitive C-reactive protein; Hcy, homocysteine.

Table 2 – Gender-stratified weighted prevalence of cardiovascular disease risk markers.

Risk factor	Total $n = 1111$	Men $n = 511$	Women $n = 600$	P
CHD risk ≥10%[a]	23.7 (20.9–26.8)	34.6 (29.9–39.5)	14.4 (11.4–18.0)	<0.001
Smoking (daily)	27.8 (25.2–30.5)	36.1 (32.2–40.4)	20.5 (17.5–24.0)	<0.001
High blood pressure	31.1 (28.5–33.9)	36.8 (32.8–41.0)	26.1 (22.8–29.9)	<0.001
Metabolic syndrome	22.9 (20.6–25.6)	29.1 (25.4–33.1)	17.5 (14.6–20.8)	<0.001
Type-2 diabetes	3.3 (2.4–4.5)	4.0 (2.7–6.1)	2.7 (1.7–4.4)	0.219
High glucose	8.8 (7.3–10.7)	10.5 (8.2–13.6)	7.4 (5.5–9.7)	0.054
Overweight (BMI)[b]	55.0 (51.8–57.6)	6 2.6 (58.4–66.7)	48.3 (44.4–52.4)	<0.001
Central obesity	46.3 (43.5–49.3)	48.5 (44.3–52.8)	44.5 (40.5–48.5)	0.181
High TC	62.3 (59.3–65.0)	63.8 (59.6–67.8)	60.9 (57.0–64.8)	0.333
High LDL-C	66.1 (63.2–68.7)	70.7 (66.7–74.5)	62.0 (58.0–65.8)	0.002
Low HDL-C	14.6 (12.7–16.8)	14.5 (11.7–17.7)	14.7 (12.0–17.7)	0.994
High TG	23.5 (21.1–26.1)	30.9 (27.2–35.1)	17.0 (14.4–20.2)	<0.001
High ApoB-100	80.7 (78.2–82.9)	77.3 (73.5–80.7)	83.7 (80.4–86.4)	0.008
Low ApoA-1	1.3 (0.8–2.1)	2.4 (1.3–4.0)	0.3 (0.1–1.2)	0.003
High Lp(a)	25.0 (22.5–27.6)	24.1 (20.5–27.9)	25.7 (22.4–29.4)	0.529
High fibrinogen	10.8 (8.9–12.6)	7.2 (5.3–9.9)	14.0 (11.2–16.9)	0.001
High hsCRP	18.5 (16.3–20.9)	18.3 (15.2–21.9)	18.7 (15.7–22.0)	0.874
High Hcy	55.9 (52.9–58.8)	66.5 (62.3–70.4)	46.5 (42.4–50.5)	<0.001

Values are percentage (95% confidence interval).
[a] Framingham Risk Score calculated for subjects aged 30–65 years; calculation included 446 men and 529 women (total $n = 975$).
[b] Obese included. P values calculated using Pearson chi-square test. For abbreviations, please see the footnote of Table 1.

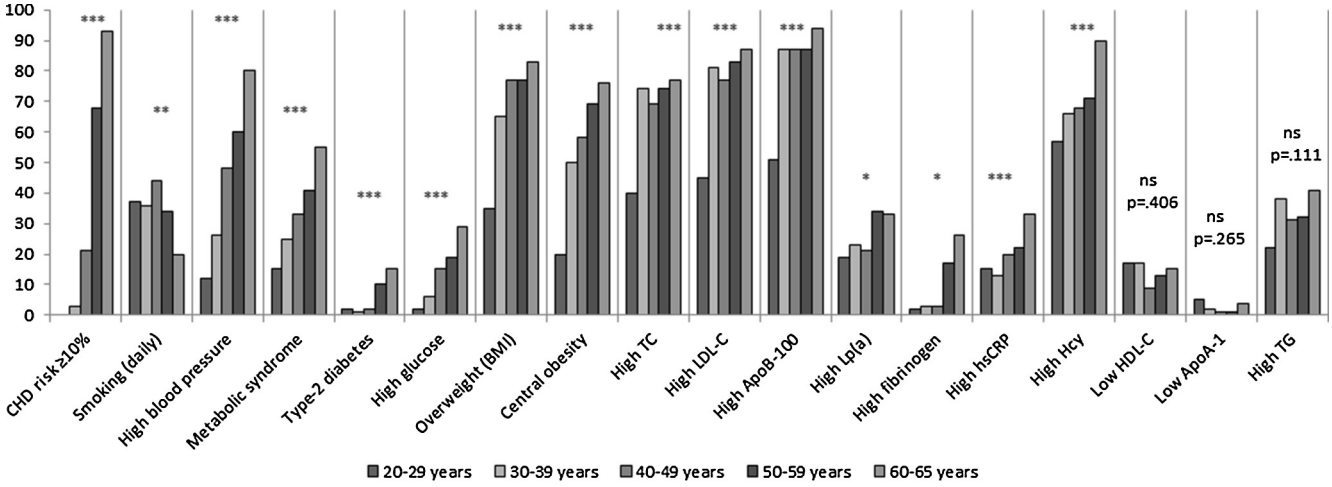

Fig. 1 – Age-stratified prevalence of cardiovascular disease risk factors amongst men. *P < 0.05; **P < 0.01; ***P < 0.001; ns, nonsignificant.

triglycerides showed association with ethnicity amongst women. In addition, Russian women and female subjects from ethnic groups other than Estonian had a significantly higher prevalence of metabolic syndrome, being overweight, and central obesity. The proportion of persons having CHD event risk ≥ 10% was highest amongst Russians.

4. Discussion

For the last three decades, much attention has been devoted to cardiovascular risk factors present in younger people. As a result, the view is increasingly accepted that prevention of the appearance of risk factors and the early manifestations of atherosclerotic and hypertensive cardiovascular diseases requires intervention before adulthood. Despite this knowledge, the risk factor prevalence was found to be high amongst young subjects. Additionally, participant distribution showed that the study response rate, the awareness of high blood pressure, high LDL-C, and TC levels was the lowest in young subjects. Lack of awareness suggests that greater attention to health-related knowledge and behavior, especially among young people, is needed.

Considering neighboring geographical locations, the prevalence of high blood pressure (31%, Table 3) seems to be lower compared to other reported results [18–20]. High blood pressure determinants in Estonian adults are discussed in greater depth in our previously published article [12].

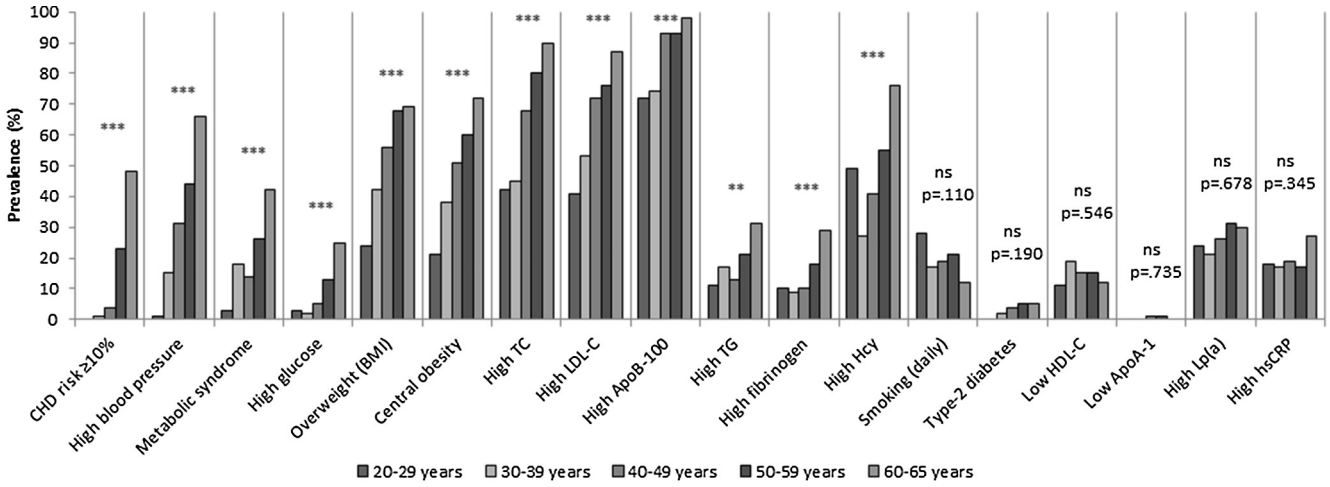

Fig. 2 – Age-stratified prevalence of cardiovascular disease risk factors amongst women. **P < 0.01; ***P < 0.001; ns, nonsignificant.

Overall prevalence of metabolic syndrome (23%, Table 3) was quite similar to that of a study previously carried out in Estonia [21]. The slight difference might stem from a rural vs. urban-rural population. As discussed in the study by Eglit et al. [21], we also found that a younger proportion of participants had a fairly high prevalence of metabolic syndrome. Although no unifying pathophysiological mechanism of metabolic syndrome has been identified, it should be taken into account as a useful clinical tool for identifying life-time CVD risk [21,22].

High prevalence of obesity (based on BMI) and central obesity (based on WC) was found. However, they presented different distribution patterns – a quarter of the subjects showed obesity whereas a half showed central obesity. Central obesity is more strongly associated with CVD risk than obesity based on BMI [23] and thus, measuring both simultaneously might confer additional risk information.

In regards to biochemical risk factors, elevated ApoB-100 had the highest prevalence followed by high TC, and LDL-C levels. While LDL-C measurement has been the cornerstone in CVD risk assessment, awareness is gradually developing that ApoB can be a more representative indicator. For example, Contois et al. [24] have suggested including ApoB-100 in national guidelines next to LDL-C due to the enhanced

Table 3 – Weighted prevalence of cardiovascular disease risk factors according to ethnicity in males.

Risk factor	Estonians $n = 293$	Russians $n = 162$	Other $n = 56$	P
Age, mean ± SD, years	45.5 ± 13.2	46.3 ± 11.3	47.0 ± 9.4	NS
CHD risk ≥10%[a]	28.3 (22.8–35.2)	37.8 (30.2–45.9)	55.3 (39.7–69.9)	0.012
Smoking (daily)	32.7 (27.6–38.7)	42.8 (35.8–50.1)	43.2 (28.7–58.9)	0.186
High blood pressure	30.2 (25.0–35.9)	40.8 (34.1–47.8)	59.0 (44.4–72.3)	0.001
Metabolic syndrome	25.6 (20.9–31.3)	31.5 (25.3–38.3)	42.9 (29.7–57.8)	0.144
Type-2 diabetes	3.7 (1.9–6.9)	3.1 (1.3–6.9)	4.6 (1.0–16.7)	1.000
High glucose	9.6 (6.5–13.9)	12.4 (8.2–18.0)	11.6 (4.7–25.9)	1.000
Overweight (BMI)[b]	60.3 (54.2–66.1)	62.4 (55.1–69.1)	75.0 (59.4–86.3)	0.525
Central obesity	45.6 (39.6–51.7)	48.7 (41.6–55.9)	63.6 (48.9–76.2)	0.252
High TC	61.2 (55.5–67.0)	62.6 (55.9–69.4)	84.9 (70.6–92.1)	0.027
High LDL-C	66.2 (60.4–71.5)	73.2 (66.6–78.9)	87.8 (73.3–93.6)	0.027
Low HDL-C	13.6 (9.9–18.4)	17.0 (12.2–23.2)	15.9 (7.2–30.7)	1.000
High TG	30.1 (24.8–36.0)	29.9 (23.7–37.0)	40.9 (26.7–56.7)	0.987
High ApoB-100	74.9 (69.1–79.8)	76.7 (70.4–82.7)	92.3 (77.4–97.1)	0.117
Low ApoA-1	2.2 (1.0–5.0)	3.1 (1.3–7.0)	0.0 (0.0–10.0)	1.000
High Lp(a)	23.7 (18.9–29.3)	26.9 (20.9–33.9)	20.5 (10.3–35.8)	1.000
High fibrinogen	5.8 (3.4–9.6)	8.0 (4.7–13.1)	13.6 (5.7–28.1)	0.516
High hsCRP	18.0 (13.7–23.2)	19.6 (14.4–26.0)	18.2 (8.7–33.2)	1.000
High Hcy	68.0 (62.1–73.5)	61.5 (54.2–68.3)	72.7 (57.0–84.6)	0.624

Values are percentage (95% confidence interval) unless otherwise indicated.

[a] Framingham Risk Score calculated for subjects aged 30–65 years; calculation included 245 Estonians, 147 Russians and 54 other.

[b] Obese included. *P* values calculated using Pearson chi-square test and corrected for multiple comparisons (Bonferroni). NS, not significant, calculated using the Mann–Whitney *U* test. For abbreviations, please see the footnote of Table 1.

Table 4 – Weighted prevalence of cardiovascular disease risk factors according to ethnicity in females.

Risk factor	Estonians $n = 319$	Russians $n = 243$	Other $n = 38$	P
Age, mean ± SD, years	45.7 ± 12.4	45.5 ± 12.0	47.1 ± 11.0	NS
CHD risk ≥10%[a]	10.4 (7.1–15.0)	19.2 (14.1–25.6)	19.4 (9.8–35.0)	0.093
Smoking (daily)	17.8 (13.9–22.6)	23.4 (18.2–29.5)	18.2 (8.7–33.2)	0.786
High blood pressure	22.2 (18.0–27.0)	29.3 (23.8–35.5)	39.6 (25.7–53.4)	0.057
Metabolic syndrome	12.9 (9.7–17.0)	22.7 (17.9–28.7)	27.7 (16.3–41.9)	0.006
Type-2 diabetes	2.1 (1.0–4.6)	3.0 (1.3–6.4)	6.8 (1.8–19.7)	0.627
High glucose	5.2 (3.2–8.4)	10.9 (7.2–15.7)	6.8 (1.8–19.7)	0.117
Overweight (BMI)[b]	41.2 (35.9–46.8)	55.4 (48.5–61.6)	65.3 (51.1–78.1)	<0.001
Central obesity	38.2 (32.8–43.2)	51.0 (44.0–56.8)	58.4 (43.3–71.6)	0.006
High TC	59.1 (53.5–64.4)	62.8 (56.2–69.0)	63.6 (47.7–77.2)	1.000
High LDL-C	59.7 (54.1–65.0)	63.8 (57.2–69.9)	69.8 (53.7–82.3)	1.000
Low HDL-C	9.7 (6.9–13.8)	19.3 (15.4–26.1)	27.9 (15.5–43.0)	0.001
High TG	13.7 (10.4–18.2)	20.3 (15.4–26.1)	30.9 (19.1–47.7)	0.021
High ApoB-100	82.6 (77.9–86.5)	85.3 (79.9–89.5)	81.8 (66.8–91.3)	1.000
Low ApoA-1	0.3 (0.02–2.0)	0.4 (0.02–2.8)	0.0 (0.0–10.0)	1.000
High Lp(a)	22.9 (18.5–28.0)	29.0 (23.3–35.4)	23.3 (12.–39.0)	0.759
High fibrinogen	14.6 (11.0–19.2)	11.1 (7.4–16.1)	16.3 (7.3–31.3)	1.000
High hsCRP	19.4 (15.3–24.2)	17.2 (12.7–22.9)	27.3 (15.5–43.0)	0.888
High Hcy	49.7 (44.1–55.3)	40.9 (34.5–47.5)	45.5 (30.7–61.0)	0.369

Values are percentage (95% confidence interval) unless otherwise indicated.
[a] Framingham Risk Score calculated for subjects aged 30–65 years; calculation included 280 Estonians, 213 Russians and 36 other.
[b] Obese included. P values calculated using Pearson chi-square test and corrected for multiple comparisons (Bonferroni). NS, not significant, calculated using the Mann–Whitney U test. For abbreviations, please see the footnote of Table 1.

reliability. The reason behind this is that ApoB-100 is not only a part of LDL, it is also a part of very low-density lipoproteins (VLDL), intermediate-density lipoproteins (IDL), and Lp(a) [25]. Therefore, through one marker, it is possible to measure the number of atherogenic lipoprotein particles in the circulation. Additionally, LDL particles are extremely heterogeneous when it comes to the amount of stored cholesterol—for example, patients with the same LDL-C concentration levels can have a very different LDL particle count [26]. Thus ApoB-100 measurement in conjunction with standard lipid testing might give a more accurate overview about a patient's risk of developing a cardiovascular event.

As for inflammatory markers, there is still debate about whether to use them in risk prediction or not because no additional value is seen when traditional risk factors are known [27]. However, homocysteine is an independent CHD risk factor [4] with each increase of 5 μmol/L in the marker level raising the risk of CHD events by approximately 20% [28]. A relatively high homocysteine level was found amongst men and women in all age groups. The exact reason for this is unclear but could be explained by several lifestyle-related factors such as smoking, psychological stress, alcohol intake, and low fruit consumption [29].

Based on data from Statistics Estonia, Russians are the largest minority (25%) in the country. In the capital, Tallinn, ethnic Estonians make up only 53% of the population (38% are Russians and 9% are other minorities). Volozh et al. [10] have published a study, describing CHD risk factors amongst Estonian and Russian inhabitants of Tallinn (aged 10–14 and 30–54) for the period of 1984–1986. Smoking, total- and HDL cholesterol, triglycerides, body mass index, and blood pressure were investigated. Overall, the risk factor levels tended to be higher amongst Estonians compared to Russians and the authors suggested the importance of a differentiated preventive approach in respect to the composition of the ethnic population. Our study results are some extent opposite to those of 30 years ago, that compared to Estonians, Russians and subjects from other ethnic groups have a higher prevalence of several CVD risk factors and also a higher prevalence of moderate-to-high 10-year CHD risk (Tables 3 and 4). Although there is no information in Estonia about other minorities, Leinsalu et al. [30] have shown that Russians have a higher mortality rate in all age groups and for almost all selected causes including heart disease related to psychological factors, alcohol consumption, and poor diet.

As previously discussed, predictive interventions would be most beneficial if conducted before adulthood. Early life programming could be very important because early life events play a powerful role in mid-life, influencing later susceptibility to certain chronic diseases [31]. Estonians, when compared to Russians and other minorities, differ from one another in language, culture, historic, and socio-economic aspects. It is quite possible the social exclusion, unemployment and limited access to higher education present during the political and economic restructuring throughout and following the end of the Soviet era has negatively impacted the cardiovascular disease factor profile we see today. Soviet times have given certain input into social exclusion, unemployment, and limited access to higher education – life factors acting long time ago can influence cardiovascular disease factor profile seen today.

4.1. *Limitations*

Only regional data (Tallinn) was analyzed. Future research should consider a larger sample including participants from rural populations.

Considering response rate younger portion of the population especially was underrepresented. Young people are often

asymptomatic, thus the outcome of this age group in reality might be worse. Also, in any age group, it can be assumed that the respondents might be more responsible and interested in their health status than non-respondents.

Additional medications taken by the subjects may interfere with metabolic variables and body weight, which can consequently influence the results.

Despite the limitations given, the study contributes to our understanding of trends in the biochemical risk markers of this population. While this is the most comprehensive epidemiological study conducted so far among Estonian inhabitants, it is also the first time when the prevalence of apolipoprotein and inflammation marker levels are reported.

5. Conclusions

This cross-sectional population-based study established a high prevalence of cardiovascular disease risk factors in the Tallinn population (20–65 years of age) and provides a certain baseline for further studies. In the future special attention should be given to the younger portion of the population, as this group exhibits low CVD risk factor awareness. Also, when designing relevant interventions, ethnicity should be emphasized – Russians and subjects from other ethnic groups showed a higher prevalence of several CVD-related markers and also moderate-to-high 10-year CHD risk compared to Estonians.

Conflict of interest

The authors report no conflicts of interest.

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