PREVALENCE OF CARDIOVASCULAR RISK FACTORS IN ADULTS REGISTERED IN A PRIMARY HEALTH UNIT OF PORTO

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ABSTRACT

Purpose. The study aims to describe the adult's overall cardiovascular disease risk factors prevalence, including arterial stiffness and physical inactivity.

Methods. The cross-sectional study involved 197 adults (males: 42%; mean age: 47 ± 13 years) from a Portuguese health centre. Traditional cardiovascular disease risk factors were measured during clinical assessment. Arterial stiffness was evaluated with carotid-femoral pulse wave velocity (≥ 10 m/s). Physical inactivity (< 30 min/day of moderate to vigorous physical activity) was objectively assessed with accelerometry. The statistical procedures included descriptive analysis (means, medians, and frequencies) and between-gender comparisons (chi-square test and *t*-test) for cardiovascular disease risk factors.

Results. Cardiovascular disease risk factors prevalence was as follows: dyslipidaemia: 71%, physical inactivity: 51%, hypertension: 43%, metabolic syndrome: 36%, arterial stiffness: 31%, smoking: 29%, and obesity: 20%. The prevalence of cardiovascular disease risk factors increases with age and is higher in males than in females. The prevalence of hypertension and metabolic syndrome was higher in participants with a lower educational level. The majority of hypertensive patients were more physically inactive (56.5%) than active (43.5%; *p* = 0.044).

Conclusions. The overall prevalence of cardiovascular disease risk factors was high, with 1/3 having augmented arterial stiffening and half being physically inactive.

Key words: epidemiology, prevalence, cardiovascular risk factors, lifestyle, physical activity

Introduction

Despite the reduced trend observed in the recent decades, coronary artery disease and stroke are still among the major causes of premature death in Portugal and across the European countries [1]. Both conditions contribute to disability and to the mounting costs of healthcare, which could be primarily reduced through early detection and management of their determinants [2].

Cardiovascular diseases (CVD) are strongly related to biological, socio-demographic, and lifestyle risk factors, historically divided into modifiable or non-modifiable ones [1]. The presence of only one risk factor augments the probability of a CVD event, but a grouping of several implies an exponential growth in that probability [3]. Metabolic syndrome (MetS) is a good example of a cluster of risk factors which doubles the probability of cardiovascular events [4].

Apart from the traditional, well-known risk factors, other biological markers have been proposed as independent predictors of cardiovascular events and cardiovascular mortality [5]. However, some of them are not yet included in cardiovascular surveillances and risk stratifications owing to methodological constraints in clinical practice. Arterial stiffness is an adequate example of one biological marker that could help in the early detection of high-risk patients and, consequently, in the early control and management of cardiovascular risk [5, 6]. Arterial stiffness denotes an augmented rigidity of the central large arteries, and is a consequence of intima and media layers structural changes resulting from multiple cellular insults across the lifespan [7]. Previous evidence reports that indi-

viduals with high compared with those with normal arterial stiffness have a superior cardiovascular event risk (relative risk: 2.26; 95% confidence interval: 1.89–2.70) and cardiovascular mortality (relative risk: 2.02; 95% confidence interval: 1.68–2.42) after adjusting for traditional risk factors [5]. Nevertheless, measuring arterial stiffness as a cardiovascular risk factor in primary care setting is still unusual, and, generally, reports on CVD risk factors do not encompass it.

Regular surveillance and screening for CVD risk factors are relevant in clinical settings because they allow to properly manage each risk factor and thus to diminish the risk of CVD events and mortality. Therefore, the study aims to describe the overall prevalence of CVD risk factors, including arterial stiffness and physical inactivity, in adults registered in a Portuguese primary care centre.

Material and methods

Study design

The study was carried out in a public primary care medical centre (Porto, Portugal). The inclusion criterion was age between 18 and 65 years. Participants with a history of severe hypertension, peripheral arterial disease, arrhythmia, acute coronary syndrome, thyroid disorders, severe renal or pulmonary disorders, infectious or chronic immunological diseases, neurological or orthopaedic deficiencies, known CVD, or cognitive disorders were excluded.

Participants

The patients were recruited from the medical centre archives that involved 8000 citizens. Overall, 4600 individuals were considered candidates following the inclusion criteria, and 1200 were randomly chosen (see Figure 1). They were invited to the study via phone calls. More details can be found elsewhere [8].

Data collection

Data were collected during 2 appointments. In the first appointment, a physician checked eligibility conditions, collected socio-demographic information and habitual medication data. After that, the participants were evaluated for anthropometry and haemodynamics. Finally, each subject received an accelerometer to be worn for the next 7 days. A week later, the patients revisited the health centre in order to return the accelerometers and provide blood samples.

Cardiovascular risk factors

Socio-demographic factors

Age was categorized in decades, and education involved 3 categories based on the number of years schooling (< 6 years, 6–12 years, and > 12 years).

Tobacco consumption

The participants had to indicate whether they smoked or not.

Obesity

Body weight (kg) (Tanita Inner Scan BC-522, Tokyo, Japan) and height (m) (standard wall-mounted stadiometer) were measured with the patients barefoot and wearing light clothing. Body mass index (BMI) was calculated as the ratio of weight (kg) and squared height (m), and classified as underweight (≤ 18.49 kg/m^2), normal weight (18.50–24.99 kg/m^2), overweight (25–29.99 kg/m^2), and obese (≥ 30 kg/m^2).

Blood pressure

Blood pressure was measured (Colin, BP 8800 monitor, Critikon, USA) on the participants' left arm after 20-minute resting, in the supine position. Three measurements were performed spaced 1 minute apart. The average value was used as the final blood pressure. Additional measurements were performed when differences between readings surpassed 5 mm Hg, being the extreme value discarded for the final calculation of blood pressure. Hypertension was defined as stated in the European Society of Cardiology and European Society of Hypertension guidelines (i.e., systolic blood pressure ≥ 140 and/or diastolic blood pressure ≥ 90 mm Hg or presence of antihypertensive medication) [3].

Arterial stiffness

Arterial stiffness was measured with the gold-standard method known as carotid-femoral pulse wave velocity (cfPWV) by using applanation tonometry (SphygmoCor device; AtCor Medical, Australia). The procedures followed the international best practices described elsewhere [6]. Two acceptable attempts of cfPWV were achieved, and the average between them was used for statistical procedures. The cut-off point of cfPWV ≥ 10 m/s was used to establish augmented

arterial stiffness [6]. Room temperature was set at ca. 21°C, and the space was both quiet and semi-dark.

Dyslipidaemia

A nurse collected blood samples after 12-hour fasting. An automated clinical chemistry analyser Olympus AU5400 (Beckman-Coulter) was used to measure total cholesterol, triglycerides, high-density lipoprotein cholesterol, and serum glucose. The Friedewald equation was applied to calculate low-density lipoprotein cholesterol concentration. Any lipid impairment in accordance with the international reference values [9] and/or a current prescription of lipid-lowering medication was stated as the presence of dyslipidaemia [3].

Metabolic syndrome

MetS is a condition defined as the simultaneous coexistence of a minimum of 3 metabolic risk factors out of the following: central obesity (male waist circumference ≥ 102 cm, female waist circumference ≥ 88 cm); systolic ≥ 130 mm Hg and/or diastolic ≥ 85 mm Hg blood pressures and/or antihypertensive medication; low levels of high-density lipoprotein cholesterol (males: < 40 mg/dl, females: < 50 mg/dl) or specific lipid treatment; triglycerides ≥ 150 mg/dl and/or specific lipid-lowering treatment; and fasting glucose ≥ 100 mg/dl or medication for elevated glucose level [4].

Physical activity

Physical activity was accurately captured with accelerometry (ActiGraph GT1M, USA). The apparatus was placed on the right hip during waking hours for 7 consecutive days, but removed during water-based activities.

Raw activity (i.e., counts/min) was transformed into daily physical activity (ActiLife 6.9 software, ActiGraph, USA). The accelerometry data were validated when the participants had at least 8 hours of use per day, and 4 days of use. The cut-off point ≥ 2020 counts/min was applied to ascertain moderate to vigorous physical activity [10]. The total time spent above the cut-off point was summed and averaged per day. The risk factor of physical inactivity was set as < 30 min/day in moderate to vigorous physical activity [11].

Statistical analysis

Normality was verified by using the Kolmogorov-Smirnov test. Variables that were not normally distributed (BMI, systolic blood pressure, cfPWV, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, total cholesterol, triglycerides, and fasting glucose) were transformed into their natural logarithm for analysis and then transformed back to the original scale for the purpose of clarity. These variables are expressed as mean ± standard deviation. Moderate to vigorous physical activity was not normally distributed and normalization was not possible. In this sense, moderate to vigorous physical activity is expressed as median and interquartile range. Between-gender comparisons were performed by using the independent *t*-test and the non-parametric test for 2 independent samples, as appropriate. Categorical variables are expressed as frequencies, and between-group comparisons were performed with the chi-squared test.

The procedures were carried out with the IBM SPSS 20 software (SPSS, Chicago, USA), and the results were considered significant at $p < 0.05$.

Ethical approval

The research related to human use has complied with all the relevant national regulations and institutional policies, has followed the tenets of the Declaration of Helsinki, and has been approved by the Ethics Committee of the Portuguese North Regional Health Authority (I.P. 25/2010).

Informed consent

Informed consent has been obtained from all individuals included in this study.

Results

A total of 318 patients from the sampled 1200 missed the phone call. Additional 244 refused to participate, and 348 met at least one of the exclusion criteria. Overall, 290 scheduled the first appointment, but 33 missed it.

Out of the 257 adults who participated in the study, 197 had simultaneous data for biochemical analyses, physical activity, and arterial stiffness; they comprised the final sample (Figure 1).

Table 1 depicts the sample characteristics. The genders were significantly different for total cholesterol (males: 189.2 ± 37.5 mg/dl, females: 203.1 ± 38.5 mg/dl; $p < 0.05$), diastolic blood pressure (males: 76.5 ± 10.3 mm Hg, females: 73.3 ± 11.0 mm Hg; $p < 0.05$), triglycerides (males: 121.6 ± 63.3 mg/dl, females: 103.8 ± 48.6 mg/dl; $p < 0.05$), and high-density lipoprotein (males: 48.0 ± 10.9 mg/dl, females: 61.7 ± 15.3 mg/dl; $p < 0.001$).

Population (*n* = 8000)

Assessed for eligibility (*n* = 4600)

Randomized (*n* = 1200)

Missed phone calls (*n* = 318)
Declined to participate (*n* = 244)
Exclusion criteria (*n* = 348)
Missed the appointment (*n* = 33)

Assessed (*n* = 257)

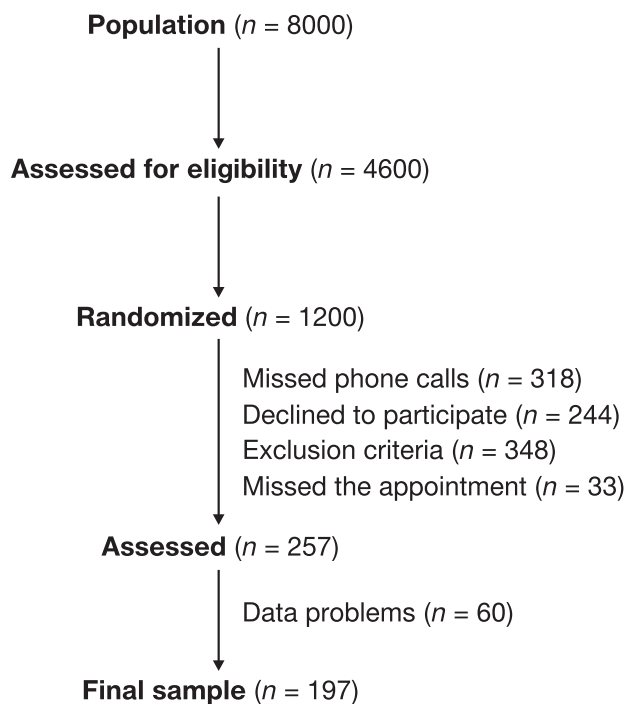Data problems (*n* = 60)

Final sample (*n* = 197)

Figure 1. Study flowchart

The prevalence of CVD risk factors is depicted in Table 2. Overall, 71% of the participants had dyslipidaemia. Physical inactivity was the second most prevalent risk factor (overall prevalence: 51%), with females showing a tendency to be more physically inactive compared with males (*p* = 0.053). The cfPWV value ≥ 10 m/s was the fifth most prevalent risk factor (31%). A significant difference was observed between genders in smoking, with males (40%) having a higher prevalence than females (22%; *p* < 0.01).

The prevalence of hypertension, dyslipidaemia, and MetS rises with age. These occurred more often in the overweight BMI category and among non-smokers (Table 3). In females, hypertension was significantly more frequent in those physically inactive (67%) than in physically active (33%; *p* = 0.044).

Regarding arterial stiffness, the prevalence of hypertension, dyslipidaemia, and MetS were higher in the category of cfPWV ≥ 10 m/s compared with the normal, but the differences were not significant (*p* = 0.568, 0.081, and 0.296, respectively).

Table 1. Sample characteristics with between-gender comparisons

Characteristics	Males (*n* = 83)	Females (*n* = 114)	Overall (*n* = 197)
Socio-demographic			
Age (years)	47.5 ± 13.7	47.3 ± 12.4	47.4 ± 12.9
Educational level (*n* [%])			
< 6 years	25 (30%)	43 (38%)	68 (35%)
6–12 years	21 (26%)	18 (16%)	39 (20%)
> 12 years	36 (44%)	52 (46%)	88 (45%)
Anthropometric			
Height (cm)	170 ± 6.5	157 ± 6.5**	163 ± 9.3
Weight (kg)	78.9 ± 12.0	65.8 ± 12.5**	71.3 ± 13.9
Body mass index (kg/m^2)	27.1 ± 3.9	26.6 ± 4.6	26.8 ± 4.3
Haemodynamic			
Systolic blood pressure (mm Hg)	127.7 ± 14.2	126.1 ± 17.7	126.8 ± 16.3
Diastolic blood pressure (mm Hg)	76.5 ± 10.3	73.3 ± 11.0*	74.7 ± 10.8
Pulse wave velocity (m/s)	9.2 ± 1.8	8.9 ± 2.0	9.1 ± 1.9
Lipid and metabolic profile			
Triglycerides (mg/dl)	121.6 ± 63.3	103.8 ± 48.6*	111.4 ± 55.9
LDL cholesterol (mg/dl)	117.4 ± 35.1	120.0 ± 36.6	118.9 ± 35.8
HDL cholesterol (mg/dl)	48.0 ± 10.9	61.7 ± 15.3**	55.9 ± 15.2
Total cholesterol (mg/dl)	189.2 ± 37.5	203.1 ± 38.5*	197.1 ± 38.6
Fasting glucose (mg/dl)	98.1 ± 18.2	94.7 ± 35.7	96.1 ± 29.5
Physical activity			
MVPA (min/day)	33 (20–54)	26 (16–48)	30 (18–50)

LDL – low-density lipoprotein, HDL – high-density lipoprotein, MVPA – moderate to vigorous physical activity
* *p* < 0.05, ** *p* < 0.001

Table 2. Prevalence of risk factors with between-gender comparisons

Risk factors	Overall	Males	Females	Statistical inference
Dyslipidaemia (*n* [%])	140 (71%)	61 (74%)	79 (69%)	χ^2: 0.411 (1); *p* = 0.316
Physical inactivity (*n* [%])	100 (51%)	36 (43%)	64 (56%)	χ^2: 3.132 (1); *p* = 0.053
Hypertension (*n* [%])	85 (43%)	40 (48%)	45 (40%)	χ^2: 1.489 (1); *p* = 0.141
Metabolic syndrome (*n* [%])	81 (41%)	38 (46%)	43 (38%)	χ^2: 1.290 (1); *p* = 0.161
Arterial stiffness (*n* [%])	61 (31%)	28 (34%)	33 (29%)	χ^2: 0.601 (1); *p* = 0.267
Smoking (*n* [%])	58 (29%)	33 (40%)	25 (22%)	χ^2: 7.350 (1); *p* < 0.01
Obesity (*n* [%])	40 (20%)	17 (20%)	23 (20%)	χ^2: 3.400 (2); *p* = 0.183

Table 3. Cardiovascular risk factors depending on socio-demographic, body mass index, lifestyle,
and arterial stiffness characteristics

Factors	Hypertension			Dyslipidaemia			Metabolic syndrome		
	Males (*n* = 40; 48%)	Females (*n* = 45; 40%)	Overall (*n* = 85; 43%)	Males (*n* = 61; 74%)	Females (*n* = 79; 69%)	Overall (*n* = 140; 71%)	Males (*n* = 38; 46%)	Females (*n* = 43; 38%)	Overall (*n* = 81; 41%)
Socio-demographics									
Age categories (years)									
Under 30 (*n* [%])	0 (0%)	0 (0%)	0 (0%)	4 (7%)	3 (4%)	7 (5%)	0 (0%)	1 (2%)	1 (1%)
31–40 (*n* [%])	2 (5%)	4 (9%)	6 (7%)	6 (10%)	11 (14%)	17 (12%)	3 (8%)	5 (12%)	8 (10%)
41–50 (*n* [%]))	5 (13%)	7 (16%)	12 (14%)	12 (20%)	17 (22%)	29 (21%)	7 (18%)	3 (7%)	10 (12%)
51–60 (*n* [%])	17 (43%)	17 (38%)	34 (40%)	20 (33%)	28 (35%)	48 (34%)	16 (42%)	15 (35%)	31 (39%)
61–65 (*n* [%])	16 (40%)	17 (38%)	33 (39%)	19 (31%)	20 (25%)	39 (28%)	12 (32%)	19 (42%)	31 (38%)
Statistical inference	χ^2: 0.739 (3); *p* = 0.869			χ^2: 1.546 (4); *p* = 0.819			χ^2: 4.421 (4); *p* = 0.353		
Education									
< 6 years (*n* [%])	16 (41%)	24 (55%)	40 (48%)	21 (34%)	33 (42%)	54 (39%)	15 (40%)	26 (62%)	41 (51%)
6–12 years (*n* [%])	10 (26%)	8 (18%)	18 (22%)	16 (26%)	9 (12%)	25 (18%)	9 (24%)	5 (12%)	14 (18%)
> 12 years (*n* [%])	13 (33%)	12 (27%)	25 (31%)	24 (39%)	36 (42%)	60 (43%)	14 (37%)	11 (26%)	25 (31%)
Statistical inference	χ^2: 1.567 (2); *p* = 0.457			χ^2: 5.023 (2); *p* = 0.081			χ^2: 4.265 (2); *p* = 0.119		
Body mass index									
Normal weight (*n* [%])	2 (5%)	10 (22%)	12 (14%)	13 (21%)	29 (37%)	42 (30%)	2 (5%)	10 (23%)	12 (14%)
Overweight (*n* [%])	24 (60%)	21 (47%)	45 (53%)	33 (54%)	32 (41%)	65 (46%)	20 (53%)	19 (44%)	39 (42%)
Obesity (*n* [%])	14 (35%)	14 (31%)	28 (33%)	15 (25%)	18 (23%)	33 (24%)	16 (42%)	14 (33%)	30 (37%)
Statistical inference	χ^2: 5.257 (2); *p* = 0.072			χ^2: 4.137 (2); *p* = 0.126			χ^2: 5.203 (2); *p* = 0.074		
Lifestyle									
Tobacco consumption									
Non-smoking (*n* [%])	28 (70%)	42 (93%)	70 (82%)	38 (62%)	62 (78%)	100 (71%)	25 (66%)	38 (88%)	63 (78%)
Smoking (*n* [%])	12 (30%)	3 (7%)	15 (18%)	23 (38%)	17 (22%)	40 (29%)	13 (34%)	5 (12%)	18 (22%)
Statistical inference	χ^2: 7.933 (1); *p* = 0.005			χ^2: 4.419 (1); *p* = 0.028			χ^2: 5.952 (1); *p* = 0.014		
Physical activity									
Physically active (*n* [%])	22 (55%)	15 (33%)	37 (43.5%)	32 (53%)	35 (44%)	67 (48%)	20 (53%)	15 (35%)	35 (43%)
Physically inactive (*n* [%])	18 (45%)	30 (67%)	48 (56.5%)	29 (48%)	44 (56%)	73 (52%)	18 (47%)	28 (65%)	46 (57%)
Statistical inference	χ^2: 4.044 (1); *p* = 0.044			χ^2: 0.917 (1); *p* = 0.216			χ^2: 2.589 (1); *p* = 0.083		
Arterial stiffness									
Normal (*n* [%])	18 (46%)	21 (47%)	39 (46%)	34 (57%)	55 (70%)	39 (46%)	20 (53%)	19 (44%)	39 (48%)
≥ 10 m/s (*n* [%])	21 (54%)	24 (53%)	45 (54%)	26 (43%)	24 (30%)	45 (53%)	18 (47%)	24 (56%)	42 (52%)
Statistical inference	χ^2: 0.002 (1); *p* = 0.568			χ^2: 2.484 (1); *p* = 0.081			χ^2: 0.576 (1); *p* = 0.296		

Discussion

In this study, the prevalence of CVD risk factors was as follows: dyslipidaemia: 71%, physical inactivity: 51%, hypertension: 43%, MetS: 41%, arterial stiffness: 31%, smoking: 29%, and obesity: 20%.

Dyslipidaemia was established as any abnormal lipid profile or even the presence of lipid-lowering medication [3]. Indeed, dyslipidaemia is a broader concept, which exceeds a single lipid disorder [9]. Prevalence studies on dyslipidaemia in Portuguese populations are normally based on total cholesterol or on each lipid concentration separately [12, 13]. In this sense, among 40–76-year-old Portuguese patients from the same geographical area as our study referred to, hypercholesterolemia prevalence was 79.7% [12].

Physical inactivity was the second most prevalent CVD risk factor (51%). In a global CVD risk factors ranking, physical inactivity appears in the 4th position [14]. This discrepancy might be somehow explained by the use of different assessment methods between the studies. It is imperative to underline that a large body of evidence in this field is based on self-report measurements, which underestimate physical inactivity [10, 15]. Considering this, it is expected that objectively measured physical activity would re-arrange the CVD risk factors ranking.

In the present study, all waking minutes identified with ≥ 2020 counts/min were summed to derive moderate to vigorous physical activity and to classify participants as physically active [10]. This approach is a study limitation and has 2 consequences. Firstly, it justifies the median of 30 min/day in moderate to vigorous physical activity of the total sample. Secondly, it may have attenuated the prevalence of physical inactivity, once physical activity recommendations state that the minimum duration of a physical activity bout should last at least 10 consecutive minutes [11]. The dose-response effect of physical activity on cardiovascular health is supported by strong evidence, and numerous physiological pathways might explain this association [16]. For example, regular physical activity improves the lipid profile, lowering especially the triglycerides [17], and improves insulin sensitivity and glycaemic control, reducing the risk of metabolic diseases [18]. An acute aerobic physical activity bout augments cardiac output and the resultant shear stress forces stimulate the release of vasodilating substances such as nitric oxide and prostaglandins, diminishing blood pressure [19]. Additionally, the hypotensive effect gathered from chronic regular physical activity is linked with a reduction in oxidative stress and in low-grade inflammation [16]. Cardiovascular risk factors damage both the intima and media layers of arterial walls, impairing arterial compliance [6, 7], and, once more, regular physical activity is protective through its effect on each of the cardiovascular risk factors and also owing to its impact on cardiorespiratory fitness, a well-known parameter related to cardiovascular health [20]. Undeniably, all risk factors are somehow positively affected by physical activity, which justifies the importance of its promotion in terms of public health for the prevention of CVD.

The third most prevalent CVD risk factor was hypertension, and our data are in accordance with previous national (ca. 42%) [21] and international (ca. 30–45%) [22] reports based on the adult general population. In participants with hypertension, this particular risk factor was significantly higher among those who were simultaneously physically inactive. Indeed, as previously discussed, several physiological mechanisms are plausible to explain the pathways of physical activity action on blood pressure regulation, including, among others, improvement in endothelial function, diminishing chronic low-grade inflammatory state, improvement in the autonomic nervous system [16].

MetS results from a complex interplay between environmental and genetics factors and doubles the risk of developing CVD in 5–10 years' time [4]. Prevention of MetS onset is linked to a proper managing of lifestyle risk factors, highlighting physical activity [23]. Among our participants, 36% had MetS, and this is a higher prevalence compared with the global data (20–25%) [23]. The large variability in MetS prevalence might arise from the criteria used to define the condition [23]. In this sense, Santos and Barros [24] observed that within the same sample (from the same geographical area as ours), MetS varied between 26.4% and 41.4%, depending on the selected criteria.

Overall, we found that 31% of our sample presented a cfPWV ≥ 10 m/s stated by the Reference Values for Arterial Stiffness' Collaboration [6]. Between-study comparisons are difficult because data are almost inexistent. Nonetheless, the prevalence of cfPWV ≥ 10 m/s was 18.7% in 2542 Portuguese citizens aged between 18 and 96 years [25]. Raised arterial stiffness has been proposed as a pivotal CVD risk factor because it independently predicts both CVD and mortality [5]. Arterial stiffness is mainly determined by age and blood pressure [6], but other health conditions (dyslipidaemia, insulin resistance, obesity) and lifestyle risk factors (physical activity, sedentary time, diet, and tobacco consumption) exert effects that cannot be over-

looked [5, 8]. Augmented arterial stiffness means that central arteries are losing its cushion capacity and, consequently, the blood flows faster, at higher pressures [7]. The arterial tree adapts to this flow pattern, and in terms of haemodynamic response, accelerated forward and backward waves are generated, intensifying the end-systolic pressure and reducing the coronary perfusion pressure [7]. The altered blood flow pattern in stiff arteries damages the heart, brain, and kidneys, and increases the risk of adverse events [6]. Early vascular aging means that the arteries are older compared with the chronological age. To some extent, it is expected in individuals with a constellation of risk factors [6]. Nevertheless, it is also possible to occur in apparently healthy people, free from traditional risk factors that are covered by the well-known cardiovascular risk algorithms [3]. Considering this, the additional predictive value of arterial stiffness as a cardiovascular biomarker appears to be relevant in the general population and must be confirmed in future studies.

The smoking prevalence was 29%, which is close to the recently reported 28.3% for a Portuguese sample [26]. We did not collect information on tobacco load or the time since tobacco consumption cessation, which constitutes a limitation of the study. Surprisingly, the prevalence of CVD risk factors was significantly higher in non-smokers than in smokers. Others have already published the same pattern [27] and there is no plausible explanation. We might guess that some of the non-smokers could be ex-smokers. As previously mentioned, the methodology used to assess smoking habits represents a limitation of this report and could at least partially explain these results. Additionally, it is eventually possible to speculate that the respondents may not have been totally honest when answering the smoking status question. If so, future studies might test for cotinine in order to verify the smoking status.

The prevalence of obesity (20%) was higher than previously reported for the Portuguese population [28]. Data from 2014 showed an obesity prevalence of 16.4%, which had risen by 1.2% since 2005 [28]. It is well known that obesity worsens most of the CVD risk factors [29], a reason why within participants with overweight or obesity, the prevalence of hypertension, dyslipidaemia, and MetS increases.

Taking into account the socio-demographic factors, the prevalence of hypertension, dyslipidaemia, and MetS increases with age, and is superior in males compared with females, as well as among those with the lowest educational level. These results are in agreement with the literature [22]. The educational level is the widest measure of the socioeconomic status, and

in the last 40 years, an inverse association between education level and CVD prevalence has been consistently reported [30]. We can hypothesize that lower educational levels might be associated with worse lifestyle choices and with an eventually lower income, which reduce the level of information and health literacy, and limit the adoption of a healthy lifestyle owing to economic constraints.

Limitations

The sample was small and not representative of all geographical areas of the country, impeding generalizations for the entire Portuguese population. Seasonality influences the amount of daily physical activity and this was not considered in the analysis. The utilization of a specific cut-off point of moderate to vigorous physical activity might result in some misclassification of participants who were near this value; physical activity was considered as the sum of all minutes regardless the minimum duration of 10 minutes. In this study, the exclusion criteria could constitute a constraint for the generalization of the results. However, we intended to assess asymptomatic individuals who might be at risk of the initiation and progression of a pathology underlying ischemic cardiovascular diseases.

Conclusions

Our report shows a high prevalence of CVD risk factors within a sample of Portuguese citizens. Augmented arterial stiffening was observed in 1/3 of our sample, and physical inactivity was the second most prevalent risk factor, present in 51% of the sample.

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