eISSN 2255-0569

ORIGINAL

Relationship between alcohol consumption and other variables with the values of different cardiovascular risk factors in 139634 Spanish workers

Relación entre el consumo de alcohol y otras variables con los valores de diferentes factores de riesgo cardiovascular en 139.634 trabajadores españoles

Joan Obrador de Hevia[1], Ángel Arturo López-González[1], José Ignacio Ramírez-Manent[1,2], Hernán Paublini Oliveira[1], Pedro Juan Tárraga López[3], Pere Riutord-Sbert[1]

1. ADEMA-Health Group of IUNICS. Balearic Islands. Spain
2. Primary Care in Mallorca. Spain
3. Professor of Medicine University of Castilla la Mancha. Spain

Corresponding author
Pedro Juan Tárraga López
E-mail: pjtarraga@sescam.jccm.es

Received: 18 - VII - 2024
Accepted: 15 - VIII - 2024

doi: 10.3306/AJHS.2024.39.06.132

Abstract

Introduction and objectives: Alcohol is a toxic substance widely consumed in the world and is responsible for a multitude of pathologies of different organs and body systems. The aim of this study is to determine the effect of alcohol consumption on arterial hypertension, dyslipidaemia and hyperglycaemia.
Methodology: A double study was carried out, on the one hand a descriptive and transversal study in 139634 workers and on the other hand a retrospective longitudinal study in 40431 workers. The effect of alcohol consumption and other variables and healthy habits on three cardiovascular risk factors such as arterial hypertension, dyslipidaemia and hyperglycaemia was assessed.
Results: All the variables analysed have an influence on the cardiovascular risk factors studied, with alcohol consumption being one of the most influential.
Conclusions: Alcohol consumption and other variables favour the appearance of arterial hypertension, dyslipidaemia and hyperglycaemia in our sample.

Key words: Alcohol consumption, cardiovascular risk, arterial hypertension, dyslipidemia, hyperglycaemia.

Resumen

Introducción y objetivos: El alcohol es un tóxico muy consumido en el mundo y es responsable de multitud de patologías de diferentes órganos y sistemas corporales. El objetivo de este estudio es conocer el efecto del consumo de alcohol sobre la hipertensión arterial, dislipemia e hiperglucemia.
Metodología: Se realiza un doble estudio, por una parte un estudio descriptivo y transversal en 139634 trabajadores y por otra un estudio longitudinal retrospectivo en 40431 trabajadores. Se valora el efecto del consumo de alcohol y de otras variables y hábitos saludables en tres factores de riesgo cardiovascular como son la hipertensión arterial, la dislipemia y la hiperglucemia.
Resultados: Todas las variables analizadas influyen sobre los factores de riesgo cardiovascular estudiados, el consumo de alcohol es uno de los que más influyen.
Conclusiones: El consumo de alcohol y otras variables favorecen la aparición de hipertensión arterial, dislipemia e hiperglucemia en nuestra muestra.

Palabras clave: Consumo de alcohol, riesgo cardiovascular, hipertensión arterial, dislipemia, hiperglucemia.

Cite as: Obrador de Hevia J, López-González ÁA, Ramírez-Manent JI, Paublini Oliveira H, Tárraga López PJ, Riutord-Sbert P. Relationship between alcohol consumption and other variables with the values of different cardiovascular risk factors in 139634 Spanish workers. *Academic Journal of Health Sciences 2024*;39 (6): 132-141 doi: 10.3306/AJHS.2024.39.06.132

Introduction

Alcohol consumption is a complex and multifaceted phenomenon that has been the subject of intense scientific and public health scrutiny due to its varied effects on human health[1]. While moderate alcohol consumption has been associated with certain health benefits, such as a reduced risk of coronary heart disease[2,3], prolonged heavy drinking can have serious adverse consequences[4]. Among the most critical conditions that can be exacerbated or induced by alcohol consumption are various cardiometabolic pathologies[5].

Hypertension is one of the most common and potentially fatal conditions related to excessive alcohol consumption[6]. Epidemiological studies have shown a consistent relationship between alcohol consumption and an increase in blood pressure[7,8]. Alcohol-induced hypertension is a well-documented phenomenon, where the consumption of significant amounts of alcohol, especially on a chronic basis, is associated with substantial increases in systolic and diastolic blood pressure levels. The mechanism behind alcohol-induced hypertension involves several physiological and biochemical factors. Alcohol can increase the activity of the sympathetic nervous system, resulting in vasoconstriction and an increase in blood pressure[9-12]. In addition, alcohol influences electrolyte balance, especially sodium and potassium, which may further contribute to elevated blood pressure[13,14].

In addition, the pattern of alcohol consumption has been found to play a crucial role. Episodic heavy episodic drinking (binge drinking)[15] has a more pronounced impact on blood pressure compared to moderate and regular drinking. This is because episodes of binge drinking can cause acute fluctuations in blood pressure, as well as endothelial damage and oxidative stress, contributing to the development of long-term hypertension[16].

Dyslipidaemia, which refers to altered blood lipid levels, is another condition that can be significantly influenced by alcohol consumption[17]. Dyslipidaemia is characterised by elevated levels of triglycerides and LDL (low-density lipoprotein) cholesterol, as well as low levels of HDL (high-density lipoprotein) cholesterol, which increases the risk of cardiovascular disease.

Alcohol has complex effects on lipid metabolism. On the one hand, moderate alcohol consumption has been associated with elevated levels of HDL-cholesterol, which is considered cardioprotective[18,19]. However, excessive alcohol consumption has detrimental effects on the lipid profile[20]. Alcohol increases hepatic triglyceride synthesis and can cause alcoholic fatty liver disease, a condition that contributes to elevated triglyceride levels in the blood[21,22] .

In addition, alcohol can affect apolipoprotein B (apoB), an essential protein in the formation of very low density lipoprotein (VLDL) and LDL[23]. The alcohol-induced increase in VLDL production by the liver results in increased levels of LDL in the circulation[24]. This process can be exacerbated by the presence of other risk factors, such as a diet rich in saturated fat and a sedentary lifestyle.

Hyperglycaemia is a metabolic condition that can be directly influenced by alcohol consumption. The relationship between alcohol and blood glucose is complex, as alcohol can affect glucose homeostasis in several ways, depending on the amount consumed, the frequency and the presence of other metabolic conditions.

Chronic alcohol consumption can lead to insulin resistance, a state in which the body's cells do not respond adequately to insulin, the hormone responsible for regulating blood glucose levels. Insulin resistance is a major risk factor for the development of type 225 diabetes. Studies have shown that excessive alcohol consumption can decrease insulin sensitivity and disrupt insulin signalling, leading to elevated blood glucose levels[26].

In addition, alcohol can interfere with hepatic gluconeogenesis[27], the process by which the liver produces glucose from non-carbohydrate precursors. Acute alcohol consumption can inhibit this process, resulting in hypoglycaemia in the short term. However, chronic alcohol consumption can lead to hyperglycaemia due to impaired liver function and inadequate glucose production[28].

The influence of alcohol on hyperglycaemia also depends on the pattern of consumption. Moderate and regular alcohol consumption may have less detrimental effects compared to episodic and heavy drinking. Excessive alcohol consumption can cause rapid and extreme fluctuations in glucose levels, increasing the risk of metabolic complications[29].

Although it is well known that alcohol consumption favours the appearance of cardiometabolic diseases, the quantification of this effect has been less studied, so the aim of this study is to find out how much alcohol consumption affects, among other variables, the appearance of high values of different cardiometabolic risk factors in a group of Spanish workers.

Material and methods

A double study was carried out, firstly a descriptive and cross-sectional study on 139634 workers from different Spanish regions and belonging to almost all employment sectors (56352 women and 83282 men) selected from those who attended the periodic medical check-ups carried out in the different participating companies.

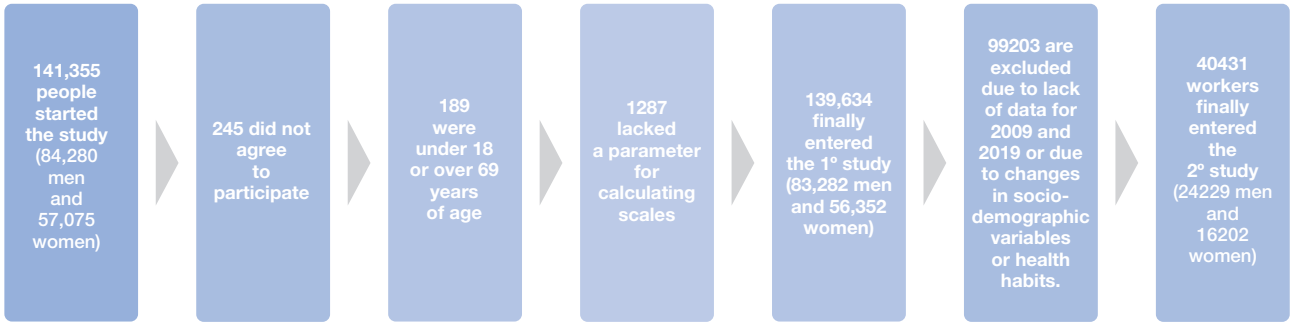
A second, retrospective longitudinal study was carried out on a sample selected from the previous one and which included 40431 workers (24229 men and 16202 women) and covered the period 2009-2019.

All variables, analytical, anthropometric and clinical, were obtained by the health professionals of the different companies once the protocols were homogenised to avoid inter-observer bias.

The following inclusion criteria were established:
- Being between 18 and 69 years of age (working age).
- Be employed by one of the participating companies and not be in a situation of temporary incapacity at the time of data collection.
- Have all the variables to calculate the different cardiovascular risk scales.
- Agree to participate in the study and to cede the data for epidemiological studies.
- To have data from 2009 and 2019 and that the socio-demographic variables and healthy habits had not changed in that period (only for the retrospective longitudinal study).

The selection of participants in both studies is presented in the flow chart (**Figure 1**).

Figure 1: Flow chart of the participants.



Determination of variables

The various companies' health specialists were in charge of gathering all the clinical, analytical, and anthropometric data required to determine an individual's heart age. The measurements were standardized to reduce interobserver bias. With the subject standing and their abdomen relaxed, the waist circumference was measured using a tape measure at the level of the last rib. After the patient had rested for ten minutes, blood pressure was taken using an OMRON M3 sphygmomanometer while the patient was seated. The mean was found after three measurements were made. Following a 12-hour fast, the study was carried out utilizing precipitation techniques for HDL cholesterol and enzymatic procedures for blood glucose, triglycerides, and cholesterol. The Friedewald formula was used to indirectly calculate LDL cholesterol. All analytical parameters were expressed in mg/dL.

The Joint National Committee (JNC-8) criteria[30], which classify hypertension as values of arterial tensile stress above 140 mmHg and/or arterial diastolic stress above 90 mmHg and/or be using antihypertensive therapy, are applied to classify arterial tensile stress values.

If there are high total cholesterol, low HDL cholesterol, low LDL cholesterol, or triglicéridos cholesterol values, or if treatment is being received, then dislipemia is considered[31].

The American Diabetes Association's recommendations were followed in classifying the baseline blood glucose results. The person was classified as having hyperglycemia if the values were >= 100 mg/dl in two separate determinations, if the person also had a HbA1c ≥ 6.0%, or if the person was on hypoglycemic medication[32].

A person was classified as a smoker if, within the previous thirty days, they had consumed one or more cigarettes each day, or the equivalent in other consumption modalities, or had given up smoking less than a year prior.
The PREDIMED study's "Mediterranean diet adherence questionnaire" was used to evaluate the heart-healthy diet. There are 14 questions total, using a 0-1 point system. A heart-healthy diet is indicated by values of 9 or higher, which also imply strong adherence[33].

The International Physical Activity Questionnaire (IPAQ), which evaluates physical activity throughout the previous week, is used to measure physical activity[34].

Units of alcohol (UA) are used to measure alcohol intake. One UA is equivalent to ten grams of pure ethanol in Spain. High consumption was defined as consuming 14 UA or more per week for women and 21 UA for males[35].

The workers were categorized into three social classes using the 2011 National Classification of Occupations (CNO-11) and the Spanish Society of Epidemiology's standards[36]. I. University professionals and managers. II.

Skilled independent contractors and intermediate jobs. III. Workers lacking skill.

The educational level was divided into three categories: elementary school, high school, and university.

Ethical considerations.

The 2013 Declaration of Helsinki and the institutional research committee's ethical guidelines were upheld throughout the investigation. The confidentiality and anonymity of the information gathered may always be ensured. The Research Ethics Committee of the Balearic Islands (CEI-IB) approved the study (IB 4383/20).

Every worker's data that was part of the study was coded, so only the person in charge of the study could determine who was who. The study participants will be able to exercise their rights to access, rectification, cancellation, and opposition of the collected data, as guaranteed by the research team's strict adherence to the Organic Law 3/2018, of December 5, on the protection of personal data and guarantee of digital rights.

Statistical analysis.

The Student's t-test was employed to ascertain the mean and standard deviation for quantitative data. The chi-square test was used to find prevalences for qualitative variables. Multinomial logistic regression was used to carry out the multivariate analysis. To perform the statistical analysis, SPSS 28.0 was employed. A p-value of less than 0.05 was considered statistically significant.

Results

The anthropometric and clinical details of the study participants are displayed in **table I**. The analyses comprised 139.634 individuals, of which 83.282 men (59.6%) and 56.352 women (40.4%) were included. The sample's average age was just over 40, with the majority of the group being in the 30- to 49-year-old age range. Men had more negative anthropometric, clinical, and analytical findings. With only an elementary education, the majority of the labor force belonged to social class III. The majority of men did not follow a balanced diet or engage in regular physical activity (the situation was better for women). Approximately one-third of the employees smoked.

Table II shows the mean values of the different clinical and analytical parameters analysed according to the different socio-demographic variables and healthy habits. We observe that all of them increase in value as age increases, as the socioeconomic level (social class and education) decreases and when healthy habits are not present, i.e. in smokers, regular alcohol consumers, sedentary people and those with low adherence to the Mediterranean diet. This situation is repeated in both sexes, although the mean values are always higher in men.

Table I: Characteristics of the population.

	Men n=83,282	Women n=56,352	
	Mean (SD)	Mean (SD)	p-value
Age (years)	41.4 (10.7)	40.1 (10.4)	<0.001
Height (cm)	173.8 (7.1)	161.2 (6.5)	<0.001
Weight (kg)	83.2 (14.6)	66.3 (13.9)	<0.001
Systolic blood pressure (mmHg)	126.2 (15.9)	115.6 (15.7)	<0.001
Diastolic blood pressure (mmHg)	76.6 (10.9)	71.1 (10.7)	<0.001
Total cholesterol (mg/dL)	199.6 (38.6)	194.6 (36.9)	<0.001
HDL-cholesterol (mg/dL)	50.0 (7.7)	54.7 (9.2)	<0.001
LDL-cholesterol (mg/dL)	122.6 (37.4)	121.5 (37.1)	<0.001
Triglycerides (mg/dL)	133.8 (95.6)	90.8 (49.7)	<0.001
Glucose (mg/dL)	93.0 (25.4)	86.8 (18.1)	<0.001
	%	%	p-value
< 30 years	15.1	18.0	<0.001
30-39 years	29.6	31.0	
40-49 years	30.2	30.3	
50-59 years	20.9	17.7	
60-69 years	4.2	3.0	
Social class I	7.5	13.6	<0.001
Social class II	23.8	32.1	
Social class III	68.7	54.1	
Elementary school	66.4	48.1	<0.001
High school	26.9	40.0	
University	6.7	11.9	
Non-smokers	66.8	67.9	<0.001
Smokers	33.2	32.1	
Non physical activity	62.4	51.4	<0.001
Yes physical activity	37.6	48.6	
Non Mediterranean diet	65.8	52.8	<0.001
Yes Mediterranean diet	34.2	47.2	
Non alcohol consumption	67.3	84.4	<0.001
Yes alcohol consumption	32.7	15.6	

LDL- Low density lipoprotein-cholesterol. HDL-c high density lipoprotein-cholesterol. SD Standard deviation.

Table II: Mean values of different cardiovascular risk parameters according sociodemographic variables and healthy habits by gender.

		SBP	DBP	TC	LDL-c	TG	Glucose
Men	n	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
< 30 years	12558	120.3 (12.9)	70.6 (9.4)	171.7 (32.9)	98.6 (32.0)	97.1 (62.5)	84.4 (14.5)
30-39 years	24648	122.0 (13.2)	74.2 (10.0)	193.8 (36.0)	118.1 (34.8)	123.0 (92.6)	88.3 (18.3)
40-49 years	25178	126.9 (15.0)	78.5 (10.5)	208.8 (38.3)	124.4 (36.8)	145.7 (103.2)	93.8 (26.4)
50-59 years	17370	133.3 (18.0)	80.3 (10.1)	212.0 (35.6)	130.3 (36.5)	153.4 (73.6)	101.5 (31.7)
60-69 years	3528	137.0 (18.4)	80.9 (10.8)	212.2 (31.9)	133.6 (36.9)	154.3 (102.4)	107.4 (34.9)
Social class I	6234	124.1 (14.1)	75.8 (10.2)	192.2 (34.6)	121.6 (38.3)	123.2 (79.9)	91.2 (22.0)
Social class II	19856	126.0 (15.9)	76.4 (10.8)	198.7 (39.3)	123.8 (33.8)	133.2 (89.7)	92.2 (24.7)
Social class III	57192	127.5 (16.4)	77.6 (11.0)	202.5 (37.5)	125.2 (35.8)	135.1 (99.1)	93.4 (26.0)
Elementary school	55306	127.3 (16.4)	77.5 (11.0)	201.4 (37.4)	125.0 (38.1)	138.4 (100.0)	100.5 (38.8)
High school	22408	126.0 (15.8)	76.6 (10.9)	199.8 (34.2)	123.4 (36.7)	132.7 (95.2)	92.4 (23.0)
University	5568	124.4 (14.1)	76.2 (10.1)	198.8 (39.4)	122.0 (38.1)	125.1 (80.1)	90.0 (16.6)
Non-smokers	55618	125.5 (16.1)	75.8 (11.1)	198.4 (40.6)	120.9 (39.1)	129.6 (88.3)	92.7 (26.6)
Smokers	27664	126.6 (15.8)	77.0 (10.7)	200.2 (37.5)	123.5 (36.6)	142.1 (108.5)	93.1 (24.8)
Non physical activity	51984	129.4 (16.3)	79.1 (10.7)	211.4 (39.0)	130.4 (39.9)	163.9 (108.4)	96.2 (27.2)
Yes physical activity	31298	120.9 (13.7)	72.5 (9.7)	179.9 (28.6)	109.8 (28.6)	83.6 (28.4)	87.6 (21.0)
Non Mediterranean diet	54792	129.0 (16.3)	78.9 (10.7)	211.5 (38.4)	130.7 (39.5)	159.8 (107.1)	95.7 (26.9)
Yes Mediterranean diet	28490	120.9 (13.7)	72.3 (9.7)	176.7 (26.7)	107.2 (27.1)	83.8 (30.1)	87.7 (21.4)
Non alcohol consumption	56022	124.1 (15.3)	75.1 (10.8)	194.6 (38.1)	120.5 (36.9)	115.0 (75.4)	87.2 (11.8)
Yes alcohol consumption	27260	130.7 (16.2)	79.8 (10.4)	209.7 (37.5)	127.0 (38.1)	172.3 (118.6)	104.9 (38.4)
Women	n	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
< 30 years	10110	109.3 (11.6)	66.6 (8.7)	175.2 (32.1)	101.7 (32.5)	80.5 (38.7)	81.5 (12.1)
30-39 years	17460	110.9 (12.9)	68.5 (9.7)	184.9 (33.1)	112.9 (34.0)	83.0 (43.2)	84.0 (14.5)
40-49 years	17094	117.3 (15.4)	72.6 (10.7)	199.4 (34.0)	126.9 (34.3)	92.2 (52.4)	88.1 (19.3)
50-59 years	9984	124.7 (17.3)	76.3 (10.9)	218.2 (35.6)	143.7 (35.8)	108.0 (58.8)	93.1 (21.5)
60-69 years	1704	129.7 (18.1)	77.3 (10.9)	222.9 (34.2)	144.7 (36.4)	115.5 (54.7)	98.3 (26.4)
Social class I	7632	109.9 (12.5)	68.4 (9.3)	185.8 (34.4)	111.2 (35.8)	83.4 (41.2)	84.7 (13.6)
Social class II	18112	114.8 (15.5)	70.3 (10.8)	194.7 (35.5)	121.9 (35.5)	88.4 (49.6)	85.9 (16.2)
Social class III	30608	117.4 (16.2)	72.2 (10.8)	196.8 (37.9)	123.9 (38.0)	94.0 (51.3)	88.0 (19.9)
Elementary school	27086	118.3 (16.4)	72.3 (10.9)	197.9 (37.8)	125.3 (37.4)	93.6 (51.8)	87.4 (17.7)
High school	22574	114.0 (15.0)	70.3 (10.7)	193.6 (36.0)	120.4 (36.6)	89.7 (49.1)	86.8 (19.5)
University	6692	109.6 (12.5)	68.6 (9.3)	184.9 (33.9)	110.2 (35.4)	83.0 (40.7)	85.1 (14.1)
Non-smokers	38252	114.4 (15.1)	70.2 (10.4)	193.2 (37.6)	120.4 (36.9)	89.6 (48.9)	85.9 (17.8)
Smokers	18100	116.1 (15.9)	71.4 (10.8)	195.3 (36.5)	122.1 (37.2)	93.1 (51.2)	87.3 (18.2)
Non physical activity	28962	120.5 (16.5)	74.4 (11.0)	210.5 (38.5)	136.0 (39.0)	109.6 (60.0)	90.5 (20.7)
Yes physical activity	27390	110.3 (12.8)	67.5 (9,0)	177.8 (26.2)	106.3 (27.8)	70.8 (22.1)	83.0 (13.8)
Non Mediterranean diet	29764	120.2 (16.5)	74.3 (11.0)	210.9 (38.1)	136.1 (38.7)	107.4 (59.7)	90.1 (20.4)
Yes Mediterranean diet	26588	110.4 (12.9)	67.4 (9.1)	176.3 (24.9)	105.3 (27.3)	72.2 (24.1)	83.2 (14.1)
Non alcohol consumption	47536	113.4 (14.3)	69.7 (10.0)	191.4 (35.8)	119.2 (36.6)	84.5 (40.8)	83.3 (10.2)
Yes alcohol consumption	8816	127.1 (17.8)	78.4 (11.4)	211.9 (37.9)	134.1 (37.5)	124.7 (73.5)	106.1 (33.0)

SBP Systolic blood pressure DBP Diastolic blood pressure. TC Total cholesterol. LDL- Low density lipoprotein-cholesterol. HDL-c high density lipoprotein-cholesterol. TG Triglycerides. SD Standard deviation

Table III, which displays the prevalence of the cardiovascular risk factors that have been examined, shows a trend that is comparable to the median values, that is, an influence from advanced age, lower socioeconomic class, and unhealthy habits. In this instance, the prevalences in varones are also higher.

The multivariate analysis (**Table IV**) shows us how all of the variables examined in this study affect the appearance of the three cardiometabolic risk factors (hypertension, dyslipemia, and hyperglycemia), with age being the factor that influences it the most. However, both alcohol consumption and sedentarism as well as poor adherence to the Mediterranean diet have a notable impact.

In the long-term retrospective study covering the years 2009 to 2019, we find that the disparity in the prevalence of the three cardiovascular risk factors examined in this study during that time increases with age, declines in socioeconomic status, and continued use of unhealthy habits (alcohol, tobacco, sedentarism, and poor adherence to Mediterranean diet). The biggest differences in both sexes are associated with alcohol use, sedentary lifestyles, and diets. In every instance, the differences found are smaller in women. The complete set of data may be viewed in **table V**.

Table III: Prevalence of high values of different different cardiovascular risk parameters according sociodemographic variables and healthy habits by gender.

		Hypertension	Dislipidemia	Hyperglycaemia
Men	**n**	**%**	**%**	**%**
< 30 years	12558	10.5	29.9	8.2
30-39 years	24648	15.4	52.4	13.1
40-49 years	25178	26.7	70.1	23.0
50-59 years	17370	39.9	76.6	35.8
60-69 years	3528	49.1	80.8	47.4
Social class I	6234	18.5	56.0	16.9
Social class II	19856	24.8	60.1	19.1
Social class III	57192	26.1	63.6	22.9
Elementary school	55306	26.8	62.9	22.8
High school	22408	24.3	59.9	18.9
University	5568	19.0	58.1	18.1
Non-smokers	55618	23.5	59.7	21.3
Smokers	27664	25.1	61.0	21.9
Non physical activity	51984	31.8	78.7	26.3
Yes physical activity	31298	12.5	30.6	12.7
Non Mediterranean diet	54792	30.8	77.5	26.0
Yes Mediterranean diet	28490	12.7	28.2	12.9
Non alcohol consumption	56022	20.3	53.0	14.1
Yes alcohol consumption	27260	33.4	76.2	36.8
Women	**n**	**%**	**%**	**%**
< 30 years	10110	2.3	26.1	4.0
30-39 years	17460	4.5	36.7	6.9
40-49 years	17094	12.0	54.1	13.4
50-59 years	9984	24.3	75.8	23.3
60-69 years	1704	30.9	80.5	34.5
Social class I	7632	3.5	37.1	7.4
Social class II	18112	9.4	48.3	9.7
Social class III	30608	13.3	51.1	14.6
Elementary school	27086	13.8	52.8	14.1
High school	22574	3.5	46.5	10.9
University	6692	3.1	36.3	7.9
Non-smokers	38252	9.2	46.5	11.5
Smokers	18100	11.4	49.2	12.4
Non physical activity	28962	17.2	68.7	18.5
Yes physical activity	27390	3.8	26.8	5.2
Non Mediterranean diet	29764	17.0	68.4	13.6
Yes Mediterranean diet	26588	3.9	27.8	5.5
Non alcohol consumption	47536	7.3	44.5	4.6
Yes alcohol consumption	8816	28.8	68.9	52.6

Table IV: Multinomial logistic regression.

	Hypertension	Dyslipidemia	Hyperglycaemia
	OR (95% CI	**OR (95% CI**	**OR (95% CI**
Women	1	1	1
Men	2.42 (2.34-2.50)	1,45 (1.41-1.48)	1.36 (1.27-1.46)
< 30 years	1	1	1
30-39 years	1.39 (1.31-1.47)	1.25 (1.16-1.34)	1.41 (1.28-1.54)
40-49 years	2.43 (2.28-2.58)	1.96 (1.82-2.11)	2.38 (2.14-2.63)
50-59 years	4.50 (4.21-4.79)	3.35 (3.10-3.61)	3.28 (2.91-3.65)
60-69 years	6.27 (5.79-6.76)	6.03 (5.56-6.51)	3.89 (3.28-4.49)
Social class I	1	1	1
Social class II	1.15 (1.10-1.21)	1.19 (1.13-1.25)	1.21 (1.15-1.27)
Social class III	1.52 (1.42-1.62)	1.68 (1.52-1.82)	1.72 (1.58-1.86)
University	1	1	1
High school	1.12 (1.08-1.17)	1.20 (1.14-1.26)	1.23 (1.17-1.30)
Elementary school	1.48 (1.40-1.57)	1.60 (1.48-1.73)	1.70 (1.60-1.81)
Non-smokers	1	1	1
Smokers	1.14 (1.09-1.20)	1.12 (1.08-1.16)	1.19 (1.15-1.24)
Yes physical activity	1	1	1
Non physical activity	1.98 (1.85-2.11)	2.61 (2.50-2.73)	2.48 (2.01-2.96)
Yes Mediterranean diet	1	1	1
Non Mediterranean diet	1.30 (1.22-1.39)	2.18 (1.98-2.39)	2.12 (1.89-2.27)
Non alcohol consumption	1	1	1
Yes alcohol consumption	1.19 (1.15-1.23)	1.35 (1.26-1.45)	2.33 (2.02-2.64)

OR Odds ratio. In all cases p<0.001

Table V: Longitudinal retrospective study by gender.

		Hypertension		Dyslipidemia		Hyperglycaemia	
Men	**n**	**%PRE-%POST**	**Difference**	**%PRE-%POST**	**Difference**	**%PRE-%POST**	**Difference**
30-39 years	8477	13.7-15.4	12.8	47.2-52.3	10.8	11.1-13.2	18.5
40-49 years	8583	22.1-26.4	19.3	54.5-63.2	15.9	19.2-23.6	22.9
50-59 years	5908	32.7-40.2	22.8	58.5-70.1	19.8	28.1-36.1	28.3
60-69 years	1261	39.6-51.3	29.7	64.1-78.4	22.4	34.9-46.3	32.5
Social class I	1900	15.9-17.9	12.5	48.7-54.9	12.8	12.0-14.4	20.3
Social class II	5769	21.5-25.0	16.5	52.3-60.1	14.9	14.0-17.5	24.8
Social class III	16560	21.5-26.0	20.9	54.5-63.6	16.7	18.3-23.2	26.9
University	1706	16.2-18.3	12.8	49.9-56.2	12.6	25.3-30.4	20.0
High school	6501	21.0-24.4	16.3	52.0-59.7	14.8	15.5-19.3	24.9
Elementary school	16022	22.5-27.1	20.7	54.4-63.6	17.0	14.6-18.5	27.0
Non-smokers	16244	20.6-24.3	17.9	52.3-60.0	14.8	17.3-21.4	23.8
Smokers	7985	20.5-24.9	21.3	52.1-60.7	16.5	18.0-22.7	26.3
Yes physical activity	9184	11.6-12.6	8.9	28.5-30.8	7.9	11.5-13.0	12.8
Non physical activity	15045	25.2-32.1	27.4	63.3-78.6	24.2	19.8-27.2	37.3
Yes Mediterranean diet	8363	11.7-12.8	9.2	26.1-28.5	8.2	11.5-13.0	13.0
Non Mediterranean diet	15866	24.4-31.0	26.8	62.4-77.3	23.9	19.3-26.5	37.0
Non alcohol consumption	16258	17.8-20.2	13.5	47.9-52.8	10.3	12.5-14.4	15.2
Yes alcohol consumption	7971	26.2-33.9	29.5	60.3-76.2	26.4	26.6-37.0	39.3
Global	24229	20.8-24.7	18.9	52.2-60.5	15.9	17.5-21.8	24.6
Women	**n**	**%PRE-%POST**	**Difference**	**%PRE-%POST**	**Difference**	**%PRE-%POST**	**Difference**
30-39 years	6169	3.6-4.0	9.8	33.6-36.1	7.3	5.9-6.7	13.1
40-49 years	5954	10.9-12.3	12.8	48.7-54.5	11.8	11.5-13.4	16.8
50-59 years	3494	21.1-24.7	17.2	66.6-76.3	14.6	18.6-22.4	20.3
60-69 years	585	25.0-30.8	23.4	66.7-79.8	19.6	27.3-34.2	25.4
Social class I	2128	2.7-3.0	9.8	34.4-37.6	9.3	6.1-7.0	14.5
Social class II	5290	7.9-8.8	11.2	42.0-47.1	12.2	6.7-7.8	15.9
Social class III	8784	11.7-13.5	15.3	45.0-51.5	14.4	12.0-14.3	19.6
University	1848	2.5-2.8	9.7	33.9-37.0	9.2	6.0-6.9	14.3
High school	6518	7.7-8.6	11.3	40.6-45.5	12.0	6.8-7.9	15.8
Elementary school	7836	12.3-14.2	15.2	46.4-53.2	14.7	12.0-14.4	19.9
Non-smokers	10992	8.1-9.1	12.8	41.3-46.2	12.0	7.7-9.0	16.8
Smokers	5210	10.0-11.4	14.3	43.2-49.2	13.8	10.1-12.1	19.6
Yes physical activity	7875	3.2-3.4	5.2	25.1-26.6	5.8	5.0-5.4	8.5
Non physical activity	8227	14.2-17.2	21.4	57.5-68.7	19.5	13.9-17.7	26.9
Yes Mediterranean diet	7570	3.5-3.7	5.5	24.0-25.5	6.3	5.1-5.6	8.9
Non Mediterranean diet	8632	13.8-16.7	20.6	57.3-68.2	19.1	13.6-17.1	26.1
Non alcohol consumption	13707	6.7-7.2	6.8	40.5-44.6	10.2	4.0-4.5	11.9
Yes alcohol consumption	2495	23.0-29.4	27.9	53.3-68.5	28.4	39.9-51.5	29.2
Global	16202	9.4-10.6	13.2	42.9-48.2	12.4	10.011.8	17.8

PRE year 2009. POST year 2019

Discussion

In our study alcohol consumption, in addition to physical activity, Mediterranean diet, tobacco consumption, age, socioeconomic status and gender influence the occurrence of hypertension, dyslipidaemia and hyperglycaemia.

A study that selected large prospective cohort studies in the Japanese population[37] assessed the effect of alcohol consumption on hypertension and other cardiovascular diseases and concluded, as we do, that irrespective of age and gender, alcohol consumption was positively associated with an increased risk of hypertension. Somewhat different data were found in a review from 2020 where in the case of women, alcohol consumption had a J-shaped relationship with hypertension. About 1 to 2 standard drinks per day were associated with a lower risk of developing hypertension, while for men, the relationship was relatively linear[38].

Another cross-sectional study in 12,285 people aged 37-66 years[39] using, as in our investigation, multiple logistic regression models and including several potential confounders such as age, education, marital status, body mass index (BMI), physical activity, smoking, coffee consumption and statin use concluded that consumption of 0.1-15.0 g/day was associated with a lower risk of hypertension in women ($p < 0.05$). In men, in the adjusted model, there were no associations between alcohol consumption and the presence of hypertension.

A systematic review and meta-analysis with 2865 participants (2464 men and 401 women) in 36 trials[40] found that in people who drank two drinks or less per day, a decrease in alcohol intake did not correlate with a significant decrease in blood pressure; however, in people who drank more than two drinks per day, a decrease in alcohol intake correlated with a more significant decrease

in blood pressure. People who drank six or more drinks per day experienced a more significant reduction in systolic blood pressure (mean difference -5.50 mm Hg, 95% CI -6.70 to -4.30) and diastolic blood pressure (-3.97, -4.70 to -3.25).

We can highlight as strengths of the study the large size of the two samples analysed and the wide range of variables studied, as well as the fact that the influence of these variables has been assessed over a long period of time (10 years).

As a major problem, we found that people younger than 18 years and older than 69 years did not participate in the study because they were not of working age. As a result, our findings may not be generalisable to the whole population.

According to epidemiological evidence, moderate consumption of red wine reduces cardiovascular mortality and the incidence of diabetes. It is not yet known whether these effects are the result of ethanol or the non-alcoholic components of red wine. A randomised crossover trial examined 67 men at high cardiovascular risk[41]. After a preparation period, each received red wine (30 g alcohol per day) and the equivalent amount of de-alcoholised red wine and gin (30 g alcohol per day), in a randomised order for four weeks. Fasting plasma glucose and insulin, plasma lipoproteins, apolipoproteins and adipokines were measured at baseline and after each intervention, as well as the homeostasis model of insulin resistance (HOMA-IR). Adjusted mean plasma insulin and HOMA-IR decreased after red wine and de-alcoholised red wine, while fasting glucose remained constant throughout the study. Red wine and gin increased HDL-cholesterol and apolipoprotein A-I and A-II. The red wine intervention reduced lipoprotein(a). These findings support the positive effect of the non-alcoholic fraction of red wine (mainly polyphenols) on insulin resistance. Red wine is more protective against cardiovascular disease than other alcoholic beverages.

These same authors conducted a systematic review of human clinical studies and meta-analyses[42] on moderate alcohol consumption and cardiovascular disease between 2000 and 2012 and concluded that heavy alcohol consumption increases morbidity and mortality. However, moderate alcohol consumption, particularly of polyphenol-rich alcoholic beverages such as wine and beer, appears to have cardiovascular health benefits in healthy subjects and even in patients with documented cardiovascular disease.

An article[43] examines the connection between alcohol intake and the elements of the metabolic syndrome, as well as the epidemiological data supporting alcohol's potential vascular preventive benefits and the likely molecular processes that underlie them. The protective, harmful, or J-shaped relationships between alcohol intake and metabolic syndrome are discussed in this article. The intricate mechanical link between alcohol intake and each metabolic syndrome component may be the cause of this debate, and practically all studies have distinct flaws and limits. In light of this, he draws the conclusion that further research is required to verify the link between alcohol use and the prevalence of metabolic syndrome as well as to evaluate the impact of drinking habits and other potential risk factors like smoking, physical activity, socioeconomic status, education, employment, diet, and exercise.

Alcohol consumption has a significant influence on high blood pressure, dyslipidaemia and hyperglycaemia. These adverse effects underline the importance of moderating alcohol consumption and considering its potential impacts on metabolic and cardiovascular health. Scientific evidence suggests that while moderate alcohol consumption may have some benefits, prolonged heavy drinking has considerable negative effects that may increase the risk of cardiovascular and metabolic diseases.

It is essential that health professionals educate their patients about the risks associated with heavy drinking and encourage responsible drinking habits. The implementation of public health policies that reduce excessive alcohol consumption and promote a healthy lifestyle is also crucial to mitigate the risks associated with these conditions.

Conclusion

The profile of the person at greatest risk of developing arterial hypertension, dyslipidaemia and hyperglycaemia will be a male, older, regular alcohol consumer, smoker, sedentary, with low adherence to the Mediterranean diet and with a low socioeconomic level.

Conflict of interest
None

Funding
None

References

1. Hendriks HFJ. Alcohol and Human Health: What Is the Evidence? Annu Rev Food Sci Technol. 2020 Mar 25;11:1-21. doi: 10.1146/annurev-food-032519-051827.

2. Masip J, Germà Lluch JR. Alcohol, health and cardiovascular disease. Rev Clin Esp (Barc). 2021 Jun-Jul;221(6):359-368. doi: 10.1016/j.rceng.2019.07.001

3. Braillon A, Wilson M. Does moderate alcohol consumption really have health benefits? BMJ. 2018 Sep 17;362:k3888. doi: 10.1136/bmj.k3888.

4. Anderson P. Lower-Strength Alcohol Products and Public Health. Nutrients. 2023 May 9;15(10):2240. doi: 10.3390/nu15102240.

5. Rosoff DB, Davey Smith G, Mehta N, Clarke TK, Lohoff FW. Evaluating the relationship between alcohol consumption, tobacco use, and cardiovascular disease: A multivariable Mendelian randomization study. PLoS Med. 2020 Dec 4;17(12):e1003410. doi: 10.1371/journal.pmed.1003410.

6. Roerecke M. Alcohol's Impact on the Cardiovascular System. Nutrients. 2021 Sep 28;13(10):3419. doi: 10.3390/nu13103419.

7. Klatsky AL. Alcohol and cardiovascular diseases: where do we stand today?. Journal of Internal Medicine 2015;278(3):238-50.

8. Vacca A, Bulfone L, Cicco S, Brosolo G, Da Porto A, Soardo G, et al. Alcohol Intake and Arterial Hypertension: Retelling of a Multifaceted Story. Nutrients. 2023 Feb 15;15(4):958. doi: 10.3390/nu15040958.

9. DeLalio LJ, Sved AF, Stocker SD. Sympathetic Nervous System Contributions to Hypertension: Updates and Therapeutic Relevance. Can J Cardiol. 2020 May;36(5):712-720. doi: 10.1016/j.cjca.2020.03.003.

10. Seravalle G, Mancia G, Grassi G. Sympathetic Nervous System, Sleep, and Hypertension. Curr Hypertens Rep. 2018 Jul 6;20(9):74. doi: 10.1007/s11906-018-0874-y.

11. da Silva AA, do Carmo JM, Wang Z, Hall JE. Melanocortin-4 Receptors and Sympathetic Nervous System Activation in Hypertension. Curr Hypertens Rep. 2019 Apr 26;21(6):46. doi: 10.1007/s11906-019-0951-x.

12. Greenlund IM, Cunningham HA, Tikkanen AL, Bigalke JA, Smoot CA, Durocher JJ, et al. Morning sympathetic activity after evening binge alcohol consumption. Am J Physiol Heart Circ Physiol. 2021 Jan 1;320(1):H305-H315. doi: 10.1152/ajpheart.00743.2020.

13. Kawano Y. Physio-pathological effects of alcohol on the cardiovascular system: its role in hypertension and cardiovascular disease. Hypertension Research 2010 33(3), 193-202.

14. Grillo A, Salvi L, Coruzzi P, Salvi P, Parati G. Sodium Intake and Hypertension. Nutrients. 2019 Aug 21;11(9):1970. doi: 10.3390/nu11091970.

15. Voskoboinik A, Prabhu S, Ling LH, Kalman JM, Kistler PM. Alcohol and Atrial Fibrillation: A Sobering Review. J Am Coll Cardiol. 2016 Dec 13;68(23):2567-2576. doi: 10.1016/j.jacc.2016.08.074.

16. Roerecke M, Rehm J. Alcohol consumption, drinking patterns, and ischemic heart disease: a narrative review of meta-analyses and a systematic review and meta-analysis of the impact of heavy drinking occasions on risk for moderate drinkers. BMC Medicine 2017;12:182.

17. Klop B, do Rego AT, Cabezas MC. Alcohol and plasma triglycerides. Curr Opin Lipidol. 2013 Aug;24(4):321-6. doi: 10.1097/MOL.0b013e3283606845.

18. Mukamal KJ. Alcohol consumption and lipids: one more challenge to the cardiologist?. Heart 2012; 98(11):823-24.

19. Kovář J, Zemánková K. Moderate alcohol consumption and triglyceridemia. Physiol Res. 2015;64(Suppl 3):S371-5. doi: 10.33549/physiolres.933178.

20. Zhou JY, Park S. Regular exercise, alcohol consumption, and smoking interact with the polygenetic risk scores involved in insulin sensitivity and secretion for the risk of concurrent hyperglycemia, hypertension, and dyslipidemia. Nutrition. 2021 Nov-Dec;91-92:111422. doi: 10.1016/j.nut.2021.111422.

21. Brinton EA. Effects of ethanol intake on lipoproteins. Current Atherosclerosis Reports 2010;12(3):166-71.

22. Tsuruya K, Yoshida H, Nagata M, Kitazono T, Iseki K, Iseki C, et al. Association of Hypertriglyceridemia With the Incidence and Progression of Chronic Kidney Disease and Modification of the Association by Daily Alcohol Consumption. J Ren Nutr. 2017 Nov;27(6):381-394. doi: 10.1053/j.jrn.2017.05.002

23. Chen Z, Tao T, Huang G, Tong X, Li Q, Su G. Analysis of the association between serum antiaging humoral factor klotho and cardiovascular disease potential risk factor apolipoprotein B in general population. Medicine (Baltimore). 2023 Jun 23;102(25):e34056. doi: 10.1097/MD.0000000000034056.

24. Gaziano JM, Buring JE, Breslow JL, Goldhaber SZ, Rosner B, VanDenburgh M, et al. Moderate alcohol intake, increased levels of high-density lipoprotein and its subfractions, and decreased risk of myocardial infarction. New England Journal of Medicine 2000; 332(15):1085-9.

25. Koppes LL, Dekker JM, Hendriks HF, Bouter LM, Heine RJ. Moderate alcohol consumption lowers the risk of type 2 diabetes: a meta-analysis of prospective observational studies. Diabetes Care 2005;28(3):719-25.

26. Baliunas DO, Taylor BJ, Irving H, Roerecke M, Patra J, Mohapatra S, et al. Alcohol as a risk factor for type 2 diabetes: A systematic review and meta-analysis. Diabetes Care 2009;32(11):2123-32.

27. Srinivasan MP, Shawky NM, Kaphalia BS, Thangaraju M, Segar L. Alcohol-induced ketonemia is associated with lowering of blood glucose, downregulation of gluconeogenic genes, and depletion of hepatic glycogen in type 2 diabetic db/db mice. Biochem Pharmacol. 2019 Feb;160:46-61. doi: 10.1016/j.bcp.2018.12.005.

28. Tappy L, Jequier E. Alcohol and glucose metabolism. In Metabolic effects of alcohol 2010 (pp. 15-30). CRC Press.

29. Tolstrup JS, Grønbæk M, Tybjaerg-Hansen A, Nordestgaard BG. Alcohol intake, alcohol dehydrogenase genotypes, and risk of diabetes: a prospective study and meta-analysis. Alcoholism: Clinical and Experimental Research 2008; 32(4):655-66.

30. James PA, Oparil S, Carter BL, Cushman WC, Dennison-Himmelfarb C, Handler J, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). JAMA. 2014 Feb 5;311(5):507-20. doi: 10.1001/jama.2013.284427. Erratum in: JAMA. 2014 May 7;311(17):1809.

31. Leone A, Vizzuso S, Brambilla P, Mameli C, Ravella S, De Amicis R, et al. Evaluation of Different Adiposity Indices and Association with Metabolic Syndrome Risk in Obese Children: Is there a Winner? Int J Mol Sci. 2020 Jun 8;21(11):4083. doi: 10.3390/ijms21114083

32. Esquivel Zuniga R, DeBoer MD. Prediabetes in Adolescents: Prevalence, Management and Diabetes Prevention Strategies. Diabetes Metab Syndr Obes. 2021 Nov 25;14:4609-4619. doi: 10.2147/DMSO.S284401.

33. Mitchell LJ, Ball LE, Ross LJ, Barnes KA, Williams LT. Effectiveness of Dietetic Consultations in Primary Health Care: A Systematic Review of Randomized Controlled Trials. J Acad Nutr Diet. 2017 Dec;117(12):1941-1962. doi: 10.1016/j.jand.2017.06.364.

34. Atehortúa A, Gkontra P, Camacho M, Diaz O, Bulgheroni M, Simonetti V, et al. Cardiometabolic risk estimation using exposome data and machine learning. Int J Med Inform. 2023 Nov;179:105209. doi: 10.1016/j.ijmedinf.2023.105209.

35. Li Y, Schoufour J, Wang DD, Dhana K, Pan A, Liu X, et al. Healthy lifestyle and life expectancy free of cancer, cardiovascular disease, and type 2 diabetes: prospective cohort study. BMJ. 2020 Jan 8;368:l6669. doi: 10.1136/bmj.l6669.

36. Domingo-Salvany A, Bacigalupe A, Carrasco JM, Espelt A, Ferrando J, Borrell C; et al. Propuestas de clase social neoweberiana y neomarxista a partir de la Clasificación Nacional de Ocupaciones 2011. Gac Sanit. 2013 May-Jun;27(3):263-72. Spanish. doi: 10.1016/j.gaceta.2012.12.009.

37. Ikehara S, Iso H. Alcohol consumption and risks of hypertension and cardiovascular disease in Japanese men and women. Hypertens Res. 2020 Jun;43(6):477-481. doi: 10.1038/s41440-020-0417-1.

38. Piano MR, Thur LA, Hwang CL, Phillips SA. Effects of Alcohol on the Cardiovascular System in Women. Alcohol Res. 2020 Jul 30;40(2):12. doi: 10.35946/arcr.v40.2.12.

39. Suliga E, Kozieł D, Ciesla E, Rebak D, Głuszek-Osuch M, Naszydłowska E, et al. The Consumption of Alcoholic Beverages and the Prevalence of Cardiovascular Diseases in Men and Women: A Cross-Sectional Study. Nutrients. 2019 Jun 12;11(6):1318. doi: 10.3390/nu11061318.

40. Roerecke M, Kaczorowski J, Tobe SW, Gmel G, Hasan OSM, Rehm J. The effect of a reduction in alcohol consumption on blood pressure: a systematic review and meta-analysis. Lancet Public Health. 2017 Feb;2(2):e108-e120. doi: 10.1016/S2468-2667(17)30003-8.

41. Chiva-Blanch G, Urpi-Sarda M, Ros E, Valderas-Martinez P, Casas R, Arranz S, et al. Effects of red wine polyphenols and alcohol on glucose metabolism and the lipid profile: a randomized clinical trial. Clin Nutr. 2013 Apr;32(2):200-6. doi: 10.1016/j.clnu.2012.08.022.

42. Chiva-Blanch G, Arranz S, Lamuela-Raventos RM, Estruch R. Effects of wine, alcohol and polyphenols on cardiovascular disease risk factors: evidences from human studies. Alcohol Alcohol. 2013 May-Jun;48(3):270-7. doi: 10.1093/alcalc/agt007.

43. Fujita N, Takei Y. Alcohol consumption and metabolic syndrome. Hepatol Res. 2011 Apr;41(4):287-95. doi: 10.1111/j.1872-034X.2011.00787.x.