RESEARCH ARTICLE

Dysglycemic states and hypertension: A relationship dependent on low-grade inflammation [version 1; peer review: 1 approved, 1 approved with reservations]

Juan Salazar ¹, Valmore Bermúdez ^{1,2}, Wheeler Torres¹, Víctor Arias², María Sofía Martínez¹, Mervin Chavez-Castillo¹, Sandra Wilches-Durán², Modesto Graterol-Rivas², Nelson Villasmil¹, Carla Navarro¹, Rosemily Graterol-Silva², Rosanna D'Addosio ³, Kyle Hoedebecke ^{4,5}, Manuel Velasco⁶, Joselyn Rojas ⁷

¹Endocrine and Metabolic Research Center, School of Medicine, University of Zulia, Maracaibo, Venezuela

²Grupo de Investigación Altos Estudios de Frontera (ALEF), Universidad Simón Bolívar, Barranquilla, Colombia

³Department of Public Health, School of Medicine, University of Zulia, Maracaibo, Venezuela

⁴WONCA Polaris - USA, Bangkok, 10500, Thailand

⁵Yongsan Health Clinic, Seoul, 96205, South Korea

⁶Clinical Pharmacology Unit, José María Vargas School of Medicine, Central University of Venezuela, Caracas, Venezuela

⁷Pulmonary and Critical Care Medicine Department, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

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Abstract

Background: Hypertension (HTN) is a prominent cardiovascular risk factor, affecting over 1 billion people worldwide. Identification of closely associated cardiometabolic conditions may be crucial for its early detection. The objective of this study was to identify factors associated with HTN and prehypertension (PHT) in an adult population sample from Maracaibo City, Venezuela.

Methods: A randomized multi-staged sampling cross-sectional study was performed in 2230 individuals from Maracaibo City Metabolic Syndrome Prevalence Study database. PHT and HTN were defined according to JNC-7 criteria. Multiple logistic regression analysis was used to assess the main risk factors for each condition.

Results: 52.6% (n=1172) of the subjects were female, the prevalence of HTN was 32% (n=714), while the prevalence of PHT was 31.1% (n=693). The main risk factors for HTN were age ≥ 60 years (odds ratio [OR]: 40.99; 95%CI: 16.94-99.19; $p < 0.001$) and the local indigenous ethnic group (OR: 3.06; 95%CI: 1.09-8.62; $p = 0.03$). Adjustment for high sensitivity C-reactive protein levels increased the OR of these factors and diminished the impact of other factors. Meanwhile, age ≥ 60 years (OR: 3.39; 95%CI: 1.41-8.18; $p = 0.007$) and alcohol consumption (OR: 1.49; 95%CI: 1.06-2.00; $p = 0.02$) were the main risk factors for PHT.

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1. **Luis M. Ruilope**, Hospital 12 de Octubre, Madrid, Spain

2. **Harry HX Wang**, Sun Yat-sen University, Guangzhou, China

Any reports and responses or comments on the article can be found at the end of the article.

Conclusion: There are significant differences in the risk factor profiles for HTN and PHT. Additionally, low-grade inflammation appears to link multiple metabolic factors and preexisting vascular characteristics.

Keywords

hypertension, prehypertension, risk factors, inflammation, C-reactive protein, Hispanic

Corresponding authors: Valmore Bermúdez (valmore@gmail.com), Kyle Hoedebecke (khoedebecke@gmail.com)

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Introduction

Hypertension (HTN) is the leading risk factor contributing to mortality worldwide, associated with approximately 9.4 million deaths every year¹. In addition, it is the main modifiable risk factor for the development of cardiovascular disease (CVD)², which causes the greatest numbers of death globally, with 17.5 million deaths in 2012³ and projections of up to 23.3 million deaths annually by 2030⁴. Given its complex etiology, HTN has been classified as primary when appearing without any identifiable cause (90% of all cases) and secondary HTN when resulting as a direct consequence of another known preexisting condition, such as cardiovascular or kidney disease⁵.

Research on primary HTN has focused on identifying different risk factors, which could help define its specific etiology⁶. Various genetic and environmental conditions have been categorized as modifiable and non-modifiable risk factors: Family history of HTN is considered to be one of the main non-modifiable risk factors⁷, along with age, sex and race⁸. On the other hand, high sodium intake, hypercaloric diets, smoking, alcohol consumption, obesity, and a sedentary lifestyle all represent modifiable risk factors^{9,10}.

Likewise, low-grade inflammation has been suggested to play a great role in the pathophysiology of HTN, due to the stimulation of the proliferation of endothelial vascular smooth muscle cells in addition to vascular remodeling and endothelial dysfunction¹¹. Several epidemiological studies have reported high levels of inflammatory markers, such as C-reactive protein (CRP) and proinflammatory cytokines in patients with HTN. Furthermore, these findings correlate with a higher risk of developing HTN, as well as target organ damage in healthy individuals¹².

Considering the high prevalence of HTN in the Venezuelan population¹³, it becomes imperative to identify the conditions associated with both HTN and prehypertension (PHT) in order to aid in early diagnosis. The objective of this study was to determine the main associated risk factors for HTN and PHT in adult patients from Maracaibo City, Venezuela.

Methods

Ethical considerations

This study was approved by the Bioethics Committee of the Endocrine and Metabolic Research Center – University of Zulia (approval number: BEC-006-0305). All participants enrolled in the study signed a written consent before any questioning, intervention, or physical examination. This ethical approval includes all future studies using this data set.

Population and sample selection

This report is part of the Maracaibo City Metabolic Syndrome Prevalence Study¹⁴, a cross-sectional study whose purpose is to identify metabolic syndrome and cardiovascular risk factors in the adult population of Maracaibo, the second largest city of Venezuela. The study sample was calculated based on city population estimates by the [National Institute of Statistics](#): (1,428,043 inhabitants for the year 2007), amounting to 1,986 individuals. An additional 244 subjects (12%) were included for oversampling in order to increase accuracy of the estimates obtained from

smaller subgroups within the overall sample, totaling 2,230 individuals¹⁴.

Maracaibo City is divided into parishes, where each parish was proportionally sampled through a multi-staged cluster selection method. In the first stage, clusters were represented by sectors from each of the 18 parishes with proportional representation. Four sectors were selected from each parish by simple randomized sampling. In the second phase, the clusters were represented by city blocks within each sector, which were selected by simple randomized sampling using a random number generator. From the overall population, 2,026 individuals were selected on the basis of availability of insulin determination. Further details of the sampling process have been previously published¹⁴.

Subject evaluation

A full medical history and blood samples for biochemical evaluation were obtained from each participant by trained personnel. A full physical examination was also performed. Collected data included age, gender, ethnic group, socioeconomic status (according to the method of Graffar modified by Mendez Castellano¹⁵), smoking habit and family history of HTN. High risk alcohol intake was defined as those consuming ≥ 1 gram of alcohol daily.

Physical activity (PA) was measured using the International PA Questionnaire-Long Form (IPAQ-LF)¹⁶. Each domain of the questionnaire was clustered in quintiles according to the calculated metabolic equivalents (METs) and classified according to the PA pattern in one of six categories: very low, low, moderate, high, and very high in addition to a category for inactive individuals or those whose METs equaled zero.

In addition, an anthropometric evaluation was performed: weight was determined using a digital scale (Tanita, TBF-310 GS Body Composition Analyzer, Tokyo-Japan) while height was measured with a previously calibrated stadiometer on a flat surface. Body Mass Index (BMI) was calculated with the Quetelet's formula [$\text{Weight}/(\text{Height})^2$] and individuals were categorized based on the World Health Organization (WHO) classification¹⁷. Waist circumference was measured with a plastic measuring tape graduated in centimeters and millimeters, taking an equidistant point between the costal margin and the anterior superior iliac spine as an anatomical reference, according to the protocol proposed by the US National Health Institute (NHI)¹⁸.

Blood pressure measurement

Blood pressure (BP) was taken through auscultation with subjects sitting down and feet on the floor following 15 minutes of rest, determined with a calibrated mercury sphygmomanometer. Korotkoff's phases I and V were identified as systolic and diastolic BP, respectively. BP was determined 3 times with 15 minutes intervals between each registration on two different days. BP was categorized according to the Seventh Joint National Committee on Prevention, Detection, Evaluation and Treatment of Hypertension (JNC-7) criteria¹⁹. PHT is classified as systolic BP between 120 mmHg and 139 mmHg or diastolic BP between 80 mmHg to 89 mmHg. HTN is classified as systolic BP greater than 140 mmHg or diastolic BP over 90 mmHg.

Laboratory analysis

Overnight fasting determination of glucose, total cholesterol, triacylglycerides (TAG), and HDL-C was performed with an automated analyzer (Human Gesellschaft für Biochemica und Diagnostica mbH, Germany); the intra-assay variation coefficients for total cholesterol, TAG, and HDL-C were 3%, 5%, and 5%, respectively. Serum hs-CRP was quantified through immunoturbidimetric assays (Human Gesellschaft für Biochemica und Diagnostica mbH, Germany). Insulin was quantified using ultra-sensitive ELISA double-sandwich methodology (DRG Instruments GmbH, Germany, Inc.).

Definitions

HOMA2-IR was utilized for the evaluation of insulin resistance (IR) as proposed by Levy *et al.*²⁰ computed with the HOMA-Calculator v2.2.2 software application with IR defined as HOMA2-IR ≥ 2 ²¹. Elevated hs-CRP was defined as levels ≥ 0.765 mg/L²²; hypertriacylglyceridemia was defined as fasting TAG ≥ 150 mg/dL; and low HDL-C as fasting HDL-C < 50 mg/dL in females or < 40 mg/dL in males²³. Regarding glycemic state, subjects were classified as: a) Normoglycemic, individuals with fasting glucose < 100 mg/dL; b) Impaired Fasting Glucose (IFG), those with fasting glycemia between 100–126 mg/dL; and c) Type 2 diabetes mellitus (DM2), those with fasting glycemia > 126 mg/dL or a previously established diagnosis²⁴. Likewise, waist circumference cutoff points ≥ 90 cm (females) and ≥ 95 cm (males) for the definition of abdominal obesity were used²⁵. We followed the methods similar to previous analyses with other risk factors²⁶.

Statistical analysis

Variables with non-normal distribution underwent logarithmic transformation, showing a normal distribution after the Geary test, qualitative variables were expressed in absolute and relative frequencies, evaluating association through the χ^2 test. Multiple logistic regression analysis was used to study the risk factors associated with PHT and HTN. Two models were devised for the latter, where the first model was adjusted by sex, age groups, ethnic groups, family history of hypertension, smoking, alcohol consumption, household physical activity, leisure time physical activity, hypertriglyceridemia, low HDL-C, glycemic status, BMI categories and elevated waist circumference. The second model was adjusted for all these factors and high serum hs-CRP levels. The multiple logistic regression model for PHT was adjusted for the same factors as the second model. Results were shown in Odds Ratios (95%CI) being statistically significant when $p < 0.05$. Data analysis was performed using the Statistical Software SPSS v.19 for Windows (IBM inc. Chicago, IL).

Results

Prevalence of hypertension and prehypertension

Normotensive individuals comprised 36.9% of the sample ($n=823$). The prevalence of PHT was 31.1% ($n=693$), while 32% ($n=714$) of the subjects were hypertensive. Therefore, a combined total of 63.1% of the sample showed altered BP levels (Figure 1).

General characteristics of the population

Table 1 and Table 2 show the distribution of subjects according to the presence of PHT and HTN. Age ($\chi^2=438.82$; $p < 0.0001$) and home PA ($\chi^2=39.53$ $p < 0.001$) were the main sociodemographic and psychobiologic factors associated with these conditions, respectively; whereas BMI was the main clinical-metabolic factor associated with HTN and PHT in the univariate analysis ($\chi^2=264.36$; $p < 0.0001$).

Risk factors for hypertension

In the first model (Table 3), the main risk factors for HTN were age ≥ 60 years (OR=33.79; 95%CI: 17.15-66.61; $p < 0.01$), and the diagnosis of DM2 (OR=2.92; 95%CI: 1.33-6.41; $p < 0.01$). After adjustment for elevated hs-CRP levels in the second model, the impact of age ≥ 60 years (OR=40.99; 95%CI: 16.94-99.19; $p < 0.01$) and family history of HTN (OR=2.70; 95%CI: 1.77-4.13; $p < 0.01$) increased; whereas characteristics such as the indigenous ethnic group (OR=3.06; 95%CI: 1.09-8.62; $p=0.03$) and IR (OR=1.50; 95%CI: 1.00-2.25; $p=0.05$) became significant risk factors, while conditions like hypertriacylglyceridemia, IFG, DM2 lost significance.

Risk factors for prehypertension

Table 4 depicts the multivariate analysis for factors associated with PHT. The main risk factors were age ≥ 60 years (OR=3.39; 95%CI: 1.41-8.18; $p < 0.01$) and age 30-59 years (OR=1.85; 95%CI: 1.29-2.65; $p < 0.01$); as well as alcohol intake ≥ 1 g/day (OR=1.49; 95%CI: 1.06-2.10; $p=0.02$) However, moderate PA demonstrated a protective effect (OR=0.62; 95%CI: 0.38-1.01; $p=0.05$).

Dataset 1. Raw data for the 2231 patient sample used in this study

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Discussion

HTN represents the main modifiable risk factor for the development of CVD², which is why it is alarming that only 4 of 10 individuals in this sample exhibit normal BP levels. According to the WHO, the prevalence of HTN in the Americas is 35%²⁷, similar to those in our study at 32%. In a cross-sectional study by Wang and Wang²⁸, based on the data obtained in the 1999–2000 National Health and Nutrition Examination Survey (NHANES) and using JNC-7 classification, the prevalence of HTN was slightly lower than ours (27.1%), while also reporting a similar prevalence for PHT (31%). Following the same methodology, Erem *et al.*²⁹ assessed a population of 4809 individuals in Turkey and reported results markedly differing from ours, with a prevalence of 44% for HTN and 14.5% for PHT. Lastly, another study by Do *et al.*³⁰ on a population of 17,199 Vietnamese individuals described a vastly different picture, where the prevalence of HTN was lower than PHT (20.7% vs 41.8%), which illustrates the heterogeneous distribution of this disease around the world.

Figure 1. Prevalence of hypertension and prehypertension in adult individuals from Maracaibo, Venezuela. 2016.

In regards to the non-modifiable risk factors for HTN, our multi-variate models showed that men had a higher risk of developing this disease, similar to several studies in Turkey²⁹, China³¹ and Guatemala³². However, age appears to be the main risk factor in our sample. In this context, a cross-sectional study by Awoke *et al.*³³ on 679 adult individuals aged ≥ 35 years ascertained greater HTN risk for individuals aged ≥ 55 years in comparison to those aged 35–44 years. Similarly, Katulanda *et al.*³⁴ and Mc Donald Posso *et al.*³⁵ found higher risk in individuals aged ≥ 70 years and ≥ 60 years, respectively. This association could be explained by the development of atherosclerosis with age³⁶. Family history of HTN was also a significant risk factor in our study. Other studies around the world, such as the one published by Ranasinghe *et al.*³⁷, have reported a higher risk in adult individuals with family history of HTN, similarly to Awoke *et al.*³³; these findings support the relevance of the genetic component in the pathogenesis of HTN⁷.

Among the modifiable risk factors assessed, both hypertriglyceridemia and dysglycemic states showed a close link with HTN, in harmony with previous reports³⁸. Nevertheless, it should

be noted that both of these factors lost significance in the multi-variate models after adjusting for hs-CRP, underlining the dependent relationship between low-grade inflammation and HTN in regards to alterations of carbohydrate and lipid metabolism. This link was ascertained notwithstanding adjustment for IR, the hallmark etiopathogenic mechanism found in the metabolic syndrome³⁹.

Concerning obesity, BMI-classified obese and overweight individuals, as well as those with increased waist circumference, showed a higher risk for HTN even after adjustment for hs-CRP. Similarly, Guwatudde *et al.*⁴⁰ studied 3906 individuals in sub-Saharan Africa and reported BMI as the only modifiable risk factor for HTN with a higher risk for overweight and obese individuals. Indeed, the predictive value of waist circumference as a risk factor for HTN tends to vary in a population-specific fashion. In a Croatian cohort of 9070 subjects, Ivičević Uhernik *et al.*⁴¹ ascertained greater HTN risk in individuals with waist circumference ≥ 94 cm for males, and ≥ 88 cm for females. These results coincide with previous investigations that show the propensity of HTN to present alongside other cardiometabolic

Table 1. General characteristics of the population according to JNC-7 classification.
Maracaibo, Venezuela. 2016.

	Normotensive		Prehypertension		Hypertension		χ^2 (p)
	n	%	n	%	n	%	
Sex							31.39 (<0.0001)
Female	496	42.3	328	28.0	348	29.7	
Male	327	30.9	365	34.5	366	34.6	
Age Group (years)							438.82 (<0.0001)
<30	439	57.7	244	32.1	78	10.2	
30–59	365	29.7	407	33.2	455	37.1	
≥60	19	7.9	42	17.4	181	74.8	
Ethnic Groups							3.33 (0.91)
Mixed Race	630	37.2	520	30.7	542	32.0	
Hispanic White	126	35.8	112	31.8	114	32.4	
Afro-Venezuelans	22	33.3	20	30.3	24	36.4	
Indigenous Americans	38	35.8	38	35.8	30	28.3	
Others	7	50.0	3	21.4	4	28.6	
Socioeconomic Status							18.73 (0.02)
Class I: High	11	30.6	15	41.7	10	27.8	
Class II: Middle-High	174	42.1	124	30.0	115	27.8	
Class III: Middle	317	36.1	296	33.7	265	30.2	
Class IV: Working	286	35.8	222	27.8	290	36.3	
Class V: Poverty	35	33.3	36	34.3	34	32.4	
Family History of HTN							17.98 (<0.001)
No	519	35.5	432	29.5	513	35.0	
Yes	304	39.7	261	34.1	201	26.2	
Alcohol Intake[‡]							9.43 (0.009)
No	605	38.8	460	29.5	493	31.6	
Yes	218	32.4	233	34.7	221	32.9	
Smoking Habit							27.83 (<0.001)
No	615	39.8	462	29.9	467	30.2	
Current Smoker	114	34.4	118	35.6	99	29.9	
Past Smoker	93	26.9	110	31.8	143	41.3	
Physical Activity (Leisure)							35.60 (<0.001)
Inactive	470	34.7	418	30.8	467	34.5	
Very Low	54	32.3	53	31.7	60	35.9	
Low	66	36.7	57	31.7	57	31.7	
Moderate	69	39.7	51	29.3	54	31.0	
High	69	41.3	50	29.9	48	28.7	
Very High	95	50.8	64	34.2	28	15.0	
Physical Activity (Home)							39.53 (<0.001)
Inactive	173	28.6	207	34.3	224	37.1	
Very Low	132	42.6	93	30.0	85	27.4	
Low	140	42.8	86	26.3	101	30.9	
Moderate	132	39.3	91	27.1	113	33.6	
High	134	41.6	107	33.2	81	25.2	
Very High	112	33.8	109	32.9	110	33.2	

HTN= Hypertension

[‡] Drinker defined as intake ≥1 gram of alcohol per day.

Table 2. Clinical-metabolic characteristics of the population according to JNC-7 classification.
Maracaibo, Venezuela. 2016.

	Normotensive		Prehypertension		Hypertension		χ^2 (p)
	n	%	n	%	n	%	
BMI Classification (kg/m²)							264.36 (<0.0001)
<25	397	57.1	194	27.9	104	15.0	
25–29.9	274	34.9	265	33.7	247	31.4	
≥30	152	20.3	234	31.2	363	48.5	
Glycemic Status							178.22 (<0.001)
Normoglycemia	677	42.2	528	32.9	401	25.0	
Impaired Fasting Glucose	122	28.0	127	29.2	186	42.8	
DM2	22	11.8	38	20.3	127	67.9	
HDL-C							15.88 (0.001)
Normal	386	41.0	293	31.1	263	27.9	
Low	437	33.9	400	31.1	451	35.0	
Hypertriacylglyceridemia							112.34 (<0.001)
Absent	681	42.2	514	31.8	419	26.0	
Present	142	23.1	179	29.1	295	47.9	
Elevated Waist Circumference							256.28 (<0.001)
Absent	539	51.8	326	31.3	176	16.9	
Present	284	23.9	367	30.9	538	45.2	
Insulin Resistance							62.09 (<0.001)
Absent	460	42.5	351	32.4	272	25.1	
Present	285	30.2	271	28.7	387	41.0	
hs-CRP (mg/L)							30.45 (<0.001)
<0.765	438	41.1	324	30.4	304	28.5	
≥0.765	98	27.5	105	29.5	153	43.0	

BMI= Body Mass Index; DM2= Type 2 Diabete Mellitus; hs-CRP=high sensitivity C-Reactive Protein

disorders^{42,43}. In this regard, IR has been suggested to play a paramount role as the common denominator connecting the pathophysiology of these conditions⁴⁴, promoting vascular alterations independently of the impact of concurrent low-grade inflammation, including increased expression of vascular cell adherence molecules, oxidative stress, and a decrease in nitric oxide availability⁴⁵. On the other hand, an increase in adiposity has also been associated with higher angiotensin II and aldosterone levels^{46,47}, postulating obesity as a state of inappropriate activation of the renin angiotensin aldosterone system; thus, participating in conjunction with IR in the pathogenesis of HTN and other associated cardiometabolic diseases⁴⁶.

The link between immunity, inflammation, and the development of HTN has been a subject of intense research in recent decades¹². Increased levels of inflammatory biomarkers, such as hs-CRP, have been independently associated with higher cardiovascular risk⁴⁸. In this study, after adjusting for hs-CRP levels

and several other variables, indigenous populations were shown to be at a higher risk for HTN in contrast with the data analyzed by NHANES 2011–2012⁴⁹. Afro-Venezuelan individuals had the highest prevalence for HTN when compared to non-Hispanic white individuals, Hispanic, and Asian groups. This may be explained by the fact that this ethnic group often suffers from lower socioeconomic status in addition to chronic stress, worse healthcare, or discrimination⁵⁰.

IR also behaved as a risk factor after adjusting for hs-CRP, comparable to results of El Bcheraoui *et al.*⁵¹, reported after studying 10,735 individuals aged ≥15 years where subjects with DM2 had a higher risk for HTN. The risk in prediabetes, however, was not significant. Similarly, a study by Sung *et al.*⁵² in 8347 individuals showed that increased hs-CRP levels were an independent risk factor for the development of HTN, analogous to our findings. CRP is involved in the innate immune response with diverse functions that include stimulating monocytes for the

Table 3. Risk factors for hypertension in adult individuals from Maracaibo, Venezuela. 2016.

	Model 1*				Model 2**	
	Crude Odds Ratio (95% CI ^a)	p ^b	Adjusted Odds Ratio (95% CI ^a)	p ^b	Adjusted Odds Ratio (95% CI ^a)	p ^b
Sex						
Female	1.00	-	1.00	-	1.00	-
Male	1.60 (1.30 - 1.95)	0.65	2.16 (1.53 - 3.05)	< 0.01	1.81 (1.18 - 2.79)	< 0.01
Age Group (years)						
<30	1.00	-	1.00	-	1.00	-
30–59	7.02 (5.32 - 9.26)	< 0.01	4.57 (3.12 - 6.67)	< 0.01	5.59 (3.46 - 9.05)	< 0.01
≥60	53.62 (31.54 - 91.13)	< 0.01	33.79 (17.15 - 66.61)	< 0.01	40.99 (16.94 - 99.19)	< 0.01
Family History of HTN						
No	1.00	-	1.00	-	1.00	-
Yes	1.49 (1.21 - 1.86)	< 0.01	2.30 (1.65 - 3.22)	< 0.01	2.70 (1.77 - 4.13)	< 0.01
Ethnic Groups						
Mixed Race	1.00	-	1.00	-	1.00	-
Hispanic White	1.05 (0.79 - 1.39)	0.72	1.02 (0.67 - 1.56)	0.92	1.32 (0.78 - 2.23)	0.29
Afro-Venezuelans	1.27 (0.70 - 2.29)	0.43	1.24 (0.53 - 2.87)	0.62	1.32 (0.45 - 3.90)	0.62
Indigenous Americans	0.92 (0.56 - 1.50)	0.73	1.64 (0.70 - 3.85)	0.25	3.06 (1.09 - 8.62)	0.03
Others	0.66 (0.19 - 2.28)	0.52	0.58 (0.08 - 4.38)	0.59	0.91 (0.04 - 18.52)	0.95
HDL-C						
Normal	1.00	-	1.00	-	1.00	-
Low	1.52 (1.23 - 1.86)	< 0.01	1.03 (0.75 - 1.40)	0.87	1.01 (0.69 - 1.49)	0.95
Hypertriacylglyceridemia^c						
Absent	1.00	-	1.00	-	1.00	-
Present	3.38 (2.67 - 4.27)	< 0.01	1.74 (1.23 - 2.46)	< 0.01	1.25 (0.80 - 1.94)	0.33
Glycemic Status						
Normoglycemia	1.00	-	1.00	-	1.00	-
Impaired Fasting Glucose	2.57 (1.99 - 3.34)	< 0.01	1.55 (1.08 - 2.21)	0.02	1.15 (0.72 - 1.84)	0.56
DM2	9.75 (6.09 - 15.58)	< 0.01	2.92 (1.33 - 6.41)	< 0.01	2.24 (0.87 - 5.73)	0.09
Elevated Waist Circumference						
Absent	1.00	-	1.00	-	1.00	-
Present	6.02 (4.82 - 7.51)	< 0.01	2.23 (1.33 - 3.09)	< 0.01	2.21 (1.29 - 3.78)	< 0.01
BMI Classification (kg/m²)						
<25	1.00	-	1.00	-	1.00	-
25–29.9	3.44 (2.61 - 4.54)	< 0.01	1.56 (1.02 - 2.37)	0.04	1.69 (0.99 - 2.90)	0.05
≥30	9.12 (6.84 - 12.15)	< 0.01	2.36 (1.37 - 4.06)	< 0.01	2.32 (1.17 - 4.60)	0.02
Insulin Resistance						
Absent	1.00	-	1.00	-	1.00	-
Present	2.30 (1.85 - 2.85)	< 0.01	1.17 (0.85 - 1.61)	0.35	1.50 (1.00 - 2.25)	0.05
hs-CRP (mg/L)						
<0.765	1.00	-	-	-	1.00	-
≥0.765	2.25 (1.68 - 3.01)	< 0.01	-	-	1.75 (1.13 - 2.73)	0.01

a Confidence Interval (95%); b Significance level; c Triglycerides ≥150 mg/dL; d HOMA2-IR≥2

* **Model 1:** Adjusted for sex, age groups, ethnic groups, socioeconomic status, family history of hypertension, smoking habit, alcohol intake, home physical activity per the IPAQ-LF, leisure physical activity per the IPAQ-LF, low HDL-C, hypertriacylglyceridemia, glycemic status, BMI categories, insulin resistance, elevated waist circumference (≥95cm for males; ≥90cm for females).

** **Model 2:** Adjusted for sex, age groups, ethnic groups, socioeconomic status, family history of hypertension, smoking habit, alcohol intake, home physical activity per the IPAQ-LF, leisure physical activity per the IPAQ-LF, low HDL-C, hypertriacylglyceridemia, glycemic status, BMI categories, insulin resistance, elevated waist circumference (≥95cm for males; ≥90cm for females); AND elevated hs-CRP levels.

Table 4. Risk factors for prehypertension in adult individuals from Maracaibo, Venezuela. 2016.

	Crude Odds Ratio (95% CI ^a)	<i>p</i> ^b	Adjusted Odds Ratio (95% CI ^a)	<i>p</i> ^b
Age Group (years)				
<30	1.00	-	1.00	-
30–59	7.02 (5.32 - 9.26)	< 0.01	1.85 (1.29 - 2.65)	< 0.01
≥60	53.62 (31.54 - 91.13)	< 0.01	3.39 (1.41 - 8.18)	< 0.01
Alcohol Intake				
<1 g/day	1.00	-	1.00	-
≥1 g/day	1.49 (1.21 - 1.86)	< 0.01	1.49 (1.06 - 2.10)	0.02
HDL-C				
Normal	1.00	-	1.00	-
Low	1.52 (1.23 - 1.86)	< 0.01	1.46 (1.06 - 2.00)	0.02
Physical Activity (Leisure)				
Inactive	1.00	-	1.00	-
Very Low	3.38 (2.67 - 4.27)	< 0.01	0.72 (0.43 - 1.21)	0.22
Low	3.38 (2.67 - 4.27)	< 0.01	0.70 (0.42 - 1.16)	0.17
Moderate	3.38 (2.67 - 4.27)	< 0.01	0.62 (0.38 - 1.01)	0.05
High	3.38 (2.67 - 4.27)	< 0.01	0.65 (0.39 - 1.07)	0.08
Very High	3.38 (2.67 - 4.27)	< 0.01	0.97 (0.59 - 1.57)	0.99

a Confidence Interval (95%); b Significance level; c Triglycerides ≥150 mg/dL; d HOMA2-IR≥2

Model adjusted for sex, age groups, ethnic groups, socioeconomic status, family history of hypertension, smoking habit, alcohol intake, home physical activity per the IPAQ-LF, leisure physical activity per the IPAQ-LF, low HDL-C, hypertriglyceridemia, glycemic status, BMI categories, insulin resistance, elevated waist circumference (≥95cm for males; ≥90cm for females); AND elevated hs-CRP levels.

release of pro-inflammatory cytokines, such as interleukin(IL)-6, IL-1 β and tumor necrosis factor alpha, as well as the expression of intercellular adhesion molecules (ICAM-1) and vascular cell adherence molecules⁵³. These effects underlie the link between inflammation and the development of HTN.

Age, alcohol consumption and low HDL-C were identified as risk factors for PHT in this study, echoing the findings by Wu *et al.*⁵⁴ where individuals with PHT were older and had low HDL-C. Furthermore, Singh *et al.*⁵⁵ reported a higher risk for PHT with older age in both males and females, while the risk associated with alcohol consumption was only significant for men. On the other hand, PA at home was determined to be a protective factor for PHT according to our model, in contrast to the higher risk reported for sedentary individuals⁵⁶. Poor lifestyle habits have been previously highlighted as the main determinants of PHT¹⁹, possibly able to modify early pathophysiologic alterations found in these subjects, such as high levels of IL-17⁵⁷.

Our findings reflect the importance of focusing efforts towards decreasing the presence of various risk factors in the adult population of Maracaibo City, particularly considering the high prevalence of PHT and HTN and the differences in risk profiles

for each state. The key modulating role of low-grade inflammation was also prominent in our study.

The limitations of this study include its cross-sectional design, which prevents the assessment of causality. However, the data analysis is comparable to current literature and provides important information about non-communicable diseases in Latin American communities - allowing for the design and implementation of primary care programs to detect individuals at risk of PHT and HTN in this context.

Data availability

Dataset 1: Raw data for the 2231 patient sample used in this study. Doi, [10.5256/f1000research.12531.d182355](https://doi.org/10.5256/f1000research.12531.d182355)⁵⁸

Competing interests

No competing interests were disclosed.

Grant information

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Harry HX Wang

School of Public Health, Sun Yat-sen University, Guangzhou, China

This was a cross-sectional study that explored factors associated with hypertension (HT) and pre-hypertension (Pre-HT), respectively, among a sample of population in a single city in Venezuela. The merits of this paper included a very detailed description of the sampling methodology and a clear demonstration of main results.

However, factors that contribute to increasing level of high blood pressure (BP) have already been well reported in many existing literature, and thus the novelty of the research question in the current study seems to be limited.

As the study identified different sets of factors associated with HT and Pre-HT in the two separate logistic regression models, I would suggest conducting a sensitivity analysis using the BP values as dependent variable in a multiple linear regression model. This could provide further evidence to illustrate a picture showing how variables currently identified for both HT and Pre-HT are linked with BP values.

The seventh paragraph in the Discussion section (page 9 of 13) argued that household physical activity was found as a protective factor for pre-HT. However in Table 4, the variable was leisure time physical activity ($p=0.05$). On this particular variable, the authors can add more texts to describe what is defined as a "moderate physical activity" so that people who are at risk for higher BP can be benefited in adjusting their physical activity level from the findings of this study.

The title of the paper highlighted dysglycemic states and HT; however, this was not closely reflected by the main results of the study.

Is the work clearly and accurately presented and does it cite the current literature?

Yes

Is the study design appropriate and is the work technically sound?

Yes

Are sufficient details of methods and analysis provided to allow replication by others?

Yes

If applicable, is the statistical analysis and its interpretation appropriate?

Partly

Are all the source data underlying the results available to ensure full reproducibility?

Yes

Are the conclusions drawn adequately supported by the results?

Yes

Competing Interests: No competing interests were disclosed.

Reviewer Expertise: Primary health care, cardiovascular diseases

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

Reviewer Report 05 February 2018

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Luis M. Ruilope

Institute of Investigation, Hospital 12 de Octubre, Madrid, Spain

This paper contains data clearly presented showing the relevance of inflammation in dysglycemic states. The group investigated is adequate in size and selection of the population in the city of Maracaibo. It is interesting the demonstration of different factors promoting prehypertension and sustained arterial hypertension and how inflammation acts when blood pressure is above 140/90 mmHg. The article in its present format is scientifically sound albeit the only caveat is that it refers only to the population of a single city.

Is the work clearly and accurately presented and does it cite the current literature?

Yes

Is the study design appropriate and is the work technically sound?

Yes

Are sufficient details of methods and analysis provided to allow replication by others?

Yes

If applicable, is the statistical analysis and its interpretation appropriate?

Yes

Are all the source data underlying the results available to ensure full reproducibility?

Yes

Are the conclusions drawn adequately supported by the results?

Yes

Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

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