

# Prevalence and Risk Factors associated with Impaired Fasting Glucose in Adults from Maracaibo City, Venezuela

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## Abstract

**Objective:** The purpose of this study was to evaluate the prevalence and risk factors associated with impaired fasting glucose (IFG) in adult individuals from Maracaibo city, Venezuela.

**Materials and methods:** 2230 patients from the Maracaibo Metabolic Syndrome Prevalence Study were selected. IFG was defined according to the 2016 ADA criteria. A multiple logistic regression model was constructed in order to assess risk factors associated with IFG.

**Results:** In the general population, the prevalence of IFG was 19.5% (n=435), with 46.4% (n=202) being women and 53.6% (n=233) being men, p=0.004. The main risk factors associated with IFG were age (≥60 years: OR=2.31; CI 95%=1.23-4.35; p<0.01), alcohol consumption, abdominal obesity and insulin resistance. After evaluating individuals with IFG exclusively, the major risk factor was the presence of elevated high-sensitivity C-Reactive Protein levels (OR=2.03; CI 95%=1.13-3.67; p<0.02).

**Conclusions:** In Maracaibo the prevalence of IFG is similar to that of international reports. It is associated with a variety of risk factors, especially abdominal obesity, insulin resistance and low-grade inflammation, demonstrating the close link between adiposopathy and alterations in glucose metabolism.

**Keywords:** Impaired fasting glucose; Insulin resistance; Obesity; Physical activity; Hypertension; Inflammation; Diabetes

## Introduction

Type 2 Diabetes Mellitus (T2D) is responsible for approximately 4.9 deaths a year worldwide, with a projected 59% increase in these estimates by year 2035 [1]. In 2003, the American Diabetes Association (ADA) recognized an intermediate metabolic stage in which blood glucose levels are higher than normal range but do not reach the diagnostic criteria for T2D, leading to the birth of the concepts of impaired fasting glucose (IFG) and impaired glucose tolerance (IGT) [2]; often grouped together and termed "Pre-Diabetes" [3]. Recently, the prevalence of this metabolic alteration in American adults was found to be 37% in the general population, and 51% in subjects older than 65 years of age [4]. In subjects classified as pre-diabetics, IFG appears to be twice as frequent as IGT [5], representing an important risk factor for T2D [6].

The pathophysiology of IFG has been described to involve hepatic insulin resistance and a deficient early phase insulin secretion [7]. Therefore, IFG is often associated with overweight and obesity, both common in populations with deleterious habits and risk factors such as physical inactivity, high-calorie diets, excessive alcohol consumption and smoking [8]; all of which contribute to progressive dysfunction of pancreatic beta cells, a mechanism shared with T2D, but present in a less severe form in IFG [9].

Identification of patients with IFG is clinically relevant, considering that changes in lifestyle and potential pharmacological intervention could reduce the risk of advancing into T2D and favor regression to euglycemia [10,11]. In view of the scarcity of reports on the epidemiological behavior of this metabolic disorder in our locality, the purpose of this study was to evaluate the prevalence and risk factors associated with impaired fasting glucose (IFG) in adult individuals from the Maracaibo city, Venezuela.

## Materials and Methods

### Subjects selection

The sample method has been already published in the Maracaibo City Metabolic Syndrome Prevalence Study cross-sectional proposal [12], yet the main aspects will be mentioned. This was a cross-sectional, descriptive, randomized, multistage study which enrolled a total of 2,230 subjects. The study was approved by the Bioethics Committee of the Endocrine and Metabolic Diseases Research Center – University of Zulia, and all participants signed a written consent before being interrogated and physically examined by a trained team.

### Subjects evaluation

Data were collected through completion of a full clinical record carried out by trained personnel, who encompassed interrogation on personal and family history of endocrine and cardiovascular disease, and determination of socioeconomic status by the Graffar scale modified by Méndez-Castellano [13].

Based on information obtained during the clinical interview, subjects were categorized by their smoking habits as follows [14]: a)

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Received April 25, 2016; Accepted June 29, 2016; Published June 30, 2016

**Citation:** Bermúdez V, Salazar J, González R, Ortega A, Calvo M, et al. (2016) Prevalence and Risk Factors associated with Impaired Fasting Glucose in Adults from Maracaibo City, Venezuela. J Diabetes Metab 7: 683. doi:10.4172/2155-6156.1000683

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Non-Smokers, individuals who had never smoked, or had smoked <100 cigarettes in their lifetime; b) Current Smokers, subjects who had smoked  $\geq 100$  cigarettes in their lifetime, or reported current habitual smoking at the time of evaluation, or had quit smoking less than one year prior to our assessment; and c) Past Smokers, individuals who had consumed  $\geq 100$  cigarettes in their lifetime and quit over one year prior to our questioning. Regarding alcohol intake, drinkers were defined as subjects who consumed  $\geq 1$  gram of alcohol daily [15].

Physical activity (PA) was assessed with the International Physical Activity Questionnaire (IPAQ) [16]. For statistical analysis, PA was evaluated in 4 domains: Occupational, Household, Transport and Leisure. In each of these domains, subjects were categorized as: a) Inactive, MET/week=0; or b) Active, MET/week>0. The latter were then subcategorized by gender-specific MET/week quintiles in each domain.

### Clinical evaluation

Blood pressure (BP) was taken with subjects sitting down with their feet on the floor following 15 minutes of rest, determined through the auscultatory method with a calibrated mercury sphygmomanometer; identifying Korotkoff's phases I and V as systolic and diastolic BP respectively. BP was determined 3 times, with 15 minutes in between each take, on two different days; results were classified by the Eighth Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-8) guidelines [17].

An electrical bioelectric scale was used to obtain weight (Tanita, TBF-310 GS Body Composition Analyzer, Tokyo – Japan). Height was measured using a calibrated metric measurement tape, with the subject standing up barefoot. Body Mass Index (BMI) was calculated with the formula: [weight/height<sup>2</sup>] expressing results as kg/m<sup>2</sup>. According to their BMI, subjects were sorted in 3 categories: a) BMI  $\leq 24.9$ ; b) 25-29.9; and c)  $\geq 30$  [18]. Waist circumference (WC) was evaluated with calibrated measuring tapes in accordance to the anatomical landmarks proposed by the USA National Institutes of Health protocol [19].

### Laboratory analysis

Overnight fasting determination of glucose, total cholesterol, triacylglycerides (TAG), and HDL-C was done 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. LDL-C and VLDL-C levels were calculated applying Friedewald's formula [20] but when TAG levels were <400 mg/dL. LDL-C concentrations were directly measured through lipoprotein electrophoresis and densitometry with a BioRad GS-800 optical densitometer. Insulin was quantified using ultrasensitive ELISA double-sandwich methodology (DRG Instruments GmbH, Germany, Inc.). Serum high-sensitivity C-Reactive Protein (hs-CRP) was quantified through immunoturbidimetric assays (Human Gesellschaft für Biochemica und Diagnostica mbH, Germany).

### Definitions

Subjects having the following characteristics were operatively classified as having T2D: a) Those with a previously established diagnosis of T2D; and b) Those without personal history of T2D but with fasting glucose  $\geq 126$  mg/dL (2). On the other hand, non-diabetic subjects were classified as follows: a) normoglycemic (NG), individuals with fasting glucose <100 mg/dL; and b) Impaired Fasting Glucose (IFG) those fasting glycemia 100-126 mg/dL [21].

As per the IDF/NHLBI/AHA/WHF/IAS/IASO-2009 consensus

criteria [22], abdominal obesity was defined as waist circumference  $\geq 80$  cm in females or  $\geq 90$  cm in males. Likewise, we use local waist circumference cutoff points  $\geq 90$  cm (females) and  $\geq 95$  cm (males) in abdominal obesity definition [23].

Regarding a lipid definition, hypertriglyceridemia was defined as fasting TAG  $\geq 150$  mg/dL; and low HDL-C as fasting HDL-C <50 mg/dL in females or <40 mg/dL in males. HOMA2-IR was utilized for the evaluation of insulin resistance (IR) as proposed by Levy et al. computed with the HOMA-Calculator v2.2.2 software application, IR was defined as HOMA2-IR  $\geq 2$  [24] and elevated hs-CRP was defined as levels  $\geq 0.765$  mg/L [25].

### Statistical Analysis

Qualitative variables were expressed as absolute and relative frequencies, evaluating association through Pearson's Chi-squared ( $\chi^2$ ) test. Quantitative variables were evaluated for distribution normality with Geary's test and were expressed as arithmetic means  $\pm$  SD. Variables with non-normal distribution underwent logarithmic transformation; when normalization could not be achieved (TAG, HDL-C hs-CRP), these variables were expressed as medians (25th percentile-75th percentile). One-Way ANOVA or Kruskal-Wallis tests were applied to evaluate differences between means or medians, respectively.

A multiple logistic regression model was constructed in order to estimate odds ratios (Confidence Interval 95%) for the presence of IFG, adjusted by gender, age groups, ethnic groups, socioeconomic status, educational status, marital status, occupational status, smoking habits, leisure-domain PA, presence of high TAG, presence of low HDL-C, JNC-8 classification, presence of elevated WC, BMI classification, presence of elevated hs-CRP and presence of IR. A second model was constructed, introducing adjustment for presence of elevated hs-CRP, and local abdominal obesity definition to waist circumference (Females:  $\geq 90$  cm; Males:  $\geq 95$  cm).

In addition, an ordinal logistic regression model was constructed, with fasting glucose tertiles among subjects with IFG set as the dependent variable. The independent variables included gender, age groups, ethnic groups, abdominal obesity, hypertriglyceridemia, low HDL-C, BMI classification, JNC-8 classification, TAG/HDL-C ratio tertiles, elevated hs-CRP and presence of IR. Regression coefficients ( $\beta$ ) were calculated with their corresponding confidence intervals (95% CI) along with Odds Ratios ( $e^\beta$ ) and their 95% CI. Goodness-of-fit parameters were calculated and parallel line testing was performed. Data were analyzed with the Statistical Package for the Social Sciences (SPSS) v.21 for Windows (IBM Inc. Chicago, IL), and de results were considered statistically significant when  $p < 0.05$ .

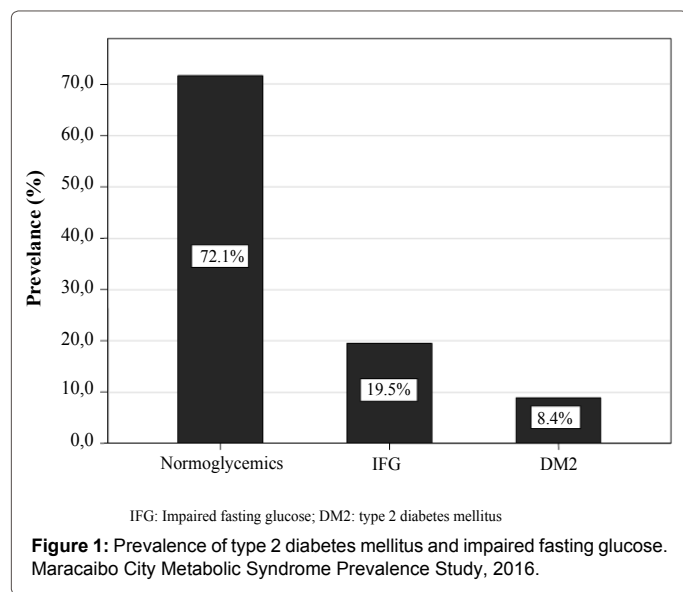
## Results

### Characteristics of the general population

The sample had 2,230 individuals, 52.6% were women (n=1172) with a mean age of  $39.3 \pm 15.4$ . The prevalence of DM2 was 8.4% (n=187) and the prevalence of IFG was 19.5% (n=435) (Figure 1).

### Sociodemographic and psychobiological characteristics according to IFG

Table 1 shows the prevalence of IFG according to sociodemographic and psychobiological habits of the population. Age groups showed the greatest degree of association, displaying a rising prevalence of IFG, from 12.1% (n=91) in individuals aged <30 and years, to 31% in those



aged  $\geq 60$  years ( $n=54$ ,  $\chi^2=63.47$ ;  $p<0.001$ ). In contrast, regarding leisure-time PA, prevalence of IFG was highest in subjects classified as inactive (22.1%;  $n=270$ ), and lowest in subjects in Q5 (11%;  $n=20$ ),  $\chi^2=15.80$ ;  $p=0.007$ .

### Clinical characteristics according to IFG

Among all clinical features studied (Table 2), IR appeared to be the most closely related to IFG, with these disorders coexisting in 31.4% of subjects ( $n=252$ ) ( $\chi^2=75.44$ ;  $p<0.0001$ ). BP classification by the JNC-8 was also tightly linked to IFG, with a 15.7% prevalence in normotensive subjects vs 32.9% in hypertensives;  $\chi^2=50.01$ ;  $p<0.0001$ .

### Risk factors for IFG

Evaluation of the correlation between risk factors in a multivariate context revealed the presence of IR to be the most important risk factor for IFG in our population (OR=2.51; 95%CI=1.79-3.52;  $p<0.01$ ), followed by age groups ( $\geq 60$  years: OR=2.31; 95%CI=1.23-4.35;  $p<0.01$ ) (Table 3).

### Clinical and biochemical characteristics of subjects with IFG

After assessing the behavior of various clinical and biochemical variables according to fasting glucose tertiles among subjects with IFG, a progressive worsening across tertiles was observed in most variables (Table 4). Although many variables showed a significant association with these fasting glucose tertiles, the presence of elevated hs-CRP was the main risk factor identified in the multivariate analysis (OR=2.03; 95%CI=1.13 to 3.67;  $p<0.02$ ).

### Discussion

IFG is an intermediate disglycemic stage that precedes T2D [5], which has displayed an alarming rise in prevalence in recent years [26,27]. Because of the possible disease-modifying potential of early therapeutic intervention in these subjects, it is of vital importance to evaluate the epidemiological behavior of this metabolic alteration and determine the main risk factors associated with it in our population.

In Latin America, the CARMELA study reported a prevalence of IFG of only 2%, with Mexico and Bogota boasting the highest figures. Meanwhile, the city of Barquisimeto in Venezuela reported only 1%

		Normoglycemics		Impaired Fasting Glucose		$\chi^2$ (p)
		n	%	n	%	
Gender	Females	870	81.2	202	18.8	8.21 (0.004)
	Males	736	76.0	233	24.0	
Age groups (years)	<30	664	87.9	91	12.1	63.47 (<0.001)
	30-59	822	73.9	290	26.1	
	$\geq 60$	120	69.0	54	31.0	
Ethnic groups	Mixed race	1224	78.7	332	21.3	6.09 (0.19)
	Hispanic White	248	77.3	73	22.7	
	Afro-Venezuelan	42	72.4	16	27.6	
	Amerindian	81	86.2	13	13.8	
	Others	11	91.7	1	8.3	
Socioeconomic status	Classes I y II	319	75.8	102	24.2	4.71 (0.09)
	Class III	660	80.9	156	19.1	
	classes IV y V	627	78.0	177	22.0	
Occupational Status	Un- Employed	799	76.3	248	23.7	3.12 (0.08)
	Employed	668	79.7	170	20.3	
Family history	No	859	79.2	226	20.8	3.95 (0.27)
	T2D	702	78.5	192	21.5	
	Type 1 Diabetes Mellitus	25	80.6	6	19.4	
	Both of them	20	64.5	11	35.5	
Smoking Habits	Non-Smokers	1143	79.6	293	20.4	2.82 (0.24)
	Current Smokers	226	75.6	73	24.4	
	Past Smokers	230	77.2	68	22.8	
Alcohol Consumption	No	1139	80.9	269	19.1	13.19 (<0.001)
	Yes	467	73.8	166	26.2	
Physical activity (Work)	Inactive	1187	78.3	329	21.7	1.90 (0.86)
	Q1	83	79.8	21	20.2	
	Q2	82	76.6	25	23.4	
	Q3	83	79.8	21	20.2	
	Q4	89	83.2	18	16.8	
	Q5	82	79.6	21	20.4	
Physical activity (Transportation)	Inactive	569	78.6	155	21.4	3.92 (0.56)
	Q1	203	81.5	46	18.5	
	Q2	206	76.6	63	23.4	
	Q3	210	81.4	48	18.6	
	Q4	214	78.1	60	21.9	
	Q5	180	76.3	56	23.7	
Physical activity (Household)	Inactive	402	75.0	134	25.0	11.78 (0.04)
	Q1	236	81.1	55	18.9	
	Q2	252	81.8	56	18.2	
	Q3	257	82.1	56	17.9	
	Q4	220	75.1	73	24.9	
	Q5	239	79.7	61	20.3	
Physical activity (Leisure)	Inactive	949	77.9	270	22.1	15.80 (0.007)
	Q1	113	72.4	43	27.6	
	Q2	127	77.9	36	22.1	
	Q3	130	78.8	35	21.2	
	Q4	126	80.3	31	19.7	
	Q5	161	89.0	20	11.0	
Total <sup>§</sup>		1606	78.7	435	21.3	

<sup>§</sup>Subjects with T2D were excluded from the analysis

**Table 1:** Sociodemographic and psychobiological characteristics associated with the diagnosis of Impaired Fasting Glucose. Maracaibo City Metabolic Syndrome Prevalence Study, 2016.

	Normoglycemics		Impaired Fasting Glucose.		$\chi^2$ (p)
	n	%	n	%	
<b>Waist circumference<sup>†</sup></b>					30.53 (<0.0001)
Normal	470	87.0	70	13.0	
Elevated	1136	75.7	365	24.3	
<b>Waist circumference<sup>‡</sup></b>					48.49 (<0.0001)
Normal	856	85.1	150	14.9	
Elevated	750	72.5	285	27.5	
<b>Triacylglycerides (mg/dL)</b>					47.25 (<0.0001)
<150	1256	82.3	270	17.7	
≥150	350	68.0	165	32.0	
<b>HDL-C</b>					3.43 (0.06)
Normal	711	80.6	171	19.4	
Lower	895	77.2	264	22.8	
<b>BMI</b>					28.78 (<0.001)
Normal weight	566	84.2	106	15.8	
Overweight	579	79.3	151	20.7	
Obesity	461	72.1	178	27.9	
<b>JNC-8 Classification</b>					50.01 (<0.0001)
Normotension	708	84.3	132	15.7	
Prehypertension	613	79.0	163	21.0	
Hypertension	285	67.1	140	32.9	
<b>hs-CRP (mg/L)</b>					4.86 (0.03)
<0.765	817	83.0	167	17.0	
≥0.765	237	77.5	69	22.5	
<b>Insulin Resistance<sup>§</sup></b>					75.44 (<0.0001)
Absent	892	85.4	152	14.6	
Present	550	68.6	252	31.4	
<b>Total<sup>§</sup></b>	<b>1606</b>	<b>78.7</b>	<b>435</b>	<b>21.3</b>	

BMI=Body Mass Index; hs- CRP= High-sensitivity C-reactive protein; JNC-8=Eighth Joint National Committee

<sup>†</sup>Criteria by IDF Men: ≥90 cm; Women: ≥80 cm.

<sup>‡</sup>Criteria according local criteria (Men: ≥95 cm; Women: ≥90 cm).

<sup>§</sup>HOMA2-IR ≥ 2

<sup>§</sup>They are excluded from the analysis of subjects with diabetes mellitus

**Table 2:** Clinical features associated with Impaired Fasting Glucose. Maracaibo City Metabolic Syndrome Prevalence Study, 2016.

	Model 1*				Modelo 2**	
	Crude Odds Ratio (CI 95% <sup>a</sup> )	p <sup>b</sup>	Adjusted Odds Ratio (CI 95% <sup>a</sup> )	p <sup>b</sup>	Adjusted Odds Ratio (CI 95% <sup>a</sup> )	p <sup>b</sup>
<b>Age groups (years)</b>						
<30	1.00	-	1.00	-	1.00	-
30-49	2.57 (1.99 - 3.33)	<0.01	1.99 (1.46 - 2.71)	<0.01	2.09 (1.39 - 3.14)	<0.01
≥60	3.28 (2.23 - 4.84)	<0.01	2.47 (1.54 - 3.96)	<0.01	2.31 (1.23 - 4.35)	<0.01
<b>Alcohol consumption<sup>c</sup></b>						
No	1.00	-	1.00	-	1.00	-
Yes	1.51 (1.21 - 1.88)	<0.01	1.28 (0.98 - 1.67)	0.07	1.49 (1.04 - 2.12)	0.03
<b>Hypertriglyceridemia<sup>d</sup></b>						
Absent	1.00	-	1.00	-	1.00	-
Present	2.19 (1.75 - 2.75)	<0.01	1.42 (1.09 - 1.86)	0.01	1.35 (0.95 - 1.94)	0.09
<b>JNC-8 Classification</b>						
Normotense	1.00	-	1.00	-	1.00	-
Pre-hypertensive	1.43 (1.11 - 1.84)	<0.01	1.05 (0.78 - 1.40)	0.77	0.93 (0.54 - 1.35)	0.68
Hypertensive	2.64 (2.00 - 3.47)	<0.01	1.61 (1.15 - 2.25)	<0.01	1.19 (0.77 - 1.85)	0.44
<b>Elevated Waist circumference</b>						
Absent	-	-	1.00	-	1.00	-
Present	-	-	1.13 (0.79 - 1.59)	0.51	1.62 (1.07 - 2.45)	0.02
<b>Insulin-resistance<sup>e</sup></b>						
Absent	1.00	-	1.00	-	1.00	-
Present	2.69 (2.14 - 3.38)	<0.01	2.33 (1.81 - 2.99)	<0.01	2.51 (1.79 - 3.52)	<0.01

<sup>a</sup> Confidence interval (95%); <sup>b</sup> Significance level; <sup>c</sup> Alcohol consumption > 1 gr/daily; <sup>d</sup> Triglycerides ≥ 150 mg/dL; <sup>e</sup> HOMA2-IR ≥ 2

\*Model 1: Adjustment for gender, age groups, ethnic groups, family history of diabetes mellitus, employment status, smoking habits, alcohol consumption, home-sphere physical activity, leisure-time physical activity, hypertriglyceridemia, low HDL -C, JNC-8 classification, BMI categories, presence of insulin resistance and elevated waist circumference: Men: ≥90 cm; Women: ≥80 cm.

\*\*Model 2: Similar adjustment to model 1 but elevated waist circumference is adjusted (Men: ≥95 cm; Women: ≥90 cm) and elevated hs-CRP (≥0,765 mg / L) is added.

**Table 3:** Logistic regression models of risk factors for Impaired Fasting Glucose. Maracaibo City Metabolic Syndrome Prevalence Study, 2016.

	Tertile 1 (100-102.9 mg/dL) [A]		Tertile 2 (103-107.9 mg/dL) [B]		Tertile 3 (≥ 108 mg/dL) [C]		p*	A vs B	A vs C	B vs C
	Media	DE	Media	DE	Media	DE				
Age (years)	40.6	15.1	44.2	14.9	46.2	14.2	0.003	0.08	0.002	0.41
BMI (Kg/m <sup>2</sup> )	28.5	6.5	29.1	5.6	31.5	7.9	<0.001	0.75	0.001	0.006
Waist Circumference (cm)	95.6	15.1	97.7	13.3	104.0	18.8	<0.001	0.40	<0.001	0.004
Basal glycemia (mg/dL)	100.9	0.8	104.8	1.5	113.8	4.8	<0.001	<0.001	<0.001	<0.001
Insulin (UI/L)	15.8	8.6	18.2	14.3	18.7	10.9	0.100	0.60	0.09	0.43
HOMA2-βcell	133.2	48.2	134.9	65.5	120.2	46.9	0.03	0.91	0.05	0.09
HOMA2-S	53.1	26.9	53.1	47.9	45.3	25.1	0.03	0.51	0.02	0.22
HOMA2-IR	2.36	1.21	2.70	1.89	2.88	1.59	0.03	0.49	0.02	0.24
Total cholesterol (mg/dL)	193.4	42.0	203.9	60.1	208.1	47.7	0.03	0.24	0.02	0.52
Triacylglycerides (mg/dL)	132.8	96.7	143.8	112.8	167.2	109.5	<0.001	0.57	<0.001	0.008
HDL-C (mg/dL)	45.1	11.5	43.1	13.2	41.3	10.5	0.02	0.19	0.02	0.53
VLDL-C (mg/dL)	26.6	19.4	28.8	22.6	33.6	21.9	0.02	0.66	0.02	0.13
LDL-C (mg/dL)	122.8	36.2	130.9	44.2	134.5	39.9	0.09	0.51	0.07	0.51
Cholesterol Non HDL	148.2	41.5	160.8	60.2	166.8	48.0	0.009	0.11	0.007	0.56
Index TAG/HDL <sup>†</sup>	2.4	1.5-4.3	2.9	1.9-4.4	3.5	2.3-5.5	<0.001	-	-	-
Lipoprotein(a) (mg/dL)	28.6	12.9	26.9	15.9	25.4	14.5	0.209	0.62	0.18	0.65
Systolic blood pressure (mmHg)	120.9	17.9	122.8	17.6	125.7	16.7	0.04	0.59	0.03	0.24
Diastolic blood pressure (mmHg)	78.8	12.5	79.7	11.9	80.6	11.8	0.40	0.75	0.37	0.79
hs-CRP (mg/L) <sup>†</sup>	0.397	0.208-0.758	0.383	0.139-0.674	0.607	0.231-1.027	0.05	-	-	-

BMI= Body mass index; hs-CRP= high-sensitivity C-reactive protein.  
\*One-Way ANOVA (after logarithmic transformation) Post-hoc Tukey test.  
<sup>†</sup>Expressed in median (p25-p75). Kruskal-Wallis Test comparison.

**Table 4:** Clinical and biochemical characteristics in subjects with Impaired Fasting Glucose according to fasting glucose tertiles. Maracaibo City Metabolic Syndrome Prevalence Study, 2016.

[28] of IFG prevalence. All of these are lower than estimations in the USA (26%) [4]. In contrast to the *CARMELA* study, the *PERUDIAB* study recently evaluated 1,677 Peruvian individuals aged ≥ 25 years, estimating a general IFG prevalence of 22.4% [29]. Reports on pre-diabetes are scarce in Venezuela, although there is a report from a rural population in Merida State, in the Andean Region, which found 18.6% of the individuals to have IFG [30], a similar rate to the findings in our study (19.5%).

Evaluation of IFG by gender showed a higher prevalence in males as has been described in other populations [31,32], similarly echoing previous reports [27,28,33], prevalence increasing with age in both univariate and multivariate analysis (Table 5). This might be associated with senescent changes, such as increased visceral adiposity [34] decreased lean mass [35] and reduced PA [36] all having a direct influence on IR development. The decrease in PA may play a particularly important role in this process, as PA improves glucose metabolism by favoring its uptake by target organs, depleting muscular glycogen, and inducing favorable changes in lipid metabolism [37,38].

Furthermore, alcohol consumption was associated with a higher risk of having this metabolic alteration, similar to findings reported by Cullmann et al. [39] in 111 pre-diabetic Swedish individuals, where alcohol consumption was recognized as a risk factor in men, mainly in beer drinkers (OR: 1.84, 95% CI: 1.13-3.01, p<0.05). This effect appears to be dependent on the amount and type of alcohol consumed, as for women, for example, the moderate consumption of wine was found to be protective, while the excessive consumption of spirituous beverages was identified as a risk factor (OR: 2.41, 95% CI 1.47-3.96). Additionally, our research group has demonstrated that the effect of alcohol consumption on the components of the metabolic syndrome appears to be dose-dependent in our population, with an approximate

intake of 4-6 beers, 3-5 spirituous beverages or 4-7 cups of wine found to be a risk factor for hyperglycemia among males (OR: 1.99, 95%CI: 1.20-3.33; p<0.01) [40].

Regarding clinical characteristics, in the multivariate context the results showed that only subjects with abdominal obesity had a higher risk of IFG similar to results by Diaz et al. [8] in the *PREDAPS* study, where 1184 Spanish pre-diabetic individuals were evaluated. In this study, abdominal obesity was considered a risk factor in both sexes, indeed obesity is closely related to metabolic disorders of carbohydrates, especially visceral adiposity. In the context of adiposopathy –characterized by increased signaling of pro-inflammatory cytokines such as TNF-α, MCP-1, IL-1β and macrophage infiltration in adipose tissue- [41] TNF-α interrupts the insulin signaling cascade by phosphorylating serine-threonine sidechains in the insulin receptor substrate (IRS) altering its enzymatic activity and preventing GLUT4 translocation in insulin-dependent tissues, being a molecular mechanism of utmost importance in IFG appearance [42]. In addition, TNF-α increases lipolysis and decreases biosynthesis of triacylglycerols via peroxisome proliferator-activated receptor gamma (PPARγ) signaling in adipose tissue, consequently causing an increase in free fatty acids (FFA) release into splanchnic circulation (43). According to Shulman et al. [43] this phenomenon favors FFA storage in ectopic tissues such as the liver and muscle, increasing the intracellular concentration of intermediates of fatty acid metabolism such as acyl-CoA, ceramides and diacylglycerol which are also involved in the phosphorylation of IRS and therefore in inhibiting effects [44], suggesting a close relationship between obesity, the presence of IR and the appearance of IFG.

Hypertension was also found to be a risk factor for IFG in our population, in resemblance to the *PREDAPS* study, where hypertensive subjects had a risk 2.33 times higher risk of IFG [8]. Nevertheless, this

	Tertile 1 (100-102.9 mg/dL)		Tertile 2 (103-107.9 mg/dL)		Tertile 3 (≥ 108 mg/dL)		$\chi^2$ (p)	$\beta^*$ (CI95%); p	OR (CI95%)
	n	%	n	%	n	%			
<b>Waist circumference‡</b>							11.26 (0.004)		
Normal	55	36.7	53	35.3	42	28.0		0	
Elevated	68	23.9	96	33.7	121	42.5		0.12 (-0.88 – 0.65); 0.76	1.13 (0.41 – 1.91)
<b>Triacylglycerides (mg/dL)</b>							9.04 (0.01)		
<150	86	31.9	97	35.9	87	32.2		0	0
≥150	37	22.4	52	31.5	76	46.1		0.18 (-1.09 – 0.73); 0.69	1.19 (0.33 – 2.07)
<b>HDL-C</b>							4.42 (0.11)		
Normal	58	33.9	54	31.6	59	34.5		0	0
Low	65	24.6	95	36.0	104	39.4		-0.13 (-0.48 – 0.74); 0.67	0.87 (0.61 – 2.09)
<b>BMI Classification</b>							11.56 (0.02)		
Normal weight	37	34.9	39	36.8	30	28.3		0	0
Overweight	47	31.1	53	35.1	51	33.8		-0.15 (-0.95 – 0.62); 0.63	0.86 (0.39 – 1.86)
Obesity	39	21.9	57	32.0	82	46.1		0.63 (-0.82 – 0.95); 0.86	1.88 (0.44 – 2.59)
<b>JNC-8 Classification</b>							6.46 (0.17)		
Normotense	43	32.6	47	35.6	42	31.8		0	0
Pre-hypertensive	49	30.1	56	34.4	58	35.6		0.03 (-0.57 – 0.61); 0.96	1.03 (0.56 – 1.84)
Hypertensive	31	22.1	46	32.9	63	45.0		0.50 (-0.22 – 1.17); 0.17	1.64 (0.80 – 3.22)
<b>hs-CRP (mg/L)</b>							12.02 (0.002)		
<0.765	54	32.3	65	38.9	48	28.7		0	0
≥0.765	17	24.6	16	23.2	36	52.2		<b>0.71 (0.12 – 1.30); 0.02</b>	<b>2.03 (1.13 – 3.67)</b>
<b>Insulinresistance<sup>1</sup></b>							2.09 (0.35)		
Absent	44	28.9	58	38.2	50	32.9		0	0
Present	66	26.2	85	33.7	101	40.1		0.39 (-0.19 – 0.96); 0.17	1.48 (0.82 – 2.61)
<b>TAG/HDL ratio</b>							16.59 (<0.01)		
<1.74	39	31.7	34	22.8	22	13.5		0	
1.74-3.36	39	31.7	55	36.9	54	33.1		0.28 (-1.23 – 1.12); 0.93	1.32 (0.29 – 3.07)
>3.36	45	36.6	60	40.3	87	53.4		-0.05 (-0.49 – 1.04); 0.48	0.95 (0.61 – 2.83)
<b>Total§</b>	<b>123</b>	<b>28.3</b>	<b>149</b>	<b>34.3</b>	<b>163</b>	<b>37.4</b>			

BMI = body mass index; JNC-8 = 8th National Joint Committee for Hypertension

<sup>‡</sup>Criteria according to EPSMM (Men: ≥95 cm; Women: ≥90 cm).

<sup>1</sup>HOMA2-IR ≥ 2

<sup>§</sup>Subjects with diabetes mellitus were excluded from the analysis

<sup>\*</sup>Ordinal regression model adjusted for gender, age groups, ethnic groups, leisure time physical activity, abdominal obesity, hypertriglyceridemia, low HDL-C, BMI classification, JNC-8 classification, elevated hs-CRP and presence of insulin resistance.

Model adjustment information: ( $\chi^2 = 34.69$ ;  $p = 0.007$ )

Pseudo R-Squared: Cox and Snell (0.14) - Nagelkerke (0.16) - McFadden (0.07)

Parallel line test ( $\chi^2 = 14.17$ ;  $p = 0.66$ )

**Table 5:** Clinical characteristics in patients with Impaired Fasting Glucose according to fasting glucose tertiles. Maracaibo City Metabolic Syndrome Prevalence Study, 2016.

association was no longer significant after adjusting the model by abdominal circumference and elevated hs-CRP, which might imply that this co-relation between hypertension and alteration of IFG depends on other factors different from IR, such as a certain level of chronic inflammation and oxidative stress, as has been proposed in previous publications [45].

Finally, when evaluating individuals with IFG exclusively, we observed that clinical and metabolic parameters are altered by increasing levels of glycemia. Moreover, when multivariate analysis revealed the presence of elevated hs-CRP to be the factor to most closely related to the highest tertile of fasting glycemia, similar to the findings of Jaiswal et al. [46]. Indeed, prospective studies have described elevated hs-CRP as an independent risk factor for the development of both T2D [47] and pre-diabetes [48], being considered as a chronic inflammation marker closely related to IR, and playing a central role in the development and progression of IFG, metabolic syndrome and T2D [49]. It is also important to note that elevated hs-CRP was a significant risk factor only when analyzing subjects with

IFG. Molecular mechanisms that could explain this unique correlation in this group of subjects may be an upregulation of CRP expression by IR state, counteracting the physiological effects of insulin in early phase synthesis of hepatic proteins [50], existing major acute phase protein synthesis, which might explain why hs-CRP is a better predicting factor only in patients with established metabolic disorders.

Limitations of this study include its cross-sectional design, which does not allow establishment of causality, and the lack of assessment of nutritional habits, which represent an important factor contributing to dysglycemia. Likewise, even though there are many markers for low grade inflammation, hs-CRP was the only one who rendered significant results in the multivariate analysis. However, more research is required in order to properly depict the mechanistic of this association in our population. Finally, the design of the study did not originally include the evaluation of 2 hr oral glucose tolerance test or postprandial measurements. Nevertheless, in the upcoming second phase of the project, we are considering this variable as a new inclusion in our research.

In conclusion, our results highlight the responsibility of clinicians for the early detection of subjects with IFG, especially in populations with high prevalence of risk factors such as obesity, physical inactivity and metabolic syndrome [51-53]. IFG prevalence in our population is similar to worldwide reports, in contrast to the low prevalence previously reported in Latin America in past decades. IFG is also linked to various cardiovascular risk factors in our population such as age, alcohol consumption, abdominal obesity; suggesting these patients as candidates for early therapeutic intervention as recommended by the ADA [54]. Lastly, hs-CRP may be a useful predicting factor for T2D among subjects with IFG.

### Funding

This work was supported by research grant N° CC-0437-10-21-09-10 from the Technological, Humanistic, and Scientific Development Council (Consejo de Desarrollo Científico, Humanístico y Tecnológico; CONDES), University of Zulia, and research grant N°. FZ-0058-2007 from Fundacite-Zulia.

### Disclosure

The authors have no conflicts of interest to disclose.

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**Citation:** Bermúdez V, Salazar J, González R, Ortega A, Calvo M, et al. (2016) Prevalence and Risk Factors associated with Impaired Fasting Glucose in Adults from Maracaibo City, Venezuela. *J Diabetes Metab* 7: 683. doi:[10.4172/2155-6156.1000683](https://doi.org/10.4172/2155-6156.1000683)

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