Associations of Insulin Resistance with Systolic and Diastolic Blood Pressure: A Study from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL)

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Abstract

Insulin resistance is hypothesized to contribute to increases in blood pressure (BP) through both the renin-angiotensin-aldosterone and sympathetic nervous systems. Prior large-scale studies examining this association are confounded by overt diabetes, obesity, and antihypertensive medication use. In a cross-sectional analysis of 10,810 Hispanic Community Health Study/ Study of Latinos (HCHS/SOL) participants without diabetes and not taking anti-hypertensive medications, we examined the associations of insulin resistance, quantified by HOMA-IR, with systolic BP (SBP) and diastolic BP (DBP) overall and stratified by sex and pre-diabetes status in

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unadjusted and adjusted analyses. The total sample included 52% women, mean (SD) age 37.2 (13.4) years, 39% of participants had pre-diabetes, mean (SD) HOMA-IR 2.8 (2.2). Stage 1 or 2 hypertension was present in 26% of participants; mean (SD) SBP 116.8 (15) mmHg and DPB 71.0 (10.4) mmHg. Overall, there was a significant linear association between HOMA-IR and both SBP (β 2.64±0.44) and DBP (β 1.49±0.35). We found a significant interaction with sex and the association between HOMA-IR and SBP; the association was linear in men and nonlinear in women. There was no statistically significant interaction between pre-diabetes status and the associations between HOMA-IR and BP. In conclusion, in a large community-based sample of Hispanic/Latino adults of diverse national backgrounds not taking anti-hypertensive medications and free from diabetes, insulin resistance was positively associated with both SBP and DBP. Future longitudinal studies, with adequate power to examine sex-specific differences, are needed to examine the independent contribution of insulin resistance to the development of hypertension.

Keywords

insulin resistance; pre-diabetes; blood pressure; hypertension; sex; Hispanic/Latino

INTRODUCTION

There is a significant burden of diabetes and hypertension (HTN) in the United States (US).[1] Among US adults, 26 million (9.8%) have diagnosed diabetes, 9.4 million (3.7%) have undiagnosed diabetes and 91.8 million (37.6%) have prediabetes based on NHANES (National Health and Nutrition Examination Survey) 2013-2016 data;[1,2] and 45.6% have HTN using the new blood pressure (BP) thresholds from the 2017 American College of Cardiology/ American Heart Association (ACC/AHA) guidelines.[3, 4] Diabetes and HTN commonly co-exist especially in Hispanic/Latino individuals who are disproportionally affected by diabetes, have higher proportion of untreated diabetes and HTN, and are at increased risk of HTN related mortality compared to Whites.[5-7]

Insulin resistance, and the compensatory hyperinsulinemia that accompany it over time, are posited to not only result in the development of diabetes, but also to contribute to the development of elevated BP and HTN through dysregulation of the renal sodium metabolism,[8, 9] renin-angiotensin-aldosterone[10, 11] and sympathetic nervous systems.[12] Although several prior epidemiologic studies have demonstrated associations between insulin resistance and BP and HTN, their findings have been inconsistent.[13-15] The extent to which insulin resistance is independently associated with increased BP and HTN in Hispanic/Latino adults, a population disproportionately affected by insulin resistance and diabetes mellitus, is limited to very few studies that report conflicting results.[16-18] In the Insulin Resistance Atherosclerosis Study (IRAS), Saad et al. found an association between insulin resistance (lower levels of insulin sensitivity index) and higher systolic BP (SBP) and diastolic BP (DBP) and HTN among nondiabetic Whites and Hispanics;[17] whereas other studies report an association in Whites but not Hispanics.[16, 18] It is hypothesized that some of this variability may be due to small sample sizes, confounding from concomitant antihypertensive medication use, and nulling of the associations after adjustment for obesity.

In this study, our goal was to examine the associations between insulin resistance and BP early in the spectrum of insulin resistance, prior to the development of overt diabetes. To overcome the limitations of prior studies, we examined the associations between insulin resistance, measured using the homeostasis model assessment of insulin resistance (HOMA-IR), and SBP and DBP in adults without diabetes who were not taking antihypertensive medications from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL). In a sensitivity analysis, we repeated all analyses in the subset of participants with a body mass index (BMI) in the normal range (18.5-25 kg/m^2).

METHODS

Data from the HCHS/SOL cohort are publicly available to researchers upon application to NHLBI BIOLINCC (National Heart, Lung, and Blood Institute Biologic Specimen and Data Repository Information Coordinating Center).

Study Sample

We studied participants of the HCHS/SOL, a community-based cohort of adults representing diverse Hispanic/Latino backgrounds (Central American, Cuban, Dominican, Mexican, Puerto Rican, and South American).[19] The sampling methods and design of HCHS/SOL have been detailed previously.[20, 21] In brief, self-identified Hispanic/Latino men and women were recruited between March 2008 to June 2011 from 4 communities in the United States (Bronx, NY; Chicago, IL; Miami, FL; and San Diego, CA) using a multi-stage area probability sample design. At each stage of sampling, oversampling occurred, and sampling weights were generated to reflect the probabilities of selection. The institutional review board at each HCHS/SOL center approved the study protocol, and all participants provided written informed consent.

Of the 16,415 HCHS/SOL participants who attended the baseline examination, we excluded individuals with missing data for BP measurement (N=23), BMI (N=41), heart rate (N=6), diabetes status (N=1), glycosylated hemoglobin A1c (HbA1c) (N=131), and (HOMA-IR) (N=92). We also excluded participants who self-reported coronary heart disease or heart failure (N=703) or had missing data (N=406). Individuals taking antihypertensive medications (N=2,172) or missing information on medications (N=359), and those with diabetes (N=1,671) were also excluded.[22] The final sample size was 10,810 participants (Figure 1).

HCHS/SOL Study Procedures

At the baseline examination, all study participants completed questionnaires regarding demographic, acculturation and social factors, medical history, and dietary patterns. Physical activity was self-reported using the modified Global Physical Activity Questionnaire (GPAQ) to derive physical activity in minutes/day.[23, 24]

Participants were asked to fast and refrain from smoking for a minimum of 8-hours before the HCHS/SOL baseline clinic examination. Venous blood and urine specimens were collected, processed, and frozen on site and sent to the HCHS/SOL central laboratory for analysis. A hexokinase enzymatic method (Roche Diagnostics Corp, Indianapolis,

IN) was used to assay fasting plasma glucose. Hemoglobin A1C was measured in EDTA whole blood using a Tosoh G7 automated high-performance liquid chromatography analyzer (Tosoh Bioscience Inc, San Francisco, CA). Fasting insulin was measured with 2 commercial immunoassays (ELISA, Mercodia AB, Uppsala, Sweden; and sandwich immunoassay on a Roche Elecsys 2010 Analyzer, Roche Diagnostics, Indianapolis, IN).[25] To account for the change in assays, insulin levels collected prior to October 29, 2009 using the Mercodia analyzers were calibrated using the following regression equation: $y = 1.00494x - 1.4504$, where y = adjusted insulin value, x = original insulin value.

BP Measurement

Height, weight, waist and hip circumferences, heart rate and BP were measured by trained study staff using standardized protocols previously described.[26] Prior to BP measurement, the appropriate cuff size was selected based on arm circumference at the midpoint of the upper arm; cuff size was determined as follows: small (17-22ccm), adult (22-32cm), large (32-42cm) and XLarge (42-50cm). BP and heart rate were measured in triplicate while in the seated position following 5 minutes of rest using an automated BP device (Omron model HEM-907 XL; Omron Healthcare Inc, Bannockburn, IL).[27] The means of the three measurements (SBP, DBP, heart rate) were used for all calculations. Normal BP was defined as SBP <120 mmHg and DBP <80 mmHg, elevated BP as SBP 120-129 mmHg and DBP <80 mmHg, untreated stage 1 HTN as SBP 130-139 mmHg and/or DBP 80-89 mmHg, and untreated stage 2 HTN as SBP 140 mmHg and/or DBP 90 mmHg) based on the 2017 ACC/AHA BP guidelines.[4]

Definition of Pre-diabetes and BMI categories

Pre-diabetes were defined according to the American Diabetes Association guideline as the presence of a fasting plasma glucose ranging from 100-125 mg/dL and/or HbA1c value of 5.7-6.4%.[28] Normal blood glucose was a fasting plasma glucose <100 mg/dL and HbA1c <5.7%. Insulin resistance was calculated using the HOMA-IR score [fasting insulin (mU/L) x fasting glucose (mg/dL)/405].[29] Normal BMI was defined as BMI 18.5-25 kg/m^2, overweight as BMI 25.1-29.9 kg/m^2, and obese as BMI 30 kg/m^2. The presence of metabolic syndrome was defined as having any three or more of the following criteria: *1)* waist circumference 102 cm in men and 88 cm in women; *2)* triglyceride level 150 mg/dL or use of triglyceride lowering medication; *3)* HDL-C level <40 mg/dL in men and <50 mg/dL in women; *4)* blood pressure 130 mmHg systolic or 85 mmHg diastolic; and *5)* fasting glucose level 100 mg/dL.[30]

Statistical Analyses

The results of all analyses (including model-based means, percentages, and p-values) are weighted using sampling weights to adjust for unequal sampling probability and nonresponse and accounted for the complex survey design in HCHS/SOL.[20, 21] Continuous variables are summarized as means [standard deviation (SD)] for and categorical variables as percentage (%). All variables were assessed for normality. HOMA-IR was found to be skewed, and all subsequent analyses were performed using log-base transformed values.

In linear regression analyses, we examined the age- and sex-adjusted univariate associations of SBP and DBP with demographic and clinical characteristics. We examined the associations of insulin resistance, quantified as log(HOMA-IR), with SBP and DBP using multivariable adjusted restricted cubic spline models with 3 knots (placed at the 10th, 50th, and 90th percentile).[31] All models were adjusted for HCHS/SOL site, self-identified Hispanic/Latino subgroup, age, sex, and all co-variates that were found to be statistically significant in univariate analyses (P<0.05). Wald tests were used to assess all models for non-linearity and significance, as well as to test for equality and interactions in the pre-defined subgroups of sex (men/women) and pre-diabetes status (normal blood glucose/ pre-diabetes).

We tested for interactions between BMI category (normal, overweight, obese) and the association of insulin resistance with SBP or DBP. In sensitivity analyses, we repeated all statistical procedures outlined above including only individuals with a normal BMI to examine the associations of insulin resistance with SBP and DBP in the 2,524 individuals free from the potential confounding effects of excess or inadequate weight.

All analyses were performed using Stata v13.1 (College Station, TX). A two-sided p-value <0.05 was the threshold for statistical significance.

RESULTS

The demographic and clinical characteristics of the HCHS/SOL participants included in the current analysis are shown in Table 1. The mean (SD) age was 37.2 (13.4) years and 52% were women. Overall, 39% had pre-diabetes, 35% were obese, and 22% had metabolic syndrome. In this study population that did not include any individuals taking anti-hypertensive medications; 59% of individuals had normal BP, 15% had elevated BP, 17% had untreated stage 1 HTN, and 9% had untreated stage 2 HTN based on the 2017 ACC/AHA BP guidelines.[4] BMI, SBP, DBP, mean arterial pressure, pulse pressure, cholesterol levels (total, LDL, triglycerides), HbA1c, fasting glucose, HOMA-IR were significantly higher in individuals with pre-diabetes compared to those with normal blood glucose (Table 1). Demographic and clinic characteristics stratified by the 2017 ACC/AHA BP categories are shown in Table S1.

The associations of clinical and metabolic characteristics with SBP and DBP are shown in Table 2. Both SBP and DBP were significantly associated with male sex, age, BMI, waist/hip ratio, protein-urine albumin/creatine, heart rate, alcohol use, all lipid measures, HbA1c, and fasting glucose.

Associations between Insulin Resistance and Systolic and Diastolic Blood Pressure

In multivariable-adjusted analyses, insulin resistance was positively associated with both SBP (β 2.64±0.44) and DBP (β 1.49±0.35) (p<0.001 for both). There was a significant interaction between sex and the association of HOMA-IR with SBP (p=0.047). In men, the association of HOMA-IR with SBP was positive and linear (linear trend p <0.001, multi-variable adjusted β-coefficient 2.56±0.53), but in women it was nonlinear (overall trend p<0.001, multi-variable adjusted β 3.62±0.69 before the inflection point) (Figure 2A).

For DBP there was no significant interaction with sex, and the association with HOMA-IR was positive and linear (multi-variable adjusted β 1.49±0.35) for both men and women (Figure 2B).

When examined by pre-diabetes status (normal blood glucose vs. presence of pre-diabetes), there was no significant interaction between pre-diabetes status and the association of HOMA-IR with either SBP or DBP. In participants with and without prediabetes there was a significant, positive linear association between SBP and HOMA-IR (multi-variable adjusted β 2.64±0.44) (Figure 3A). For DBP, there was a significant linear association with HOMA-IR only in participants with normal blood glucose (multi-variable adjusted β 1.47±0.44) (Figure 3B).

Sensitivity Analyses Examining Individuals with a Normal BMI

There was no statistically significant interaction between BMI category (normal, overweight, obese) and the association of HOMA-IR with SBP or DBP (p>0.5). In sensitivity analyses including only normal BMI individuals (N=2,524), the association between HOMA-IR and both SBP (β 1.94±0.76) and DBP (β 1.04±0.46) was positive, linear and statistically significant. There was no statistically significant interaction between sex or pre-diabetes status and the association of HOMA-IR with either SBP or DBP (p>0.05).

DISCUSSION

In the largest US community-based sample of Hispanics/Latino adults of diverse national backgrounds across the age spectrum with a relatively high (~40%) burden of untreated elevated BP and HTN we demonstrate that insulin resistance was positively associated with both SBP and DBP in individuals without diabetes. This association was not confounded by antihypertensive medications and was independent of abnormal BMI. We also demonstrate a sex-specific interaction exists in the association between insulin resistance and SBP; in women the association was non-linear and stronger than the non-linear association seen in men. Further, we found a linear association between insulin resistance and SBP in individuals with both normal blood glucose and prediabetes, whereas the association with DBP was only significant in individuals with normal blood glucose.

The mechanisms through which insulin resistance alters BP are not fully understood. The hypersecretion of high levels of insulin in response to insulin resistance is known to affect renal sodium excretion,[8, 9] the sympathetic nervous system,[12] and the renin-angiotensin-aldosterone system.[10, 11] Insulin resistance has also been associated with impaired endothelium-dependent vasodilatation, atherosclerosis, and inflammation.[32] Over the long-term, these toxic effects of insulin resistance on the vasculature may contribute to BP elevation, and as shown in the current study this appears to be independent of diabetes status. The association between insulin resistance and DBP in individuals with normal blood glucose also suggests that insulin resistance may affect DBP even prior to the development of the hyperinsulinemia associated with pre-diabetes. These findings may help us understand why DBP is the strongest predictor of coronary heart disease in young and middle-age adults.[33, 34]

Consistent with previous studies, we found a positive association between insulin resistance and BP.[13-15] Unlike prior work, we were able to examine this association in a large number of individuals not taking antihypertensive medications, thus eliminating potential confounding effects of therapies such as thiazide diuretics and beta-blockers, which are known to increase insulin resistance.[17, 35-37] It is also known that visceral adipose tissue secretes cytokines that may result in inflammation and contribute to insulin resistance.[38-40] Unlike prior studies which reported attenuation or nulling of the association between insulin resistance and BP after adjustment for BMI,[41-43] the associations remained significant after multivariable adjustment that included BMI, as well as in sensitivity analyses examining only individuals with a normal BMI. This consistency lends strength to the hypothesis that the relationship between insulin resistance and BP is independent of the contribution of obesity to insulin resistance. It is possible that the underlying changes in adipose tissue resulting in insulin resistance may also contribute to BP elevation, and future studies are needed to examine the potential direct effects of metabolically active adipose on BP.

It is increasingly recognized that there are sex-specific differences in the association between BP and HTN, though prior studies have reported conflicting results.[44-47] We found a sex-specific interaction in the association between insulin resistance and SBP, with a stronger, non-linear association in females. This is consistent with the findings of Goodfriend et al. who reported an association between insulin resistance in normotensive women but not in men.[45] These findings suggest that the underlying physiologic mechanism of blood pressure and insulin regulation may differ in men and women and may partially explain the heightened cardiovascular risk seen in women with diabetes relative to men with a similar risk factor profile.[48, 49]

Our study has several strengths and limitations that should be considered. First, our study population included individuals aged 18-76 years, with a mean age of 37 years. This represents an age group most likely to benefit from interventions aimed at preventing the development of HTN, a major cause of morbidity and mortality. Next, the use of HOMA-IR as a measure of insulin resistance has been extensively validated for the evaluation of insulin resistance in individuals with and without diabetes.[29, 50-54] Although multiple statistical tests were performed, all analyses were prespecified, and the consistency of our findings across the different models argue against a type I error as an explanation for our findings. Our sample was composed of an ethnically diverse population of Hispanic/Latino women and men, a group that has a heightened risk of diabetes and hypertension, which has historically been understudied. Despite the large sample size, we did not have the power to study these associations by Hispanic/Latino subgroup. Our findings also require confirmation in other populations of different ethnic backgrounds. Another limitation was the cross-sectional nature of our data, which precludes us from investigating potential causal associations between insulin resistance and the development of elevated BP and overt HTN. Lastly, although BP was measured using a rigorous protocol, the measurements were all made during one visit, rather than over at least 2 separate visits as recommended by the ACC/AHA guidelines.[4]

In summary, this study provides a unique opportunity to examine the association between insulin resistance and BP in a large, ethnically diverse, community-based cohort of

Hispanics/Latino adults free from the potentially confounding effects of overt diabetes, abnormal BMI, and anti-hypertensive medications. We found strong, positive associations between insulin resistance and both SBP and DBP as well as sex-based differences in some associations. Our findings support the hypothesis that insulin resistance adversely affects the vasculature early in the disease process, before the development of diabetes, and thus may contribute to the development of HTN, particularly in women. Future, longitudinal sex-specific studies are needed to examine whether there is a casual relationship between insulin resistance and the development of elevated BP and HTN.

PERSPECTIVES

In light of the rising burden of both HTN and diabetes it is imperative that we better understand the associations between insulin resistance and BP. Prior large-scale studies examining these associations have reported conflicting results and their interpretation is limited by potential confounding from overt diabetes, abnormal BMI, and antihypertensive medication use. In this large, ethnically diverse, community-based cohort of Hispanics/ Latino adults, insulin resistance was associated with both SBP and DBP suggesting that insulin resistance may adversely affect the vasculature prior to the development of diabetes independent of confounders. Our findings also suggest the association of insulin resistance and SBP is stronger in women.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGEMENTS

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NOVELTY AND SIGNIFICANCE

What is new:

There are sex-differences in the association between insulin resistance and systolic blood pressure.

What is relevant:

Insulin resistance is associated with systolic and diastolic blood pressure in individuals without diabetes independent of potential confounders such as antihypertensive medications and abnormal body mass index.

Summary:

The positive association between insulin resistance and both SBP and DBP in individuals without diabetes suggests that insulin resistance may adversely affect the vasculature prior to the development of dysglycemia, and this association may be stronger in women than men.
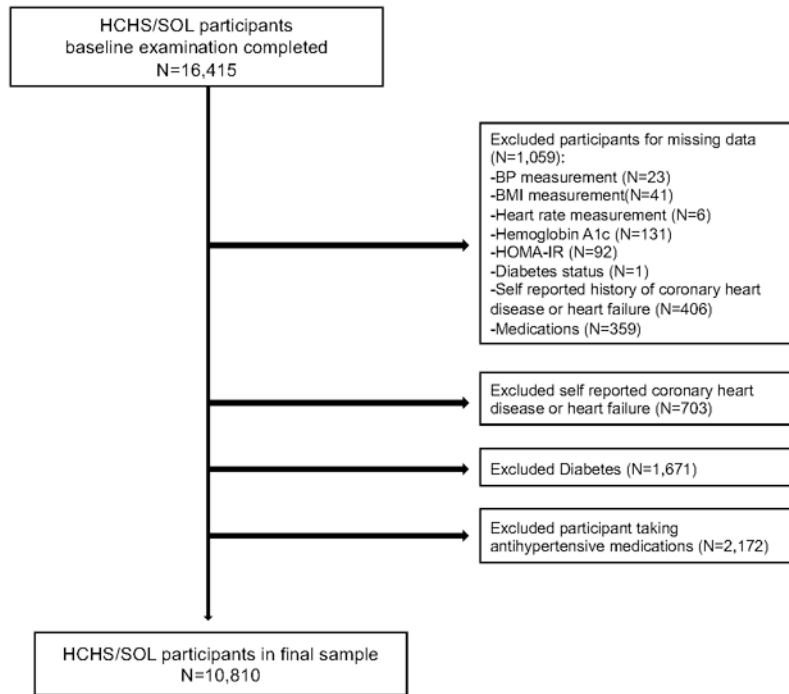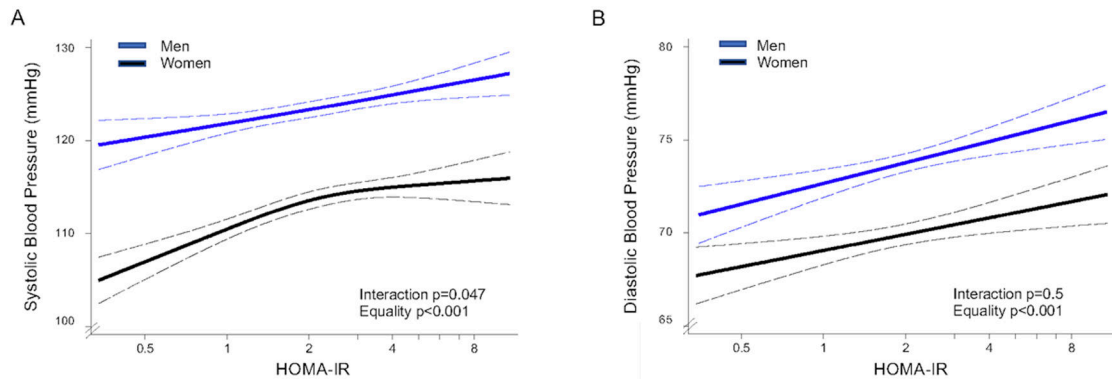
Figure 1.
Study Flowchart

Figure 2. Associations of insulin resistance with systolic and diastolic blood pressure by sex.
Multivariable-adjusted model showing association between HOMA-IR and systolic blood pressure [A], diastolic blood pressure [B] by sex. Models were adjusted for any variable with $p<0.05$ in the univariate analysis: sex, age, years living in the US, body mass index, waist/hip ratio, urine albumin/creatine, heart rate, alcohol use, total physical activity, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, HbA1c, fasting glucose. The dashed black curves represent the upper and lower 95% confidence limits.
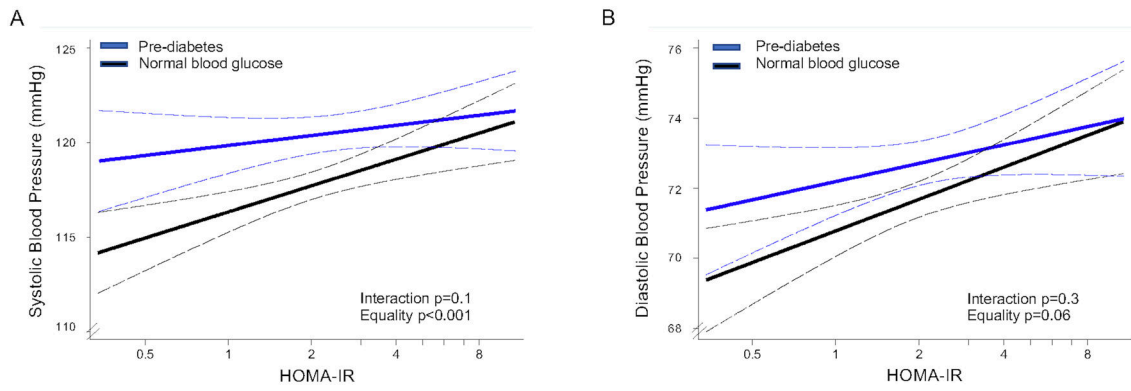
Figure 3. Association of insulin resistance with systolic and diastolic blood pressure by pre-diabetes status.

Multivariable-adjusted linear model showing association between HOMA-IR and systolic blood pressure [A], diastolic blood pressure [B] by pre-diabetes status (normal blood glucose vs. pre-diabetes). Model was adjusted for any variable with $p<0.05$ in the univariate analysis: sex, age, years living in the US, body mass index, waist/hip ratio, urine albumin/creatine, heart rate, alcohol use, total physical activity, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, HbA1c, fasting glucose. The dashed black curves represent the upper and lower 95% confidence limits.

Table 1:

Demographics and Clinical Characteristics Overall and by Pre-Diabetes Status in the Hispanic Community Health Study/Study of Latinos at Visit 1 (2008–2011)

Characteristics at Visit 1	Overall (n = 10,810)	Normal Blood Glucose (n = 5,972)	Pre-diabetes (n = 4,838)	P value
Demographics				
Female, %	52.0	54.7	47.8	<0.001
Age, yrs, mean (SD)	37.2 (13.4)	33.2 (11.9)	43.4 (13.2)	<0.001
Annual Family Income $40,000, %	22.1	23.3	20.4	0.083
Years living in U.S., yrs, mean (SD)	18.6 (13.4)	17.4 (12.3)	20.6 (14.7)	<0.001
Health Insurance, %	46.3	46.8	45.6	0.436
Hispanic/Latin Background, %				0.181
Dominican	9.9	11.1	8.0	
Central/South American	13.2	13.3	13.1	
Cuban	19.1	18.2	20.6	
Mexican	39.0	38.3	40.0	
Puerto-Rican	14.3	14.2	14.4	
Other or more than 1 heritage	4.5	4.9	4.0	
Clinical Characteristics				
2017 ACC/AHA BP Category *, %				<0.001
Normal	59.3	67.4	46.5	
Elevated	14.9	13.8	16.6	
Stage 1 Hypertension	16.9	13.6	22.1	
Stage 2 Hypertension	9.0	5.2	14.9	
CKD, %	2.3	1.4	3.6	<0.001
Metabolic Syndrome, %	21.6	10.2	39.5	<0.001
Current smoker, %	21.6	21.3	22.1	0.522
Alcohol use, drinks/week, mean (SD)	5.4 (8.0)	5.5 (8.1)	5.2 (7.8)	0.359
TPA, metabolic min/day, mean (SD)	145.1 (196.2)	146.8 (193.6)	142.4 (200.4)	0.528
Clinical Measures				
Body mass index, kg/m2, mean. (SD)	28.7 (5.8)	27.7 (5.6)	30.1 (5.8)	<0.001

Characteristics at Visit 1	Overall (n = 10,810)	Normal Blood Glucose (n = 5,972)	Pre-diabetes (n = 4,838)	P value
Waist/hip ratio, mean (SD)	0.9 (0.1)	0.9 (0.1)	0.9 (0.1)	<0.001
Heart rate, bpm, mean (SD)	64.9 (9.4)	64.5 (9.2)	65.7 (9.8)	<0.001
Systolic BP, mmHg, mean (SD)	116.8 (15.0)	113.7 (13.5)	121.6 (15.9)	<0.001
Diastolic BP, mmHg, mean (SD)	71.0 (10.4)	69.1 (9.8)	74.1 (10.5)	<0.001
MAP, mmHg, mean (SD)	86.3 (11.1)	84.0 (10.3)	89.9 (11.4)	<0.001
Pulse pressure, mmHg, mean (SD)	45.8 (10.2)	44.7 (9.2)	47.5 (11.3)	<0.001
Laboratory Measures				
Total cholesterol, mg/dL, mean (SD)	191.6 (41.7)	183.6 (39.1)	204.1 (42.6)	<0.001
HDL cholesterol, mg/dL, mean (SD)	48.9 (12.8)	50.0 (12.8)	47.3 (12.7)	<0.001
LDL cholesterol, mg/dL, mean (SD)	118.6 (35.4)	112.4 (33.3)	128.4 (36.4)	<0.001
Triglycerides, mg/dL, mean (SD)	122.8 (91.0)	107.2 (75.8)	147.0 (106.3)	<0.001
Proteinuria, %	5.5	5.0	6.3	0.027
HbA1c, %, mean (SD)	5.4 (0.4)	5.2 (0.3)	5.6 (0.3)	<0.001
Fasting glucose, mg/dL, mean (SD)	93.1 (8.1)	89.6 (5.7)	98.4 (8.4)	<0.001
HOMA-IR, mean (SD)	2.8 (2.2)	2.3 (1.6)	3.5 (2.6)	<0.001

BP, blood pressure; CKD, chronic kidney disease (eGFR <60 mL/min/1.73m^2); Global Physical Activity Questionnaire was used to derive total physical activity (TPA) in min/day; MAP, mean arterial pressure; HDL, high density lipoprotein; LDL, low density lipoprotein; proteinuria (urine albumin/creatinine >30mg/g); HbA1c, hemoglobin A1c; HOMA-IR, Homeostasis model assessment-Insulin Resistance

*
2017 ACC/AHA Blood Pressure Category: normal BP (<120 mmHg and DBP <80 mmHg), elevated BP (SBP 120-129 mmHg and DBP <80 mmHg), stage 1 hypertension (SBP 130-139 mmHg or DBP 80-89 mmHg), stage 2 hypertension (SBP 140 mmHg or DBP 90 mmHg)

Table 2:

Association of clinical and metabolic characteristics with systolic and diastolic blood pressure

Characteristics at Visit 1	Systolic Blood Pressure (mmHg)		Diastolic Blood Pressure (mmHg)	
	B-coefficient (95% CI)	P value	B-coefficient (95% CI)	P value
Male Sex	9.06 (8.37 - 9.76)	<0.001	3.17 (2.61 - 3.73)	<0.001
Age (per 10 years)	4.92 (4.66 - 5.19)	<0.001	2.45 (2.24 - 2.66)	<0.001
Annual Family Income ≥ $40,000	−0.66 (−1.55 - 0.24)	0.149	−0.49 (−1.21 - 0.23)	0.183
Years living in U.S. (per 5-year increase)	−0.07 (−0.20 - 0.06)	0.302	0.13 (0.02 - 0.24)	0.017
Currently Has Health Insurance, %	−0.05 (−0.74 - 0.64)	0.894	−0.52 (−1.07 - 0.03)	0.062
Body mass index (per 1 kg/m² increase)	0.25 (0.18 - 0.31)	<0.001	0.55 (0.49 - 0.60)	<0.001
Waist/hip ratio (per 0.2 increase)	1.90 (0.88 - 2.92)	<0.001	4.78 (4.02 - 5.54)	<0.001
Current smoker	−0.73 (−1.54 - 0.09)	0.082	−0.20 (−0.88 - 0.49)	0.572
Proteinuria	5.45 (3.70 - 7.20)	<0.001	3.82 (2.65 - 5.00)	<0.001
Chronic kidney disease (eGFR <60)	2.82 (0.04 - 5.61)	0.047	0.48 (−1.22 - 2.17)	0.582
Heart rate (per 5 bpm increase)	0.57 (0.39 - 0.75)	<0.001	1.50 (1.37 - 1.64)	<0.001
Alcohol use (per 1 drink per week increase)	0.14 (0.09 - 0.19)	<0.001	0.12 (0.07 - 0.17)	<0.001
TPA (per 100 metabolic min/day increase)	0.001 (−0.193 - 0.195)	0.993	−0.24 (−0.39 - −0.09)	0.002
Total cholesterol (per 10 mg/dL increase)	0.33 (0.24 - 0.43)	<0.0001	0.39 (0.32 - 0.46)	<0.001
HDL cholesterol (per 10 mg/dL increase)	0.22 (−0.03 - 0.47)	0.080	−0.71 (−0.92 - −0.50)	<0.001
LDL cholesterol (per 10 mg/dL increase)	0.29 (0.18 - 0.40)	<0.001	0.40 (0.32 - 0.48)	<0.001
Triglycerides (per 10 mg/dL increase)	0.11 (0.07 - 0.16)	<0.001	0.18 (0.15 - 0.22)	<0.001
HbA1c (per 1 % increase)	1.64 (0.48 - 2.80)	0.006	2.90 (1.99 - 3.80)	<0.001
Fasting glucose (per 10 mg/dL increase)	1.94 (1.46 - 2.41)	<0.001	1.77 (1.38 - 2.15)	<0.001
HOMA IR (per 1unit increase)	0.80 (0.63 - 0.97)	<0.001	1.18 (0.99 - 1.37)	<0.001

All are adjusted for age and sex, age is adjusted for only sex and sex is adjusted for only age.

HDL, high density lipoprotein; LDL, low density lipoprotein; proteinuria (urine albumin/creatinine >30mg/g); HbA1c, hemoglobin A1c; Global Physical Activity Questionnaire was used to derive total physical activity (TPA) in minutes/day (min/day); HOMA-IR, Homeostasis model assessment-Insulin Resistance; eGFR, estimated glomerular filtration rate;