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Relationship among Age, Insulin Resistance, and Blood Pressure

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Abstract

The effect of age to modify the relationship between insulin resistance and hypertension are unclear. In this retrospective, cross-sectional study, median age was used to create two age groups (<52 vs. ≥52 years), and comparisons were made of metabolic characteristics, including steady-state plasma glucose (SSPG) concentrations measured during the insulin suppression test to quantify insulin resistance. Individuals were stratified into SSPG tertiles and categorized as having normal BP, prehypertension, or hypertension. SSPG concentrations were similar in the two age groups (161 vs. 164 mg/dL). In the most insulin resistant tertile, distribution of normal BP, prehypertension, and hypertension was equal in those <52 years, whereas in those ≥52 years, prevalence of hypertension was increased approximately five-fold as compared to those with normal BP. Multivariate regression analysis demonstrated significant interaction between age and SSPG in predicting systolic BP (p=0.023). In stratified analysis, SSPG, but not age, was an independent predictor of systolic BP and diastolic BP in ≥52 year group, whereas the reverse was true in the younger group. The adverse impact of insulin resistance on blood pressure was accentuated in older individuals and may have a greater impact than further aging.

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Disclosure

All authors declared no conflict of interest.

Keywords

age; Insulin resistance; steady-state plasma glucose; blood pressure

Introduction

Although it is well-recognized that the prevalence of hypertension increases with age, [1,2] the explanation for this relationship is not as well-established. As pointed out by Lakatta and Levy,[3] "increasing age contributes to an increased exposure time to other age-dependent risk factors." Thus, one possibility is that the adverse impact of any given abnormality will increase as a simple function of duration of exposure. Alternatively, they suggested that the effect of the abnormality in question may "change with time related to an aging process," and that its adverse impact in older persons "is due to age-disease interactions."

These alternatives are straight-forward, but deciding between them is not so easy. For example, there are several lines of evidence suggesting a relationship between insulin resistance and essential hypertension. Patients with essential hypertension are more glucose intolerant and insulin resistant than those with normal blood pressure.[4,5] Normotensive first degree relatives of patients with essential hypertension are insulin resistant.[6] Finally, insulin resistance predicts the development of essential hypertension.[7,8] It is also known that aging is associated with increased insulin resistance.[9,10] However, the interrelationship among age, insulin resistance and blood pressure has not been fully explored. It could be argued that the longer the duration of insulin resistance, the more likely the development of hypertension. Alternatively, is there something intrinsic to the aging process that accentuates the known untoward effect of insulin resistance on blood pressure in older persons, increasing the likelihood of an elevation in blood pressure? The current analysis is an attempt to begin addressing these unresolved issues in order to increase understanding of the complicated relationship that exists between age, insulin resistance and elevated blood pressure.

Methods

Study design and subjects

Participants included 493 individuals in our ongoing registry, who were recruited between 2003 and 2013. All had signed informed consent and participated in our research studies related to insulin resistance approved by the Institutional Review Board at Stanford University. Age ranged from 22 to 71 years, and 66% of the total population was non-Hispanic white. All were free of diabetes, cardiovascular, liver, or kidney disease. Nondiabetic status of the subjects was determined based on no medical history of diabetes, no use of antihyperglycemic medications and fasting glucose level less than 126 mg/dL. All participants had an insulin suppression test to quantify insulin-mediated glucose disposal and measurement of blood pressure.

Anthropometric and biochemical measurements

All procedures were performed in the Stanford Clinical and Translational Research Unit after fasting for 12 hours. Body weight and height were measured to the nearest 0.1 kg and

0.1 cm, respectively. Body mass index (BMI) was calculated as weight (kg) divided by height (m^2). Waist circumference (WC) was taken midway between the inferior margin of the last rib and the crest of the ilium in the horizontal plane while in an upright position. WC was measured in duplicate with an anthropometric tape while the participants were wearing light clothing. Blood pressure (BP) and heart rate were measured from the brachial artery with an appropriate cuff size using a Dinamap automatic recorder (GE Healthcare). The patients were instructed to avoid alcohol, nicotine, caffeine, and exercise within 30 minutes before BP measurement. Before the BP and heart rate measurements, individuals were seated quietly in a chair with back support, with both feet flat on the floor for 5 minutes and one arm was supported at heart level. Three measurements were taken at 1-minute intervals and the mean of these readings was used for data analysis. Pulse pressure was obtained by the numeric difference between systolic and diastolic BP (systolic BP minus diastolic BP).

Definition of Elevated Blood Pressure

Subjects (n=493) were divided into 3 groups according to established clinical guidelines[11] as having normal blood pressure (<120 mm Hg systolic and <80 mm Hg diastolic), prehypertension (120–139 mm Hg systolic or 80–89 mm Hg diastolic), or hypertension (≥140 mm Hg systolic or ≥90 mm Hg diastolic or use of BP-lowering medication). Based on these definitions, the experimental population contained 163 persons with normal BP and 330 with elevated BP, subdivided into prehypertension (n=146) and hypertension (n=184).

Quantification of insulin-mediated glucose disposal

Differences in degree of insulin resistance were obtained by quantifying insulin-mediated glucose disposal by the modified version[12] of the insulin suppression test as initially introduced and validated by our research group.[12,13] Briefly, after an overnight fast, an intravenous catheter was placed in each of the subject's arms. One arm was used for the administration of a 180-minute infusion of octreotide (0.27 μg/m^2 per min), insulin (32 mU/m^2 per min), and glucose (267 mg/m^2/min); the other arm was used for collecting blood samples. Blood was drawn at 10-minute intervals from 150 to 180 minutes of the infusion to determine the steady-state plasma glucose (SSPG) and insulin (SSPI) concentrations. Since the SSPI concentrations are comparable in all subjects, the SSPG concentration provides a direct measure of the ability of insulin to mediate disposal of an infused glucose load. Higher SSPG concentration signifies greater degree of insulin resistance. Estimates of insulin resistance obtained with the insulin suppression test are highly correlated to those determined with the hyperinsulinemic, euglycemic clamp technique.[13,14] Since prospective studies have shown that the most insulin resistant third of an apparently healthy population is at increased risk for a number of clinical syndromes including hypertension,[15,16] SSPG concentrations were used to stratify the entire population into tertiles; tertile 1 (lowest), tertile 2 (intermediate), and tertile 3 (highest). Participants in SSPG tertile 3 were classified as being insulin resistant.

Statistical analysis

In order to examine the relationship between age, insulin resistance and elevated BP, participants were divided into 2 groups based on the median age of 52 years (<52, range: 22–51 years vs. ≥52, range: 52–71 years). Data were reported as mean ± standard deviation

(SD) for normally distributed variables, as median (range) for non-normally distributed variables or as number of participants (percentages). Statistical differences in demographic and clinical characteristics between groups by age were evaluated by means of chi-square test for categorical variables and Student's t-test or Mann-Whitney U test for continuous variables. Chi-square tests were used to compare prevalence rates of hypertension status across SSPG tertiles within each age group (<52 years vs. ≥52 years).

Univariate and multivariate regression analyses were performed to evaluate the prediction of age and SSPG on levels of BP and an interaction between age and SSPG. BP was regressed on age, SSPG, sex, ethnicity, BMI, WC, heart rate, and a multiplicative interaction term of age and SSPG. Systolic BP, diastolic BP and SSPG were presented as continuous variables, while age was categorized at two levels, namely, <52 years and ≥52 yrs. The coefficient for every 10 year increase in age and every 50 mg/dL increase in SSPG was calculated. For sensitivity analyses, a similar process was conducted in those individuals not taking BP lowering drugs.

A 2-tailed probability value of <0.05 was considered statistically significant in all analyses. All the statistical analysis was performed using SPSS 21.0 (SPSS Inc, Chicago, IL, U.S.A).

Results

Table 1 compares demographic and metabolic characteristics of the two age groups. The prevalence of prehypertension was comparable in the 2 groups. However, prevalence of hypertension was significantly increased in the older group (45% vs. 29%), associated with substantially more individuals in the younger group with normal BP (42% vs. 25%). There were no differences in either BMI, WC, or mean SSPG concentration in the 2 groups, but the older group contained a greater percentage of non-Hispanic Whites, was more often being treated with BP-lowering drugs, had a higher systolic BP, as well as higher fasting plasma glucose, total cholesterol, and HDL-cholesterol concentrations.

In view of the difference in use of BP-lowering drugs, a similar analysis was performed, excluding patients on these medications (Supplemental Table 1). Although there were fewer subjects, the comparisons between the two groups were comparable. In particular, SSPG concentrations did not differ between the two groups, and there were relatively more subjects in the older group with either hypertension (18% vs. 14%) or prehypertension (45% vs. 35%) (p=0.019).

In Table 2, we evaluated the univariate and multivariate association between age (<52 and ≥52), SSPG, and either systolic or diastolic BP. Systolic BP was significantly associated with age, sex, BMI, WC, and SSPG concentration in univariate analysis. In multivariate analysis, there was a significant interaction between age and SSPG (p=0.023). For diastolic BP, sex and heart rate were significantly associated, and there was no interaction between age and SSPG.

Given the significant interaction between age and SSPG in predicting systolic BP, we conducted stratified analysis by age. In Table 3, we compared the two age groups in terms of the prevalence of BP categories by SSPG tertiles. The ≥52 age group had higher prevalence

of hypertension regardless of SSPG tertile. Perhaps the most dramatic age-related difference is the comparison of the distribution of BP status in the most insulin resistant subset (SSPG tertile 3) of the two groups. Thus, the prevalence of normal BP, prehypertension, and hypertension is almost identical in the <52 age group, whereas the prevalence of hypertension in those ≥52 years is increased approximately 5-fold as compared to those with normal BP. At the other extreme, focusing on SSPG tertile 1, the prevalence of hypertension and normal BP in the older subjects was reasonably comparable. As before, a sub-analysis was undertaken that included only participants free of BP-lowering drugs, and the findings in Supplemental Table 2 reveal the same age-related differences.

Table 4 shows the results of multivariate regression analyses stratified by age. In <52 year age group, age, but not SSPG, was an independent predictor of systolic BP and diastolic BP, when adjusted for sex, ethnicity, BMI, WC, and heart rate (Model 2). In ≥52 year age group, SSPG was an independent predictor of systolic BP and diastolic BP, whereas age was not.

Discussion

Consistent with known observations,[1–3] we observed an increased prevalence of hypertension in the older age group. However, this finding cannot be attributed to an age difference in insulin resistance, *per se*, as the SSPG concentration (quantitative measure of insulin-stimulated glucose uptake) was essentially identical in the two age groups. Despite the fact that overall insulin resistance in the two groups did not vary, there were substantial age-related differences in the relationship between insulin resistance and blood pressure regulation. For example, essentially equal number of insulin resistant persons (SSPG tertile 3) in the <52 years age group had normal BP, prehypertension, or hypertension. In contrast, in those ≥52 years, only 11% of the most insulin resistant individuals (SSPG tertile 3) were able to maintain a normal BP. Furthermore, age modified the relationship between age and SSPG concentration, such that only older individuals had a significantly higher BP associated with an increased degree of insulin resistance. To put it simply, the untoward effect of insulin resistance on blood pressure was more evident in the older individuals.

Although certainly not definitive, the results of this analysis are consistent with the view that the impact of insulin resistance on blood pressure will “change with time related to an aging process,” and that its modulation of the prevalence of elevated blood pressure in older persons “is possibly due to age-disease interactions.” While exact mechanisms are unknown, the aging vasculature may be more susceptible to insults that can raise blood pressure. For example, the compensatory hyperinsulinemia that prevents frank type 2 diabetes in insulin resistant persons acts on normally insulin sensitive tissues, e.g., kidney and sympathetic nervous system, to enhance sodium reabsorption and increase sympathetic tone—changes that can certainly contribute to elevated BP.[17,18] Perhaps, the older the person, the greater is the magnitude of the untoward impact of compensatory hyperinsulinemia on kidney and/or the sympathetic nervous system. Another speculation is that aging may impair the ability to compensate for these effects of insulin resistance. For example, aging is associated with greater likelihood of salt-sensitive hypertension[19,20] which may represent an age-related decrease in compensation for insulin-induced increase in sodium reabsorption and blood pressure. On the other hand, in the absence of longitudinal observations, we cannot rule out

the possibility that the enhanced impact of insulin resistance on blood pressure elevation in the older group is simply secondary to having been insulin resistant for a longer period of time.

Finally, there are limitations to our study. As this was a cross-sectional study, we cannot establish causal links between insulin resistance and blood pressure. In addition, we cannot rule out that the duration of insulin resistance had an impact on increasing blood pressure in older vs. younger groups. We also did not have direct measurements of body composition, which may differ with age. BMI and WC did not differ, but we did not evaluate other measurements such as hip circumference. Finally, since we included individuals with hypertension, we cannot rule out effects of antihypertensive medications on insulin resistance. However, subgroup analyses on untreated individuals showed similar results.

In conclusion, our findings suggest that the relationship between insulin resistance and blood pressure may be modified by age, and not simply because the older group was more insulin resistant. Instead, older individuals seemed to be more susceptible to the effects of insulin resistance to raise blood pressure, and this is particularly interesting in that enhancing insulin sensitivity appears effective in reducing blood pressure in older populations with hypertension.[21–24] Therefore, improving insulin resistance may be an important, modifiable risk factor for hypertension in older individuals.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Burt VL, Cutler JA, Higgins M, Horan MJ, Labarthe D, Whelton P, et al. Trends in the prevalence, awareness, treatment, and control of hypertension in the adult US population. Data from the health examination surveys, 1960 to 1991. Hypertension. 1995; 26:60–9. [PubMed: 7607734]
2. Guo F, He D, Zhang W, Walton RG. Trends in prevalence, awareness, management, and control of hypertension among United States Adults, 1999 to 2010. J Am Coll Cardiol. 2012; 60:599–606. [PubMed: 22796254]
3. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a “set up” for vascular disease. Circulation. 2003; 107:139–46. [PubMed: 12515756]
4. Ferrannini E, Buzzigoli G, Bonadonna R, Giorico MA, Oleggini M, Graziadei L, et al. Insulin resistance in essential hypertension. N Engl J Med. 1987; 317:350–7. [PubMed: 3299096]
5. Pollare T, Lithell H, Berne C. Insulin resistance is a characteristic feature of primary hypertension independent of obesity. Metabolism. 1990; 39:167–74. [PubMed: 2405235]

6. Allemann Y, Horber FF, Colombo M, Ferrari P, Shaw S, Jaeger P, et al. Insulin sensitivity and body fat distribution in normotensive offspring of hypertensive parents. Lancet. 1993; 341:327–31. [PubMed: 8094112]
7. Raitakari OT, Porkka KVK, Rönnemaa T, Knip M, Uhari M, Akerblom HK, et al. The role of insulin in clustering of serum lipids and blood pressure in children and adolescents. Diabetologia. 1995; 38:1042–50. [PubMed: 8591817]
8. Reaven GM. Insulin resistance/compensatory hyperinsulinemia, essential hypertension, and cardiovascular disease. J Clin Endocrinol Metab. 2003; 88:2399–403. [PubMed: 12788834]
9. Reaven GM, Reaven E. Effects of age on various aspects of glucose and insulin metabolism. Mol Cell Biochem. 1980; 31:37–47. [PubMed: 6993916]
10. DeFronzo RA. Glucose intolerance and aging. Diabetes Care. 1981; 4:493–501. [PubMed: 7049632]
11. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003; 42:1206–52. [PubMed: 14656957]
12. Pei D, Jones CN, Bhargava R, Chen YD, Reaven GM. Evaluation of octreotide to assess insulin-mediated glucose disposal by the insulin suppression test. Diabetologia. 1994; 37:843–5. [PubMed: 7988789]
13. Greenfield MS, Doberne L, Kraemer F, Tobey T, Reaven G. Assessment of insulin resistance with the insulin suppression test and the euglycemic clamp. Diabetes. 1981; 30:387–92. [PubMed: 7014307]
14. Knowles J, Assimes TL, Tsao PS, Natali A, Quertemous E, Reaven GM, et al. Measurement of insulin-mediated glucose uptake: Direct comparison of the modified insulin suppression test and the euglycemic, hyperinsulinemic clamp. Metabolism. 2013; 62:548–53. [PubMed: 23151437]
15. Yip J, Facchini FS, Reaven GM. Resistance to insulin-mediated glucose disposal as a predictor of cardiovascular disease. J Clin Endocrinol Metab. 1998; 83:2773–6. [PubMed: 9709945]
16. Facchini FS, Hua N, Abbasi F, Reaven GM. Insulin resistance as a predictor of age-related diseases. J Clin Endocrinol Metab. 2001; 86:3574–8. [PubMed: 11502781]
17. Reaven GM, Lithell H, Landsberg L. Hypertension and associated metabolic abnormalities; the role of insulin resistance and the sympathoadrenal system. N Engl J Med. 1996; 334:374–81. [PubMed: 8538710]
18. Reaven GM. The kidney: An unwilling accomplice in syndrome X. Am J of Kidney Dis. 1997; 30:928–31. [PubMed: 9398143]
19. DeFronzo RA, Cooke RC, Anders R, Faloona GR, Davis PJ. The effects of insulin on renal handling of sodium, potassium, calcium and phosphate in man. J Clin Invest. 1975; 55:845–55. [PubMed: 1120786]
20. Khaw KT, Barrett-Connor E. Increasing sensitivity of blood pressure to dietary sodium and potassium with increasing age. A population study using casual urine specimens. Am J Hypertens. 1990; 3:505–11. [PubMed: 2369500]
21. Ikeda T, Gomi T, Hirawa N, Sakurai J, Yoshikawa N. Improvement of insulin sensitivity contributes to blood pressure reduction after weight loss in hypertensive subjects with obesity. Hypertension. 1996; 27:1180–6. [PubMed: 8621214]
22. Blumenthal JA, Sherwood A, Gullette EC, Babyak M, Waugh R, Georgiades A, et al. Exercise and weight loss reduce blood pressure in men and women with mild hypertension: effects on cardiovascular, metabolic, and hemodynamic functioning. Arch Intern Med. 2000; 160:1947–58. [PubMed: 10888969]
23. Su HY, Sheu WH, Chin HM, Jeng CY, Chen YD, Reaven GM. Effect of weight loss on blood pressure and insulin resistance in normotensive and hypertensive obese individuals. Am J Hypertens. 1995; 8:1067–71. [PubMed: 8554729]
24. Mertens IL, Van Gaal LF. Overweight, obesity, and blood pressure: the effects of modest weight reduction. Obesity Res. 2000; 8:270–8.

Highlights

- We performed this study to investigate the effect of age to modify the relationship between insulin resistance and hypertension.
- We used steady-state plasma glucose (SSPG) concentrations measured during the insulin suppression test to quantify insulin resistance.
- Multivariate regression analysis demonstrated significant interaction between age and SSPG in predicting systolic BP.
- SSPG, but not age, was an independent predictor of systolic BP and diastolic BP in ≥52 year group, whereas the reverse was true in the younger group.
- Our findings suggest that the relationship between insulin resistance and blood pressure may be modified by age, and not simply because the older group was more insulin resistant.

Table 1

Clinical characteristics of study participants stratified by age

Variable	<52 yr (n=230)	≥52 yr (n=263)	P-value
Age (year)	43 ± 6	58 ± 5	<0.001
Male, n (%)	90 (39.1)	112 (42.6)	0.246
Body mass index (kg/m^2)	31 (20–60.8)	30 (18.8–53.8)	0.616
Waist circumference (cm)	101 ± 15	102 ± 12	0.163
Non-Hispanic White, n(%)	124 (54)	200 (76)	<0.001
Treated HTN, n(%)	40 (17.4)	87 (33.1)	<0.001
Systolic BP (mmHg)	121 ± 15	126 ± 15	<0.001
Diastolic BP (mmHg)	75 ± 10	75 ± 9	0.995
Pulse pressure (mmHg)	47 ± 10	51 ± 12	0.057
Heart rate (/min)	71 ± 10	67 ± 9	0.544
NBP/PreHTN/HTN, n (%)	98/66/66 (42/29/29)	65/80/118 (25/30/45)	<0.001
SSPG, mg/dL	161 (41–340)	164 (43–332)	0.907
SSPG tertile, n(%)	79/74/74 (34/33/33)	85/84/94 (32/32/36)	0.705
FPG (mg/dL)	95 (65–123)	100 (71–126)	<0.001
Total cholesterol (mg/dL)	186 (116–404)	194 (113–361)	0.011
LDL-cholesterol (mg/dL)	119 ± 34	121 ± 33	0.359
HDL-cholesterol (mg/dL)	45 ± 12	49 ± 14	<0.001
Triglycerides (mg/dL)	107 (32–800)	118 (29–1217)	0.453

Data are shown as mean±SD, median (range) or as number (%).

NBP: normal blood pressure; PreHTN: prehypertension; SSPG: steady-state plasma glucose; LDL: low density lipoprotein; HDL: high density lipoprotein

Table 2

Interaction between age and insulin resistance (SSPG) in predicting systolic and diastolic blood pressure

Variable	Univariate		Multivariate		Multivariate with interaction term		
	B	95% CI	B	95% CI	B	95% CI	p-value
Systolic blood pressure							
Age(≥52 vs. <52)	4.31**	1.68–6.93	4.98**	2.24–7.75	−2.12	−8.86–4.60	
Sex(male vs. female)	5.23**	2.58–7.88	6.36**	3.38–9.34	8.03**	6.21–9.84	
Ethnicity (nonHispanic white)	1.70	−1.08–4.49	1.35	−1.57–4.26	−0.68	−2.45–1.09	
BMI	0.49**	0.24–0.74	0.67**	0.18–1.16	0.04	−0.26–0.34	
WC (cm)	0.22**	0.12–0.31	−0.13	−0.32–0.06	−0.06	−0.18–0.06	
Heart rate (/min)	0.11	−0.03–0.25	0.11	−0.04–0.25	0.12	0.03–0.21	
SSPG(mg/dL)	0.04**	0.02–0.06	0.02**	0.00–0.05	0.01	−0.003–0.03	
Age*SSPG					−0.04*	0.01–0.08	0.023
Diastolic blood pressure							
Age(≥52 vs. <52)	−0.006	−1.68–1.67	0.49	−1.19–2.16	−2.79	−6.88–1.31	
Sex(male vs. female)	7.48**	5.92–9.05	8.03**	6.21–9.84	8.05**	6.24–9.85	
Ethnicity (nonHispanic white)	−0.14	−1.90–1.62	−0.68	−2.45–1.09	−0.72	−2.45–1.09	
BMI	−0.02	−0.18–0.14	0.04	−0.26–0.34	0.06	−0.24–0.35	
WC (cm)	−0.02	−0.18–0.14	−0.06	−0.18–0.06	−0.06	−0.18–0.06	
Heart rate (/min)	0.09*	0.01–0.18	0.12**	0.03–0.21	0.13**	0.04–0.22	
SSPG(mg/dL)	0.009	−0.002–0.02	0.01	−0.003–0.03	−0.001	−0.02–0.02	
Age*SSPG					0.02	−0.003–0.04	0.086

Univariate and multivariate analyses predicting systolic and diastolic BP are shown.

Last column includes the interaction term between age and SSPG.

Data are given as regression coefficients (B) and 95% confidence intervals.

** p<0.01,

* p<0.05

Table 3

Prevalence of HTN status according to SSPG tertiles

Variable	SSPG Tert1	SSPG Tert2	SSPG Tert3	p value
Age <52 years				
NBP	38 (48.1%)	36 (46.8%)	24 (32.4%)	0.217
Prehypertension	23 (29.1%)	18 (23.4%)	25 (33.8%)	
Hypertension	18 (22.8%)	23 (29.9%)	25 (33.8%)	
Age ≥ 52 years				
NBP	35 (41.2%)	20 (23.8%)	10 (10.6%)	<0.001
Prehypertension	19 (22.4%)	28 (33.3%)	33 (35.1%)	
Hypertension	31 (36.5%)	36 (42.9%)	51 (54.3%)	

Data was presented as frequency (percentage).

P-value was calculated by chi-squared test.

Table 4

Age and insulin resistance (SSPG) as predictors of systolic and diastolic blood pressure in younger and older individuals.

Variable	Model1				Model2			
	B	SE	β	p	B	SE	β	p
Systolic blood pressure								
In Age<52 (N=230)								
Age (10year)	5.83	1.45	0.26	**<0.001**	6.42	1.44	0.29	<0.001
SSPG (50mg/dL)	1.53	0.67	0.15	**0.024**	−0.34	0.82	−0.03	0.682
In Age≥52 (N=263)								
Age (10year)	3.42	2.05	0.1	0.096	3.72	2.11	0.11	0.079
SSPG (50mg/dL)	2.41	0.68	0.22	**<0.001**	2.06	0.80	0.19	0.010
Diastolic blood pressure								
In Age<52 (N=230)								
Age (10year)	3.88	0.95	0.27	**<0.001**	3.76	0.93	0.26	<0.001
SSPG (50mg/dL)	0.39	0.44	0.06	0.38	0.22	0.53	0.03	0.675
In Age≥52 (N=263)								
Age (10year)	−1.85	1.31	−0.09	0.157	−1.94	1.25	−0.09	0.122
SSPG (50mg/dL)	0.99	0.43	0.14	**0.023**	0.96	0.47	0.14	0.043

B=regression coefficient; SE=standard error; β=standardized regression coefficient.

Model1 were calculated after adjustment for age and SSPG as covariates.

Model2 were calculated after adjustment for age, sex, ethnicity, BMI, WC, heart rate, and SSPG as covariates.