Greater Effect of Glycemia on Incidence of Hypertension in Women Than in Men

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OBJECTIVE— In subjects with NIDDM, diabetic women have a greater relative excess of CHD relative to nondiabetic women than do diabetic men relative to nondiabetic men. This excess in diabetic women is explained partially by the particularly atherogenic pattern of lipoproteins in this group. We hypothesize that diabetic women also may have a higher incidence of hypertension than diabetic men.

RESEARCH DESIGN AND METHODS— We examined the effect of NIDDM and IGT relative to NGT on the incidence of hypertension separately in men (n = 844) and women (n = 618) in the 8-yr follow-up of the San Antonio Heart Study, a population-based study of diabetes and cardiovascular disease.

RESULTS— Women had a greater risk of hypertension with worsening glucose tolerance (RR NIDDM/NGT = 2.65 and RR IGT/NGT = 1.94) compared with men (RR NIDDM/NGT = 1.61 and RR IGT/NGT = 0.91). Controlling for other possible confounding variables such as age, obesity, body fat distribution, and fasting insulin concentration did not alter the interaction of sex and glycemia on incidence of hypertension.

CONCLUSIONS— The especially increased risk of hypertension in women with abnormal glucose tolerance may explain partly the high risk of CHD in this group.

The extent to which NIDDM augments the risk of CHD has been reported to be greater in women than in men in many (1–3), although not all, studies (4,5). One possible explanation for this phenomenon could be a more adverse effect of diabetes on the lipid and lipoprotein pattern (especially decreased high-density lipoprotein cholesterol) in diabetic women than diabetic men (6–8). This particularly adverse effect on the lipids and lipoproteins of diabetic women could occur even before the onset of clinical diabetes (9).

Hypertension is common in patients with NIDDM (10,11). However, few data are available on the effect of glycemia on the incidence of hypertension. Furthermore, no prospective study has examined whether there is a sex difference in this effect. In this report, we examine the effects of glycemia on the incidence of hypertension separately in men and women in the 8-yr follow-up of the San Antonio Heart Study. We also examine whether these associations are explained by sex differences in the effects of obesity, body fat distribution, and fasting insulin concentrations on hypertension incidence. In a previous article on this population, we reported a greater effect of body fat distribution on the incidence of NIDDM in women compared with men (12).

RESEARCH DESIGN AND METHODS

— The San Antonio Heart Study is a population-based study of diabetes and cardiovascular disease in Mexican Americans and non-Hispanic whites. From 1979 to 1982, we randomly sampled households from several San Antonio census tracts: two low-income (barrio) census tracts (99% Mexican American), two middle-income (transitional) census tracts (60% Mexican American and 40% non-Hispanic white), and a cluster of high-income (suburban) census tracts (10% Mexican American and 90% non-Hispanic white) (13). Only Mexican Americans were sampled in the barrio. Stratified random sampling was used in the middle-income and suburban census tracts to ensure the inclusion of approximately equal numbers from each ethnic group in the study sample from these neighborhoods. All men and nonpregnant women 25–64 yr of age residing in the randomly selected

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NIDDM, NON-INSULIN-DEPENDENT DIABETES MELLITUS; CHD, CORONARY HEART DISEASE; IGT, IMPAIRED GLUCOSE TOLERANCE; NGT, NORMAL GLUCOSE TOLERANCE; RR, RELATIVE RISK; BMI, BODY MASS INDEX; WHO, WORLD HEALTH ORGANIZATION; ANOVA, ANALYSIS OF VARIANCE.

households were eligible for study. A total of 1288 Mexican Americans and 929 non-Hispanic whites was included in the 1979–1982 survey. Mexican Americans were defined as individuals whose ancestry and cultural traditions derived from a Mexican national origin (14). A detailed description of the 1979–1982 survey has been published previously (13). The overall response rate to the baseline survey was 63.9%. The study was approved by the Institutional Review Board of the University of Texas Health Science Center at San Antonio, and all subjects gave informed consent.

At the baseline examination, blood specimens were obtained after a 12- to 14-h fast and 1 and 2 h after administration of a 75-g glucose equivalent load (Glucola, Ames, Elkhart, IN). Plasma glucose concentrations were measured with an Abbott Bichromatic Analyzer (South Pasadena, CA). Insulin was measured by a commercial radioimmunoassay (Diagnostic Products, Los Angeles, CA) (15). Postload (2-h) insulin concentrations were not measured at baseline. Diabetes mellitus was diagnosed according to WHO criteria (fasting plasma glucose level ≥7.8 mM (140 mg/dl) and/or 2-h postload glucose level ≥11.1 mM [200 mg/dl]) (16). Subjects who did not meet WHO plasma glucose criteria but were under treatment with oral antidiabetic agents or insulin also were considered to have diabetes. IGT also was diagnosed according to WHO criteria (fasting plasma glucose level <7.8 mM (140 mg/dl) and 2-h plasma glucose between 7.8 and 11. 1 mM [140 and 200 mg/dl]). Subjects with NGT had both a fasting and a 2-h plasma glucose <7.8 mM (140 mg/dl). Diabetic subjects who were not taking insulin were considered to have NIDDM. Diabetic subjects taking insulin but whose age at disease onset was >40 yr and who had BMI ≥30 kg/m^2 also were considered to have NIDDM. Other insulin-taking diabetic subjects were considered to have insulin-dependent diabetes mellitus or to be unclassifiable and were excluded from analyses involving diabetes.

Height, weight, and subscapular and triceps skin folds were determined after each participant had removed his or her upper garments and donned an examination gown (17). BMI was calculated as weight (kg) divided by height squared (m^2). The ratio of subscapular-to-triceps skin fold (centrality index) was used as a measure of central adiposity. (Although information on waist and hip circumferences were available at the follow-up examination, this information was not collected at baseline.)

Systolic (1st-phase) and diastolic (5th-phase) blood pressures were measured with a random-zero sphygmomanometer (Hawksley-Gelman, Lancing, Sussex, UK) to the nearest even digit on the right arm of the seated participant after at least a 5-min rest period. Three readings were recorded for each individual, and the subject's blood pressure was defined as the average of the second and third readings. The definition of hypertension was a diastolic blood pressure ≥95 mmHg or current use of antihypertensive medications (Hypertension Detection and Follow-up Program; 18). (The use of a definition of hypertension including both elevated systolic [≥160 mmHg] and diastolic blood pressure yielded similar results to those presented in this report.)

In October 1987, an 8-yr follow-up was begun to determine the incidence of NIDDM and cardiovascular disease. Vital status was ascertained on 97.4% of the Mexican Americans and 97.8% of the non-Hispanic whites who had participated in the 1979–1982 baseline survey originally. The follow-up examination consisted of a home or telephone interview, followed by a medical examination performed in a mobile clinic located in the participant's neighborhood. Forty-eight Mexican Americans and 29 non-Hispanic whites died before the follow-up interview. Two Mexican Americans and two non-Hispanic whites were ineligible for the interview because of physical or mental disabilities. The follow-up interview was completed by 96.8% of surviving eligible Mexican Americans and 97.1% of surviving eligible non-Hispanic whites. One Mexican American and one non-Hispanic white died after completing the home interview but before completing the medical examination. Two additional Mexican Americans completed the home interview but were then considered ineligible for the medical examination because of physical disabilities. The response rate to the medical examination was 78.9% of Mexican Americans and 73.4% of non-Hispanic whites who completed the home interview. Thus, the overall response rate was 76.4% for Mexican Americans (0.968 × 0.789) and 71.3% for non-Hispanic whites (0.971 × 0.734). This report is restricted to the 867 Mexican Americans and 595 non-Hispanic whites who were free of hypertension at the baseline examination and who attended the medical examination 8 yr later. The methods used for anthropometric and blood pressure measurements at the follow-up examination were identical to those used at the baseline examination. A complete description of the follow-up has been published previously (19).

Differences in clinical characteristics between men and women were evaluated with two-way ANOVA with sex and conversion status to hypertension as grouping variables. Spearman (nonparametric, univariate) correlations were determined separately by sex. We have shown previously that the incidence of hypertension is similar in Mexican Americans and non-Hispanic whites (20). Interactions between ethnicity and sex were also tested in both ANOVA and logistic regression; in each case, the interactions were not statistically significant ($P > 0.20$), suggesting that the effect of independent variables on the incidence of hypertension was not different in the two ethnic groups. Therefore, the ethnic groups have been pooled for ease of presentation and to increase sta-

Table 1—*Baseline demographic and clinical variables by sex and conversion status at 8-yr follow-up*

	Conversion status					
	Women			Men		
	Normal	Hypertensive	*P*	Normal	Hypertensive	*P*
N	768	76		560	58	
Mexican American (%)	59	55	0.551	59	62	0.622
Age (yr)	50.8 ± 0.4	55.7 ± 1.1	<0.001	51.3 ± 0.5	56.0 ± 1.1	0.002
BMI (kg/m^2)	25.3 ± 0.2	27.6 ± 0.5	<0.001	26.6 ± 0.2	27.7 ± 0.7	0.042
Skin fold						
Subscapular	20.8 ± 0.3	25.5 ± 1.0	<0.001	19.1 ± 0.3	20.4 ± 1.1	0.196
Triceps	22.9 ± 0.3	25.7 ± 0.9	0.005	14.3 ± 0.3	14.8 ± 0.9	0.606
Subscapular-triceps skin fold ratio	0.927 ± 0.012	1.039 ± 0.04	0.016	1.43 ± 0.22	1.52 ± 0.08	0.255
Glucose (mM)						
Fasting	5.2 ± 0.1	5.8 ± 0.3	0.001	5.4 ± 0.1	5.6 ± 0.3	0.502
2-h	6.5 ± 0.11	8.4 ± 0.7	<0.001	0.68 ± 0.2	6.70 ± 0.3	0.971
Fasting insulin (pM)	87.8 ± 17.2	76.3 ± 3.6	0.043	89.2 ± 15.6	75.5 ± 5.0	0.071

tistical power. Differences in the incidence of hypertension, according to the level of various risk factors, were tested by χ^2 statistic. Analyses of fasting glucose and insulin concentrations and 2-h glucose concentrations were performed on the log-transformed variables to normalize their distributions, and these variables then were back transformed to their natural units for presentation in the tables. All statistical analyses (except logistic regression analysis) were performed with SYSTAT statistical software (21). The test for trend used a χ^2 test. Logistic regression analyses were performed with the package developed by Dallal (22). Independent variables were considered as categorical variables in Tables 3 and 4 and as continuous variables in Table 5. Odds ratios were calculated for a 10-yr age difference and for a 5-kg/m^2 BMI difference. Odds ratios were calculated by exponentiating the logistic regression coefficients.

RESULTS— Table 1 shows baseline demographic and clinical characteristics of subjects by sex and subsequent conversion status. Women who converted to hypertension were older and had significantly higher BMIs, subscapular and triceps skin folds, ratios of subscapular-triceps skin fold (centrality index), fasting and 2-h glucose, and fasting insulin concentrations at baseline compared with women who did not convert to hypertension. In men, converters to hypertension had higher BMIs and were older than subjects who remained normotensive at follow-up. No other significant differences were noted between converters and nonconverters in men. In neither sex was ethnicity associated with the incidence of hypertension. In table 2, Spearman correlation coefficients are shown separately by sex for possible predictors of hypertension incidence.

Table 3 shows the incidence of hypertension by level of selected variables. In women, age, BMI, subscapular and triceps skin folds, centrality index, glucose tolerance status, fasting and 2-h glucose, and fasting insulin were all significantly related to the incidence of hypertension. In men, only age and fasting insulin were significantly related to the incidence of hypertension. Women had a greater risk of hypertension with worsening glucose tolerance than men. For example, the RR of developing hypertension in NIDDM subjects compared with those with NGT was 2.65 for women and 1.61 for men. Similarly, the RR of developing hypertension in subjects with IGT compared with those with NGT was 1.94 in women and 0.84 in men. In subjects with NGT, men had a higher incidence of hypertension than women (9.3 vs. 7.1%, respectively). However, women had a higher incidence of hypertension in the IGT (13.8 vs. 8.6%, respectively) and in the NIDDM (18.8 vs. 15.0%, respectively) categories.

Table 4 shows the results of multiple logistic regression analyses with incidence of hypertension as the dependent variable. Glucose tolerance is treated as a categorical variable in these analyses. Two different models are presented. Model 1 includes all subjects, and model 2 excludes insulin-taking subjects to be able to add plasma insulin concentrations to the model. In model 1, age, BMI, centrality index, and NIDDM are significantly related to the incidence of hypertension in women, whereas ethnicity and IGT are not. In men, only age and BMI are significantly related to the incidence of hypertension. In model 2, fasting insulin was not significantly re-

Table 2—*Spearman correlation coefficients between metabolic variables*

	Age	Glucose		Fasting insulin
		Fasting	2 h	
Men				
Age	1.000			
Ethnicity	−0.057			
Glucose				
Fasting	0.300*	1.00		
2 h	0.344*	0.429*	1.00	
Fasting insulin	0.118†	0.371*	0.361*	1.00
Skin fold				
Subscapular	0.045	0.239*	0.292*	0.350*
Triceps	−0.070	0.079	0.188*	0.228*
Subscapular-triceps skin fold ratio	0.137*	0.175*	0.097‡	0.091‡
BMI	0.082‡	0.263	0.319	0.413*
Women				
Age	1.000			
Ethnicity	−0.139*			
Glucose				
Fasting	0.314*	1.00		
2 h	0.202*	0.462*	1.00	
Fasting insulin	0.071‡	0.393*	0.298*	1.000
Skin fold				
Subscapular	0.054	0.428*	0.386*	0.496*
Triceps	0.117*	0.338*	0.241*	0.354*
Subscapular-triceps skin fold ratio	−0.030	0.232*	0.273*	0.284*
BMI	0.172*	0.451*	0.379*	0.470*

*$P < 0.001$.
†$P < 0.01$.
‡$P < 0.05$.

lated to the incidence of hypertension in either sex. None of the other independent (or predictor) variables were affected appreciably by the addition of fasting insulin to the model. Restriction of the analyses to only nondiabetic subjects also yielded similar results.

Table 5 shows the results of multiple logistic regression analysis in which fasting and 2-h glucose are treated as continuous variables. In men, neither fasting nor 2-h glucose are related significantly to the incidence of hypertension. The odds ratios for glucose in men is slightly <1.0, suggesting a mildly protective effect. In women, both fasting and 2-h glucose are related positively to the incidence of hypertension, although only the relationship for 2-h glucose is statistically significant. We also fit a pooled model, including men and women with a sex × 2-h glucose interaction term. The interaction term for sex × 2-h glucose was almost statistically significant ($P = 0.051$), suggesting a greater effect of glycemia on the incidence of hypertension in women than in men. None of the other first-order interaction terms involving sex (e.g., sex × BMI) was related significantly to the incidence of hypertension ($P > 0.10$) and therefore were excluded from the final model.

One possible explanation for an apparent greater effect of glycemia on the incidence of hypertension in women than men might be that men with increased glucose concentrations or blood pressure might have a relatively higher mortality than women with these disorders and thus be more likely to be removed before ascertainment at follow-up. In the 8-yr follow-up of the San Antonio Heart Study, the mortality rate was 30.0% for men with diabetes, 10% for men with IGT, and 2.4% for normal, nondiabetic men. The corresponding rates for women were 18.8, 2.8, and 1.7%. (Subjects with hypertension at baseline were excluded from this analyses.)

CONCLUSIONS — We have shown in this report that glycemia has a greater effect on the incidence of hypertension in women than in men. This sex difference does not appear to be caused by other confounding variables, such as overall adiposity, body fat distribution, or insulinemia. Although the incidence of hypertension is somewhat higher in normoglycemic men than in normoglycemic women (Table 3), the opposite is true for subjects with IGT or NIDDM. Thus, as in the case of lipids and lipoproteins (6–8), NIDDM appears to exert a greater adverse effect in women than in men. Our results on hypertension incidence may partially explain the relatively greater excess risk of CHD in diabetic women than in diabetic men (1–3).

We found a somewhat higher mortality in men with increased glucose concentrations than in women with increased glucose concentrations. However, for this increased mortality to explain the lesser effect of glycemia on the hypertension incidence in men, we would have to hypothesize that men who died were also more likely to have developed hypertension before death than women who died. We have no ready way to assess this possibility.

We also found a greater effect of body fat distribution on the incidence of hypertension in women than in men, although the formal test of interaction was

Table 3—*Eight-year incidence of hypertension by selected demographic and clinical variables at baseline*

	Men			Women		
	Cutoff points	*N*	Hypertensive (%)	Cutoff points	*N*	Hypertensive (%)
Age (yr)						
25–34		163	3.1		223	3.6
35–44		155	7.7		246	9.4
45–54		170	16.5		214	8.9
55–64		130	10.0		161	16.2
Total		618	9.4		844	9.0
Test for trend (*p*)			0.002			<0.001
BMI (kg/m^2)						
Low	<24.9	206	7.8	<22.7	281	3.6
Medium	24.9–27.7	206	9.2	22.7–26.3	281	7.8
High	>27.7	206	11.2	>26.3	281	15.7
Test for trend (*p*)			0.083			<0.001
Subscapular skin fold (mm)						
Low	<15	204	9.8	<15.8	281	5.3
Medium	15.0–21.7	204	6.9	15.8–24.3	281	10.3
High	>21.7	205	11.7	>24.3	282	13.1
Test for trend (*p*)			0.513			0.002
Triceps skin fold						
Low	<10.7	205	9.8	<20.0	281	5.3
Medium	10.7–15.7	205	8.8	20.0–25.7	281	8.9
High	>15.7	205	9.8	>25.7	282	12.8
Test for trend (*p*)			0.511			0.012
Subscapular-triceps skin fold ratio						
Low	<1.20	204	9.3	<0.76	281	4.6
Medium	1.20–1.53	205	7.8	0.76–1.06	281	10.7
High	>1.53	205	11.2	>1.06	282	11.7
Test for trend (*p*)			0.511			0.003
Glucose tolerance status						
NGT		461	9.3		635	7.1
IGT		70	8.6		109	13.8
NIDDM		40	15.0		48	18.8
Test for trend (*p*)			0.921			<0.001
Fasting glucose (mm/L)						
Low	<4.9	204	8.3	<4.8	277	5.8
Medium	4.9–5.4	204	9.7	4.8–5.1	278	7.9
High	>5.4	205	11.2	>5.1	278	13.0
Test for trend (*p*)			0.311			0.002
2-h Glucose (mm/L)						
Low	<5.3	190	7.9	<5.3	263	3.8
Medium	5.3–6.7	190	11.6	5.3–6.7	263	9.1
High	>6.7	191	8.9	>6.7	263	13.3
Test for trend (*p*)			0.472			<0.001
Fasting insulin (pm/L)						
Low	<59.0	179	6.2	<52.5	259	6.6
Medium	59.0–98.6	179	9.5	52.5–84.2	260	8.9
High	>98.6	179	13.4	>84.2	260	13.1
Test for trend (*p*)		0.021			0.009	

*p < 0.05; **p < 0.01; ***p < 0.001

Table 4—*Multiple logistic regression analyses for incidence of hypertension*

	Men			Women		
	Odds ratio	95% Confidence interval	*P*	Odds ratio	95% Confidence interval	*P*
Model 1						
Age (10-yr difference)	1.49	1.13–1.96	0.005	1.42	1.10–1.83	0.006
BMI (5-kg/m^2 difference)	1.40	1.00–1.95	0.045	1.36	1.08–1.71	0.008
Subscapular-triceps skin fold ratio (1-U difference)	1.08	0.65–1.79	0.761	2.12	1.08–4.18	0.030
Glucose tolerance test						
NGT	1.00			1.00		
IGT	0.91	0.34–2.38	0.839	1.25	0.79–1.97	0.338
NIDDM	1.59	0.47–5.42	0.456	2.11	1.12–4.09	0.032
Ethnic group (Mexican American/nonHispanic white)	0.81	0.45–1.46	0.482	0.86	0.50–1.51	0.604
Model 2 (excluding insulin-taking diabetic subjects)						
Age (10-yr difference)	1.55	1.16–2.07	0.003	1.44	1.12–1.86	0.004
BMI (5-kg/m^2 difference)	1.30	0.95–1.78	0.099	1.50	1.17–1.92	0.002
Subscapular-triceps skin fold ratio (1-U difference)	0.96	0.53–1.72	0.882	2.47	1.20–5.09	0.014
Glucose tolerance test						
NGT	1.00			1.00		
IGT	0.89	0.31–2.63	0.833	1.30	0.82–2.17	0.301
NIDDM	1.49	0.42–5.62	0.515	2.21	1.17–4.17	0.015
Ethnic group (Mexican American/nonHispanic white)	0.89	0.48–1.66	0.713	0.78	0.45–1.38	0.396
Fasting insulin (7.5 pM)	1.00	0.98–1.03	0.908	0.98	0.95–1.01	0.115

not significant (data not shown, $P = 0.11$). In this population, we previously reported a sex difference in the effect of body fat distribution on the incidence of NIDDM (greater in women) (12).

Interestingly, we did not find a sex difference in the effect of fasting insulin on the incidence of hypertension. In univariate analyses, we found a significant effect of insulin concentrations on the incidence of hypertension in both sexes. These results were similar in both ethnic groups (data not shown). These data are in contrast to the recently published data of Saad et al. (24), who in cross-sectional data observed an effect of insulin resistance on blood pressure in whites but not in blacks or Pima Indians. In our population, no evidence of an ethnic difference was found in the effect of fasting insulin on the incidence of hypertension (25). Laakso et al. (26) found a stronger relation between insulin resistance and hypertension in lean compared with obese subjects. We also found a stronger relationship between fasting insulin concentration and the incidence of hypertension in lean compared with obese subjects (25), and because Mexican Americans, although obese (13), are less obese than Pima Indians, they could still be in the range of adiposity where insulin resistance could play a role in the pathogenesis of hypertension. In multivariate analyses, no significant relationship was observed between fasting insulin concentrations and the incidence of hypertension, suggesting that the insulin–blood pressure relationship in the overall population may be because of other confounding variables such as glucose tolerance, obesity, and/or body fat distribution (Table 5). On the other hand, as mentioned earlier, the association between glucose tolerance, body fat distribution, and incidence of hypertension does not appear to be mediated by changes in fasting insulin concentrations. This stands in contrast to the situation with NIDDM, where the effect of body fat distribution appears to be mediated by hyperinsulinemia (23).

In conclusion, we have shown a greater effect of glycemia and body fat distribution on the incidence of hypertension in women than in men. These effects are not dependent on confounding variables such as overall obesity or hyperinsulinemia. Our results suggest an additional reason, aside from adverse effects on lipids and lipoproteins, why diabetes worsens the cardiovascular risk of women more than men.

Table 5—*Multiple logistic regression analyses for incidence of hypertension with glycemia treated as a continuous variable*

	Men			Women		
	Odds ratio	95% Confidence interval	*P*	Odds ratio	95% Confidence interval	*P*
Model 1 (fasting glucose)						
Age (10-yr difference)	1.47	1.13–1.91	0.005	1.43	1.13–1.82	0.003
Ethnic group (Mexican American/non-Hispanic white)	0.84	0.47–1.49	0.551	0.91	0.53–1.56	0.732
BMI (5-kg/m^2 difference)	1.30	0.95–1.78	0.103	1.33	1.07–1.65	0.010
Subscapular-triceps skin fold ratio (1-U difference)	1.18	0.73–1.90	0.498	2.08	1.08–4.01	0.029
Fasting glucose (0.6 mM/L difference)	0.99	0.89–1.10	0.838	1.05	0.98–1.12	0.153
Model 2 (2-h glucose)						
Age (10-yr difference)	1.50	1.13–1.99	0.004	1.35	1.05–1.73	0.020
Ethnic group (Mexican American/non-Hispanic white)	0.79	0.44–1.43	0.433	0.86	0.49–1.50	0.501
BMI (5-kg/m^2 difference)	1.41	1.02–1.94	0.036	1.34	1.07–1.68	0.010
Subscapular-triceps skin fold ratio (1-U difference)	1.13	0.69–1.85	0.640	2.00	1.02–3.95	0.045
2-h Glucose (0.6 mM/L difference)	0.92	0.79–1.07	0.283	1.05	1.01–1.09	0.008

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