Elevated Incidence of Type 2 Diabetes in San Antonio, Texas, Compared With That of Mexico City, Mexico

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OBJECTIVE — To compare the incidence of type 2 diabetes between low-income Mexican-Americans residing in San Antonio, Texas, and low-income residents in Mexico City, Mexico.

RESEARCH DESIGN AND METHODS — Using data from the San Antonio Heart Study and the Mexico City Diabetes Study, we compared the incidence of type 2 diabetes in 35- to 64-year-old low-income Mexican-American residents of San Antonio with similarly aged low-income residents of Mexico City. Because of the different follow-up times in the two studies, Poisson regression was used to compare the rates of diabetes. Potential risk factors for diabetes were also analyzed to determine whether they explained or contributed to a difference in incidence.

RESULTS — The age- and sex-adjusted incidence of type 2 diabetes was significantly higher in San Antonio (RR 2.01) compared with Mexico City. This difference was seen primarily in the oldest age group (55–64 years of age) and remained statistically significant after adjusting for a number of diabetes risk factors, including demographic, anthropometric, and metabolic variables. Follow-up rates were similar in both cities.

CONCLUSIONS — We conclude that there was a higher incidence of type 2 diabetes in San Antonio than in Mexico City, and that difference occurred primarily in individuals in the oldest age group. The potential mediating factors we examined did not account for this difference. Other factors, such as exercise and diet, which were not available for analysis in this study, in addition to a cohort effect, may have contributed to the difference in incidence of type 2 diabetes in the two cities. In addition, there was no evidence of a higher case fatality among diabetic individuals from Mexico City compared with San Antonio.

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We have previously reported a lower prevalence of type 2 diabetes in Mexico City compared with San Antonio (1). However, it is unknown whether this difference is caused by a higher incidence of diabetes in San Antonio or a higher case fatality in Mexico City, where access to and utilization of health care resources may be lower than in San Antonio. Incidence data are therefore necessary to determine whether rates of incident diabetes are actually higher in San Antonio.

Using data from the San Antonio Heart Study (SAHS) and the Mexico City Diabetes Study, we compared the incidence of type 2 diabetes in the two populations. From the SAHS, we examined low-income Mexican-Americans and compared them to Mexicans living in six low-income colonias in Mexico City. In addition, we examined risk factors that may mediate potential differences in the incidence rates between the two cities.

RESEARCH DESIGN AND METHODS

— The Mexico City Diabetes Study (2) was conducted in six low-income colonias in Mexico City and was designed to determine the incidence of type 2 diabetes in this population. A complete enumeration of the colonias was carried out, and 3,326 study-eligible individuals consisting of men and nonpregnant women 35–64 years of age were identified. Of these, 2,813 (84.5%) completed a home interview, and 2,282 (68.5%) completed a medical examination in a clinic. A follow-up examination was performed an average of 6.3 years later. An interim follow-up examination at 3.1 years was also conducted, but only data from the 6.3-year follow-up was used in the present analyses. A total of 1,754 subjects (76.9%) came to the Mexico City second follow-up.

SAHS is a population-based study of diabetes and cardiovascular disease in Mexican-Americans and non-Hispanic whites. The study initially enrolled 3,301 Mexican-American and 1,857 non-Hispanic white men and nonpregnant women in two phases between 1979 and 1988. Participants were 25–64 years of age at enrollment and were randomly selected from low-, middle-, and high-income neighborhoods in San Antonio, Texas. A 7- to 8-year follow-up to examine the incidence of type 2 diabetes and cardiovascular disease began in 1987 and was completed in the fall of 1996. A total of 3,682 individuals (73.7% of survivors) from the two phases completed the follow-up examination.

Because the Mexico City Diabetes Study involved 35- to 64-year-old individuals living in low-income colonias, the San Antonio data presented in this paper are restricted to Mexican-Americans in the same age range and residing in the

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Abbreviations: IFG, impaired fasting glucose; IGT, impaired glucose tolerance; SAHS, San Antonio Heart Study; STR, subscapular skin fold–to–triceps skin fold ratio.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Table 1—*Incidence of type 2 diabetes* (per 100 person-years) in 6–8 years of follow-up of Mexico City residents and San Antonio Mexican-Americans†*

Age at baseline (years)	Men			Women		
	Mexico City	San Antonio	*P*	Mexico City	San Antonio	*P*
35–44	1.15 (22/295)	1.48 (4/37)	0.660	1.13 (29/402)	1.77 (10/77)	0.220
45–54	1.98 (26/204)	1.93 (6/43)	0.960	1.46 (27/291)	2.77 (18/89)	0.037
55–64	1.07 (7/104)	4.35 (15/47)	0.002	0.96 (9/148)	4.15 (22/73)	<0.001
Total	1.42 (55/603)	2.70 (25/127)	0.008	1.21 (65/841)	2.86 (50/239)	<0.001

Data are incidence (*n*). *1999 World Health Organization criteria; †from Poisson regression offset by the logarithm of the years of observation.

low-income San Antonio barrio neighborhoods (1). Because of the substantial time differences between phase I of the SAHS (enrollment period from 1979 to 1982) and the Mexico City Diabetes Study (enrollment period from 1990 to 1992), only participants from the second phase of the SAHS (enrollment period from 1984 to 1988) are included in the present analyses. This is particularly important because of the increasing trend in the incidence of type 2 diabetes seen in the SAHS population (3). A total of 466 Mexican-Americans from the SAHS who resided in the low-income barrio neighborhoods and who completed both baseline and follow-up clinical examinations are included in the present analyses. The study protocol was approved by the institutional review boards of the Centro de Estudios en Diabetes in Mexico City and the University of Texas Health Science Center at San Antonio, and all subjects gave informed consent.

Descriptions of the survey procedures used at the baseline and follow-up examinations for the SAHS (4–6) and for the Mexico City survey (1,2,4,7) have been previously published. At both sites, height, weight, subscapular and triceps skin folds, waist-hip circumferences, systolic and diastolic blood pressures (random-zero sphygmomanometer; Hawksley-Gelman, London) were measured as previously described (8–11). BMI was calculated as weight (kilograms) divided by height (meters) squared and was used as an index of overall adiposity. Individuals were considered overweight if their BMI was ≥25 kg/m^2 and <30 kg/m^2 and obese if their BMI was ≥30 kgm^2. The ratios of waist to hip circumference and subscapular skin fold–to–triceps skin fold ratio (STR) were used as measures of body fat distribution.

Blood chemistry measurements from both San Antonio and Mexico City were made in San Antonio in the Division of Clinical Epidemiology laboratory. Fasting plasma glucose concentration and fasting serum insulin, total and HDL cholesterol, and triglyceride concentrations were determined as previously described (11). Glucose and insulin concentrations were also measured 2 h after a standardized 75-g oral glucose load (12). In Mexico, serum was stored in a −70°C freezer until it was shipped to San Antonio on dry ice at ~4- to 6-week intervals. Shipments usually arrived in San Antonio within 24 h and in no case later than 48 h.

Subjects were considered to have type 2 diabetes if they met the modified World Health Organization plasma glucose criteria, i.e., fasting glucose ≥7.0 mm/l (126 mg/dl) or 2-h glucose ≥11.1 mm/l (200 mg/dl) (13). Subjects who gave a history of diabetes and who were under treatment with either insulin or oral antidiabetic agents were also considered to have diabetes regardless of plasma glucose levels. Diabetic subjects who were not taking insulin were considered to have type 2 diabetes. Those taking insulin were considered to have type 2 diabetes if they had BMI >27 kg/m^2 and age at onset >30 years.

Statistical analyses were performed using SAS statistical software (14). We used Poisson regression to analyze the data. This approach enabled us to account for the difference in average follow-up times between studies (average 6.3 years for Mexico City vs. 7.5 years for San Antonio) and for the variation in follow-up times within study. To perform these analyses we applied PROC GENMOD in SAS with the relation between the logarithm of the number of events and the logarithm of the person-year incidence offset by the logarithm of the observation time. Analysis of covariance and logistic regression were used to determine statistical differences in baseline means and proportions, respectively. We used stepwise logistic regression to identify variables to include in a multivariate Poisson regression model. A 5 and 10% significance level was used for entrance and retention in the model, respectively.

RESULTS — Table 1 presents the incidence of type 2 diabetes in Mexico City and San Antonio stratified by age and sex. In men, the crude incidence was 90.1% higher in San Antonio than in Mexico City. This difference was statistically significant. There was a particularly elevated risk in San Antonio men 55–64 years of age, who had a fourfold increase compared with those of Mexico City. However, in men 35–44 and 45–54 years of age, there was no statistically significant city difference in incidence of type 2 diabetes.

In women, the crude incidence of type 2 diabetes was more than twofold higher in San Antonio than in Mexico City (Table 1). This difference was also highly statistically significant. In women 55–64 years of age, as in men, there was a fourfold increase in San Antonio compared with Mexico City. In women 45–54 years of age, there was also a significant increased incidence in San Antonio compared with Mexico City. There was no significant difference in women 35–44 years of age.

The age-adjusted baseline type 2 diabetes risk factors in Mexico City and San Antonio residents, stratified by sex, are presented in Table 2. The effect of city within the sexes and the main effect of the city with both sexes combined were tested. A mixed pattern was observed, with some diabetes risk factors more favorable in Mexico City (e.g., BMI, fasting and 2-h glucose, 2-h insulin, hypertension, and impaired glucose tolerance [IGT]) and others more favorable in San Antonio (e.g., fasting insulin, triglycerides, and HDL cholesterol). Significant interactions between city and sex were seen for waist circumference and fasting glucose.

Table 3 presents age-, sex-, and risk factor–adjusted city relative risks of developing type 2 diabetes in San Antonio Mexican-Americans compared with Mexico City residents. The age- and sex-adjusted risk of developing type 2 diabetes

Table 2—*Age-adjusted baseline risk factors in Mexico City residents and San Antonio Mexican-Americans*

Risk factor	Men		Women		*P* for main effect of city
	Mexico City	San Antonio	Mexico City	San Antonio	
n	608	129	850	244	
BMI (kg/m^2)	27.1*	28.6	28.8†	30.1	<0.0001
Waist circumference (cm)§	94.0*	95.6	98.2†	90.0	<0.0001
Fasting glucose (mg/dl)§	84.7	89.0	84.6	86.9	<0.0001
2-hour glucose (mg/dl)	95.9*	109.0	109.3†	117.9	<0.0001
Fasting insulin (μU/L)	14.6	13.7	16.3‡	14.7	0.1558
2-hour insulin (μU/L)	78.5†	86.1	104.3†	125.5	0.0075
Triglycerides (mg/dl)	247.3‡	181.2	178.1†	144.3	<0.0001
HDL cholesterol (mg/dl)	29.9†	42.4	34.7†	46.9	<0.0001
IGT (%)	10.2‡	18.0	13.7†	24.2	<0.0001
IFG (%)	2.3	2.6	2.5	2.7	0.78
Hypertension (%)‖	11.6‡	19.6	11.2	15.00	0.0040

*$P < 0.01$ for city comparison within sex; †$P < 0.0001$ for city comparison within sex; ‡$P < 0.05$ for city comparison within sex; §significant interaction; ‖systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or taking antihypertensive medications.

in San Antonio relative to Mexico City was 2.01, which was highly statistically significant. The relative risk remained statistically significant after adjusting for each risk factor individually. There were significant city–risk factor interactions for waist circumference and BMI, which was a significant predictor in San Antonio but not in Mexico City. The multivariate model is presented in Table 4. City, along with BMI, fasting glucose, 2-h glucose, and HDL cholesterol, were entered into the model and were thus independent predictors of the development of type 2 diabetes.

Figure 1 presents the type 2 diabetes incidence per 100 person-years stratified by city, age, and overweight/obesity. Because of the small numbers, it is difficult to analyze each category separately. However, some distinct patterns emerge. In San Antonio residents, the incidence of type 2 diabetes increased consistently by age group, as expected. However, such a pattern was not seen in Mexico City residents, where the incidence in the oldest group (aged 55–64 years) was not higher than that of the younger age groups. In general, the incidence of type 2 diabetes increased as weight increased from normal to overweight to obese, as expected. However, an exception appeared in men and women aged 55–64 years from Mexico City, where the incidence remained similar among the three weight categories.

The return–follow-up rates by city, age, and diabetes/IGT status at baseline are presented in Fig. 2. In individuals who had diabetes at baseline, there was no significant difference in return rate between the two cities. In individuals with IGT or impaired fasting glucose (IFG) at baseline, there was also no overall significant difference in return rate between the two cities. However, San Antonio residents aged 35–44 years were less likely to attend follow-up, and San Antonio residents age 55–64 were more likely to attend follow-up than Mexico City residents in the corresponding age groups. Overall, San Antonio residents with normal glucose tolerance were less likely than Mexico City residents to attend follow-up, but this appeared to be caused entirely by differences in the youngest age group.

CONCLUSIONS — This study examined the incidence of type 2 diabetes in low-income Mexican-Americans living in San Antonio and low-income Mexicans living in Mexico City. The incidence of type 2 diabetes was significantly higher in residents of San Antonio than those of Mexico City. This difference, seen primarily in the oldest age group, was present in both sexes and remained statistically significant after controlling for numerous risk factors for type 2 diabetes. There were significant differences in baseline risk factors between the two populations, some favoring Mexico City and some favoring San Antonio. Adjusting for the former attenuated the excess incidence in San Antonio, and adjusting for the latter widened it. In neither case, however, did adjustment for these differences eliminate the significant association between city and incidence of type 2 diabetes. Thus, there

Table 3—*Age-, sex-, and risk factor–adjusted risk ratio of developing type 2 diabetes in San Antonio Mexican-Americans compared with Mexico City residents*

	RR	*P*	95% CI
San Antonio versus Mexico City	2.01	<0.0001	1.49–2.69
BMI (5 kg/m^2)*	1.49	<0.0001	1.32–1.67
San Antonio versus Mexico City	1.72	0.0004	1.27–2.32
Waist circumference (15 cm)*	1.42	<0.0001	1.28–1.56
San Antonio versus Mexico City	2.14	<0.0001	1.59–2.87
Fasting glucose (15 mg/dl)	2.28	<0.0001	1.92–2.69
San Antonio versus Mexico City	1.78	0.0001	1.32–2.40
2-h glucose (50 mg/dl)	3.15	<0.0001	2.57–3.87
San Antonio versus Mexico City	1.53	0.0059	1.13–2.07
Fasting insulin (10 μU/l)	1.12	0.0002	1.05–1.19
San Antonio versus Mexico City	2.08	<0.0001	1.54–2.80
Triglycerides (80 mg/dl)	1.14	<0.0001	1.08–1.20
San Antonio versus Mexico City	2.20	<0.0001	1.62–2.97
HDL cholesterol (15 mg/dl)*	0.60	<0.0001	0.48–0.76
San Antonio versus Mexico City	2.93	<0.0001	2.08–4.09
IGT	4.49	<0.0001	3.35–6.01
San Antonio versus Mexico City	1.65	0.0012	1.22–2.24
IFG	3.51	<0.0001	2.02–5.66
San Antonio versus Mexico City	2.01	<0.0001	1.49–2.70

*$P < 0.05$ for city–risk factor interaction.

Table 4—*Multivariate ORs and 95% CIs*

	OR	95% CI
San Antonio versus Mexico City	1.79	(1.24–2.55)
Male versus female	1.25	(0.91–1.71)
Age 10 years	1.08	(0.90–1.30)
BMI 5 kg/m^2	1.28	(1.11–1.47)
Fasting glucose (15 mg/dl)	1.60	(1.31–1.95)
2-h glucose (50 mg/dl)	2.29	(1.82–2.89)
HDL cholesterol (15 mg/dl)	0.76	(0.59–0.97)

Risk factors included in the multivariate model, along with the demographic variables (age and sex), were selected using stepwise logistic regression with significance criteria set at 5% to enter and 10% to stay. To account for differences in observation time, the SAS Proc Logistic events/trials syntax was used with diabetes incidence for the number of events and observation time for the number of trials. Once selected, the variables were included in a Poisson regression model as previously explained.

was no specific explanation for the difference in incidence of type 2 diabetes between the two cities.

A possible explanation for the difference in incidence of type 2 diabetes may be differences in environmental factors, such as diet and physical activity. In a previous study (4), we examined the differences in diets in samples from Mexico City and San Antonio. Major differences in fat and carbohydrate intake as a percent of total kilocalories were observed. Mexico City residents consumed ~18–21% of calories from fat and 68–72% from carbohydrates compared with 29–33% from fat and 48–52% from carbohydrate for Mexican-Americans from San Antonio. Unfortunately, data were obtained only for a subset of SAHS participants, and thus we are unable to use them as covariates in our analyses.

The higher triglyceride levels in Mexico City compared with San Antonio were not unexpected, because as shown above, carbohydrate intake is higher in Mexico City (4), and international data indicate an inverse association between carbohydrate intake and average triglyceride levels in various populations (15). High carbohydrate intakes are typical of underdeveloped societies, where diabetes rates are typically low, so it is interesting that in the present study, triglycerides emerged as a positive risk factor for diabetes not only in San Antonio but also in Mexico City.

A potential confounding factor in these analyses could be a difference in genetic susceptibility to type 2 diabetes between the two populations. In our previous prevalence study of these two populations (1), we inferred genetic susceptibility to type 2 diabetes from the percentage of Native American genetic admixture, as estimated from skin reflectance measurements. Genetic susceptibility to type 2 diabetes was estimated to be similar in the two groups. Therefore, this is an unlikely explanation for differences in incidence rates between the two populations.

It is noteworthy that the risk of developing type 2 diabetes in the oldest Mexico City residents did not increase as weight category increased from normal to overweight to obese. One explanation could be the lower return rate in the oldest Mexico City age group with IFG or IGT compared with that of San Antonio, perhaps reflecting a higher case fatality in overweight and obese individuals in this age

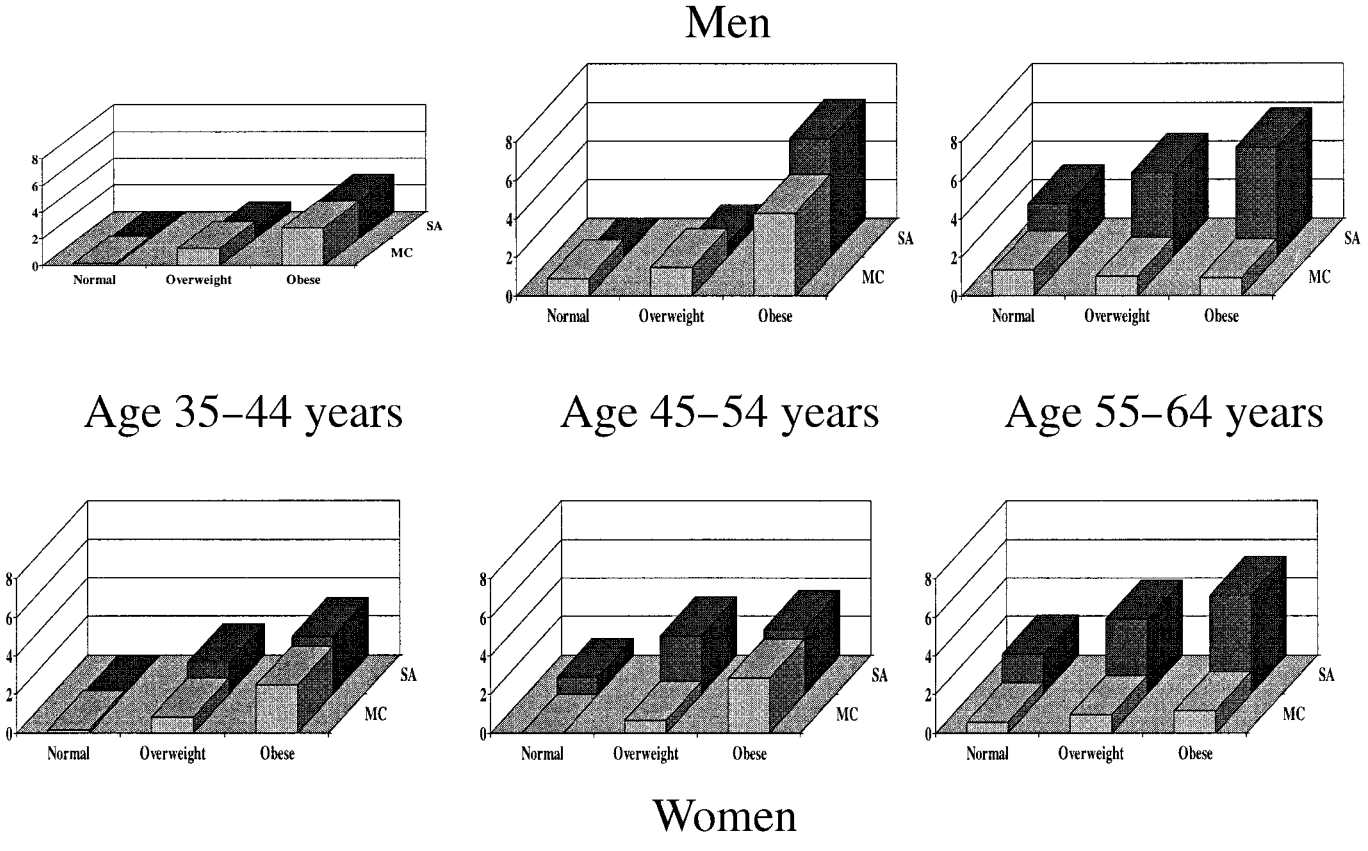

Figure 1—*Incidence of diabetes per 100 person-years. MC, Mexico City; SA, San Antonio.*

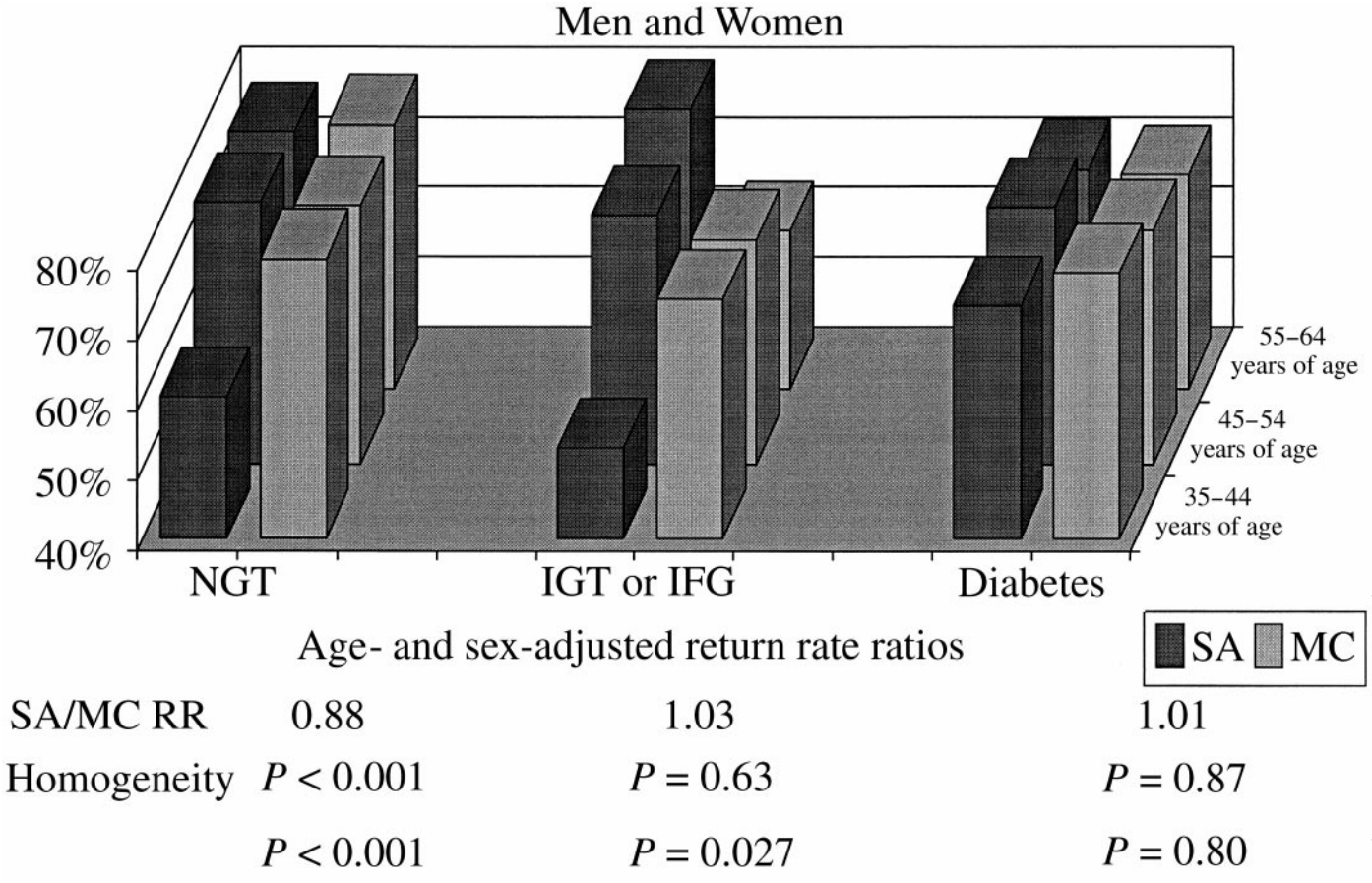

Figure 2—*Percent return to follow-up by diabetes/IFG/IGT status.*

group who develop diabetes in Mexico City, where access to and utilization of health care resources may be lower than in San Antonio.

However, it is interesting to note that there was no significant city difference in return rates among those with diabetes at baseline. This suggests that a higher case fatality rate among individuals with diabetes in Mexico City is not a likely explanation for the markedly lower diabetes incidence in the oldest age group in Mexico City. A possible explanation for this difference could be a cohort effect, i.e., Mexico appears to be in the midst of a transition from a traditional to a "Westernized culture," primarily affecting younger individuals, with the oldest individuals having lived most of their lives before this transition. This would explain the significantly lower incidence of diabetes in the oldest age group of Mexico City compared with that of San Antonio, whereas the differences in the younger age groups were much less marked. On the other hand, it is possible that the lower return rates in 66- to 65-year-old Mexico City residents with IGT or IFG could have led to an underestimation of diabetes incidence in this subgroup.

In conclusion, we have found a significantly increased incidence of type 2 diabetes in low-income Mexican-American residents of San Antonio, Texas, compared with low-income residents of Mexico City. This difference was seen primarily in the oldest age group and remained statistically significant after adjusting for a variety of diabetes risk factors. A number of risk factors for type 2 diabetes, particularly diet and physical activity, were only available for a subset of the SAHS population and thus could not be included as covariates. It is possible that these factors, along with a cohort effect, may explain part of this difference.

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