Determinants of Fasting Glucose in Young Guatemalan Adults

Andrea J. Conlisk, MPH; Aryeh D. Stein, PhD, MPH; Dirk G. Schroeder, ScD, MPH; Benjamin Torun, MD, PhD; Ruben Grajeda, MD; Reynaldo Martorell, PhD

Objective: To examine correlates of fasting glucose, a precursor for type 2 diabetes mellitus, in young adults in Guatemala, a country undergoing an epidemiologic transition.

Design: Cross-sectional.

Methods: Anthropometric, lifestyle, dietary, serum lipid, and socioeconomic characteristic data were collected on 189 men and 201 women (mean age 24.4 years) born in four villages in Eastern Guatemala. We used linear regression to identify parsimonious predictive models, including 2-way interactions.

Results: In men, mean fasting glucose was 87.3 mg/dL (SD 10.2); our model explained 30% of variance. Body mass index (BMI), abdomen-to-hip ratio (AHR), and total cholesterol showed graded positive effects. BMI and AHR interacted ($P<.001$); men with high BMI and high AHR had the highest fasting glucose levels. No dietary factors independently predicted fasting glucose. In women, mean fasting glucose was 83.9 mg/dL (SD 8.5); 22% of variance was explained by BMI, energy-adjusted fat intake, physical activity, birth village, and current residence (rural/urban). BMI and fasting glucose were positively related. Urban residence interacted with birth village ($P=.06$) and physical activity ($P=.13$).

Conclusions: The major conclusion drawn from this study is that increased adiposity, even among lean individuals, is the largest environmental predictor of fasting glucose. Prevention and control of obesity in young adults in transitioning countries are key strategies for the prevention of diabetes. (*Ethn Dis.* 2001;11:585–597)

Key Words: Blood Glucose, Guatemala, Risk Factors, Obesity

Introduction

Non-insulin dependent diabetes mellitus (type 2 DM) is a disease generally thought to afflict individuals with a sedentary lifestyle characterized by obesity and a high-fat diet, as these are considered the principal environmental determinants of diabetes.[1] Until recently, type 2 DM was considered to be a problem only in industrialized nations; however, recent reports indicate that its prevalence is increasing rapidly in the developing world, particularly in countries undergoing rapid industrialization.[2,3] The prevalence of type 2 DM is predicted to increase by 48% from 1995 to 2025, in developing countries, compared to 27% in industrialized nations.[2] Developing countries are experiencing a considerable excess of diabetes in urban areas and people are developing the disease at younger ages than in developed countries, with the prevalence highest among those aged 45 to 64 years.[2]

From the Department of International Health, Rollins School of Public Health (AJC, ADS, DGS, RM); Biological and Biomedical Sciences Division, Nutrition and Health Sciences Program, Emory University, Atlanta, Georgia, USA (AJC); Instituto de Nutricion de Centro America y Panama (INCAP), Guatemala City, Guatemala (BT, RG)

Address correspondence and reprint requests to Andrea J. Conlisk, MPH; Department of International Health; Rollins School of Public Health; 1518 Clifton Rd. NE; Atlanta, GA 30322; 404-727-9713; 404-727-4590 (fax); aconlis@emory.edu

Hyperglycemia and clinical diabetes predispose to microvascular complications, neurological impairments, and cardiovascular disease,[1] so the earlier development of diabetes is likely to lead to earlier onset of these complications. This increased disease burden will have major implications for medical costs and loss of human capital in developing countries.

Studies examining the determinants of type 2 DM have concentrated primarily on industrialized populations with a high prevalence of risk factors, particularly obesity. Differences in the prevalence of diabetes between industrialized and developing countries may result from a differential distribution of risk factors, or from variability in gene-environment interactions across ethnic groups.[3] Since environmental and behavioral factors may be modifiable, identification of the specific risk factors operating in specific populations should be a priority. Our goal was to assess correlates of fasting glucose in young men and women in Guatemala, a Latin American country currently undergoing economic development and urban migration.

Research design and methods

Study population

Individuals interviewed for this study were participants in a longitudinal study on growth and development conducted by the Institute of Nutrition of Central America and Panama (INCAP) in four Ladino (ie, Spanish-speaking) villages of eastern Guatemala between 1969 and 1977. A detailed description of the original study and subsequent follow-up surveys appear elsewhere.[4] During 1998–1999, data were collected on anthropometry, levels of blood glucose and lipids, dietary and lifestyle habits, and sociodemographic characteristics. Participants eligible for this follow-up study were born between 1969 and 1977, had recorded birth weight, and had at least one year of growth monitoring in childhood. Participants also had to reside, or be available for interview, in one of the four original study villages or in Guatemala City. Of 762 eligible cohort members, 473 (62%) subjects were examined (237 men and 236 women). Reasons for non-examination included death (N = 3), relocation to a distant or unknown place (N = 151), not being at home on multiple visits (N = 36), serious handicaps or chronic illness (N = 4), women pregnant or nursing babies <6 months old (N = 25), and refusal to participate in the study (N = 70).

Data collection

Levels of glucose, total cholesterol, and HDL-cholesterol were determined by solid-phase enzymatic reactions (Cholestech LDX System, Hayward, California). The Cholestech system is a compact, portable analyzer that provides fast results using a small sample of capillary blood from a finger prick.[7] Lipid measurements were calibrated against Emory University's Lipid Research Laboratory using 55 venous blood samples that were obtained at the time of the finger prick.[8] Participants were instructed to fast overnight before morning blood analysis. Duration of fasting was determined by asking participants the time they consumed their last meal, dessert, unsweetened coffee or tea, and fruit juice or sweetened beverage, and subtracting the most recent time of food consumption from the time of the blood analysis.

Standardized anthropometry was used to measure height; weight; arm, natural waist (smallest), abdomen (umbilical), and hip circumferences; and triceps and subscapular skinfolds. Measurements were taken in triplicate with weighing scales that were calibrated periodically, measuring tapes, and Holtein-Harpenden skinfold calipers. All measurements were taken standing.[9] Body mass index (BMI), waist-to-hip ratio (WHR), and abdomen-to-hip ratio (AHR) were then calculated. Percent body fat was also estimated using predictive equations

from hydrostatic weighing in a similar population (M. Ramirez, INCAP, unpublished data). BMI or percent body fat was used to estimate overall obesity, while central obesity was estimated by either WHR or AHR.

Diet was assessed using a semi-quantitative food frequency questionnaire that was developed by INCAP nutritionists. The questionnaire asked for information about food consumption over the past three months, including portion size, and incorporated foods known to be available in the study villages and Guatemala City. The INCAP food composition database,[10] supplemented where necessary with databases from the region and the United States Department of Agriculture,[11] was used to estimate the average daily intake of carbohydrates, fat, fiber, alcohol, β-carotene, vitamin E, and energy.

Customary physical activity level (PAL) during the past 12 months was estimated using a detailed semi-quantitative questionnaire developed for the study.[12] The questionnaire asked about patterns, duration, and intensity of activities at home, work, and leisure. Average daily energy expenditure was calculated from the time spent in each activity and from predictive equations of basal metabolism.[13] Men were classified as having very light, light, or moderate/heavy habitual activity when their estimated PAL was, respectively, <1.48, 1.48–1.65, >1.65 metabolic equivalents (METS).[14] The corresponding PAL categories for women were <1.48, 1.48–1.59, >1.59 METS.

Other relevant covariates were ascertained by standardized interview. Smoking habit was categorized as current or non-current. Current place of residence was characterized as rural or urban.

Exclusions

Participants who had not fasted for at least eight hours before blood draw (men: $N = 10$, women: $N = 8$) or who were missing data on any independent variable (men: $N = 15$, women: $N = 20$) were excluded from the analysis. We also excluded participants (men: $N = 20$, women: $N = 7$) who had unreliable dietary data defined as an estimated daily energy intake >5500 kcal or an individual food (other than tortillas, the local staple food) accounting for more than 25% of total energy intake. The final population for analysis consisted of 189 men and 201 women.

Statistical methods

Men and women were analyzed separately. We compared values between the study population and those excluded from analysis. Energy-adjusted intakes of carbohydrates, fat, and fiber were computed as the residuals from a regression model with total energy intake as the independent variable and absolute nutrient intake as the dependent variable.[15] Vitamins A and E were log transformed to increase normality. Few women reported cigarette ($N = 2$) or alcohol ($N = 4$) use; therefore, these variables were not used in the analysis of women. Three categories of alcohol intake were defined for men: nondrinkers, those consuming less than or equal to 3.5 grams per day (the median alcohol intake of drinkers), and those consuming greater than 3.5 grams per day.

We used linear regression models to evaluate the relationship between fasting glucose and five classes of predictor variables: anthropometry; lifestyle; diet; lipids; and sociodemographic characteristics. To build the most parsimonious and predictive model, we first examined the available predictor variables within each class. Sequentially adding variables, and their centered interactions, to linear models, we used the extra sum of squares approach[16] to choose the combination of the fewest predictor variables that explained the greatest amount of fasting glucose variability. We declared the observed partial F-test significant at $P<.05$ for main effects. For interaction terms, $P<.15$ was considered significant. A non-

Table 1.—Selected characteristics of young Guatemalan adults participating in the 1998–99 INCAP cardiovascular disease follow-up study

	Men			Women		
	Complete N = 189	N	Missing	Complete N = 201	N	Missing
Glucose (mg/dL)	87.3 ± 10.2	20	87.2 ± 6.9	83.9 ± 8.5	19	94.9 ± 43.1
Age (yrs)	24.5 ± 2.4	48	24.1 ± 2.4	24.3 ± 2.2	35	24.4 ± 2.4
Anthropometry						
Weight (kg)	59.8 ± 7.6	41	60.8 ± 9.8	53.8 ± 9.8	30	58.5 ± 12.4*
Height (cm)	164.4 ± 6.7	41	165.0 ± 5.6	151.8 ± 5.3	30	151.8 ± 4.2
BMI (kg/m²)	22.1 ± 2.4	41	22.3 ± 2.7	23.3 ± 3.9	30	25.4 ± 5.1*
Body fat (%)	14.2 ± 5.6	41	13.2 ± 5.8	29.8 ± 6.2	18	30.3 ± 5.1
Waist/hip ratio	0.87 ± 0.04	41	0.87 ± 0.03	0.80 ± 0.05	26	0.84 ± 0.05*
Abdomen/hip ratio	0.90 ± 0.04	41	0.90 ± 0.05	0.94 ± 0.05	26	0.97 ± 0.04
Lifestyle						
Physical activity (METS/d)	1.63 ± 0.28	18	1.64 ± 0.27	1.51 ± 0.10	35	1.55 ± 0.07
Alcohol (g)	4.2 ± 10.3	28	2.6 ± 5.3	0.03 ± 0.30	28	0.02 ± 0.08
Current smokers (%)	41.1	48	37.5	0.5	35	2.86
Daily dietary intake						
Kilocalories	3153 ± 870	28	3184 ± 1041	2133 ± 570	28	2276 ± 527*
Fat (g)	74.2 ± 26.6	28	74.8 ± 32.1	55.9 ± 19.7	28	61.6 ± 19.6
Carbohydrate (g)	585.4 ± 156.3	28	596.5 ± 183.9	426.2 ± 103.6	28	439.8 ± 104.4
Total fiber intake (g)	17.3 ± 16.0	28	48.0 ± 18.0	34.3 ± 9.8	28	35.2 ± 9.9
β-carotene (µg)	2228 ± 3139	28	1684 ± 1180	1433 ± 1375	28	1059 ± 924
Vitamin E (mg)	4.1 ± 3.2	28	5.2 ± 6.2	3.2 ± 3.0	28	3.1 ± 2.8
Copper	2.5 ± 0.8	28	2.4 ± 0.8	1.8 ± 0.5	28	1.9 ± 0.8
Lipid Profile						
Total cholesterol (mg/dL)	158.8 ± 28.2	20	148.9 ± 28.6	169.7 ± 29.9	19	184.9 ± 46.8*
HDL (mg/dL)	37.1 ± 7.6	20	38.0 ± 6.5	41.8 ± 10.2	18	38.8 ± 8.2
Current residence						
Migrated to urban areas (%)	34	48	25	19	35	11

predictive variable was also retained if adding it to the model altered the effect of a predictive variable by more than 20%. After determining the most predictive variables within each class, we used the same procedure to combine classes and develop a final model. We used regression diagnostics, including examination of residuals and influential points, to test model assumptions. Statistical analyses were conducted with the SPSS program for Windows (version 8.0; SPSS Inc, Chicago, Illinois).

Results

Characteristics of the study participants are summarized in Table 1. Men excluded from the analysis did not differ from our analytic population on any tested variable. Means for fasting glucose, total cholesterol, and BMI were greater among women excluded from the analysis relative to those included in the analysis; however, this was due to a previously diagnosed diabetic woman, who was excluded from subsequent analysis due to missing covariate data. Other than this woman, there were no persons with a fasting glucose diagnostic of diabetes (fasting glucose ≥126 mg/dL).[17] Although, 3% of men and 1% of women were considered to have impaired fasting glucose (110 mg/dL≤fasting glucose <126 mg/dL). Obesity (BMI≥30 kg/m^2) was present in 1% of men and 8% of women. An additional 9% of men and 18% of women were classified as overweight (25 kg/m^2≤BMI<30 kg/m^2). Central obesity (WHR greater than 0.90 for men and 0.85 for women) was present in 22% of men and 15% of women. For both men and women, 20% of energy intake was from fat. Only 44% of men and 17% of women were considered moderately or heavily active. Among men, 52% drank alcohol, but the quantity consumed was low. Total cholesterol was greater than 200 mg/dL among 8% of men and 16% of women.

Table 1.—Continued

	Men				Women			
	Complete N = 189	N	Missing		Complete N = 201	N	Missing	
Village of birth								
Santo Domingo (13)	20		21*		25		29	
Comacastc (31)	29		31		31		31	
Espiritu Santo (40)	23		40		26		20	
San Juan (16)	28		8		18		20	

X ± SD unless otherwise specified

* P value for difference between the study and excluded populations < .05

Analysis of independent variables by variable class

Bivariate associations between fasting glucose and the independent variables are presented in Table 2. Further analysis of each variable class was conducted both with and without age adjustment. Adjustment for age did not significantly change the results (data not shown).

Anthropometry

Among men, BMI, percent body fat, WHR, and AHR were each independently and positively predictive of fasting glucose. An interaction term between measures of obesity and central obesity significantly added to the explained variance of fasting glucose ($\Delta R^2 = 0.06$; $P<.001$). When modeled together, BMI, AHR, and the BMI-AHR interaction explained the greatest variance ($R^2 = 0.10$) compared to all other combinations of anthropometric measures (data not shown). To investigate this interaction, we categorized men by AHR tertiles (Figure 1). BMI was negatively associated with fasting glucose among men in the lowest AHR tertile ($\beta = -1.67$ mg/dL per kg/m^2; 95% confidence interval [CI] −3.00, −0.33) and positively associated with fasting glucose in the highest tertile ($\beta = 1.24$ mg/dL per kg/m^2; 95% CI 0.24, 2.21). No association was found between BMI and fasting glucose among men in the middle AHR tertile ($\beta = 0.89$ mg/dL per kg/m^2; 95% CI −0.89, 1.73).

In women, both BMI and percent body fat predicted fasting glucose ($R^2 = 0.05$ for each). To maintain comparability with men, BMI was chosen to estimate obesity. Fasting glucose level increased 0.50 mg/dL per kg/m^2 increase in BMI (95% CI 0.20, 0.79). Measures of central obesity (WHR or AHR) did not contribute significantly to the prediction of fasting glucose, either alone or in a model containing BMI.

Lifestyle

Among men, physical activity, alcohol, and smoking did not explain variance in fasting glucose, whether modeled alone or as a group. Among women, physical activity did not significantly predict variance in fasting glucose in any model. Few women reported smoking or consuming alcohol.

Lipids

Fasting glucose increased by 0.07 mg/dL per mg/dL increase in total cholesterol among men (95% CI 0.02, 0.12), accounting for four percent of fasting glucose variance. HDL was borderline predictive when modeled alone ($R^2 = 0.02$, $P = .06$), but was no longer significant when added to a model containing total cholesterol. When total cholesterol and HDL were entered as a ratio (total cholesterol/HDL), lipids were no longer associated with fasting glucose. Neither total cholesterol, HDL, nor the total cholesterol/HDL ratio was associated with fasting glucose in women.

Diet

Energy-adjusted fat and carbohydrates intakes were highly inversely correlated in both men and women ($r = -0.89$ and -0.95, respectively). To avoid collinearity, we tested models using only one macronutrient variable at a time. Among men, the interaction between energy-adjusted fat and β-carotene intake was statistically significant ($R^2 = 0.03$, $P = .06$). Graphical analysis of the relationship between fasting glucose, energy-adjusted fat intake, and β-carotene intake suggested that this relationship was complex (not shown), and characterization of this relationship was not explored further. Among men, no other dietary variables explained variance in fasting glucose. Among women, only energy-adjusted fat explained variance in fasting glucose ($R^2 = 0.02$, $P = 0.05$). However, both energy intake and energy-adjusted fiber intake confounded the relationship between energy-adjusted fat intake and fasting glucose, and, hence, were retained in the model.

Sociodemographic characteristics

Among men, neither village of birth nor current residence was predictive of fasting

Table 2.—Bivariate regression coefficients for independent variables and fasting glucose among young Guatemalan adults participating in the 1998–99 INCAP cardiovascular disease follow-up study

	Men			Women		
	β	95% Confidence Interval		β	95% Confidence Interval	
Independent Variable	(mg/dL)	Lower	Upper	(mg/dL)	Lower	Upper
Age (yr)	0.45	−0.16	1.06	0.71	−0.42	0.64
Anthropometry						
BMI (kg/m²)	0.70*	0.08	1.31	0.50†	0.20	0.79
Body fat (%)	0.30*	0.04	0.56	0.32†	0.14	0.51
Waist/hip ratio (WHR)	41.82*	6.64	76.99	3.30	−20.37	26.96
Abdomen/hip ratio (AHR)	46.06*	6.86	85.27	10.72	−13.82	35.25
Lifestyle						
Physical activity (METS/d)	−1.13	−6.44	4.19	0.37	−11.46	12.20
Alcohol intake						
Non-drinker	0	referent category		‡		
1–3.5 g/day	0.57	−2.96	4.10			
>3.5 g/day	−1.30	−4.50	1.89			
Smoking history						
Current nonsmoker	0	referent category		‡		
Smoker	0.57	−2.42	3.55			
Diet						
Kilocalories/day	0.001	−0.001	0.003	0.002	0.000	0.004
Fat (adjusted for kcal) (g/day)	−0.01	−0.10	0.08	0.09*	0.001	0.19
Fiber (adjusted for kcal) (g/day)	−0.01	−0.16	0.13	−0.15	0.32	0.02
β-carotene (log μg/d)	0.64	−1.85	0.56	0.80	−0.26	1.86
Vitamin E (log mg/d)	−0.08	−2.06	1.91	0.35	−1.31	2.01
Lipids						
Cholesterol (mg/dL)	0.07†	0.02	0.12	0.02	−0.02	0.06
HDL (mg/dL)	0.19	−0.01	0.38	−0.06	−0.18	0.05
Sociodemographic characteristics						
Village of birth						
Santo Domingo	0	referent category		0	referent category	
Conacaste	1.45	−1.77	4.68	0.29	−2.26	2.84
Espiritu Santo	−2.43	−5.89	1.03	1.50	−1.19	4.20
San Juan	−2.43	−5.69	0.84	−5.12	−8.12	−2.12
Current residence						
Rural	0	referent category		0	referent category	
Urban	−1.09	−4.18	2.01	−0.34	−3.33	2.65

All models unadjusted: fasting glucose = independent variable.

* $P<.05$.

† $P<.01$.

‡ Not applicable as very few women smoked or consumed alcohol.

glucose when modeled alone or together. Among women, village of birth significantly added to explained variance (R^2 = 0.06, P = .008), while current residence did not. However, an interaction between village of birth and current residence was evident (P = .06). In analyses stratifying by village, only women born in Santo Domingo had a mean fasting glucose that differed between those currently living in a rural (86.31 mg/

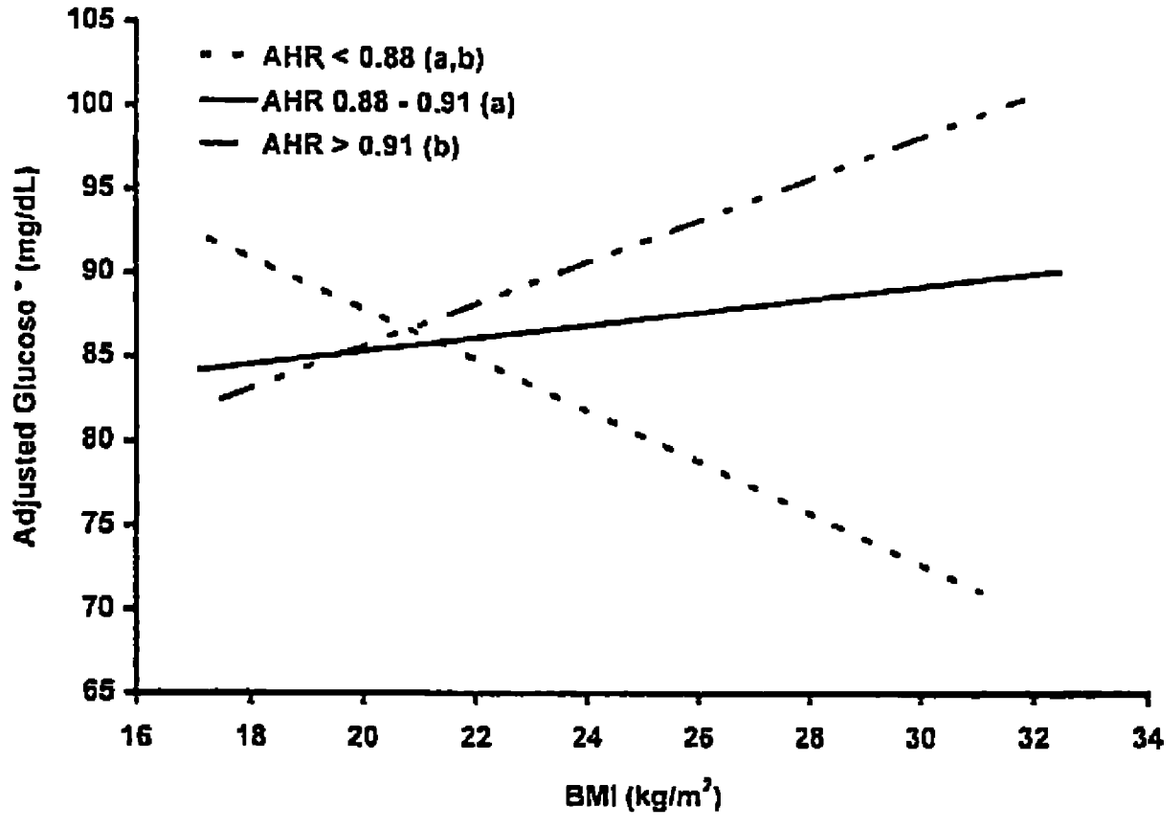

Fig 1.—The relationship between BMI (kg/m²) and fasting glucose (mg/dL) stratified by abdomen-to-hip ratio tertiles among young Guatemalan men. *Adjusted for total cholesterol and alcohol intake. Pairwise comparison $P<.05$ for abdomen-hip ratio tertiles with the same letter.

dL, SEM = 1.23) or urban (78.00 mg/dL, SEM = 3.69) environment.

Interactions between variable classes

Among men, village of birth interacted with HDL ($P = .003$). HDL was positively related to fasting glucose in both San Juan ($\beta = 0.60$ mg/dL; 95% CI 0.23, 0.98) and Santo Domingo ($\beta = 0.27$ mg/dL; 95% CI −0.22, 0.76), negatively related to fasting glucose in Espiritu Santo ($\beta = -0.84$ mg/dL; 95% CI −1.70, 0.02) and had no relationship to fasting glucose in Conacaste ($\beta = -0.10$ mg/dL; 95% CI −0.39, 0.19).

Among women, village of birth interacted with energy-adjusted fat intake ($P = .13$) and fasting glucose. Energy-adjusted fat intake was positively associated with fasting glucose in Santo Domingo ($\beta = 0.36$, 95% CI 0.03, 0.68) and Espiritu Santo ($\beta = 0.20$ mg/dL, 95% CI 0.01, 0.40) and negatively associated with fasting glucose in San Juan ($\beta = -0.32$ mg/dL, 95% CI −0.52, −0.12). No relationship was found in Conacaste ($\beta = -0.08$ mg/dL, 95% CI −0.36, 0.20).

Current residence interacted with physical activity among women ($P = .13$). Among women currently living in a rural environment, fasting glucose increased by 8.24 mg/dL for each METS/day of physical activity (95% CI −6.18, 22.65). In contrast, fasting glucose among women currently living in an urban setting was found to decrease 16.50 mg/dL per METS/day of physical activity (95% CI −35.11, 2.10).

Final models

All independent variables within each variable class that either significantly explained variance of fasting glucose, or confounded the relationship between fasting glucose and another predictive variable were entered as a group into a model (Table 3, reduced model). All other previously rejected variables were then entered individually into this model to test for confounding or additional explained variance. Among men, alcohol intake confounded the association between AHR and fasting glucose. Alcohol intake was negatively associated

Table 3.—Reduced and full models for prediction of fasting glucose among young Guatemalan men

	Reduced Model R^2=0.30			Full Model R^2=0.31		
	β (mg/dL)	95% Confidence Interval		β (mg/dL)	95% Confidence Interval	
		Lower	Upper		Lower	Upper
Constant	84.90	73.26	96.55	82.26	62.52	102.00
BMI centered (kg/m²)	0.05	−0.63	0.74	−0.02	−0.77	0.73
Abdomen/hip ratio centered (AHR)	30.34	−10.26	70.94	31.12	−12.32	74.56
BMI AHR	25.27	13.12	37.42	26.39	13.69	39.08
Alcohol intake						
Non-drinker		referent category			referent category	
1–3.5 g/day	0.00	−3.55	3.55	−0.29	−4.00	3.42
>3.5 g/day	−4.38	−7.70	−1.07	−4.52	−8.09	−0.94
Total cholesterol (mg/dL)	0.06	0.01	0.11	0.06	0.01	0.12
HDL centered (mg/dL)	0.33	−0.09	0.75	0.34	−0.09	0.77
Kilocalories/day	0.002	0.0004	0.004	0.002	0.0001	0.004
Energy-adjusted fiber (g/d)	0.10	−0.04	0.23	0.15	−0.04	0.33
β-carotene (log μg/d)	−1.30	−2.49	−0.12	−1.58	−2.92	−0.25
Village of birth						
Santo Domingo		referent category			referent category	
Conacaste	−3.45	−7.48	0.58	−3.52	−7.71	0.68
Espiritu Santo	−7.41	−11.96	2.87	−7.47	−12.21	−2.73
San Juan	−5.98	−10.00	1.96	−6.01	−10.32	−1.70
Village of birth HDL						
Santo Domingo HDL		referent category			referent category	
Conacaste HDL	−0.38	−0.87	0.11	−0.42	−0.92	0.09
Espiritu Santo HDL	−0.92	−1.69	−0.16	−0.94	−1.72	−0.16
San Juan HDL	0.27	−0.28	0.82	0.24	−0.32	0.81
Age (y)				0.14	−0.45	0.73
Physical activity (METS/d)				0.28	−5.56	6.13
Current smoker				0.76	−2.22	3.74
Energy-adjusted fat (g/d)				0.05	−0.07	0.16
Vitamin E (log mg/d)				0.26	−1.96	2.48
Urban residence				0.17	−3.25	3.60

with fasting glucose, with the lowest fasting glucose found among those consuming more than 3.5 g/day. Energy (R^2 = 0.02, P = .05) and β-carotene intake (R^2 = 0.02, P = .03) were found to explain additional variance after adjusting for other covariates. The association between β-carotene intake and fasting glucose was confounded by both energy and energy-adjusted fiber intake. The final model among men included BMI, AHR, alcohol intake, total cholesterol, HDL, birth village, energy, energy-adjusted fiber and β-carotene intake, and two interaction terms (between BMI and AHR, and HDL and birth village, respectively) explaining 30% of fasting glucose variance (Table 3, reduced model). Inclusion of all other *a priori* independent variables explained no additional fasting glucose variance (P = .94) (Table 3, full model).

Among women, 21% of fasting glucose variance was explained by BMI, physical activity, energy intake, energy-adjusted fat and fiber intake, birth village, current residence and three interaction terms, namely birth village and energy-adjusted fat intake, birth village and current residence, and current residence and physical activity level (Table 4, reduced model). An additional 3% of variance (P = .41) was explained by in-

Table 4.—Reduced and full models for prediction of fasting glucose among young Guatemalan women

	Reduced Model R^2=0.21			Full Model R^2=0.24		
	β (mg/dL)	95% Confidence Interval		β (mg/dL)	95% Confidence Interval	
		Lower	Upper		Lower	Upper
Constant	72.56	64.34	80.79	83.94	59.28	108.60
BMI centered (kg/m²)	0.45	0.16	0.74	0.45	0.09	0.80
Physical activity (METS/d)	9.03	−4.40	22.46	13.03	−1.09	27.10
Kilocalories/day	0.002	0.000	0.004	0.001	−0.001	0.004
Energy-adjusted fat centered (g/d)	0.12	−0.12	0.36	0.09	−0.16	0.34
Energy-adjusted fiber (g/d)	−0.12	−0.37	0.13	−0.16	−0.42	0.11
Village of birth						
Santo Domingo		referent category			referent category	
Conacaste	−4.65	−8.02	−1.28	−4.55	−8.29	−0.81
Espiritu Santo	−3.62	−7.29	0.06	−3.21	−7.05	0.62
San Juan	−8.17	−12.27	−4.07	−9.04	−13.35	−4.73
Urban residence	−10.85	−18.38	−3.32	−11.30	−18.90	−3.69
Village of birth·energy-adj fat						
Santo Domingo fat		referent category			referent category	
Conacaste fat	−0.09	−0.35	0.17	−0.08	−0.34	0.19
Espiritu Santo fat	0.07	−0.21	0.35	0.08	−0.20	0.36
San Juan fat	−0.24	−0.51	0.04	−0.27	−0.56	0.01
Village of birth Urban residence						
Santo Domingo urban		referent category			referent category	
Conacaste urban	14.08	5.05	23.11	14.21	5.06	23.36
Espiritu Santo urban	8.21	−1.14	17.57	8.13	−1.31	17.58
San Juan urban	12.54	2.93	22.15	13.61	3.89	23.32
Urban residence physical activity	−25.24	−51.37	0.88	−25.61	−52.40	1.19
Age (y)				−0.18	−0.72	0.36
Waist/hip ratio				−16.60	−45.54	12.33
Total cholesterol (mg/dL)				0.04	−0.001	0.09
HDL centered (mg/dL)				−0.08	−0.20	0.05
β-carotene (log ug/d)				0.52	−0.71	1.75
Vitamin E (log mg/d)				0.22	−1.75	2.19

clusion of all other tested variables (Table 4, full model).

Our results suggest that a shift from the 75th to the 25th percentile in obesity (men and women) and central obesity (men) is associated with a 3.2 mg/dL (men) and a 2.4 mg/dL (women) lower fasting glucose. A shift from the 75th to the 25th percentile for all other modifiable risk factors (men: intake of energy and β-carotene, total cholesterol and HDL: women: fat intake) is associated with an additional decrease of 3.9 mg/dL among men and 1.6 mg/dL among women.

Discussion

A considerable rise in diabetes prevalence over the next 25 years is projected in developing countries.[2] Prolonged life expectancy and improved economic development with urban migration are the primary forces driving this diabetes epidemic. As a region, Latin America is expected to have a substantial increase in diabetes prevalence, with a marked excess among urban residents and women.[2]

Guatemala is currently undergoing rapid economic development and rural to urban

migration.[12] Economic development and urban migration are associated with increased consumption of dietary fats and sugars and decreased physical activity,[12] factors which may promote the development of obesity and, hence, diabetes. Although the majority of men and women in our study still resided in a rural environment, overweight and obesity were common among women, central obesity was common among men, and the majority of men and women engaged only in light physical activity. There were no undiagnosed cases of diabetes in the young men and women in our study. Impaired fasting glucose levels were present, however, though not yet at a prevalence comparable to that among young Mexican Americans in the United States.[18]

In our analysis of fasting glucose correlates, we found obesity and central obesity (men only) to be the most important predictors of fasting glucose. Obesity, particularly central obesity, has long been recognized as a risk factor for diabetes in industrialized countries.[19] In the United States, one in five adults who are at least 25 percent over their ideal body weight has an elevated fasting glucose,[20] and more than one third of adult diabetics are obese.[21] Additionally, there is a greater prevalence of central obesity in those with diabetes or impaired glucose tolerance than in those with normal glucose levels, especially among men.[21] While the young Guatemalan men and women in our study were considered lean relative to US populations,[22] we found obesity and central obesity to be the largest environmental influence on fasting glucose variance. BMI and AHR were positively associated with fasting glucose, and men who had both high BMI and AHR showed the highest fasting glucose. BMI was also positively related to fasting glucose among women. However, unlike other reports,[21,23] we found that central obesity was not an independent predictor of fasting glucose among women.

Independent of their associations with obesity, dietary fat intake and physical inactivity have been shown to influence the development of diabetes.[1] Dietary fat intake in our study population was considerably lower than usual fat intakes found in US populations.[24] However, similar to other studies supporting an association between fasting glucose and fat intake,[25,26] we found energy-adjusted dietary fat intake to be a significant predictor of fasting glucose among women. The association between fasting glucose and physical activity was present only among women and was dependent upon current place of residence. It is unclear why physical activity would be associated with an increase in fasting glucose among women living in a rural environment. In the context of a rural setting, increased physical activity may be indicative of a lower socioeconomic status which may coincide with other risk factors for disease. The negative relationship between physical activity and fasting glucose found among women in urban settings was considerably stronger and consistent with other studies.[1]

Fasting glucose level was inversely associated with β-carotene intake among men in our study. Other observational studies have reported positive associations between glucose tolerance and β-carotene intake,[27] or β-carotene serum concentration.[28,29] However, in a randomized, double-blind, placebo controlled clinical β-carotene supplementation trial among male physicians, incidence of type 2 DM did not differ between treatment groups.[30] While our study supports a relationship between β-carotene and type 2 DM, it is possible that other compounds present in β-carotene rich foods may play a role.

Village of birth was not only a significant predictor of fasting glucose among women, but also modified the association between fasting glucose and energy-adjusted fat intake or current residence among women, and HDL among men. The relationship between village of birth and fasting glucose may be a marker for genetic differences be-

tween villages, or for unmeasured environmental influences operating at a village level. Further exploration into village-level differences may be warranted.

To our knowledge, no other study has assessed the correlates of fasting glucose in young men and women living in Guatemala. Identifying risk factors for hyperglycemia is especially important for Latin American countries, as diabetes prevalence in Latin America is expected to increase 48% from 1995 to 2025.[2] We were able to assess multiple socioeconomic, behavioral, and clinical risk factors known to be associated with the development of diabetes.

This was, however, a cross-sectional study and causal links between these risk factors and fasting glucose cannot be established. Our study is also limited by the use of a measure of fasting blood glucose level that has not, to our knowledge, been validated. It is possible that the measures of fasting glucose are imprecise or biased. While these limitations may result in an unreliable population mean for fasting glucose, any associations observed are still valid and our results may, in fact, underestimate the true strength of the associations observed. Although our analytical approach was based on empirical evidence, the variables we chose to test were a subset of possible variables thought to be physiologically relevant *a priori*. Our approach did not allow intra-class variables to change after combination with variables from other classes. However, this was only relevant to the variable class of anthropometry where we used more than one variable to represent obesity and central obesity. Additionally, fasting glucose measures may not be the best indicator of pre-clinical disease. Oral glucose tolerance tests may be more predictive of disease development than are fasting glucose,[17] but were not feasible in the context of this study.

The emerging epidemic of type 2 DM in developing countries[1] necessitates the identification of the predictors of fasting glucose operating in populations which are at increased risk for developing diabetes, in order to promote early glucose management before the onset of clinical disease. Only about one quarter of fasting glucose variance could be explained in the young men and women of this Guatemalan population. This is similar to reports on other populations in developed countries,[23,31,32] and suggests that glucose metabolism is tightly regulated biologically. Nevertheless, we have identified several factors known to increase risk for developing diabetes that may be amenable to change and, thus, may contribute to lower fasting glucose concentrations. Most importantly, our study reinforces the need to prevent the onset of obesity.

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