Disparities in prediabetes and type 2 diabetes prevalence between indigenous and nonindigenous populations from Southeastern Mexico: The Comitan Study

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ABSTRACT

Aims: In this research we assessed the prevalence of prediabetes and type 2 diabetes and its association with social determinants such as indigenous origin and residence area in population from Comitan, Chiapas, Mexico.
Methods: The Comitan Study is a population-based study carried out from 2010 to 2012 that included 1844 participants aged ≥ 20 years, 880 indigenous and 964 nonindigenous participants. Ethnicity was ascertained by self-report and speaking an indigenous language was also recorded. Prediabetes was defined as fasting serum glucose 5.6–6.9 mmol/l or 2-hour post load serum glucose 7.8–11.0 mmol/l. Type 2 diabetes was defined as fasting serum glucose ≥ 7.0 mmol/l or 2-h post load serum glucose ≥ 11.1 mmol/l or previous clinical diagnosis.
Results: Age-sex-adjusted prevalence of prediabetes and type 2 diabetes was 18.0% (95%CI 15.3–20.6) and 11.0% (95%CI 8.9–131.1) in nonindigenous and 10.6% (95%CI 8.4–12.7) and 4.7% (95%CI 3.3–6.1) in indigenous individuals, respectively. After stratifying by ethnicity, in both indigenous and nonindigenous participants the probability of prediabetes and type 2 diabetes increased with age and BMI. In both indigenous and nonindigenous participants the probability of type 2 diabetes was lower in those living in rural compared with urban areas.
Conclusions: The prevalence of prediabetes and type 2 diabetes was significantly lower in indigenous than in nonindigenous participants. Also, the prevalence of type 2 diabetes was lower in those living in rural areas. Health benefits of a traditional lifestyle may partially account for these differences.

Introduction

The prevalence of type 2 diabetes has increased worldwide over the past three decades and the disease will affect roughly half a billion people by 2030 [1,2]. Ethnic differences have an influence in the prevalence and incidence of type 2 diabetes, perhaps stemming from genetic variations and environmental risk factors. Some studies suggest that disadvantaged groups, including many indigenous populations, are at particularly high risk for diabetes owing to their lower socioeconomic status (SES), lower education level, and lack of accessible health care [3–5]. The increased risk of obesity and diabetes in indigenous populations may be explained in part by the acculturation

process that occurs inside or outside their communities. In general, the degree of acculturation is related to the level of modernization, which has an influence on daily activities, including a more sedentary lifestyle and access to poor quality food and soft drinks [6,7]. Whether this is the case regarding indigenous populations of Mexico remains to be determined.

In Mexico, diabetes has become more common at younger ages, which is due in part to the adoption of obesogenic lifestyles. Nationwide, the prevalence of type 2 diabetes in individuals 20 years and older was estimated at 6.7%, 7.5%, and 14.4% in 1993, 2000, and 2006, respectively [8–10]. Few studies have specifically determined the prevalence of the disease in Mexican indigenous population, which ranges from 2% to 9% across indigenous groups in individuals 20 years and older [11–13]. Likewise, data about the prevalence of prediabetes in indigenous people are scarce, and the estimates range from 14.6% to 21% [14,15]. This study aimed to assess the prevalence of prediabetes and type 2 diabetes and its association with social determinants, such as indigenous origin and residence area, in population from the Municipality of Comitan de Dominguez, in the State of Chiapas, Mexico. We hypothesized that a less urbanized environment is a protective factor for indigenous individuals in relation to lower probability of prediabetes and type 2 diabetes.

Materials and Methods

Study population

The Comitan Study is a population-based investigation carried out from 2010 to 2012 to determine the prevalence of type 2 diabetes, cardiovascular diseases, nephropathy, and associated risk factors among indigenous and nonindigenous adult population from the Municipality of Comitan de Dominguez, in the Mexican State of Chiapas, which is among the four poorest states in Mexico, and is the second state with the highest proportion of indigenous population. A census was conducted in 5 rural and 3 urban areas chosen at random and by convenience sampling, respectively. From the rural areas, all eligible indigenous individuals were invited to participate, whereas urban nonindigenous persons were chosen randomly. Indigenous origin was ascertained by self-report. Indigenous individuals speaking either only Spanish or both their native language and Spanish (bilinguals) were identified. Participants who considered themselves as nonindigenous constitute a group of population, the largest in Mexico, known as mestizos (of mixed racial parentage, mainly of indigenous and European origin) [16]. The degree of community marginalization was categorized in accordance with Mexico's National Population Council (CONAPO) criteria as low, medium, high, and highest degree depending on the level of education, type of dwelling, and economic wealth. Rural communities were defined following Mexico's National Institute of Statistics and Geography (INEGI) criteria as those with 2499 inhabitants or less [17].

The research evaluation included questionnaires on medical history, dietary information (semi-quantitative Food Frequency Questionnaire, FFQ, designed with the methodology of Willett et al.), and physical activity (International Physical Activity Questionnaire, IPAQ, short version). A physical examination and laboratory tests were done as well. Height, weight, and waist circumference were measured with the participants wearing lightweight clothing and no shoes. Body mass index (BMI) was calculated as weight divided by the square of height in kg/m^2. Systolic blood pressure (SBP) was measured at the first and diastolic blood pressure (DBP) at the fourth Korotkoff sound in the right arm three times after a 5-minute rest, while the individual was seated. Values for the last two readings were averaged. Hypertension was defined as SBP ≥ 140 mmHg, DBP ≥ 90 mmHg, or use of anti-hypertensive medicine, regardless of blood pressure values.

Fasting and 2 h venous blood samples were collected, centrifugated and stored until analysis. An oral glucose tolerance test was performed

after at least 8 h of overnight fast and a venous blood sample was obtained before and 2 h after a 75-g oral glucose load. Prediabetes was defined according to the Expert Committee on Diagnosis and Classification of Diabetes Mellitus 1997 and 2003 definitions as fasting serum glucose concentration from 5.6 to 6.9 mmol/l (100 to 125 mg/dl) or 2-hour post load serum glucose concentration from 7.8 to 11.0 mmol/l (140 to 199 mg/dl). Type 2 diabetes was defined as fasting serum glucose concentration ≥ 7.0 mmol/l (≥126 mg/dl) or 2-hour post load serum glucose concentration ≥ 11.1 mmol/l (≥200 mg/dl) or previous clinical diagnosis determined by self-report in individuals taking hypoglycemic medication. Glucose concentrations were measured by ion-selective potentiometry. Total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol, creatinine, and albumin concentrations were measured by spectrophotometry. All analytical measurements were performed in the Central Laboratory at the National Institute of Medical Sciences and Nutrition, Salvador Zubiran in Mexico City, Mexico. The study was performed in accordance with the STROBE for cross-sectional studies and was conducted in accordance with the guidelines of the Declarations of Helsinki. The Institutional Review Boards of Research, Ethics and Biosecurity of The National Institute of Public Health in Mexico approved the study protocol. All participants gave written informed consent.

Statistical analysis

Comparisons of different risk factors between indigenous and nonindigenous individuals were performed using χ^2, Student's *t* test, or the Wilcoxon rank-sum test, when appropriate. The main variables included in the analysis were level of education (< elementary vs. ≥ elementary), area of residence (rural vs. urban), degree of marginalization (high/highest vs. medium), degree of social inequality (high/highest vs. low/medium), BMI, waist circumference, physical activity (inactive/low vs. moderate/high physical activity), calorie intake, SBP and DBP, and lipids profile. Age- and age-sex-adjusted prevalence of prediabetes and type 2 diabetes and their 95% confidence intervals (CI) in indigenous and nonindigenous individuals were estimated by multiple logistic regression analysis. The association between prediabetes and type 2 diabetes and indigenous origin was examined by multiple multinomial regression analysis, calculating odds ratios (OR) and their 95%CI using the nonindigenous population as the reference group. First order interaction between indigenous origin and family history of diabetes, residence area, BMI, and physical activity was evaluated by the log-likelihood ratio test. The model fits were tested by Hosmer-Lemeshow goodness of fit, evaluation of outliers, and influence statistics. All analyses were performed using STATA/MP 15.1 (Stata Corporation, College Station, TX, USA.).

Results

Description of the study population

Of 2949 potential participants (1393 indigenous and 1556 nonindigenous) 1,940 individuals aged ≥ 20 years at the time of interview were examined (response rate 74% for indigenous and 58% for nonindigenous individuals) and 1844 had complete information for this analysis (response rate 93% for indigenous and 97% for nonindigenous individuals). The main reason for nonparticipation of indigenous persons was working for 6 months a year in other states of the country, whereas nonindigenous individuals had no time because of self-employment. 880 individuals included in this study were of indigenous origin (mainly from the Tojolabal ethnic group); 47.1% were bilingual. On the other hand, 964 persons were of nonindigenous origin. No differences by age, sex, and education level were observed between the individuals that participated in the study and those that did not participate. In a comparison between indigenous and nonindigenous participants, a great proportion of indigenous participants lived in rural

Table 1
Social determinants and clinical variables of indigenous and nonindigenous populations of Comitan, Chiapas.

	Indigenous population n = 880	Nonindigenous population n = 964	p value[*]
Age, years (mean, s.d.)	43.1 (16.3)	41.7 (14.3)	0.050
Sex (women), no (%)	527 (59.9)	683 (70.9)	< 0.001
Education level, no (%)			
None	337 (38.3)	225 (23.3)	< 0.001
Elementary school	414 (47.0)	505 (52.4)	
Secondary school or higher	129 (14.7)	234 (24.3)	
Occupation, no (%)			
Housewife	481 (54.7)	534 (55.4)	< 0.001
Farmer	257 (29.2)	132 (13.7)	
Other	142 (16.1)	298 (30.9)	
Speaking an indigenous language, no (%)	414 (47.1)	–	–
Area of residence, no (%)	484 (53.6)		
Urban	213 (24.2)	717 (74.4)	< 0.001
Rural	667 (75.8)	247 (25.6)	
Degree of marginalization, no (%)			
Medium	201 (22.8)	559 (58.0)	< 0.001
High/Highest	679 (77.2)	405 (42.0)	
Degree of social inequality, no (%)			
Low/medium	434 (49.3)	789 (81.8)	< 0.001
High/highest	446 (50.7)	175 (18.2)	
Family history of diabetes, no (%)	87 (9.9)	175 (18.2)	< 0.001
Smoking, no (%)			
Nonsmoker	621 (70.6)	749 (77.7)	< 0.001
Former smoker	119 (13.5)	124 (12.9)	
Current smoker	140 (15.9)	91 (9.4)	
Alcohol consumption, no (%)			
Nonconsumer	408 (46.4)	469 (48.7)	0.090
Former consumer	129 (14.7)	164 (17.0)	
Current consumer	343 (38.9)	331 (34.3)	
Total energy, kcal (mean, s.d.)	1829.6 (497.0)	1827.1 (492.8)	0.915
Protein calorie intake, g (mean, s.d.)	75.8 (18.5)	77.5 (18.5)	0.053
Fat calorie intake, g (mean, s.d.)	51.8 (18.5)	58.9 (18.9)	< 0.001
Carbohydrates calorie intake, g (mean, s.d.)	270.0 (83.6)	250.6 (79.7)	< 0.001
Physical activity, no (%)			
Inactive/low	125 (14.2)	201 (20.9)	< 0.001
Moderate	393 (44.7)	517 (53.6)	
Vigorous	362 (41.1)	246 (25.5)	
Hypertension, no (%)	154 (17.5)	210 (21.8)	0.021
SBP (mmHg), (mean, s.d.)	113.6 (13.4)	117.6 (15.9)	< 0.0001
DBP (mmHg), (mean, s.d.)	74.9 (9.0)	76.6 (9.5)	0.0001
Waist circumference (cm) (mean, s.d.)			
Women	88.5 (10.3)	91.0 (11.2)	0.0001
Men	86.1 (8.7)	90.0 (10.6)	< 0.0001
Body mass index (kg/m^2)	25.5 (4.0)	27.3 (4.8)	< 0.0001
Body mass index (kg/m^2)			
Normal weight	449 (51.3)	347 (36.4)	< 0.001
Overweight	315 (36.0)	359 (37.6)	
Obesity	111 (12.7)	248 (26.0)	
Total cholesterol, mmol/l (mean, s.d.)	4.02 (1.08)	4.23 (1.15)	< 0.0001
HDL-cholesterol, mmol/l (mean, s.d.)			
Women	0.85 (0.33)	0.95 (0.31)	< 0.0001
Men	0.88 (0.36)	0.92 (0.28)	0.155
Triglycerides, mmol/l (median, RIQ)	1.87 (1.34–2.70)	1.80 (1.32–2.64)	0.186

Missing values: total calories, protein, fat, and carbohydrate calories for 6 indigenous and 15 nonindigenous participants; SBP and DBP for 3 indigenous and 7 nonindigenous participants; waist circumference for 4 indigenous and 7 nonindigenous participants; BMI for 5 indigenous and 10 nonindigenous participants; total cholesterol, HDL-cholesterol, and triglycerides for 3 indigenous and 8 nonindigenous participants.

[*] χ^2 test for proportion comparisons was used. *T* student and Wilcoxon test for mean and median comparisons, respectively, were used. All values in parentheses are percentages, except when noted.

areas (75.8% vs. 25.6%) in conditions of high marginalization (77.2% vs. 42.0%), high social inequality (50.7% vs. 18.2%), and had lower level of education (38.3% vs. 23.3%). On the other hand, indigenous participants were leaner compared with nonindigenous participants (Table 1).

Additional comparisons were made by dividing the indigenous participants into 2 groups: Spanish-only speakers and bilinguals, living in urban and rural areas. Those of both groups living in rural areas had lower BMI and fat intake and significantly higher carbohydrate intake compared with their counterparts from urban areas. Bilinguals had lower BMI and fat intake and higher carbohydrate intake compared with Spanish-only speakers for both areas. Differences by physical activity were also observed. For both groups, a higher amount of METs (mainly related to work activities) was expended in rural than in urban areas.

Of 290 participants in the study with prediabetes, 13.4% were diagnosed by both fasting and 2-hour serum glucose; 52.4% only by fasting serum glucose, and 34.2% only by 2-hour serum glucose. Of 170 participants with type 2 diabetes, 65.3% had previous medical diagnosis and 12.4% were detected at the research examination by both fasting and 2-hour serum glucose; 11.7% only by fasting serum glucose, and 10.6% only by 2-hour serum glucose.

Prevalence of prediabetes and type 2 diabetes

The age-sex-adjusted prevalence of prediabetes (11.0%, 95%CI 8.9–13.1) was significantly lower (p < 0.001) in indigenous than in nonindigenous individuals (18.0%, 95%CI 15.3–20.6). In addition, the age-sex-adjusted prevalence was lower in indigenous (8.9%, 95%CI 6.6–11.1) than in nonindigenous (15.5%, 95%CI 10.7–20.2) participants living in rural areas (p = 0.010), but showed no difference in indigenous compared with nonindigenous individuals of urban areas. The age-sex-adjusted prevalence was lower in indigenous (10.3%, 95%CI 8.0–12.5) than in nonindigenous (17.0%, 95%CI 14.1–19.8) participants without family history of diabetes (p < 0.010), but showed no difference in indigenous compared with nonindigenous individuals with family history of diabetes. (Table 2)

The age-sex-adjusted prevalence of type 2 diabetes was significantly lower (p < 0.001) in indigenous (4.7%, 95%CI 3.3–6.1) than in nonindigenous individuals (10.6%, 95%CI 8.4–12.7). The age-sex-adjusted prevalence was lower in indigenous (3.6%, 95%CI 2.3–4.9) than in nonindigenous (9.0%, 95%CI 7.0–11.2) participants without family history of diabetes (p < 0.001), but showed no difference in indigenous compared with nonindigenous individuals without family history of diabetes. (Table 3)

Risk factors associated with the prevalence of prediabetes and type 2 diabetes

After adjustment for age, sex, indigenous origin, residence area, family history of diabetes, BMI, physical activity, and triglyceride levels, a significant interaction occurred between indigenous origin and family history of diabetes for type 2 diabetes but not for prediabetes. Indigenous participants had lower probability of prediabetes (OR = 0.65, 95%CI 0.46–0.91, p = 0.012) compared with nonindigenous participants. As for type 2 diabetes, in absence of family history of diabetes the probability was lower in indigenous (OR = 0.58, 95%CI 0.36–0.94, p = 0.028) compared with nonindigenous participants, whereas with family history of diabetes the probability was higher in indigenous (OR = 3.10, 95%CI 1.28–7.47, p = 0.028)

Table 2
Adjusted prevalence (%) of prediabetes in indigenous and nonindigenous populations of Comitan, Chiapas.

	Indigenous population N = 880 Prevalence (95% CI)	Nonindigenous population N = 964 Prevalence (95% CI)	Indigenous vs. nonindigenous population	
			Ratio (95% CI)	P value
Total	11.0 (8.9–13.1)	18.0 (15.3–20.6)	0.61 (0.47–0.75)	< 0.001
Sex†				
Women	11.7 (8.9–14.5)	20.3 (17.2–23.5)	0.57 (0.41–0.73)	< 0.001
Men	11.0 (7.6–14.3)	15.4 (11.1–19.7)	0.71 (0.42–0.99)	0.102
Family history of diabetes				
Yes	19.4 (10.3–28.6)	24.3 (16.8–31.8)	0.80 (0.37–1.22)	0.392
No	10.3 (8.0–12.5)	17.0 (14.1–19.8)	0.60 (0.46–0.76)	< 0.001
Education level				
Elementary school or higher	12.1 (9.2–15.0)	20.6 (17.4–23.8)	0.58 (0.42–0.75)	< 0.001
None	7.4 (4.2–10.6)	9.3 (5.1–13.5)	0.79 (0.45–1.14)	0.326
Area of residence				
Urban	16.7 (11.5–21.8)	19.1 (15.9–22.4)	0.87 (0.58–1.16)	0.398
Rural	8.9 (6.6–11.1)	15.5 (10.7–20.2)	0.57 (0.35–0.79)	0.010
Degree of marginalization				
Medium	16.9 (11.6–22.3)	21.0 (17.2–24.8)	0.80 (0.52–1.09)	0.209
High/highest	9.0 (6.8–11.3)	14.6 (11.0–18.2)	0.62 (0.41–0.82)	0.006
Degree of social inequality				
Low/medium	14.4 (11.0–17.8)	19.1 (16.0–22.2)	0.75 (0.55–0.95)	0.031
High/highest	7.3 (4.8–9.8)	11.9 (7.0–16.9)	0.61 (0.29–0.92)	0.091
Body mass index				
Normal	7.8 (5.2–10.4)	7.6 (4.7–10.6)	1.02 (0.53–1.51)	0.924
Overweight	10.5 (7.0–13.9)	19.8 (15.3–24.2)	0.53 (0.32–0.73)	< 0.001
Obesity	27.3 (18.4–36.2)	32.3 (25.1–39.6)	0.84 (0.54–1.14)	0.332
Physical activity				
Vigorous	11.1 (7.8–14.4)	18.6 (13.6–23.7)	0.59 (0.35–0.83)	0.013
Moderate	11.0 (7.7–14.2)	16.1 (12.5–19.8)	0.67 (0.45–0.90)	0.020
Inactive/low	8.7 (3.6–13.7)	17.9 (11.7–24.0)	0.48 (0.19–0.77)	0.008

Logistic regression models were used to estimate prevalence and its 95% CI.
[*]Age-sex-adjusted prevalence.
[†]Age-adjusted prevalence.
Missing values: BMI for 5 indigenous and 10 nonindigenous participants.

Table 3
Age- and sex-adjusted prevalence of type 2 diabetes in indigenous and nonindigenous populations of Comitan, Chiapas.

	Indigenous population N = 880 Prevalence (95% CI)	Nonindigenous population N = 964 Prevalence (95% CI)	Indigenous vs. nonindigenous population	
			Ratio (95% CI)	P value
Total	4.7 (3.3–6.1)	10.6 (8.4–12.7)	0.44 (0.29–0.59)	< 0.001
Sex†				
Women	5.8 (3.8–7.8)	11.5 (9.0–14.0)	0.50 (0.31–0.70)	< 0.001
Men	3.5 (1.6–5.5)	10.4 (6.8–14.1)	0.34 (0.13–0.54)	< 0.001
Family history of diabetes				
Yes	17.4 (0.8–26.3)	18.1 (11.3–24.9)	0.95 (0.40–1.52)	0.888
No	3.6 (2.3–4.9)	9.0 (7.0–11.2)	0.39 (0.24–0.55)	< 0.001
Level of education				
Elementary school or higher	4.5 (2.7–6.4)	10.3 (7.8–12.8)	0.44 (0.24–0.64)	< 0.001
None	6.3 (3.4–9.3)	12.8 (7.4–18.1)	0.49 (0.25–0.74)	0.011
Area of residence				
Urban	10.3 (6.3–14.4)	12.7 (9.9–15.5)	0.81 (0.48–1.14)	0.300
Rural	3.0 (1.7–4.4)	4.0 (1.5–6.5)	0.75 (0.21–1.30)	0.479
Degree of marginalization				
Medium	11.0 (6.7–15.4)	14.5 (11.1–17.8)	0.76 (0.44–1.08)	0.177
High/highest	2.9 (1.6–4.2)	5.6 (3.3–8.0)	0.51 (0.23–0.79)	0.028
Degree of social inequality				
Low/medium	8.5 (5.8–11.2)	12.5 (9.9–15.1)	0.68 (0.44–0.91)	0.020
High/highest	1.2 (0.1–2.2)	2.8 (0.3–5.3)	0.41 (0.12–1.20)	0.199
Body mass index				
Normal	1.7 (0.5–2.8)	6.4 (3.7–9.2)	0.26 (0.06–0.45)	0.001
Overweight	5.1 (2.7–7.6)	11.4 (7.8–15.0)	0.45 (0.22–0.68)	0.001
Obesity	16.2 (8.9–23.6)	14.2 (8.9–19.6)	1.14 (0.55–1.72)	0.622
Physical activity				
Vigorous	3.9 (1.9–5.8)	9.5 (5.7–13.3)	0.40 (0.15–0.66)	0.007
Moderate	4.7 (2.6–6.8)	7.8 (5.2–10.4)	0.60 (0.31–0.89)	0.037
Inactive/low	6.4 (2.0–10.8)	18.4 (12.3–24.5)	0.34 (0.10–0.59)	< 0.001

Logistic regression models were used to estimate prevalence and its 95%CI.
[*]Age-sex-adjusted prevalence.
[†]Age-adjusted prevalence.
Missing values: BMI for 5 indigenous and 10 nonindigenous participants

compared with nonindigenous participants. No significant interaction with BMI and residence area was found.

After adjustment for the same variables mentioned above, prediabetes was associated with family history of diabetes among indigenous participants (OR = 2, 95%CI 1.04–3.85, p = 0.038) but not among nonindigenous individuals. Prediabetes was also associated with BMI in both indigenous (OR = 1.13, 95%CI 1.07–1.19, p < 0.001) and nonindigenous (OR = 1.14, 95%CI 1.09–1.18, p < 0.001) participants. As for type 2 diabetes, a stronger association with family history of diabetes was noted in indigenous (OR = 5.71, 95%CI 2.74–11.89, p < 0.001) than in nonindigenous (OR = 2.43, 95%CI 1.44–4.09, p = 001) participants. Also, the probability of type 2 diabetes was lower in both indigenous (OR = 0.31, 95%CI 0.16–0.60, p < 0.001) and nonindigenous (OR = 0.32, 95%CI 0.15–0.66, p = 0.002) participants from rural areas compared with those from urban areas. In addition, BMI was associated with type 2 diabetes in both indigenous (OR = 1.15, 95%CI 1.07–1.25, p < 0.001) and nonindigenous (OR = 1.09, 95%CI 1.03–1.14, p = 0.002) persons. Finally, low physical activity was associated with type 2 diabetes in nonindigenous (OR = 1.83, 95%CI 1.10–3.04, p = 0.021) participants, but not in indigenous ones. (Table 4)

Discussion

In this study, the prevalence of prediabetes and type 2 diabetes was significantly lower in indigenous than in nonindigenous population, after adjustment for recognized risk factors. This disparity may be accounted for by a variety of conditions, such as differences in lifestyle behaviors (i.e. calorie intake and level of physical activity), social determinants (i.e. illiteracy, residence area, and degree of marginalization), access to varied high-quality food, and other environmental factors not yet identified.

The prevalence of prediabetes varies widely around the world, due to in part to the ascertainment of this condition. Nevertheless, its increase is undeniable in both developed and developing countries [2,18]. In a study that included adults from seven Latin-American cities, fasting plasma glucose was used to define prediabetes, and the highest prevalence was reported in Mexico City, Mexico, and in Bogota, Colombia (3%). However, no estimations by indigenous origin were reported [19]. Another research conducted in six Central American countries including adults reported that 18.6% of participants had impaired glucose tolerance/impaired fasting glucose, ranging from 12.1%

in Nicaragua to 28.2% in Guatemala [20]. In a study that included Zapotec and Mixe communities from Oaxaca, 9.9% and 4.7% of participants, respectively, had impaired glucose tolerance [14]. A 2010 study of Pima (7.4%) and non-Pima Mexicans (8.5%) found a similar prevalence of abnormal glucose tolerance [21].

In many developed countries, indigenous populations have a higher prevalence of diabetes compared with nonindigenous ones, but this pattern is not consistent in developing countries. Studies carried out in American and Canadian Indians and in Australian aborigines have reported a higher prevalence of type 2 diabetes in these native groups than in nonindigenous populations, with the differences related to the adoption of obesogenic lifestyles and genetic predisposition [7,22]. Other studies suggest that the prevalence of type 2 diabetes is greater in population groups with higher levels of poverty and marginalization, particularly when they adopt urbanized lifestyles [23,24]. In the Mexican Family Life Survey, which oversampled communities with high proportions of indigenous people, the differences in the probability of diabetes were partially explained by lower SES [25]. On the other hand, a comparison between indigenous Pimas from Maycoba, Sonora, Mexico, and Pima Indians from Phoenix, Arizona, USA, showed that the latter had a 3-fold higher prevalence of diabetes than the Pimas from Mexico [11]. Although these 2 groups share similar genetic background, Pimas from Mexico are leaner, more physically active, and have a lower caloric intake than those from USA. Because Pimas from Mexico remain a relatively geographically and economically isolated population, the acculturation process has been less extensive than that probably occurring in less isolated populations [6,7,26,27]. When Pima (9%) and non-Pima Mexicans (10.5%) living in the same conditions are compared, slight differences in the prevalence of diabetes can be observed [21]. In our study, we found that the prevalence of type 2 diabetes was lower in indigenous than in nonindigenous population, perhaps due to their poverty and marginalization levels which, in this context, are associated with lower energy intake and thus lower obesity. Moreover, the prevalence of type 2 diabetes was similar in urban-dwelling indigenous and nonindigenous persons, which suggests that as acculturation occurs (i.e. lifestyles change) the risk for diabetes increases in indigenous population [11]. Relevant effects of acculturation include changes in dietary habits, physical activity, language of communication, among others, which all may have health implications [6,21,28]. Similar to other studies, we used the condition of speaking an indigenous language as a subrogation of the acculturation process. In this research, as in studies in Mexican-Americans and Latino

Table 4
Risk factors associated with prediabetes and type 2 diabetes in indigenous and nonindigenous populations of Comitan, Chiapas.

	Indigenous population n = 873		Nonindigenous population n = 952	
	OR* (95%CI)	p value	OR* (95%CI)	p value
Prediabetes				
Age (per 5 years)	1.22 (1.14–1.31)	< 0.001	1.19 (1.12–1.27)	< 0.001
Women	1.06 (0.67–1.66)	0.803	1.27 (0.84–1.91)	0.255
Rural area of residence	0.65 (0.40–1.06)	0.084	1.12 (0.72–1.74)	0.619
Family history of diabetes	2.00 (1.04–3.85)	0.038	1.41 (0.90–2.20)	0.135
Body mass index (per kg/m^2)	1.13 (1.07–1.19)	< 0.001	1.14 (1.09–1.18)	< 0.001
Low physical activity	0.84 (0.45–1.56)	0.579	1.16 (0.76–1.79)	0.489
Levels of triglycerides (per mmol)	1.30 (1.12–1.52)	0.001	1.36 (1.19–1.55)	< 0.001
Type 2 diabetes				
Age (per 5 years)	1.24 (1.11–1.37)	< 0.001	1.41 (1.30–1.53)	< 0.001
Women	1.43 (0.72–2.82)	0.302	1.19 (0.72–1.98)	0.500
Rural area of residence	0.31 (0.16–0.60)	< 0.001	0.32 (0.15–0.66)	0.002
Family history of diabetes	5.71 (2.74–11.89)	< 0.001	2.43 (1.44–4.09)	0.001
Body mass index (per kg/m^2)	1.15 (1.07–1.25)	< 0.001	1.09 (1.03–1.14)	0.002
Low physical activity	0.73 (0.31–1.74)	0.479	1.83 (1.10–3.04)	0.021
Levels of triglycerides (per mmol)	1.62 (1.34–1.94)	< 0.001	1.61 (1.39–1.88)	< 0.001

Missing values: for BMI 10 indigenous and 5 for nonindigenous individuals; for triglycerides 8 indigenous and 3 nonindigenous individuals.
 * Multiple multinomial logistic regression models were used to estimate ORs and their 95%CI.

populations, those less acculturated were less likely to have prediabetes and type 2 diabetes. Although 47% of the indigenous people from our study spoke their native language and < 3% were monolingual, 76% of them were living in rural areas, so they had a lower possibility of dealing with an obesogenic environment [6,7].

With respect to Latin America, a study with adults from seven cities the prevalence of diabetes ranged from 4% in Lima, Peru to 8.9% in Mexico City, Mexico [19]. In another study in Central America including adults from six cities the prevalence of diabetes varied from 5.4% in Honduras to 12.4% in Belize [20]. In a study comprising Bolivian (about 80% of participants of indigenous origin) and Chilean native populations, the prevalence of diabetes was 7.8% for Bolivians and 6.9% in Aymara natives and 8.2% in Mapuche indians for Chileans [29].

In Mexico, according to 2000 national estimates, the prevalence of type 2 diabetes was 8.1% in urban and 6.5% in rural areas [9], and in 2006 the prevalence rose to 15.5% and 10.4%, respectively [10]. In adults of Amerindian origin from Mexico, the prevalence ranged from 2% to 11.7% [11–14] with the highest prevalence found in Mayans (11.7%) from Yucatan [30], Zapotecos (8.7%) from Oaxaca [14], and Pimas from Maycoba, Sonora (8.6%) [11]. Few studies have distinguished between urban and rural areas regarding prevalence. In Mayan communities, the prevalence of diabetes was 1.3% [31]; however, recent estimations have shown a prevalence of 11.7% in both rural and urban areas [30]. In a study of rural areas in the State of Durango, the prevalence of type 2 diabetes was 3.2%, although the population was not classified considering indigenous or nonindigenous ethnicity [32]. Noteworthy, in the communities of Mexicaneros, Huicholes, and Tepehuanos from Durango, no cases of type 2 diabetes were reported [33].

In our study, indigenous population from rural areas had a decreased risk of prediabetes and type 2 diabetes. Rural poverty may be creating conditions that are favorable to cardio-metabolic health. Besides, indigenous population has a greater intake of carbohydrates compared with nonindigenous people; their intake of fat is lower, and their level of physical activity is higher. Therefore, their energy expenditure is more efficient with a positive impact on BMI reduction. On the other hand, indigenous persons living in urban areas increase their fat intake and decrease their physical activity, which is perhaps influenced by a greater access to high-density food and a more sedentary lifestyle. Another important aspect relates to the purchasing power of the population that allows accessing to a greater variety of best quality foods. Indigenous persons living in rural areas with a high degree of marginalization and social inequality have limited access to varied food because more than half of them eat only the food they grow. Moreover, this population has a high level of physical activity due to their working occupations, lacks means of transportation inside their community, and hence walks long distances every day.

Among the strengths of our research was the number of individuals enrolled in both the indigenous and nonindigenous groups of the study, which helped us to evaluate several social determinants and lifestyle risk factors. Although the nonresponse rate was moderate because of similar reasons in both groups of participants, no differences in social determinants between respondents and nonrespondents were found, thus reducing the probability of selection bias. On the other hand, because of the definition of indigenous origin we used, the probability of misclassification bias exists; nevertheless, self-reported indigenous origin is more related to social and cultural factors rather than biological or genetic aspects per se, which was more in accordance with the goals of this study [16]. Also, the cross-sectional design of the study made it difficult to determine the impact of changes on food intake and physical activity as well as on social determinants related to the prevalence of prediabetes and diabetes.

In summary, our results seem to support the hypothesis that environmental factors associated with traditional ways of life, such as moderate/vigorous physical activity and reduced access to energy-dense diet, protect the indigenous population from diabetes. Unfortunately, the process of acculturation, which is entailed by poverty reduction strategies in developing countries, may increase the prevalence of diabetes in these groups [6]. It is thus advisable to implement well-designed and comprehensive strategies and public policies against community isolation and poverty that promote education and healthcare while preserving at the same time healthy lifestyles.

Author Contrubution

A.J.-C. designed the study, researched data, conducted data analysis and wrote the manuscript. R.G.N. contributed to study design, data analysis, reviewed and edited the manuscript, and contributed to discussion. M.E.J.-C. contributed to study design, reviewed the manuscript and contributed to discussion. P.W.F. reviewed the manuscript and contributed to discussion. C.A.A.-S. reviewed the manuscript and contributed to discussion. S.H.J. reviewed the manuscript and contributed to discussion. E.O.G.-H. reviewed the manuscript and contributed to discussion. M.H.-A. reviewed the manuscript and contributed to discussion. A.J.-C. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Previuosly Presented

Part of this study was presented in abstract at the 73rd Scientific Session of The American Diabetes Association, June 21-25 2013, Chicago, IL, USA.

Conflicts of Interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jcte.2019.100191.

References

[1] Wild S, Roglic G, Green A, Sicree R, King H. Global Prevalence of Diabetes. Estimates for the year 2000 and projections for 2030. Diabetes Care 2004;27(5):1047–53.
[2] Shaw J, Sicree R, Zimmet P. Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes Res Clin Pract 2010;87(1):4–14.
[3] Barcelo A, Rajpathak S. Incidence and prevalence of diabetes mellitus in the Americas. Rev Panam Salud Publica 2001;10(5):300–8.
[4] Harris M, Hadden W, Knowler W, Bennett P. Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in the U.S. population aged 20–74 yrs. Diabetes 1987;36(4):523–34.
[5] Harris M. Epidemiological correlates of NIDDM in Hispanic, whites and blacks in

U.S. population. Diabetes Care 1991;14(7):639–48.

[6] Garcia L, Gold E, Wang L, Yang X, Mao M, Schwartz A. The Relation of Acculturation to Overweight, Obesity, Pre- Diabetes and Diabetes Among US Mexican-American Women and Men. Ethn Dis 2012;22(1):58–64.

[7] Schultz L, Chaudhari LO. High-Risk Populations: The Pimas of Arizona and Mexico. Curr Obes Rep 2015;4(1):92–8. https://doi.org/10.1007/s13679-014-0132-9.

[8] Dirección General de Epidemiología/Instituto Nacional de Nutrición Salvador Zubiran. Encuesta Nacional de Enfermedades Crónicas 1993. Secretaria de Salud, Mexico 1993.

[9] Olaiz-Fernandez G, Rojas R, Aguilar-Salinas C, Rauda J, Villalpando S. Diabetes mellitus in Mexican adults: results from the 2000 National Health Survey. Salud Publica Mex 2007;49(suppl 3):S331–7.

[10] Villalpando S, De la Cruz V, Rojas R, et al. Prevalence and distribution of type 2 diabetes mellitus in Mexican adult population. A probabilistic survey. Salud Publica Mex 2010;52(suppl 1):S19–26.

[11] Ravussin E, Valencia M, Esparza J, Bennett P, Schulz L. Effects of traditional life-style on obesity in Pima Indians. Diabetes Care 1994;17(9):1067–74.

[12] Alvarado-Osuna C, Milian-Suazo F, Valles-Sánchez V. Prevalencia de diabetes mellitus e hiperlipidemias en indígenas otomíes [Article in Spanish]. Salud Publica Mex 2001;43(5):459–63.

[13] Castro S, Escobedo P. La prevalencia de la diabetes Prevalence of non insulin dependent diabetes mellitus and associated risk factors in the Mazatec population of the State of Oaxaca, Mexico [Article in Spanish]. Gac Med Mex 1997;133(6):527–34.

[14] Escobedo J, Chavira I, Martınez L, Velasco X, Escandon C, Cabral J. Diabetes and other glucose metabolism abnormalities in Mexican Zapotec and Mixe Indians. Diabet Med 2010;27:412–6.

[15] Zvarova K, Zvarova Z, Callas P, Malone-Rising D. New estimates of pre-diabetes and type 2 diabetes prevalence in Mexican Quintana Roo. Int J Diabetes Dev Ctries 2013;33(1):8–12.

[16] Martínez-Cortes G, Salazar-Flores J, Laura G, et al. Admixture and Population Structure in Mexican-Mestizos Based on Paternal Lineages. J Hum Genet 2012;57(9):568–74.

[17] Consejo Nacional de Población, México. Estimaciones del CONAPO con base en el INEGI, Censo de Población y Vivienda 2010, Principales resultados por localidad. http://conapo.gob.mx/en/CONAPO/Indice_de_Marginacion_por_Localidad_2010. 2012.

[18] Danaei G, Finucane MY, Lu Y, et al. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2·7 million participants. Lancet 2011;378(9785):31–40.

[19] Escobedo J, Buitrón L, Velasco M, et al. High prevalence of diabetes and impaired fasting glucose in urban Latin America: the CARMELA Study. Diabet Med 2009;26(9):864–71.

[20] Barcelo A, Gregg E, Gerzoff R, et al. Prevalence of diabetes and intermediate hyperglycemia among adults from the first multinational study of noncommunicable diseases in six Central American countries: the Central America Diabetes Initiative (CAMDI). Diabetes Care 2012;35(4):738–40.

[21] Esparza-Romero J, Valencia M, Urquidez-Romero R, Chaudhari L, et al. Environmentally Driven Increases in Type 2 Diabetes and Obesity in Pima Indians and Non-Pimas in Mexico Over a 15-Year Period: The Maycoba Project. Diabetes Care 2015;38(11):2075–82.

[22] Dunstan D, Zimmet P, Welborn T, et al. The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study. Diabetes Care 2002;25(5):829–34.

[23] Rojas-Martínez R, Aguilar-Salinas CA, Jiménez-Corona A, Gómez-Pérez FJ, Barquera S, Lazcano-Ponce E. Prevalence of obesity and metabolic syndrome components in Mexican adults without type 2 diabetes or hypertension. Salud Publica Mex 2012;54(1):7–12.

[24] Leonard D, McDermott R, Odea K, et al. Obesity, diabetes and associated cardiovascular risk factors among Torres Strait Islander people. Aust N Z J Public Heal 2002;26(2):144–9.

[25] Stoddard P, Handley M, Vargas-Bustamante A, Schillinger D. The influence of indigenous status and community indigenous composition on obesity and diabetes among Mexican adults. Soc Sci Med 2011;73(11):1635–43.

[26] Valencia M, Bennett P, Ravussin E, Esparza J, Fox C, Schulz L. The Pima Indians in Sonora, Mexico. Nutr Rev 1999;57(5 Pt 2):S55–7.

[27] Schulz L, Bennett P, Ravussin E, et al. Effects of traditional and western environments on prevalence of type 2 diabetes in Pima Indians in Mexico and the U.S. Diabetes Care 2006;29(8):1866–71.

[28] Fisher-Hoch S, Vatcheva K, Rahbar M, McCormick J. Undiagnosed Diabetes and Pre-Diabetes in Health Disparities. PLoS ONE 2015;10(7):e0133135https://doi.org/10.1371/journal.pone.0133135.

[29] Carrasco E, Perez F, Angel B, et al. Prevalence of type 2 diabetes and obesity in two Chilean aboriginal populations living in urban zones [Article in Spanish]. Rev Medica Chil 2004;132(10):1189–97.

[30] Vargas-Azcona L. Epidemiología de la Diabetes Mellitus, Intolerancia a la glucosa y factores de riesgo aterogénico en Yucatán México. Rev Biomed 1994;5(3):151–9.

[31] Chavez A, Balam G, Zubiran S. Epidemiologic study of diabetes in 3 communities of the sisal producing area of the State of Yucatan [Article in Spanish]. Rev Invest Clin 1963;15:333–44.

[32] Guerrero Romero J, Rodriguez Moran M, Sandoval-Herrera F. The prevalence of non-insulin-dependent diabetes mellitus in the rural population of Durango, Mexico. Rev Panam Salud Publica 1997;2(6):386–91.

[33] Guerrero-Romero J, Rodríguez-Moran F, Sandoval-Herrera F. Low prevalence of non-insulin-dependent diabetes mellitus in indigenous communities of Durango Mexico. Arch Med Res 1997;29(19):137–40.