Epidemiology of Diabetes in Mexico and Associated Coronary Risk Factors

Israel Lerman-Garber MD and Juan Antonio Rull Rodrigo MD
Department of Endocrinology and Metabolism, National Institute of Nutrition Salvador Zubirán, Mexico City, Mexico

Key words: diabetes, epidemiology, coronary risk factors, Mexico

Type 2 diabetes in the Mexican population

The prevalence of type 2 or non-insulin-dependent diabetes varies widely. Most at risk are populations of developing countries, minority groups, and disadvantaged communities in industrialized countries [1]. Projections made from current epidemiological data in Mexican and Mexican-American populations suggest that the incidence of type 2 diabetes in this ethnic group will continue to escalate, being closely related to increased rates of obesity, genetic background, and the trend for diminished physical activity [2–4]. Diabetes-related complications will occur more frequently because of the early appearance of the disease, its underdiagnosis and undertreatment, and the high prevalence of other coronary risk factors such as hypertension, dyslipidemias and smoking [5–8].

The population of Mexico is currently estimated at 100 million. During the last few decades the country has undergone dramatic and very rapid socioeconomic changes that have led to profound demographic and epidemiological changes. Internal migration has been massive, the proportion of the population in urban areas having increased from 42.6% in 1950 to 71.4% in 1990. Infant mortality dropped by 14.4% from 1970 to 1990 and life expectancy rose from 63.5 to 73.9 years during the same period. While around 50% of the Mexican population are youngsters, in the 65 years and older age group the growth rate is 4%, i.e., twice the actual growth of the total population. Current estimates suggest that by the year 2030 there will be 14 millions Mexicanos in this age group [9].

Mexico is on the ascending limb of the diabetes epidemic. There has been a stepwise increase in the prevalence from 2–3% in 1963 when the first available data were obtained to around 8–9% in the most recent surveys [10–13]. According to the Chronic Diseases National Survey, the distribution of diabetes increased in states with the largest urban concentrations, was associated with advanced age and increased body mass index, and affected mostly the lowest income groups with the lowest levels of education. Almost 25% of people aged 65 and above are diabetics, however 0.5% of the population in the 20–30 age group and 3.0% in the 30–40 age group have diabetes. Based on our current population pyramid with its large base of younger individuals, extrapolation of these data allow a calculation of approximately 300,000 diabetics in the 20–40 age group, which represents a tremendous impact on our public health facilities in the years to come [13].

The thrifty genotype

Newly arrived migrants to the large cities in Mexico rapidly adapt to the urban milieu, losing their rural nutritional and activity habits. In addition, it is well known that Mexicans, particularly those of Indian or mixed origin, share with other Indian-American groups a high genetic susceptibility to type 2 diabetes [14]. Considering diabetes prevalence rates separately, one sees almost a fivefold difference between rural and urban communities.

The thrifty genotype hypothesis proposes that in traditional populations subject to periods of “feast and famine,” a survival advantage was conferred on those whose metabolism stored energy with maximum efficiency [15]. With modernization and the accompanying assured supply of highly refined calories, coupled with a sedentary lifestyle, the thrifty genotype became disadvantageous, leading to obesity, hyperinsulinemia, insulin resistance, and eventually to pancreatic beta cell decompensation and diabetes [16]. This is evident in the Mexican population where the genotype probably has a high prevalence and penetration.

Of great concern is how changes in the physical environment and lifestyle, such as have occurred in Mexican-Americans, can result in the major causes of morbidity and mortality [14] and even override genetic susceptibility in the expression of type 2 diabetes and other traits. Unfortunately, as demonstrated by recent surveys, the conditions that predispose to an increased prevalence of diabetes and related complications are already present in Mexico [8,12,17–19].

Diabetes and coronary heart disease

Cardiovascular disease represents the greatest burden of diabetes, at both the individual and population level. Approximately 75% of patients with diabetes will die from cardiovascular disease [20–22]. Diabetic patients have more extensive
atherosclerosis than non-diabetics, a higher incidence of multi-
vessel disease, a greater propensity for suffering cardiovascular
sequelae, and a poorer short-term and one year prognosis after
a first myocardial infarction. The mortality rate from CHD is at
least three times greater, the numbers being particularly high for
women [23,24]. In a recent study, the 7 year incidence of
myocardial infarction in non-diabetic subjects (age 45–64 years)
with a prior myocardial infarction at baseline was shown to be
similar to that of diabetic subjects without a prior myocardial
infarction (18.8% vs. 20.2% respectively) [25,26].

While the role of diabetes as a risk factor for cardiovascular
disease is well established, several epidemiological and clinical
studies have also shown an association between increased
insulin concentrations and the metabolic syndrome with an
increased risk for atherosclerotic heart disease [27–29]. There
are important associated coronary risk factors that may coexist
in diabetic patients, such as hyperglycemia, dyslipidemia,
insulin resistance, platelets and hemostatic abnormalities,
oxidative stress, vascular dysfunction, advanced glycosylation
of proteins, hypertension, smoking, obesity, lack of exercise,
and a positive family history of premature CHD [20–22]. A
decline in CVD mortality has been shown in the general U.S.
population, but not in diabetic patients, particularly in women;
therefore, aggressive management of cardiovascular risk factor
in diabetic subjects must be initiated before the onset of overt
CHD.

Cardiovascular diseases are the leading cause of death in
Mexico. Results of the National Survey of Chronic Diseases [13]
showed that arterial hypertension was twice as common and
myocardial infarction and stroke three to four times more
frequent in diabetic than in non-diabetic individuals. Some
conditions, such as peripheral vascular disease, are more
prevalent in Mexican than in non-Hispanic whites [5]. Haffner
et al. [30] compared the prevalence of hypertension in 1,500
Mexican Americans who participated in the San Antonio Heart
Study and 2,280 low income Mexicans who participated in the Mexico City
Study. The crude prevalence of mild hypertension was 17.1% and 17.4% in
Mexican men and women, compared to
24.4% and 22.0% in Mexican American
men and women (P < 0.001 and
P < 0.005 respectively). These
differences could be related to greater
physical activity, lower body mass index,
and the consumption of a high carbo-
hydrate low fat diet in the Mexican
population.

The results of cross-cultural epidemi-
ological studies on changes in mor-
tality and morbidity from CHD related
to changes in lifestyles and coronary risk factors give strong
support to the concept that environment and lifestyle are
powerful determinants of the frequency of CHD in populations
[6,7,16,18,28,31].

Diabetes and associated coronary risk factors in Mexico

An epidemiological survey in Mexico City to determine the
prevalence of diabetes and associated coronary risk factors [12]
was conducted in a sample of 805 individuals aged 20–90 years,
who were selected by the method of multistage cluster sampling
with proportional allocation. The crude rate prevalence of type
2 diabetes was 8.7%, with an age-adjusted rate of 10.6% for
females and 6.0% for males. Although age strongly influenced
the prevalence of diabetes, a significant proportion (5.9%) of
younger individuals (35-44 years of age) was affected by the
disease. Diabetes was associated with advanced age, had a
greater impact in the low income classes, and showed increased
odds ratios for hypertension, dyslipidemias and myocardial
infarction in men and women and for obesity only in women.
Diabetes from the poor income class had increased BMI and a
trend for higher triglycerides and lower high density lipopro-
tein-cholesterol values. Individuals with impaired fasting
glucose or newly diagnosed diabetes with fasting plasma glucose
between 126 and 140 mg/dl had a higher atherogenic risk profile
than individuals with a normal carbohydrate metabolism.

The prevalence of insulin-resistant related metabolic dis-
orders was high in this random sample of the Mexico City
population [Table 1]. The mean values of BMI, waist measure-
ment, waist-to-hip ratio, systolic and diastolic blood pressure,
triglycerides, glucose and the atherogenic index increased
significantly at higher insulin levels. A significant inverse
tendency was observed for the mean concentration of HDL-
cholesterol. Other variables like age, lipoprotein(a), total and
LDL-cholesterol had no association with insulin levels.

| Table 1. Anthropometric and metabolic variables according to insulin quartile in males from Mexico City |
|-----------------|----------------|----------------|----------------|----------------|
|                 | Quartile 1     | Quartile 2     | Quartile 3     | Quartile 4     |
| Insulin (μU/ml) | < 5.6          | 5.6–8.5        | 8.5–12.7       | > 12.7         |
| N               | 99             | 99             | 101            | 97             |
| Age (yr)        | 41 ± 14        | 37 ± 13        | 39 ± 12        | 41 ± 13        |
| BMI (kg/m²)     | 24.3 ± 2.6     | 25.1 ± 2.5     | 26.6 ± 3.0     | 28.7 ± 4.2     |
| WHR             | 0.94 ± 0.07    | 0.94 ± 0.06    | 0.95 ± 0.06    | 0.98 ± 0.08    |
| SBP (mmHg)      | 118 ± 17       | 119 ± 13       | 122 ± 17       | 125 ± 18       |
| DBP (mmHg)      | 75 ± 10        | 75 ± 10        | 77 ± 12        | 80 ± 11        |
| TC (mg/dl)      | 206 ± 44       | 205 ± 51       | 205 ± 58       | 212 ± 36       |
| TG (mg/dl)      | 141 ± 75       | 154 ± 97       | 172 ± 91       | 206 ± 107      |
| LDL-C (mg/dl)   | 140 ± 39       | 139 ± 45       | 140 ± 33       | 141 ± 33       |
| HDL-C (mg/dl)   | 43 ± 11        | 41 ± 10        | 38 ± 9         | 38 ± 9         |
| Lp(a) (mg/dl)   | 19.6 ± 28.3    | 16.9 ± 21.3    | 19.1 ± 28.5    | 13.6 ± 29.7    |
| Glucose (mg/dl)| 89 ± 10        | 97 ± 24        | 102 ± 34       | 99 ± 22        |

CHD = coronary heart disease
CVD = coronary vascular disease
BMI = body mass index
WHR = waist-to-hip ratio, SBP = systolic blood pressure, DBP = diastolic blood pressure, TC = total cholesterol, TG = triglycerides.
The values are expressed as mean ± standard deviation. *ANOVA
similar pattern was observed in women, however statistical significance was only obtained for BMI, waist measurement, triglycerides, glucose and HDL-cholesterol concentrations. In this study, there was a three- to fivefold increase in the risk of presenting two, three or more cardiovascular risk factors in subjects with higher fasting insulin levels. Lp(a) mean levels were inversely related to fasting insulin concentrations [18].

Mexican Americans are known to have a prevalence of type 2 diabetes three times higher than non-Hispanic whites, as well as higher mean plasma insulin levels and a more centralized upper body adiposity. An anthropometric and metabolic profile was consistent with our findings in the Mexico City population. The rich carbohydrate diet that is characteristic of the Mexican population has been linked to increased insulin and triglyceride values, however the clustering of metabolic disorders cannot be explained solely on the basis of diet, obesity or body fat distribution [27].

**Diabetes in the elderly**

In the elderly age group in Mexico, diabetes is the second cause of mortality after cardiovascular disease and the main cause of mortality and hospitalization documented by the Mexican Institute of Social Security [32]. These data must be tempered by two parallel tendencies: the change in age distribution to greater absolute numbers of older people, who have higher prevalence and incidence rates of diabetes and its complications; and the increasing urbanization of the rural population.

A study was recently undertaken to determine the prevalence of diabetes and to examine its association with food intake, anthropometric and metabolic variables and other coronary risk factors in urban and rural elderly Mexican populations [33]. In this cross-sectional study of three different Mexican communities (urban area of medium-low income, urban area of low income, and a rural area), individuals were randomly selected and a personal interview assessed demographic information, personal medical history and functional status. A 24 hour diet recall was obtained. The physical examination included anthropometric and blood pressure measurements, and a fasting blood sample was obtained for measurements of lipids, insulin and glucose. Findings showed that diabetes prevalence was higher in males than females for all age groups – 16.7 vs. 9.5% in adults and 30.8 vs. 22.8% in the elderly. Using a multivariate stepwise logistic regression, elderly individuals had the following variables independently associated with diabetes: male gender odds ratio 2.1, \( P < 0.009 \), diminished carbohydrate intake (OR 0.77, \( P < 0.03 \)), central distribution of the adiposity (OR 1.9, \( P < 0.03 \)) and functional disability (OR 2.3, \( P < 0.01 \)).

Rural-urban comparisons offer an exceptional opportunity to study the spectrum of socioeconomic and environmental factors and their relationship to the health of the population.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Urban: Middle-low</th>
<th>Urban: Low</th>
<th>Rural</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>n = 45</td>
<td>n = 36</td>
<td>n = 40</td>
</tr>
<tr>
<td>Fiber (g/day)</td>
<td>10 ( \pm ) 6.6</td>
<td>93</td>
<td>20 ( \pm ) 11</td>
</tr>
<tr>
<td>% total protein</td>
<td>15 ( \pm ) 0.4</td>
<td>13 ( \pm ) 0.3</td>
<td>13 ( \pm ) 0.1</td>
</tr>
<tr>
<td>% CHO</td>
<td>52 ( \pm ) 0.11</td>
<td>60 ( \pm ) 0.09</td>
<td>70 ( \pm ) 0.8</td>
</tr>
<tr>
<td>% fat</td>
<td>33 ( \pm ) 0.10</td>
<td>27 ( \pm ) 0.08</td>
<td>18 ( \pm ) 0.7</td>
</tr>
<tr>
<td>Alcohol intake (%)</td>
<td>13.3 ( \pm ) 13.9</td>
<td>147 ( \pm ) 14</td>
<td></td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>114 ( \pm ) 49</td>
<td>108 ( \pm ) 51</td>
<td>83 ( \pm ) 37</td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>213 ( \pm ) 41</td>
<td>208 ( \pm ) 30</td>
<td>191 ( \pm ) 37</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>41 ( \pm ) 11</td>
<td>42 ( \pm ) 18</td>
<td>50 ( \pm ) 14</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td>151 ( \pm ) 40</td>
<td>142 ( \pm ) 28</td>
<td>122 ( \pm ) 35</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>183 ( \pm ) 157</td>
<td>165 ( \pm ) 60</td>
<td>147 ( \pm ) 130</td>
</tr>
<tr>
<td>Insulin (mU/ml)</td>
<td>42 ( \pm ) 68</td>
<td>26 ( \pm ) 47</td>
<td>12 ( \pm ) 10</td>
</tr>
</tbody>
</table>

**CHO**% of total = carbohydrate percent of total, alcohol intake = significant alcohol intake.

Data are presented as means SD. Mean comparisons between two groups were performed by Student’s \( t \)-test for independent variables.

Urban middle-low vs. urban low \( P \leq 0.05, **P \leq 0.01 \)

Urban middle vs. rural \( P \leq 0.05, ***P \leq 0.001 \)

Urban low vs. rural \( P \leq 0.05, \#P \leq 0.001 \)
Table 3. Diabetes mellitus and associated coronary risk factors in adults and elderly people

<table>
<thead>
<tr>
<th>Variable</th>
<th>DM (%)</th>
<th>No DM (%)</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;30% fat diet</td>
<td>56.3</td>
<td>37.3</td>
<td>2.2</td>
<td>1.0-4.6</td>
<td>0.04</td>
</tr>
<tr>
<td>BMI &gt; 27</td>
<td>60</td>
<td>49.4</td>
<td>1.5</td>
<td>0.7-3.3</td>
<td>0.27</td>
</tr>
<tr>
<td>Hypertension</td>
<td>50.0</td>
<td>38.4</td>
<td>1.6</td>
<td>0.8-3.4</td>
<td>0.21</td>
</tr>
<tr>
<td>Smoker</td>
<td>32.3</td>
<td>25.1</td>
<td>1.4</td>
<td>0.6-3.2</td>
<td>0.39</td>
</tr>
<tr>
<td>HDL-C</td>
<td>40.6</td>
<td>16.5</td>
<td>3.5</td>
<td>1.6-7.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Low HDL-C</td>
<td>40.6</td>
<td>53.6</td>
<td>0.6</td>
<td>0.3-1.3</td>
<td>0.17</td>
</tr>
<tr>
<td>HTG</td>
<td>53.1</td>
<td>26.6</td>
<td>3.1</td>
<td>1.5-6.6</td>
<td>0.002</td>
</tr>
</tbody>
</table>

| Elderly           |        |           |      |        |       |
| 30% fat diet      | 48.2   | 41.4      | 1.3  | 0.8-2.2| 0.27  |
| History of MI     | 14.5   | 5.8       | 2.8  | 1.2-6.2| 0.01  |
| BMI > 27          | 40.2   | 37.3      | 1.1  | 0.7-1.9| 0.64  |
| Hypertension      | 42.4   | 36.3      | 1.3  | 0.7-2.4| 0.38  |
| Smoker            | 14     | 12.6      | 1.1  | 0.6-2.3| 0.74  |
| HDL-C             | 33.3   | 34.3      | 1.0  | 0.5-1.7| 0.89  |
| Low HDL-C         | 41.9   | 37.2      | 1.2  | 0.7-2.1| 0.49  |
| HTG               | 35.5   | 21.2      | 2.0  | 1.4-3.7| 0.02  |

DM = diabetes mellitus; CI = confidence intervals; MI = myocardial infarction in Mexican Americans and non-Hispanics whites; The San Antonio Heart Study. Circulation 1991;83(45).1


Correspondence: Dr. I. Lerman, Departamento de Diabetes y Metabolismo de Lípidos, Instituto Nacional de la Nutrición Salvador Zubirán, Vasco de Quiroga #15, Tlalpan, 14000 México City, México. Phone: (52-5) 573-1200 ext 2405, Fax: (52-5) 655-1076, email: lerman@netserve.com.mx

---

**Capsule**

**Fruits and vegetables to fight cancer**

Some epidemiologic studies suggest that elevated fruit and vegetable consumption is associated with a reduced risk of breast cancer. However, most have been case-control studies in which recall and selection bias may influence the results. In addition, publication bias may have influenced the literature on associations for specific fruit and vegetable subgroups.

Smith-Warner et al. assessed the association between breast cancer and total and specific fruit and vegetable group intakes using standardized exposure definitions. Their analyses included eight prospective studies that had at least 200 incident breast cancer cases, assessed usual dietary intake and completed a validation study of the diet assessment method or a closely related instrument. The studies comprised 7,377 incident invasive breast cancer cases occurring among 351,825 women whose diet was analyzed at baseline. For comparisons of the highest vs. lowest quartiles of intake, weak non-significant associations were observed for total fruits (relative risk 0.93), total vegetables (RR 0.96), and total fruits and vegetables (RR 0.93). No additional benefit was apparent in comparisons of the highest and lowest deciles of intake. No associations were observed for green leafy vegetables, 8 botanical groups, and 17 specific fruits and vegetables. The authors conclude that fruit and vegetable consumption during adulthood is not significantly associated with reduced breast cancer risk.

*JAMA* 2001;281:769

---

**Capsule**

**Influenza prevention by zanamivir inhalation**

As prophylaxis against influenza in families, amantadine and rimantadine have had inconsistent effectiveness, partly because of the transmission of drug-resistant variants from treated index patients. In their double-blind, placebo-controlled study of inhaled zanamivir for the treatment and prevention of influenza in families, Hayden et al. enrolled families (with two to five members and at least one child aged 5 or older) before the 1998–1999 influenza season. If an influenza-like illness developed in one member, the family was randomly assigned to receive either inhaled zanamivir or placebo. The family member with the index illness was treated with either 10 mg of inhaled zanamivir (163 subjects) or placebo (158) twice a day for 5 days, and the other family members received either 10 mg of zanamivir (414 subjects) or placebo (423) once a day as prophylaxis for 10 days. The primary end point was the proportion of families in which at least one household contact had symptomatic, laboratory-confirmed influenza.

The results showed that zanamivir provided protection against both influenza A and influenza B. A neuraminidase-inhibition assay and sequencing of the neuraminidase and hemagglutinin genes revealed no zanamivir-resistant variants. When combined with the treatment of index cases, prophylactic treatment of family members with once-daily inhaled zanamivir is well tolerated and prevents the development of influenza. In this study there was no evidence of the emergence of resistant influenza variants.