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Myocardial Infarction in Mexican-Americans and Non-Hispanic Whites

The San Antonio Heart Study

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Mexican-American men experience reduced cardiovascular mortality compared with non-Hispanic white men. There is no corresponding ethnic difference in cardiovascular mortality in women. The difference in men could result either from a lower incidence of cardiovascular disease or a lower case fatality rate among Mexican-Americans. Although the incidence of cardiovascular disease in Mexican-Americans is unknown, we have collected data on prevalence of myocardial infarction in 5,148 individuals examined in the San Antonio Heart Study, a population-based survey of cardiovascular disease conducted between 1979 and 1988 in Mexican-Americans and non-Hispanic whites aged 25–64 years. Myocardial infarction was assessed by Minnesota-coded electrocardiograms and by a self-reported history of a physician-diagnosed heart attack. For both end points, the age-adjusted prevalence of myocardial infarction was lower in Mexican-American men than in non-Hispanic white men. After adjustment for age and diabetes status (present/absent), the odds of a myocardial infarction, as defined by either criterion, was approximately one third lower in Mexican-American men than in non-Hispanic white men ($p=0.06$). In women, the prevalence of both myocardial infarction end points was slightly higher in Mexican-Americans than in non-Hispanic whites, although neither of these differences was significant. Although the ethnic differences in prevalence in this study were not statistically significant, their pattern parallels the pattern in the mortality due to cardiovascular diseases. Therefore, the results support the hypothesis that the reduced cardiovascular mortality rate observed in Mexican-American men reflects a lower incidence of myocardial infarction rather than a reduced case fatality rate because the latter would result in a higher prevalence. (*Circulation* 1991;83:45–51)

On the basis of their cardiovascular risk factor profile, Mexican-Americans appear to be a population at high risk for coronary heart disease (CHD). Compared with non-Hispanic whites, for example, Mexican-Americans have a threefold higher prevalence of diabetes^{1,2} and are more likely to be obese.^{1–5} In addition, triglyceride levels tend to be higher in Mexican-Americans,^{1,3,4,6} and high density lipoprotein (HDL) cholesterol levels tend to be lower.^{1,6} Even though Mexican-Americans tend to smoke less than non-Hispanic whites,^{2,6} their Framingham risk scores for mortality due to cardio-

vascular diseases are significantly higher than those for non-Hispanic whites.⁶

Despite the high levels of cardiovascular risk factors of Mexican-Americans, mortality studies from California,^{7,8} New Mexico,^{9,10} and Texas^{11–14} have documented that men in this ethnic group experience lower cardiovascular mortality rates (15–36% lower) than do non-Hispanic white men. Interestingly, this protection does not appear to extend to Mexican-American women, who have cardiovascular mortality rates similar to those of non-Hispanic white women (Table 1). The ethnic difference in cardiovascular mortality observed in men could be even greater than reported because Mexican-Americans are known to be underestimated in the US census relative to non-Hispanic whites,¹⁵ thus overestimating their cardiovascular mortality rates.

Two possible explanations might account for the lower cardiovascular mortality rates of the Mexican-American men. First, despite a less-favorable cardiovascular risk profile, Mexican-American men may

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TABLE 1. Studies Comparing Cardiovascular Mortality Between Mexican-Americans and Non-Hispanic Whites

Geographic area	Time period	Mortality end point	Men			Women		
			Rate*			Rate		
			MA	NHW	MA/NHW	MA	NHW	MA/NHW
New Mexico ¹⁰	1978–1982	Ischemic heart disease	159.8	231.4	0.69	91.6	109.8	0.83
New Mexico ⁹	1969–1975	Ischemic heart disease	193.1	299.6	0.64	—	—	—
Texas ¹¹	1969–1971	Ischemic heart disease	346.1	441.4	0.78	225.0	217.2	1.04
Texas ¹⁴	1979–1981	Acute myocardial infarction	344.7	403.4	0.85	168.8	175.7	0.96
California ⁷	1969–1971	Cardiovascular disease	—	—	0.82	—	—	1.00

*All rates are age-adjusted and expressed per 100,000 people. (See individual references for standard population used and for *International Classification of Diseases, Ninth Revision* codes used to define disease category.)

MA, Mexican-American; NHW, non-Hispanic white.

experience a lower incidence of cardiovascular disease than do non-Hispanic white men. Alternatively, Mexican-American men may experience the same or higher incidence of cardiovascular disease, but their disease could be characterized by a lower case fatality rate. Unfortunately, incidence data on cardiovascular disease in Mexican-Americans are not available to distinguish between these two possibilities. In this report, we present data on the prevalence of myocardial infarction, as assessed by Minnesota-coded electrocardiograms (ECGs) and by self-reported history of physician-diagnosed heart attack, obtained from 5,148 Mexican-Americans and non-Hispanic whites examined in the San Antonio Heart Study, a population-based study of cardiovascular disease and diabetes.

Methods

Details of the study design, sampling and recruitment procedures, response rates, and field procedures of the San Antonio Heart Study have been previously reported.^{1,2,5,16–18} Briefly, subjects were recruited into the San Antonio Heart Study in two phases, the first between October 1979 and November 1982, and the second between October 1984 and October 1988. Households were randomly sampled from three types of neighborhoods: a low-income barrio, a middle-income transitional neighborhood, and a high-income suburb. All men and nonpregnant women between 25 and 64 years of age residing in the selected households were considered eligible for the study and were invited to undergo a physical examination in a mobile clinic. The combined response rate for both phases of the study was 65.3%.

Ethnicity was defined on the basis of a previously published algorithm that considers parental surnames and birthplaces, stated ethnicity of grandparents, and participant's preferred ethnic identity when it indicated a distinct national origin.¹⁹ Compared with the reference standard of at least three grandparents of Mexican origin this algorithm outperforms other ethnic classification schemes based on surname alone.¹⁹ Persons who were identified as belonging to an ethnic group other than Mexican-American or non-Hispanic white are excluded from these analyses. The protocol was approved by the University of

Texas Health Science Center Institutional Review Board, and all subjects gave informed consent.

At the time of the clinic visit, subjects were asked if they had ever been told by a physician that they had had a heart attack. Fasting blood samples were then drawn for plasma glucose and for serum lipid and lipoprotein determinations (including total cholesterol, triglyceride, and HDL cholesterol levels). Glucose, lipid, and lipoprotein methods have been previously described.^{1,16,18} A 2-hour oral glucose tolerance test was administered, and diabetes was diagnosed according to the plasma glucose criteria of the National Diabetes Data Group.²⁰ Individuals who did not meet these criteria but who gave a history of diabetes and who were currently taking either oral antidiabetic agents or insulin were also considered to have diabetes. Individuals with insulin-dependent diabetes mellitus (defined as persons currently taking insulin, age of diabetes onset less than 40 years of age and body mass index less than 30 kg/m²) are excluded from the present analyses ($n=16$).

Standard 12-lead ECGs were obtained from all subjects and sent to the ECG Coding Laboratory of the University of Minnesota to be read. The same two experienced readers analyzed all the tracings according to the Minnesota Code criteria,²¹ and a third reader arbitrated all disagreements. Tracings were then classified on the basis of their Q and QS patterns as definite, possible, or unlikely myocardial infarction. Definite myocardial infarction included pathological Q and QS patterns (all 1.1 codes) and prominent Q and QS patterns accompanied by negative T waves (all 1.2 codes accompanied by codes 5.1 or 5.2, with the exception of 1.2.6 and 1.2.8). Possible myocardial infarction included minor Q and QS patterns (all 1.3 codes and codes 1.2.6 and 1.2.8), and unlikely myocardial infarction included all other codes.

Although ECGs were obtained for all subjects, the ECGs have currently been analyzed according to Minnesota Code criteria only on phase 1 subjects ($n=2,080$ coded ECGs; response rate, 94.0%) plus all diabetic subjects from both phases ($n=192$ coded ECGs for phase 1 and $n=260$ coded ECGs for phase 2; combined response rate, 97.4%). Because of the

TABLE 2. Mean Values of Risk Factors for Cardiovascular Diseases in Mexican-Americans and Non-Hispanic Whites

Risk factor	Men		Women	
	MA	NHW	MA	NHW
Subjects (<i>n</i>)	1,384	827	1,894	1,036
Age (yr)	43.0	44.8*	42.9	44.8*
Body mass index (kg/m ²)	28.0	27.3*	28.4	26.7*
Total cholesterol (mg/100 ml)	207.1	205.6	200.5	200.8
HDL cholesterol (mg/100 ml)	42.7	43.2	49.4	52.3*
Triglycerides (mg/100 ml)	149.0	137.3*	117.1	105.4*
Systolic blood pressure (mm Hg)	121.2	119.8*	115.0	113.0*
With NIDDM (%)	11.1	4.5*	12.7	4.0*
Cigarette smoking				
Current smokers (%)	36.7	30.4*	21.0	26.8*
Quantity (cigarettes/day; current smokers)	12.9	18.9*	8.1	13.9*

Mean values of continuous variables represent age-adjusted means by analysis of covariance. Percent with NIDDM and percent current smokers obtained by direct age adjustment.

MA, Mexican-American; NHW, non-Hispanic white; HDL, high density lipoprotein; NIDDM, noninsulin-dependent diabetes mellitus.

**p* < 0.001 for MA vs. NHW.

small number of ECG-confirmed myocardial infarctions, the definite (*n*=21) and possible (*n*=72) categories for this end point were combined and considered as a positive response.

In addition to the two myocardial infarction end points (ECG-documented myocardial infarction and self-report of a physician-diagnosed myocardial infarction) two composite measures were created: 1) myocardial infarction by either ECG or self-report; and 2) myocardial infarction by both ECG and self-report. The age-adjusted prevalence of each myocardial infarction end point was calculated by the direct method,²² using the pooled population of Mexican-Americans and non-Hispanic whites in the San Antonio Heart Study as the standard. Because ECGs have not yet been coded according to Minnesota criteria for the nondiabetic subjects from phase 2, analysis of this end point and of the two composite end points is based on the phase 1 sample only. Analyses were also stratified by diabetes status, and myocardial infarction prevalence rates among diabetic subjects are based on phases 1 and 2.

Little difference was observed in the prevalence rates of myocardial infarction between phase 1 (1979–1982) and phase 2 (1984–1988) of the study. Allowance for an effect of examination period did not alter the results of any of the ethnic comparisons reported in the present results. Therefore, the two study periods are pooled in the analyses presented in this paper.

Age-adjusted ethnic odds ratios were computed using the Mantel-Haenszel procedure.²³ Simultaneous adjustment for the effects of age and diabetes status was performed with the multiple logistic regression model.²⁴ To account for the less-favorable cardiovascular risk profile of Mexican-Americans, we also used the multiple logistic regression model to estimate the effect of ethnicity on myocardial infarction prevalence after adjusting for conventional car-

diovascular risk factors. The risk factors included in this model were age (years), diabetes (present/absent), body mass index (kg/m²), systolic blood pressure (mm Hg), total cholesterol level (mg/dl), triglyceride level (mg/dl), HDL cholesterol level (mg/dl), and smoking status (current cigarette smoker/not a current smoker).

Results

A total of 3,281 Mexican-Americans and 1,867 non-Hispanic whites were examined in the San Antonio Heart Study. Table 2 presents ethnic comparisons of the conventional cardiovascular risk factors. Except for total cholesterol levels and cigarette smoking, all cardiovascular risk factors were significantly less favorable in Mexican-Americans than in non-Hispanic whites, although the HDL cholesterol level difference was statistically significant only in women. Table 3 shows the number of subjects examined for each end point according to study phase and ethnicity. The small number of subjects examined in phase 2 for the ECG only and for the composite either and both end points reflects the fact that at the present time ECGs have been coded according to Minnesota Code criteria only for diabetic subjects in phase 2.

Age-specific and age-adjusted prevalence rates for the four myocardial infarction end points are presented in Table 4. The prevalence of each end point was higher in men than in women in both ethnic groups. In men, the age-adjusted prevalence of each of the four myocardial infarction end points was higher in non-Hispanic whites than in Mexican-Americans, with the age-adjusted ethnic odds ratios (Mexican-American versus non-Hispanic white) ranging from 0.47 to 0.88 for the four end points. In women, the opposite trend was observed, with Mexican-Americans having a higher prevalence of myocardial infarction than non-Hispanic whites (ethnic

TABLE 3. Subjects Examined According to Study Phase and Ethnicity

End point	Phase 1				Phase 2			
	MA		NHW		MA		NHW	
	<i>n</i>	no of events	<i>n</i>	no of events	<i>n</i>	no of events	<i>n</i>	no of events
Men								
ECG	520	20	374	22	79*	13	16*	4
Self-reported	541	19	398	23	834	35	422	19
Either	512	35	369	37	80*	19	16*	7
Both	549	4	403	8	82*	5	16*	1
Women								
ECG	698	17	488	8	150*	8	15*	1
Self-reported	720	12	521	11	1,157	15	510	5
Either	686	28	483	18	149*	13	15*	1
Both	732	1	526	1	157*	0	15*	0

For the end point both, subjects were considered noncases when either self-report or ECG was normal (even when the other end point was missing). For the end point either, subjects were considered noncases only when both end points were normal. Thus, the number of subjects available for analysis was greater for the both criterion than for the either criterion.

MA, Mexican-American; NHW, non-Hispanic white; ECG, electrocardiogram.

*Diabetic subjects only.

odds ratios ranging from 1.08 to 1.82). In neither sex, however, were any of these ethnic differences statistically significant.

To increase the power of our data to detect ethnic differences, age-adjusted myocardial infarction prevalence rates were computed for diabetic and nondiabetic subjects separately (Table 5). In both sexes, myocardial infarction prevalence was considerably

higher in diabetic subjects than in nondiabetic subjects, and the adjusted odds ratios (diabetic versus nondiabetic) ranged from 2.20 to 3.86 in men and from 1.91 to 2.55 in women. The association between ethnicity and myocardial infarction was similar in diabetic and nondiabetic subjects (diabetes-specific ethnic odds ratios not shown), and therefore, only the diabetes-adjusted ethnic odds ratios are presented.

TABLE 4. Age-Adjusted and Age-Specific Prevalence of Myocardial Infarction According to Gender and Ethnicity

	ECG		Self-reported		Either		Both	
	MA	NHW	MA	NHW	MA	NHW	MA	NHW
Men								
Age (yr)								
25-44	2.2	2.4	1.2	0.7	3.7	3.7	0.0	0.0
45-54	5.0	7.4	5.2	5.6	9.3	10.6	0.7	2.9
55-65	6.7	9.6	10.1	12.8	11.9	18.6	2.7	4.1
Age-adjusted prevalence	4.0	5.5	4.1	4.6	7.2	9.2	0.8	1.8
Age-adjusted OR	0.72		0.88		0.76		0.47	
95% CI	0.38-1.35		0.58-1.35		0.58-1.35		0.14-1.55	
<i>p</i>	0.30		0.56		0.27		0.20	
Women								
Age (yr)								
25-44	1.5	0.0	0.7	0.4	2.2	0.9	0.0	0.0
45-54	4.2	2.6	1.3	1.3	5.6	4.3	0.0	0.0
55-65	3.2	3.2	3.6	4.0	8.3	7.2	0.7	0.6
Age-adjusted prevalence	2.5	1.4	1.5	1.4	4.4	3.2	0.2	0.1
Age-adjusted OR	1.82		1.08		1.40		1.25	
95% CI	0.75-4.43		0.57-2.03		0.75-2.61		0.08-20.12	
<i>p</i>	0.19		0.82		0.30		0.89	

Age-adjusted values were adjusted to the age distribution of the pooled (Mexican-American plus non-Hispanic white) San Antonio Heart Study population.

ECG, electrocardiogram; MA, Mexican-American; NHW, non-Hispanic white; OR, odds ratio; CI, confidence interval.

Age-adjusted odds ratios by Mantel-Haenszel procedure.

TABLE 5. Age-Adjusted Prevalence of Myocardial Infarction According to Diabetes Status and Ethnicity

End point	Diabetic		Nondiabetic		Diabetes-adjusted ethnic odds ratio		<i>p</i>
	MA	NHW	MA	NHW	MA/NHW	95% CI	
Men							
ECG	13.5	19.8	3.2	4.6	0.65	0.37–1.15	0.14
Self-reported	12.6	13.3	3.1	4.0	0.79	0.51–1.22	0.29
Either	21.6	29.9	5.6	8.2	0.65	0.41–1.03	0.06
Both	4.8	2.6	0.5	1.8	0.58	0.21–1.59	0.29
Women							
ECG	6.3	4.5	1.8	1.1	1.49	0.65–3.33	0.34
Self-reported	4.2	7.2	1.1	1.2	0.99	0.52–1.89	0.96
Either	10.7	11.7	3.1	2.5	1.24	0.68–2.26	0.49
Both	0.0	0.0	0.2	0.1		Too few events	

MA, Mexican-American; NHW, non-Hispanic white; CI, confidence interval; ECG, electrocardiogram.

In men, the age- and diabetes-adjusted ethnic odds ratios (Mexican-American versus non-Hispanic white) for all four myocardial infarction end points were nearly identical (ranging from 0.58 for composite both to 0.79 for self-report). When the composite either end point was used as the end point, the magnitude of the ethnic odds ratio was almost statistically significant (OR, 0.65; 95% CI, 0.41–1.03; $p=0.06$).

In women, the age- and diabetes-adjusted ethnic odds ratios (Mexican-American versus non-Hispanic white) showed no consistent trends, and the adjusted odds ratios ranged from 0.99 for self-reported to 1.49 for ECG-documented myocardial infarction. None of these differences was statistically significant. It was not possible to compute an adjusted odds ratio with both the ECG-documented and self-reported myocardial infarction as a composite end point, because only two women experienced both of these events concurrently.

Multiple logistic regression analyses were performed in men to evaluate the magnitude of the ethnic effect on myocardial infarction as defined by either ECG criteria or by self-report after accounting for conventional cardiovascular risk factors. The adjusted ethnic odds ratio (Mexican-American versus non-Hispanic white) obtained from this model was 0.62 (95% CI, 0.38–1.01; $p=0.056$). When ECG-documented myocardial infarction alone was used as the end point, the estimated odds ratio was 0.60 ($p=0.09$), and for self-reported heart attack alone, the estimated odds ratio was 0.86 ($p=0.54$). Consideration of ethnic-covariate interaction terms did not provide any evidence for a differential effect of any of the cardiovascular risk factors between the two ethnic groups.

Discussion

The present report provides the first data on the prevalence of coronary heart disease in Mexican-Americans. The observed trends in myocardial infarction prevalence are consistent with previously documented differences in mortality due to cardiovascular causes, namely, that relative protection is

seen in Mexican-American men but not women. As in the case of mortality due to cardiovascular disease, which is 15–36% lower in Mexican-American men (Table 1), the age- and diabetes-adjusted prevalence of myocardial infarction is 21–42% lower in Mexican-American men than in non-Hispanic white men. Although a nearly significant difference was observed in only one of the four myocardial infarction end points examined ($p=0.06$ for the composite either end point) the magnitudes of the ethnic effects were quite similar for all four end points, and the differences were all in the anticipated direction.

There are other examples of populations having lower rates of cardiovascular disease than would be expected on the basis of their cardiovascular risk profiles. For example, CHD appears to be relatively uncommon among certain native American populations^{25–27} despite high prevalences of both diabetes mellitus and obesity. However, cholesterol levels and cigarette consumption tend to be low in these populations. The observed CHD experience of men in Honolulu,²⁸ Puerto Rico,²⁸ and Yugoslavia²⁹ was less than predicted based on their Framingham risk scores. Similarly, the incidence of cardiovascular disease in men enrolled in the Seven Countries Study was considerably less than that predicted using risk equations generated from investigations in US railroad men.³⁰

The hypothesis that genetic factors may confer relative protection against CHD in Mexican-American men is suggested by their shared ancestry with native Americans,^{31,32} who, as noted above, also have lower rates of CHD. If protective genetic factors are involved, however, they must either be expressed only in men or else must be overwhelmed by other factors in women. Also of interest is the fact that unlike Pima Indians, Mexican-American men do not have low cholesterol levels (Table 2).

A variety of social and cultural factors have been invoked to explain the failure of conventional cardiovascular risk factors to account for differences in CHD prevalence between populations. Some of these proposed sociocultural factors are cultural mo-

bility (changes in customs, values, and beliefs that occur with movement from one "social world" to another),³³ social stress,³⁴ and absence of social networks.³⁵ Studies of biethnic populations may provide particularly valuable insight into clarifying the role of some of these factors. For example, in a study of migrant Japanese living in Hawaii, Reed et al³⁶ examined the effects of acculturation (defined as cultural change from a traditional Japanese to a Western lifestyle) on CHD and found acculturation to be associated with the prevalence, but not incidence, of CHD. In Mexican-Americans in San Antonio, we have observed an inverse association between acculturation and prevalence of noninsulin-dependent diabetes mellitus.³⁷ The relationship between acculturation and CHD in Mexican-Americans has not yet been explored.

The protective effect of Mexican-American ethnicity among men was similar in diabetic and nondiabetic subjects. Of interest, the prevalence of myocardial infarction is less among diabetic Mexican-American men than diabetic non-Hispanic white men because the opposite ethnic difference is seen in the case of microvascular complications. Specifically, Mexican-American diabetics have a higher prevalence of retinopathy¹⁷ and proteinuria³⁸ as well as more severe glycemia^{2,39} than non-Hispanic white diabetics.

There are inherent limitations associated with the assessment of myocardial infarction in epidemiological studies. First, the end points used in this study measure slightly different manifestations of myocardial infarction. For example, ECG tracings may detect silent, as well as symptomatic, heart attacks. On the other hand, self-reported histories of physician-diagnosed heart attack may be more sensitive in detecting symptomatic myocardial infarctions that occurred long ago because abnormal ECGs may revert to normal over time. However, an important limitation of the self-report end point is that it may be influenced by access to medical care. In fact, in the San Antonio Heart Study, Mexican-Americans are less likely to have visited a physician in the past year than are non-Hispanic whites.⁴⁰ Last, neither of the myocardial infarction end points used in this study were validated through other means, such as a review of the medical charts. Despite these limitations, the ethnic differences observed in myocardial infarction prevalence were remarkably similar for both end points separately as well as for the composite end point defined by the presence of either criterion.

In conclusion, the present data on myocardial infarction prevalence, with previously published studies on mortality due to cardiovascular disease, suggest that Mexican-American men are at less risk of developing CHD than are non-Hispanic white men. This observation is of interest because Mexican-Americans generally have a less-favorable cardiovascular risk profile, including a higher prevalence of diabetes, than that of non-Hispanic whites. Thus, a potentially fruitful direction for future studies may

involve examination of the sociocultural factors related to CHD in Mexican-Americans.

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