Risk Factors for Mortality Among Patients With Diabetes

The Translating Research Into Action for Diabetes (TRIAD) Study

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OBJECTIVE — We sought to examine demographic, socioeconomic, and biological predictors of all-cause, cardiovascular, and noncardiovascular mortality in patients with diabetes.

RESEARCH DESIGN AND METHODS — Survey, medical record, and administrative data were obtained from 8,733 participants in the Translating Research Into Action for Diabetes Study, a multicenter, prospective, observational study of diabetes care in managed care. Data on deaths ($n = 791$) and cause of death were obtained from the National Death Index after 4 years. Predictors examined included age, sex, race, education, income, duration, and treatment of diabetes, BMI, smoking, microvascular and macrovascular complications, and comorbidities.

RESULTS — Predictors of adjusted all-cause mortality included older age (hazard ratio [HR] 1.04 [95% CI 1.03–1.05]), male sex (1.57 [1.35–1.83]), lower income (<$15,000 vs. >$75,000, HR 1.82 [1.30–2.54]; $15,000–$40,000 vs. >$75,000, HR 1.58 [1.15–2.17]), longer duration of diabetes (≥9 years vs. <9 years, HR 1.20 [1.02–1.41]), lower BMI (<26 vs. 26–30 kg/m^2, HR 1.43 [1.13–1.69]), smoking (1.44 [1.20–1.74]), nephropathy (1.46 [1.23–2.73]), macrovascular disease (1.46 [1.23–1.74]), and greater Charlson index (≥2–3 vs. <1, HR 2.01 [1.04–3.90]; ≥3 vs. <1, HR 4.38 [2.26–8.47]). The predictors of cardiovascular and noncardiovascular mortality were different. Macrovascular disease predicted cardiovascular but not noncardiovascular mortality.

CONCLUSIONS — Among people with diabetes and access to medical care, older age, male sex, smoking, and renal disease are important predictors of mortality. Even within an insured population, socioeconomic circumstance is an important independent predictor of health.

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Diabetes is the sixth leading cause of death in the U.S. (1), and age-adjusted mortality in people with diabetes is approximately twice that of people without diabetes (2). People with diabetes are at increased risk of death from causes specific to diabetes (e.g., acute metabolic events such as diabetic ketoacidosis), from cardiovascular disease (ischemic heart disease, heart failure), and from other causes (e.g., infectious diseases such as influenza and pneumonia) (2,3).

Despite the high and increasing prevalence of diabetes, diabetes is underreported on death certificates as the underlying cause of death, a contributing cause of death, or a significant condition related to death (1–6). Therefore, the optimal approach to determine mortality rates among people with diabetes is to use a prospective cohort design where the diagnosis of diabetes is established at the start and participants are followed until death. With this approach, investigators can verify the diagnosis of diabetes before death and are able to obtain information about risk factors directly from the participants and their medical records.

Before the release of the NHANES (National Health and Nutrition Examination Survey)-I National Epidemiologic Follow-Up Survey (1982–1984), mortality studies of individuals with diabetes were limited to select (nongeneralizable) populations or had low statistical power (small numbers of deaths) (7). Since the late 1980s, investigators interested in diabetes mortality have used the NHANES-I Follow-Up Survey and other national data to increase the generalizability of their findings and to study larger populations. Recent studies performed in the U.K. (8–10), Finland (11), Sweden (12), Italy (13), and the U.S. (14,15) have confirmed that mortality is substantially higher in people with diabetes than in those without diabetes. Unfortunately, specific risk factors for death often were not studied, and few studies examined risk factors for noncardiovascular mortality in individuals with diabetes. In addition, characteristics of the U.S. population and standards of care and available treatments have changed over time, underscoring the need for newer studies. Relative to people living in the U.S. in 1990, current inhabitants are more likely to be of nonwhite race/ethnicity, more obese, less likely to smoke, and more likely to be treated with effective blood pressure and lipid-lowering medications (16–18). Compared with diabetic patients in Europe, diabetic patients in the U.S. are more likely to be of nonwhite race/ethnicity, more obese, less likely to smoke, and more likely to be treated with effective lipid-lowering medications (19).

To further our understanding of mor-

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Abbreviations: NDI, National Death Index; TRIAD, Translating Research Into Action for Diabetes.

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

tality among people with diabetes, we conducted a 4-year follow-up study of mortality in the Translating Research Into Action for Diabetes (TRIAD) Study. TRIAD has carefully characterized a large population with diabetes using data from patients, physicians, and health care systems. The TRIAD population is well suited to this follow-up study because of its large size, detailed measures, and diversity in terms of age, sex, race/ethnicity, geography, and health status.

RESEARCH DESIGN AND METHODS

— TRIAD has been described in detail elsewhere (20). In brief, six centers collaborate with 10 managed health care plans and 68 provider groups that serve ~180,000 individuals with diabetes. Patients ≥18 years of age were sampled. Institutional review boards at each participating site approved the study. All participants provided informed consent.

A baseline survey was administered to all TRIAD participants either by computer-assisted telephone interview or in writing by mail. Medical records and health plan administrative data for each participant were also reviewed. TRIAD decedents were identified from electronic searches of the National Death Index (NDI) Plus (21). Deaths were verified by matching name, sex, date of birth, and social security number (available for 52% of participants) of the decedent with data supplied by the NDI. The sensitivity of NDI ranges from 87 to 98% (22). Different combinations of identifiers excluding social security numbers correctly identify 83 to 92% of dead persons (i.e., they classify individuals known to have died as deceased) and 92 to 99% of living persons, underscoring the accuracy of NDI Plus (23).

Vital status was determined for all TRIAD participants (n = 11,927) as of 1 January 2004. All information regarding ICD-10 codes for the underlying and contributing causes of death was derived from the NDI file. For our analyses, we included all TRIAD participants who had complete survey and medical record review data for all possible exposure variables (n = 8,733), 791 (9%) of whom were identified as having died before 1 January 2004. This excluded 27% of the original sample, 98% of whom did not consent to review of their medical records. The demographic characteristics (age, sex, race, income, education, smoking, and duration of diabetes) of the included population were similar to those of the entire cohort. Missing values for age, sex, race and ethnicity, education, income, duration of diabetes, BMI, and smoking status from the patient survey were imputed using single imputation with the transcan function in S-PLUS (edition 6.1; Insightful, Seattle, WA). Variables that were imputed had <15% missing data.

Main outcome measures and covariates

We used National Center for Health Statistics definitions to classify cardiovascular versus noncardiovascular causes of death and the American Heart Association definitions to subclassify cardiovascular causes of death according to the underlying cause (1,24). The categories were as follows: cardiac (ICD-10 code I00–09, I11, I13, I20–25, I27, and I30–52), cancer (C00–99), diabetes (E10–14), cerebrovascular disease (I60–69), respiratory (J00–09 and J19–99), all other cardiovascular disease (I10, I12, I14–19, I28–29, I53–59, and I70–99), infections (A00–99 and B00–99), accidents/suicides/assaults (V00–99, W00–99, X00–99, and Y00–99), renal failure (N17–19), influenza/pneumonia (J10–18), and others (all other ICD-10 codes).

We investigated all-cause mortality, cardiovascular mortality (ICD-10 codes I00-I99 for underlying cause of death), and noncardiovascular mortality (all other ICD-10 codes for underlying cause of death). Covariates included age; sex; race; education; income; duration and treatment of diabetes; BMI; current smoking; history of hypertension, dyslipidemia, macrovascular disease, retinopathy, nephropathy, and peripheral neuropathy, measured at baseline; and Charlson index. Macrovascular disease included (from medical record review) history of transient ischemic attack, cerebrovascular accident, angina, myocardial infarction, congestive heart failure, other coronary heart disease or coronary artery disease, or peripheral vascular disease. The Charlson index, an extensively studied, accurate, and valid measure that weights various comorbid conditions by the strength of their associations with mortality, was used to quantify the comorbidity burden (25,26).

Statistical analyses

Bivariate analyses were performed for all-cause mortality and separately for cardiovascular and noncardiovascular mortality. We summarized continuous variables using means and tested differences between means using t tests. We summarized categorical variables using frequencies and tested their differences by χ^2. Unadjusted hazard rate ratios (HR) were constructed using each variable singularly in a Cox proportional hazards model predicting all-cause mortality, cardiovascular mortality, or noncardiovascular mortality.

Before constructing the models, we assessed Spearman's correlations between all possible exposure variables. Although education and income were correlated (r = 0.52), we included both in the multivariable models because they may have different predictive properties when used to model health-related outcomes (27). The Charlson index was correlated with history of macrovascular disease (r = 0.49), retinopathy (r = 0.35), nephropathy (r = 0.40), and peripheral neuropathy (r = 0.37), but because the Charlson index is constructed from a weighted examination of comorbid conditions, this was not unexpected (26). We decided to keep these four variables as well at the Charlson index in the model because the index represents more than just those diseases and provides a valid measure of comorbidity (26).

To simultaneously adjust for all possible covariates predicting mortality, we constructed multivariable Cox proportional hazards models. For these analyses, all-cause mortality, cardiovascular mortality defined by underlying cause of death, and noncardiovascular mortality were modeled separately, with each compared with the total alive population. We included the variables that were significant in bivariate tests of association between each predictor and the outcomes of interest. We also included smoking, which was found in previous studies to predict mortality in persons with diabetes (2). We used Cox proportional hazards models with a dummy variable for each health plan and provider group combination to account for the clustered study design (health plan, provider group, and participant levels) and the correlation among participant characteristics within health plans and provider groups. All analyses were performed using SAS (version 9.1; Research Triangle Institute, Research Triangle Park, NC).

RESULTS

— Of the 8,733 individuals included in our analyses, 791 (9%) died before 1 January 2004. The average length of follow-up was 3.7 years. A total

Table 1—*Distributions for factors associated with mortality in the TRIAD population, 2000–2004*

Characteristic	Percent distribution of characteristics in the TRIAD cohort	Percent distribution of characteristics in decedents (all causes)
n	8,733	791
Age	61 ± 13	69 ± 11
Sex (male)	4,061 (47)	428 (54)
Race or ethnicity		
Non-Hispanic white	3,673 (42)	404 (51)
Hispanic	1,419 (16)	97 (12)
African American	1,453 (17)	152 (19)
Asian/Pacific Islander	1,413 (16)	77 (10)
Other	775 (9)	61 (8)
Education		
Some high school or less	2,090 (24)	281 (35)
High school graduate	2,551 (29)	228 (29)
Some college	2,478 (28)	187 (24)
College graduate or more	1,614 (18)	95 (12)
Income		
<$15,000	2,740 (31)	372 (47)
$15,000–$40,000	2,200 (25)	260 (33)
$40,000–$75,000	2,192 (25)	107 (14)
>$75,000	2,180 (25)	52 (7)
Duration of diabetes (years)		
<9	4,293 (49)	274 (35)
≥9	4,440 (51)	517 (65)
Diabetes treatment		
Diet or exercise only	673 (8)	65 (9)
Oral medication	5,367 (61)	397 (50)
Oral medication plus insulin	1,069 (12)	112 (14)
Insulin only	1,624 (19)	217 (28)
BMI (kg/m^2)		
<26	1,803 (21)	223 (28)
≥26 to <30	2,289 (26)	193 (24)
≥30 to <35	2,366 (27)	213 (27)
≥35	2,275 (26)	162 (20)
Currently some or every day smoker	1,604 (18)	163 (21)
Hypertension	6,224 (71)	643 (81)
Dyslipidemia	4,599 (53)	383 (48)
Macrovascular disease	2,977 (34)	510 (65)
Retinopathy	1,559 (18)	210 (27)
Nephropathy	1,620 (19)	240 (30)
Peripheral neuropathy	1,511 (17)	204 (26)
Charlson index		
<1	331 (4)	10 (1)
≥1 to <2	2,940 (34)	96 (12)
≥2 to <3	2,538 (29)	148 (19)
≥3	2,924 (33)	537 (68)

Data are means ± SD or *n* (%).

of 54% of decedents were men, and the distribution of age (in years) at death was 25–44 (1%), 45–64 (27%), 65–84 (63%), and ≥85 (8%). Of decedents, 12% were Hispanic, 19% black, 51% non-Hispanic white, 10% Asian/Pacific Islander, and 8% of other race/ethnicity (Table 1). The underlying causes of death were listed as cardiac (34%), cancer (20%), diabetes (12%), other (11%), cerebrovascular disease (6%), respiratory (5%), infections (4%), all other cardiovascular disease (3%), accidents/suicides/assaults (2%), renal failure (2%), and influenza/pneumonia (1%). Of the 791 deaths, 336 (42%) had a cardiovascular cause listed as the underlying cause of death, and 455 (58%) had a noncardiovascular cause listed as the underlying cause of death; 534 deaths (68% of the total) had any cardiovascular cause listed in either part I or part II of the death certificate (ICD-10 codes I00–99).

The unadjusted characteristics of the entire TRIAD cohort with diabetes and those who died are shown in Table 2. In analyses of associations with all-cause, cardiovascular, and noncardiovascular mortality, we found that 13 variables predicted all three outcomes: older age, male sex, non-Hispanic white race (vs. Hispanic or Asian/Pacific Islander), lower education, lower income, greater duration of diabetes, lower BMI, hypertension, macrovascular disease, retinopathy, nephropathy, peripheral neuropathy, and a higher Charlson score (Table 2). For all-cause mortality, we also found associations with other race and treatment with insulin (Table 2). For cardiovascular mortality, we also found associations with treatment with insulin. Finally, for noncardiovascular mortality, we also found an association with other race (Table 2).

In fully adjusted multivariable analyses that examined risk for all-cause, cardiovascular, and noncardiovascular mortality, we found that older age, male sex, lower income, lower BMI, smoking, and nephropathy predicted all three outcomes (Table 2). For all-cause mortality, non-Hispanic white race (vs. Hispanic), longer duration of diabetes, macrovascular disease, and higher Charlson score also predicted mortality (Table 2). For cardiovascular mortality, longer duration of diabetes and macrovascular disease also predicted mortality. Noncardiovascular mortality was also associated with non-Hispanic white race (vs. black race or other) and higher Charles score (Table 2).

CONCLUSIONS — Our findings that older age, male sex, smoking, nephropathy, and cardiovascular disease predicted all-cause mortality are consistent with previous studies. While obesity is highly predictive of diabetes, we, like others, found that being underweight is a greater predictor of mortality among patients with diabetes (28,29). Despite an earlier finding of worse risk factor control (30), we did find that the Hispanic race was associated with lower all-cause mortality. This "Hispanic paradox" has been shown in other mortality studies (31). While

Table 2—*Unadjusted and adjusted* Cox proportional hazards regression models predicting all-cause, cardiovascular, and noncardiovascular mortality in the TRIAD population* (n = *8733), 2000–2004*

Characteristic	Unadjusted HR (95% CI) for all-cause mortality	Adjusted HR (95% CI) for all-cause mortality	Unadjusted HR (95% CI) for cardiovascular mortality	Adjusted HR (95% CI) for cardiovascular mortality	Unadjusted HR (95% CI) for noncardiovascular mortality	Adjusted HR (95% CI) for noncardiovascular mortality
n	791 of 8,733	791 of 8,733	336 of 8,278	336 of 8,278	455 of 8,397	455 of 8,397
Age (years)	1.06 (1.05–1.07)	1.04 (1.03–1.05)	1.07 (1.06–1.08)	1.05 (1.04–1.06)	1.06 (1.05–1.06)	1.04 (1.03–1.05)
Sex (ref. female)	1.39 (1.21–1.60)	1.57 (1.35–1.83)	1.34 (1.08–1.66)	1.54 (1.21–1.94)	1.46 (1.21–1.75)	1.65 (1.35–2.02)
Race/ethnicity (ref. non-Hispanic white)						
Hispanic	0.61 (0.49–0.77)	0.72 (0.55–0.96)	0.58 (0.41–0.82)	0.65 (0.41–1.01)	0.63 (0.47–0.84)	0.74 (0.51–1.06)
African American	0.94 (0.78–1.13)	0.80 (0.64–1.00)	1.01 (0.76–1.34)	0.87 (0.61–1.23)	0.89 (0.69–1.14)	0.71 (0.53–0.97)
Asian/Pacific Islander	0.51 (0.40–0.65)	0.81 (0.59–1.13)	0.46 (0.32–0.69)	0.85 (0.51–1.41)	0.53 (0.38–0.72)	0.78 (0.52–1.20)
Other	0.71 (0.54–0.93)	0.80 (0.60–1.06)	0.97 (0.68–1.40)	1.13 (0.76–1.68)	0.52 (0.35–0.78)	0.58 (0.38–0.89)
Education (ref. college graduate or more)						
Some high school or less	2.35 (1.86–2.96)	1.28 (0.98–1.68)	2.68 (1.85–3.87)	1.31 (0.86–1.99)	2.22 (1.64–2.99)	1.28 (0.90–1.82)
High school graduate	1.55 (1.22–1.97)	1.13 (0.88–1.47)	1.68 (1.15–2.46)	1.16 (0.77–1.74)	1.48 (1.09–2.01)	1.14 (0.81–1.59)
Some college	1.30 (1.01–1.66)	1.17 (0.91–1.51)	1.45 (0.99–2.16)	1.26 (0.84–1.88)	1.20 (0.87–1.96)	1.09 (0.78–1.53)
Income (ref. >$75,000)						
<$15,000	3.53 (2.64–4.72)	1.82 (1.30–2.54)	4.67 (2.87–7.59)	2.20 (1.28–3.79)	3.07 (2.14–4.41)	1.60 (1.04–2.45)
$15,000–$40,000	2.51 (1.86–3.37)	1.58 (1.15–2.17)	3.11 (1.89–5.11)	1.78 (1.05–3.00)	2.23 (1.54–3.24)	1.44 (0.97–2.15)
$40,000–$75,000	1.37 (0.99–1.91)	1.19 (0.85–1.67)	1.64 (0.95–2.83)	1.33 (0.76–2.32)	1.24 (0.82–1.89)	1.07 (0.70–1.63)
Duration of diabetes (years) (ref. <9)						
≥9	1.88 (1.63–2.18)	1.20 (1.02–1.41)	2.28 (1.81–2.88)	1.37 (1.06–1.77)	1.68 (1.39–2.03)	1.08 (0.87–1.33)
Treatment of diabetes (ref. diet or exercise)						
Oral medication plus insulin	0.76 (0.58–0.98)	0.61 (0.46–0.80)	0.89 (0.58–1.38)	0.72 (0.45–1.14)	0.68 (0.49–0.94)	0.51 (0.36–0.72)
Oral medication only	1.08 (0.80–1.47)	0.70 (0.50–0.98)	1.42 (0.87–2.32)	0.88 (0.52–1.52)	0.90 (0.61–1.33)	0.57 (0.37–0.87)
Insulin only	1.40 (1.06–1.85)	0.84 (0.62–1.14)	1.74 (1.10–2.75)	1.03 (0.62–1.71)	1.24 (0.87–1.76)	0.71 (0.48–1.04)
BMI (kg/m^2) (ref. ≥26 to <30)						
<26	1.50 (1.24–1.82)	1.43 (1.13–1.69)	1.61 (1.20–2.16)	1.50 (1.10–2.04)	1.45 (1.13–1.87)	1.35 (1.04–1.76)
≥30 to <35	1.06 (0.87–1.29)	1.21 (0.99–1.48)	1.11 (0.82–1.50)	1.30 (0.95–1.77)	1.03 (0.79–1.33)	1.19 (0.91–1.54)
≥35	0.83 (0.67–1.02)	1.11 (0.89–1.38)	0.86 (0.62–1.19)	1.21 (0.86–1.71)	0.80 (0.61–1.06)	1.05 (0.78–1.40)
Smoking	1.16 (0.97–1.37)	1.44 (1.20–1.74)	1.08 (0.82–1.41)	1.45 (1.09–1.95)	1.23 (0.98–1.53)	1.46 (1.15–1.86)
Hypertension	1.78 (1.49–2.13)	1.12 (0.92–1.36)	2.28 (1.69–3.06)	1.26 (0.92–1.73)	1.55 (1.24–1.95)	1.07 (0.83–1.36)
Dyslipidemia	0.85 (0.74–0.98)	0.76 (0.66–0.89)	0.91 (0.73–1.13)	0.77 (0.61–0.98)	0.81 (0.67–0.98)	0.73 (0.59–0.89)
Macrovascular disease	3.74 (3.23–4.32)	1.46 (1.23–1.74)	5.99 (4.70–7.64)	2.40 (1.79–3.20)	2.88 (2.39–3.47)	1.10 (0.88–1.37)
Retinopathy	1.72 (1.47–2.02)	1.04 (0.87–1.24)	1.61 (1.26–2.07)	0.90 (0.68–1.19)	1.84 (1.50–2.27)	1.17 (0.93–1.48)
Nephropathy	2.04 (1.75–2.37)	1.46 (1.23–1.73)	2.13 (1.69–2.68)	1.54 (1.18–2.01)	2.03 (1.66–2.48)	1.46 (1.16–1.84)
Peripheral neuropathy	1.70 (1.45–1.99)	0.90 (0.75–1.07)	1.64 (1.28–2.10)	0.77 (0.58–1.01)	1.78 (1.45–2.20)	0.99 (0.79–1.24)
Charlson index (ref. <1)						
≥1 to <2	1.08 (0.56–2.08)	1.44 (0.74–2.80)	0.57 (0.25–1.28)	0.67 (0.29–1.56)	2.28 (0.72–7.27)	3.27 (1.01–10.58)
≥2 to <3	1.96 (1.03–3.72)	2.01 (1.04–3.90)	1.15 (0.53–2.52)	0.91 (0.40–2.09)	3.89 (1.23–12.30)	4.57 (1.42–14.73)
≥3	6.66 (3.56–12.45)	4.38 (2.26–8.47)	4.36 (2.06–9.24)	1.84 (0.80–4.22)	12.93 (4.15–40.31)	10.92 (3.40–35.06)

*Adjusted for all characteristics listed in the table.

both lower income and poorer education were associated with all-cause, cardiovascular, and noncardiovascular mortality in unadjusted models, only lower income remained a significant predictor in models that included both. We assume a causal pathway where income mediates the relationship between education and mortality, and thus it was expected that adjustment for income attenuated the education-mortality relationship. Income has not previously been shown to predict mortality in people with diabetes after adjustment for biological risk factors. Our results suggest that even in an insured population with good access to health care, socioeconomic circumstances remain an important predictor of health.

Because cardiovascular disease is a major contributor to all-cause mortality in diabetes, one would expect the biological predictors of cardiovascular disease, such as hypertension, dyslipidemia, and current smoking, to be associated with both all-cause and cardiovascular mortality. Indeed, previous studies have shown age, sex, duration of diabetes, treatment with insulin, high blood pressure, and smoking to be associated with cardiovascular mortality (7,8,14,32). In contrast, we did not find treatment with insulin to be predictive of cardiovascular mortality.

Unlike earlier studies, we did not find significant associations between all-cause mortality and education, treatment with insulin, or history of hypertension (7,8, 14,32). A likely explanation for the absence of some previously reported predictors of cardiovascular mortality is our inclusion of additional variables in the models. For example, we included history of macrovascular disease as well as hypertension, dyslipidemia, and smoking. This has the effect of attenuating to nonsignificance the more remote factors. In addition, controlling for the Charlson index, which includes multiple comorbidities, may have attenuated the associations with some individual risk factors. The absence of some variables in these predictive models should not be interpreted to mean they are not causally important, given that our goal was to identify key predictors of mortality rather than to model causality. When we left macrovascular disease out of the model, hypertension became a significant predictor. We also observed joint confounding of smoking by all other variables in the multivariate model, which may have attenuated to nonsignificance some other causally important variables.

In our population, using only the underlying cause of death, more than one-half (58%) of the deaths were not related to cardiovascular disease. Noncardiovascular mortality has not often been studied in individuals with diabetes. Studies have reported that a longer duration of diabetes and treatment with oral hypoglycemic medications, as opposed to insulin, were associated with lower noncardiovascular mortality (7,32). We, too, found that treatment with oral antidiabetic agents with or without insulin was associated with lower noncardiovascular mortality. We hypothesized that risk factors for cardiovascular disease would not be associated with noncardiovascular mortality, and this was generally the case. As expected with competing risks for mortality, history of macrovascular disease was not significantly associated with noncardiovascular mortality.

The major limiatation of this study was its examination of a select population. Because our population consisted of those with physician-diagnosed diabetes for at least 18 months before the survey, our sample did not include individuals with new-onset diabetes. The study sites were not representative of all managed care populations and may not be generalizable to settings outside of managed care. Furthermore, the TRIAD population had good access to and quality of care, which may have attenuated the impact of biological risk factors. This high quality of care and the focus on prevention of cardiovascular disease may have accounted for the lower rate of cardiovascular mortality in the TRIAD population compared with other U.S. population samples (2).

In conclusion, we have shown the importance of demographic risk factors such as age, sex, and income on short-term, all-cause, cardiovascular, and noncardiovascular disease mortality. We have also shown that even in an insured population where diabetes and cardiovascular risk factors are well controlled (30), socioeconomic circumstance is an important independent predictor of mortality.

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