# 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.


#### Abstract

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 ( $\geq 9$ years vs. $<9$ years, HR $1.20[1.02-1.41]$ ), lower BMI ( $<26$ vs. $26-30 \mathrm{~kg} / \mathrm{m}^{2}$, HR 1.43 [1.13-1.69]), smoking (1.44 [1.20-1.74]), nephropathy (1.46 [1.232.73]), macrovascular disease ( 1.46 [1.23-1.74]), and greater Charlson index ( $\geq 2-3 \mathrm{vs} .<1$, HR 2.01 [1.04-3.90]; $\geq 3$ 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 ageadjusted 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 metabolicevents 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 under-

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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 Systeme International (SI) units and conversion factors for many substances.
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reported 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. (810), 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 (1618). 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 lipidlowering medications (19).

To further our understanding of mor-
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 $\sim 180,000$ individuals with diabetes. Patients $\geq 18$ 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 participantseither by computerassisted 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 100-09, I11, I13, I20-25, I27, and I30-52), cancer (C00-99), diabetes (E10-14), cerebrovascular disease (160-69), respiratory (J00-09 and J19-99), all other cardiovascular disease (I10, I12, I14-19, I2829, 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 (J1018), and others (all other ICD-10 codes).

We investigated all-cause mortality, cardiovascular mortality (ICD-10 codes I00-199 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 $\chi^{2}$. 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, 20002004

| Characteristic | Percent distribution of characteristics in the TRIAD cohort | Percent distribution of characteristics in decedents (all causes) |
| :---: | :---: | :---: |
| $n$ | 8,733 | 791 |
| Age | $61 \pm 13$ | $69 \pm 11$ |
| 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) |
| $\geq 9$ | 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 ( $\mathrm{kg} / \mathrm{m}^{2}$ ) |  |  |
| $<26$ | 1,803 (21) | 223 (28) |
| $\geq 26$ to $<30$ | 2,289 (26) | 193 (24) |
| $\geq 30$ to $<35$ | 2,366 (27) | 213 (27) |
| $\geq 35$ | 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) |
| $\geq 1$ to $<2$ | 2,940 (34) | 96 (12) |
| $\geq 2$ to $<3$ | 2,538 (29) | 148 (19) |
| $\geq 3$ | 2,924 (33) | 537 (68) |

Data are means $\pm$ 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 $\geq 85$ ( $8 \%$ ). 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 allcause 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

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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 ed-ucation-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 onehalf ( $58 \%$ ) 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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## References

1. Arias E, Anderson RN, Kung HC, Murphy SL, Kochanek KD: Deaths: final data for 2001. Natl Vital Stat Rep 52:1-115, 2003
2. Geiss LS, Herman WH, Smith PJ: Mortality in non-insulin-dependent diabetes. In Diabetes in America. 2nd ed. Bennett PH, Boyko EJ, Cowie CC, Harris MI, Stern MP, Reiber GE, Eds. Bethesda, MD, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, 1995, p. 233-258 (NIH publ. no. 95-1468)
3. McEwen LN, Kim C, Haan M, Ghosh D, Lantz PM, Mangione CM, Safford MM, Marrero D, Thompson TJ, Herman WH: Diabetes reporting as a cause of death: results from the Translating Research Into Action for Diabetes (TRIAD) study. Diabetes Care 29:247-253, 2006
4. Bild DE, Stevenson JM: Frequency of recording of diabetes on U.S. death certificates: analysis of the 1986 National Mortality Followback Survey. J Clin Epidemiol 45:275-281, 1992
5. Will JC, Vinicor F, Stevenson J: Recording of diabetes on death certificates: has it improved? J Clin Epidemiol 54:239-244, 2001
6. Thomason MJ, Biddulph JP, Cull CA, Holman RR: Reporting of diabetes on death certificates using data from the UK Prospective Diabetes Study. Diabet Med 22:1031-1036, 2005
7. Kleinman JC, Donahue RP, Harris MI, Finucane FF, Madans JH, Brock DB: Mortality among diabetics in a national sample. Am J Epidemiol 128:389-401, 1988
8. Vaccaro O, Stamler J, Neaton JD: Sixteenyear coronary mortality in black and white men with diabetes screened for the Multiple Risk Factor Intervention Trial (MRFIT). Int J Epidemiol 27:636-641, 1998
9. Laing SP, Swerdlow AJ, Slater SD, Botha JL, Burden AC, Waugh NR, Smith AW, Hill RD, Bingley PJ, Patterson CC, Qiao Z, Keen H: The British Diabetic Association Cohort Study, II: cause-specific mortality in patients with insulin-treated diabetes mellitus. Diabet Med 16:466-471, 1999
10. Roper NA, Bilous RW, Kelly WF, Unwin NC, Connolly VM: Cause-specific mortality in a population with diabetes: South Tees Diabetes Mortality Study. Diabetes Care 25:43-48, 2002
11. Koskinen SV, Reunanen AR, Martelin TP, Valkonen T: Mortality in a large popula-tion-based cohort of patients with drugtreated diabetes mellitus. Am J Public Health 88:765-770, 1998
12. Weiderpass E, Gridley G, Nyren O, Pen-
nello G, Landstrom AS, Ekbom A: Causespecific mortality in a cohort of patients with diabetes mellitus: a populationbased study in Sweden. J Clin Epidemiol 54:802-809, 2001
13. de Marco R, Locatelli F, Zoppini G, Verlato G, Bonora E, Muggeo M: Cause-specific mortality in type 2 diabetes: the Verona Diabetes Study. Diabetes Care 22: 756-761, 1999
14. Gu K, Cowie CC, Harris MI: Mortality in adults with and without diabetes in a national cohort of the U.S. population, 1971-1993. Diabetes Care 21:11381145, 1998
15. Bertoni AG, Krop JS, Anderson GF, Brancati FL: Diabetes-related morbidity and mortality in a national sample of U.S. elders. Diabetes Care 25:471-475, 2002
16. Census UBot: Census of Population 1990, General Population Characteristics (1990 CP-1), and 1990 Census of Housing, General Housing Characteristics (1990 CH-1), report series published 19921993; and Summary Tape File (STF) 1A, series released 1991. Washington, DC, U.S. Census Bureau. Available from http://factfinder.census.gov/servlet/basic factsservlet. Accessed May 2003
17. Census UBot: Census 2000. Washington, DC, U.S. Census Bureau. Available from http://factfinder.census.gov/servlet/basic factsservlet. Accessed May 2003
18. Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, Marks JS: Prevalence of obesity, diabetes, and obesityrelated health risk factors, 2001. JAMA 289:76-79, 2003
19. Viberti G, Lachin J, Holman R, Zinman B, Haffner S, Kravitz B, Heise MA, Jones NP, O'Neill MC, Freed MI, Kahn SE, Herman WH: A Diabetes Outcome Progression Trial (ADOPT): baseline characteristics of type 2 diabetic patients in North America
and Europe. Diabet Med 23:1289-1294, 2006
20. TRIAD: The Translating Research Into Action for Diabetes (TRIAD) study: a multicenter study of diabetes in managed care. Diabetes Care 25:386-389, 2002
21. Centers for Disease Control and Prevention, National Center for Health Statistics: National Death Index (NDI), NDI Plus Searches. Available from http://www.cdc. gov/nchs/r\&d/ndi/what_is_ndi.htm. Accessed April 2003
22. Cowper DC, Kubal JD, Maynard C, Hynes DM: A primer and comparative review of major US mortality databases. Ann Epidemiol 12:462-468, 2002
23. Williams BC, Demitrack LB, Fries BE: The accuracy of the National Death Index when personal identifiers other than Social Security number are used. Am J Public Health 82:1145-1147, 1992
24. Luepker RV, Apple FS, Christenson RH, Crow RS, Fortmann SP, Goff D, Goldberg RJ, Hand MM, Jaffe AS, Julian DG, Levy D, Manolio T, Mendis S, Mensah G, Pajak A, Prineas RJ, Reddy KS, Roger VL, Rosamond WD, Shahar E, Sharrett AR, Sorlie P, Tunstall-Pedoe H: Case definitions for acute coronary heart disease in epidemiology and clinical research studies: a statement from the AHA Council on Epidemiology and Prevention; AHA Statistics Committee; World Heart Federation Council on Epidemiology and Prevention; the European Society of Cardiology Working Group on Epidemiology and Prevention; Centers for Disease Control and Prevention; and the National Heart, Lung, and Blood Institute. Circulation 108:2543-2549, 2003
25. Charlson ME, Pompei P, Ales KL, MacKenzie CR: A new method of classifying prognostic comorbidity in longitudinal studies: development and validation.

J Chronic Dis 40:373-383, 1987
26. de Groot V, Beckerman H, Lankhorst GJ, Bouter LM: How to measure comorbidity: a critical review of available methods. J Clin Epidemiol 56:221-229, 2003
27. Robbins JM, Vaccarino V, Zhang H, Kasl SV: Socioeconomic status and type 2 diabetes in African American and non-Hispanic white women and men: evidence from the Third National Health and Nutrition Examination Survey. Am J Public Health 91:76-83, 2001
28. Ross C, Langer RD, Barrett-Connor E: Given diabetes, is fat better than thin? Diabetes Care 20:650-652, 1997
29. Klein R, Klein BE, Moss SE: Is obesity related to microvascular and macrovascular complications in diabetes? The Wisconsin Epidemiologic Study of Diabetic Retinopathy. Arch Intern Med 157:650-656, 1997
30. Brown AF, Gregg EW, Stevens MR, Karter AJ, Weinberger M, Safford MM, Gary TL, Caputo DA, Waitzfelder B, Kim C, Beckles GL: Race, ethnicity, socioeconomic position, and quality of care for adults with diabetes enrolled in managed care: the Translating Research Into Action for Diabetes (TRIAD) study. Diabetes Care 28:2864-2870, 2005
31. Karter AJ, Gazzaniga JM, Cohen RD, Casper ML, Davis BD, Kaplan GA: Ischemic heart disease and stroke mortality in African-American, Hispanic, and nonHispanic white men and women, 1985 to 1991. West J Med 169:139-145, 1998
32. Ford ES, DeStefano F: Risk factors for mortality from all causes and from coronary heart disease among persons with diabetes: findings from the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study. Am J Epidemiol 133:1220-1230, 1991


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