Associations of Diabetes Mellitus With Total Life Expectancy and Life Expectancy With and Without Cardiovascular Disease

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Background: Diabetes mellitus is a recognized risk factor for cardiovascular disease (CVD) and mortality. However, limited information exists on the association of diabetes with life expectancy with and without CVD. We aimed to calculate the association of diabetes after age 50 years with life expectancy and the number of years lived with and without CVD.

Methods: Using data from the Framingham Heart Study, we built life tables to calculate the associations of having diabetes with life expectancy and years lived with and without CVD among populations 50 years and older. For the life table calculations, we used hazard ratios for 3 transitions (healthy to death, healthy to CVD, and CVD to death), stratifying by the presence of diabetes at baseline and adjusting for age and confounders.

Results: Having diabetes significantly increased the risk of developing CVD (hazard ratio, 2.5 for women and 2.4 for men) and of dying when CVD was present (hazard ratio, 2.2 for women and 1.7 for men). Diabetic men and women 50 years and older lived on average 7.5 (95% confidence interval, 5.5-9.5) and 8.2 (95% confidence interval, 6.1-10.4) years less than their nondiabetic equivalents. The differences in life expectancy free of CVD were 7.8 and 8.4 years, respectively.

Conclusions: The increase in the risk of CVD and mortality from diabetes represents an important decrease in life expectancy and life expectancy free of CVD. Prevention of diabetes is a fundamental task facing today’s society in the pursuit of healthy aging.

Arch Intern Med. 2007;167:1145-1151

GLOBALIZATION OF THE Western lifestyle led to diabetes mellitus being a major and progressive health care problem worldwide. By 2000, there were more than 171 million individuals with diabetes in the world, and this number is expected to double in 25 years. Many years of research have shown that diabetes poses an increased risk of mortality and morbidity among those who have it. For instance, diabetic subjects have a greater than 2-fold–increased risk of developing cardiovascular disease (CVD). However, there is limited information about the potential association of diabetes with total life expectancy (LE), LE with CVD, and LE without CVD. This association is not easy to predict, since the associations of different risk factors for CVD with LE and the association of LE with CVD can follow unexpected directions. For instance, hypertension is associated with shorter LE and more years of life spent with CVD. On the other hand, smoking is associated with a shorter LE but also with a decrease in LE with CVD, since smokers generally experience higher levels of non-CVD mortality at younger ages.

We aimed to calculate the association of diabetes mellitus at age 50 years (and older) with LE, with special attention to the number of years of life spent with and without CVD.

METHODS

Using data from the Framingham Heart Study (FHS), we built multistate life tables to calculate the associations of having diabetes in total LE and years lived with and without CVD at age 50 years in the general population.

DATA SOURCES

The FHS recruited 5209 (64% men) respondents aged 28 to 62 years, residing in Framing-
ham, Mass, between 1948 and 1951. The cohort has been examined biannually and followed for more than 46 years. A further description can be found elsewhere.7

STUDY SAMPLE

We selected 3 nonoverlapping follow-up periods of 12 years to allow analyses including all potential confounders in the relationships between diabetes and mortality and CVD. We considered that physical activity—among others—could be an important determinant of the evaluated associations of diabetes. In the FHS, physical activity was not measured at every evaluation; therefore, we selected periods that included a measurement of physical activity. The selected follow-up periods started at examinations 4 (1956-1958), 11 and 12 (1969-1973), and 19 and 20 (1985-1989). Using the pooling of the repeated observations method,8 we considered the follow-up information during the 3 periods, yielding a total of 9773 observation intervals. Participants may thus be followed during the 3 periods until the event (CVD or death) occurs or the subject is censored. However, follow-up time and diabetes status were measured anew for each interval. After excluding participants with missing data on potential confounders (n=592) and baseline CVD (n=148), 4121 subjects were available from examination 4, 3260 from examinations 11 and 12, and 1652 from examinations 19 and 20, yielding a total of 9033 observation intervals.

Ethical approval was not required because this study was a secondary data analysis.

ASSESSMENT OF DIABETES

Presence of diabetes was defined at baseline of each of the selected follow-up periods. In the FHS, a casual glucose rather than a fasting measure has been used for the diagnosis of diabetes so that the diagnostic criteria remain the same over time. Diabetes was defined by having a random glycemic measurement (measured using the Somogyi-Nelson method)9,10 of 200 mg/dL or greater (≥11.1 mmol/L) or being treated with insulin and/or oral hypoglycemic agents. In the 3 different baseline periods included in our analyses, medication use was evaluated by self-report rather than by medicines brought to the clinic.

OUTCOME ASSESSMENT

The primary outcome of our study was incident or fatal CVD. Cardiovascular disease was defined as the presence of 1 or more definite manifestations of coronary heart disease (angina pectoris, coronary insufficiency, myocardial infarction, and sudden or not sudden death as consequence of coronary disease), congestive heart failure, stroke, transient ischemic attack, and intermittent claudication. A panel of 3 physicians evaluated all events; agreement of all 3 was required. Clinical, electrocardiographic, and enzymatic information was used to establish the diagnosis of myocardial infarction. Sudden death from coronary heart disease was defined as that in an individual who had been apparently well and was observed to have died within 60 minutes of the onset of symptoms in the absence of any other cause. Stroke was defined as intracranial hemorrhage, cerebroembolus, and atherothrombotic brain infarction. Transient ischemic attacks were considered to have occurred when there was a history of documented, focal, neurologic deficit that lasted less than 24 hours. Intermittent claudication consisted of a cramping discomfort in one or both calves of the leg that was provoked by walking and relieved by rest. Symptoms were recorded by a physician interviewer using a structured questionnaire for uniform assessment. In addition, a second physician immediately confirmed all suspected cases of angina pectoris and intermittent claudication. More detail on the evaluation of cardiovascular outcomes in the FHS is available elsewhere.11

POTENTIAL CONFOUNDERS

All analyses were adjusted for age and stratified by sex. We considered the following potential confounders: education (eight grade or less or higher than eight grade), smoking (never, former, or current), marital status (single, married, widowed, separated or divorced), comorbidity present at baseline (cancer, left ventricular hypertrophy, arthritis, ankle edema, or pulmonary disease), physical activity (low, moderate, or high), total cholesterol level, and the examination at the start of follow-up (examinations 4, 11 and 12, or 19 and 20). Hypertension and body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) were considered as intermediate variables, since both are established risk factors for the development of diabetes and may represent a step in the causal chain between diabetes and CVD and mortality. The examination at the start of follow-up was included to correct for a potential cohort and period effect, since the participants could belong to 3 different periods of follow-up and different birth cohorts.

To evaluate physical activity levels, participants were asked to estimate the time spent in a typical day at various levels of activity: sleeping, resting, or engaged in light, moderate, and heavy activity. The reported levels of activity were weighted to reflect metabolically expended time and added to calculate a daily physical activity score. Further details can be found elsewhere.7 Hypertension was defined as systolic blood pressure of 140 mm Hg or higher or diastolic blood pressure of 90 mm Hg or higher.12 For BMI, the following 4 categories were defined: less than 18.5, 18.5 to 24.9, 25.0 to 29.9, and 30.0 or greater.13

DATA ANALYSIS

We created life tables to calculate the differences in LE and years lived with and without CVD by presence of diabetes. We used a period multistate life table. This type of life table combines information from people of different ages and from different birth cohorts.14,15 We considered 3 different health states: free of CVD, CVD, and death. Participants could experience the following transitions: from free of CVD to CVD or death and from CVD to death. Participants were not allowed to move back from a disease state (eg, from having CVD to not having CVD), and only the first entry into a state was considered.16

To evaluate the differences in risk of mortality and CVD among persons 50 years and older by presence of diabetes at baseline, we first calculated the overall sex- and age-specific rates for each transition. Hazard ratios (HRs) comparing diabetic with nondiabetic subjects were calculated using Poisson regression (“Gompertz” distribution) in 3 final models.13,14 Model 1 adjusted for age; model 2 adjusted for confounders that substantially changed the association of diabetes with CVD or mortality in addition to age; and model 3 adjusted for intermediate variables (hypertension and BMI) in addition to the variables in model 2.

Finally, we calculated 3 sets of transition rates for diabetic and nondiabetic subjects using the overall transition rates, the adjusted HRs for CVD and mortality, and the prevalence of diabetes by sex and presence of CVD. Similar calculations have been described previously7,13,14,15,17. The Excel worksheets (Microsoft Corp, Redmond, Wash) are available on request.

For our statistical analyses, we used STATA version 8.2 for Windows (StataCorp, College Station, Tex; 2003).

We built life tables stratified by sex and presence of diabetes. The multistate life table started at age 50 years and closed
at age 100 years. We calculated confidence intervals for all LEs and their differences using Monte Carlo simulation (parametric bootstrapping). To calculate the confidence intervals, we used @RISK software (MathSoft Inc, Cambridge, Mass; 1999), with 10,000 runs.

SENSITIVITY ANALYSES

In a previous report, we found an increasing size of the association of physical activity with mortality and CVD with decreasing lengths of follow-up. This effect was likely due to the misclassification of exposure, since physical activity levels change through time. Diabetes is a more definite diagnosis. We might hence expect that less misclassification would occur, but nevertheless we evaluated the effect of length of follow-up (10, 5, and 6 years) on LE estimates. Furthermore, we repeated our analyses considering a 20% and 50% decrease or increase on the HRs for the 3 transitions considered.

BASELINE CHARACTERISTICS

Overall, diabetic subjects were older compared with those without diabetes (mean age, 68 vs 59 years) (Table 1). In subjects with vs without diabetes, there were fewer women (49% vs 57%), fewer smokers (27% vs 41%), and twice as many participants with family history of diabetes (30% vs 16%) among the diabetic subjects. Diabetic subjects had a higher mean systolic blood pressure (149 mm Hg vs 137 mm Hg) and BMI (27.5 vs 26.0) compared with their counterparts without diabetes. Marital status was similar for diabetic and nondiabetic subjects as well as the distribution of education, physical activity level, diastolic blood pressure, cholesterolemia, and cancer at baseline (Table 1).

DIABETES MELLITUS AND RISK OF CVD AND DEATH

Among men and women, having diabetes represented an increased risk of developing CVD, of mortality among those with CVD, and of mortality among those free of CVD (Table 2). Hazard ratios changed only slightly after adjusting for potential confounders.

Besides age, we adjusted for the following variables: education level, marital status, smoking, cancer at baseline, physical activity levels, and the starting date of follow-up. Other variables such as total cholesterol and presence of left ventricular hypertrophy, arthritis, ankle edema, or pulmonary disease at baseline were also tested, but they were not included in the adjusted analysis because they did not alter the HRs for CVD and mortality.

After adjustment for age and selected confounders (model 2), the effect of diabetes mellitus was statistically significant for both sexes (P < .05, 2-sided) for 2 of the 3 transitions (incident CVD and CVD to death). Diabetic women had a 2.5-times increased risk of developing CVD and a 2.2-times increased risk of mortality once CVD had developed compared with nondiabetic women at a similar age (Table 2).

The directions of the associations were similar among men but slightly smaller. Further adjustment for intermediate variables (model 3) did not represent a substantial change in the associations of diabetes.

DIABETES MELLITUS AND LE

Total LE and LE free of CVD were significantly decreased among men and women at age 50 years with diabetes compared with their nondiabetic equivalents (Figure). Differences in number of years lived with CVD between nondiabetic and diabetic subjects were minimal and not statistically significant (Table 3).

Women and men with diabetes who were 50 years and older were expected to live on average 8.2 and 7.5 years less, respectively, than their nondiabetic equivalents. In women and men, diabetes led to 8.4 and 7.8 years less in LE free of CVD, respectively, and 0.2 and 0.3 (non-significant) years more in LE with CVD, respectively.

SENSITIVITY ANALYSES

The increased risk of CVD and mortality among diabetic subjects was already seen at 6 years of follow-up and was reasonably consistent for lengths of follow-up between 6 and 12 years. The magnitude of the association increased as the period of follow-up was reduced; the shorter the period, the higher the differences in LEs between diabetic and nondiabetic subjects (Table 4).
Men, compared with nondiabetic men, had more than doubled the risk of developing CVD and a 1.7 times higher risk of dying once CVD was present.

Life expectancy at age 50 years and older for diabetic women was 8.2 years less than for women at the same age but without diabetes. Diabetic women also lived on average more than 8 years less free of CVD. Although these differences in LEs were slightly smaller for men, the size of the association was very similar.

The larger total LE among nondiabetic subjects was predominantly the result of the larger number of years lived without CVD and a slightly shorter—but nonsignificant—LE with CVD.

The shorter CVD-free LE among diabetic subjects is due to their higher incidence of CVD combined with a higher risk of non-CVD mortality. We found no significant difference between the years spent with CVD between diabetic and nondiabetic subjects. This is not surprising, given that while those with diabetes are at a greater risk of developing CVD, once they have it, they are at a greater risk of dying.

The HRs that we used in our life tables fall well within the range of the published associations of diabetes with CVD and mortality. The sensitivity analysis illustrates the effects of smaller or larger associations of diabetes, none of which altered our conclusions.

We found that the risk of CVD and mortality was more increased, although at a lower level than previously described, for diabetic women than for diabetic men. Different mechanisms have been described to explain these sex differences. First, diabetes provokes worse levels of conventional risk factors among women. In addition, a preferential treatment for men with diabetes, compared with diabetic women, has been described. Diabetic men tend to receive more adequate pharmacological treatment for CVD prevention compared with their female counterparts.

Having diabetes at age 50 years and older represents not only a significant increase in the risk of developing CVD and mortality but also an important decrease in LE and LE free of CVD.

Women with diabetes had more than double the risk of developing CVD and, among those already with CVD, mortality compared with nondiabetic women. Diabetic men, compared with nondiabetic men, had more than double the risk of developing CVD and a 1.7 times higher risk of dying once CVD was present.

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ing that some of the data we used from the FHS are from decades (the 1950s to the 1980s) when statins were not available and the coverage with hypertension treatment was well below current standards, it is possible that the sex differences in treatment played a less significant role in our analyses. Finally, this difference could be because nondiabetic women have a lower risk of CVD compared with nondiabetic men, since additional risk factors (eg, dyslipidemia) are more prevalent in men than in women.

The differences in LE observed between diabetic and nondiabetic subjects is similar to those found in previous studies. Using data from NHANES I, Gu et al found that median LE was 8 years lower for diabetic subjects aged 55 to 64 years. Similarly, using cross-sectional data from the National Health Interview Surveys (NHIS) and Markov models, Narayan et al estimated that the presence of diabetes among non-Hispanic, 50-year-old men would result in a loss of 8 years in LE. However, none of these studies calculated the association of diabetes with years lived with and without CVD, and both studies had a shorter length of follow-up.

A strength of our study is that we used a long period of follow-up in a well-organized historic cohort. We updated information on covariates and outcomes. The diagnosis of diabetes was based on the combination of glycemia tests and pharmacological treatment of diabetes recorded biannually. This assures a reasonably correct and timely identification of subjects with the disease. Underdiagnosis is unlikely to have occurred. However, since we used information on diabetes status that was updated every 12 years, delayed diagnosis may have generated a nondifferential misclassification of exposure and hence underestimation of the association of diabetes with CVD and mortality. Indeed, differences in LE increased with consideration of shorter follow-up intervals (Table 4).

Some limitations of our study must be considered. Participants included in the FHS were mainly white; therefore, our results may not apply to other ethnic groups. However, because of a higher risk of diabetes in other ethnic groups, the public health burden of diabetes-associated loss in LE may be even greater in those populations. Furthermore, the historical character of the FHS limits the extrapolation of findings to the population at present. Great advances in health promotion and in the diagnosis, prevention, and treatment of CVD have occurred since the FHS started. Note, however, that we did not use data from the first examination, and our follow-up period includes data from the 1980s and 1990s. Nevertheless, the incidence of diabetes has largely increased in recent years owing to detrimental changes in lifestyle; hence, our calculations may underestimate the true association of diabetes with CVD and LE. Finally, the diagnostic criteria of diabetes that we used may have suboptimally identified all exist-

### Table 3. Life Expectancy (LE) in Years at Age 50 Years and Older, Stratified by Sex

<table>
<thead>
<tr>
<th>LE</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Total LE</td>
<td>27.9 (27.3 to 28.6)</td>
<td>28.8 (28.1 to 29.7)</td>
</tr>
<tr>
<td>Difference in total LE</td>
<td>NA</td>
<td>Ref†</td>
</tr>
<tr>
<td>LE free of CVD</td>
<td>21.2 (20.5 to 22.0)</td>
<td>22.0 (21.2 to 22.9)</td>
</tr>
<tr>
<td>Difference in LE free of CVD</td>
<td>NA</td>
<td>Ref†</td>
</tr>
<tr>
<td>LE with CVD</td>
<td>6.7 (6.2 to 7.1)</td>
<td>6.8 (6.2 to 7.4)</td>
</tr>
<tr>
<td>Difference in LE with CVD</td>
<td>NA</td>
<td>Ref†</td>
</tr>
</tbody>
</table>

Abbreviations: CVD, cardiovascular disease; NA, not applicable; Ref, reference.
*Data are given as years (95% confidence interval). All life expectancies have been calculated with hazard ratios adjusted for age, education level, marital status, smoking, cancer at baseline, starting date of follow-up, level of physical activity, body mass index, and hypertension.
†Differences are calculated using the “no diabetes” group as the Ref.
‡Differences are significant at the P<.05 level.

### Table 4. Effect of Different Lengths of Follow-up on Life Expectancy at Age 50 Years and Older, Stratified by Sex

<table>
<thead>
<tr>
<th>Length of Follow-up</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>6 y of follow-up</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total LE</td>
<td>29.1 (29.0 to 29.3)</td>
<td>35.0 (34.8 to 35.2)</td>
</tr>
<tr>
<td>Diff total LE†</td>
<td>−8.2 Ref</td>
<td>−8.4 Ref</td>
</tr>
<tr>
<td>Diff LE free of CVD†</td>
<td>−7.8 Ref</td>
<td>−8.6 Ref</td>
</tr>
<tr>
<td>Diff LE with CVD†</td>
<td>−0.4 Ref</td>
<td>0.2 Ref</td>
</tr>
<tr>
<td>10 y of follow-up</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total LE</td>
<td>29.2 (29.1 to 29.4)</td>
<td>35.4 (35.2 to 35.6)</td>
</tr>
<tr>
<td>Diff total LE†</td>
<td>−8.2 Ref</td>
<td>−8.7 Ref</td>
</tr>
<tr>
<td>Diff LE free of CVD†</td>
<td>−7.1 Ref</td>
<td>−8.7 Ref</td>
</tr>
<tr>
<td>Diff LE with CVD†</td>
<td>−1.1 Ref</td>
<td>0.0 Ref</td>
</tr>
<tr>
<td>6 y of follow-up</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total LE</td>
<td>29.4 (29.3 to 29.5)</td>
<td>36.0 (35.8 to 36.2)</td>
</tr>
<tr>
<td>Diff total LE†</td>
<td>−8.7 Ref</td>
<td>−9.7 Ref</td>
</tr>
<tr>
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</tr>
<tr>
<td>Diff LE with CVD†</td>
<td>−1.0 Ref</td>
<td>0.0 Ref</td>
</tr>
</tbody>
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Abbreviations: CVD, cardiovascular disease; Dif, difference; LE, life expectancy; Ref, reference.
*Data are given as years. All LEs have been calculated with hazard ratios adjusted for age, education level, marital status, smoking, cancer at baseline, starting date of follow-up, level of physical activity, body mass index, and hypertension.
†Differences are calculated using the “no diabetes” group as the reference.
ing diabetic cases. Therefore, it is possible that some diabetic subjects contributed to the nondiabetic group, generating a further underestimation of the deleterious association with diabetes.

CONCLUSIONS

We analyzed a large and continuously followed cohort and showed the detrimental effects of having diabetes mellitus at age 50 years and older. The life table perspective allowed us to demonstrate a major shortening of total LE free of CVD. Although this association was larger among women, diabetic men also had a large decrease in years lived without CVD. These findings underscore the importance of diabetes prevention for the promotion of healthy aging. Toward this end, it is essential to implement global strategies to change the current “Western” lifestyle and to promote the adoption of physical activity and healthy diets.36 In this regard, diet and lifestyle interventions that have proven successful should be considered for implementation in the general population.36 Taking into consideration that treatment of diabetes and its complications accounts for at least 10% of health care expenditure in many countries,2 effectively preventing diabetes will not only represent an increase in LE and years lived without CVD but also may represent important savings for health care, at least with respect to direct medical costs. Prevention of diabetes is a fundamental task facing today’s society, with the aim to achieve populations living longer and healthier lives.

Accepted for Publication: February 10, 2007.
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Author Contributions: Dr Franco as guarantor of this article accepts full responsibility for the integrity of the data and the accuracy of the data analysis, had full access to all the data in the study, and controlled the decision to publish. Study concept and design: Franco, Mackenbach, and Nusselder. Acquisition of data: Franco. Analysis and interpretation of data: Franco, Steyerberg, Hu, Mackenbach, and Nusselder. Drafting of the manuscript: Franco. Critical revision of the manuscript for important intellectual content: Franco, Steyerberg, Hu, Mackenbach, and Nusselder. Statistical analysis: Franco and Nusselder. Obtained funding: Mackenbach and Nusselder. Supervision: Franco, Steyerberg, Mackenbach, and Nusselder.

Financial Disclosure: None reported.

Funding/Support: Drs Franco, Mackenbach, and Nusselder were partly funded by the Netherlands Organization for Scientific Research (ZON-MW grant 014-91-054). Dr Hu is a receipt of American Heart Association Established Investigator Award.

Role of the Sponsors: The funding organizations did not participate in the design and conduct of the study; in the collection, management, analysis, and interpretation of the data; and in the preparation, review, or approval of the manuscript.

Acknowledgment: We thank the Framingham Heart Study coordinators for access to the original dataset. The Framingham Study is conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with the Framingham Heart Study Investigators. The manuscript has been reviewed by the NHLBI for scientific content and consistency of data interpretation with previous Framingham Heart Study publications, and significant comments have been incorporated prior to submission for publication.

REFERENCES


