Impact of Diabetes on Cardiovascular Disease Risk and All-Cause Mortality in Older Men

Influence of Age at Onset, Diabetes Duration, and Established and Novel Risk Factors

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Background: We have examined the influence of age at onset and duration on the impact of diabetes mellitus on cardiovascular disease risk and all cause-mortality among men aged 60 to 79 years.

Methods: A prospective study of 4045 men aged 60 to 79 years followed up for a mean of 9 years, during which there were 372 major coronary heart disease (CHD) events (fatal and nonfatal myocardial infarction [MI]), 455 deaths from cardiovascular disease, and 1112 deaths from all causes. Men were classified as having (1) no history of MI and diabetes, (2) late-onset diabetes (diagnosed at ≥60 years or undiagnosed diabetes [fasting blood glucose level, >126.1 mg/dL]), (3) early-onset diabetes (diagnosed before age 60 years), or (4) prior MI.

Results: Men who had both MI and diabetes were excluded. Both early and late onset of diabetes were associated with a significantly increased risk of major CHD events and all-cause mortality compared with nondiabetic men who had no CHD, even after adjustment for conventional risk factors and novel risk markers (levels of C-reactive protein and von Willebrand factor and renal dysfunction). Only men with early-onset diabetes (associated with a duration of 16.7 years) showed risk similar to those with previous MI and no diabetes. The adjusted relative risks (95% confidence intervals) for major CHD events were 1.00 (reference), 1.54 (1.07-2.21), 2.39 (1.41-4.05), and 2.51 (1.88-3.36) for groups 1 through 4, respectively.

Conclusion: Both early and late onset of diabetes are associated with increased risk of major CHD events and mortality, but only early onset of diabetes (associated with >10 years’ duration) appears to be a CHD equivalent.

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See Invited Commentary at end of article

Diabetes mellitus is associated with increased risk of cardiovascular disease (CVD), including coronary heart disease (CHD), in middle-aged and older populations, although the relative effects of diabetes on CVD are weaker among older adults. The increased CVD risk associated with diabetes is not fully explained by traditional CVD risk factors. Although diabetes is a well-established risk factor for CHD, whether diabetes alone is a CHD equivalent in assessing the risk of future CVD events is controversial. In some but not all studies, CHD risk among people with diabetes has been shown to be equivalent to that among people with prior myocardial infarction (MI). Moreover, the influence of age at onset and duration on the impact of diabetes on CVD risk is not fully established. Earlier studies have suggested that the impact of diabetes on mortality decreases with increasing age at onset. It has been reported that the characteristics of those diagnosed before age 65 years differ from the characteristics of those diagnosed after age 65 years, and some studies have suggested that onset at a later age is not associated with increased mortality. However, these findings have not been supported by all studies. The impact of late onset of diabetes on CHD risk in older adults, in whom absolute CHD risk is high, and the issue of whether late onset (and thus shorter duration) of diabetes confers a risk similar to that of prior MI have been less well studied. A better understanding of the relationship between diabetes and CHD in older people and in relation to diabetes duration could be important for strategies aimed at reducing cardiovascular risk in older adults with diabetes. We have examined the re-
relationship between age at onset of diabetes and risk of major CHD events, CVD, and all-cause mortality among men aged 60 to 79 years. Because markers of inflammation, endothelial dysfunction, and renal dysfunction are related to CVD and diabetes, we have assessed the contributing role of these factors and linked CVD risk markers with the increased CVD risk associated with diabetes. Our analyses may help reconcile apparently contradictory results on the risk of CHD in patients with diabetes.

METHODS

The British Regional Heart Study is a prospective study of CVD involving 7735 men aged 40 to 59 years drawn from 24 British towns who underwent screening from 1978 through 1980. The population studied was socioeconomically representative of British men but consisted almost entirely of white Europeans (>99%). In 1998 to 2000, all surviving men, then aged 60 to 79 years, were invited for a 20-year follow-up examination (Q20). All participants provided written informed consent to the investigation, and ethical approval was provided by all relevant local research ethics committees. The men completed a questionnaire that included information about their medical history and lifestyle behavior. They were instructed to fast for a minimum of 6 hours, during which time they were to drink only water and to attend for measurement at a prespecified time ranging from 8 AM to 6 PM. All men were asked to provide a blood sample, collected using a closed blood collection system (Sarstedt Monovette; Sarstedt, Numbrecht, Germany). Four thousand two hundred fifty-two men (77% of survivors) attended the Q20 examination. Blood measurements were available in 4045 men.

CVD AND NOVEL RISK FACTORS

At the Q20 examination, anthropometric measurements, including body weight, height, and waist circumference, were performed. Details of measurements and classification methods for smoking status; physical activity; body mass index; waist circumference; social class; blood pressure; levels of high-density lipoprotein cholesterol, triglycerides, and glucose; and measures of lung function (forced expiratory volume in 1 second and hemostatic and inflammatory markers) have been described elsewhere. Insulin resistance was estimated according to the homeostasis model assessment (HOMA). Predicted glomerular filtration rate (eGFR), estimated from serum creatinine levels using the Modification of Diet in Renal Disease equation developed by Levey et al, was used as a measure of renal function. C-reactive protein (CRP) level was assayed by ultrasonic nephelometry (Dade Behring, Milton Keynes, England). Plasma levels of von Willebrand factor (vWF) antigen were measured with enzyme-linked immunosorbent assays (DAKO, High Wycombe, England).

FOLLOW-UP

All men have been followed up for all-cause mortality, CVD morbidity, and the development of diabetes from the initial screening to June 2008. The analyses in this study are based on follow-up from the Q20 examination (1998-2000) through June 2008, a mean follow-up period of 9 years. Information on death was collected through the established tagging procedures provided by the National Health Service registers. A nonfatal MI was diagnosed according to World Health Organization criteria. Evidence regarding CHD events was obtained by reports from general practitioners, by biennial reviews of the patients’ notes (including hospital and clinic correspondence) through the end of the study period, and from postal questionnaires to surviving participants. Major CVD events include nonfatal MI or CHD death. Cardiovascular deaths include all those with codes 400 to 499 from the International Classification of Diseases, Ninth Revision. We also used a composite end point referred to as cardiovascular events, which included major CVD events or CVD death. New cases of diabetes were ascertained by postal questionnaires to surviving participants at year 5 and 12 to 14 years after the initial examination, by systematic biennial reviews of primary care records and by review of all death certificates for any reference to diabetes. All events in the period through June 2008 have been recorded, and follow-up has been achieved for 99% of the cohort.

MEN WITH PHYSICIAN-DIAGNOSED MI AND/OR DIABETES

At the Q20 examination, the men were asked whether a physician had ever told them that they had had an MI (ie, heart attack or coronary thrombosis) or been given the diagnosis of diabetes and, if so, what their age was at diagnosis. They were also asked about a number of other disorders. Men with prior MI consisted of those with physician-diagnosed MI at Q20 and those who had a major nonfatal MI event before Q20 based on the regular surveillance of the general practitioners’ records, including hospital and clinic correspondence (n=434). Diabetic participants included men with recall of a physician diagnosis of diabetes at Q20, confirmed cases of diabetes based on general practitioner’s records between the initial examination and Q20 completion (n=285), and men with a fasting blood glucose level of at least 126.1 mg/dL at Q20 (to convert glucose levels to millimoles per liter, multiply by 0.0555). Self-report of physician-diagnosed diabetes has been shown to be a valid measure of recording diabetes in the present study, agreeing with the results of medical record review in 97% of cases. Diabetic men without MI (n=414) were initially divided according to the time of diagnosis of diabetes as having (1) undiagnosed diabetes based on a fasting blood glucose level of 126.1 mg/dL or greater with no physician diagnosis at Q20, (2) a clinical diagnosis at 60 years or older, and (3) a clinical diagnosis before age 60 years.

On the basis of their combined diabetic and MI status at Q20, the men were divided into the following 4 groups:

1. Participants without diabetes and without MI (reference group) (n=3197);
2. Men without MI and with a diagnosis of diabetes at 60 years or older or with undiagnosed diabetes (late-onset diabetes; n=307);
3. Men with a diagnosis of diabetes before age 60 years without MI (early-onset diabetes; n=107); and
4. Men with a diagnosis of MI without (n=368) or with (n=66) diabetes (total, 434).

Characteristics of each group are described in Table 1. To assess whether diabetes is a CHD risk equivalent, the small number of men with both diabetes and MI (n=66) were excluded from analyses in Table 2. Men with undiagnosed diabetes were grouped with those diagnosed at 60 years or older because all these men with undiagnosed diabetes (minimum age, 60 years) would be at least 60 years of age when diagnosed. In a subsidiary analysis, we separated late onset of diabetes and examined separately the risk of those with undiagnosed diabetes and those diagnosed at 60 years or older. We also performed analyses dividing those with preexisting MI into...
those with early-onset MI (diagnosed before age 60 years) and those with late-onset MI (diagnosed at 60 years or older).

### STATISTICAL METHODS

We used a Cox proportional hazards model to assess the age- and multivariate-adjusted hazards ratios (relative risk [RR]) with 95% confidence intervals (CIs) for each category compared with the reference group. To assess the contributing role of established and novel risk markers, we adjusted in a stepwise manner. In the adjustment (model 2, Table 2), we included conventional biological risk factors that are well established as being associated with CHD. To assess the contribution of novel risk markers, we performed additional adjustments for documented novel risk markers for CVD (model 3), including markers of inflammation (CRP level), endothelial dysfunction (vWF level), and renal dysfunction (eGFR) because these have been shown to be independently associated with CVD risk in this study.20 In the adjustment, the following variables were fitted as categorical: smoking (never, long-term ex-smokers [≥15 years], recent ex-smokers [<15 years], and current smokers), social class (manual vs nonmanual work), physical activity (4 groups), alcohol intake (5 groups), lung function (lowest quartile of forced expiratory volume in 1 second vs rest), and eGFR (<60, 60-69, and ≥70 mL/min per 1.73 m²). Body mass index, blood lipid levels (high-density lipoprotein and total cholesterol), HOMA, and systolic blood pressure, CRP, and vWF levels were fitted as continuous variables. To assess duration of diabetes and risk of CVD and mortality, participants with undiagnosed diabetes were considered to have a duration of 0 years. Because duration of diabetes changes during the course of follow-up, duration of diabetes was fitted as a time-dependent covariate in the analyses presented in Table 3.

### RESULTS

During the mean follow-up period of 9 years, there were 372 major CHD events (including 263 CHD deaths) and 1112 deaths from all causes (including 455 attributed to cardiovascular causes) in the 4045 men. Table 1 shows the characteristics and mean levels of biological markers by the 4 diabetes/MI status groups. The average duration of diabetes mellitus in participants with early onset of diabetes was 16.7 years compared with 1.9 years in those with late onset (undiagnosed or diagnosis at ≥60 years). Those with early-onset diabetes showed signifi-
cantly higher levels of HOMA (P < .001), blood glucose (P < .001) and hemoglobin A₁c (P = .006) than those with late-onset diabetes.

**CARDIOVASCULAR DISEASE**

Table 2 shows the relationship between the 4 diabetes/MI status groups and incident major CHD events. Men with both diabetes and MI were excluded from the analyses. Men with diabetes or MI all showed significantly greater risk of major CHD than those with no MI and no diabetes, even after adjustment for conventional risk factors and lung function. Men with early onset of diabetes showed risk similar to those with a history of MI. Further adjustment for novel and other risk markers associated with diabetes status that have been shown to be independently associated with CVD risk in this study (CRP and vWF levels and eGFR) attenuated the association.

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**Table 2. Diabetes and MI Status and Rates per 1000 Person-years and Adjusted Hazards Ratios of Major CHD and CVD Events and All-Cause Mortality**

<table>
<thead>
<tr>
<th>Event</th>
<th>Study Group</th>
<th>No Prevalent Diabetes or Prior MI (n = 3197)</th>
<th>Men With Prevalent Diabetes With No Prior MI</th>
<th>Men With Prior MI With No Prevalent Diabetes (n = 368)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate (No. of events)</td>
<td>Major CHD (n = 353)</td>
<td>8.7 (229)</td>
<td>15.7 (36)</td>
<td>21.7 (18)</td>
</tr>
<tr>
<td>Age</td>
<td>Model 1</td>
<td>1.70 (1.20-2.42)</td>
<td>2.93 (1.81-4.74)</td>
<td>2.73 (2.08-3.56)</td>
</tr>
<tr>
<td>Model 2</td>
<td>1.69 (1.18-2.41)</td>
<td>2.86 (1.76-4.64)</td>
<td>2.62 (1.99-3.44)</td>
<td></td>
</tr>
<tr>
<td>Model 3</td>
<td>1.55 (1.09-2.11)</td>
<td>2.63 (1.56-4.42)</td>
<td>2.61 (1.96-3.49)</td>
<td></td>
</tr>
</tbody>
</table>

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**Table 3. Duration of Diabetes in 414 Diabetic Men Without Previous MI Aged 60-79 Years and Rates per 1000 Person-years and Adjusted HRs of Major CVD Events and All-Cause Mortality**

<table>
<thead>
<tr>
<th>Duration of Diabetes Mellitus, y</th>
<th>0-1</th>
<th>2-7</th>
<th>≥8</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of participants</td>
<td>202</td>
<td>103</td>
<td>109</td>
</tr>
</tbody>
</table>

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**Table 3.** Duration of Diabetes in 414 Diabetic Men Without Previous MI Aged 60-79 Years and Rates per 1000 Person-years and Adjusted HRs of Major CVD Events and All-Cause Mortality.

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Abbreviations: CHD, coronary heart disease; CVD, cardiovascular disease; MI, myocardial infarction.

a Each CVD event includes a fatal or nonfatal MI. We excluded 66 men with diabetes and MI (32 deaths and 19 major CHD events). Unless otherwise indicated, data are expressed as hazards ratio (95% confidence interval). Early and late onset of diabetes are described in Table 1.

b Includes age, smoking, alcohol consumption, social class, body mass index, physical activity, and previous stroke.

c Includes model 1 plus systolic blood pressure, high-density lipoprotein and total cholesterol levels, low forced expiratory volume in 1 second.

d Includes model 2 plus C-reactive protein and von Willebrand factor levels and estimated glomerular filtration rate.

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tions slightly, and the increased risks associated with diabetes and MI remained significant. When examined separately, participants with undiagnosed diabetes (duration, 0 years) and those diagnosed at 60 years or older (duration, 49 years) showed a similar risk of major CHD events after adjustment for conventional risk factors (model 2) compared with those with no MI and no diabetes (adjusted RR, 1.74 [95% CI, 1.06-2.86] and 1.63 [95% CI, 1.01-2.63], respectively). To increase power, we also examined the relationships with all CVD events (CVD deaths or nonfatal MI combined). Both late and early onset of diabetes were associated with significantly increased risk of CVD events, even after adjustment for conventional and novel risk markers. Most of the men with early-onset diabetes were diagnosed at 30 years or older. To exclude the possibility of including men with type 1 diabetes, we excluded those diagnosed before age 30 years (n=4). This exclusion made no difference to the findings.

Among men with diabetes, early onset showed a significantly increased risk of major CVD events compared with late onset (adjusted RR, 1.95 [95% CI, 1.08-3.53]) after adjustment for conventional and novel risk markers and remained significantly higher after additional adjustment for HOMA (adjusted RR, 2.05 [95% CI, 1.10-3.81]).

We separated the men with preexisting MI and no diabetes into those who developed MI before age 60 years (n=215) and those who developed MI at 60 years or older (n=153). Early onset of diabetes showed intermediate risk compared with early and late development of MI. Compared with those with no MI or diabetes, the RRs (95% CIs) for major CVD events after adjustment for conventional risk factors were 1.42 (1.05-1.91), 2.32 (1.49-3.61), 1.67 (1.18-2.36), and 2.98 (2.22-3.99) for late onset of diabetes, early onset of diabetes, late development of MI, and early development of MI, respectively. The difference in risk between early MI and early onset of diabetes was not significant (adjusted RR, 1.31 [95% CI, 0.76-2.25]).

### ALL-CAUSE MORTALITY

Men with diabetes and those with previous MI showed significantly higher total mortality than those with no diabetes and no MI, even after adjustment for conventional and novel risk markers, with the highest mortality risk in those with early onset of diabetes (Table 2).

### DURATION OF DIABETES

We also examined duration of diabetes and risk of major CVD events (CVD mortality or nonfatal MI) and total mortality among all diabetic participants without previous MI (Table 3). Only those with longer duration (≥8 years) showed a significantly increased risk of CVD and total mortality compared with those with less than 2 years’ duration after adjustment for established and novel risk markers. Further adjustment for HOMA made little difference to the findings. Because duration of diabetes and age at onset of diabetes in these men were highly correlated (83 men [76.1%] with ≥8 years’ duration were diagnosed before age 60 years), it was not possible to statistically discriminate between the effects of age at onset and duration.

**COMMENT**

There remains considerable confusion and debate over the extent to which diabetes mellitus does or does not reflect a CHD risk equivalent to that of a previous MI. Our present analysis shows clearly the importance of the timing of development of diabetes (linked to the duration of diabetes) on such risk. Specifically, later onset of diabetes (those diagnosed at ≥60 years, associated with a mean diabetes duration of 4.9 years and undiagnosed diabetes) had a CHD risk approximately half that in persons who developed diabetes before age 60 years and had a mean duration of 16.7 years. Moreover, the RRs for vascular events and mortality in persons with early onset of diabetes were comparable to those in men with prior MI, suggesting that a longer duration of diabetes may be necessary to raise risks toward a CHD risk equivalent.

These findings help reconcile data from prior studies. In the seminal report from Haffner and colleagues, in which they notably proposed diabetes as a CHD risk equivalent, mean diabetes duration was longer than 8 years in a population with an average age of 59 years. By contrast, in a much larger cohort and prospective analysis from Scotland, CVD mortality risk was approximately 3-fold lower in persons with newly diagnosed diabetes (mean age, 66 years) compared with patients with recent MI. In line with such findings, we have also shown that duration matters among diabetic individuals, with a duration of 8 or more years associated with significantly increased risk of CVD among diabetic participants independent of risk factors and insulin resistance (HOMA). Overall, our observations plus prior research extend data from previous studies suggesting that CHD risk in patients with diabetes escalates significantly with disease duration and approaches CHD risk equivalence only when disease duration is beyond 8 years.

There may be alternative explanations as to why late-onset diabetes (at ≥60 years) should be associated with a lower risk of CHD events than early-onset diabetes. Persons developing diabetes later in life may be phenotypically different (ie, less insulin resistant and retaining greater beta-cell dysfunction). Those with late-onset diabetes had lower HOMA compared with those with early-onset diabetes, but adjusting for HOMA did not account for the differential risk between the 2 diabetes groups. Adjustments for other risk factors, including a much more extensive list of variables than was used in previous studies, also did not alter findings (Table 2). Further work is needed to examine this issue, but data currently argue against easily identifiable phenotypic differences as the major explanation.

If diabetes duration, more than other features, is associated with an increase in CHD risk, what are the responsible mechanisms? A longer exposure to chronic hyperglycemia may be the explanation. However, other factors, such as worsening beta-cell function and thus in-
sulin insufficiency, could also play a role because endogenously released insulin has effects on many pathways/tissues beyond those relevant simply to control of glucose levels.21 Because chronic worsening hyperglycemia and insulin insufficiency generally coexist in persons with longer diabetes duration, it is difficult to determine their independent roles, and beta-cell function is not easy to measure. Irrespective of mechanisms, the clinical implication is that, although 10-year CHD risk for newly diagnosed diabetes may not be very high, CHD risk beyond 10 years or indeed lifetime risk will be much higher. This pattern of substantially worsening risk with time emphasizes the need to be aggressive with CHD risk reduction (ie, statin use and blood pressure modification) in patients with type 2 diabetes diagnosed at a relatively young age (eg, age 40 years).

Finally, neither adjustment for traditional risk factors nor a range of novel risk factors (including markers of inflammation, endothelial dysfunction, and renal dysfunction) explained the excess CHD risk in patients with known diabetes, particularly in those with early onset. These men still showed an almost 3-fold increase in risk after adjustment. These data concur with and extend recent findings from the Emerging Risk Factor Collaboration meta-analysis, which also showed that established risk factors and novel risk markers, including CRP and fibrinogen levels and eGFR, do not explain excess CHD risk in persons with diabetes.6

Our work has a number of strengths and limitations. The presentation of 4 groups of men with differing baseline characteristics according to 2 levels of diabetes status and prior MI status enabled us to show clear patterns of risk dependent on history. We also had a high level of ascertainment of events and deaths and access to baseline phenotyping extending beyond traditional risk factors to a wide range of novel measures. The present findings are based on standard epidemiological approaches (including the use of routine information sources for ascertainment of CHD events) that are likely to be valid.32 Our work concerns only men; further studies in women are needed, as well as extension to other ethnicities. Given that our study was conducted in elderly men (aged 60-79 years), it would be useful to repeat the present study in younger men with and without diabetes (eg, diabetes diagnosed before 50 years and at 50 years or older), but of course a much larger study would be required to achieve meaningful results given lower CVD event rates in younger individuals.

In conclusion, we have demonstrated that, among elderly men (aged 60-79 years), both early and late onset of diabetes are associated with increased risk of major CVD events and all-cause mortality, but only early onset of diabetes (associated with more than a decade of mean duration of diabetes) appears to be equivalent in risk of CVD to a previous MI. In the light of current trends of rising prevalence of type 2 diabetes combined with a decline in the average age at onset,33 these findings emphasize the critical importance of age at development of diabetes and thus of diabetes duration on cardiovascular risk and the seriousness of the CVD consequences of the emerging type 2 diabetes epidemic.

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Author Contributions: Dr Wannamethee had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Wannamethee and Sattar.

Acquisition of data: Whincup and Lennon. Analysis and interpretation of data: Wannamethee, Shaper, and Whincup. Drafting of the manuscript: Wannamethee and Sattar. Critical revision of the manuscript for important intellectual content: Wannamethee, Shaper, Whincup, Lennon, and Sattar. Statistical analysis: Wannamethee. Obtained funding: Wannamethee, Whincup, and Sattar. Administrative, technical, and material support: Lennon.

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REFERENCES


Diabetes and Cardiovascular Risk Equivalency

Do Age at Diagnosis and Disease Duration Affect Risk Stratification?

The term CHD risk equivalence for individuals with diabetes has become popular and controversial following the report by Haffner et al1 more than a decade ago of higher rates of cardiac events in persons with diabetes. For individuals in this category, the updated version of the National Cholesterol Education Adult Treatment Panel III advocates a target reduction of low-density lipoprotein cholesterol levels to less than 100 mg/dL (to convert to millimoles per liter, multiply by 0.0259) despite the lack of evidence to support this target value. Given the rising prevalence of diabetes and related atherosclerotic events since the publication of the widely cited study, the enthusiasm for this concept is further driven by the availabilities of lipid-lowering therapies (mainly statins), which lower rates of CHD events among individuals with high-risk profiles. Thus, basing target lipid levels on readily ascertained characteristics such as the presence or absence of diabetes appears practicable. However, this needs to be balanced against the potential harms and significant resource implications of advocating blanket treatment targets to all patients on the basis of a diagnosis of diabetes. In our enthusiasm to extrapolate the findings from Haffner et al1 to a host of population groups, we have somehow overlooked some of the study’s important limitations, namely, the lack of power to detect differences between the 2 groups of patients being studied. Furthermore, the study was restricted to self-selected Finnish patient groups aged 45 to 64 years. The significant heterogeneity to the risk of developing CHD in patients with diabetes was further evident by the observation from the Detection of Ischemia in Asymptomatic Diabetics (DIAD) Study.2 When targeting a “healthier” population of asymptomatic patients with diabetes, the observed annual cardiac event rate in this study was very low at 0.69%. In the Strong Heart Study, which included communities with high risk of developing diabetes and CHD, the rates of CHD in patients with diabetes appear to depend in part on coexisting risk factors,3 and only those with multiple risk factors had rates of CHD events equivalent to those of patients with established CHD. Not surprisingly, subsequent large observational studies from different population groups have provided contradictory findings, with some supporting diabetes as a CHD risk equivalence4 and others not.5,6

Most of the studies investigating the concept of diabetes risk equivalency have not taken into account the patient’s lifetime risk of developing CHD. For example, a middle-aged person who was found to have diabetes during early adulthood may have a higher lifetime CHD...