Use of national data sources in diabetes epidemiology

Health authorities worldwide have been collecting vast quantities of data for many years. Historically, such data—generally obtained for administrative, financial, and clinical reasons—have rarely been used for research because of problems with access, scale, and linkages. However, things are changing, with a move towards open data access, improved computing power, and a realisation by governments that routine data might be able to answer important health questions efficiently and cost-effectively if records are appropriately linked while maintaining anonymity. The UK, where patients can be traced from primary care consultations to death, offers one of the world’s most promising sources of routine data in terms of research potential. The key issue is whether such data can be used to produce good-quality research that is similar to, or even enhances, bespoke data collection. Up to now, evidence of this type of data being used in such a way to study diabetes has been scant, but this gap has now been addressed by Anoop Dinesh Shah and colleagues1 in The Lancet Diabetes & Endocrinology.

The investigators have linked longitudinal data for almost 2 million people aged 30 years or older without previous cardiovascular disease, across general practices, hospitals, the national register of acute coronary syndromes, and death registrations in the CALIBER study.2 They analysed the relation between type 2 diabetes status (recorded as positive in 34,198 individuals in the cohort) and twelve initial manifestations of incident cardiovascular disease. This study is the first, of which I am aware, to look at subtypes of cardiovascular disease outcomes in such fine detail. They report that peripheral arterial disease is the most common first presentation of cardiovascular disease for people with type 2 diabetes (reported in 992 [16%] of 6137 who had a cardiovascular event), and people with diabetes were almost three times as likely to have peripheral arterial disease as an initial presentation than were those without diabetes (10,074 [9%] of 107,501; adjusted HR 2.98 [95% CI 2.76–3.22]; p<0·0001). However, diabetes was not associated with sudden death, and was negatively associated with subarachnoid haemorrhage and abdominal aortic aneurysm, which provide intriguing contrasts with the other nine initial manifestations of cardiovascular disease analysed, all of which showed positive associations with diabetes. Importantly, the hazard ratios reported for other types of cardiovascular disease that are often analysed in clinical trials and observational epidemiology (ie, myocardial infarction and stroke) are broadly consistent with past cohort studies.3 4 Furthermore, the investigators’ data suggest that diabetes has more of an effect on cardiovascular disease in young women than in young men, consistent with results of the most recent large meta-analyses.5 6 Finally, in line with large epidemiological studies,3 4 Shah and colleagues’ results show attenuation of the effects of diabetes on cardiovascular disease with age. So the study not only shows that use of routine data can give sensible results, but also adds value in terms of fine detail and precision (narrow confidence intervals).

The study’s results should be interpreted with caution. Unusually, the investigators analysed initial presentations rather than all manifestations of, for example, peripheral arterial disease. Most researchers would include, in presentations of risk and relative risk, peripheral arterial disease events that came after other cardiovascular disease events. This limitation makes direct comparisons with earlier studies, including those in the preceding paragraph, approximate. However, since the risk of having several cardiovascular disease events is not high, the approximation should be quite accurate. Whether or not this method gives a more reasonable estimate of the true effect of diabetes, unencumbered by intervention after another cardiovascular disease event, is debatable—hazard ratios for diabetes might be expected to be lower than with the usual all-events method, because of the greater risk of most competing cardiovascular disease events in individuals with diabetes.

Shah and colleagues have shown that careful processing of routinely obtained linked national health data, and not a little hard labour,2 can produce an excellent basis for aetiological research.7 Their study suggests that the limited quality of routine data—including incomplete event ascertainment and missing data—can be overcome in observational analyses. The question remains as to how the new epidemiological results on subsets of cardiovascular disease outcomes in diabetes might affect practice. As the investigators...
imply, new clinical trials in diabetes should now consider including (at least) peripheral arterial disease as part of any composite cardiovascular disease outcome, as should cardiovascular disease risk scores for people with diabetes. The varying levels and directions of association for different initial manifestations of cardiovascular disease might well prompt readers to instigate new research.

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Obesity, diabetes, and the moving targets of healthy-years estimation

Many studies have attempted to quantify the effect of obesity on death, fueling a sustained controversy about which levels of bodyweight can harm health. However, many investigators have argued that life expectancy does not capture the essence of the damage that obesity causes across a lifetime and that better long-term metrics are needed to convey risk, judge interventions, and motivate behaviour. In The Lancet Diabetes & Endocrinology, Steven Grover and colleagues model the effect of diabetes and cardiovascular disease in people who are overweight or obese and show what is intuitively known, but not often quantified, about obesity—that its effect on the number of number of healthy-years lost is far greater than its effect on total years of life.

Constructing a model from cohort studies about the probability of transition to diabetes, cardiovascular disease, and death, Grover and colleagues study used data for obesity, blood pressure, glucose concentrations, lipid concentrations, and other risk factors from 3992 non-Hispanic white participants from the US National Heath and Nutrition Examination Surveys 2003–10 to estimate the life years and healthy life-years lost associated with different levels of overweight and obesity.

Among young adults aged 20–39 years, obesity (BMI 30–35 kg/m²) reduced healthy life-years by 11·8 years (95% CI 9·9–13·7) in men and 14·6 years (12·0–17·2) in women, and reduced life expectancy by 5·9 years (4·4–7·4) in men and 5·6 years (4·1–7·1) in women. Among young adults aged 20–39 years, being very obese (BMI >35 kg/m²) reduced healthy life-years by 18·8 years (95% CI 16·8–20·8) in men and 19·1 years (16·7–21·5) in women, and shortened male life expectancy by 8·4 years (7·0–9·8) and female life expectancy by 6·1 years (4·6–7·6). On the basis of available life tables, these data mean that from young adulthood, obese and very obese people will spend a quarter to a third of their remaining lives with diabetes or cardiovascular disease. Being overweight (BMI 25–30 kg/m²) was also associated with small but significant reductions in healthy life-years, probably because of this BMI category’s strong association with diabetes. These findings are generally consistent with previous studies showing that young adults who are obese have more than a 50% lifetime risk of diabetes and death, Grover and colleagues’ study model the effect of diabetes and cardiovascular disease in people who are overweight and show what is intuitively known, but not often quantified about obesity—that its effect on the number of healthy-years lost is far greater than its effect on total years of life.