

Epidemiology of type 2 diabetes

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The rising prevalence of type 2 diabetes is a public health problem as it is a major cause of coronary heart disease, end-stage renal disease, non-traumatic amputations and preventable visual loss. Epidemiology can contribute to understanding and dealing with this emerging epidemic in a number of different ways. Firstly, it can provide the basic descriptive data that allow the epidemic to be tracked. Secondly, it can contribute to understanding the aetiology and pathogenesis of the condition. Finally it can contribute to efforts to identify risk sub-groups and inform preventive action. The extent and rapidity of the rising prevalence of the condition is dramatic, but it does not affect all parts of the world equally. Wild et al estimated in 2004 that the prevalence of diabetes for all age-groups would rise from 171 million in 2000 to 366 million in 2030¹. In absolute terms, the rise is greatest in developing countries among people aged 45-64 years. In developed countries most people with diabetes are in the >65 year category and as this population group is projected to rise over the next 20 years, this is also the stratum in which the prevalence of diabetes will also increase most. Overall the growing epidemic hits most dramatically those countries least able to cope with the costs of dealing with the large numbers of people with the condition.

The global distribution of the prevalence of type 2 diabetes suggest that this disorder originates from a complex interaction between genetic susceptibility, early growth and programming and lifestyle behaviours throughout life². The quality of our information about the variation of prevalence and incidence of type 2 diabetes by place and person is in marked contrast to the clear gaps in our knowledge about variation of diabetes incidence over time. The apparent changes in prevalence^{3,4} and future population prevalence projections¹ are dramatic and the public media is full of reports of the increasing problems associated with diabetes. However, some authorities have surprisingly questioned whether there is truly an epidemic of diabetes⁵. In one sense, the demonstration of a rising absolute number of people with the condition over time in many countries is sufficient justification of the use of the term epidemic by virtue of its pressure on medical services. However, absolute prevalence estimates are driven by a number of factors, most particularly the age structure of the population. As the number of older people is rising in most countries, it follows that the absolute number of people with age-related disorders, of which type 2 diabetes is a clear example, will also rise. In order to remove the effect of ageing in the population as a cause for the epidemic, one needs to examine temporal trends in age and sex specific stratum prevalence

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rates⁶. These too clearly indicate a rising prevalence over time⁷. However, prevalence is also affected by other factors such as improved survival among the diabetic population and outward migration of healthy individuals and inward migration of at-risk groups⁶. Even if these explanations are discounted, most estimates of prevalence are based on clinical register rather than the true prevalence of the condition. The likelihood of being on a registers is a function of the completeness of that register as a record of all those with clinically recognised diabetes. It is possible that the advent of financial incentives linked to the number of people with diabetes as a part of a process of quality improvement in primary care in some countries has led to a greater proportion of people with clinically recognised diabetes being registered. The proportion of people who truly have diabetes who are clinically recognised is also subject to change and might have altered with the advent of early detection programmes. Early data from the 1960s and 70s⁸ suggests that only 50% of those with detectable diabetes are clinically recognised. More recent estimates from the 1990s⁹ and 2000s suggest that this proportion has not changed much.

Perhaps the clearest form of evidence of an epidemic of diabetes would come from repeated measurement of the incidence of the disease in the same population over time. There is some evidence in the United States to suggest that incidence has increased¹⁰, but it is surprisingly difficult to find such data. One issue relates to definitions as studies differ as to whether they study the true incidence of the disease, both biochemical and clinical, or more usually just the clinically detected disease. Studies with repeated oral glucose tolerance testing (OGTT) which are often described as being the optimal way of measuring true incidence have an additional complexity related to the frequency of testing. An OGTT is a poorly reproducible test and even in studies with short-term repeat testing, there is a high degree of variability¹¹. As with all physiological variables that are measured with error, such imprecision leads to regression to the mean. If we superimpose this day-to-day variability on an overall upward trajectory as in figure 1, we can readily see that the interval between repeat OGTTs will influence the incidence of disease if people are censored or classified as diabetic on the first occasion that they have a result beyond the diagnostic threshold. Thus we tend to observe higher incidence rates in studies using more frequent OGTTs as the way of assessing progression to diabetes. This is common in trials but the estimates of incidence will not be generalisable to the real world where repeat testing is much less frequent. This phenomenon is particularly important for those people with "pre-diabetes" defined as those with either impaired glucose tolerance or impaired fasting glucose. In trials of high risk subgroups who are tested frequently, high rates of progression are seen¹². More population-based studies with a longer interval between repeats tend to observe lower incidence rates. Indeed, in the Ely population-based

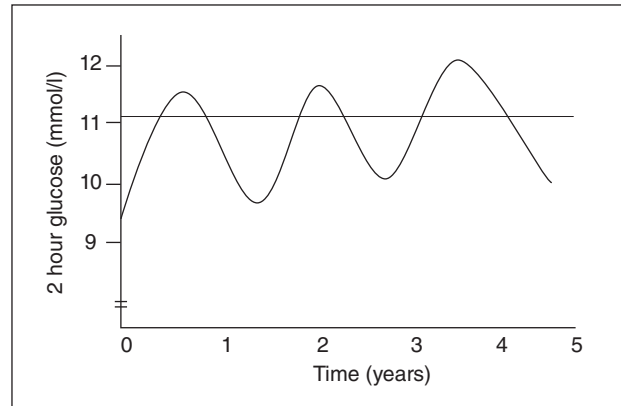


Fig. 1. Theoretical variation and upward shift of glucose levels over time showing how frequent testing with censoring at the diabetic threshold would result in higher incidence.

study only a small proportion of people with pre-diabetes progressed onto to get diabetes over 10 years with the majority reverting to normal glucose tolerance¹³. Overall the quality of the population-level information about the changing patterns of the incidence of type 2 diabetes over time is relatively poor and there has been an undue reliance on small scale cohort studies whose primary focus has been on the investigation of aetiological hypotheses rather than the production of high quality descriptive epidemiological data. Future population monitoring systems need to take into account multiple forms of data to allow the pattern of the emerging epidemic to be more accurately described.

The traditional epidemiological paradigm of incidence and prevalence is driven by an underlying notion that people can be classified as being normal or abnormal and that there is a clear point at which someone crosses that boundary. The variability issues of the standard measures of hyperglycaemia described above already show that it is difficult to determine when someone moves across the boundary. However, even the existence of a boundary at all is in question. Diabetes is typically defined as a distinct entity because of the threshold effect seen in the relationship between measures of hyperglycaemia and microvascular complications. However, no such threshold is demonstrable for the cardiovascular complications of hyperglycaemia¹⁴ and more recent studies have even questioned whether there is a threshold at all for microvascular disease¹⁵. As glucose is normally distributed in the population and the relationship between glucose levels and cardiovascular disease is linear, it follows that the greatest impact on public health will come from shifting the population mean of glucose levels rather than focusing on the relatively small group of people with high levels. In this sense, glucose is similar to blood pressure and cholesterol and the approach to prevention follows the principles outlined by Geoffrey Rose (fig. 2)¹⁶. Moving the emphasis away from individuals to populations also concentrates attention onto the determinants of glucose levels at the

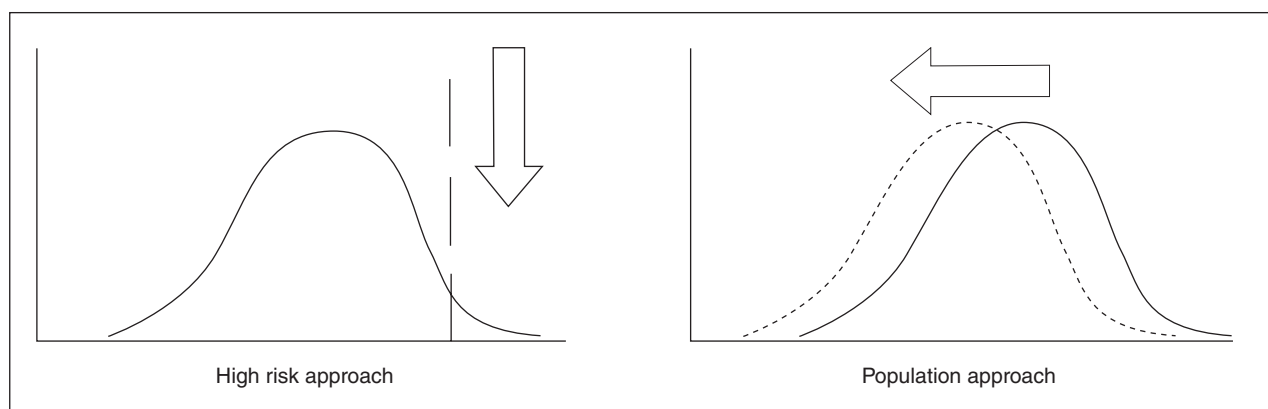


Fig. 2. Contrasting high risk and population approaches to prevention.

population level¹⁷, an understanding of the wider determinants of dietary and physical activity behaviour and on developing strategies for prevention that recognise the societal influences on these behaviours.

Conflict of interest

The author declares he has no conflict of interest.

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