Is the Recent Rise in Type 2 Diabetes Incidence From 1984 to 2007 Explained by the Trend in Increasing BMI?

Evidence from a prospective study of British men

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OBJECTIVE — To estimate the extent to which increasing BMI may explain the rise in type 2 diabetes incidence in British men from 1984 to 2007.

RESEARCH DESIGN AND METHODS — A representative cohort ratio of 6,460 British men was followed-up for type 2 diabetes incidence between 1984 (aged 45–65 years) and 2007 (aged 67–89 years). BMI was ascertained at regular intervals before and during the follow-up.

RESULTS — Between 1984–1992 and 1999–2007, the age-adjusted hazard of type 2 diabetes more than doubled (hazard ratio 2.33 [95% CI 1.75–3.10]). Mean BMI rose by 1.42 kg/m^2 (95% CI 1.10–1.74) between 1984 and 1999; this could explain 26% (95% CI 17–38) of the type 2 diabetes increase.

CONCLUSIONS — An appreciable portion of the rise in type 2 diabetes can be attributed to BMI changes. A substantial portion remains unexplained, possibly associated with other determinants such as physical activity. This merits further research.

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In the U.K., incidence of type 2 diabetes has increased by two-thirds in the last decade (1), and in the U.S. incidence has doubled over the last 30 years (2). Understanding the reasons for these unfavourable trends will help inform efforts to curb future type 2 diabetes increases. Marked rises in population adiposity have also occurred (3–5). However, few attempts have been made to quantify the contribution of changes in adiposity to the observed time trend in type 2 diabetes, partly reflecting the paucity of studies that have simultaneously monitored both type 2 diabetes and BMI levels in the same population over an extended period. We have estimated the proportion of the time-trend in type 2 diabetes incidence in British men over 24 years that may be explained by increasing population adiposity levels.

RESEARCH DESIGN AND METHODS

— Data came from the British Regional Heart Study (6), a socially and geographically representative cohort of middle-aged British men recruited between 1978 and 1980. The men were followed-up from baseline for all-cause mortality through National Health Service central registers and from 1984 for type 2 diabetes (the principal outcome) through a combination of biennial reviews of general practitioner (GP) records and self-report in questionnaires that were confirmed by GP records (7). Follow-up has been maintained for >99% of surviving men.

Type 2 diabetes incidence was compared in three consecutive follow-up periods, separated by intermittent questionnaires or examination sessions: period 1 from 1984 to 1992; period 2 from 1992 to 1999; and period 3 from 1999 to 2007 (Fig. 1). The principal exposure was BMI recorded in the questionnaire or examination at the start of each period (measurement techniques are detailed elsewhere [8]). BMI data were also available at recruitment in 1978–80. Analyses are based on 6,460 surviving men, aged 45–65 years at the start of the follow-up in 1984, with no prior diagnosis of diabetes. Men diagnosed within one year of the start of each period were excluded to limit the effect of reverse causality.

The proportion of the trend in type 2 diabetes hazard from period 1 to period 3 that may be statistically explained by increasing BMI was estimated by the expression $(\beta_0-\beta_1)/\beta_0$, where β_0 is the coefficient of an indicator for time period 3 in a Cox proportional hazard model for incident type 2 diabetes that only included time period, and β_1 is the coefficient of the indicator for period 3 in a Cox proportional hazard model adjusting additionally for time-varying BMI (as a continuous variable with all significant powers) (9). A 95% CI for this estimate was obtained using bias-corrected bootstrap resampling (10). Age was used as the underlying time scale in the models, which were thereby automatically adjusted for age.

RESULTS

— The hazard rate of type 2 diabetes increased more than twofold from period 1 (1984–1992) to period 3 (1999–2007) (hazard ratio[HR] 2.33[95% CI 1.75–3.10], adjusting for age) (Fig. 1). An estimated 25.9% (95% CI 16.5–38.3) of this increase in type 2 diabetes hazard could be statistically explained by a population-averaged age-adjusted increase in BMI from 1984 to 1999 of 1.42 kg/m^2 (95% CI 1.10–1.74). A smaller percentage, 20.5% (95% CI 12.6–31.4), could be explained by "lagged" BMI changes, i.e., BMI ~5–8 years prior to each follow-up

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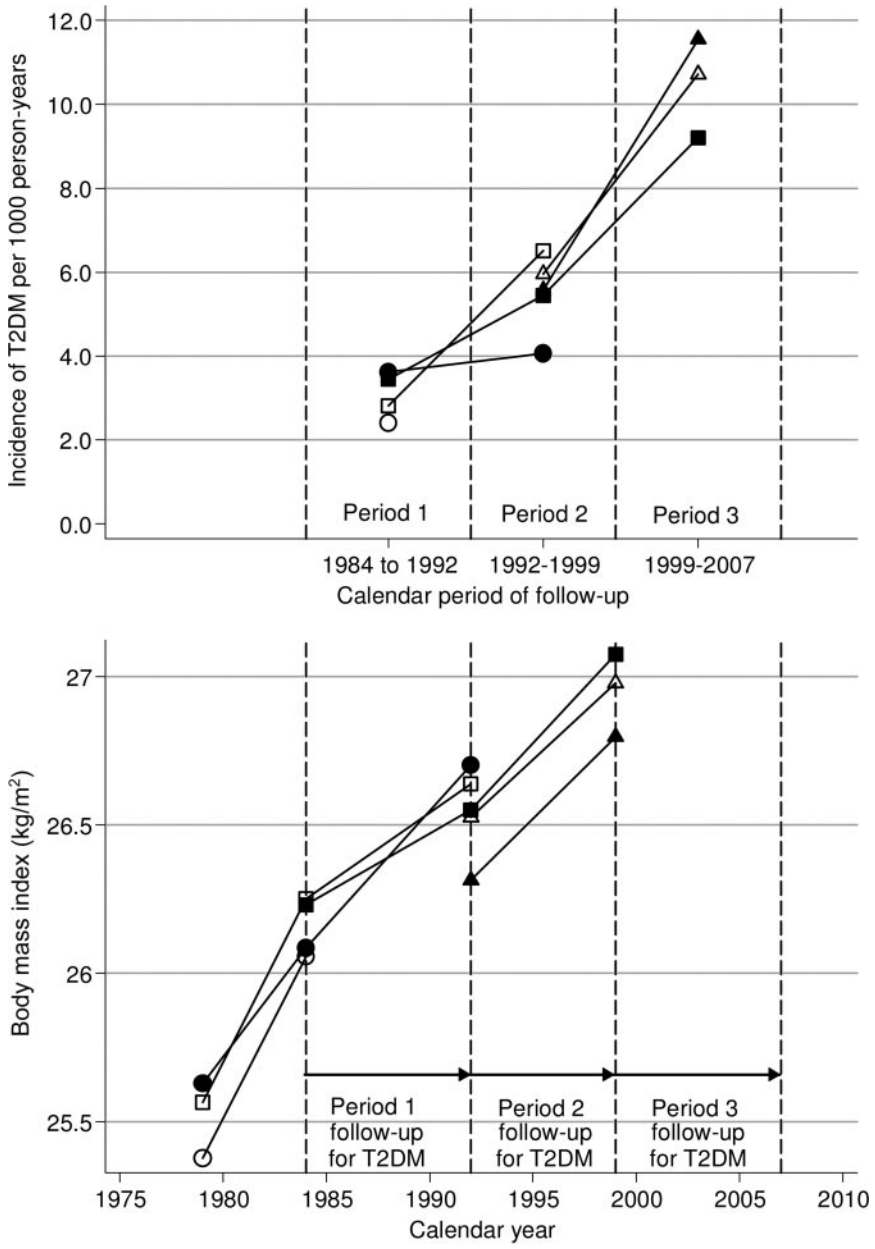

Figure 1—*Trend over time in incidence of type 2 diabetes and in mean BMI, by age-group.* ○*, age d 45–49 years;* ●*, age d 50–54 years;* □*, age d 55–59 years;* ■*, aged 60–64 years;* △*, aged 65–69 years;* ▲*, aged 70–74 years.*

period, corresponding to looking at the BMI increase over an earlier period from 1979 to 1992 of 1.13 kg/m^2 (95% CI 0.95–1.31). The combination of current and lagged measurements did not increase the contribution of BMI (percentage explained 25.8% [95% CI 16.8–38.1]).

The contribution of increasing BMI to the trend in type 2 diabetes incidence was examined separately in earlier and later portions of the follow-up. Between periods 1 (1984–1992) and 2 (1992–1999), incidence of type 2 diabetes increased by about a half (HR 1.59[95% CI 1.23–2.05]). Between periods 2 (1992–1999) and 3 (1999–2007), a similar increase was observed (HR 1.47[1.17–1.84]). Of the increase in type 2 diabetes risk between periods 1 and 2, 21.7% (95% CI 11.9–47.9) could be statistically explained by an age-adjusted increase in BMI from 1984 to 1992 of 0.40 kg/m^2 (95% CI 0.29–0.52). During periods 2 and 3, 31.1% (95% CI 16.8–80.6) of the increase in type 2 diabetes could be statistically explained by an age-adjusted increase in BMI from 1992 to 1999 of 0.61 kg/m^2 (95% CI 0.44–0.78).

CONCLUSIONS — Increasing adiposity (assessed by BMI) appears to have accounted for about a quarter of the increase in type 2 diabetes incidence in periods 1 and 3 between 1984 and 2007 in this survivor cohort of British men. Control and reversal of the rise in adiposity levels is therefore an important priority in controlling the diabetes epidemic. A substantial proportion of the rise in type 2 diabetes remained unexplained. This may partly reflect study limitations; including the use of BMI as opposed to central adiposity measures and possible overestimation of the type 2 diabetes increase due to improved case ascertainment, changing diagnostic criteria, and the changing method of case identification (self-report in earlier years versus later use of GP records). However, trends in other determinants of type 2 diabetes, including declining physical activity levels (11) and dietary changes (12), could be playing an important role. Physical activity has a strong protective effect on type 2 diabetes risk, which operates at least partly independently of BMI (13). Dietary factors associated with reducing type 2 diabetes risk include a high fiber diet (14), and daily consumption of fiber declined between 1987 and 2000 (12).

Studies assessing population attributable risks of adiposity in type 2 diabetes have reported estimates similar to those in the present analysis (15). However in such studies, which examine overall type 2 diabetes incidence in the population, the contribution of modifiable risk factors, such as adiposity, are necessarily affected by contributions of static variables including genetic factors. In contrast our analysis examines time trends in type 2 diabetes, attributable only to modifiable factors that have changed over time in the cohort, thus carrying more immediate public health implications. Time-trend studies in other populations are needed to verify the findings and establish the roles of other risk factors. The presence of other contributing factors would suggest the need for a more multifactorial approach to combat rising type 2 diabetes in the population.

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