

# The relative contributions of different levels of overweight and obesity to the increased prevalence of diabetes in the United States: 1976–2004<sup>☆</sup>

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## Abstract

**Background.** Policy makers are divided on whether to focus public health efforts to prevent type 2 diabetes on subpopulations at high risk or on the entire population. We examined the extent to which increases in the prevalence of overweight, obesity, and severe obesity have contributed to the increase in diabetes prevalence among U.S. adults between 1976–1980 and 1999–2004.

**Methods.** Using assembled data of 37,606 U.S. adults aged 20 to 74 years from 3 consecutive U.S. national surveys (NHANES II, III, and NHANES 1999–2004), we compared the body mass index distributions among prevalent diabetes cases over time and divided changes in prevalence of 5 diabetes-body mass index categories by changes in the diabetes prevalence observed in the total population.

**Results.** Prevalence of diabetes among adults aged 20 to 74 increased from 5.08% in 1976–1980 to 8.83% in 1999–2004. Of the 3.75 additional cases per hundred that existed in 1999–2004 as compared to 1976–1980, we estimated that –8% were among persons of normal or below normal weight (body mass index <25); 27% were among those who were overweight (body mass index 25 to 30); and 32%, 23%, and 26% among those with class I (body mass index 30 to 35), class II (body mass index 35 to 40), and class III obesity (body mass index >40), respectively. Thus, of the additional prevalent diabetes cases that existed in 1999–2004 as compared to 1976–1980, 81% were obese (i.e. body mass index >30) and 49% had class II or III obesity (body mass index >35), a group that increased in prevalence from 4% to 13% of the overall adult population.

**Conclusions.** The increases in diabetes prevalence over recent decades have been disproportionately comprised of persons with extreme levels of obesity.

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**Keywords:** Diabetes; Prevalence; Obesity

## Introduction

Although obesity and weight gain are established causes of type 2 diabetes, it is currently unclear how best to target weight-reduction interventions designed to reduce diabetes incidence (Colditz et al., 1995; Edelman et al., 1997; Ford et al., 1997; Zimmet et al., 2001; Tuomilehto et al., 2001; Knowler et al., 2002; Williamson et al., 2004). Major diabetes prevention trials have been limited to predominantly obese “high-risk” persons with impaired glucose tolerance (IGT) (Pan et al., 1997; Tuomilehto et

al., 2001; Knowler et al., 2002) and observational studies have highlighted the particular diabetes risks associated with extreme obesity (Field et al., 2001; McTigue et al., 2006). However, in two classic articles on public health approaches to disease control, Rose argued that the entire population, rather than only subgroups at high risk for a particular condition, should be targeted for intervention (Rose, 1985; Rose and Day, 1990).

Cohort studies of diabetes incidence have provided a rich body of literature (Colditz et al., 1995; Edelman et al., 1997; Ford et al., 1997; Leibson et al., 2001; Burke et al., 2003) on the association of BMI and diabetes and have shown, for example, that about half of incident diabetes cases are among people with a BMI >30 and that one-fourth to one-sixth are among those with a BMI greater than 35 (Leibson et al., 2001; Burke et al., 2003). However, previous studies have not estimated the relative impact of overweight and different levels of obesity on

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Table 1

Trends in the prevalence of 5 BMI categories and prevalence of diabetes according to BMI group among U.S. adults with diabetes, 1976–1980 through 1999–2004

| BMI category              | Proportion of U.S. adults in BMI categories |                        |                         | Prevalence of total diabetes (diagnosed and undiagnosed combined <sup>a</sup> ) |                              |                               |
|---------------------------|---|------------------------|-------------------------|---|------------------------------|-------------------------------|
|                           | 1976–1980<br>(n=11761)                      | 1988–1994<br>(n=14301) | 1999–2004<br>(n=11,270) | 1976–1980<br>(n=11761, 5856)  | 1988–1994<br>(n=14301, 6793) | 1999–2004<br>(n=11,270, 4911) |
| <25 (normal/under weight) | 54.4 (52.7, 56.0)                           | 45.6 (43.8, 47.4)      | 34.7 (33.4, 36.0)       | 3.1 (2.4, 3.8)  | 2.9 (2.3, 3.6)               | 4.3 (3.2, 5.3)                |
| 25–30 (overweight)        | 31.4 (30.2, 32.6)                           | 32.0 (30.8, 33.3)      | 33.9 (32.6, 35.3)       | 4.4 (3.5, 5.3)  | 6.4 (5.4, 7.4)               | 6.9 (5.8, 7.8)                |
| 30–35 (class I obesity)   | 9.9 (9.2, 10.6)                             | 14.3 (13.5, 15.2)      | 18.5 (17.5, 19.4)       | 9.6 (10.2, 22.4)  | 12.1 (10.2, 14.1)            | 11.6 (9.7, 12.8)              |
| 35–40 (class II obesity)  | 3.1 (2.8, 3.4)                              | 5.2 (4.5, 6.1)         | 7.8 (7.2, 8.5)          | 16.3 (10.2, 22.4)   | 19.4 (13.7, 25.1)            | 17.9 (14.7, 20.0)             |
| 40+ (class III obesity)   | 1.3 (1.0, 1.5)                              | 2.9 (2.4, 3.4)         | 5.1 (4.5, 5.8)          | 44.3 (24.3, 64.3)   | 18.4 (13.8, 23.0)            | 28.1 (21.9, 31.1)             |
| Total                     | 100   | 100                    | 100                     | 5.1   | 7.02                         | 8.8                           |

<sup>a</sup> Sample sizes within parentheses refer to the number in the examination sample used for computation of diagnosed diabetes, followed by the number used for computation of undiagnosed diabetes. All estimates are standardized by age and sex to the year 2000 census.

the trends in diabetes prevalence in the United States that have occurred over recent decades. Understanding the contributors to secular trends over time requires independent, serial cross-sections or cohorts. For this study, we compare the BMI composition among prevalent diabetes cases of recent national surveys (1999–2002) to that of previous decades (1976–1980) to estimate the contribution of different levels of obesity to recent trends in diabetes prevalence.

**Methods**

We assembled data from 3 consecutive nationally representative surveys, the NHANES II (1976–1980) and NHANES III (1988–1994) and from NHANES 1999–2004 (McDowell, 1981; National Center for Health Statistics, 1994, 2003). Our analyses are based on 11,761; 14,301; and 11,544 non-pregnant participants aged 20–74 in the 3 surveys, respectively who attended the mobile examination clinic. Prevalence of diagnosed diabetes was determined by self-report during an interview of all participants. Random sub-samples of 5856,

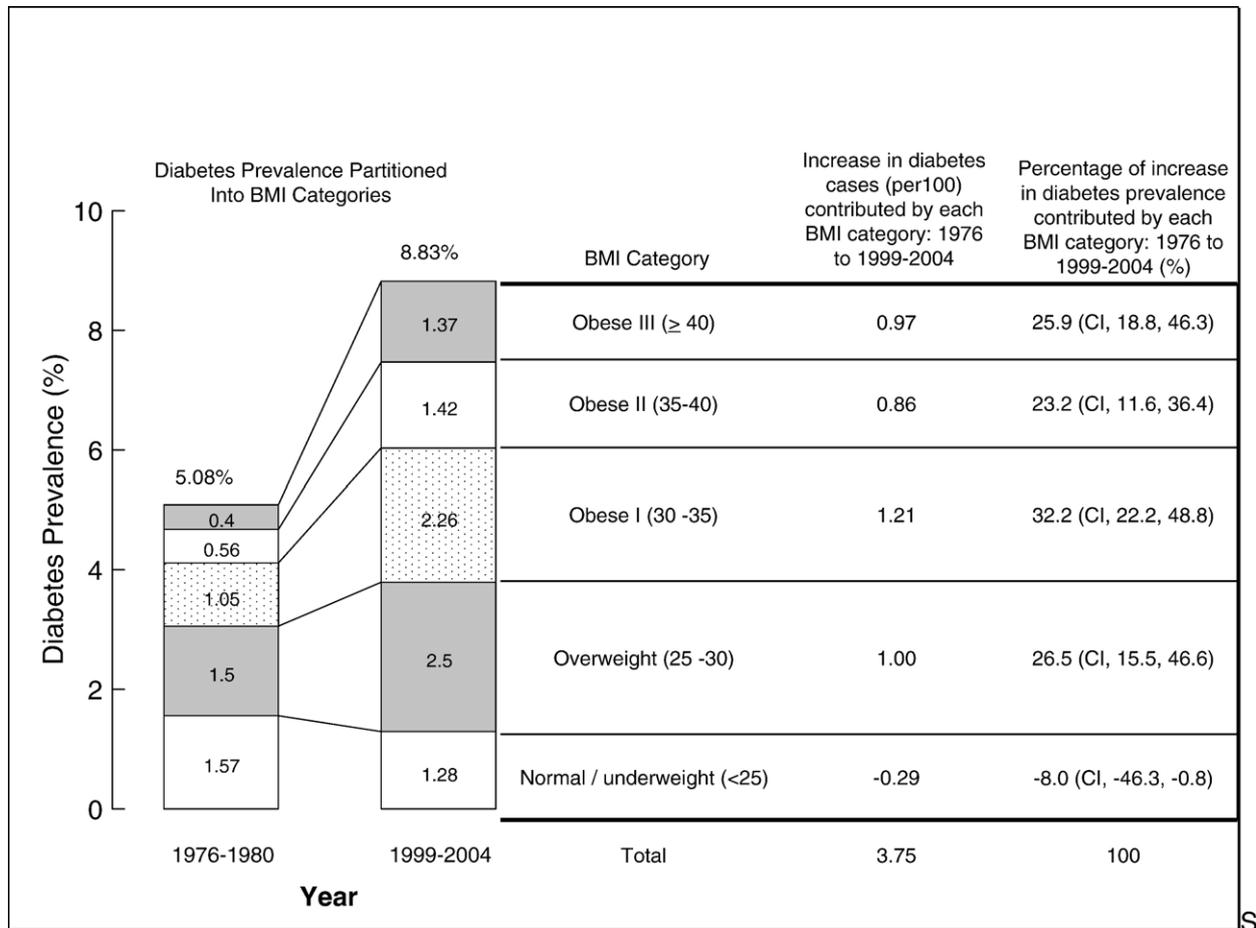


Fig. 1. Contribution of 5 BMI categories to the overall prevalence of diabetes per 100 U.S. adults in 1976–1980 and in 1999–2004. Sample size for BMI-diabetes estimates were as follows: NHANES II: 11,761 (total), 6043 (BMI<25), 3855 (overweight), 1302 (class I obese), 390 (class II obese), and 171 (class III obese). NHANES 1999–2004: 11,270 (total), 3566 (BMI<25), 3938 (overweight), 2218 (class I obese), 933 (class II obese), 615 (class III obese).

6793, and 4911 among the 3 surveys underwent a fasting blood glucose measurement for determination of undiagnosed diabetes (FBG > 126) (Flegal et al., 1991; Harris et al., 1998; Cowie et al., 2006). Prevalence of undiagnosed diabetes was adjusted to account for the fact that individuals with diagnosed diabetes were not part of the fasting morning sample denominator and appropriate survey weights were used such that the sum of sample weights for all diagnostic categories (no diabetes, undiagnosed diabetes, diagnosed diabetes) equal the total U.S. adult population aged 20–74 (Flegal et al., 1991; Harris et al., 1998; Cowie et al., 2006). We used subjects' measured weight and height to divide them into 5 body mass index (BMI, kg/m<sup>2</sup>) categories: normal or underweight (<25); overweight (25 to <30), class I obesity (30 to <35), class II obesity (35 to <40), and class III obesity (≥40).

To estimate the proportion of the increase in total diabetes prevalence contributed by changes in specific BMI categories, we compare the BMI composition of prevalent cases in 1999–2004 to that of 1976–1980. To do this, we first defined 6 mutually exclusive groups: 1 group of people without diabetes (irrespective of their BMI category) and 5 groups of people with diabetes grouped by their BMI status (i.e. percent with BMI < 25 and with diabetes; percent with BMI 25 to 30 and with diabetes, etc.) and calculated the age- and sex-adjusted percentage of each of these 6 groups in the U.S. population at the time of each survey. This step was conducted separately for diagnosed and undiagnosed diabetes and adjusted for age and sex to the U.S. 2000 census. These percentages and their standard errors were estimated using SUDAAN (version 9.0), using survey weights to account for the complex survey design. Second, we divided the change for each diabetes-BMI specific category between the 1st and 3rd survey by the increase in total diabetes prevalence for the overall population. We then aggregated diagnosed and undiagnosed diabetes for each of the BMI-specific subgroups such that total diabetes is the sum of diagnosed and undiagnosed diabetes. Standard errors for these resulting quotients (i.e. the estimated contribution of each group to the total excess prevalence) were computed using a simulation method that accounts for the correlation of the components (Buckland, 1984).

## Results

Consistent with published findings (Centers for Disease Control and Prevention; Gregg et al., 2004; Ogden et al., 2006), we found that the percentage of U.S. adults who were overweight increased only slightly from 1976–1980 to 1999–2004, but that the prevalence of class I and II obesity each more than doubled, prevalence of class III obesity quadrupled, and total diabetes prevalence increased from 5.1% to 8.8% (Table 1). Across all survey years, diabetes prevalence was strongly associated with BMI group. Compared with the diabetes prevalence among people of normal weight (3% to 4%), the prevalence was about 3 times as high among those with class I obesity (10% to 12%), 4 to 5 times as high among those with class II obesity (16% to 18%), and 6 to 10 times as high among those with class III obesity (18% to 44%).

In 1976–1980, 5.08% of U.S. adults had diabetes (see Fig. 1). When the 5.08 prevalent cases (per 100) were partitioned into BMI categories (see Fig. 1), most of the prevalent diabetes cases were either normal/underweight or overweight (1.57% and 1.50%, respectively), whereas 1.05% were class I obese with diabetes, 0.56% were class II obese with diabetes, and 0.4% were class III obese with diabetes (see Fig. 1). In 1999–2004, 8.83% of U.S. adults had diabetes, representing an increase of 3.75 prevalent cases per 100. The greatest relative increase in prevalent cases was among the most obese. Again partitioned into the respective BMI categories, 1.28% had diabetes and were normal/underweight (down 0.29% percentage points since NHANES II), 2.50% were overweight and had diabetes (up 1.00 percentage point), 2.26% were class I obese with diabetes

(up 1.21 percentage point), 1.42% were class II obese with diabetes (up 0.86 percentage point), and 1.37% were class III obese with diabetes (up 0.97 percentage point).

Thus, of the additional prevalent cases that existed in 1999–2004 compared to 1976–1980 (i.e. 3.75 additional cases per 100), 26.5% were among people who were overweight, and 32.2%, 23.2%, and 25.9% were among those with class I, class II, and class III obesity, respectively. (The percentages add to more than 100% because of the decreased diabetes prevalence among people of normal weight.) Eighty-one percent of the additional cases were among persons who were obese (BMI > 30), and 49% were among persons from the two highest BMI categories (BMI > 35).

When we conducted analyses with diabetes defined only by self-report, we found similar percentages. Of the additional cases of diagnosed diabetes that existed in 1999–2004, 20.0% were overweight, and 28.4%, 27.8%, and 21.4% were among those with class I, II, and III obesity, respectively.

## Discussion

We found that obese persons, particularly those of class II and III obesity, have been disproportionately represented in the increase in national diabetes prevalence over the past 25 years. Of the additional prevalent diabetes cases that exist in 1999–2004 as compared to 1976–1980, four-fifths have BMI ≥ 30 and about half of the additional diabetes cases have BMI ≥ 35, a group that comprised between 4% and 13% of the BMI distribution during this time period. Further, one-fourth of the additional prevalent diabetes cases had BMI > 40, a group comprising between 1% and 5% of the BMI distribution.

Our findings cannot explain why the increase in prevalent cases disproportionately came from the most obese segments of the population. Presumably this is a reflection of the rapid population increases in prevalence at the high end of the BMI distribution, combined with the strong causal link between diabetes and obesity. In other words, the population attributable fraction that obesity places on diabetes appears to have increased due to the increased prevalence of extreme obesity, presumably due to factors ranging from decreasing energy expenditure to increasing energy intake associated with glycemic-rich foods, high fat intake, increasing portion sizes, etc (Gross et al., 2004). However, several non-causal factors could also increase the prevalence of very obese persons in the U.S. diabetic population. We previously documented a greater increase in the ratio of diagnosed to undiagnosed diabetes among very obese persons, suggesting preferential ascertainment is one potential contributor (24). However, our sensitivity analyses showed that the contributions of BMI categories to additional diabetes cases were similar for diagnosed diabetes as when we included cases of undiagnosed diabetes. Another possibility is that mortality rates have declined more among very obese persons, leading to greater increases in prevalence of very obese persons with diabetes (Flegal et al., 2002; Gregg et al., 2004). Secular changes in the average amount of weight loss after the diagnosis of diabetes due to changes in therapeutic approaches could also affect BMI distributions among the diabetic population, but we

lack data on this issue to speculate which direction this would influence the findings. Finally, our attempts to correlate ecological trends in obesity may be complicated by a time lag between the two phenomena that has not been clarified.

The proportion of prevalent diabetes cases contributed by the very obese categories is more than twice what would be expected based on the BMI contribution of diabetes cases in 1976–1980. However, our study should be interpreted differently from cohort studies that examine the contribution of BMI to incidence of diabetes. For example, in the San Antonio Heart Study, 13% of white diabetic cases and 23% of Hispanic diabetic cases were very obese (Ford et al., 1997; Burke et al., 2003). Risk of diabetes in a defined cohort may be influenced by many demographic, genetic, and behavioral factors independent of obesity. Whereas cohort studies examine the etiologic association of BMI with diabetes incidence, our study examined the composition of change in population-wide prevalence and thus may be influenced by changes in the prevalence of obesity over time.

These findings raise the question of whether public health programs should ultimately aim to reduce levels of moderate and extreme obesity rather than using limited resources to achieve better weight control more broadly for the entire population. This question is a reminder of the classic debate between the “high risk” approach and the “population approach” described by Rose, who argued that prevalence of adverse risk factors (e.g. extreme obesity) are so highly correlated with the mean values of the risk factor (e.g. mean BMI) in the population, that modest reductions in risk factors applied to the whole population achieve substantial reductions in incidence of the condition (Rose, 1985; Rose and Day, 1990).

The “high risk” approach to diabetes prevention that has been previously described involves following the model of the diabetes prevention trials, wherein overweight and obese persons with impaired glucose tolerance are identified and submitted to intensive lifestyle change (Tuomilehto et al., 2001; Knowler et al., 2002; Williamson et al., 2004). Our study suggests, in addition to impaired glucose tolerance, aggressively pursuing weight loss and prevention of further weight gain among obese persons to avoid progression to extreme obesity may be an efficient strategy because of the large number of cases contributed by a minority segment of the BMI distribution. Such an approach will depend on efficient identification and effective intervention among persons who are likely to progress to extreme obesity. It is also noteworthy, however, that overweight persons contributed 27% of the additional cases, despite the fact that prevalence of overweight remained virtually unchanged in the overall population over the past 25 years. This raises the question of whether factors independent of the changes in BMI – perhaps dietary factors or body fat distribution not reflected in BMI – have also contributed to the additional cases. In the end, the breadth of the epidemic may require a combination of both a high risk and a population approach. Our study suggests that to curtail the current trends in diabetes prevalence, it will be crucial to stem the increased prevalence in extreme obesity. However, since many of the diabetes cases still continue to come from overweight and moderately obese persons, it will ultimately be

important to consider the full range of modifiable risk factors for diabetes in the development of interventions.

Several limitations to these analyses should be considered. First, because the “diagnosed diabetes” status of study subjects was based on their self-reports, our diabetes prevalence estimates may have been influenced by recall biases and by changes over time in public awareness of diabetes, in diabetes detection, and in diagnostic definitions of diabetes. However, our observation that the contributions of the various BMI groups to the increase in the prevalence of diagnosed diabetes were similar to that of total diabetes suggest these influences were not major. Second, because we used data from a series of observational surveys, we could not determine the effectiveness of interventions to reverse the association of obesity with diabetes. However, our study design has a unique advantage of being able to examine contributors to the population over time, and thus may lend itself to inferences about how to slow the current epidemic. Third, BMI is an imperfect surrogate measure for the underlying aspects of obesity that contribute to diabetes. More accurate measurements of obesity are now being incorporated into national surveillance studies but did not exist when the NHANES data that we used were collected. Finally, although our analyses adjusted for age and sex, there may be variation across these and other demographic variables that we could not examine due to power limitations due to the large number of diabetes BMI  $\times$  diabetes strata.

Decisions about how to best confront the national increases in the prevalence of obesity and diabetes are controversial because of the potentially large segment of the population involved, as well as the broad implications of such decisions for the public’s health, for employers, and for health care resources. Ultimately, such decisions about resource allocation for diabetes prevention will depend on more information about the effectiveness of broad-based, non-clinical interventions, the cost of such interventions, and their impact on other health outcomes, including cardiovascular disease, disability, and quality of life. However, our study suggests that approaches aimed at reducing the prevalence of obesity and particularly extreme obesity are reasonable options to slowing the increase in diabetes prevalence. To make these decisions, policy makers will need data from empirical explorations of the relative effectiveness of diabetes-prevention approaches that focus on high-risk groups (i.e. obese or severely obese people) vs. that of more broad-based population approaches, including the health and economic trade-offs of these approaches.

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