Relation of weight gain and weight loss on subsequent diabetes risk in overweight adults

Helaine E Resnick, Paola Valsania, Jeffrey B Halter, Xihong Lin

Abstract

**Study objective**—To determine whether long term weight gain and weight loss are associated with subsequent risk of type 2 diabetes in overweight, non-diabetic adults.

**Design**—Prospective cohort. Baseline overweight was defined as BMI >27.3 for women and BMI >27.8 for men. Annual weight change (kg/year) over 10 years was calculated using measured weight at subjects’ baseline and first follow up examinations. In the 10 years after measurement of weight change, incident cases of diabetes were ascertained by self report, hospital discharge records, and death certificates.

**Setting**—Community.

**Participants**—1929 overweight, non-diabetic adults.

**Main results**—Incident diabetes was ascertained in 251 subjects. Age adjusted cumulative incidence increased from 9.6% for BMI<29 to 26.2% for BMI >37. Annual weight change over 10 years was higher in subjects who become diabetic compared with those who did not for all BMI<35. Relative to overweight people with stable weight, each kg of weight gained annually over 10 years was associated with a 49% increase in risk of developing diabetes in the subsequent 10 years. Each kg of weight lost annually over 10 years was associated with a 33% lower risk of diabetes in the subsequent 10 years.

**Conclusions**—Weight gain was associated with substantially increased risk of diabetes among overweight adults, and even modest weight loss was associated with significantly reduced diabetes risk. Minor weight reductions may have major beneficial effects on subsequent diabetes risk in overweight adults at high risk of developing diabetes.

Evidence from several cohort studies indicates that weight gain during adulthood is associated with increased risk of type 2 diabetes. While this finding is present in most studies with only a few exceptions, it is not clear whether weight gain and weight loss are associated with changes in diabetes risk in overweight adults, a group at very high risk of developing diabetes. As the prevalence of overweight is rising among both adults and adolescents, it is important to understand whether people who are already overweight are susceptible to added diabetes risk after additional weight gain, and it is equally important to clarify the association between weight loss and subsequent diabetes risk in this high risk population. Accordingly, the purpose of this study is to test the hypotheses that weight gain over approximately 10 years in a cohort of overweight adults is associated with increased risk of diabetes in the subsequent 10 years, and that weight loss over 10 years is associated with lower risk of developing diabetes in the subsequent 10 years. We further hypothesised that diabetes risk associated with weight change is independent of other diabetes risk factors including age and baseline body mass index (BMI).

**Methods**

**DATA AND STUDY DESIGN**

Figure 1 is a schematic representation of the study design, timeline, data collection and measurements of interest in this investigation. The US First National Health and Nutrition Examination Survey, Epidemiologic Follow-up Study.
Weight change and diabetes risk in overweight adults

SELECTION CRITERIA
Of the 14,407 subjects in the NHEFS, 10,225 were diabetes within BMI group. Tertiles of annual weight change (1: <−0.22 kg/y; 2: −0.22 kg/y ≤ 0.39 kg/y; 3: > 0.39 kg/y) were defined based on the sample distribution of this variable, which did not differ between men and women. The partial correlation coefficient (p) was used to determine the degree of age adjusted linear association between annual weight change and other continuous variables. The correlations are age adjusted because it is known that weight and weight change vary with age.

The logistic regression model was used to examine the association between annual weight change and subsequent diabetes risk. Age, race, skinfold ratio, systolic blood pressure and education were considered potential confounders of the association between annual weight change and diabetes risk. Additionally, educational attainment (high school education compared with less than high school), was used as a proxy for socioeconomic status. As changes in physical activity may also be associated with weight change, we contrasted subjects who reported increases and decreases in physical activity during the period that weight change was measured, with subjects who reported no change in physical activity. Baseline BMI and annual weight change were both examined as continuous variables, and quadratic terms were tested to determine if the relations between these variables and diabetes risk were non-linear.

Annual weight change is a continuous variable that can be interpreted either as the mean yearly weight change (kg/y) between baseline and first follow up, or as the slope of a subject’s weight trajectory throughout that period. Positive values reflect weight gain, and negative ones indicate weight loss. Total weight change can be approximated by multiplying annual weight change by 10.

In descriptive analyses, baseline BMI was categorised into several groups. Although somewhat arbitrary, these categories provide a basis for calculation of age standardised cumulative incidence of diabetes, and allow for presentation of descriptive associations between weight change and diabetes risk by strata of BMI, a confounding variable. Cumulative incidence of diabetes was age standardised by the direct method using the age distribution of the study sample as the reference population. We used the t test to examine differences in annual weight change between diabetic and non-diabetic subjects within body mass index group, and the χ² test for trend (χ² for trend) was used to examine linear associations between increasing tertile of annual weight change and age adjusted cumulative incidence of diabetes within BMI group. Tertiles of annual weight change (1: <−0.22 kg/y; 2: −0.22 kg/y ≤ 0.39 kg/y; 3: > 0.39 kg/y) were defined based on the sample distribution of this variable, which did not differ between men and women. The partial correlation coefficient (p) was used to determine the degree of age adjusted linear association between annual weight change and other continuous variables. The correlations are age adjusted because it is known that weight and weight change vary with age.

The logistic regression model was used to examine the association between annual weight change and subsequent diabetes risk. Age, race, skinfold ratio, systolic blood pressure and education were considered potential confounders of the association between annual weight change and diabetes risk. Additionally, educational attainment (high school education compared with less than high school), was used as a proxy for socioeconomic status. As changes in physical activity may also be associated with weight change, we contrasted subjects who reported increases and decreases in physical activity during the period that weight change was measured, with subjects who reported no change in physical activity. Baseline BMI and annual weight change were both examined as continuous variables, and quadratic terms were tested to determine if the relationships between these variables and diabetes risk were non-linear.
The best multivariate models were chosen by the likelihood ratio test, and the Hosmer-Lemeshow test was used to evaluate the goodness of fit of the final model. To evaluate the predictive accuracy of the final model, we constructed a receiver operator curve contrasting the model’s sensitivity to 1 minus its specificity. The area under the curve ranges from 0 to 1, and is represented numerically by the c statistic, with higher values indicating good predictive power of the model. All analyses were performed with SAS software, version 6.11. Using the adjusted parameter estimate for annual weight change from the final model, we predicted risk of diabetes associated with various levels of weight change.

### Results

Subjects’ mean baseline BMI was 31.3 kg/m², and these people gained an average of 0.1 kg/y (1.0 kg total) over the first 10 years of the study. Two thirds of the sample were women, and the crude incidence of diabetes was 13% (table 1).

Annual weight change followed a normal distribution (fig 2), and the median value was 0.09 kg/y, indicating that a majority of subjects gained weight in the 10 years between baseline and first follow up. However, annual weight change was highly variable, did not differ by sex, and was inversely correlated with baseline BMI (partial $\rho = -0.27$; $p<0.001$). When the highest BMI category (BMI $\geq 37$) was excluded, the inverse correlation between age adjusted annual weight change and BMI was substantially reduced, but was still statistically significant because of the large sample (partial $\rho = -0.11$; $p<0.001$). Subjects who reported no change in physical activity and those who reported decreased physical activity between baseline and first follow up both gained weight during that period (median weight change: 0.10 and 0.15 kg/y, respectively). Subjects who reported increased physical activity were weight stable (median weight change: 0.04 kg/y).

In the 10 years after measurement of weight change, the age standardised cumulative incidence of diabetes was 15.5%, and diabetes incidence increased with increasing baseline BMI (table 2). While nearly 10% of subjects in the lowest BMI group developed diabetes, over 26% of subjects in the highest group developed diabetes. On average, both diabetic and non-diabetic subjects in all but the highest BMI group either gained weight, or maintained relatively stable weight during follow up (fig 3). In contrast, both diabetic and non-diabetic subjects in the highest BMI group lost weight. For BMI $<31$ and $33 \leq $BMI$<35$, subjects who developed diabetes had significantly higher annual weight change in the previous 10 years.

### Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Annual weight change (kg/y)$\dagger$</th>
<th>Total weight change (AWC x 10)</th>
<th>Age (y)</th>
<th>Sex (% female)</th>
<th>Race</th>
<th>Skinfold ratio</th>
<th>Systolic blood pressure (mm Hg)</th>
<th>Education ($&gt;$ high school)</th>
<th>Change in physical activity$\dagger$</th>
<th>Incident diabetes$\ddagger$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.10 (0.99)</td>
<td>1.0 (0.9)</td>
<td>48.1 (13.9)</td>
<td>1261 (65.4)</td>
<td>White 1579 (81.9%)</td>
<td>Men 1.57 ± 0.57</td>
<td>139 (23)</td>
<td>1076 (56.5)</td>
<td>Same 956 (49.1)</td>
<td>251 (13.0)</td>
</tr>
<tr>
<td></td>
<td>Total weight change (AWC x 10)</td>
<td>1.0 (0.9)</td>
<td></td>
<td></td>
<td>Black 339 (17.5%)</td>
<td>0.98 ± 0.26</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age (y)</td>
<td>48.1 (13.9)</td>
<td></td>
<td></td>
<td>Other 11 (0.6%)</td>
<td>100 (5.2%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sex (% female)</td>
<td>1261 (65.4)</td>
<td></td>
<td></td>
<td>BMI (kg/m²) 31.3 (3.7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Race</td>
<td>White 1579 (81.9%)</td>
<td></td>
<td></td>
<td>0.98 ± 0.26</td>
<td>100 (5.2%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Skinfold ratio</td>
<td>Men 1.57 ± 0.57</td>
<td></td>
<td></td>
<td>139 (23)</td>
<td>1076 (56.5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Systolic blood pressure (mm Hg)</td>
<td>139 (23)</td>
<td></td>
<td></td>
<td>0.98 ± 0.26</td>
<td>100 (5.2%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Education ($&gt;$ high school)</td>
<td>1076 (56.5)</td>
<td></td>
<td></td>
<td>0.98 ± 0.26</td>
<td>100 (5.2%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Change in physical activity$\dagger$</td>
<td>Same 956 (49.1)</td>
<td></td>
<td></td>
<td>1076 (56.5)</td>
<td>0.98 ± 0.26</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Incident diabetes$\ddagger$</td>
<td>251 (13.0)</td>
<td></td>
<td></td>
<td>1076 (56.5)</td>
<td>0.98 ± 0.26</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as mean (SD) for continuous variables and n (%) for nominal variables. *Overweight defined as BMI $>27.8$kg/m² for men and BMI $>27.3$kg/m² for women. Subjects were non-diabetic between baseline and first follow up. $\dagger$Between baseline and first follow up examinations (mean interval=10 y). $\ddagger$Crude cumulative incidence calculated for diabetes diagnosed during the period 1982-94 to 1992.

**Figure 2** Distribution of annual weight change over 10 years in a cohort of 1929 overweight adults.
compared with subjects who did not develop diabetes. For instance, for BMI<29, total weight gain (annual weight change × 10) was (mean (SD) 5.6 (8.5) kg for subjects who subsequently developed diabetes, and 2.2 (8.6) kg for those who did not (p=0.004). For 29≤BMI<31, total weight gain was 5.2 (8.6) kg for subjects who developed diabetes and 0.9 (8.1) kg for those who did not (p<0.001). A significant difference in annual weight change was also observed in the 33≤BMI<35 group. Annual weight change was higher in diabetic subjects in the remaining BMI groups, but these differences were not statistically significant.

Increasing tertile of annual weight change was associated with increased age adjusted risk of diabetes at most levels of baseline BMI (fig 4). Significant trends were observed between tertile of annual weight change and diabetes risk for BMI<29 (p=0.003, χ²), 29≤BMI<31 (p=0.005, χ²), and 35≤BMI<37 (p=0.04, χ²). The greatest cumulative incidence of diabetes was observed in the third tertile of annual weight change in all but the highest BMI group.

We examined whether the association between annual weight change and subsequent diabetes risk would persist after adjustment for multiple diabetes risk factors, and we were particularly interested in whether the addition of risk variables other than baseline age and BMI would reduce or eliminate the association between annual weight change and diabetes risk. Accordingly, two models were defined (table 3). Model I included age, age² and BMI. The quadratic term for age indicates a non-linear association between age and diabetes risk. Model II included all variables in model I as well as sex, race, education, systolic blood pressure, skinfold ratio and reported change in physical activity. Regression analyses

![Figure 3](https://www.jech.com)

**Figure 3** Annual weight change over 10 years in relation to development of diabetes in the subsequent 10 years, by baseline BMI. Comparison is shown between subjects who developed diabetes and those who did not. *p* Value is for the t test of differences in means between these groups.

### Table 2 Distribution of incident* cases of diabetes, by baseline body mass index

<table>
<thead>
<tr>
<th>Baseline BMI (kg/m²)</th>
<th>Incident cases of diabetes</th>
<th>Cumulative incidence (%)†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>&lt; 29 (591)</td>
<td>56</td>
<td>9.5</td>
</tr>
<tr>
<td>29 to &lt;31 (562)</td>
<td>66</td>
<td>11.3</td>
</tr>
<tr>
<td>31 to &lt;33 (335)</td>
<td>39</td>
<td>11.6</td>
</tr>
<tr>
<td>33 to &lt;35 (190)</td>
<td>35</td>
<td>18.4</td>
</tr>
<tr>
<td>35 to &lt;37 (110)</td>
<td>16</td>
<td>14.6</td>
</tr>
<tr>
<td>≥ 37 (141)</td>
<td>39</td>
<td>27.7</td>
</tr>
<tr>
<td>Total</td>
<td>251</td>
<td>15.5</td>
</tr>
</tbody>
</table>

*Diagnosis of diabetes during the period 1982-1984 to 1992. †Age standardised. Minimum BMI = 27.8 kg/m² for men and 27.3 kg/m² for women.

**KEY POINTS**

- Weight gain has been associated with risk of type 2 diabetes in adulthood.
- It is not clear if weight gain further increases diabetes risk in overweight adults, a group already at high risk of diabetes.
- Further weight gain was associated with significantly increased risk of diabetes in overweight adults, and even minor weight loss was associated with reduced risk.
- Overweight adults should not be considered to have reached a threshold where additional weight gain fails to increase diabetes risk.
- Weight loss interventions may be effective at reducing diabetes risk in overweight people.
indicated that annual weight change was a significant predictor of subsequent diabetes risk in model I: odds ratio = 1.46, 95% confidence intervals = 1.26, 1.69. The strength of the association between annual weight change and diabetes risk was virtually unchanged after adjustment for the additional risk variables in model II: odds ratio = 1.49, 95% confidence intervals = 1.29, 1.73.

Model II indicated that race, sex, education and change in physical activity did not predict of subsequent diabetes risk in this overweight cohort. There was no evidence of interaction between annual weight change and the covariates, and there was no quadratic relation between either BMI or annual weight change with diabetes risk. The Hosmer-Lemeshow test indicated that model II fits the data well (p = 0.82). The area under the ROC curve for model II was 0.68, indicating relatively good predictive power.

We estimated from model II odds ratios for specific levels of annual weight change, and corresponding total weight change over 10 years (table 4). Relative to no weight change, weight gain was associated with increased risk of developing diabetes. Model estimates suggested significant increases in diabetes risk after minor weight gain. Risk estimates ranged from 1.04 (95% confidence intervals = 1.03, 1.06) for a 10 year weight gain of 1 kg to 2.22 (95% confidence intervals = 1.66, 2.98) for a 10 year weight gain of 20 kg. Conversely, our model indicated that weight loss was associated with significant reductions in subsequent diabetes risk relative to no weight change. A weight loss of 1 kg over 10 years was associated with a slight, but significantly decreased subsequent risk of developing diabetes: odds ratio = 0.96, 95% confidence intervals = 0.95, 0.97. Greater weight loss was associated with even larger reductions in subsequent diabetes risk. A weight loss of 10 kg over 10 years was associated with a 33% reduction in subsequent diabetes risk compared with no weight loss: odds ratio = 0.67, 95% confidence intervals = 0.58, 0.78. These associations were independent of age, baseline BMI, and other risk variables.

### Discussion

Our findings extend previous work on the association between weight change and diabetes risk by demonstrating a significant

---

**Table 3** Logistic regression analysis of the effect of average annual weight change over 10 years and baseline characteristics on risk of subsequent diabetes in a cohort of overweight adults, First National Health and Nutrition Examination Survey, Epidemiologic Follow-up Study, 1971–1992

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model I</th>
<th></th>
<th>Model II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β</td>
<td>Odds ratio</td>
<td>95%</td>
</tr>
<tr>
<td>Annual weight change (per kg/y)</td>
<td>0.380</td>
<td>1.46</td>
<td>1.26, 1.69</td>
</tr>
<tr>
<td>Age (per 10y)</td>
<td>0.033</td>
<td>1.23</td>
<td>1.09, 1.38</td>
</tr>
<tr>
<td>Age²</td>
<td>−0.001</td>
<td>−0.001</td>
<td></td>
</tr>
<tr>
<td>BMI (per kg/m²)</td>
<td>0.117</td>
<td>1.13</td>
<td>1.09, 1.16</td>
</tr>
<tr>
<td>Skinfold Ratio (per 0.2 unit)</td>
<td>0.117</td>
<td>1.13</td>
<td>1.09, 1.16</td>
</tr>
<tr>
<td>Systolic blood pressure (per 5mmHg)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*The significant non-linear association between age and diabetes risk requires calculation of the odds ratio for a specified change in age. The odds ratio in this table represents the change from age 40 to age 50.

---

**Table 4** Average annual weight change over 10 years, absolute weight change over 10 years, and estimated risk of diabetes risk in a cohort of overweight adults, First National Health and Nutrition Examination Survey, Epidemiologic Follow-up Study, 1971–1992

<table>
<thead>
<tr>
<th>Annual weight change (kg/y)</th>
<th>Absolute weight change (kg/10y)</th>
<th>Odds ratio*</th>
<th>95% confidence intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>−2.0</td>
<td>−20</td>
<td>0.45</td>
<td>0.34, 0.60</td>
</tr>
<tr>
<td>−1.5</td>
<td>−15</td>
<td>0.55</td>
<td>0.44, 0.68</td>
</tr>
<tr>
<td>−1.0</td>
<td>−10</td>
<td>0.67</td>
<td>0.58, 0.78</td>
</tr>
<tr>
<td>−0.5</td>
<td>−5</td>
<td>0.82</td>
<td>0.76, 0.88</td>
</tr>
<tr>
<td>−0.1</td>
<td>−1</td>
<td>0.96</td>
<td>0.95, 0.97</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>0.1</td>
<td>1</td>
<td>1.04</td>
<td>1.03, 1.06</td>
</tr>
<tr>
<td>0.5</td>
<td>5</td>
<td>1.22</td>
<td>1.13, 1.31</td>
</tr>
<tr>
<td>1.0</td>
<td>10</td>
<td>1.49</td>
<td>1.29, 1.73</td>
</tr>
<tr>
<td>1.5</td>
<td>15</td>
<td>1.82</td>
<td>1.46, 2.27</td>
</tr>
<tr>
<td>2.0</td>
<td>20</td>
<td>2.22</td>
<td>1.66, 2.98</td>
</tr>
</tbody>
</table>

*Odds ratios are adjusted for age, BMI, sex, race, skinfold ratio and systolic blood pressure.
Weight change and diabetes risk in overweight adults

601

Cardiovascular risk.25 Our models did not indicate a significant association between reported physical activity and subsequent diabetes risk. However, the measure of physical activity in this observational study is crude and applied only to recreational activities. It is not surprising that previous studies with exercise interventions showed positive effects of physical activity on diabetes risk factors. Availability of better data on changes in physical activity may have provided greater insight into how this factor is associated with long term weight change and diabetes risk in overweight people.

Although the age standardised cumulative incidence of diabetes in the BMI≥37 kg/m² group (n=141) was 26.2%, annual weight change was unrelated to development of diabetes in this group. This finding suggests that it is perhaps only at the highest levels of obesity that the effect of weight gain reaches a threshold beyond which this factor does not contribute added diabetes risk. It was therefore not surprising that when we repeated our regression analyses excluding the highest BMI group, the magnitude of association between annual weight gain and diabetes risk increased. The observation that both diabetic and non-diabetic subjects in the highest baseline BMI category tended to lose weight may be “regression to the mean”, rather than a biological phenomenon.26 Although there was an inverse correlation between baseline BMI and annual weight change, this association was weak across lower BMI groups. Moreover, significant differences in annual weight change between diabetic and non-diabetic subjects were observed in most of the lower BMI groups, supporting the idea that annual weight change is most useful in predicting subsequent diabetes risk in the least extreme range of overweight, which constituted 93% of this cohort.

While the ROC curve indicated relatively good predictive power of the final model, it also suggested that factors other than those in our model are needed to accurately predict diabetes in overweight people. Addition of key baseline measures, such as fasting glucose and visceral fat may have resulted in greater predictive power of the model. The absence of repeated fasting glucose measures throughout the study probably contributed to under-identification of incident cases, which also may have limited the model's predictive accuracy. However, the validity of our results rests on an accurate description of weight change before development of diabetes. Therefore, under-identification of incident cases would not have changed our findings as there is no reason to believe that the weight histories of undiagnosed and diagnosed diabetic people differ. This study is also limited by the availability of only two measured weights taken 10 years apart, precluding examination of potentially important effects of weight cycling that may have occurred in the interim period. Other potentially important factors such as family history of both diabetes and overweight were not
available in the NHEFS, but these may be critical to consider in future studies of diabetes in high risk populations.

It is important to emphasize that while our data allowed for identification of diabetes cases from three sources of information, no data on fasting or post-challenge glucose were available. The latter measures are standard screening instruments for detection of undiagnosed diabetes. Absence of these measures in the NHEFS presumably resulted in misclassification of diabetic people as non-diabetic, although it is difficult to speculated on the degree and effect of this misclassification.

Duration of overweight predicts diabetes risk, suggesting that this factor may have cumulative effects. To determine if the association between annual weight change and diabetes risk may have been attributable to overweight beginning earlier in life for subjects who became diabetic relative to those who did not, we examined diabetes status at the end of the study in relation to self reported weight at age 25. There was no difference in mean reported weight at age 25 between subjects who became diabetic and those who did not, after adjusting for sex, race, and baseline age. Although weight at age 25 is an imperfect tool for evaluating duration of overweight before entry into the study, it does provide evidence that overweight subjects in this study had comparable weights in early adulthood, and supports the role of weight gain as a risk factor for diabetes in this overweight cohort.

This study raises interesting questions about mechanisms that make some overweight people more likely to develop diabetes than others. It is known that overweight is associated with insulin resistance through a variety of mechanisms, but many overweight people with insulin resistance never develop frank diabetes, while, over time, others develop a relative insulin deficiency leading to diabetes. This observation is suggestive of biological heterogeneity among overweight people. Further investigation of factors that distinguish high risk from low risk overweight people will become more increasingly important as the prevalence of overweight continues to rise, placing more people at risk of developing diabetes. At the present time, weight loss seems to be an effective way to reduce diabetes risk in overweight adults.

In summary, our results indicate that weight gain was associated with increased diabetes risk in overweight adults and that even modest weight loss in overweight people was associated with reduced risk of developing diabetes. Weight loss interventions may be effective in reducing long term diabetes risk even among overweight people.

The authors thank Drs Patricia Peyster and MaryFran Sowers for helpful comments on an earlier draft of this manuscript. The data used in this study were collected by the National Center for Health Statistics, Bethesda, MD. Parts of this work were presented at the annual meeting of the American Diabetes Association, Chicago, IL, June, 1998.

Funding: this research was supported by Multidisciplinary Training Grant in Aging (no AG-00114) from the National Institute on Aging, an AARP/Andrus Foundation Graduate Fellowship, and a Blue Cross and Blue Shield of Michigan Student Award Program Grant (no 755-SAP97) to Dr Resnick.

Conflicts of interest: none.

Relation of weight gain and weight loss on subsequent diabetes risk in overweight adults

Helaine E Resnick, Paola Valsania, Jeffrey B Halter and Xihong Lin

*J Epidemiol Community Health* 2000 54: 596-602
doi: 10.1136/jech.54.8.596

Updated information and services can be found at:
http://jech.bmj.com/content/54/8/596

These include:

**References**
This article cites 18 articles, 9 of which you can access for free at:
http://jech.bmj.com/content/54/8/596#BIBL

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Topic Collections**
Articles on similar topics can be found in the following collections

- Health education (1537)
- Health promotion (1711)
- Obesity (public health) (542)
- Cohort studies (794)
- Epidemiologic studies (2838)

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/