Duration of Obesity Increases the Incidence of NIDDM

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The effect of duration of obesity on incidence of non-insulin-dependent diabetes mellitus (NIDDM) was determined among Pima Indians. Duration of obesity was defined as the time since body mass index (BMI) was first known to be at least 30 kg/m^2. Among 1057 participants eligible for study, there were 224 incident cases of NIDDM in 5975 person-yr of follow-up. The association of duration of obesity with incidence of diabetes adjusted for age, sex, and current BMI was highly significant ($P < 0.0001$). This adjusted incidence of diabetes in cases/1000 person-yr of obesity was 24.8 for people with less <5 yr of obesity, 35.2 for people with 5–10 yr of obesity, and 59.8 for people with at least 10 yr of obesity. There was no apparent excess risk of diabetes for people who had a BMI of at least 30 kg/m^2 and then lost weight. They had a slightly nonsignificantly higher rate than people who had not attained a BMI of at least 30 kg/m^2 and a lower rate than people whose BMI remained 30–35 kg/m^2. The relationship of duration of obesity with serum insulin concentrations among nondiabetic people was determined controlling for sex and age, BMI, and plasma glucose concentrations at the time of a glucose tolerance test. Duration of obesity was inversely associated with fasting serum insulin concentration through most of the range of fasting plasma glucose concentrations ($P < 0.001$) and tended to be inversely associated with 2-h postload serum insulin concentration through the entire range of postload plasma glucose concentrations ($P = 0.058$).
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Obesity is one of the most important risk factors for incidence of non-insulin-dependent diabetes mellitus (NIDDM; 1–3). It is not known whether duration of obesity influences diabetes incidence independently of the current degree of obesity. In other words, of two individuals with the same degree of obesity, who is at greater risk of diabetes—the individual who has been obese for many years or the one who has become obese only recently? The answer to this question is of potential importance in considering mechanisms for the development of NIDDM and as a public health problem among obese young adults. We examined incidence of NIDDM according to duration of obesity among Pima Indians, who are at high risk of NIDDM. In addition, we examined the relationship of duration of obesity to serum insulin concentrations obtained during an oral glucose tolerance test (OGTT).

RESEARCH DESIGN AND METHODS

Subjects. Residents of the Gila River Indian community of southern Arizona have participated since 1965 in a study of the occurrence and effects of diabetes. The current analysis included data collected through 1988. Community residents were requested approximately every 2 yr to undergo a standardized examination, which included measures of height and weight and a modified OGTT. Before 1975, venous plasma was obtained for glucose measurement 2 h after ingestion of 75 g of carbohydrate (Dexcola, Custom, Baltimore, MD; or Glucola, Ames, Elkhart, IN). Since 1975, most laboratory tests were performed after an overnight fast and included measurements of fasting and 2-h postload plasma glucose and serum insulin concentrations. Diabetes was diagnosed according to World Health Organization Criteria if the plasma glucose concentration was at least 7.8 mM during fasting or at least 11.1 mM 2 h after carbo-

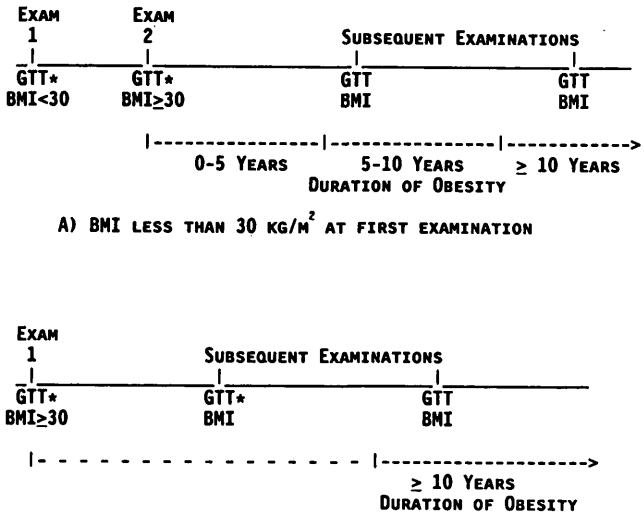

FIG. 1. Methods for determining duration of obesity. GTT, oral glucose tolerance test; BMI, body mass index (kg/m^2). *A*: people with 2 examinations to determine date of onset of obesity could have follow-up of 0–5, 5–10, and ≥10 yr. *B*: people who were obese at the first examination could only have follow-up of ≥10 yr.

hydrate loading (4). The degree of obesity was estimated by body mass index (BMI [wt/ht^2 in kg/m^2]). This study was limited to people at least 15 yr of age.

Obesity is common among Pima Indians: ~50% of nondiabetic adults have a BMI of at least 30 kg/m^2, and 25% have a BMI of at least 35 kg/m^2. Women and people aged 20–45 yr are more likely to be obese (2). For this study, a BMI of 30 kg/m^2 was chosen as an indicator of obesity (5,6). Duration of obesity was defined as the period between the first observed BMI of at least 30 kg/m^2 and the last examination or onset of diabetes, although some people met this definition who subsequently had a BMI <30 kg/m^2. Duration of obesity was determined by two mutually exclusive methods (Fig. 1). Most people had two examinations to determine the date of onset of obesity. At each of these examinations, the subject was required to have a nondiabetic OGTT. At the first examination, the BMI was <30 kg/m^2, and at the second examination, it was at least 30 kg/m^2. The duration of obesity was the time from the date of the second examination (i.e., onset of obesity) until the last examination or diagnosis of diabetes and ranged from 0.1 to 18 yr. This requirement that all people have follow-up after a nondiabetic OGTT excluded 55 people with diabetes diagnosed on the date of onset of obesity and 391 people who had no follow-up after the onset of obesity. Among the latter group were 33 who died without follow-up and 159 who were last examined within 3 yr of the close of the study, a reasonable period in which to have expected an examination. There remained for analysis 845 people for whom the duration of obesity was determined by this method. Follow-up among these participants was grouped into categories of 0–5 yr, 5–10 yr, or at least 10 yr of obesity. A precise duration of obesity could not be calculated for 627 people who had a BMI of at least 30 kg/m^2 at the first examination. However, after 10 yr of follow-up, 216 could be grouped into the at-least-10-yr duration-of-obesity category. They were not included in the shorter-duration categories.

The effect of weight loss on the incidence of diabetes was evaluated among the 171 people with a BMI of at least 30 kg/m^2 that subsequently fell to <30 kg/m^2. Their incidence was compared with the incidence among two groups: 927 people with BMI 27–30 kg/m^2 who had not attained a BMI of at least 30 kg/m^2 and 839 people with BMI 30–35 kg/m^2.

Among the 845 people who had had a date of onset of obesity determined by two examinations, there were 507 people who also had subsequent measurements of fasting and post–carbohydrate load serum insulin concentrations (7). Insulin was measured in microunits per milliliter and converted to picomolar with a conversion factor of 6. The relationship of duration of obesity to insulin concentrations was determined at the last nondiabetic OGTT, with duration of obesity defined as the time from the date of onset of obesity until this OGTT.

Statistical analyses. Incidence of NIDDM was calculated as the number of newly diagnosed cases of diabetes divided by time at risk for diabetes after BMI of at least 30 kg/m^2 was first observed. Incidence rates were expressed as new cases per 1000 person-yr at risk. Person-years at risk of diabetes were stratified by sex and by variables (age, current BMI, and duration of obesity) that changed over time. Age categories were changed at each subject's 15th, 25th, and 45th birthdays. The category of current BMI could change with each examination and included <30, 30–32, 32–35, and 35–40 kg/m^2. Category of obesity duration changed with the 5th- and 10th-yr anniversaries of the date that BMI was first observed to be at least 30 kg/m^2. If, on a subsequent examination, the BMI fell below 30 kg/m^2, duration of obesity continued to increase, but in the current BMI category of <30 kg/m^2. There was insufficient follow-up time to include a current BMI category of at least 40 kg/m^2 or the four men who were in the category ≥45 yr old with BMI at least 35 kg/m^2, leaving 1057 people for calculation of NIDDM incidence. Incidence rates for each duration-of-obesity group were directly standardized to a population with a uniform distribution across age, sex, and current BMI groups to determine age, sex, and current BMI adjusted incidence rates of diabetes and their 95% confidence intervals (CIs). A test for linear association of duration of obesity with diabetes incidence was made with an extension of the Mantel-Haenszel χ^2 statistic (8,9). In a similar manner, adjusted diabetes incidence rate ratios and their 95% CIs were calculated from the incidence among people who lost weight relative to the incidence (adjusted for age and sex) among people who had not attained a BMI of 30 kg/m^2 and to the incidence (adjusted for age, sex, and duration of obesity) among people with BMI 30–35 kg/m^2.

Cross-sectional relationships of duration of obesity to fasting and to 2-h post–carbohydrate load serum insulin

TABLE 1
Incidence of diabetes (cases/1000 person-yr) according to current body mass index, sex, age, and duration of obesity

Current body mass index	Duration of obesity (yr)								
	0–5			5–10			≥10		
	Cases	Person-yr	Incidence	Cases	Person-yr	Incidence	Cases	Person-yr	Incidence
<30 kg/m^2									
Male (age)									
15–24 yr	0	17.3	0	0	17.2	0	0	4.1	0
25–44 yr	0	20.8	0	0	33.8	0	1	39.2	25.5
≥45 yr	0	16.7	0	2	22.8	87.6	4	40.5	98.9
Female (age)									
15–24 yr	0	48.6	0	0	33.8	0	0	9.9	0
25–44 yr	2	50.2	39.9	2	54.0	37.0	0	23.6	0
≥45 yr	0	17.7	0	1	21.7	46.1	4	47.7	83.8
Total	2	171.3	6.7*	5	183.3	28.5*	9	165.0	34.7*
30–32 kg/m^2									
Male (age)									
15–24 yr	3	184.8	16.2	1	44.1	22.7	0	12.2	0
25–44 yr	4	126.9	31.5	1	49.2	20.3	4	48.0	83.3
≥45 yr	3	78.2	38.3	2	35.7	56.0	2	42.3	47.3
Female (age)									
15–24 yr	6	415.7	14.4	0	51.4	0.0	0	10.7	0
25–44 yr	5	270.1	18.5	6	108.1	55.5	4	60.0	68.6
≥45 yr	5	58.3	85.8	2	29.3	68.3	6	57.1	105.1
Total	26	1134.0	34.1*	12	317.8	37.2*	16	230.3	50.4*
32–35 kg/m^2									
Male (age)									
15–24 yr	1	234.1	4.3	2	60.2	33.2	0	7.7	0.0
25–44 yr	6	93.3	64.3	5	69.3	72.2	5	50.4	99.2
≥45 yr	2	16.9	118.3	0	13.8	0.0	9	79.1	113.8
Female (age)									
15–24 yr	10	447.1	22.4	3	103.8	28.9	3	14.0	214.3
25–44 yr	6	301.5	19.9	17	225.3	75.5	8	128.6	62.2
≥45yr	2	45.5	44.0	2	38.7	51.7	8	126.7	63.1
Total	27	1138.4	45.5*	29	511.1	43.6*	33	406.5	92.1*
35–40 kg/m^2									
Male (age)									
15–24 yr	3	124.0	24.2	3	53.5	56.1	1	12.9	77.6
25–44 yr	0	55.0	0	3	70.9	42.3	9	109.8	82.0
Female (age)									
15–24 yr	2	245.6	8.1	5	164.8	30.3	1	38.8	25.8
25–44 yr	3	142.4	21.1	5	196.9	25.4	15	234.8	63.9
≥45 yr	0	12.1	0	0	14.0	0	15	241.5	62.1
Total	8	579.1	10.7*	16	500.1	30.8*	41	637.8	62.3*

*Age-sex–adjusted incidence.

concentrations were determined with linear regression models. Models included the logarithm of fasting or postload serum insulin concentrations as the dependent variable and controlled for the plasma glucose concentration drawn at the same time and for sex, age, and current BMI. Duration of obesity was evaluated as a continuous variable. Duration of obesity and its quadratic form, duration of obesity squared, were included together in each model because they jointly improved the model fit. All two-way interaction terms with the obesity-duration terms and quadratic terms for BMI, age, and glucose concentrations were tested in the models and included at a significance level of 0.05. The joint statistical significance of all terms that contained a specific variable (interaction and quadratic terms as well as the variable itself) was determined from a partial F statistic for the contribution of these terms to the model (10). A plot of predicted serum insulin concentration according to plasma glucose concentration and obesity duration was made with regression coefficients and means from the regression model. The approximate 25th, 50th, and 75th percentiles of duration of obesity (3, 6, and 9 yr, respectively) were used in this plot.

RESULTS

The incidence of NIDDM according to categories of age, sex, current BMI, and duration of obesity is shown in Table 1. There were 224 incident cases of diabetes in 5975 person-yr of follow-up. Within current BMI groups, the age-sex–adjusted incidence of diabetes generally increased with increasing duration of obesity. The only exceptions were for current BMI groups 30–32 and 32–35 kg/m^2 in which the age-sex–adjusted incidences for 0–5 and 5–10 yr of obesity were similar. The incidence rates of NIDDM increased markedly with increasing duration of obesity, controlling for age, sex, and current BMI (Fig. 2). Per 1000 person-yr, these rates (with

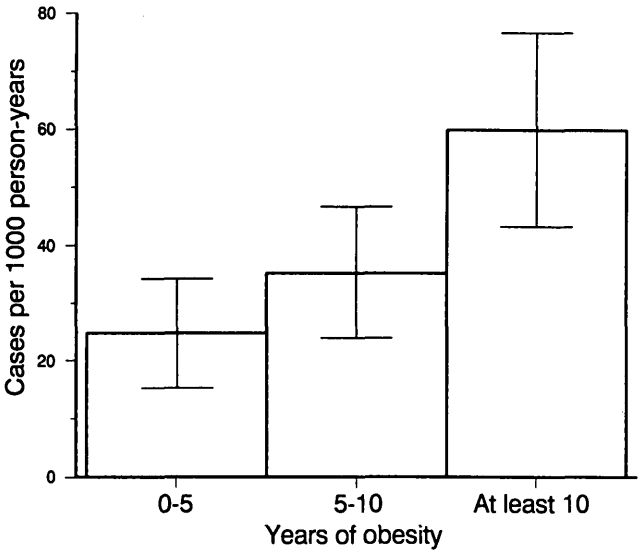

FIG. 2. Incidence with 95% confidence intervals (*error bars*) of non-insulin-dependent diabetes mellitus according to duration of obesity, adjusted for sex, age, and current body mass index.

95% CIs in parentheses) were 24.8 (15.4–34.3) during 0–5 yr of obesity, 35.2 (23.9–46.5) during 5–10 yr of obesity, and 59.8 (43.1–76.4) after ≥10 yr of obesity. A test for linear association of duration of obesity with NIDDM incidence was strongly positive with a χ^2 of 26.9 for 1 df and $P < 0.0001$.

The incidence of NIDDM for people whose BMI had decreased to <30 kg/m^2 (from mean maximum BMI 32.3 kg/m^2) was compared to the incidence among people whose BMI had not exceeded 30 kg/m^2. These two groups had the same mean current BMI of 28.4 kg/m^2. The incidence rate among the formerly obese was 1.1 times (95% CI 0.68–1.9) the rate of people who had not been obese. The incidence of NIDDM for people whose BMI had decreased to <30 kg/m^2 was 0.58 times (95% CI 0.34–0.98) the rate of people with current BMI 30–35 kg/m^2.

To attempt to understand mechanisms that might explain the effect of duration of obesity on incidence of NIDDM, the relationships of serum insulin concentrations to duration of obesity were examined at the last nondiabetic OGTT. Duration of obesity and fasting serum insulin concentrations were associated, but the direction of the association was dependent on the fasting plasma glucose concentration (Table 2; Fig. 3). At plasma glucose concentrations <6.1 mM, longer duration of obesity was associated with lower fasting serum insulin concentrations. But at higher glucose concentrations, longer duration of obesity was associated with higher insulin concentrations. Only 12.6% of people had fasting plasma glucose concentrations >6.1 mM, of whom 67% had impaired glucose tolerance. Two-hour postload serum insulin concentrations were higher in people with the shortest duration of obesity over the entire range of postload plasma glucose levels, but this association was of borderline significance (Table 2).

DISCUSSION

Few studies have been published on the effects of duration of obesity on occurrence of NIDDM, and none have had adequate measures of duration of obesity. A population study in Israel that determined the prevalence of diabetes with a 100-g OGTT had a single measure of height and weight from a previous examination ~10 yr earlier (11). Among people with a BMI of at least 27 kg/m^2 at the time of the OGTT, the prevalence of NIDDM was highest in those whose BMI was also at least 27 kg/m^2 at the earlier examination. From analysis of data gathered in an earlier study, a strong correlation of duration of obesity

TABLE 2
Linear regression analysis of $\log_{10}$ of fasting and 2-h post–carbohydrate load serum insulin concentrations (pM) among 507 Pima Indians without diabetes

	Fasting insulin			Post-load insulin		
Characteristic	Coefficient	Partial *F* statistic	*P*	Coefficient	Partial *F* statistic	*P*
Duration of obesity (yr)	−0.2766	8.7		−0.0262	5.4	0.058
(Duration of obesity)^2	0.0131	5.2	<0.001	0.0014	4.2	
Plasma glucose concentration (mM)						
Fasting × duration of obesity	0.0449	6.9				*
Fasting × (duration of obesity)^2	−0.0021	4.2	<0.001			*
Fasting	−0.0518	0.7				*
2-h postload			*	0.3574	63.4	<0.001
(2-h postload)^2			*	−0.0180	33.5	
Current body mass index (kg/m^2)	0.0220	90.6	<0.001	0.0134	27.8	<0.001
Age (yr)	0.0001	0.0	0.99	0.0007	0.4	0.524
Sex (female)	0.0182	0.8	0.38	0.0620	7.5	0.006
Intercept	1.9495			0.9229		

Duration of obesity was the number of years since body mass index first became ≥30 kg/m^2. Partial *F* statistics are for 1505 df.
*Not included in this analysis

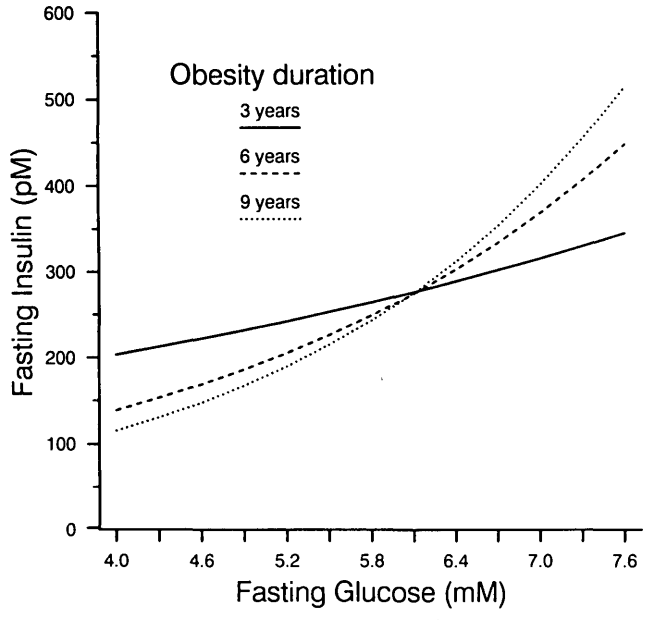

FIG. 3. Predicted fasting serum insulin concentration among 507 Pima Indians without diabetes according to fasting plasma glucose concentration and duration of obesity, controlled for sex, age, and current body mass index.

to glucose intolerance was suggested (12,13). However, obesity was not defined, and no information was provided on how duration of obesity was determined. Similar limitations existed in a study of glucocorticoid-stressed intravenous glucose tolerance among obese, nondiabetic people, in which neither degree nor duration of obesity was associated with abnormal glucose tolerance (14).

In this study, we found that incidence of NIDDM increased from the time that BMI became at least 30 kg/m^2, regardless of current BMI. Thus, independent of degree of obesity, duration of obesity was a significant risk factor for diabetes. The risk of NIDDM associated with rapid weight gain or weight cycling was not evaluated, so we could not exclude possible effects of these obesity-related factors on diabetes incidence. We also could not determine whether longer duration of obesity was associated with either increased overall adiposity or increased intra-abdominal adiposity (or both) for a given current BMI.

If applicable to other populations, the findings of this study support the general concern regarding obesity among the young. Young, obese adults who remain obese may be at particularly high risk of diabetes by middle age. Obesity is common among the youth of many North American Indian tribes and, by middle age, so is diabetes (15–17). A high percentage of members of other ethnic groups also become overweight at a young age. For example, in the age-group 18–24 yr, black women have 2.5 times the prevalence of being overweight as white women (18). If the findings in this study apply generally, then, as these populations age, long-term obesity may result in a high incidence of NIDDM that could not be explained completely by the degree of current obesity.

If obese people lose weight, do they remain at increased risk of diabetes? This study did not find such an effect. Despite a mean maximum BMI 3.9 kg/m^2 higher than their current mean BMI, people who lost weight to <30 kg/m^2 had an incidence of diabetes only 10% higher than people whose BMI remained <30 kg/m^2. However, the imprecision of this estimate of a small increased risk, as shown by the wide 95% CI, does not eliminate the possibility that the risk could be more substantial. Current BMI should not have confounded the comparison because the two groups had the same mean current BMI of 28.4 kg/m^2. Also, there seemed to be benefits from weight loss among the obese; among people whose current BMI had decreased to <30 kg/m^2, incidence of diabetes was a statistically significant 40% lower than people whose current BMI remained 30–35 kg/m^2. Thus, the effect of transient obesity on risk of diabetes declined with weight loss. This is not to say that a BMI of <30 kg/m^2 is protective of diabetes; Pima Indians with BMI 27–30 kg/m^2 are still at higher risk of developing NIDDM than those with lower BMI (2).

If prolonged obesity increases the risk of diabetes, what is the mechanism? Although the results were inconclusive, inference into the mechanism may be gained by examining the association of duration of obesity with serum insulin concentrations. For the 87% of the nondiabetic people with fasting glucose concentrations <6.1 mM, longer duration of obesity was associated with lower fasting serum insulin concentration when also controlling for fasting plasma glucose, age, sex, and current BMI. Only among people with the highest fasting glucose concentrations was there an association of higher insulin concentrations with longer duration of obesity. Therefore, for most of the study population, longer duration of obesity was associated with lower fasting serum insulin concentration. For a given postload plasma glucose concentration, longer duration of obesity tended to be associated with lower postload serum insulin concentrations throughout the entire range of nondiabetic postload glucose concentrations.

Two possible deleterious effects of prolonged duration of obesity on glucose homeostasis would be increased resistance to glucose disposal and decreased secretion of insulin. Resistance to glucose disposal is strongly associated with obesity and results in high fasting and postload serum insulin concentrations. Prolonged duration of obesity could conceivably worsen this resistance and result in even higher serum insulin concentrations. Yet, in general, the opposite was found in this study; for a given BMI, there was a tendency for longer duration of obesity to be associated with lower serum insulin concentrations. Thus, long duration of obesity did not appear to worsen resistance to glucose disposal, at least as reflected by insulin concentrations.

Rather than worsen resistance to glucose disposal, the diabetogenic effect of prolonged obesity may be to impair insulin secretion. Although the association of prolonged obesity with lower serum insulin concentrations in this study supported this hypothesis, the evidence was not compelling. The cross-sectional measurement of plasma glucose and serum insulin concentrations could

not control for the dynamic relationship of these two variables. As noted above, the association of long duration of obesity with low serum insulin concentrations was not clear-cut. Also, the measurement of low fasting and postload serum insulin concentrations is, at best, a crude indicator of impaired insulin secretion.

If, with better tests, prolonged obesity were found to be associated with either impaired insulin secretion or resistance to glucose uptake, the mechanism would still require explanation. As current hypotheses for the role of obesity in the development of NIDDM are tested and new hypotheses are developed, the role of duration of obesity should be considered.

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