# Effects of Childbearing on Glucose Tolerance and NIDDM Prevalence

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**The goal of this study was to estimate the effects of childbearing on subsequent glucose tolerance and non-insulin-dependent diabetes mellitus (NIDDM) prevalence. A sample of subjects from 64 different locations in the United States were recruited for inclusion in the Second National Health and Nutrition Examination Survey. A complex survey design was used to select a probability sample of subjects from each location. A total of 4577 women were recruited, of whom 3057 underwent clinical and laboratory evaluation for the presence of diabetes mellitus. Women were classified as to their glucose tolerance based on the results of an oral glucose tolerance test or previous physician diagnosis of diabetes mellitus combined with current use of hypoglycemic medication. Childbearing was defined as number of live births experienced by each woman at the time of the interview. Fasting plasma glucose increased linearly with increasing number of live births (coefficient 0.009, 95% confidence interval [CI] 0.006–0.012), as did the 2-h value (coefficient 0.015, 95% CI 0.009–0.021). Adjustment for age, body mass index (BMI), education, and income substantially reduced the magnitude of the association between childbearing and either plasma glucose measurement. When the prevalence of NIDDM in relation to childbearing was examined with logistic regression analysis, a significant linear increase in diabetes prevalence was seen with increasing number of live births (relative prevalence of NIDDM, 1 birth vs. 0 = 1.73, 95% CI 1.39–2.15), but adjustment for age, BMI, education, and income greatly reduced the magnitude of this association (relative prevalence of NIDDM, 1 birth vs. 0 = 1.07, 95% CI 0.98–1.17). These data do not support a role for childbearing in the subsequent development of NIDDM.**
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Many researchers have reported that childbearing increases the subsequent risk of developing diabetes mellitus in women. To our knowledge, 24 studies have directly addressed this question (1–24). We evaluated the data from these studies according to adjustment for age or obesity. Adjustment for age or obesity was considered adequate when direct standardization, regression analysis, or an equivalent method was used to calculate an effect measure adjusted for the potential confounding factor. Twelve studies found no association between childbearing experience and prevalence of diabetes mellitus or glucose intolerance but had not been adequately adjusted for age or obesity (4–15). When adequate adjustment was made for age and obesity together or age or obesity alone, no study found no association. With inadequate adjustment for age or obesity, 9 studies found higher parity or gravidity in diabetic subjects or a higher diabetes prevalence in more parous or gravid women (16–24). With adequate adjustment for age and obesity together, 2 studies found higher parity or gravidity in diabetic women or higher diabetes prevalence in more parous or gravid women (1,2). When adequate adjustment for either age or obesity was made separately, 1 study found higher parity or gravidity in diabetic women or higher diabetes prevalence in more

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parous or gravid women (3). Twelve of these studies reported an increase in diabetes mellitus prevalence with increasing number of pregnancies or live births (1–3,16–24). Few studies, however, have considered whether correlates of childbearing, e.g., age and weight gain (25,26), account for the higher diabetes mellitus prevalence seen with increasing parity, because both age and obesity have been reported as risk factors for the most common type of diabetes, non-insulin-dependent diabetes mellitus (NIDDM; 27). Only 2 of 12 studies finding a positive association between childbearing and diabetes mellitus prevalence adjusted simultaneously for the potential confounding effects of age and adiposity (1,2). Only 1 of 2 studies reported the magnitude of the association between number of births and NIDDM prevalence (1).

Because of the relatively scarce amount of information on age- and obesity-adjusted effects of childbearing on glucose tolerance and diabetes mellitus prevalence, we studied this association in a sample of 3057 women in the United States who underwent clinical and laboratory evaluation for the presence of diabetes mellitus as part of a comprehensive health survey.

## RESEARCH DESIGN AND METHODS

The Second National Health and Nutrition Examination Survey (NHANES II) conducted between 1976 and 1980 focused on a probability sample of people selected from 64 different locations in the U.S. This survey was designed to reflect the target population consisting of civilian noninstitutionalized U.S. residents (including Alaska and Hawaii) between ages 6 mo and 74 yr. Certain subgroups of special interest for nutritional assessment (preschool-aged children, the elderly, and the poor) were oversampled to improve the statistical precision of measurements in these subgroups. The 17,390 people (9316 women, 8074 men) aged 20–74 yr selected for NHANES II were alternatively assigned to a subsample that was offered the oral glucose tolerance test (OGTT) in addition to the standard survey protocol, which included a medical history and physical examination (28). A total of 8686 men and women comprised the OGTT subsample (28).

Subjects selected for the survey underwent an interview, medical examination, and laboratory evaluation. The examination and laboratory evaluation were conducted in specially designed mobile examination centers that were transported to each sample location to provide standardized equipment and conditions. Subjects in the OGTT subsample reported to the examination center in the morning for an OGTT after having fasted overnight between 10 and 16 h. Subjects with previously diagnosed diabetes mellitus in the OGTT subsample underwent glucose tolerance testing, except for those currently treated with insulin. Pregnant women were also invited to take the OGTT. Eligible subjects underwent venipuncture for a fasting blood sample, followed by ingestion of an oral 75-g glucose load. One- and 2-h post–glucose load blood samples were also drawn. Frozen plasma was shipped to the Centers for Disease Control for glucose determinations with an automated modification of the National Glucose Reference Method developed at the Centers for Disease Control (29).

For the analysis reported herein, participants were classified as having diabetes mellitus based on data from the medical history and OGTT. Subjects currently treated with insulin or oral hypoglycemic agents were considered to have this metabolic condition, whereas other subjects were classified as to their glucose tolerance status with the National Diabetes Data Group criteria (27). Body mass index (BMI) was computed with the standard formula ($kg/m^2$). Height and weight were obtained by direct measurement of study subjects. Childbearing was defined as the number of live births experienced by the women at the time of the survey. Information was also obtained during an interview on other factors associated with NIDDM, e.g., family history of diabetes mellitus, ethnicity, educational level, and income (27).

Because we did not want to consider the effects of childbearing on glucose metabolism in women with insulin-dependent or gestational diabetes, we excluded pregnant women and women with diabetes onset before age 45 yr from the analysis. The latter exclusion effectively eliminates women with insulin-dependent diabetes mellitus because this condition rarely develops after age 45 yr. This age-based exclusion also probably eliminated subjects from the analysis whose childbearing plans may have been influenced by the development of diabetes. Additional exclusions specific to linear and logistic regression analyses are described below.

**Statistical methods.** A multiple linear regression analysis program that accounted for the complex sampling design of the study (SUPERCARP) was used to determine whether a linear relationship existed between the independent variable (number of live births) and the dependent variables (fasting and 2-h post–glucose load plasma glucose) while adjusting for potential confounding factors (30). The natural logarithm of plasma glucose was used in this analysis to reduce the skewness of the distribution of plasma glucose values. Additional exclusions for this analysis were use of oral hypoglycemic agents, because these medications alter glucose tolerance, and use of insulin, because subjects on insulin did not undergo the OGTT.

We examined the relative prevalence of diabetes mellitus in relation to childbearing using logistic regression analysis. Because the comparison of interest for this analysis was normal glucose tolerance versus diabetes, subjects with impaired glucose tolerance were excluded. To adjust the standard errors of the logistic regression coefficients for the NHANES II sampling design, we used the linear regression program in SUPERCARP to calculate simple random sample and complex survey coefficient standard errors for the exposures of interest in relation to presence or absence of diabetes mellitus. A complex survey design effect was then com-

puted by dividing the complex survey model variance by that obtained from the simple random sample model. The logistic regression coefficient standard error was then multiplied by the design effect to adjust for the complex survey design. Because statistical analysis of complex survey data usually leads to greater error estimates than analysis of random samples, the design-effect adjustment was only used on logistic regression coefficients that significantly differed from zero (31). A similar method has been used previously to estimate confidence intervals (CIs) for logistic regression coefficients obtained from NHANES II data analysis (32).

## RESULTS

Of 9316 women between ages 20 and 74 yr selected for NHANES II, 4577 were recruited to take an OGTT and 3057 (67% of 4577) reported for this test. A total of 1936 women (42% of 4577) were included in the analysis of childbearing and fasting plasma glucose after exclusion for diabetes mellitus diagnosed before age 45 yr ($n$ = 126), missing information on number of live births ($n$ = 19), current pregnancy ($n$ = 45), current use of oral hypoglycemic agents ($n$ = 59), and missing fasting plasma glucose value ($n$ = 976). Missing plasma glucose values were primarily due to inadequate fasting, subjects reporting in the afternoon, or technical errors, e.g., unsuccessful venipuncture or improper processing of specimens. Subtracting the total number of women excluded for various reasons from 3057 does not lead to the number included in the analyses, because women frequently met more than one exclusion criterion. A smaller number of women ($n$ = 1878, 41% of 4577) were included in the analysis of 2-h plasma glucose level and childbearing, due to a lower completion rate for the 2-h measure. A total of 1874 women were available for logistic regression analysis after including women taking insulin ($n$ = 40) and oral hypoglycemic agents ($n$ = 59) but excluding women with impaired glucose tolerance ($n$ = 115).

The level of fasting plasma glucose was positively associated with the number of live births in this sample (Table 1). Model 1 in Table 1 suggests that each live birth increases fasting plasma glucose by 0.056 mM. The effects of several possible confounding factors on the relationship between fasting plasma glucose and childbearing are shown in Table 1. Inclusion of race/ethnicity (defined as White, Black, Mexican American, or Native American) or family history of diabetes had no effect on the magnitude of the childbearing coefficient. Few subjects were in the high-risk ethnic categories for NIDDM (Mexican Americans, 2.5%; Native Americans, 1.4%). On the other hand, the inclusion of age and BMI substantially reduced the magnitude of the childbearing coefficient so that it no longer differed significantly from zero. Further adjustment for education and income did not change this result.

Examination of the relationship between 2-h post–glucose load plasma glucose and childbearing yielded similar findings. Number of live births appeared strongly related to 2-h glucose (Table 2, model 1), with each live birth associated with a 0.056-mM increase in plasma glucose. After adjustment for the confounding effects of age and BMI, however, the value of the coefficient for childbearing diminished to nearly zero (Table 2, model 4).

Mean numbers of live births by glucose tolerance are compared in Table 3. Mean number of live births was higher in women with NIDDM compared to those with normal glucose tolerance. The mean number of live births among women with impaired glucose tolerance was higher than in healthy women but not as high as among women with NIDDM.

Logistic regression analysis revealed a statistically significant increase in the relative prevalence of diabetes in relation to number of live births (Table 4). After adjustment for age, BMI, education, and income, the effect of childbearing on the relative prevalence of diabetes declined. When childbearing was entered into the logistic regression model as a set of categorical (dummy) variables with values shown in Table 4, no consistent linear relationship was seen between the relative prevalence of diabetes and number of live births. After adjustment of the categorical (dummy) variable model for age, BMI, education, and income, the magnitude of the childbearing-NIDDM relationship decreased markedly and no linear increase was apparent.

To investigate the potential effect of OGTT nonresponse on the above findings, we repeated the analysis of the association between NIDDM and childbearing using a medical history–based definition of diabetes status that permitted inclusion of a larger proportion of sampled subjects in the analysis. By redefining diabetes mellitus as self-report of a physician diagnosis of this disorder, data on this metabolic outcome became available for 6260 of 9316 women aged 20–74 yr selected for NHANES II, thereby increasing the response rate to 67%. We examined the relationship between childbearing and physician diagnosis of diabetes mellitus using logistic regression analysis, after excluding women diagnosed with diabetes before age 45 yr, women currently pregnant, and those missing data on number of live births. The relative prevalence of NIDDM in this analysis in relation to childbearing increased 1.19-fold for one live birth compared to none. After adjustment for age, BMI, education, and income, the relative prevalence diminished to 1.08. These results are similar to those obtained when the analysis included subjects who were classified based on OGTT results or use of hypoglycemic medication or insulin (Table 4). We further examined the association between parity and physician diagnosis of diabetes in the 1874 women who successfully completed the OGTT or were taking hypoglycemic medication. The relative prevalence of diabetes associated with one live birth was 1.22, which fell to 1.15 after adjustment for age, BMI, education, and income.

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**TABLE 1**
**Multiple linear regression analysis of logarithmic transformation of fasting plasma glucose in relation to childbearing (per live birth) among 1936 women**

| Regression model* | Constant | Coefficient | 95% confidence interval |
|---|---|---|---|
| 1 | | | |
| Childbearing | 4.48 | 0.009 | 0.006–0.012 |
| 2 | | | |
| Childbearing | 4.37 | 0.004 | 0.001–0.007 |
| Age | | 0.003 | 0.003–0.003 |
| 3 | | | |
| Childbearing | 4.26 | 0.005 | 0.002–0.008 |
| Body mass index | | 0.009 | 0.007–0.011 |
| 4 | | | |
| Childbearing | 4.20 | 0.001 | −0.002–0.004 |
| Age | | 0.002 | 0.002–0.002 |
| Body mass index | | 0.008 | 0.006–0.010 |
| 5 | | | |
| Childbearing | 4.48 | 0.009 | 0.006–0.012 |
| Mexican American | | −0.014 | −0.039–0.011 |
| Native American | | −0.013 | −0.046–0.020 |
| Black | | 0.018 | −0.010–0.046 |
| 6 | | | |
| Childbearing | 4.47 | 0.009 | 0.006–0.012 |
| Family history of diabetes mellitus | | 0.012 | 0.008–0.032 |
| 7 | | | |
| Childbearing | 4.66 | 0.007 | 0.004–0.010 |
| Education | | −0.028 | −0.040–−0.016 |
| Income | | −0.005 | −0.008–−0.002 |
| 8 | | | |
| Childbearing | 4.28 | 0.001 | −0.002–0.004 |
| Age | | 0.002 | 0.002–0.003 |
| Body mass index | | 0.008 | 0.006–0.010 |
| Education | | −0.006 | −0.016–0.003 |
| Income | | −0.003 | −0.006–−0.0002 |

*The following coding scheme was used for variables entered into analyses: childbearing (total number of live births), age in years, body mass index (kg/m$^2$), education (0, no grade school; 1, some grade school; 2, some high school; 3, some college), yearly income was divided into 12 ordered categories, lowest being <$1000/yr and highest being >$25,000/yr, and remaining variables were coded 1 for presence of characteristic or 0 otherwise.

diabetes onset before age 45 yr had on the analysis, we reexamined the relationship between fasting and 2-h plasma glucose and childbearing by linear regression analysis while excluding women with age of onset of diabetes of <30 yr. Thirty-six additional women were included in this analysis. Childbearing regression coefficients for fasting (coefficient 0.001, 95% CI −0.002–0.004) and 2-h (coefficient 0.0003, 95% CI −0.009–0.007) plasma glucose adjusted for age and BMI were similar to those obtained from the analyses that excluded women with age of onset before 45 yr.

## DISCUSSION

This analysis confirmed that fasting plasma glucose, 2-h post–glucose load plasma glucose, and prevalence of NIDDM all increase with increasing number of live births. The small detrimental effect of childbearing on glucose tolerance shown in these data, however, can be explained almost entirely by its correlation with other risk factors for NIDDM. After adjustment for these correlates, childbearing had no important or statistically significant effect on glucose tolerance or NIDDM prevalence. The most important correlates of childbearing, with regard to their effect on glucose tolerance, appear to be age and BMI, because the age- and BMI-adjusted effect of childbearing on both fasting and 2-h plasma glucose is similar to this effect when adjusted for educational level and income as well (Tables 1 and 2, models 4 and 8). Furthermore, the categorical (dummy) variable analysis did not support childbearing experience as a cause of NIDDM, because this analysis did not show a consistent increase in NIDDM prevalence with increased childbearing after adjustment for age, BMI, education, and income. This analysis indicates that women who have given birth have a greater tendency to develop diabetes primarily due to their greater age and BMI. This analysis did not find evidence to suggest that bearing children independently caused an increase in NIDDM prevalence. These

**TABLE 2**
**Multiple linear regression analysis of logarithmic transformation of 2-h post-glucose load plasma glucose in relation to childbearing (per live birth) among 1878 women**

| Regression model* | Constant | Coefficient | 95% confidence interval |
|---|---|---|---|
| 1 | | | |
| Childbearing | 4.63 | 0.015 | 0.009–0.021 |
| 2 | | | |
| Childbearing | 4.40 | 0.004 | −0.002–0.010 |
| Age | | 0.006 | 0.005–0.007 |
| 3 | | | |
| Childbearing | 4.16 | 0.006 | −0.002–0.014 |
| Body mass index | | 0.019 | 0.015–0.023 |
| 4 | | | |
| Childbearing | 4.04 | −0.001 | −0.009–0.007 |
| Age | | 0.005 | 0.004–0.006 |
| Body mass index | | 0.017 | 0.013–0.021 |
| 5 | | | |
| Childbearing | 4.62 | 0.015 | 0.009–0.021 |
| Mexican American | | 0.087 | −0.019–0.192 |
| Native American | | −0.077 | −0.195–0.041 |
| Black | | 0.037 | −0.006–0.080 |
| 6 | | | |
| Childbearing | 4.56 | 0.015 | 0.009–0.021 |
| Family history of diabetes mellitus | | 0.077 | 0.020–0.134 |
| 7 | | | |
| Childbearing | 4.97 | 0.011 | 0.005–0.017 |
| Education | | −0.049 | −0.080–−0.018 |
| Income | | −0.010 | −0.018–−0.002 |
| 8 | | | |
| Childbearing | 4.17 | −0.001 | −0.009–0.007 |
| Age | | 0.005 | 0.004–0.005 |
| Body mass index | | 0.017 | 0.013–0.020 |
| Education | | −0.004 | −0.030–−0.022 |
| Income | | −0.006 | −0.012–0.0003 |

*The following coding scheme was used for variables entered into analyses: childbearing (total number of live births), age in years, body mass index (kg/m$^2$), education (0, no grade school; 1, some grade school; 2, some high school; 3, some college), yearly income was divided into 12 ordered categories, lowest being <$1000/yr and highest being >$25,000/yr, and remaining variables were coded 1 for presence of characteristic or 0 otherwise.

findings imply that the excess risk of NIDDM associated with increasing number of births may be reduced to some degree through the shedding of excess weight associated with fertility.

The exclusion of women who developed diabetes before age 45 yr and pregnant women from this analysis were unlikely to have affected the results. As shown above, no relationship was observed between fasting or 2-h plasma glucose and number of live births when women with diabetes onset at ≥30 yr of age were included in the analysis. Women who were pregnant at the time of the survey were also excluded because of the possibility of including subjects with gestational diabetes in our analysis, which was intended to focus on women with NIDDM. This exclusion is unlikely to have biased the results toward the null value by eliminating parous women with poor glucose tolerance from this analysis, because only one pregnant woman had a history of diabetes mellitus. Furthermore, no pregnant women who completed the OGTT met the National Diabetes Data Group criteria for NIDDM or had either a fasting, 1-, or 2-h plasma glucose value exceeding the threshold values for gestational diabetes according to the O'Sullivan criteria (33).

A more important limitation of this study is that the result is biased due to the large nonresponse rate. To some extent, we were able to examine whether bias was present by comparing the associations between reported

**TABLE 3**
**Mean number of live births in relation to presence of normal glucose tolerance, impaired glucose tolerance, or non-insulin-dependent diabetes mellitus (NIDDM) among 1989 women**

| | *n* | Live births (mean *n*) | 95% confidence intervals |
|---|---|---|---|
| Normal glucose tolerance | 1694 | 2.27 | 2.16–2.38 |
| Impaired glucose tolerance | 115 | 2.76 | 2.01–3.51 |
| NIDDM | 180 | 3.27 | 2.89–3.65 |

**TABLE 4**
**Relative prevalence of non-insulin-dependent diabetes mellitus in relation to childbearing among 1874 women**

| Logistic regression model* | Relative prevalence (95% confidence interval) |
|---|---|
| 1 | |
| Childbearing† | 1.73 (1.39–2.15) |
| 2 | |
| Childbearing† | 1.07 (0.98–1.17) |
| Age (yr) | 1.08 (1.06–1.10) |
| Body mass index | 1.12 (1.08–1.16) |
| Education | 0.71 (0.51–0.98) |
| Income | 0.99 (0.92–1.07) |
| 3 | |
| 0 live births | 1.00 (referent) |
| 1 live birth | 1.02 (0.47–2.19) |
| 2 live births | 1.04 (0.53–2.04) |
| 3 live births | 2.48 (1.34–4.61) |
| 4 live births | 2.14 (1.03–4.48) |
| 5 live births | 1.62 (0.65–4.04) |
| ≥6 live births | 4.51 (2.32–8.77) |
| 4 | |
| 0 live births | 1.00 (referent) |
| 1 live birth | 0.76 (0.34–1.72) |
| 2 live births | 0.55 (0.26–1.16) |
| 3 live births | 1.47 (0.74–2.93) |
| 4 live births | 1.14 (0.51–2.55) |
| 5 live births | 0.54 (0.18–1.60) |
| ≥6 live births | 1.66 (0.79–3.52) |
| Age (yr) | 1.08 (1.06–1.10) |
| Body mass index | 1.11 (1.07–1.16) |
| Education | 0.70 (0.51–0.96) |
| Income | 0.99 (0.92–1.07) |

*The following coding scheme was used for variables entered into analyses: childbearing (total number of live births), age in years, body mass index (kg/m$^2$), education (0, no grade school; 1, some grade school; 2, some high school; 3, some college), yearly income was divided into 12 ordered categories, lowest being <$1000/yr and highest being >$25,000/yr, and remaining variables were coded 1 for presence of characteristic or 0 otherwise.
†One vs. 0 live births.

history of diabetes mellitus and childbearing after adjustment for confounding factors in the NHANES II sample and the OGTT subsample. Women in the larger NHANES II sample did not appear to differ from those who successfully completed the OGTT, with regard to several risk factors for diabetes mellitus and childbearing history. Mean age (42.1 yr), BMI (25.1), and number of live births (2.3) in the NHANES II sample ($n$ = 6260) appeared similar to the smaller OGTT subsample ($n$ = 2042, mean age 43.2 yr, mean BMI 25.2, mean number of live births 2.3), as did the proportion of physician-diagnosed diabetic women in the larger group (5.1%) compared to the smaller group (4.3%). These results argue against the presence of a response bias in this analysis due to the OGTT nonresponse rate.

Results of this study differ from the only other recently published research conducted in a U.S. population on the effects of childbearing on future diabetes mellitus prevalence (1). We considered the possibility that the difference in findings was because we examined the effects of number of live births on NIDDM prevalence, whereas Kritz-Silverstein et al. (1) examined the effects of total number of pregnancies (gravidity). To make our methods more directly comparable with those of Kritz-Silverstein et al., we analyzed the relative prevalence of NIDDM in relation to gravidity in our data while adjusting for the covariates shown in Table 4, model 2. The results of this analysis (relative prevalence 1.06, 95% CI 0.99–1.16) were nearly identical to our original analysis using number of live births as the exposure of interest. Therefore, the different definitions of childbearing do not account for the different results in two studies. A potential reason for the smaller effect of childbearing on NIDDM prevalence in this study compared with that of Kritz-Silverstein et al. is that the latent period between childbearing and NIDDM onset may be longer than we were able to detect. The study of Kritz-Silverstein et al. would have had an advantage in detecting an association between childbearing and NIDDM prevalence in the elderly if it existed, because their population was much older (mean age ~70 yr) and chronologically more distant from the childbearing years than ours (mean age 43 yr). Last, the effects of childbearing on the later development of NIDDM may differ in the Rancho Bernardo community studied by Kritz-Silverstein et al. compared to a national sample of women.

Although Kritz-Silverstein et al. conclude that the risk of NIDDM increased 1.16-fold/pregnancy, the data they present in Fig. 1 of their article suggest that an increase in risk does not appear until after the number of pregnancies exceeds five. For fewer than six pregnancies, the relative risk appears to be near one. The presence of a clear dose-response gradient in their data with respect to risk of NIDDM would have provided more convincing evidence for a causal relationship between reproductive history and NIDDM incidence (34). If it were true that six or more pregnancies are required to increase NIDDM prevalence, then childbearing is unlikely to have a significant impact on the prevalence of this disorder in the U.S., where few women experience more than five pregnancies.

Note, that while our results differ from those of Kritz-Silverstein et al. with regard to whether the null hypothesis was rejected, the 95% CI in our study for the age- and BMI-adjusted effects of childbearing (per live birth) on NIDDM prevalence included 1.16, the increase in diabetes prevalence detected by Kritz-Silverstein et al. for one pregnancy (1). Despite the fact that our results do not show a statistically significant increase in NIDDM prevalence with increasing number of live births, the results of the logistic regression model (Table 4, model 2) comparing number of live births to NIDDM prevalence do not exclude up to a 17% increase in prevalence per birth, as judged by the upper end of the 95% CI for the relative prevalence associated with live births.

In conclusion, we found no long-term independent

effects of childbearing on the prevalence of developing NIDDM. These results indicate that parous women who reduce their body weight to the levels of women of similar age and socioeconomic status who have not borne children may not be at higher risk for developing NIDDM.

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