Evidence Against Association Between Parity and NIDDM From Five Population Groups

Veronica R. Collins, BSc
Gary K. Dowse, MBBS, MSc
Paul Z. Zimmet, MD, PhD

Objective: To determine whether a reported positive association between parity and the development of non-insulin-dependent diabetes mellitus (NIDDM) and impaired glucose tolerance (IGT) is reproducible in other populations. Research Design and Methods: We investigated the relationship in data from population-based surveys in four Pacific and Indian Ocean island nations. Women ≥40 yr of age at the time of the survey, excluding those in whom diabetes developed before 40 yr of age, were included in this study of Micronesians from Nauru (*n* = 204) and Kiribati (*n* = 562), Fiji Melanesians (*n* = 390), Fiji Indians (*n* = 247), and mixed-ethnic Mauritians (*n* = 1333). Subjects in each survey underwent a 75-g oral glucose tolerance test, and glucose tolerance status was ascertained with 1985 World Health Organization criteria. Obstetric information and family history of diabetes were determined by interview. Results: Age and body mass index (BMI)–adjusted mean parity increased slightly with worsening glucose tolerance in only two groups, decreased in one group, and was inconsistent in the other two (none were statistically significant). We also found an inconsistent relationship between the number of full-term pregnancies and the prevalence of IGT and NIDDM, although in each population, there was a higher prevalence of NIDDM in the highest parity group (≥10 pregnancies) compared with the lowest parity group (1–3 pregnancies). In logistic regression analyses accounting for age, BMI, and family history of diabetes, odds ratio estimates for NIDDM and IGT associated with each pregnancy were not significantly greater than unity. Conclusions: The results indicate that there is little if any independent association between parity and the development of abnormal glucose tolerance in these populations. *Diabetes Care* **14:975–81, 1991**

A recent report has once again raised the controversial issue of the relationship between parity and the later development of non-insulin-dependent diabetes mellitus (NIDDM; 1). As early as 1933, there were reports that death from diabetes was more commonly recorded among married or widowed women than single women, suggesting an association between parity and diabetes (2,3). Subsequently, various studies have demonstrated that the frequency of diabetes, specifically the high rate in women, was associated with increasing parity (4–6). Furthermore, the association could not be explained entirely by the greater body mass of parous compared with nonparous women (7). Another study that used death certificates of women who have ever been married in England and Wales between 1938 and 1960 also reported that parous women died more frequently from diabetes mellitus than nonparous women (8).

Therefore, until ~1960, it was generally accepted that there was a positive association between parity and NIDDM. The subsequent appearance of several better controlled studies led to a reappraisal. In 1978, West (9) reviewed the literature and concluded that there was little evidence to support an independent relationship between parity and NIDDM. For example, West and Kalbfleisch (10) studied 10 populations and found a positive association in only 2, and in these, it may have been attributable to the greater adiposity of multiparous women. Similarly, a large survey conducted in the

From the International Diabetes Institute, Melbourne, Australia.
Address correspondence and reprint requests to Veronica Collins, BSC, International Diabetes Institute, Caulfield General Medical Centre, 260 Kooyong Road, Caulfield South, Victoria 3162, Australia.
Received for publication 7 January 1991 and accepted in revised form 27 June 1991.

United States found little relationship between parity and glucose levels after correcting for age and skin-fold thickness (11). Results from a study by Sicree et al. (12), which looked primarily at stillbirth rates in five Pacific island populations, indicated that any association between parity and abnormal glucose tolerance was very weak. These and other studies, along with observations that the prevalence of NIDDM is low in many underdeveloped countries where multiparity is common and that generally the prevalence of NIDDM is similar in men and women (13,14), led to the consensus position that parity conveyed little or no risk of NIDDM (9,14). Therefore, there has been little interest in this area until it was revived by a study of Californian women, which found a slight increase in the risk of NIDDM or impaired glucose tolerance (IGT) with increasing parity independent of the effects of obesity (1). Subsequently, Boyko et al. (15) presented data from the Second National Health and Nutrition Examination Survey (NHANES II), which did not support a role for the number of live births in the development of NIDDM after adjustment for age, body mass index (BMI), and income.

This study investigated the putative relationship between parity and NIDDM in five Pacific and Indian Ocean island populations, each of which demonstrated both high fertility and a high prevalence of abnormal glucose tolerance.

RESEARCH DESIGN AND METHODS

Five population groups from four medical surveys performed between 1980 and 1987 in Micronesians of Kiribati (1981) and Nauru (1987), Melanesians and migrant Asian Indians of Fiji (1980), and the mixed-ethnic population of Mauritius (1987) comprised this study. Full details of sampling procedures and response rates are published elsewhere but will be described here briefly (16–19). In Mauritius, two-stage cluster-sampling was used, whereby 10 areas were randomly selected based on census divisions, each with 500–600 residents. In addition, a specific area was chosen with a high proportion of Chinese residents to give a total of 11 areas. All adults 25–74 yr of age within these areas were eligible to attend the survey (19). In Fiji, what were considered to be typical rural villages or urban settlements were chosen on a deliberate basis. All adults ≥20 yr of age who lived within the defined geographic boundaries of those chosen areas were eligible to attend (16). Similarly, in Kiribati, a rural atoll where a traditional lifestyle had been maintained was selected to contrast with the islet of Betio, the most urbanized region of Kiribati. All adults ≥20 yr of age who resided in these two defined communities were eligible to attend, but in Betio, they were required to have been a resident for at least 5 yr (17). In Nauru, the eligible population was all Nauruans who had attended either or both of two previous diabetes surveys (78% of Nauruans ≥25 yr of age were thus eligible). The age distribution of the eligible population was similar to that of the total Nauruan population (18). Response rates exceeded 81% in each of these surveys (16–19).

Only women ≥40 yr of age at the time of each survey, excluding those in whom diabetes developed before 40 yr of age, were included in this study. This was to ensure that pregnancies (if any) occurred before the onset of diabetes and also to be consistent with the methods of Kritz-Silverstein et al. (1). Although the Mauritian population comprised three ethnic groups (Asian Indians, Creoles, and Chinese), the prevalence of abnormal glucose tolerance was similar in each (19), and they were combined into one group for analyses. However, the indigenous Melanesians and migrant Fiji Indians were analyzed separately because the prevalence of IGT and NIDDM was significantly higher in the Fiji Indians (16). The number of women included in this study were as follows: 204 Nauruans, 1333 Mauritians, 562 Kiribati islanders, 390 Fiji Melanesians, and 247 Fiji Indians (Table 1).

Methods were similar for each survey and are described elsewhere (16–20). Obstetric data were obtained through personal interview for information on

TABLE 1
Characteristics of study populations

	Nauruans	Mauritians	Kiribati islanders	Fiji Melanesians	Fiji Indians
n	204	1333	562	390	247
Median age (yr)	54.0 (40–80)	53.0 (40–74)	50.0 (40–81)	51.0 (40–87)	50.0 (40–95)
Prevalence ± SE					
Impaired glucose tolerance (%)	14.2 ± 2.4	23.8 ± 1.2	21.5 ± 1.7	15.4 ± 1.8	17.4 ± 2.4
Non-insulin-dependent diabetes mellitus (%)	49.0 ± 3.5	20.0 ± 1.1	10.0 ± 1.3	12.3 ± 1.7	23.1 ± 2.7
Mean ± SE					
Body mass index	35.2 ± 0.6	25.0 ± 0.1	26.0 ± 0.2	29.4 ± 0.3	25.3 ± 0.4
Parity	7.24 ± 0.28	5.48 ± 0.09	5.67 ± 0.14	5.36 ± 0.18	6.26 ± 0.22
Live births	6.92 ± 0.27	5.25 ± 0.08	4.65 ± 0.12	5.21 ± 0.18	6.09 ± 0.21
Positive family history of diabetes (%)	40.2 ± 3.4	19.3 ± 1.1	6.2 ± 1.0	9.6 ± 1.5	27.5 ± 2.8

Age ranges are given in parentheses.

number of full-term pregnancies, number of live births, number of stillbirths, and congenital malformations. A full-term pregnancy was defined as any delivery after the second trimester, and a live birth was any nonstill delivery. Parity was defined as the total number of full-term pregnancies, i.e., the number of live births plus stillbirths. Information on family history of diabetes was also obtained by personal interview. It has been recorded as a dichotomous variable, i.e., presence or absence of diabetes in first-degree family members.

In each survey, weight and height were measured with standard apparatus, and BMI was calculated as weight (kg) divided by height $(m)^2$.

Oral glucose tolerance tests (OGTTs) were performed with standardized methods (16–19). After an overnight fast, blood samples were taken into heparinized fluoridated tubes. Glucose loads (75 g) were administered, and 2 h later, further blood samples were taken. Specimens were centrifuged immediately, and plasma glucose was determined on site with a YSI glucose analyzer (Yellow Springs, OH) that used a glucose oxidase method. In Fiji and Kiribati, OGTTs were performed on all subjects (16,17). However, in Nauru and Mauritius, OGTTs were not administered to diabetic subjects taking hypoglycemic drugs or insulin or to subjects who reported a history of diabetes and were not being treated pharmacologically but who had a fasting glucose value ≥7.8 mM (18,19).

Classification of glucose tolerance was based on current World Health Organization criteria modified for epidemiological studies (21). Diabetes was diagnosed on the basis of a 2-h plasma glucose concentration ≥11.1 mM. Subjects who reported a history of diabetes, were taking insulin or oral hypoglycemic drugs, and/or had a fasting plasma glucose level ≥7.8 mM were also classified in the diabetes category, but only women diagnosed at ≥40 yr of age were included in this study. IGT was defined by a fasting plasma glucose level <7.8 mM and a 2-h plasma glucose concentration ≥7.8 mM but <11.1 mM. Others were classified as having normal glucose tolerance.

Means were adjusted for age and BMI by analysis of covariance, and differences across groups were assessed by the F ratio with SPSSx software (22). The prevalence of IGT and NIDDM across levels of increasing parity were tested for trend with a χ^2 test with 1 df (23).

Multiple logistic regressions were performed with BMDP statistical software (24) to obtain estimates of the odds ratio (OR) of IGT or NIDDM associated with parity, adjusted for the effects of age, obesity, and family history of diabetes. Parity, age, and BMI were entered as continuous variables such that OR estimates referred to a unit change in these variables. Family history of diabetes was entered as a dichotomous variable as previously described. For both IGT and NIDDM, comparison was with the group with normal glucose tolerance. A separate regression analysis was done for each of the five groups. Two-way interaction terms were tested for inclusion in the full models, and 0.05 was chosen as the discriminating level of significance. Statistically sig-

TABLE 2
Mean parity and live births adjusted for age and body mass index by glucose tolerance

Population	*n*	Parity		Live births	
		Unadjusted	Adjusted	Unadjusted	Adjusted
Nauruan					
Normal	74	7.39	7.56	7.14	7.27
IGT	29	6.72	6.75	6.52	6.53
Diabetes	99	7.30	7.10*	6.90	6.76*
Mauritian					
Normal	746	5.30	5.56	5.10	5.33
IGT	317	5.50	5.50	5.28	5.28
Diabetes	266	5.93	5.67*	5.64	5.40*
Kiribati islanders					
Normal	383	5.45	5.45	4.49	4.45
IGT	121	5.93	5.92	4.93	4.92
Diabetes	56	6.48	6.49*	5.13	5.17*
Melanesian					
Normal	281	5.34	5.38	5.20	5.20
IGT	59	5.05	5.06	4.88	4.91
Diabetes	48	5.92	5.86*	5.71	5.68*
Indian					
Normal	147	5.91	6.11	5.76	5.93
IGT	43	6.54	6.42	6.33	6.23
Diabetes	57	6.95	6.85*	6.79	6.71*

IGT, impaired glucose tolerance.
*Differences in the means across glucose tolerance groups were not significant at the 0.05 level, according to the F test.

nificant interaction terms were explored further to determine the nature of the interaction and were either retained or removed from the models depending on the biological plausibility or importance of the interaction.

RESULTS

Characteristics of the study populations are in Table 1. The median age was slightly higher for Nauruans and Mauritians, although the range was wider for the other three groups. The populations showed considerable variation with respect to abnormal glucose tolerance and obesity, with Nauruans having the highest prevalence of NIDDM and the highest mean BMI. Mean parity was also highest in Nauruans, whereas it was similar for Mauritians, Kiribati islanders, and Fiji Melanesians.

Table 2 shows mean parity and live births unadjusted and adjusted for age and BMI according to glucose tolerance. Adjusting for age and BMI made little difference to the results. Only Kiribati islanders and Fiji Indians showed a trend of increasing parity with worsening glucose tolerance, although the changes were not statistically significant. Mauritians and Fiji Melanesians showed higher mean parity among the diabetic than normal or IGT groups, but conversely, adjusted mean parity for Nauruans with diabetes was lower than for those with normal glucose tolerance. The magnitude of these differences was minor and not statistically significant. These patterns were reproduced when live births only were considered.

The proportion of subjects with IGT or NIDDM according to parity is shown in Table 3. The relationship between parity and the prevalence of IGT was inconsistent. Only in Nauruans did there appear to be a positive association of IGT and parity (trend not statistically significant), although there was a marked decline in prevalence in the group with the highest parity. There was a significant trend for increasing prevalence of NIDDM with increasing parity only in Mauritians and Kiribati islanders, although in all populations, there was a higher prevalence of NIDDM in the high-parity group compared with the low-parity group. For combined abnormal glucose tolerance, the trend across parity groups just failed to reach statistical significance for Mauritians and Kiribati islanders.

Logistic regression analyses were performed to determine the effect of parity on IGT or NIDDM after accounting for the effects of age, obesity, and family history of diabetes. Table 4 lists the standardized parameter estimate and OR estimate for NIDDM for each population. The OR estimates per pregnancy were marginally elevated in three of the five populations but were not statistically significant in Fiji Melanesians (OR = 1.04) or Fiji Indians (OR = 1.07) and were of borderline significance in Kiribati-islander women (OR = 1.09, $P = 0.055$). However, in Nauruans and Mauritians, the OR estimates were less than unity, although again they were not statistically significant.

TABLE 3
Proportion (%) of impaired glucose tolerance (IGT) and non-insulin-dependent diabetes mellitus by parity groups

	Parity groups					
	0	1–3	4–5	6–9	≥10	χ^{2*}
Nauruan						
n	8	29	38	62	65	
IGT	12.5	13.8	15.8	21.0	7.7	2.44
Diabetes	25.0	48.3	57.9	43.5	52.3	0.38
IGT + diabetes	37.5	62.1	73.7	64.5	60.0	0.01
Mauritian						
n	84	306	291	503	149	
IGT	28.6	21.2	24.1	24.5	23.5	0.06
Diabetes	19.0	18.0	18.2	20.5	26.8	4.29†
IGT + diabetes	47.6	39.2	42.3	45.0	50.3	3.57
Kiribati islanders						
n	44	104	112	227	75	
IGT	20.5	18.3	20.5	23.8	21.3	0.16
Diabetes	6.8	6.7	10.7	9.7	16.0	3.89†
IGT + diabetes	27.3	25.0	31.2	33.5	37.3	3.72
Melanesian						
n	46	87	57	147	52	
IGT	17.4	17.2	15.8	16.3	7.7	1.74
Diabetes	4.3	13.8	12.3	12.9	15.4	1.16
IGT + diabetes	21.7	31.0	28.1	29.2	23.1	0.08
Indian						
n	13	38	54	100	42	
IGT	7.7	21.1	16.7	15.0	23.8	0.49
Diabetes	15.4	13.2	27.8	24.0	26.2	1.65
IGT + diabetes	23.1	34.3	44.5	39.0	50.0	2.70

*χ^2 (1 df) test for trend (23).
†$P < 0.05$.

Table 5 shows a similar analysis for IGT. Again, in Nauru and Mauritius, parity had a marginally negative nonsignificant association with IGT (OR 0.94 and 0.99, respectively). Fiji Melanesians also showed a negative association between parity and IGT, whereas Kiribati islanders had an OR of 1.05 for each pregnancy (not statistically significant). In Fiji Indians, there was a significant interaction between parity and family history of diabetes. When this interaction was investigated further, there was an apparent positive association between the prevalence of IGT and the total number of pregnancies for subjects without a family history of diabetes but not for those with a family history of diabetes.

All logistic regression analyses were repeated with live births rather than parity, but results were similar to those reported in Tables 4 and 5 and are not shown here.

CONCLUSIONS

A study in American women reported a statistically significant, albeit small, increased risk of abnormal glucose

TABLE 4
Regression coefficients and odds ratio estimates for non-insulin-dependent diabetes mellitus for each population

Population	Variable	β coefficient	Odds ratio	95% Confidence interval	*P*
Nauruan (*n* = 168)	Parity	−0.035	0.97	0.89–1.05	0.426
	Age	0.083	1.09	1.04–1.13	<0.001
	BMI	−0.007	0.99	0.95–1.04	0.774
	FH	0.789	2.20	1.08–4.48	0.032
Mauritian (*n* = 1010)	Parity	−0.007	0.99	0.95–1.04	0.762
	Age	0.075	1.08	1.06–1.10	<0.001
	BMI	0.114	1.12	1.09–1.16	<0.001
	FH	0.626	1.87	1.29–2.71	<0.001
Kiribati islanders (*n* = 439)	Parity	0.085	1.09	1.00–1.19	0.055
	Age	0.041	1.04	1.01–1.07	0.007
	BMI	0.098	1.10	1.05–1.16	<0.001
	FH	1.208	3.35	1.38–8.11	0.008
Fiji Melanesian (*n* = 325)	Parity	0.035	1.04	0.95–1.13	0.415
	Age	0.067	1.07	1.04–1.10	<0.001
	BMI	0.042	1.04	0.99–1.10	0.107
	FH	0.819	2.27	0.86–5.95	0.091
Fiji Indian (*n* = 204)	Parity	0.066	1.07	0.97–1.18	0.196
	Age	0.014	1.01	0.98–1.05	0.418
	BMI	0.133	1.14	1.07–1.22	<0.001
	FH	0.185	1.20	0.58–2.48	0.622

FH, family history of diabetes; BMI, body mass index.

tolerance associated with increasing parity, independent of age, family history of diabetes, and obesity (1). In contrast to these findings in the white upper-middle class population of Rancho Bernardo, our results from five Pacific and Indian Ocean populations do not support the hypothesis implicating parity as a risk factor for

TABLE 5
Regression coefficients and odds ratio estimates for impaired glucose tolerance for each population

Population	Variable	β coefficient	Odds ratio	95% Confidence interval	*P*
Nauruan (*n* = 101)	Parity	−0.066	0.94	0.84–1.05	0.256
	Age	0.052	1.05	1.00–1.11	0.065
	BMI	0.029	1.03	0.97–1.09	0.361
	FH	0.009	1.01	0.37–2.78	0.986
Mauritian (*n* = 1061)	Parity	−0.009	0.99	0.95–1.03	0.646
	Age	0.031	1.03	1.02–1.05	<0.001
	BMI	0.081	1.08	1.05–1.12	<0.001
	FH	0.231	1.26	0.89–1.78	0.189
Kiribati islanders (*n* = 504)	Parity	0.044	1.05	0.98–1.11	0.168
	Age	0.021	1.02	1.00–1.04	0.058
	BMI	0.088	1.09	1.05–1.14	<0.001
	FH	0.007	1.01	0.40–2.51	0.988
Fiji Melanesian (*n* = 336)	Parity	−0.032	0.97	0.89–1.05	0.441
	Age	0.048	1.05	1.02–1.08	0.001
	BMI	0.007	1.01	0.96–1.06	0.759
	FH	0.865	2.37	0.99–5.68	0.053
Fiji Indian (*n* = 190)	Parity*	0.097			
	Age	0.015	1.02	0.98–1.05	0.392
	BMI	0.153	1.16	1.08–1.24	<0.001
	FH*	1.645			
	Parity FH	−0.305	0.74	0.55–0.99	0.043

FH, family history of diabetes; BMI, body mass index.
*Odds ratio estimates for parity and FH as main effect terms are not meaningful due to the interaction term, therefore they have not been shown in the table.

NIDDM and IGT. Our findings are similar to those recently published from the NHANES II study, which did not confirm the data from Rancho Bernardo (15). However, the NHANES II study used different methodology, in that they examined the association with the number of live births rather than parity, and they only excluded women <45 yr of age if they had diabetes. Our methods were deliberately similar to those of the Rancho Bernardo study (1), in that all women included were ≥40 yr of age, and those with diabetes were included only if they had not been diagnosed before 40 yr of age. These restrictions effectively limit the study to women who completed their families before onset of diabetes, whereby although our surveys were performed cross sectionally, the data are prospective.

Kritz-Silverstein et al. (1) found a significant increase in the mean number of pregnancies with worsening glucose tolerance. Results for our populations did not support this finding, although two groups showed an unconvincing trend in this direction. Similarly, our independent OR estimates for the risk of NIDDM or IGT associated with parity are not consistent with those of Kritz-Silverstein et al. (1). The magnitude of the effects found in that study, i.e., OR of 1.16 for NIDDM and 1.10 for IGT, are very unlikely in our study, given the confidence intervals described in Tables 4 and 5. Considered together, our data suggest that the true independent risk associated with parity is probably not elevated in these groups.

Obesity is a known risk factor for NIDDM and has been suggested as the underlying factor in the apparent parity-NIDDM relationship in some studies (10), because multiparous women tend to be more obese than nonparous. The women in the Rancho Bernardo study (1) were described as relatively lean, and the authors suggested that this enabled them to study the relationship between parity and NIDDM with little confounding by obesity. In any case, their results indicated that BMI had no appreciable effect on parity. In our study, the degree of obesity varied considerably between populations (Table 1), but mean BMI in Mauritians, Fiji Indians, and Kiribati islanders was in fact similar to that of the Rancho Bernardo population. Nonetheless, a relationship was not apparent in any group regardless of whether adjustment was made for the effect of obesity within populations. Although an increase in the number of live births with worsening glucose tolerance was found in the NHANES II study (15), this association could be explained almost entirely by the effects of BMI and age.

We used a similar methodology to Kritz-Silverstein et al. (1), but there were still differences between the study populations that might have accounted for the conflicting results. Factors involved in the development of NIDDM in the ethnic groups of our study (i.e., Micronesian, Melanesian, Indian, Creole, and Chinese) may be different from those for the white women of Rancho Bernardo. However, it seems unlikely that the mechanism responsible for an association between parity and NIDDM will be operating only in white women and not in other ethnic groups. Moreover, Boyko et al. (15) reported a lack of association in another group of white women. A potentially more important difference is that women in our study were considerably younger (median ages 50–54 yr) than those in the Rancho Bernardo study (mean age ~71 yr). If the time span between childbearing and its effect on the development of IGT or NIDDM is long, we may not detect the effect until our populations become older. Boyko et al. (15) have also suggested that younger age was a potential difficulty in their study (mean age 43 yr). However, note that age-specific data for the Rancho Bernardo study did not indicate a stronger effect in older age-groups, except perhaps a slight increase in women ≥80 yr of age (1).

Boyko et al. (15) noted that the reported increase in the risk of NIDDM in the Rancho Bernardo study is not actually a linear relationship and does not become apparent until the number of pregnancies exceeds five. A similar finding was reported in an earlier study (25) based on nondiabetic and diabetic women attending an outpatient clinic. The expected number of diabetic women within each level of parity (based on distribution of nondiabetic women within parity groups) was exceeded significantly by the observed number of women only after six pregnancies. Our results did not show a linear trend for the prevalence of NIDDM, IGT, or combined abnormal glucose tolerance across parity groups nor was there a particular change after five or six pregnancies. Therefore, these data from a number of populations call into question the general applicability of the Rancho Bernardo findings. Certainly, the lack of a dose-response effect in that study also raises questions about whether the relationship is causal.

This study in five populations and that of Boyko et al. (15) in a representative sample of American women do not provide supporting evidence for an association between parity and abnormal glucose tolerance. In 1978, West (9) stated that ". . . the effect of pregnancy is probably negligible or trivial in many societies." Quite possibly, "many" should be amended to most or all.

ACKNOWLEDGMENTS

This study was supported in part by the National Institute of Diabetes and Digestive and Kidney Diseases Grant DK-25446.

We gratefully acknowledge the support of the governments and people of the participating countries in the conduct of these surveys. The World Health Organization contributed to the Fiji and Mauritius surveys, and the Australian International Development Assistance Bureau supported the work in Kiribati. The Mauritian data are included on behalf of the Mauritius Noncommunicable Disease Study Group (19). We also thank Angela Fischer for preparation of the manuscript.

REFERENCES

1. Kritz-Silverstein D, Barrett-Connor E, Wingard D: The effect of parity on the later development of non-insulin-dependent diabetes mellitus or impaired glucose tolerance. *N Engl J Med* 321:1214–19, 1989
2. Mosenthal HO, Bolduan C: Diabetes mellitus—problems of present-day treatment. *Am J Med Sci* 186:605, 1933
3. Joslin EP, Dublin LI, Marks HH: Studies in diabetes mellitus. IV. Etiology. *Am J Med Sci* 191:759–775, 1936
4. Pyke DA: Parity and the incidence of diabetes. *Lancet* 270:818–21, 1956
5. Fitzgerald MG, Malins JM, O'Sullivan DJ, Wall M: The effect of sex and parity on the incidence of diabetes mellitus. *Q J Med* 30:57, 1961
6. Middleton GD, Caird FI: Parity and diabetes mellitus. *Br J Prev Soc Med* 22:100–104, 1968
7. Pyke DA, Please NW: Obesity, parity and diabetes. *J Endocrinol* 15:26–33, 1957
8. Beral V: Long term effects of childbearing on health. *J Epidemiol Community Health* 39:343–46, 1985
9. West KM: *Factors associated with occurrence of diabetes. Epidemiology of Diabetes and Its Vascular Lesions*. Elsevier, New York, 1978, p. 221–24
10. West KM, Kalbfleisch JM: Diabetes in Central America. *Diabetes* 19:656–63, 1970
11. O'Sullivan JB, Gordon T: *Childbearing and Diabetes Mellitus*. Washington, DC, U.S. Govt. Printing Office, 1966 National Center for Health Statistics (publ. no. 1000, ser. 11, no. 21)
12. Sicree R, Hoet J, Zimmet P, King H, Coventry J: The association of non-insulin-dependent diabetes with parity and still-birth occurrence amongst five Pacific populations. *Diabetes Res Clin Pract* 2:113–22, 1986
13. King H, Zimmet P: Trends in the incidence and prevalence of diabetes: non-insulin-dependent diabetes mellitus. *World Health Stat Q* 41:190–96, 1988
14. Dowse G, Zimmet P: The prevalence and incidence of non-insulin dependent diabetes mellitus. In *Frontiers of Diabetes Research: Current Trends in Non-Insulin-Dependent Diabetes Mellitus*. Alberti KGMM, Mazze R, Eds. Amsterdam, Elsevier, 1989, p. 37–59
15. Boyko EJ, Alderman BW, Keane EM, Baron AE: Effects of childbearing on glucose tolerance and NIDDM prevalence. *Diabetes Care* 13:848–54, 1990
16. Zimmet P, Taylor R, Ram P, King H, Sloman G, Raper LR, Hunt D: Prevalence of diabetes and impaired glucose tolerance in the biracial (Melanesian and Indian) population of Fiji: a rural-urban comparison. *Am J Epidemiol* 118: 673–88, 1983
17. King H, Taylor R, Zimmet P, Pargeter K, Raper LR, Beriki T, Tekanene J: Non-insulin-dependent diabetes (NIDDM) in a newly independent Pacific nation: the Republic of Kiribati. *Diabetes Care* 7:409–15, 1984
18. Collins V, Dowse G, Finch C, Zimmet P, Linnane A: Prevalence and risk factors for micro- and macroalbuminuria in diabetic subjects and entire population of Nauru. *Diabetes* 38:1602–10, 1989
19. Dowse G, Gareeboo H, Zimmet P, Alberti KGMM, Tuomilehto J, Fareed D, Brissonnette LG, Finch C: High prevalence of NIDDM and impaired glucose tolerance in Indian, Creole, and Chinese Mauritians. *Diabetes* 39:390–96, 1990
20. Dowse G, Zimmet P, Gareeboo H, Alberti KGMM, Tuomilehto J, Finch C, Chitson P, Tulsidas H: Abdominal obesity and physical inactivity are risk factors for both NIDDM and impaired glucose tolerance in Indian, Creole, and Chinese Mauritians. *Diabetes Care* 14:271–82, 1991
21. World Health Organization: *Diabetes Mellitus: Report of a WHO Study Group*. Geneva, World Health Org., 1985 (Tech. Rep. Ser., no. 727)
22. SPSS Inc.: *SPSSx User's Guide*. 2nd Ed., New York, McGraw-Hill, 1986
23. Armitage P, Berry G: *Statistical Methods in Medical Research*. 2nd ed. Oxford, UK, Blackwell, 1987, p. 372–74
24. Dixon WJ (Ed.): *BMDP Statistical Software Manual*. Berkeley, Univ. of Calif. Press, 1985
25. Jackson WPU: Is pregnancy diabetogenic? *Lancet* 2: 1369–71, 1961