Early Infant Diet and Risk of IDDM in Blacks and Whites

A Matched Case-Control Study

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OBJECTIVE — To investigate the role of early infant feeding in the development of insulin-dependent diabetes mellitus (IDDM) and to determine whether an association exists in both blacks and whites.

RESEARCH DESIGN AND METHODS — Black and white diabetic subjects were recruited from the Allegheny County and Children's Hospital of Pittsburgh IDDM Registries. Extensive infant diet histories were obtained from the diabetic subjects and their nondiabetic siblings, who were used as nondiabetic control subjects. Each diabetic subject was matched outside his/her family to an unrelated nondiabetic control subject on birth order, birth year (±2 yr), and race, which resulted in 211 case-control pairs with a mean birth year of 1967.

RESULTS — In whites, diabetic subjects were less likely to have been breast-fed than control subjects (odds ratio [OR] 0.5, 95% confidence interval [CI] 0.3,0.9). Breast-feeding prevalence did not differ between black diabetic subjects and control subjects. Duration of overall and exclusive breast-feeding did not differ between diabetic and control subjects in the black and white cohorts. The following analyses, which examined whether the timing of the first breast milk substitute to which the infant was exposed differed between diabetic and control subjects, were conducted for exposure to any breast milk substitute and to breast milk substitutes that were cow's milk based. In whites, age at exposure to any breast milk substitutes and cow's milk–based substitutes were similar between diabetic and control subjects. In blacks, the first exposure to breast milk substitutes occurred significantly earlier for any substitute (5.1 vs. 11.9 wk, $P = 0.02$) and marginally earlier for cow's milk–based substitutes (3.9 vs. 8.5 wk, $P = 0.07$) in diabetic subjects compared with control subjects. The first exposure to breast milk substitutes was more likely to occur by 3 mo of age in black diabetic subjects compared with black control subjects (OR 3.3, 95% CI 1.1–10.0) after adjusting for maternal age at birth. The addition of breast-feeding status to the model only slightly weakened this association in blacks.

CONCLUSIONS — The analyses of this study cohort suggest that the observed protective effect of breast-feeding on the risk of IDDM may be related to differences in the age at exposure to breast milk substitutes in blacks but not in whites.

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Ecological data from Scandinavia that demonstrated an inverse relationship between rates of insulin-dependent diabetes mellitus (IDDM) and breast-feeding suggested that breast-feeding may protect the infant from IDDM later in life (1). Case-control studies of the role of breast-feeding in the etiology of IDDM have both supported (1–4) and contradicted (5–7) this hypothesis. The inconsistent results may be attributed to inadequate measures of exposure (i.e., breast-feeding status or duration), particularly if breast-feeding is acting as a surrogate for perinatal factors that are causally associated with IDDM (i.e., foods given to the infant in place of breast milk). For example, a recent study from Finland reported that diabetic children were younger at the introduction of supplementary milk feeding compared with nondiabetic children (8). Also, animal models have shown that exposure to cow's milk protein sources at weaning can trigger diabetes (9–11). In addition, the inconsistent results may be due, in part, to confounding of the infant nutrition data by maternal age, birth order, and birth year. For example, because one's birth order within a family is inversely related to the likelihood of being breast-fed (12), birth order is an important confounder when coupled with the observation that higher birth order is related to increased risk of IDDM (13–15). Although the previous studies did not account for differences in birth order, their positive findings could have been influenced by the aforementioned birth-order effect.

Previous studies were conducted in relatively homogeneous populations at medium-to-high risk of IDDM (1–7). When evaluating a potential etiologic factor, it is important to determine whether the risk factor exerts a similar influence in populations with varying rates of IDDM and risks of exposure. American blacks have a lower incidence of IDDM (9.6/100,000 person-yr [PY]) (16) and markedly different infant nutrition practices (17,18) compared with

Table 1—*Percentage of individuals with insulin-dependent diabetes mellitus (IDDM) by sibship size and birth order in study cohort*

SIBSHIP SIZE	2	3	4	5
FAMILIES (N)	73	65	37	18
BIRTH ORDER (% W/IDDM)				
1ST	44	32	13	11
2ND	56	29	27	17
3RD		39	22	17
4TH			38	17
5TH				39
X^2 TEST OF TREND (P)	0.14	0.46	0.03	0.06

American whites, whose IDDM incidence is 15.4/100,000 PY (16).

This retrospective study of infant diet and IDDM was conducted to determine whether the relationship between breast-feeding and IDDM could be attributed to other characteristics of the infant diet (i.e., exposure to specific breast-milk substitutes and the age at which this occurred) while controlling for confounders such as birth order. Our infant diet data provided us with the ability to distinguish between cow's milk–based and non-cow's milk–based breast milk substitutes. Moreover, this study was designed to determine whether the association between infant diet and IDDM exists in both blacks and whites.

RESEARCH DESIGN AND METHODS

— To obtain a study cohort of black diabetic subjects sufficiently large enough to evaluate the aforementioned relationship given the low incidence of IDDM in blacks, both the Allegheny County IDDM Registry and Children's Hospital of Pittsburgh (CHP) IDDM Registry were used to recruit black diabetic subjects and their families. The Allegheny County IDDM Registry is a population-based registry that was developed through hospital record review of all the hospitals in Allegheny County and of two hospitals outside the county. Independent pediatrician validation has revealed that ascertainment is >90%. The patients registered were all newly diagnosed with IDDM, residents of Allegheny County, and on insulin at the time of hospital discharge (19). In addition to those in the Allegheny County IDDM Registry, blacks who were diagnosed with IDDM at CHP or seen at CHP within 1 yr of diagnosis for a diabetes evaluation (20) were also recruited for the study.

Criteria for inclusion in the infant diet study were *1*) <17 yr of age at diabetes diagnosis, *2*) diagnosed between 1965 and 1989, *3*) currently living, and *4*) diabetic subject's mother currently living, which was necessary to obtain accurate infant diet information. Seventy-five percent (n = 79) of the 105 eligible black subjects agreed to participate. There were no differences in birth year, age at diagnosis, year of diagnosis, sex, current age of mother, or sibship size between black diabetic subjects who agreed to participate and those who refused to participate (data not shown). To recruit a comparable white cohort, two white IDDM subjects of the same sex and birth year (±1 yr) as a participating black IDDM subject and who fit the aforementioned criteria were randomly selected from the Allegheny County and CHP IDDM registries. This was done to ensure that the black and white cohorts were comparable in terms of temporal influences in the infant diet and length of maternal recall of infant diet. Of the 167 white IDDM subjects contacted, 158 participated in the study. Black diabetic subjects had a similar mean year of IDDM diagnosis (1978 vs. 1977, $P > 0.05$) and were significantly older at diabetes diagnosis (10.1 vs. 8.6 yr, $P = 0.02$) compared with white diabetic subjects.

The control subjects were selected from the pool of nondiabetic siblings of the subjects. To control for the potentially confounding effect of birth order and birth year on the analyses of infant nutrition, each diabetic subject was matched to an unrelated nondiabetic control subject (i.e., outside his/her family) on birth order, birth year (±2 yr), and race. Cases were not matched to a sibling control subject for the following reasons: *1*) it was inappropriate to compare the infant diets of a case and a sibling control subject because of the lack of independence of infant diets within families (21), 2) IDDM risk was not independent of birth order in this population (Table 1), and 3) the likelihood of being breast-fed was inversely related to birth order (12). After matching the diabetic subjects to the nondiabetic control subjects on race, birth order, and birth year, 211 pairs (55 black and 156 white) were available for analyses. The mean birth year for white and black case-control pairs was 1968 and 1965, respectively. In whites, 51% of the IDDM subjects and 54% of the control subjects were male. In blacks, 45% of the IDDM subjects and 60% of the control subjects were male. White IDDM subjects were more likely to have a mother ≥35 yr of age at their birth than were white control subjects (odds ratio [OR] 3.3, confidence interval [CI] 0.9–15.4), although this was only marginally significant. Maternal age at birth was not significantly different between IDDM subjects and control subjects in blacks (OR 4, CI 0.3–38.5).

Infant diet data

The infant nutrition data were collected via mailed questionnaires to only the mothers. Telephone and face-to-face interviews were conducted if the mothers failed to return the questionnaires by

mail. The survey questions covered several issues concerning the perinatal experience of the diabetic child and his/her siblings; the mothers were not told of the dietary focus of the study. The data on feeding in infancy included questions on the duration of overall and exclusive breast-feeding, the child's age at introduction of breast milk substitutes and solid foods into the diet, the type of breast-milk substitute (i.e., milk based, soy based or special infant formula, evaporated milk, powdered milk, fresh cow's milk, cow's milk with Lactaid, and goat's milk), and brand name of formula. Special infant formulas included formulas with hydrolyzed protein. Copies of the questionnaire are available from the author (J.N.K.).

Variables were calculated to measure the risk associated with age at exposure to breast milk substitutes. The age at which the infant was first exposed to an infant milk other than breast milk was calculated from the infant diet history by reasoning that non–breast-fed infants were exposed to breast milk substitutes at birth and breast-fed infants were exposed to breast milk substitutes when either supplementation or weaning occurred. This variable was also categorized into substitutes containing cow's milk and those not containing cow's milk. Cow's milk–based substitutes included fresh cow's milk, cow's milk with Lactaid, homemade evaporated milk formulas, powdered cow's milk, and commercial cow's milk–based infant formulas. Substitutes not based on cow's milk included commercial soy-based and special infant formulas and goat's milk.

Statistical analyses

The data analyses were performed with the SAS statistical software package (22). McNemar's matched-pairs test was used to analyze dichotomous variables, and the Wilcoxon's matched-pairs signed-rank test was used to analyze continuous variables. Wilcoxon's test was used to evaluate the difference between two survival curves depicting the age at exposure to breast milk substitutes. Conditional multiple logistic regression analyses were used to examine the relationship between the binary-dependent variable (case-control status) and the independent variables in the matched data set (23).

Data validation

Medical records were requested for the diabetic subject to compare the survey data with the data abstracted from the records to determine the validity of the survey instrument. The birth record was obtained to determine initiation of breast-feeding. The pediatric record was obtained to determine later aspects of the infant diet. Eighty-one percent of the cases (n = 191) agreed to release their records, and 145 birth records and 81 pediatric records could be obtained from the hospitals and physician's practices, respectively. Medical records (birth and/or pediatric records) were less likely to have been available for subjects born before 1970 compared with those born later (49 vs. 79%, $P < 0.05$) and for black subjects compared with white subjects (37 vs. 72%, $P < 0.05$). The survey information was in excellent agreement with the hospital and pediatric records regarding breast-feeding status (κ =0.96 and 0.90, respectively). The mean differences (d) in weeks and 95% CI between the pediatric records and the survey data for duration of breast-feeding (d −4.8, CI −15.3–10.5) and infant formula use (d 0.4, CI −6.7–7.5) and the age at introduction of infant formula (d −0.6, CI −1.9–0.6), cow's milk (d −1.8, CI −7.4,3.8), and solid food (d −0.5, CI −2.2–1.2) were not significant. Stratification by length of recall (<15 and ≥15 yr) showed adequate maternal recall of infant diet in both groups (data not shown).

RESULTS — In whites, although IDDM subjects were less likely to have been breast-fed than control subjects (OR 0.5, CI 0.3,0.9), overall breast-feeding duration (23 vs. 25 wk, P = 0.8) and exclusive breast-feeding duration (18 vs. 13 wk, P = 0.4) were similar between IDDM and control subjects. No significant difference in breast-feeding status in IDDM and control subjects was seen in blacks (OR 0.5, CI 0.2,1.4). The differences in duration of breast-feeding (17 vs. 28 wk, P = 0.13) and exclusive breast-feeding (13 vs. 27 wk, P = 0.16) between black IDDM subjects and control subjects were not statistically significant.

We examined the risk associated with the first breast milk substitute to which the infant was exposed and whether the type and age at exposure differed between IDDM and control subjects. Black IDDM subjects were exposed to any breast milk substitutes and cow's milk–based substitutes earlier than control subjects, although the latter comparison was only marginally significant (Table 2). There were little differences in age at exposure to either cow's milk–based substitutes or any breast milk substitutes between white IDDM subjects and control subjects. Comparisons of age at exposure to non–cow's milk–based substitutes were not done because of the small number of individuals (n = 19) whose first breast milk substitute was not cow's milk based.

Figure 1 shows the age at first exposure to any breast milk substitutes in blacks and whites. Approximately 60% of control subjects and 70% of the IDDM subjects were exposed to a substitute in the 1st wk of life, which is consistent with the low prevalence of breast-feeding in this population (36%). The curves are significantly different in blacks but not in whites (Wilcoxon's test, P = 0.04 and 0.33, respectively). Similar examination of age at first exposure to cow's milk–based substitutes showed no significant differences between the curves of IDDM subjects and control subjects in either blacks or whites (data not shown).

After adjusting for maternal age at birth, initial exposure to any breast-milk substitute by 3 mo of age increased

Table 2—*Timing of first exposures in infant diet in diabetic/nondiabetic pairs matched on birth order, birth year, and race by breast milk substitute*

Infant milk	Pairs (N)	Age (wk) IDDM	Age (wk) Control	P
Black				
Cow's milk–based substitute	45	3.9 ± 8.5	8.5 ± 14.3	0.07
Any substitute	54	5.1 ± 10.9	11.9 ± 18.2	0.02
White				
Cow's milk–based substitute	132	5.3 ± 9.4	6.6 ± 11.0	0.35
Any substitute	151	5.5 ± 11.0	7.1 ± 12.2	0.18

Values are means ± SD.
N pairs in which both IDDM subjects and control subjects had been exposed initially to breast milk substitute.
P value based on Wilcoxon's matched-pairs test.

IDDM risk three-fold in blacks (Table 3). The association between initial exposure to cow's milk–based substitutes and IDDM in blacks was not significant. Whites showed no association between exposure to any substitutes or cow's milk–based substitutes and IDDM status. To determine whether the effect of age at exposure to breast milk substitutes was independent of breast-feeding, breast-feeding status was added to the model. Although the addition of breast-feeding status statistically weakened the association between exposure to any substitutes before 3 mo of age and IDDM in blacks, the OR remained strong (Table 3).

CONCLUSIONS — Although the risk of IDDM associated with early introduction of breast milk substitutes has been reported (8), a specific examination of cow's milk–based substitutes has not been conducted. We investigated exposures to breast milk substitutes containing cow's milk protein, which has been considered diabetogenic (9–11), and exposures to all breast milk substitutes. Our analyses suggest that the observed protective effect of breast-feeding on the risk of IDDM may be related to differences in the age at first exposure to any breast milk substitutes in blacks but not in whites in this study cohort. Specific examination of cow's milk–based substitutes demonstrated a similar but marginally significant association in blacks.

The relationship between IDDM and age at exposure to breast milk substitutes was observed in blacks but not in whites. The power to detect an OR of 2 was 62 and 90% in blacks and whites, respectively, given the sample size and the matched design of the study. Therefore, insufficient power would not explain why the diet associations observed in the black cohort were not found in the white cohort. It is possible that low-risk populations have a smaller genetic influence (24) and may be more strongly affected by environmental factors. Alternatively, other differences within blacks and whites (i.e., maternal recall, unmeasured exposures in infancy) may explain these discrepancies.

The use of sibling control subjects, which allows the researcher to gather twice the data through one contact person (i.e., the mother of the diabetic subject and nondiabetic sibling) is a study design that is gaining popularity in epidemiological research. However, because infant diet exposures of the diabetic subject and his/her siblings are not independent, matching a case to his own sibling violates an assumption of the case-control study design (21). Therefore in this study, the diabetic subject was matched on birth order, birth year, and race to an unrelated nondiabetic in-

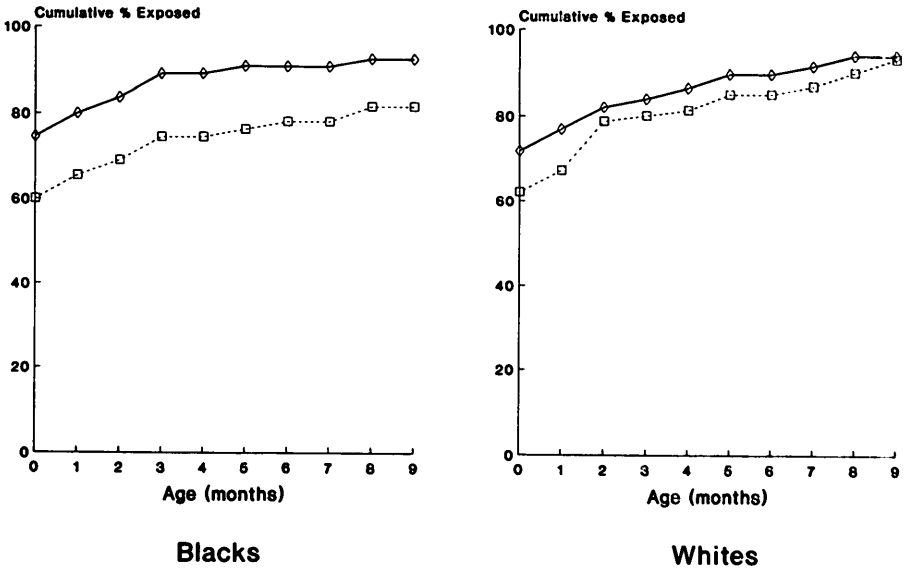

Figure 1—*Cumulative percentage of exposure to breast milk substitutes (-◇-, diabetic; -□-, nondiabetic control) in blacks (54 diabetic and 54 control subjects) and whites (151 diabetic and 151 control subjects). Curves are significantly different by case-control status in blacks (P = 0.04, Wilcoxon's test) but not in whites (P = 0.33).*

Table 3—*Odds ratio (OR) and 95% confidence interval (CI) for risk of insulin-dependent diabetes mellitus by exposure to breast milk substitutes by 3 mo of age*

	Any substitute		Cow's milk–based substitute	
	OR	95% CI	OR	95% CI
Blacks	3.3 (2.9)	1.1–10.0 (0.9–9.5)	3.0 (3.3)	0.8–11.1 (0.7–16.0)
Whites	1.1 (0.5)	0.6–2.0 (0.2–1.4)	1.2 (0.6)	0.6–2.2 (0.2–1.5)

Values are adjusted for differences in maternal age at birth, which was dichotomized (≥35 yr, <35 yr). Values in parentheses are adjusted for both breast-feeding status and maternal age at birth.

dividual from another family in the study cohort. Although there is a potential for overmatching with this design, associations were seen in the data. There is also a potential for misclassification when siblings of diabetic children are used as control subjects because they are at increased risk of IDDM with an incidence of 162/100,000 PY (25). However, most of our control subjects had passed the high-risk period and the remainder represented only 576 PY at risk for developing IDDM before the age of 20 yr; therefore the likelihood of misclassification in this cohort was minimal. A potential confounder that had not been addressed in previous studies is birth order. Individuals with a higher birth order in a family are at greater risk for IDDM (13–15) and are less likely to be breast-fed (12) than siblings born before them, which could result in spurious findings if the effect of birth order is not removed. Socioeconomic status was not directly addressed in this study. However, by controlling for race and maternal age at birth, we indirectly accounted for the effect of socioeconomic status on infant nutrition practices.

Even though matching on birth year and birth order was done primarily to control for strong temporal and familial trends in infant diet practices, it also allowed us to control for differences in maternal recall of infant diet, which is likely to vary by birth year and birth order. Comparison of the survey data with that of the medical records showed that the infant diet histories were relatively well recalled by the mothers. Accurate maternal recall of breast-feeding has been reported in the literature (26–28); however, the duration of breast-feeding may be less reliable than the prevalence (29). As demonstrated by Vobecky et al. (30), it may be more difficult to detect a significant relationship with retrospective data because of the increased variability of the data, and that a significant finding with retrospective data may lead to a conservative rather than an invalid conclusion. Recall bias could not be evaluated in this study because only IDDM subject records were obtained. However, it is unlikely that there was a recall bias in the data because mothers of diabetic children are not routinely asked to recall the infant diet of their diabetic child and are unlikely to search their child's infancy for possible causes of diabetes without prompting.

Breast milk protects the newborn infant against infections through specific secretory IgA antibodies, specifically sensitized cytotoxic T- and B-lymphocytes and nonspecific defense factors (e.g., complement components). However, the increasing trend in breast-feeding in the past decade has not resulted in the decline in IDDM incidence we would expect if breast-feeding alone were protective. Animal and ecological studies have indicated a possible relationship between cow's milk protein sources and IDDM (9,10,31). In the BB rat, the diabetogenicity of certain dietary protein sources may be dependent on an early and continuous exposure (11). Exposure to the diabetogenic agent may be most detrimental in infancy when the intestine is relatively permeable, suggesting that the critical period for exposure occurs early in life when the gut barrier may be less efficient in protecting against foreign antigens.

By investigating the environmental etiology of IDDM, we are ultimately seeking a potential mechanism for the prevention of the disease. The prevention of IDDM through a relatively benign dietary intervention such as the elimination or reduction of the dietary load of an offending antigen has major implications for public health.

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