

Proportion of Type 2 Diabetes Cases Resulting From Impaired Fetal Growth

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OBJECTIVE — During the past decade, several researchers have demonstrated a higher risk of type 2 diabetes in relation to lower birth weight. This theory, referred to as the “thrifty phenotype” hypothesis, postulates that impaired fetal growth predisposes individuals to the development of diabetes and other metabolic abnormalities. This article examines the importance of fetal growth in the etiology of diabetes by estimating the proportion of diabetes cases associated with this exposure.

RESEARCH DESIGN AND METHODS — The importance of an exposure or its correlate as a potential cause of a disease can be assessed by estimating the proportion of cases that could be prevented if the exposure was eliminated from a defined population. This proportion is referred to as the population-attributable fraction (PAF). Published studies of the association between diabetes and birth weight were reviewed and selected for further analysis if data were presented that enabled PAF calculation. In addition, PAFs were calculated for higher birth weight cutoffs because researchers have postulated that the lowest birth weight category may not capture all cases of fetal growth retardation. Studies have shown that exposure classified in this broader manner can produce unbiased PAF estimates, even if many subjects are falsely classified as exposed.

RESULTS — PAFs for the lowest birth weight category ranged from 0.01 to 0.25. In this analysis, PAFs for diabetes did not exceed 0.35. In contrast, >50% of diabetes cases in the First National Health and Nutrition Examination Survey Epidemiologic Follow-Up Study were attributable to excess adiposity as reflected by a BMI of $\geq 26 \text{ kg/m}^2$ (PAF >0.50).

CONCLUSIONS — Impaired fetal growth or its correlates account for a minority (<0.50) of type 2 diabetes cases.

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Much interest has developed during the past decade regarding what is known as the “thrifty phenotype” hypothesis (1–3). This hypothesis proposes that type 2 diabetes and associated conditions such as hypertension and lipid abnormalities result from inadequate intrauterine conditions for optimal fetal growth. Several studies have demonstrated a higher risk of diabetes or impaired glucose tolerance in relation to lower birth weight, including 2 large cohort studies of U.S. health professionals that included

thousands of subjects (1,4–8). Critics of these reports focus on the highly selected populations included in several of these studies and the likely presence of residual confounding by correlates of birth weight (9,10). Other investigators have pointed out that low birth weight probably does not causally account for a substantial proportion of diabetes cases because only a relatively small proportion (<10%) of infants fall into this category (6).

Despite these factors, the originators of the thrifty phenotype hypothesis have down-

played an important role for genetic factors in the etiology of type 2 diabetes and recently concluded that “environmental, probably nutritional factors operating in early (fetal and possibly infant) life play a major causative role in NIDDM [type 2 diabetes]” (3). Regarding the issue of a low proportion of exposed diabetes cases, those researchers made the point that low birth weight inaccurately classifies the actual cause, which they postulated to be a currently unidentified “environmental condition” (3). This adverse condition may be present even at higher birth weight categories and may result in the failure of the fetus to reach its optimal birth weight. Hales et al. (3) gave as an example a baby weighing 7.0 lb exposed to an inadequate intrauterine environment who would, under conditions of optimal nutrition, have gone on to reach a birth weight of 9.0 lb (3). I will refer to this theory as fetal underdevelopment syndrome (FUS) in this article. Currently, no methods exist to measure whether suboptimal fetal development has occurred within normal birth weight categories, although the likelihood of this occurrence is probably very low in higher birth weight categories.

A method is available to assess the proportion of cases of a disease resulting from a given exposure that is referred to as the population-attributable fraction (PAF). The PAF reflects the proportion of cases of a disease that could be prevented if the exposure was removed from a given population. Levin (11) first proposed this concept in 1953 and called it “attributable risk” (11), and it has since had other names, including “etiologic fraction” (12) and “population-attributable risk” (13), thus creating the potential for confusion because of the use of different terminology for the same concept. In this article, I assess the importance of both low birth weight and FUS as a cause of type 2 diabetes by calculating PAFs for this exposure in published studies that included the requisite data for these calculations.

RESEARCH DESIGN AND METHODS

Literature search

I searched Medline from 1991 through April 2000 for all articles written in the

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Abbreviations: FUS, fetal underdevelopment syndrome; MeSH, medical subject headings; PAF, population-attributable fraction; RR, relative risk.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

English language that contained the medical subject headings (MeSH) “diabetes mellitus (exploded),” “birth weight,” and “human.” I began the search in 1991 because an important article on the association between birth weight and diabetes risk in adult life first appeared at that time (1). This search yielded a total of 383 references, but most of these addressed issues related to immediate pregnancy outcomes in relation to type 1 and gestational diabetes versus the target issue of the effect of birth weight on the risk of diabetes development as an adult. Therefore, the search was restricted further by eliminating references using the Boolean operator “not” that contained the MeSH “diabetes, gestational,” “fetal macrosomia,” or “pregnancy outcome.” These restrictions yielded 123 potential articles for this analysis.

Potential articles had to satisfy the following eligibility criteria: assessment of diabetes as the outcome in adult life and presentation of original data in the publication on diabetes incidence in relation to birth weight that permitted calculation of the PAF. Only 5 studies fulfilled these criteria. Several studies examined glucose as a continuous measure in relation to birth weight (14,15), including 2 studies of prenatal exposure to famine (16,17), but an estimate of PAF cannot be made from this information. Other studies were not included because they considered a combined outcome (either impaired glucose tolerance or diabetes) (1,7) and therefore did not permit estimation of the PAF for the effect of birth weight on diabetes alone.

Analysis

The PAF is defined as the excess number of cases resulting from an exposure divided by the total number of cases in a defined population (13). This quantity can be calculated several different ways. If the cumulative incidence in the exposed and unexposed subjects is known along with the number of exposed subjects, then the following yields the excess number of cases resulting from the exposure or a correlate:

$$\text{Excess cases} = (\text{incidence}_{\text{exposed}} - \text{incidence}_{\text{unexposed}}) \times \text{number}_{\text{exposed}} \quad (1)$$

Dividing the excess cases by the total number of cases yields the PAF. For example, cumulative incidences of 0.2 in the exposed and 0.1 in the unexposed with 100 exposed and 900 unexposed subjects

result in a PAF of $[(0.2 - 0.1) \times 100] / (0.2 \times 100 + 0.1 \times 900) = 10/110 = 0.091$.

Algebraic manipulation of Eq. 1 results in a formula for the PAF that depends on the relative risk (RR) of the exposure and the proportion of exposed cases only.

$$\text{PAF} = \text{proportion of exposed cases} \times (\text{RR} - 1) / \text{RR} \quad (2)$$

Equation 2 demonstrates several general features of the association among the proportion of exposed cases, RR, and potential importance of the exposure as a cause of a disease in a population. As an example, consider the results of a case-control study of the association between lung cancer and cigarette smoking (18). Smoking was associated with an estimated RR of lung cancer of 52.8, and a high proportion of the 298 cases were exposed (0.983), which led to a PAF of $0.983 \times (51.8/52.8) = 0.964$. Therefore, ~96% of lung cancer cases would not have occurred during the same time period if no cigarette smoking had occurred in this study population. In general, the PAF increases as the proportion of exposed cases and the RR increase. High levels of the proportion of exposed cases or the RR do not ensure a high PAF. For example, if the RR associated with the exposure is 1.5, and all cases are exposed, then the PAF associated with this combination is 0.33. Similarly, a high RR does not ensure a high PAF. For example, an RR of 100 results in a PAF of only 0.099 when 0.10 of cases are exposed.

Although birth weight is directly measurable, FUS is not because a newborn's optimal birth weight cannot be known, only the actual birth weight can. In fact, conceiving of any method to measure optimal birth weight and failure to achieve it in humans in the near or distant future is difficult. FUS probably, however, occurs rarely or not at all in higher birth weight categories (e.g., >3,500 g or 7.7 lb). Therefore, capturing all cases of FUS may be possible if the birth weight cutoff is set appropriately high. This would undoubtedly lead to many unexposed subjects falsely included in the exposed category. To be specific, many infants with birth weights between 2,500 and 4,000 g will not have experienced FUS, yet they will be classified as positive for this exposure. This proposed classification scheme would lead to considerable bias in the estimation of RR of diabetes associated with this exposure. Interestingly, studies have shown that setting a wider range for

exposure (e.g., higher birth weight) in this manner leads to a more accurate estimate of the true PAF when considerable underascertainment of exposure occurs at a lower cutoff value, even though many truly unexposed subjects will be falsely classified as exposed in the process (19,20). I used this property of the PAF to estimate the potential importance of the FUS as a cause of type 2 diabetes, even though no direct measurement of this exposure is currently possible. This property is especially important because an overly narrow definition of exposure can underestimate the PAF. If a broad exposure definition does not capture all exposed subjects for a factor associated with higher disease risk, then the PAF will be biased toward the null value, although this effect will likely be small unless many truly exposed subjects are classified as unexposed.

I also used the distributive property of the PAF to calculate the PAF for the sum of multiple exposure categories (19). For example, if the diabetes PAF associated with a BMI >30 kg/m² is 0.3 and with a BMI 27–30 kg/m² is 0.2, then the PAF for a BMI ≥ 27 kg/m² is 0.5. Proof of these properties and further technical information regarding the PAF may be found elsewhere (13,19–22).

RESULTS

Type 2 diabetes PAF for low birth weight

Table 1 displays characteristics of the 5 eligible publications regarding birth weight and the incidence of diabetes in adult subjects in which data were presented that permitted PAF calculation. The table numbers in these publications from which data were extracted for PAF calculations are shown in Table 1. Cumulative incidences were calculated from the numerator and denominator data provided. No adjustment could be made for potential confounding factors (except as indicated below) because the results presented in these articles did not typically contain sufficiently stratified data to permit these calculations. In calculating the PAF for low birth weight, the lowest birth weight category presented in each article was compared with the remaining subjects. The crude (unadjusted) results of 2 studies demonstrated a higher diabetes risk in the higher birth weight category, which possibly reflects the presence of gestational diabetes (6,8). For these 2 studies, results were presented with the higher birth weight categories excluded so as not to underestimate the effect of low birth weight on diabetes

Table 1—Characteristics of eligible studies for the estimation of type 2 diabetes PAF in relation to low birth weight compared with all other birth weights except as indicated

Study (criteria for diabetes diagnosis)	Subjects (n)	Age (years)	Lowest birth weight category definition	Proportion of exposed cases	RR	PAF
Pima Indians, Table 1 (2-h glucose ≥ 11.1 mmol/l) (6)	1,147	20–39	<2,500 g	0.06	1.51	0.02*
Pima Indians, Table 1 (fasting glucose ≥ 7.8 mmol/l) (6)	1,147	20–39	<2,500 g	0.05	1.37	0.01*
Nurses' Health Study, Tables 2 and 3 (self-reported physician diagnosis) (8)	60,244	57.1–60.9‡	<5 lb	0.10	1.94	0.05† 0.06§
Swedish men, Table 4 (National Diabetes Data Group) (5)	1,093	60‡	<3,250 g	0.34	1.99	0.17
Swedish Men, Table 3 (World Health Organization criteria) (26)	2,294	35–56	$\leq 3,000$ g	0.34	3.52	0.25
Male Health Professionals Study, Table 3 (self-reported physician diagnosis) (4)	22,312	48–84	<5.5 lb	0.05	1.77	0.04

*Highest birth weight category ($\geq 4,500$ g) excluded from this comparison as explained in the text; †higher birth weight categories (>8.5 lb) excluded from this comparison as explained in the text; ‡mean; §subjects with a parental history of diabetes excluded.

risk. To exclude the possibility of confounding by parental diabetes history, 1 study presented data among subjects with no parental diabetes history (8). The PAF was calculated in this case using all birth weight categories. Definitions of diabetes varied by study (Table 1). The upper cutoff value and units of presentation for the lowest birth weight category also differed by study (Table 1).

By using the RR and the proportion of exposed cases (Table 1) substituted into Eq. 2, the PAFs for the studies that assessed the relationship between low birth weight and diabetes ranged from 0.01 to 0.25. Lower PAFs characterize the association between low birth weight and either a fasting glucose level of ≥ 7.8 mmol/l or a 2-h glucose level of ≥ 11.1 mmol/l among Pima Indians (0.01–0.02). An analysis of the Nurses' Health Study data that excluded any subjects with a parental history of diabetes

yielded a diabetes PAF estimate of 0.06 for the lowest birth weight category (8).

Type 2 diabetes PAF for FUS

To assess the proportion of type 2 diabetes cases attributable to the FUS, birth weight categories other than the highest in each study were considered to be exposed. This definition of exposure would be very likely to capture all or nearly all cases of fetal underdevelopment. PAFs were recalculated over multiple birth weight categories, were combined using the distributive property, and were compared with the highest (referent) birth weight category (Table 2). In all but 1 case, a higher PAF was seen for the combination of multiple birth weight categories compared with the lowest birth weight category only (Table 2). PAFs ranged from 0.01 to 0.35. The data from the Nurses Health Study excluded subjects

with a parental history of diabetes to yield the largest estimate of the PAF. In an analysis that included subjects with and without a parental history of diabetes, the PAF estimates were much lower. The Pima Indian PAF associated with a fasting plasma glucose of ≥ 7.8 mmol/l for combined birth weight categories was less than the lowest birth weight category because the next-to-lowest category had a lower incidence of this outcome than the referent category. As in the analysis of the PAF associated with the lowest birth weight category only, the highest birth weight category ($\geq 4,500$ g) was excluded from the analysis of multiple categories among Pima Indians because of the higher diabetes risk associated with higher birth weight in this population (6).

CONCLUSIONS — Low birth weight or its correlates account for only a small to

Table 2—PAF of diabetes for individual and combined birth weight categories

Study population	PAF nonreferent categories combined	PAF for each birth weight category vs. the referent category				
		<2,500 g	2,500–3,499 g	3,500–4,499 g	>4,249 g	>10 lb
Pima Indians (6)	0.01	<2,500 g	2,500–3,499 g	3,500–4,499 g		
		0.02	–0.01	Referent		
2-h glucose ≥ 11.1	0.03	0.01	0.01	Referent		
		0.03	0.01	Referent		
Nurses Health Study, no parental diabetes history (8)	0.27	<5 lb	5.0–5.5 lb	5.6–7.0 lb	7.1–10 lb	>10 lb
		0.06	0.04	0.05	0.12	Referent
Swedish men (5)	0.28	<3,250 g	3,250–3,749 g	3,750–4,249 g	>4,249 g	
		0.17	0.04	0.06	Referent	
Swedish men (26)	0.35	$\leq 3,000$ g	3,001–3,600 g	>3,600 g		
		0.25	0.10	Referent		
Male Health Professionals Study (4)	0.07	<5.5 lb	5.5–6.9 lb	7.0–8.4 lb	8.5–9.9 lb	≥ 10 lb
		0.04	0.03	0.00	0.00	Referent

Table 3—PAF for separate and combined categories of BMI in relation to risk of diabetes over 20 years of follow-up (white subjects only) in the First National Health and Nutrition Examination Survey Epidemiologic Follow-Up Study (1971–1992)

	BMI (kg/m ²)				
	≥22	≥30	26 to <30	22 to <26	<22
Women					
<i>n</i>		891	1,064	2,000	1,838
Incidence		0.26	0.12	0.06	0.02
PAF	0.82	0.43	0.23	0.16	Referent
Men					
<i>n</i>		482	1,359	1,535	683
Incidence		0.23	0.12	0.05	0.03
PAF	0.64	0.25	0.31	0.08	Referent

Adapted from Resnick et al. (24).

moderate ($\leq 35\%$) proportion of diabetes cases in these studies. Although this analysis could not adjust for most confounding factors, proper consideration of these factors would not likely result in an appreciable change to these conclusions. Two analyses that considered potential confounding factors demonstrated only small differences between crude and adjusted RR estimates (4,8). Therefore, the excess cases associated with low birth weight adjusted for confounding factors would be similar to the unadjusted estimates. A third analysis found an adjusted diabetes odds ratio of 3.8 when comparing birth weight <2,500 g with birth weights ranging from 2,500 to 4,499 g (6). This odds ratio likely considerably overestimates the true RR because diabetes occurred with greater-than-rare frequency in this Pima Indian population (23). Even assuming that the RR for this exposure is 3.8, to account for $\geq 50\%$ of diabetes cases among Pima Indians, >67% of them would have to have been exposed to low birth weight, which is ~ 17 times the reported level of exposure. The issue of whether a higher risk of diabetes associated with low birth weight in a particular subgroup would lead to a higher PAF must also be considered. If a high-risk subgroup is defined by the presence of 2 risk factors, then this will not necessarily lead to a larger PAF for the subgroup because the increase in RR would be offset by the smaller proportion of cases in the general population exposed to both factors than to either factor alone (Eq. 2).

These results also confirm that FUS or its causal correlates most likely account for no more than about one-third of the cases of type 2 diabetes in these populations. Because 2 large studies of health professionals included clinically diagnosed cases

of diabetes, this likely resulted in underascertainment of diabetes incidence. If clinically occult type 2 diabetes is more likely to be related to low birth weight than the manifest version of the disorder, then these analyses may have underestimated the RR and also the PAF of diabetes resulting from low birth weight.

In contrast, consider the PAF associated with higher BMI among participants in the First National Health and Nutrition Examination Survey Epidemiologic Follow-up Study (24). The number of subjects in different BMI categories and the incidence of diabetes during 20 years of follow-up are shown in Table 3. In assessing the association between adiposity as the exposure and diabetes risk, BMI serves as an imperfect measure of adiposity (25). Therefore, the same approach taken in the estimation of the effect of FUS on the proportion of diabetes cases resulting from this exposure can be used to estimate the effect of adiposity on this outcome by setting lower thresholds for adiposity based on measured BMI. The highest BMI category (≥ 30 kg/m²) has associated diabetes PAFs of 0.43 for women and 0.25 for men. The PAFs for overweight were 0.66 for women and 0.56 for men (BMI ≥ 30 kg/m² and 26–29 kg/m²). Setting a very low threshold for adiposity (BMI ≥ 22 kg/m²) will probably capture nearly all cases of excess adiposity. With this definition, the adiposity PAF equals 0.82 in women and 0.64 in men. Compared with adiposity, the FUS or its correlate appears far less important as a cause of diabetes from a population perspective.

As in most areas of etiological research, imperfect data exist on which to make decisions regarding the validity and relative importance of risk factors for type 2 dia-

betes. This analysis of existing data on low birth weight and the speculative FUS indicates that both conditions play a less important role than adiposity in the etiology of type 2 diabetes. In the former case, because low birth weight has usually occurred in a minority of diabetes cases (<10%), accounting for most (>50%) diabetes cases, even if the associated RR equaled infinity, would be impossible. In the latter case, a broad definition of exposure was used that was highly likely to have captured all cases of FUS, with the result being that most cases of diabetes could not have been prevented if FUS had been eliminated from the populations under study. This analysis argues that fetal events reflected by birth weight account for a minority (<50%) of diabetes cases.

Because of variability in study design and the absence of statistical methodology, no summary estimate for PAF from these 5 studies is presented. Although the larger sample size of several of these studies would have contributed greater weight to a summary estimate, the larger studies used less valid methods for assessment of diabetes status (self-report). Even if a summary meta-analytic estimate of PAF was available for multiple studies, it would be of questionable validity given these differences in methodology across publications.

Although fetal growth retardation plays a less important role than adiposity in the development of diabetes, it may nonetheless have a substantial effect on the development of diabetes in a given population, although this effect may vary, as seen in this analysis.

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