

Relative Risk of Mortality Associated With Diabetes as a Function of Birth Weight

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OBJECTIVE — Birth weight is a risk factor for both diabetes and mortality. Diabetes is a risk factor for mortality. Whether the excess mortality observed for diabetes varies with birth weight is unclear.

RESEARCH DESIGN AND METHODS — Among all 2,508 Rochester, Minnesota, residents who first met research criteria for adult-onset diabetes in 1960–1995, 171 were born locally in-hospital after 1922 (i.e., birth weights available) as singleton, term infants. Each case subject and two age- and sex-matched nondiabetic control subjects (born locally, residing locally when the case subject met the criteria for diabetes) were followed through 31 December 2000 for vital status.

RESULTS — Of the diabetic case subjects, 16% (27 of 171) died vs. 7% (25 of 342) of control subjects ($P = 0.004$). The difference was less for normal-birth-weight (NBW) (2,948–<3,856 g) individuals (12% [12 of 102] vs. 8% [20 of 246], $P = 0.31$) than for abnormal-birth-weight individuals (low birth weight [LBW] 20% [8 of 39] vs. 2% [1 of 46], $P = 0.01$; high birth weight [HBW] 23% [7 of 30] vs. 8% [4 of 50], $P = 0.16$), as confirmed with age- and sex-adjusted Cox proportional hazards (diabetes-associated hazard ratio 1.4 [95% CI 0.69–2.90] for NBW vs. 4.8 [1.7–13.3] for abnormal birth weight, test for interaction $P = 0.056$). The observed diabetes deaths were greater than expected, based on mortality for the general population (27 vs. 13.3, $P < 0.001$), with 70% of excess deaths occurring among LBW (8 vs. 2.2, $P < 0.001$) and HBW (7 vs. 3.1, $P = 0.03$) individuals.

CONCLUSIONS — The excess mortality observed for diabetes appears disproportionately concentrated among abnormal-birth-weight individuals, thus identifying a subset of at-risk diabetic individuals and reinforcing the importance of NBW deliveries.

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D iabetes is a prevalent disease with serious adverse consequences. Several studies have reported an association between diabetes and abnormal birth weight (1–11), and abnormal birth weight has been implicated as a risk factor for mortality in studies of the general population (12–15). Whether birth weight is predictive of diabetes-associated mortality is unclear. The question is important because the risk of death for people with diabetes is approximately twofold that for

their nondiabetic peers (16). If abnormal birth weight is a risk factor, it could help identify high-risk diabetic individuals. Because birth weight is modifiable, the risk of death associated with diabetes could be reduced in the future. A rare opportunity to investigate the association between birth weight and diabetes-related mortality is afforded with access to the complete detailed medical records from birth until emigration or death of all individuals born in Rochester, Minnesota, since 1922.

RESEARCH DESIGN AND METHODS

— Rochester, Minnesota (2000 census population 85,806), is relatively geographically isolated and is home to one of the world's largest private medical centers, the Mayo Clinic. Thus, nearly all medical care received by local residents is delivered by very few providers. Since 1907, every Mayo Clinic patient has been assigned a unique identifier. All information (medical history, clinical assessments, laboratory/pathology/radiology results, correspondence, and birth/death certificates) from every contact (office/emergency department/nursing home visits and hospitalizations) is contained within a single dossier for each patient (17). The diagnoses assigned are coded and entered into continuously updated computer files. Under the auspices of the Rochester Epidemiology Project (REP), this medical record linkage was expanded to include Olmsted Medical Center, a second group practice, and the few local private practitioners in the area (17).

REP resources were used to construct the Rochester diabetes incidence cohort (18–21). The cohort includes all 2,508 individuals who first met the research criteria for diabetes at ≥ 20 years of age as a Rochester resident in 1960–1995. Diabetes status was determined after review of provider-linked medical records for all laboratory glucose values (available since 1930) from date of first contact with each REP provider until date of last contact. Criteria approximate National Diabetes Data Group (NDDG) recommendations, i.e., two consecutive fasting glucose levels ≥ 7.8 mmol/l (≥ 140 mg/dl) or both 1- and 2-h levels obtained during a standard oral glucose tolerance test ≥ 11.1 mmol/l (≥ 200 mg/dl) (22). Adjustments were made for changes in laboratory methods over time (23). Individuals using antidiabetic agents for ≥ 2 weeks also qualified as case subjects. The record review was limited to candidate case subjects, i.e., residents assigned any diagnosis suggestive of diabetes (e.g., elevated blood glucose concentration, impaired glucose tolerance, rule-out diabetes, or diabetic nephropathy). We previously demonstrated that ascertainment of diabetes with this passive surveillance approach is relatively complete. A study of all Rochester dece-

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Abbreviations: HBW, high birth weight; LBW, low birth weight; NBW, normal birth weight; NDDG, National Diabetes Data Group; REP, Rochester Epidemiology Project.

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

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dents aged ≥ 45 years in 1970–1995 revealed that the median duration of medical record from date first seen by a REP provider until death was 43 years (interquartile range 24–58 years); $>25\%$ of all decedents had a diagnosis that qualified them as a candidate case subject for the REP diabetes cohort (24). In each year, the proportion of local residents aged ≥ 30 years with at least one blood glucose measurement averages 37% for men and 44% for women (19).

This study was approved by the Mayo Clinic and Olmsted Medical Center Institutional Review Boards. In accordance with a Minnesota State privacy statute, (25) 45 (2%) members of the REP diabetes incidence cohort who declined authorization to use their medical records in research after 1997 were excluded from review.

The study was limited to incident diabetic case subjects for whom we had access to recorded birth weights (i.e., those born locally in-hospital after 1922). Preterm and multiple births were excluded. Birth weight, birth number, and gestational age were obtained from hospital birth books, Olmsted County birth certificates, and mothers' medical records. The study was limited to possible type 2 diabetes, i.e., individuals who met all of the following criteria were excluded: BMI (weight in kilograms divided by the square of height in meters) <30 as of the date criteria were met, receiving insulin within 1 year of that date, and receiving insulin when last seen.

The vast majority of members of the REP diabetes incidence cohort were born before 1922 and/or moved into the area after birth. Only 171 were born locally in-hospital after 1922 (i.e., for whom we had access to recorded weights at birth) as singleton, term infants and met the criteria for type 2 diabetes. For each of the 171 case subjects, we identified two nondiabetic Rochester residents of the same sex and birth year who met the same birth criteria as case subjects and who were seen by a REP provider in the year (± 2) that the case subject first met the criteria for diabetes.

Determination of vital status

Vital status as of 31 December 2000 and date of last follow-up were determined after review of medical records, State of Minnesota death tapes, the Social Security Death Index, and telephone and mail follow-up. Confirmation that individuals were alive as of 31 December 2000 or had

died (with date of death) before that date was available for 95% of control subjects and 99% of case subjects. Follow-up for remaining individuals was censored at the date vital status was last known.

Statistical analyses

Analyses were conducted using SAS version 8.02 (SAS Institute, Cary, NC), with statistical significance set at $P < 0.05$. Univariate descriptive statistics were used to compare case subjects and control subjects for baseline characteristics. In keeping with our previous publication (11), low birth weight (LBW) was defined as 2,948 g (<6.5 lb), normal birth weight (NBW) as 2,948– $<3,856$ g (6.5– <8.5 lb), and high birth weight (HBW) as $\geq 3,856$ g (≥ 8.5 lb). Within each category, differences in mortality between case subjects and control subjects were tested using the Kaplan-Meier product limit method and a two-sample log-rank test. Cox proportional hazards modeling was used to assess the contribution of diabetes to mortality, for each birth weight category separately (adjusting for sex, age, and birth year) and for all categories combined (adjusting for sex, age, birth year, and birth weight category). To assess whether the effect of diabetes differed as a function of birth weight, we tested for an interaction between case status and birth weight category, considered both as a three-level (low versus normal and high versus normal) and a two-level (normal versus abnormal) variable.

We additionally tested whether diabetes-associated mortality differed as a function of birth weight category by comparing observed mortality for diabetic case subjects with that expected, based on sex/age/calendar year stratified rates for the white West North Central U.S. population. This approach validated the representativeness of the control cohort and provided an independent assessment of the role of birth weight in diabetes-associated mortality. Within each category, differences between observed and expected mortality were tested using the Kaplan-Meier product limit method and a one-sample log-rank test. We then examined the effect of birth weight on the relative risk of mortality after adjusting for age, sex, and birth year. Relative mortality was analyzed by creating a new time variable that took into account the actuarial life-table survival distribution for each individual. This variable was defined as $-\log[S(T)]$, where $S(T)$ is the actuarial life-table probability of survival for each indi-

vidual up until his or her date of last follow-up. Under the assumption that each individual's survival follows the expected actuarial life-table distributions, this variable has a negative exponential distribution with parameter 1.0, independent of the individual's age, sex, and year he or she met the criteria for diabetes. The departure of the individual's actual survival from expected is reflected in the departure of this "survival time" from the predicted negative exponential distribution. This transformed censored survival time was then subjected to ordinary Cox regression to determine how "relative survival" was related to birth weight, as well as to age, sex, and birth year. Within this framework, we analyzed birth weight as a continuous variable, considered both as a linear and a nonlinear function. The latter was based on several different models: 1) a quadratic function (two parameters), 2) a wedge-shaped function (linear, then flat), with the initial slope and change point estimated from the data (two parameters), and 3) a wedge-shaped function with two lines, neither of which was required to be flat (three parameters). In each case, it was the overall model χ^2 that was assessed, relative to its degrees of freedom.

RESULTS— Comparison of characteristics among diabetic case subjects and nondiabetic control subjects (Table 1) revealed significant differences in the distribution of birth weight categories, with a greater proportion of both LBW and HBW case subjects compared with control subjects.

Mortality among diabetic case subjects versus nondiabetic control subjects

Median follow-up from baseline through 31 December 2000 was 10.0 years (11.6 ± 7.4 years [mean \pm SD]; range 0–38 years). The proportion of deaths among case subjects was more than twice that for control subjects (27 of 171 [16%] vs. 25 of 342 [7%], two-sample log-rank $P = 0.004$). The difference was less apparent for NBW individuals (12 of 102 [12%] vs. 20 of 246 [8%], $P = 0.31$) than for HBW individuals (7 of 30 [23%] vs. 4 of 50 [8%], $P = 0.16$) and LBW individuals (8 of 39 [20%] vs. 1 of 46 [2%], $P = 0.01$). In models adjusted for age, sex, and birth year, the diabetes-associated hazard of death was 1.39 (95% CI 0.67–2.86, $P = 0.38$) for NBW individuals,

Table 1—Characteristics of term singleton births born locally in 1922–1971 with birth weights available who did (case subjects) and did not (control subjects) first meet research criteria for type 2 diabetes as a Rochester, Minnesota, resident in 1960–1995

	Case patients	Control subjects	P value
n	171	342	
Age (years)*	45 ± 11 (20–72)	45 ± 11 (19–72)	0.99
Men	90 (53)	180 (53)	1.00
Birth weight (lb)	7.42 ± 1.07	7.49 ± 0.95	0.47
Distribution by category			0.01
<6.5	39 (23)	46 (13)	
6.5–8.49	102 (60)	246 (72)	
≥8.5	30 (18)	50 (15)	

Data are means ± SD (range) or n (%). *As of the date the case subject first met research criteria for diabetes and the same date (±2 years) for the age- and sex-matched control subjects.

2.21 (0.61–8.05, $P = 0.23$) for HBW individuals, and 10.7 (1.32–87.1, $P = 0.03$) for LBW individuals. There was only one death among LBW control subjects.

When all 513 individuals were considered together, the adjusted diabetes-associated hazard of death was 2.19 (Table 2). Tests for interaction (Table 2) revealed that the effect of diabetes on mortality was not significantly increased for HBW versus NBW individuals but was borderline significantly increased for LBW versus NBW individuals.

Observed versus expected mortality

Whether the high diabetes-associated mortality among LBW individuals reflected “healthy” control subjects was investigated by comparing observed

mortality for diabetic case subjects with that expected for the white West North Central U.S. population of similar age, sex, and calendar year distribution. The 27 observed deaths among diabetic case subjects were twice the 13.3 expected (one-sample log-rank $P < 0.001$). When stratified by birth weight category (Fig. 1), the observed number of deaths was significantly greater than that expected among both HBW (7 vs. 3.1, $P = 0.03$) and LBW (8 vs. 2.2, $P < 0.001$) case subjects. For NBW diabetic case subjects, however, the observed number of deaths was not significantly different from expected (12 vs. 7.9, $P = 0.14$), especially over the first 2 decades of follow-up. The 40% of case subjects who were LBW or HBW accounted for 70% of excess (observed minus expected) deaths.

Table 2—Proportional hazards models for mortality through 2000 among term singleton births born locally in 1922–1971 who did (case subjects) and did not (control subjects) meet research criteria for type 2 diabetes as a Rochester resident in 1960–1995

Variable	β Coefficient	SE	HR (95% CI)	P value
Main effects				
Age (10 years)	0.589	0.241	1.78 (1.31–2.26)	0.02
Male sex (yes)	0.539	0.311	1.72 (0.93–3.15)	0.08
Birth year	−0.020	0.026	0.98 (0.93–1.03)	0.44
Diabetes (yes)	0.786	0.280	2.19 (1.26–3.79)	0.005
LBW vs. NBW	0.205	0.383	1.23 (0.58–2.60)	0.59
HBW vs. NBW	0.094	0.354	1.10 (0.55–2.19)	0.79
Test for interactions				
Age (10 years)	0.626	0.244	1.87 (1.39–2.34)	0.01
Male sex (yes)	0.517	0.312	1.68 (0.91–3.90)	0.10
Birth year	−0.019	0.026	0.98 (0.93–1.03)	0.48
Diabetes (yes)	0.341	0.368	—	0.35
LBW vs. NBW	−1.298	1.027	—	0.21
HBW vs. NBW	−0.249	0.552	—	0.65
LBW vs. NBW × diabetes	2.189	1.128	—	0.05
HBW vs. NBW × diabetes	0.703	0.732	—	0.34

Case patients $n = 171$; control subjects $n = 342$.

Whether these differences reflected fortuitous categorization was investigated by considering weight as a continuous variable. Observed versus expected mortality for diabetic case subjects is graphed in Fig. 2. When considered as a linear function, in models that adjusted for age, sex, and birth year, there was a tendency for each increasing pound to be associated with a decline in the relative risk of mortality associated with diabetes (hazard ratio [HR] 0.71 [95% CI 0.49–1.03], $P = 0.07$); the association was highly significant when case subjects who weighed $\geq 3,856$ g (8.5 lb) were excluded ($P = 0.008$). However, in the three analyses in which birth weight was considered as a nonlinear function (see RESEARCH DESIGN AND METHODS), the effect of weight on the relative risk of mortality associated with diabetes failed to reach statistical significance ($P = 0.06, 0.10, \text{ and } 0.15$, respectively).

CONCLUSIONS— Previous studies have found that abnormal-birth-weight infants are at increased risk of developing type 2 diabetes (1–11). However, because the majority of adults who develop diabetes are normal weight at birth, (2,5,10,11) the population-attributable risk of diabetes due to abnormal birth weight is probably small (26). By contrast, findings from the present study suggest that the population-attributable risk of diabetes-associated mortality due to abnormal birth weight may be large. Our finding that diabetic case subjects had an overall risk of death approximately twice that for nondiabetic control subjects is consistent with numerous observations (16). We additionally observed that the risk of death associated with diabetes varied as a function of birth weight. Although abnormal-birth-weight individuals accounted for a minority of diabetic case subjects, they accounted for the majority of excess deaths associated with diabetes.

There are few opportunities to investigate the association between birth weight and mortality in persons with adult-onset diabetes. REP resources afford access to the detailed medical records for a geographically defined population, including all local birth certificates and laboratory glucose values, for more than 7 decades. Identification of all individuals who were born locally and who met the criteria for adult-onset diabetes before emigration or death thus minimizes the potential for selective survival that is

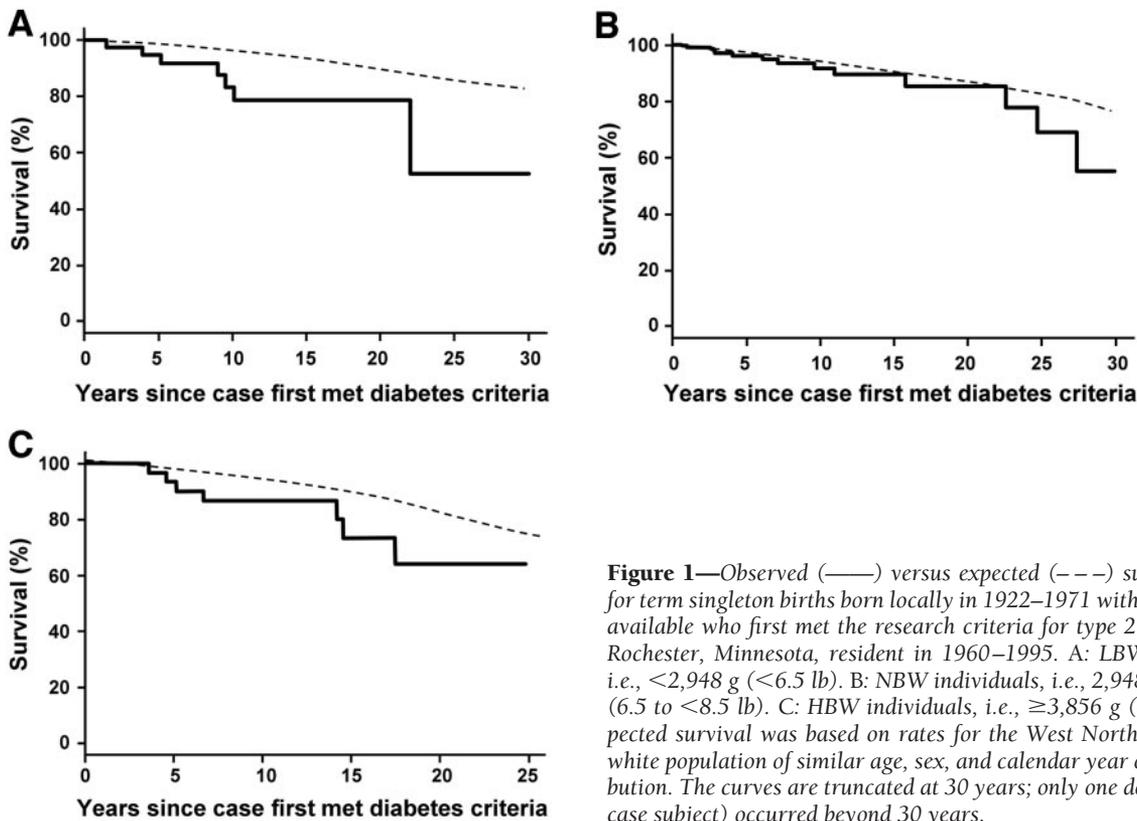


Figure 1—Observed (—) versus expected (---) survival curves for term singleton births born locally in 1922–1971 with birth weights available who first met the research criteria for type 2 diabetes as a Rochester, Minnesota, resident in 1960–1995. A: LBW individuals, i.e., <2,948 g (<6.5 lb). B: NBW individuals, i.e., 2,948 to <3,856 g (6.5 to <8.5 lb). C: HBW individuals, i.e., ≥3,856 g (≥8.5 lb). Expected survival was based on rates for the West North Central U.S. white population of similar age, sex, and calendar year of birth distribution. The curves are truncated at 30 years; only one death (an LBW case subject) occurred beyond 30 years.

problematic with studies identifying older individuals with and without prevalent diabetes. Selection bias is also minimized because REP diabetes incidence case subjects include all individuals irrespective of sex, diabetes treatment, and/or ability to participate. Additional opportunities afforded by the REP include 1) recorded birth weights (avoiding recall bias), 2) exclusion of preterm births, 3) relatively long follow-up, and 4) essentially complete information on vital status.

The few investigations of the association between birth weight and mortality have focused on the population generally; most found significant inverse associations between birth weight and the likelihood of death due to cardiovascular disease (12–15). The extent to which people with diabetes accounted for these observations is not known. Eriksson et al. (4) followed 1,319 Swedish men from age 20 years in 1933 to age 85 years and found no effect of weight as recorded at

birth on cardiovascular disease mortality. This is consistent with our finding of no significant effect of birth weight on mortality when both diabetic case subjects and control subjects were included together in the model (Table 2). In contrast to our findings, however, Eriksson et al. (4) found no significant effect of birth weight on mortality for individuals either with or without diabetes. Eriksson et al. identified diabetes from 1933 forward using hospital discharge or death certificate diagnoses. The problems with identifying diabetes from death certificates are well recognized. The limitations decreased over time (24,27). REP studies reveal that the proportion of diabetic decedents with any mention of diabetes on their death certificate averaged 34% for 1945–1970 and 49% for 1970–1994 (20,24,28). The likelihood that any individual is hospitalized and the likelihood that diabetic individuals receive a clinical diagnosis of diabetes has also increased markedly since the 1930s (21,29). If, as our findings suggest, abnormal-birth-weight diabetic individuals are at increased risk of death, such individuals in the study by Eriksson et al. (4) would have died earlier in the course of follow-up and may have been differentially misclassified as nondiabetic.

The present study defined diabetes based on review of medical records (in-

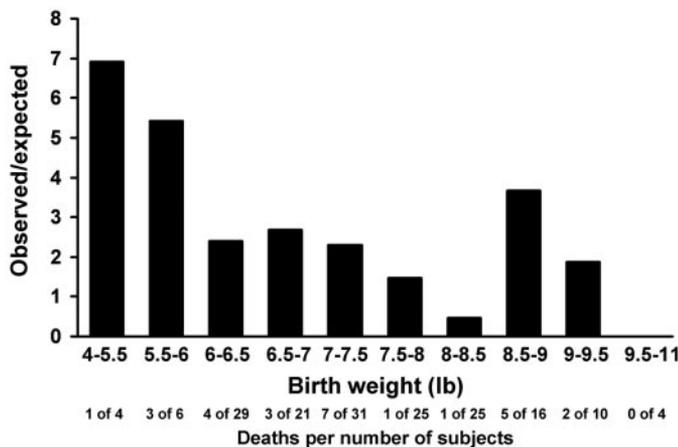


Figure 2—The ratio of observed to expected mortality as a function of birth weight (in 0.5-lb categories) for term singleton births born locally in 1922–1971 with birth weights available who first met the research criteria for type 2 diabetes as a Rochester, Minnesota, resident in 1960–1995 (n = 171).

cluding all glucose values); standardized glycemic criteria were applied throughout the study period. To minimize differences in detection over time, we used NDDG criteria rather than the more sensitive American Diabetes Association criteria (30). Thus, nondiabetic control subjects may have included individuals who never met NDDG criteria but who did meet American Diabetes Association criteria for diabetes. Control subjects may have also included individuals who would have met NDDG criteria if tested but who never received a diagnosis of diabetes or a diabetes-like condition while a local resident. The generalizability of our study findings to nonwhite individuals also cannot be assessed because the Rochester population during the period under investigation was >95% white.

The direction of our findings was consistent across all analyses. However, few comparisons reached conventional statistical significance of $P < 0.05$. Thus, caution is needed in interpreting the study findings, and the importance of replicating findings elsewhere is emphasized. If replicated, the findings have important implications for both research and clinical practice. They reinforce the need to decrease LBW deliveries and maintain tight control of glucose during pregnancy. They suggest that monitoring and treatment of diabetes complications may be especially important in the subgroup of diabetic patients whose weight at birth was outside the normal range.

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