Are All Growth-restricted Newborns Created Equal(ly)?

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Are All Growth-restricted Newborns Created Equal(ly)?

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ABSTRACT. *Background.* **Previous etiologic studies have defined intrauterine growth restriction (IUGR) based on a single cutoff.**

Objective. **To assess the relative importance of known etiologic determinants for different degrees (mild versus severe) and timing (preterm versus term) of fetal growth restriction.**

Design. **Hospital-based cohort study.**

Setting. **Tertiary-care university hospital.**

Participants. **Sixty-five thousand two hundred eighty inborn singleton infants without major congenital anomalies delivered between January 1, 1978 and March 31, 1996.**

Measurements. **Comparison of adjusted odds ratios (ORs) and 95% confidence intervals for mild IUGR (defined as birth weight 75% to <85% of the mean for gestational age, the latter cutoff equivalent to the 9.9th percentile for this cohort) and severe IUGR (<75% of mean, or 2.3rd percentile), after controlling for maternal age, education, marital status, and other potential determinants by means of multiple logistic regression.**

Results. **Maternal prepregnancy overweight (body mass index [BMI] >26.0–29.0 kg/m^2) and obesity (BMI >29.0 kg/m^2) had stronger protective effects against mild IUGR than against severe IUGR, but most of the determinants showed the opposite pattern. This was especially true for pathologic determinants; ORs (and 95% confidence intervals) for severe versus mild IUGR were 18.5 (14.5–23.8) vs 4.6 (3.6–5.8) for severe pregnancy-induced hypertension (PIH), 3.5 (2.2–5.5) vs 2.3 (1.5–3.4) for prepregnancy hypertension, and 3.4 (2.9–3.9) vs 2.2 (2.0–2.4) for smoking ≥11 cigarettes/day. Primiparity, short stature, prepregnancy BMI, maternal weight gain, and cigarette smoking had significantly larger effects on term IUGR, whereas the effect of severe PIH was more than twice as large for preterm IUGR (OR = 9.7 [7.3–13.0]) as for term IUGR (OR = 4.0 [3.0–5.3]).**

Conclusion. **Pathologic determinants of IUGR such as prepregnancy and PIH and cigarette smoking predispose to more severe fetal growth retardation, and PIH in particular seems to do so before 37 weeks. Growth-restricted newborns are not, therefore, all created equal(ly).** ***Pediatrics* 1999;103:599–602;** ***fetal growth, intrauterine growth restriction, low birth weight.***

ABBREVIATIONS. IUGR, intrauterine growth restriction; BMI, body mass index; LNMP, last normal menstrual period; BWR, birth weight ratio; PIH, pregnancy-induced hypertension.

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The etiologic determinants of intrauterine growth restriction (IUGR) are widely recognized.[1–4] In decreasing order of importance (based on their etiologic fractions, or population attributable risks) for a developed country in which ~25% of the women smoke during pregnancy, they include maternal cigarette smoking, low gestational weight gain, low prepregnancy body mass index (BMI), primiparity, preeclampsia, short stature, nonwhite racial origin, other genetic factors, and alcohol and drug use during pregnancy.

Previous etiologic studies have considered IUGR as homogeneous, based on a single cutoff point: usually, a birth weight <10th percentile for gestational age. Etiologic factors have not previously been examined in terms of the severity of IUGR. In other words, investigators have assumed that determinants have similar effects on severe IUGR as they have on milder degrees of fetal growth restriction. Similarly, although a few investigators have examined risk factors for preterm delivery accompanied by IUGR,[5–7] none (to our knowledge) has assessed whether the magnitude of their association with IUGR differs in infants born before versus at term.

We hypothesized that the effects of etiologic determinants would differ according to the severity, or degree, of IUGR. Specifically, we hypothesized that certain pathologic determinants such as prepregnancy hypertension and pregnancy-induced hypertension (PIH) would have greater effects on severe IUGR, whereas parity, maternal anthropometric factors, and cigarette smoking would have greater effects on mild IUGR. We also hypothesized that pathologic determinants would be more highly associated with preterm IUGR than with term IUGR.

METHODS

We used a hospital-based cohort design. The study setting is Montreal's Royal Victoria Hospital, a tertiary-care university hospital that serves a socioeconomically and ethnically diverse population. For several decades, this hospital has had a computerized obstetric and neonatal database that is both clinically rich and of high quality.[8] The study sample consists of 65 280 inborn singleton infants without major congenital anomalies delivered between the 1st of January 1978 and the 31st of March 1996. Gestational age was based on the first day of the last normal menstrual period (LNMP) if confirmed (±7 days) by early ultrasound or in the absence of an ultrasound recorded in the database. In the absence of a known LNMP, or when the ultrasound estimate differed more than 7 days from the LNMP estimate, the ultrasound estimate was used.

We defined the severity of IUGR based on the birth weight ratio (BWR), which is the ratio of the observed birth weight in a given infant to the hospital population's sex-specific mean birth weight for that infant's gestational age.[6] As in previous studies,[6,9] we defined BWR ≥0.85 as no IUGR, BWR ≥0.75 but <0.85 as mild

IUGR, and BWR <0.75 as severe IUGR. The two IUGR cutoffs correspond to the 9.9th and 2.3rd percentiles, respectively, of our study sample.

We studied potential sociodemographic, anthropometric, and pathologic determinants. Sociodemographic determinants included maternal age, education, marital status, and parity. Women who were legally single, widowed, or divorced were classified as unmarried. Anthropometric determinants comprised maternal height, prepregnancy BMI, and rate of net (ie, after subtracting the infant's birth weight) maternal weight gain. Analyses of prepregnancy BMI and of maternal weight gain were based on categories established by the Institute of Medicine.[10] The pathologic determinants we studied included prepregnancy hypertension, PIH, prepregnancy or gestational diabetes, and cigarette smoking. PIH was considered severe if the term "severe preeclampsia" was specifically mentioned on the discharge sheet completed by the attending obstetrician or in the presence of documented eclampsia. Data on smoking, education, and marital status were obtained by maternal self-report. Alcohol and illicit drug use were not considered in our analysis, because mothers were not routinely questioned about such use during the early years of study and because of extremely low reported levels of use thereafter.[11,12]

Bivariate associations were sought between these potential determinants and mild and severe IUGR, and between term (≥37 completed weeks) and preterm (<37 completed weeks) IUGR. Multiple logistic regression analyses were used to adjust for all the potential determinants listed above. In regression analyses for the associations of potential determinants with mild IUGR, we excluded severely growth-retarded infants. In regression analyses for severe IUGR, we excluded mildly growth-retarded infants. Analyses for term IUGR were restricted to births ≥37 weeks, whereas analyses for preterm IUGR were restricted to births <37 weeks. A determinant was considered to have a different magnitude of effect on severe than on mild IUGR if the point estimate of the adjusted odds ratio for severe IUGR lay outside the 95% confidence interval for the adjusted odds ratio for mild IUGR. Inferences about the magnitude of effect on preterm versus term IUGR were based on the same criterion.

To assess the impact of missing values on these adjusted estimates, we examined the results of three alternative logistic regression models: 1) inclusion of an indicator for unknown for those potential determinants with a substantial proportion of missing values; 2) exclusion of potential determinants with a substantial proportion of missing values (variable-wise deletion); and 3) exclusion of those cases with missing values on any potential determinants (case-wise deletion). The results of the first model were very close to those of the second, indicating little confounding of the estimates by the additional variables in the first model. The results of the third model differed slightly from those of the first two, however, suggesting a small degree of selection bias introduced by the case-wise deletion strategy. The results presented are therefore those from the first model, ie, including an indicator for unknown.

All analyses were conducted with SAS-PC (SAS Institute, Inc, Cary, NC).

RESULTS

Table 1 describes the study cohort in terms of the potential determinants under study. As shown in the last column of Table 1, values were missing for a substantial fraction of the study cohort (total n = 65 280) for maternal education, height, prepregnancy BMI, and net rate of gestational weight gain. Table 2 shows the crude (bivariate) association between each of the determinants and mild IUGR, severe IUGR, term IUGR, and preterm IUGR. Table 3 lists the results of multiple logistic regression analyses for mild and severe IUGR, and Table 4 the corresponding results for term and preterm IUGR.

Prepregnancy overweight (defined as BMI >26.0 but ≤29.0 kg/m^2) and obesity (BMI >29.0 kg/m^2) had stronger protective effects against mild IUGR than against severe IUGR. These were the only determinants studied that showed this pattern.

TABLE 1. Description of Study Cohort (Total n = 65 280)

Variable	Percent	n
Maternal age (y)		65 247
<20	2.3	
20–34	83.1	
≥35	14.6	
Unmarried	15.0	65 168
Maternal education (years completed)		45 700
≤10	11.4	
11–12	33.7	
13–15	26.2	
≥16	28.6	
Primiparity	47.1	65 255
Height <157.5 cm	28.7	41 495
Body mass index (kg/m^2)		37 164
<19.8	24.7	
19.8–26.0	61.4	
>26.0–29.0	7.4	
>29.0	6.5	
Net weight gain ≤0.17 kg/wk	17.7	36 913
Prepregnancy hypertension	0.3	64 538
Pregnancy-induced hypertension		65 280
None	91.0	
Mild	8.0	
Severe	0.9	
Diabetes (prepregnancy or gestational)	3.2	65 280
Smoking (cigarettes/day)		62 773
0	80.4	
1–10	8.2	
≥11	11.4	

The higher crude IUGR rate observed in teenage mothers (Table 2) was confounded by other risk factors, as shown by the slight (albeit nonsignificant) reduction in adjusted risk (Table 3) for both levels of IUGR relative to the reference group of women between the ages of 20 and 34 years. At the other end of the age spectrum, mothers 35 years or older had a one-third increase in adjusted risk of severe IUGR but only a trivial increase for mild IUGR. (We found no substantial interaction between the effects of age and parity on either level of IUGR.) Maternal educational attainment of <16 years and unmarried status were also associated with a substantial increase in severe IUGR but little if any increase in mild IUGR.

Primiparity, maternal short stature (height <157.5 cm), prepregnancy BMI <19.8 kg/m^2, and net gestational weight gain ≤0.17 kg per week all had independent adverse effects of similar magnitude, increasing the risk of mild IUGR by 1.5-fold and the risk of severe IUGR by approximately twofold. All the so-called "pathologic" determinants we studied also had larger effects on severe than on mild IUGR. Prepregnancy hypertension was associated with a substantially higher risk of severe than of mild IUGR. The same was true for both mild and severe PIH, for which there was no overlap whatsoever in the 95% confidence intervals for mild and severe IUGR. We were surprised to find that smoking up to 10 cigarettes per day or ≥11 cigarettes per day was also associated with a higher risk for severe IUGR than for mild IUGR, but were not surprised by the obvious dose-response relation between level of smoking and both IUGR cutoffs.

In logistic regression analyses for term versus pre-

TABLE 2. Rates (%) of IUGR Types According to Potential Determinants

Potential Determinant	IUGR Type			
	Mild*	Severe*	Term†	Preterm‡
Maternal age (y)				
<20	10.3	4.0	13.4	19.3
20–34	7.7	2.2	9.1	17.5
≥35	7.2	2.3	8.4	18.7
Marital Status				
Married	7.3	2.0	8.6	17.0
Unmarried	9.7	3.8	12.3	19.9
Maternal education (years completed)				
≤10	9.0	2.5	10.9	16.4
11–12	7.5	2.2	9.0	16.2
13–15	6.5	2.0	7.8	16.4
≥16	6.2	1.3	7.0	15.5
Parity				
Multiparous	6.2	1.5	7.1	14.5
Primiparous	9.2	3.1	11.4	21.1
Height (cm)				
≥157.5	6.5	1.8	7.7	15.6
<157.5	10.2	3.0	12.4	21.5
Body mass index (kg/m^2)				
<19.8	11.0	3.1	13.5	20.9
19.8–26.0	6.8	1.7	7.9	16.1
>26.0–29.0	5.4	2.2	6.6	17.0
>29.0	5.2	2.2	6.3	16.9
Net weight gain (kg/wk)				
>0.17	6.9	1.8	8.1	16.4
≤0.17	9.4	3.0	11.7	19.2
Pregnancy hypertension				
Absent	7.6	2.2	9.1	16.8
Present	15.8	13.9	20.1	48.6
Pregnancy-induced hypertension				
None	7.4	1.9	8.9	13.6
Mild	8.1	4.5	10.5	30.0
Severe	23.1	23.1	30.1	59.7
Diabetes				
None	7.7	2.3	9.2	17.9
Pregnancy or gestational	6.7	2.1	7.5	14.6
Smoking (cigarettes/day)				
0	6.6	1.7	7.6	16.1
1–10	9.8	3.3	12.4	20.2
≥11	13.2	5.3	17.7	23.9

Abbreviation: IUGR, intrauterine growth restriction.
* Rates expressed as of % of all births.
† Rates expressed as % of all births ≥37 completed weeks.
‡ Rates expressed as % of all births <37 completed weeks.

TABLE 3. Logistic Regression Analyses for Mild and Severe IUGR*

Potential Determinant	OR (95% CI) for Mild IUGR	OR (95% CI) for Severe IUGR
Maternal age (y)		
<20	0.85 (0.70–1.04)	0.83 (0.60–1.13)
20–34 (reference)	1.00 —	1.00 —
≥35	1.10 (1.00–1.20)	1.36 (1.16–1.61)
Unmarried	1.08 (0.99–1.17)	1.34 (1.16–1.54)
Maternal education (years completed)		
≤10	1.29 (1.13–1.46)	1.45 (1.12–1.86)
11–12	1.08 (0.97–1.19)	1.32 (1.08–1.62)
13–15	1.00 (0.90–1.11)	1.39 (1.13–1.71)
≥16 (reference)	1.00 —	1.00 —
Unknown	1.31 (1.19–1.44)	1.97 (1.63–2.38)
Primiparity	1.61 (1.51–1.72)	2.05 (1.81–2.31)
Height (cm)		
≥157.5 (reference)	1.00 —	1.00 —
<157.5	1.73 (1.60–1.87)	1.90 (1.64–2.20)
Unknown	1.22 (1.07–1.41)	1.13 (0.89–1.44)
Body mass index kg/m^2)		
<19.8	1.69 (1.55–1.85)	1.92 (1.62–2.26)
19.8–26.0 (reference)	1.00 —	1.00 —
>26.0–29.0	0.70 (0.59–0.85)	1.01 (0.75–1.35)
>29.0	0.60 (0.49–0.74)	0.72 (0.52–1.01)
Unknown	1.03 (0.89–1.20)	1.28 (0.99–1.67)
Net weight gain (kg/wk)		
>0.17 (reference)	1.00 —	1.00 —
≤0.17	1.56 (1.42–1.73)	1.89 (1.58–2.26)
Unknown	1.27 (1.17–1.38)	1.48 (1.27–1.72)
Prepregnancy hypertension	2.24 (1.47–3.39)	3.45 (2.18–5.46)
Pregnancy-induced hypertension		
None (reference)	1.00 —	1.00 —
Mild	1.10 (0.97–1.23)	2.41 (2.04–2.85)
Severe	4.55 (3.58–5.77)	18.50 (14.43–23.71)
Diabetes (prepregnancy or gestational)	0.96 (0.79–1.15)	0.77 (0.54–1.11)
Smoking (cigarettes/day)		
0 (reference)	1.00 —	1.00 —
1–10	1.54 (1.39–1.70)	1.93 (1.61–2.31)
≥11	2.21 (2.03–2.40)	3.36 (2.92–3.86)

Abbreviations: IUGR, intrauterine growth restriction; CI, confidence interval; OR, odds ratio.
* Tabulated odds ratios are adjusted for all variables and categories shown in the table.

term IUGR (Table 4), unmarried women were at slightly increased risk of term IUGR but not of preterm IUGR. Primiparity, short stature, prepregnancy BMI, maternal weight gain, and cigarette smoking had significantly larger effects on term IUGR than on preterm IUGR. Conversely, the effects of mild and severe PIH were more than twice as large for preterm IUGR as for term IUGR.

DISCUSSION

Our study has several potentially important limitations. First, the study cohort is hospital-based, not population-based. Although the study hospital serves an ethnically and socioeconomically diverse population and postnatally transferred infants were excluded from the study cohort, antenatal referrals were not. We are aware of no selection factors that would alter the magnitude of the effects we studied, but unknown sources of selection bias cannot be dismissed. Second, values were missing in a substantial minority of the study sample for maternal education, height, prepregnancy weight, and weight gain. Although the results of our three alternative logistic regression models provide some assurance against substantial selection bias or confounding, a small degree of bias because of missing data cannot be ruled out.[13] Third, the anthropometric information, even when available, does not permit analysis of prepregnancy body composition, or of the composition and timing of weight gain during the pregnancy. A fourth limitation relates to our inability to quantify the effects of alcohol and drug use during pregnancy. Fifth, to the extent that maternal smoking was underreported, we may have underestimated the magnitude of its association with IUGR, although we do not believe such underreporting would differ according to severity or timing (term or preterm). Finally, as in Canada as a whole, our study hospital does not identify the racial origin of its mothers or infants, and thus we are unable to control for the

TABLE 4. Logistic Regression Analyses for Term and Preterm IUGR*

Potential Determinant	OR (95% CI) for Term IUGR	OR (95% CI) for Preterm IUGR
Maternal age (y)		
<20	0.86 (0.72–1.05)	0.89 (0.59–1.34)
20–34 (reference)	1.00 —	1.00 —
≥35	1.13 (1.03–1.24)	1.12 (0.91–1.39)
Unmarried	1.13 (1.04–1.22)	1.01 (0.84–1.22)
Maternal education (years completed)		
≤10	1.33 (1.18–1.51)	1.11 (0.80–1.54)
11–12	1.11 (1.01–1.23)	1.02 (0.77–1.34)
13–15	1.05 (0.94–1.16)	1.15 (0.86–1.54)
≥16 (reference)	1.00 —	1.00 —
Unknown	1.40 (1.28–1.53)	1.38 (1.07–1.78)
Primiparity	1.74 (1.63–1.85)	1.49 (1.27–1.75)
Height (cm)		
≥157.5 (reference)	1.00 —	1.00 —
<157.5	1.78 (1.65–1.92)	1.58 (1.29–1.94)
Unknown	1.20 (1.05–1.37)	1.15 (0.85–1.57)
Body mass index (kg/m^2)		
<19.8	1.76 (1.61–1.91)	1.51 (1.19–1.93)
19.8–26.0 (reference)	1.00 —	1.00 —
>26.0–29.0	0.73 (0.61–0.87)	1.00 (0.67–1.49)
>29.0	0.63 (0.52–0.77)	0.58 (0.37–0.92)
Unknown	1.08 (0.93–1.25)	1.09 (0.77–1.54)
Net weight gain (kg/wk)		
>0.17 (reference)	1.00 —	1.00 —
≤0.17	1.66 (1.51–1.83)	1.35 (1.03–1.77)
Unknown	1.29 (1.20–1.40)	1.27 (1.04–1.56)
Prepregnancy hypertension	2.20 (1.40–3.44)	2.59 (1.50–4.48)
Pregnancy-induced hypertension		
None (reference)	1.00 —	1.00 —
Mild	1.17 (1.05–1.31)	2.76 (2.18–3.48)
Severe	3.97 (2.96–5.32)	9.70 (7.30–12.89)
Diabetes (prepregnancy or gestational)	0.88 (0.73–1.07)	0.81 (0.57–1.16)
Smoking (cigarettes/day)		
0 (reference)	1.00 —	1.00 —
1–10	1.65 (1.49–1.82)	1.38 (1.07–1.79)
≥11	2.50 (2.31–2.71)	1.95 (1.60–2.38)

Abbreviations: IUGR, intrauterine growth restriction; OR, odds ratio; CI, confidence interval.
* Tabulated odds ratios are adjusted for all variables and categories shown in the table.

potential confounding effects of race or assess its role as an effect modifier.

With these limitations in mind, we offer the following tentative conclusions. The effects of important determinants of IUGR seem to vary according to the severity of growth restriction. With the exception of maternal prepregnancy overweight and obesity, most determinants have larger effects on severe than on mild IUGR. All the pathologic determinants we studied, including cigarette smoking, prepregnancy hypertension, and (especially) PIH, had substantially greater effects on severe IUGR than on mild IUGR.

Most of the determinants we studied also had different magnitudes of effect on IUGR at term versus preterm. Parity and maternal anthropometric factors had larger effects on term IUGR, suggesting that these effects are mediated by placental blood flow and/or nutritional influences late in the third trimester. PIH seems to begin its effect on fetal growth earlier in gestation. The greater magnitude of association of PIH with preterm than with term IUGR is probably explained by the effect of PIH on both spontaneous and induced preterm delivery.[14]

Our results are concordant with several of our hypotheses and with some common clinical observations (such as the strong association of PIH with severe IUGR) but require confirmation in other studies. Pending such confirmation, the heterogeneity in etiologic determinants of IUGR seems to parallel the heterogeneity that investigators have recently observed with respect to prognosis.[15,16] The available evidence now suggests that both the severity and prognosis of IUGR may depend on its cause. In other words, growth-restricted newborns are not all created equal(ly).

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