cigarettes during pregnancy, regardless of smoking status before conception, and three categories were used: nonsmokers, moderate smokers (9 cigarettes per day or less) or heavy smokers (10 or more cigarettes per day). The maternal and neonatal information collected comprised of maternal demographic characteristics: medical and obstetrics histories; and antepartum, intrapartum, postpartum, and neonatal outcomes.

Statistical Analysis

Statistical analyses were undertaken to identify independent predictors of birth weight (as a continuous variable), and risk factors for SGA. Differences in proportion of categorical variables were compared using χ^2. Differences in continuous variables were determined by independent t-test or by the Mann-Whitney-Wilcoxon test for non-normally distributed data, or ANOVA. Multiple logistic regression analyses were performed to identify the risk factors for SGA. The dependent variable was SGA. Logistic regression was used in the multivariable analysis, the measure of effect being the odds ratio (OR) with its 95% confidence interval (CI) and adjusted for two potential cofounders: maternal height and gestational weeks. Differences were considered significant at $p<0.05$. Statistical analysis was carried out with SPSS for Windows, version 12.0 (SPSS Inc, Chicago, Ⅲ,USA).

Ethical considerations

This study was approved by the ethical committees at the University of Tokyo.

Results

The investigation was undertaken using data from 2972 mothers who gave birth in the metropolitan area of Tokyo between January 2002 and December 2003. Table 1 presents the maternal and neonatal demographic characteristics. Out of 2972 deliveries, 250 (8.4%) were SGA infants and 2722 (91.6%) were of normal birth weight (NBW: more than the 10th percentile for gestational age at birth). The sizes of the SGA infants, such as height, head and chest measurements were significantly lower than NBW infants.

In mothers with SGA infant, the average maternal age was 29.3±4.3. Altogether, 12.0% were over 35 years old, and 0.8% were less than 20 years old; and 42.8% of the mothers were primiparous. No statistically significant differences were found in maternal age between SGA and NBW infants, but the proportion of SGA infants among primiparous women was lower than that of NBW infants ($p<0.01$). The mean maternal weight gain was 8.4±3.7 kg in mothers with SGA infants and 1.3 kg lower than in mothers with NBW infants ($p<0.001$). Prepregnancy BMI was 20.3±2.7 kg/m^2 in mothers with SGA infants, which was 0.5 kg/m^2 lower than in those with NBW infants ($p<0.01$). Smoking status showed a higher consumption in women with SGA infants (23.4% for those with SGA infants vs 15.9% for those with NBW infants, $p<0.01$).

Table 2 presents the birth weight and proportion of SGA infants by maternal weight gain category according to prepregnancy BMI. Among the 2972 mothers, the frequencies of underweight, normal weight and obesity were 316 (10.6%), 2337 (78.7%) and 319 (10.7%)

respectively. The mean birth weight in underweight infants was lower than in the normal and obese infants (2979.1±321.4 g; 3052.5±335.6 g, and 3134±350.1 g, respectively; $p<0.001$). The proportion of SGA infants was 10.8% for underweight, 8.4% for normal and 6.3% for obese, but there was no significant difference among the prepregnancy BMI categories ($p=0.13$).

Among women of normal weight and below, the birth weight increased significantly with greater maternal weight gain ($p<0.001$ for underweight and normal). In the underweight group, the birth weight in mothers gaining less than 8.0 kg, 8.0-10.0 kg, 10.1-12.0 kg and more than 12.0 kg were 2788.5±293.9 g, 2931.9±278.6 g, 3017.6±295.5 g and 3098.9±327.4 g, respectively ($p<0.05$). The birth weight in mothers with less than 8.0 kg weight gain was 310.4 g lower than in mothers with more than 12.0 kg weight gain. In the normal group, birth weights in mothers with less than 8.0 kg, 8.0-10.0 kg, 10.1-12.0 kg and more than 12.0 kg weight gain was 2988.1±325.3 g, 3001.1±307.5 g, 3058.4±328.5 g and 3180.1±348.4 g, respectively ($p<0.05$). Birth weights in mothers with gains of less than 8.0 kg were 192 g lower than in mothers with more than 12.0 kg weight gain. On the other hand, in the obese group, birth weight increased with greater maternal weight gain, but without any statistical significance ($p=0.07$). There were significantly more SGA infants than underweight and normal infants ($p<0.05$, $p<0.01$, respectively) among mothers with weight gains of less than 8.0 kg, but not in the obese group because the sample was small ($p=0.14$).

Table 3 presents the birth weight and proportion of SGA infant by maternal weight gain category according to smoking status. Smoking prevalence was 14.1%, the prevalence of

moderate and heavy smokers being 4.2% and 9.9%, respectively. It was noticed that the birth weight was significantly decreased in smokers. The mean birth weight of heavy smokers was significantly lower than that of moderate smokers and nonsmokers (heavy smokers, 2978± 349.3 g; moderate smokers, 3035.8±310.1 g; nonsmokers, 3065.0±334.0 g; $p<0.001$). The prevalence of SGA was the highest among heavy smokers, smoking more than 10 cigarettes per day (13.7%, $p<0.01$).

Among women who smoke, birth weight was increased with increased maternal weight gain ($p<0.001$ for nonsmokers, $p<0.01$ for moderate smokers, $p<0.001$ for heavy smokers). In all categories of stratified maternal weight gain, birth weight among infants whose mothers were nonsmokers was significantly higher than in those whose mothers were moderate and heavy smokers. The proportion of SGA births among nonsmokers was significantly increased in the low maternal weight gain categories (10.9% for less than 8.0 kg, 8.3% for 8.0-10.0 kg, 7.2% for 10.1-12.0 kg, 3.4% for more than 12.0 kg; $p<0.001$), but was not significant for moderate and heavy smokers ($p=0.16$, $p=0.31$, respectively).

Table 4 presents the results of the multivariate logistic regression analysis for SGA. The odds ratio for SGA was significantly decreased by prepregnancy BMI (OR 0.89, 95%CI. 0.84-0.94, $p<0.001$). The odds ratio of SGA for stratified maternal weight gain was 1.79 (95%CI.1.24-2.58, p=<0.01) for weight gain less than 8.0 kg, 1.16 (95%CI.0.79-1.71, $p=0.45$) for weight gain of 8.0-10.0 kg, and 0.49 (95%CI.0.30-0.78, p=<0.01) for weight gain over 12.0 kg. The proportion of SGA infants was significantly increased among women who had less than 8.0

kg weight gain and decreased among those who had more than 12.0 kg weight gain. Heavy smokers were 2.3 times more likely to have an SGA infant (95%CI.1.58-3.38, $p<0.001$) than nonsmokers, but no such effect was observed among moderate smokers (95%CI.0.51-2.09, $p=0.93$). Live birth order, maternal advanced age and the sex of the infant were not associated with the proportion of SGA infants.

Discussion

We conducted an epidemiological study in which the risk factors for an SGA birth at full term were examined, because almost all reports in Japan have been focused on the risk factors for LBW. The term SGA refers not to fetal growth but to the size of the infant at birth; the term LBW is defined as birth weight less than 2500 g, regardless of gestational age. A large number of epidemiological studies have noted that gestational age, sex and parity influence on the birth weight. Therefore, this study may contribute to analyzing the factors behind the recent trend toward decreased average birth weight in Japan. In our study, prepregnancy BMI, low maternal weight gain and maternal smoking during pregnancy were identified as risk factors of SGA, but neither live birth order nor advancing maternal age was observed as a risk factor.

Some researches reported that a lower prepregnancy BMI affects the risk of adverse pregnancy outcomes, such as intrauterine growth retardation or LBW infants.[9-10] The mean birth weight in the underweight group was statistically significantly lower than that in the normal and obese, and the proportion of SGA was higher, but not significantly so. Decreased

body mass index was associated with an increase in the risk of an SGA infant. The increase in the prevalence of underweight in the ages between 20 and 29, and in 30 and 39-year-old women over the last two decades (25.2 to 26.0%, 9.9 to 15.1% respectively) is a possible cause, given the increasing incidence of SGA births in Japan.[6] The prevalence of a BMI less than the 18.5 kg/m^2 among young women was twice as high as that of other industrialized countries, such as America and Australia.[11-12]

Our data suggest that the incidence of SGA births and the birth weight vary depending on how much weight gain occurs in the mother during pregnancy. As observed in this study, women with a weight gain of less than 8.0 kg group were twice as likely to deliver SGA infants as women with weight gains of 10.1-12.0 kg. The incidence of SGA births was significantly reduced in women who gained more than 12.0 kg and birth weight showed a significant correlation with maternal weight gain among women with prepregnancy BMI of less than 24.0. Thus, an increase in total maternal weight gain in pregnancy may eliminate or reduce the frequency of SGA.

Our data show that total maternal weight gain and increased prepregnancy weight gain in Japanese women were key factors in the incidence in average birth weight and reduced the proportion of SGA infants. Care should be taken not to restrict weight gain excessively: weight gain of more than 12.0 kg is necessary to eliminate the risk of an SGA birth. Therefore, our findings call into question whether the currently recommended maternal weight gains established by the JSOG (which are 10-12 kg for women of BMI<18.0, 7-10 kg for women of

18.0≦BMI≦24.0, 5-7 kg for women of BMI>24.0) [13] are inappropriate for Japanese women, and further research is necessary to determine the relationship between maternal weight gain and prenatal outcomes.

Prenatal cigarette smoking is among the preventable causes of SGA and Intrauterine growth retardation.[14] The effects of cigarette on birth weight are probably due to several mechanisms. Carbon monoxide, by binding to fetal hemoglobin and reducing the availability of oxygen, is thought to be an important cause of the fetal growth restriction.[15] The rate of smoking in Japan among females aged 20-29 years has increased from 8.9% in 1989 to 21.3% in 1997, and continues to increase still.[16] In our study, although smoking status affects birth weight and although the incidence of SGA births was the highest among heavy smokers, maternal weight gain among smokers was not associated with a decreased proportion of SGA infants, even among moderate smokers. Consistent with the findings of Butler et.[17] the odds ratio for heavy smokers to have SGA infants was about 2.3 times higher than for nonsmokers, but did not vary significantly for moderate smokers (<10 cigarettes/day). Although the power to detect statistically significant risks for the delivery of an SGA infant among moderate smokers was limited because of the small number of women who smoke less than 10 cigarettes a day, increased birth weight was observed among moderate smokers with increased weight gain. Horta et al.[18] reported that women who stopped smoking in the first trimester of pregnancy faced risks similar to those of nonsmokers, but mothers who smoked until second or third trimester had higher risks. Our data suggest that birth weight may be increased by reducing

cigarettes consumption and by increasing maternal weight gain. Health providers should encourage women to stop smoking at conception.

There are potential limitations in our study. First, our sample size for SGA infants was small. Although we confirmed that increased maternal weight gain in pregnancy had a significant impact on birth weight, we could not determine the influence of maternal weight gain, when categorized by prepregnancy BMI or by maternal smoking status, on the incidence of SGA by logistic regression analyses.

Second, we could not obtain the details of weight changes in the three trimesters since the data used was secondary. Maternal weight gain in each trimester is a factor that allows prediction of fetal size. Brown et al. reported that maternal weight gain in the first trimester of pregnancy influences birth weight more strongly than that in the second or third trimester.[19] On the other hand, Strauss et al. demonstrated that low maternal weight gain in the second or third trimester caused a twofold increase of LBW infants compared to that in the first trimester.[20] Thus, the effect of maternal weight gain in each trimester of pregnancy on birth weight is still controversial. If we could identify the crucial times in pregnancy when weight gain most influences birth weight, early intervention might reduce the proportion of SGA births or increase the birth weight, and potentially decrease the risk later in life, of adult onset diseases.

Third, the current study lacks any data on passive smoking at the workplace and at home. Passive smoking at home among mothers presented a 1.3 times greater risk of delivery of an

LBW infant than the absence of a smoker in the workplace and home.[21] To investigate more thoroughly and accurately the effect on SGA or birth weight, it will be necessary to obtain information about smoking status that includes passive smoking throughout pregnancy.

In conclusion, although we can not claim the study subjects to be representative of the whole Japanese population, we are certain that the findings presented in this study will give a better insight into, and understanding of the current situation in the Japanese population regarding the said issue. Our study confirms the detrimental effect of low prepregnancy BMI, low maternal weight gain and maternal smoking during pregnancy on birth weight and incidence of SGA births. An increase in maternal weight gain and prepregnancy BMI, and a decrease in maternal smoking during pregnancy would be expected to reduce this incidence, as well to increase birth weight; and improved prepregnancy nutritional status, as determined from prepregnancy weight might also have a beneficial effect. Appropriate maternal BMI at conception followed by adequate weight gain during pregnancy may have a substantial influence on reducing the number of SGA infants. Recent recommendations advising additional weight gain in pregnancy need further evaluation before implementation and may offer long-term benefits to the offspring. In addition, the educational programs directed at girls and young women in order to prevent excessive dieting and smoking should be strengthened.

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Table 1. Maternal and Neonatal Characteristics.

	SGA* n=250 (8.4 %)	NBW** n=2,722 (91.6 %)	p-value
Infants			
Birth weight (g)	2492.1±181.4	3105.1±299.6	<0.001
Height at birth (cm)	46.8±1.5	49.3±1.6	<0.001
Head circumstance (cm)	31.8±1.1	33.1±1.2	<0.001
Chest circumstance (cm)	29.6±1.3	31.8±1.4	<0.001
Gestational length (week)	39.4±1.0	39.5±1.0	<0.001
Placenta weight (g)	474.2±66.3	585.1±96.5	<0.001
Mothers			
Age at delivery (year)	29.3±4.3	29.6±4.3	0.37
Over 35 years old (%)	12.0	12.0	0.99
Under 20 years old (%)	0.8	1.1	0.63
Height (cm)	157.6±5.4	158.3±5.1	0.08
Prepregnancy weight (Kg)	50.4±7.4	52.1±7.3	<0.01
Prepregnancy BMI (kg/m^2)	20.3±2.7	20.8±2.7	<0.01
Total weight gain (Kg)	8.4±3.7	9.7±3.5	<0.001
Primiparous (%)	42.8	53.1	<0.01
Smoker (%)	23.4	15.9	<0.01

Values are presented as Mean±SD or percentages (%)

p value based on χ^2 test for percent and variables Student t-test for continuous ones.

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*SGA: small for gestational age infant (the lowest 10th percentile for gestational age)

**NBW: not SGA group (more than the 10th percentile for gestational age at birth)

Table 2. Birth weight and proportion of SGA infants by maternal weight gain category and prepregnancy BMI.

	Birth weight(g)				SGA		
	n	(%)	Mean±SD	p value	n(%)		χ^2 test p values
Prepregnancy BMI							0.13
Underweight (a)	316	(10.6)	2979.1±321.4	b,c	34.0	(10.8)	
Normal (b)	2337	(78.7)	3052.5±335.6	a,c	196.0	(8.4)	
Obesity (c)	319	(10.7)	3134.0±350.1	a,b	20.0	(6.3)	
Underweight							<0.05
<8.0 Kg (a)	62	(19.6)	2788.5±293.9	b,c,d	13.0	(21.0)	
8.0–10.0 Kg (b)	75	(23.7)	2931.9±278.6	a,d	5.0	(6.7)	
10.1–12.0 kg (c)	75	(23.7)	3017.6±295.5	a	7.0	(9.3)	
>12.0 kg (d)	104	(32.9)	3098.9±327.4	a,b	9.0	(8.7)	
Normal							<0.001
<8.0 Kg (a)	656	(28.1)	2988.1±325.3	c,d	74.0	(11.3)	
8.0–10.0 Kg (b)	614	(26.3)	3001.1±307.5	c,d	59.0	(9.6)	
10.1–12.0 kg (c)	511	(21.9)	3058.4±328.5	a,b,d	42.0	(8.2)	
>12.0 kg (d)	556	(23.8)	3180.1±348.4	a,b,c	21.0	(3.8)	
Obese							0.14
<8.0 Kg (a)	185	(58.0)	3099.0±345.6	0.07	16.0	(8.6)	
8.0–10.0 Kg (b)	53	(16.6)	3144.6±353.6		3.0	(5.7)	
10.1–12.0 kg (c)	46	(14.4)	3168.2±353.6		1.0	(2.2)	
>12.0 kg (d)	35	(11.0)	3262.1±343.5		-		

Values are presented as Mean±SD or percentages(%)

p value based on χ^2 test for percent. Pediatrics International (in press)

p values were analysed by prepregnancy BMI categories (Underweight<18.0,Normal 18.0–24.0, Obesity >24.0).

Letters show the values that are significantly different(p<0.05, Tukey's test).

Table 3. Birth weight and proportion of SGA infants by maternal weight gain category and smoking status.

	Birth weight(g)			SGA	
	n (%)	Mean±SD	p values	n (%)	χ^2 test p values
Maternal smoking status					<0.01*
nonsmoker (a)	2554 (85.9)	3065.0±334.0	c	201.0 (7.9)	
moderate smoker (b)	126 (4.2)	3035.8±310.1		9.0 (7.1)	
heavy smoker (c)	292 (9.9)	2978.9±349.3	a	40.0 (13.7)	
Nonsmoker					<0.001
<8.0 Kg (a)	829 (32.5)	3005.7±338.1	c,d	90.0 (10.9)	
8.0–10.0 Kg (b)	650 (25.5)	3024.9±300.6	c,d	54.0 (8.3)	
10.1–12.0 kg (c)	543 (21.3)	3081.5±338.0	a,b,d	39.0 (7.2)	
>12.0 kg (d)	532 (20.8)	3191.5±326.9	a,b,c	18.0 (3.4)	
Moderate smoker					0.16
<8.0 Kg (a)	23 (18.3)	2907.6±311.5	d	4.0 (17.4)	
8.0–10.0 Kg (b)	27 (21.4)	2914.3±260.8	d	2.0 (7.4)	
10.1–12.0 kg (c)	33 (34.1)	3095.3±329.7		2.0 (6.1)	
>12.0 kg (d)	43 (34.1)	3134.9±264.4	a,b	1.0 (2.3)	
Heavy smoker					0.31
<8.0 Kg (a)	51 (17.5)	2885.3±331.3	d	9.0 (17.6)	
8.0–10.0 Kg (b)	65 (22.3)	2887.3±313.5	d	11.0 (16.9)	
10.1–12.0 kg (c)	56 (19.2)	2965.3±361.1		9.0 (16.1)	
>12.0 kg (d)	120 (41.1)	3074.5±333.4	a,b	11.0 (9.2)	

Values are presented as Mean±SD or percentages (%)

p value based on χ^2 test for percent. Pediatrics International (in press)

p values were analysed by maternal smoking status (Nonsmoker, Moderate smoker<10/day, Heavy smoker>10/day).

Letters show the values that are significantly different (p<0.05, Tukey's test).

Table 4. Multivariate logistic regression analysis for SGA infants.

Variables	OR*	95%CI**	P value
Prepregnancy BMI	0.89	0.84–0.94	<0.001
Maternal weight gain			
<8.0 Kg	1.79	1.24–2.58	<0.01
8.0–10.0 Kg	1.16	0.79–1.71	0.45
10.1–12.0 kg	1.0		
>12.0 kg	0.49	0.30–0.78	<0.01
Maternal smoking status			
Nonsmoker	1.0		
Moderate smoker	1.04	0.51–2.09	0.93
Heavy smoker	2.31	1.58–3.38	<0.001
Age at delivery (years)	0.98	0.94–1.01	0.13
Femal (vs male)	0.95	0.73–1.23	0.67
Live birth order			
1	1.0		
2	1.35	0.97–1.96	0.34
>3	1.07	0.64–1.77	0.81

*OR:Odds ratio, **CI: Confidence interval.
Adjusted for maternal height, gestational age
Maternal smoking status
(Nonsmoker, Moderate smoker<10/day, Heavy smoker>10/day).

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SPECIAL ARTICLE

Maternal weight gain ranges for optimal fetal growth in Japanese women

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KEYWORDS
IUGR;
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Cross-sectional survey

Abstract

Objective: To identify adequate weight gain ranges during pregnancy in Japanese women. *Method:* Obstetric records from 2001 to 2002 for 46,659 term, singleton, vaginally delivered live births was used to estimate IUGR and macrosomia risk. Total maternal weight gain was grouped according to gestational age-specific percentile values of weight gain as follows: "very low" (under the 25th), "low" (25th to 49th), "moderate" (50th to 74th), "high" (75th to 89th), and "very high" (90th and over). *Results:* About 6% of infants were identified as having IUGR and 0.9% as macrosomia. IUGR risk was elevated with low weight gains. Macrosomia risk was related to high weight gains and previous spontaneous abortions. *Conclusion:* Achieving weight gains between the 50th and 75th percentiles for gestational age was considered adequate for optimal fetal growth in Japanese pregnant women.

1. Introduction

Recent trends in industrialized countries, such as the USA, Canada [1], Sweden [2] and Norway [3], show that infants are born heavier, with increased mean birth weight and a decline in the prevalence of low birth weight (LBW: under 2.5 kg). This

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phenomenon has been attributed to an increase in heavier mothers, and decreased maternal smoking in pregnancy. By contrast, the mean birth weight of Japanese infants has steadily declined since the 1980s, despite increasing mean height of young women since World War II. Mean birth weight in male and female infants has decreased from 3.23 kg (males) and 3.14 kg (females) in 1980, to 3.06 kg (males) and 2.98 kg (females) in 2002, and the proportion of LBW has increased from 5.2% in 1980 to 9.0% in 2002. Macrosomia, as defined by a birth weight of 4.0 kg or more, has decreased from 3.0% in 1980 to 1.0% in 2002 [4]. A new fetal growth curve has had to be developed to reflect the changing growth status of Japanese infants [5] because the standard fetal growth curve developed by Nishida et al. in 1984 [6] was no longer applicable due to an increasing number of smaller infants. Moreover, increase in LBW among term singletons has been observed in the national Children and Infants Growth Survey [7], rising from 2.7% in 1980 to 4.9% in 2000.

This recent trend in reduced fetal size in Japan may be attributed to changes in maternal health. First, maternal smoking in pregnancy had increased from 6.5% in 1990 to 10.9% in 2000 [7]. This is in accordance with the rise in smoking rates among young women. Smoking rates in women aged 20–29 years has increased from 11.9% in 1990 to 20.9% in 2000 [8]. Second, the prevalence of underweight (BMI<18.5 kg/m^2) in young Japanese women has been increasing since 1976 [9]. It has increased from 15.8% in 1976–1980 to 22.9% in 1995–2000 in women aged 20–24 years, and 13.5% to 23.7% in women aged 25–29 years.

In spite of these unfavorable changes regarding maternal and child health, few attempts have been made to enhance fetal growth by promoting greater weight gain during pregnancy. To date, the only weight gain guideline [10] is for the prevention of pregnancy toxemia, not for achieving optimal fetal growth. This study was aimed at newly developing reference weight gain goals in Japanese women, using the 2001–2002 data from the JSOG Perinatal Database.

2. Methods

The data set of 112,257 deliveries in 2001 and 2002 from the 150 obstetric units participating in the Japan Society of Obstetrics and Gynecology (JSOG) Perinatal Database was obtained for analyses. Each year, the Committee requests these units to provide information on maternal health and birth outcome on all deliveries at each unit. No private information on mothers or children is available in this database, which is shared freely by JSOG members for research purposes. This database covered 4.8% of 2,324,517 births which were recorded in the Vital Statistics *reports in* 2001 and 2002 [4].

To determine referent values for weight gain in pregnancy, the data was limited to low-risk singleton term deliveries, according to the exclusion criteria as shown in Table 1. Cesarean deliveries were excluded because most of them were referrals from other hospitals for an emergent delivery. From the remaining cases, 46,659 cases with complete information available on birth weight, infant gender, gestational age (weeks), maternal age, maternal pre-pregnancy and delivery body weight, obstetric history, pregnancy complications (pregnancy-induced hypertension and diabetes), and smoking and drinking history during pregnancy, was selected. Maternal height was not recorded in this database.

The latest JSOG criteria define pregnancy-induced hypertension (PIH) as the first onset of hypertension after 20 weeks gestation [11]. PIH is further categorized into preeclampsia, which is a hypertensive state accompanied by proteinuria, and gestational hypertension, which is a hypertensive state without proteinuria. Proteinuria is defined as urinary protein excretion of 0.3 g/l or greater in a 24-h urine collection. Hypertension is defined as a systolic blood pressure ≧140 mmHg and/or a diastolic blood pressure ≧110 mmHg. As the data was collected before the revision of the PIH criteria, preeclampsia here is hypertension with heavy proteinuria (urinary protein excretion 2.0 g/l or greater).

The 1984 Nishida standard [6] was applied for identification of IUGR in our data. Mean birthweight minus 1.5 S.D. (standard deviations) was selected as a cutoff value for IUGR in this standard

Table 1 Cases excluded from analysis (total number of cases=112,257)

	N	% of total
Cesarean deliveries	30,559	27.2
Delivery method unknown	2258	2.0
Multiple gestations	8387	7.5
Preterm deliveries (<37 weeks)	19,623	17.5
Post-term deliveries (>41 weeks)	623	0.6
Stillbirths and early neonatal deaths[a]	2558	2.3
Maternal deaths	11	0.01
Congenital anomalies of the infant[b]	2449	2.2

[a] Early neonatal death=infant death within 7 days after delivery.
[b] Includes chromosomal disorders.

rather than the 10th percentile, as it is reported to be more relevant to infant morbidity rates [6]. Macrosomia was defined as a birthweight over 4000 g.

Maternal total weight gain was grouped according to the gestational age-specific percentile values of weight gain as follows: "very low" (under the 25th), "low" (25th to 49th), "moderate" (50th to 74th), "high" (75th to 89th), and "very high" (90th and over). Chi-square tests were applied to compare the prevalence of IUGR among different groups. The multivariate logistic model was applied to estimate the odds ratios for significantly related maternal and child factors to IUGR and macrosomia. A p value of less than 0.05 (two-tailed) was considered to be significant. All statistical analyses were performed using the SPSS 11.5J package program (SPSS Japan Inc., Tokyo).

3. Results

The descriptive characteristics of the 46,659 cases fulfilling our inclusion criteria are shown in Table 2. There were 410 cases (0.9%) of macrosomia, including 18 cases of infants weighing 4500 g and over. Four hundred and fifty-seven (1.0%) were conceived through in vitro fertilization (IVF). PIH was observed in 946 cases, and 196 cases of PIH (20.3%) had preeclampsia.

The distribution of birthweight according to infant gender, parity, and gestational age is shown in Table 3, together with the Nishida standards for IUGR [6]. Six percent of cases were identified as having IUGR. In males and females born to primiparas, the 10th percentile values for birth weight were less than 2500 g at 37–38 weeks. The prevalence of LBW and IUGR were higher in females than in males at the same gestational age. LBW prevalence decreased significantly with advanced gestational age in all four groups ($p<0.01$) grouped by infant gender and parity. IUGR prevalence also decreased with advanced gestational age in males born to primiparas ($p<0.01$) and in males ($p<0.01$) and females ($p<0.01$) born to multiparas. The prevalence of macrosomia increased with advanced gestational age in all four groups.

In order to categorize maternal weight gain according to gestational age, four cutoff values (the 25th, 50th, 75th, and 90th percentiles) for each gestational age were applied, as shown in Table 4. Using these cutoffs, maternal weight gains were categorized into "very low" (under the 25th), "low" (25th to 50th), "moderate" (50th to 74th), "high" (75th to 89th), and "very high" (90th and over). To examine the effect of selected factors on

Table 2 Maternal and infant characteristics of the selected sample

	Selected sample (n=46,659)	2002 National data [4] (n=1,153,855)
Infants		
Male (%)	50.4	51.4
Birth weight (g)[a]	2982±472	3020[b]
Birth weight category		
Very low (<1500 g) (%)	0.07	0.7
Low (1500–2500 g) (%)	6.2	8.3
High (>4000 g) (%)	0.9	1.1
Placental weight (g)[a]	576±112	–
Gestational length (weeks)[a]	39.2±1.1	–
Mothers		
Age at delivery (years)[a]	29.9±4.8	29.8
>34 years old (%)	16.3	12.8
<20 years old (%)	1.8	1.9
Number of prior gestations[a]	1.1±1.2	–
Number of prior deliveries[a]	0.6±0.8	–
Primiparas (%)	53.5	49.5
IVF conception	1.0	–
Past preterm deliveries (%)	1.3	–
Past still births (%)	0.9	–
Past spontaneous abortions (%)	12.6	–
Past cesarean deliveries (%)	1.9	–
Pre-pregnancy weight (kg)[a]	52.2±8.2	–
Delivery weight (kg)[a]	62.1±8.5	–
Total weight gain (kg)[a]	9.9±4.3	–
Pregnancy complications		–
Pregnancy-induced hypertension (%)	2.0	–
Diabetes (%)[c]	1.2	–
Smoking in pregnancy (%)	6.3	–
Drinking in pregnancy (%)	4.6	–

[a] Mean±S.D.
[b] Original data in kg.
[c] Gestational diabetes and pregestational diabetes.

IUGR and macrosomia, the odds ratios (ORs) for each of these factors was calculated by applying logistic regression analysis adjusted for maternal age, parity, pre-pregnancy weight, and infant gender, as shown in Table 5. Compared to the reference "moderate" weight gain group, the ORs for IUGR were significantly high in the "very low" (2.91, 95% CI: 2.59–3.26) and "low" (1.48, 95% CI: 1.34–1.65) weight gain groups. Conception by IVF or prior negative obstetric history, such as preterm deliveries, spontaneous abortions, and stillbirths, were not related to IUGR. Prior cesarean deliveries did not increase IUGR risk. Maternal PIH and diabetes were both significant factors increasing IUGR risk. Preeclamptic mothers presented a higher OR (5.58, 95% CI: 3.92–7.96) for IUGR than mothers with gestational hypertension (2.88, 95% CI: 2.27–3.66). Maternal smoking and drinking both increased IUGR risk.

Compared to the reference "moderate" weight gain group, the ORs for macrosomia were signifi-

Distribution of birthweight (g), according to infant gender, parity and gestational length (weeks)

	Primiparas				Multiparas				
	37 weeks	38 weeks	39 weeks	40 weeks	41 weeks	37 weeks	38 weeks	39 weeks	40 weeks
	N=747	*N*=1843	*N*=3691	*N*=4178	*N*=1941	*N*=875	*N*=2062	*N*=3492	*N*=3225
	2619±362	2792±331	2935±337	3049±329	3138±346	2745±346	2896±355	3047±334	3159±357
itile	2171	2394	2538	2650	2722	2315	2470	2640	2740
itile	2650	2792	2920	3042	3132	2750	2891	3042	3150
itile	3056	3191	3360	3468	3566	3184	3340	3468	3590
ht for IUGR[a]	2080	2290	2450	2560	2630	2230	2450	2620	2690
	7.6	5.7	6.1	6.3	6.4	6.4	9.4	8.9	7.5
	31.6	17.7	8.0	4.0	2.5	21.5	11.6	4.4	2.4
ia	0.0	0.1	0.2	0.6	1.1	0.0	0.4	0.5	1.4
	N=908	*N*=2263	*N*=3883	*N*=3861	*N*=1593	*N*=1022	*N*=2382	*N*=3750	*N*=2967
	2694±367	2895±341	3042±335	3139±348	3235±350	2832±349	3011±345	3155±341	3283±363
itile	2216	2466	2632	2720	2809	2413	2599	2725	2850
itile	2697	2891	3048	3130	3230	2820	3002	3144	3276
itile	3150	3315	3464	3580	3663	3259	3444	3596	3738
ht for IUGR[a]	2180	2360	2480	2560	2630	2300	2500	2660	2720
	8.4	5.4	4.4	4.0	3.8	4.9	5.7	6.5	5.1
	27.4	11.9	4.8	2.3	1.2	14.5	5.6	1.7	1.3
ia	0.1	0.2	0.3	0.7	1.6	0.4	0.8	1.3	2.4

standard [6].