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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References

1 Barker DJ, Gulkman PD, Godfery KM, *et al.* Fetal nutrition and cardiovascular disease in adult life. *Lancet.* 1993;341:938-41.

2 Lagerstrom M, Bremme K, Eneroth P, *et al.* School performance and IQ-test scores at age 13 as related to birth weight and gestational age. *Scand J Psychol*. 1991;32:316-24.

3 Rich-Edwards JW, Colditz GA, Stampfer MJ, *et al.* Birthweight and the risk for type 2 diabetes mellitus in adult women. *Ann Intern Med.* 1999;130: 278-84.

4 Li R, Haas JD, Habicht JP. Timing of the influence of maternal nutritional status during pregnancy on fetal growth. *Am J Hum Biol*. 1998;10:529-39.

5 Wiles NJ, Peters TJ, Leon DA, *et a*l. Birth weight and psychological distress at age 45-51 years: Results from the Aberdeen Children of the 1950s cohort study. *Br J Psychiatry.* 2005;187:21-8.

6 Ministry of Health and Welfare, Japan, Mothers' & Children's Health Division, *Maternal And Child Health Statistics of Japan*, Mothers' & Children's Health Organization. Tokyo, 2004.

7 Nishida H, Sakagami M, Kurati K, Asada M, Kubo S, Funakawa H. Fetal

growth curves of Japanese, *Nihon Shinseiji-gakkaishi.* 1984;20:90-7 (in Japanese).

8 Choo V. WHO reassesses appropriate body-mass index for Asian populations. *Lancet.* 2002;360:235.

9 Nandi C, Nelson MR. Maternal pregravid weight, age, and smoking status as risk factors for low birth weight births. *Public Health Rep.* 1992;107:658-62.

10 Mendelson R, Dollard D, Hall P, *et al.* The impact of the Healthiest Babies Possible Program on maternal diet and pregnancy outcome in underweight and overweight clients. *J Can Diet Assoc.* 1991;52:229-34.

11 Schoenborn CA, Adams PF, Barnes PM. Body weight status of adults:United States, 1997-98. *Adv Data Vital Health Stat.* 2002;1-15.

12 Brown WJ, Mishra G, kenardy J, *et al.* Relationship between body mass index and well-being in young Australian women. *Int J Obes Relat Metab Disord.* 2000;24:1360-8.

13 Committee of Nutritional guideline, Japan Society of Obstetrics and Gynecology, *Journal of Japan of Society Obstet & Gynecol.* 1990;51:N-507-10.

14 Wen SW, Goldenberg RL, Cutter GR, *et al.* Smoking, maternal age, fetal growth and gestational age at delivery. *Am J Obstet Gynecol.* 1990;162:53-8.

15 Abel EL. Smoking during pregnancy: A review of effects on growth and development of offspring. *Hum Biol*. 1980;52:593-625.

16 Ministry of Health, Labour and Welfare. *National Nutrition Survey.* Dai-ichi Shuppan,

Tokyo, 2002 (in Japanese).

17 Butler NR, Goldstein H, Ross EM. Cigarette smoking in pregnancy: its influence on birth weight and perinatal mortality. *Br Med J.* 1972 ;2:127-30

18 Horta BL, Victora CG, Menezes AM, et al. Low birthweight, preterm births and intrauterine growth retardation in relation to maternal smoking. *Paediatr Perinat Epidemiol.* 1997;11:140-51.

19 Brown JE, Murtaugh MA, Jacobs DR, *et al.* Variation in newborn size according to pregnancy weight change by trimester1-3. *Am J Clini Nutr.* 2002;76:205-9.

20 Strauss RS, Dietz WH. Low maternal weight gain in the second or third trimester increases the risk for intrauterine growth retardation. *J Nutr.* 1999;129:988-93.

21 Ojima T, Uehara R, Watanabe M, *et al.* Population attributable fraction of smoking to low birth weight in Japan. *Pediatr Int.* 2004;46:264-7.

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 10^{th} percentile for gestational age)

**NBW: not SGA group (more than the 10^{th} 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.

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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.

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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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ORIGINAL ARTICLE

Impact of prepregnant body mass index and maternal weight gain on the risk of pregnancy complications in Japanese women

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Abstract
Background. To analyze the association of pregnancy complications with prepregnant body mass index and weight gain during pregnancy in Japanese women. *Methods.* A retrospective cohort study was conducted with 21,718 Japanese women with a singleton pregnancy. Pregnant women were grouped by prepregnant body mass index and evaluated for association with pregnancy complications using multivariate logistic regression analysis. The women in each body mass index group were then divided into groups by weight gain during pregnancy using intervals of 0.05 kg/week to analyze the relationship between the weight gain and pregnancy complications by multivariate logistic regression association analysis. *Results.* In both nulliparous and parous women, the least pregnancy complications were found among women with medium prepregnant body mass indexes (18–23.9). Significant risks of pregnancy complications were associated with low (<18) and high (≥24) prepregnant body mass indexes, particularly high prepregnant body mass indexes. In nulliparous women, the optimal weight gain was 0.25–0.4 kg/week for low (<18) prepregnant body mass index, 0.20–0.30 kg/week for medium (18–23.9) prepregnant body mass index, and ≥0.05 kg/week for high (≥24) prepregnant body mass index. In parous women, the corresponding values were ≥0.20, 0.20–0.30, and 0.05–0.30 kg/week. *Conclusions.* Japanese women with prepregnant body mass indexes from 18 to 23.9 are least associated with pregnancy complications, although there is a broad range of prepregnant body mass indexes associated with few pregnancy complications. Optimal weight gain is roughly inversely related to prepregnant body mass index.

Key words: *Prepregnant BMI, pregnancy complication, weight gain*

Abbreviations: *BMI: body mass index, SGA: small-for-gestational-age infants, LGA: large-for-gestational-age infants, NICU: neonatal intensive care unit*

It has been reported that lean prepregnant women have an increased risk of delivering small-for-gestational-age infants (SGA) (1,2), while prepregnant obesity is associated with gestational diabetes, pre-eclampsia, eclampsia, cesarean delivery, labor complications, and macrosomia (3–6). Excessive weight gain during pregnancy has also been reported to be associated with an increased risk of pre-eclampsia, gestational diabetes, and other complications (6–10). Therefore, attempts have been made to categorize prepregnant body mass index (BMI) (3,11) and maternal weight gain in relation to the risk of pregnancy complications and adverse birth outcomes (6,8,9). However, these studies have so far been mostly on Caucasian women and little information is available on Japanese women. In this study, we addressed the issue of pregnancy complications in relation to prepregnant BMI and weight gain during pregnancy among Japanese women in a retrospective cohort study.

Material and methods

Subjects and database

We conducted a retrospective cohort study with 21,718 women with a singleton pregnancy who

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delivered the baby at term in Osaka Medical Center and Research Institute for Maternal and Child Health in 1981–1999. All pregnancies and deliveries were registered in the hospital database, which carried demographic characteristics, and antepartum, intrapartum and neonatal complications, filled out prospectively. Demographic characteristics included prepregnant BMI, age, weight gain during pregnancy, and gestational age at delivery. Antepartum complications included pre-eclampsia and severe pre-eclampsia. Intrapartum complications included blood loss of more than 1,000 ml at delivery, vacuum extraction, elective cesarean delivery (cesarean delivery), and emergent cesarean delivery. Neonatal complications included SGA, large-for-gestational-age infants (LGA), 1-min Apgar <4, 1-min Apgar score <7, and neonatal intensive care unit (NICU) admission. Pre-eclampsia was diagnosed by blood pressure of ≥140/90 mmHg or a sustained rise (≥30 mmHg in systolic blood pressure and 15 mmHg in diastolic blood pressure) or proteinuria of ≥300 mg per 24 h after the 20th week of gestation. Severe pre-eclampsia was diagnosed by systolic blood pressure of ≥160 mmHg, diastolic blood pressure of ≥110 mmHg, or proteinuria of ≥5 g per 24 h. SGA and LGA were defined as infants whose birth weights were below and above the tenth percentile of birth weights of infants of the same sex delivered at the same gestational age, respectively. The weight gain during pregnancy (kg/week) was calculated by dividing the difference between body weight at delivery and prepregnant body weight by the number of gestational weeks at delivery.

Methods

First, we performed multivariate logistic regression analysis for the association of prepregnant BMIs with antepartum, intrapartum, or neonatal complications among nulliparous and parous women. Second, we divided women into groups by an increase in prepregnant BMI of 2 kg/m^2, and analyzed each group for the association with specific pregnancy complications identified in the above analysis using multivariate logistic regression analysis with dummy variable. Relative risks of complications of various prepregnant BMI groups were evaluated using an adjusted odds ratio (OR). An association was considered significant when statistic analysis showed a p value less than 0.05 and OR was more than 1.5. Third, we analyzed women with low (<18), medium (18–23.9), and high (≥24) prepregnant BMIs for an association of weight gain during pregnancy with antepartum, intrapartum, or neonatal complications using multivariate logistic regression analysis. Each prepregnant BMI group was then analyzed for an association between weight gain of 0.05 kg/week and specific pregnancy complications associated with maternal weight gain using multivariate logistic regression analysis with dummy variable.

Statistical analysis

The statistical analysis was performed using Stat Flex (Version 5) and a p value less than 0.05 was regarded as significant.

Results

Table I presents maternal and neonatal demographic and outcome data for the 21,718 Japanese women. A significant difference between nulliparous and parous women was observed in a number of demographic variables, including not only prepregnant BMI, age, weight gain during pregnancy, and gestational age, but also many pregnancy complications like pre-eclampsia, blood loss of more than 1,000 ml at delivery, vacuum extraction, cesarean delivery, emergent cesarean delivery, SGA, and 1-min Apgar score <4.

Then the association of prepregnant BMI with antepartum, intrapartum, and neonatal complications among women with various prepregnant BMIs was evaluated using multivariate logistic regression analysis in both nulliparous and parous women (data not shown). In nulliparous women, high prepregnant BMIs showed significant association with a number of complications, including pre-eclampsia, blood loss at delivery of more than 1,000 ml, cesarean delivery, emergent cesarean delivery, LGA, and 1-min Apgar score <7. On the other hand, low prepregnant BMIs were significantly associated with SGA. Similarly, in parous women, high prepregnant BMIs were associated with pre-eclampsia, severe pre-eclampsia, blood loss at delivery of more than 1,000 ml, cesarean delivery, LGA, 1-min Apgar <4 and 7, and NICU admission, whereas low prepregnant BMIs showed significant association with SGA.

We divided the women into 7 groups by prepregnant BMI and examined each BMI group for an association with pregnancy complications listed above (Table II). Nulliparous women with prepregnant BMIs 18–19.9, 20–21.9, and 22–23.9 showed the least association with pregnancy complications. On the other hand, prepregnant BMI <18 was associated with only SGA, while prepregnant BMI ≥24 was associated with pre-eclampsia, blood

Table I. Demographic characteristics and complications between nulliparous and parous women (1981–1999)

Demographic characteristics	Nulliparaous (n = 10413)	Parous women (n = 11305)	p
Prepregnant BMI (kg/m2, mean ±SD)	20.5±2.6	21.1±3.0	<.01
Age (y, mean ±SD)	27.8±4.1	30.45±3.9	<.01
Weight gain (kg/wk, mean ±SD)	0.25±0.09	0.24±0.09	<.01
Gestational age (wk, mean ±SD)	39.8±1.2	39.3±1.2	<.01
Preeclampsia	415 (4.0)	259 (2.3)	<.01
Severe preeclampsia	59 (0.6)	47 (0.4)	NS
Blood loss >1000 ml at delivery	63 (0.6)	120 (1.1)	<.01
Vacuum extraction	552 (5.3)	175 (1.5)	<.01
Cesarean delivery	1409 (13.5)	2342 (20.7)	<.01
Emergent cesarean delivery	776 (7.5)	428 (3.8)	<.01
SGA	560 (5.4)	729 (6.5)	<.01
LGA	537 (5.2)	584 (5.2)	NS
1-min Apgar score <4	122 (1.2)	187 (1.7)	<.01
1-min Apgar score <7	99 (1.0)	83 (0.7)	NS
NICU admission	271 (2.6)	284 (2.5)	NS

NS = not significant; SD = standard deviation; SGA = small-for-gestational-age infants; LGA = large-for-gestational-age infants; NICU = neonatal intensive care unit.
Data are presented as n (%).

loss of more than 1,000 ml at delivery, cesarean delivery, emergent cesarean delivery, and LGA. Similar results were obtained with parous women.

Next, we classified the women into three prepregnant BMI groups, low (<18), medium (18–23.9), and high (≥24), and examined whether weight gains were associated with pregnancy and neonatal complications in each group using multivariate logistic regression analysis (data not shown). Among nulliparous women, the low prepregnant BMI group showed significant association between weight gain during pregnancy and pre-eclampsia, cesarean delivery, SGA, LGA, and 1-min Apgar score <4. In the medium prepregnant BMI group, weight gain during pregnancy was associated with pre-eclampsia, severe pre-eclampsia, cesarean delivery, SGA, and LGA. In the high prepregnant BMI group, weight gain during pregnancy was associated with pre-eclampsia, emergent cesarean delivery, SGA, and NICU admission. Among parous women, weight gain during pregnancy showed significant association with SGA and LGA in the low prepregnant BMI group; with cesarean delivery, SGA, LGA, 1-min Apgar score <4, and NICU admission in the medium prepregnant BMI group; and with pre-eclampsia, severe pre-eclampsia, SGA, LGA, and 1-min Apgar score <4 in the high prepregnant BMI group.

Each prepregnant BMI group was classified by weight gain of 0.05 kg/week and examined for the association with pregnancy complications listed above. In the case of nulliparous women, few complications were associated with weight gains of 0.25–0.40 kg/week in the low prepregnant BMI group, 0.20–0.30 kg/week weight gain in the medium prepregnant BMI group, and ≥0.05 kg/week weight gain in the high prepregnant BMI group (Table III). In the case of parous women, increases ≥0.20 kg/week in the low prepregnant BMI group, 0.20–0.30 kg/week in the medium prepregnant BMI group, and 0.05–0.30 kg/week in the high prepregnant BMI group were considered as low-risk weight gains (Table IV).

Discussion

This is the first analysis of prepregnant BMI and weight gain during pregnancy in relation to pregnancy complications and adverse neonatal outcomes among Japanese women. We found that the incidence of antepartum, intrapartum, and neonatal complications varied depending on prepregnant BMI among both nulliparous and parous women. Similar associations have been reported with non-Japanese women (1,2,6–8,12–19).

As there were significant differences between nulliparous and parous women in prepregnant BMI, age, weight gain during pregnancy, gestational age, and frequency of complications, these two groups of women were separately analyzed in the following study.

We found that women with prepregnant BMI 18–23.9 showed few associations with pregnancy complications studied. Women with prepregnant BMI <18 were associated with SGA in both nulliparous and parous groups. Women with prepregnant BMI ≥24 were associated with pre-eclampsia, blood loss of more than 1,000 ml at delivery, cesarean delivery,

Table II. Adjusted odds ratios for selected complications associated with prepregnant BMI among nulliparous and parous women

	BMI						
	<18	18–19.9	20–21.9	22–23.9	24–25.9	26–27.9	28 ≦
Nulliparous women	(n = 1292)	(n = 3849)	(n = 3123)	(n = 1317)	(n = 472)	(n = 167)	(n = 193)
Complication							
Preeclampsia	0.50 (0.32–0.77)	0.56 (0.42–0.75)	1.00	1.14 (0.82–1.60)	2.16 (1.45–3.22)	2.59 (1.41–4.74)	5.74 (3.58–9.21)
Blood loss > 1000 ml at delivery	0.76 (0.55–1.13)	0.86 (0.70–1.07)	1.00	1.15 (0.89–1.48)	1.55 (1.10–2.18)	2.66 (1.70–4.14)	1.60 (1.01–2.53)
Cesarean delivery	0.72 (0.56–1.08)	0.78 (0.67–0.90)	1.00	1.28 (1.07–1.53)*	1.54 (1.20–1.99)	2.97 (2.09–4.23)	3.65 (2.46–5.43)
Emergent cesarean delivery	0.78 (0.56–1.03)	0.77 (0.63–0.93)	1.00	1.12 (0.90–1.42)	1.20 (0.86–1.68)	1.82 (1.15–2.90)	3.98 (2.55–6.42)
SGA	1.71 (1.30–2.26)	1.33 (1.07–1.67)*	1.00	0.87 (0.63–1.21)	1.05 (0.67–1.65)	0.86 (0.41–1.81)	0.80 (0.41–1.55)
LGA	0.39 (0.27–0.57)	0.77 (0.66–0.91)	1.00	1.14 (0.93–1.40)	1.09 (0.80–1.48)	2.10 (1.39–3.17)	1.79 (1.02–3.14)
1-min Apgar score <7	1.02 (0.53–1.96)	0.69 (0.53–0.92)	1.00	1.16 (0.84–1.61)	1.12 (0.70–1.79)	0.70 (0.30–1.64)	1.67 (0.93–2.95)
Parous women	(n = 1067)	(n = 3461)	(n = 3521)	(n = 1829)	(n = 717)	(n = 337)	(n = 373)
Complication							
Preeclampsia	0.26 (0.10–0.59)	0.56 (0.38–0.82)	1.00	1.21 (0.83–1.74)	2.08 (1.35–3.22)	2.96 (1.75–5.01)	4.74 (3.22–7.00)
Severe preeclampsia	0.87 (0.24–3.14)	0.92 (0.39–2.20)	1.00	2.13 (0.91–4.98)	1.85 (0.56–6.01)	3.78 (1.19–11.97)	2.22 (0.61–8.04)
Blood loss > 1000 ml at delivery	0.88 (0.66–1.17)	1.02 (0.85–1.23)	1.00	1.27 (1.03–1.56)*	1.42 (1.07–1.89)*	1.26 (0.86–1.85)	1.95 (1.55–2.99)
Cesarean delivery	0.79 (0.57–1.11)	1.05 (0.81–1.35)	1.00	1.29 (0.98–1.77)	1.46 (1.00–2.13)*	1.46 (0.98–2.38)	2.59 (1.15–4.08)
SGA	2.48 (1.94–3.16)	1.18 (0.97–1.43)	1.00	0.61 (0.47–0.81)	0.42 (0.27–0.64)	0.35 (0.19–0.64)	0.26 (0.12–0.48)
LGA	0.39 (0.25–0.61)	0.59 (0.46–0.77)	1.00	1.16 (0.89–1.54)	1.87 (1.35–2.59)	3.77 (2.58–5.52)	5.09 (3.18–7.88)
1-min Apgar score <4	1.13 (0.51–2.39)	1.28 (0.82–1.99)	1.00	1.58 (0.97–2.57)	2.49 (1.39–4.45)	3.58 (1.82–7.02)	3.85 (1.80–8.70)
1-min Apgar score <7	0.97 (0.51–1.84)	1.84 (0.99–3.74)	1.00	2.08 (0.92–4.69)	5.43 (2.37–12.42)	3.92 (1.29–11.97)	6.67 (3.16–13.40)
NICU admission	0.93 (0.61–1.43)	0.76 (0.55–1.06)	1.00	0.64 (0.41–1.01)	0.77 (0.42–1.41)	2.49 (1.47–4.23)	1.77 (1.03–3.05)

Data are presented as odds ratio (95% confidence interval).
Underlined data: $p < 0.05$ and odds ratio $\geqq 1.5$.
*: $p < 0.05$ and odds ratio < 1.5.

Table III. Adjusted odds ratios for selected complications associated with weight gain during pregnancy in each BMI group among nulliparous women

	Weight gain (kg/w)						
	<0.15	0.15–0.20	0.20–0.25	0.25–0.30	0.30–0.35	0.35–0.40	0.40 ≦
Low BMI Group	(n = 79)	(n = 132)	(n = 259)	(n = 311)	(n = 252)	(n = 147)	(n = 112)
Complication							
Preeclampsia	1.06 (0.21–5.46)	0.57 (0.11–2.90)	0.84 (0.25–2.80)	1.00	0.22 (0.03–1.87)	2.72 (0.84–8.74)	3.45 (1.04–11.50)
Cesarean delivery	0.49 (0.17–1.39)	0.91 (0.43–1.89)	0.86 (0.46–1.60)	1.00	0.87 (0.43–1.74)	1.10 (0.48–2.54)	2.30 (1.06–4.98)
SGA	6.20 (2.72–14.09)	2.58 (1.14–5.87)	2.46 (1.19–5.08)	1.00	1.55 (0.67–3.58)	1.03 (0.34–3.12)	2.02 (0.74–5.52)
LGA	0.76 (0.25–2.31)	1.75 (0.86–3.56)	1.36 (0.73–2.54)	1.00	1.73 (0.91–3.28)	1.26 (0.53–3.00)	2.25 (1.03–4.94)
1-min Apgar score <4	12.24 (2.04–73.43)	2.93 (0.45–19.14)	2.49 (0.44–14.2)	1.00	1.41 (0.19–10.59)	-	1.18 (0.10–14.24)

	Weight gain (kg/w)						
	<0.15	0.15–0.20	0.20–0.25	0.25–0.30	0.30–0.35	0.35–0.40	0.40 ≦
Medium BMI Group	(n = 753)	(n = 1236)	(n = 2051)	(n = 2046)	(n = 1222)	(n = 588)	(n = 393)
Complication							
Preeclampsia	0.61 (0.35–1.05)	0.46 (0.28–0.78)	0.73 (0.49–1.08)	1.00	1.17 (0.78–1.74)	1.92 (1.24–2.98)	3.53 (2.31–5.39)
Severe preeclampsia	1.39 (0.40–4.99)	2.01 (0.69–5.81)	0.17 (0.02–1.42)	1.00	1.98 (0.66–5.90)	7.21 (2.68–19.40)	9.58 (3.44–26.64)
Cesarean delivery	1.28 (0.98–1.66)	0.86 (0.68–1.10)	0.84 (0.68–1.05)	1.00	1.10 (0.87–1.39)	1.61 (1.21–2.14)	1.68 (1.22–2.30)
SGA	2.64 (1.88–3.71)	1.60 (1.15–2.23)	1.39 (1.03–1.87)*	1.00	0.96 (0.66–1.39)	0.84 (0.51–1.37)	0.34 (0.16–0.76)
LGA	1.40 (0.86–2.79)	1.16 (0.89–1.51)	1.41 (1.31–1.76)*	1.00	1.76 (1.38–2.23)	2.34 (1.77–3.10)	2.58 (1.71–3.89)

	Weight gain (kg/w)						
	<0.05	0.05–0.10	0.10–0.15	0.15–0.20	0.20–0.25	0.25–0.30	0.30 ≦
High BMI Group	(n = 67)	(n = 84)	(n = 87)	(n = 134)	(n = 153)	(n = 112)	(n = 195)
Complication							
Preeclampsia	0.61 (0.21–1.82)	0.30 (0.08–1.14)	0.21 (0.04–1.01)	1.00	0.82 (0.34–1.98)	1.54 (0.66–3.58)	1.82 (0.86–3.89)
Emergent cesarean delivery	0.53 (0.21–1.29)	0.58 (0.24–1.40)	0.83 (0.37–1.87)	1.00	0.94 (0.47–1.88)	1.05 (0.50–2.21)	1.13 (0.58–2.19)
SGA	7.06 (2.11–23.61)	0.70 (0.13–3.85)	1.51 (0.38–6.03)	1.00	2.03 (0.64–6.43)	2.03 (0.60–6.86)	0.56 (0.14–2.26)
NICU admission	0.61 (0.12–3.13)	-	1.18 (0.30–4.62)	1.00	0.31 (0.06–1.63)	1.34 (0.40–4.56)	1.61 (0.53–4.83)

Data are presented as odds ratio (95% confidence interval).
Underlined data: $p < 0.05$ and the odds ratio ≧1.5.
*:$p < 0.05$ and the odds ratio <1.5.