

Maternal weight gain and fetal growth

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Abstract

Extrinsic factors such as maternal nutrition may affect fetal growth giving rise to short and long-term consequences. The present study sets out to explore the inter-relationship between antenatal weight gain, in part dependant on maternal nutrition, and fetal growth as determined by birth weight. The study population included 21,573 women who delivered their singleton child at term during 1999-2006. This population was subdivided into 16 subgroups according to Body Mass Index (BMI) and antenatal weight gain. The study confirms that irrespective of maternal BMI, there is a statistically significant trend towards low birth weight with decreasing antenatal weight gain and conversely macrosomia with increasing antenatal weight gain.

Key Words

Maternal nutrition, fetal complications, macrosomia, low birth weight

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Introduction

The eventual birth weight of the infant is dependant on a number of intrinsic fetal and maternal factors which determine growth potential and placental function. In addition extrinsic factors have been shown to play a role in antenatal fetal growth and eventual birth weight. Maternal nutrition as a determinant of infant birth weight has long been recognised, but was particularly emphasised by The Netherlands famine during the winter of 1944-45, when a situation of acute short-term food restriction was noted to have predisposed to low birth weight infants.¹ Similarly, increased weight gain during pregnancy had been shown to affect eventual infant birth weight in obese women.² Antenatal weight gain in the mother is partly dependant on maternal nutrition. This study attempts to investigate the inter-relationship between maternal antenatal weight gain to fetal growth.

Material and Methods

The study utilised the national database of all maternities delivering in the Maltese Islands during 1999-2006. Cases with absent maternal data regarding height, pre-pregnancy weight, and weight at term, and infant birth weight data were excluded from the study. Since preterm and multiple births influence the eventual birth weight, these cases were also excluded from the analysis. The total number of patients analysed in the study thus amounted to 21,573 cases of singleton maternities delivering at term, accounting for 66.7% of the total maternities during the period. To enable the assessment of maternal weight gain influence irrespective of original maternal body weight, the population was subdivided into four basic groups on the basis of their Body Mass Index [BMI]: Group A – BMI <20 kg/m² = 1355; Group B – BMI 20-24 kg/m² = 9313; Group C – BMI 25-29 kg/m² = 6736; and Group D – BMI ≥30 kg/m² = 4169. Each group was further subdivided into different antenatal weight gain groups – Subgroup 1 – <5 kg, Subgroup 2 – 5-9 kg, Subgroup 3 – 10-14 kg, and Subgroup 4 – ≥15 kg [Table 1]. Statistical analysis was carried out using the Chi Square test and student t test as appropriate.

Results

The mean antenatal weight gain appeared to be statistically decreased with increasing BMI values from 13.25 ± 5.41kg in the lean group to 10.32 ± 5.94kg [p<0.0001] in the obese women. Similarly, the infant's mean birth weight increased progressively

with increasing BMI from $3140 \pm 415\text{g}$ in the lean BMI group to $3416 \pm 463\text{g}$ in the obese group. The incidence of macrosomia increased progressively through increasing maternal BMI from 2.1% in the lean group to 10.2% in the obese group [$p < 0.0001$]; while the incidence of small for dates infants increased with decreasing maternal BMI [$p < 0.0001$] from 1.8% in the obese group to 5.6% in the lean group [Table 2].

Irrespective of the maternal BMI, the infant's birth weight was further dependant on the overall antenatal weight gain during pregnancy. Thus the mean birth weight increased progressively in all BMI groups with increasing antenatal weight gain [Table 3 & Figure 1]. The increase in mean infant birth weight with increasing maternal BMI and antenatal weight gain is also reflected by the statistically significant increase in the rate of macrosomia $>4000\text{g}$ rate [Table 3 & Figure 2]. There appears conversely to be a statistically significant inter-relationship between decreasing maternal BMI and antenatal weight gain values to an increase in small for gestational age infants weighing $<2500\text{g}$. This phenomenon is not noticeable in obese women with a BMI $>30\text{ kg/m}^2$ [Table 3 & Figure 2].

Table 1: Study population distribution by BMI and antenatal weight gain

Study groups	Body Mass Index	Antepartum Weight gain	No.	%
A1	<20 kg/m ²	0-4 kg	22	1.6
A2		5-9 kg	270	19.9
A3		10-14 kg	547	40.4
A4		$\geq 15\text{ kg}$	516	38.1
A1-4		Total	1355	
B1	20-24 kg/m ²	0-4 kg	346	3.7
B2		5-9 kg	1877	20.2
B3		10-14 kg	3654	39.2
B4		$\geq 15\text{ kg}$	3436	36.9
B1-4		Total	9313	
C1	25-29 kg/m ²	0-4 kg	440	6.5
C2		5-9 kg	1749	20.2
C3		10-14 kg	2361	39.2
C4		$\geq 15\text{ kg}$	2186	36.9
C1-4		Total	6736	
D1	>30 kg/m ²	0-4 kg	650	15.6
D2		5-9 kg	1378	33.1
D3		10-14 kg	1232	29.6
D4		$\geq 15\text{ kg}$	909	21.8
D1-4		Total	4169	
A-D1	Total population	0-4 kg	1458	6.8
A-D2		5-9 kg	5274	24.4
A-D3		[singleton, term	7794	36.1
A-D4		maternities]	7047	32.7
A-D1-4		Total	21573	

Discussion

The influence of maternal nutrition on fetal growth has long been assumed. In a paper read before the Medical Society of London in 1788, to allow for sub-optimal growth in cases of past cephalo-pelvic disproportion, James Lucas advocated "temperance in diet, a diminution in the usual quantity and a change in the quality of food, an increase in exercise, the occasional loss of a few ounces of blood, and the moderate use of cooling aperients. This regimen was more strictly enjoined in the last months of pregnancy from a persuasion that an observance of it in the former months would avail little if neglected in the latter".³ This idea was taken up and promulgated by Ludwig Prochownik who in 1889 proposed a basic high-protein and low-carbohydrate regimen in the last six weeks of pregnancy to control the size of the child. The inter-relationship between maternal diet and fetal growth was studied scientifically by GFD Smith in 1919. Smith found that women with poor nutritional status delivered smaller babies. A significantly lowered mean birth weight of the order of 250g was noted in

Figure 1: Mean birth weight by maternal BMI and antenatal weight gain [statistical value using t-test comparing to weight gain Subgroup 2]

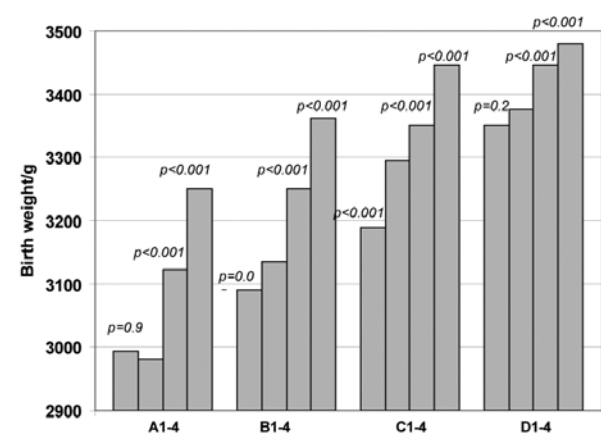
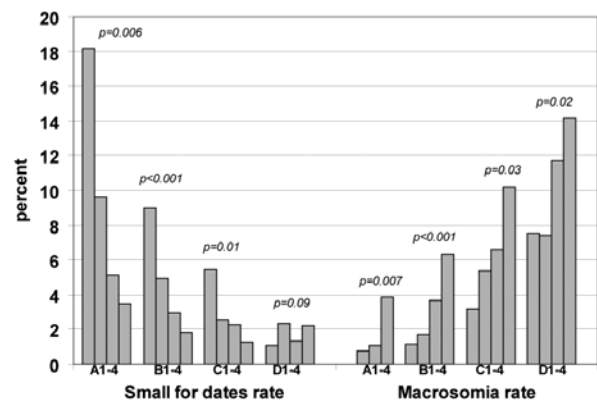


Figure 2: Small for dates and macrosomia rates by maternal BMI and antenatal weight gain



the Dutch population who experienced severe famine during the winter of 1944-45. The lowest median birth weight was reached in this population when the food restriction affected the whole second half of gestation.^{1,5} Maternal malnutrition has further been shown in animal studies to be associated with fetal growth retardation, not simply resulting from a direct effect of the lack of nutrients in the maternal blood but also from the interference with the normal physiological adjustments of pregnancy.⁶ The present study has confirmed the inter-relationship between low birth weight and poor antenatal weight gain partly contributed by poor maternal nutrition. This observation could be noted

in all women irrespective of body habitus, except in the obese group of women. Maternal antenatal weight gain in pregnancy is the sum product of various components including the weight gain related to the products of conception, the anatomical and physiological changes that occur in the mother throughout pregnancy, and changes that occur in the mother's fat stores. The absence of any statistically significant inter-relationship between poor antenatal weight gain and low birth weight in the previously obese women could be attributed to this group of individuals having a sufficient store of nutrients that could be made available to the developing fetus. The obese pregnant

Table 2: Outcomes by maternal BMI

Outcomes	A - Lean BMI <20 N = 1355	B - Normal BMI 20-24 N = 9313	C - Overweight BMI 25-29 N = 6736	D - Obese BMI >30 N = 4169
BMI [kg/m ²] Mean ± sd	18.42 ± 0.91	22.24 ± 1.36	26.64 ± 1.40	33.8 ± 3.94
Antenatal weight gain [kg] Mean ± sd	13.47 ± 5.17 p=0.16	13.25 ± 5.41	12.38 ± 5.75 p<0.0001	10.32 ± 5.94 p<0.0001
Infant birth weight [g] Mean ± sd	3140 ± 415 p<0.0001	3263 ± 416	3357 ± 438 p<0.0001	3416 ± 463 p<0.0001
Small for gestation [<2.5 kg] p<0.0001	76 (5.6%)	297 (3.2%)	150 (2.2%)	75 (1.8%)
Macrosomia [≥4.0 kg] p<0.0001	28 (2.1%)	389 (4.2%)	487 (7.2%)	424 (10.2%)

Table 3: Infant birth weight by maternal BMI and antenatal weight gain

Study subgroup	Small for dates Birth weight <2.5 kg		Macrosomia Birth weight ≥4.0 kg		Birth Weight grams	
	no	Rate %	no	Rate %	mean	sd
A1	4	18.2	0	0.0	2994	478
A2	26	9.6	2	0.7	2980	398
A3	28	5.1	6	1.1	3123	389
A4	18	3.5	20	3.9	3250	415
B1	31	9.0	4	1.2	3091	429
B2	93	5.0	32	1.7	3136	392
B3	109	3.0	135	3.7	3250	409
B4	64	1.9	218	6.3	3362	410
C1	24	5.5	14	3.2	3189	454
C2	44	2.5	94	5.4	3295	426
C3	54	2.3	156	6.6	3351	423
C4	28	1.3	223	10.2	3446	440
D1	7	1.1	49	7.5	3351	435
D2	32	2.3	102	7.4	3376	458
D3	16	1.3	144	11.7	3447	470
D4	20	2.2	129	14.2	3481	468

woman has further been repeatedly shown to gain less weight during pregnancy than their lean counterparts.

Poor intra-uterine nutrition thus appears to predispose towards retardation of fetal growth. Conversely, increased intra-uterine nutrition as occurs in the diabetic or obese mother predisposes to increase in fetal growth and a tendency towards macrosomia.⁷ The relationship between overeating and excessive birth weight was first proposed by Hugo Ehrenfest in 1919.⁴ The present study has further indicated that the tendency towards increase in fetal growth is determined by antenatal maternal nutrition irrespective of maternal body habitus, with a tendency towards macrosomia being observed with increasing antenatal weight gain in all BMI groups. While the traditional assumption is that macrosomia is predisposed by fetal hyperglycaemia causing fetal hyperinsulinism promoting growth, the role of other nutrients cannot be completely ignored and needs to be more clearly defined.

Extremes of fetal growth, both low and high birth weight infants, have significant short-term consequences resulting from an altered metabolic process predisposing to hypoglycaemia in the early neonatal period. This problem is a transient one that needs to be actively looked for to prevent disastrous consequences. However, extremes of fetal growth have been shown to have long-term consequences to health predisposing the child to a greater likelihood of developing adult-onset disease including type-2 diabetes mellitus.^{8,9} In view of the consequences

of extremes of fetal growth, it is essential that all pregnant women particularly those who are extremely lean or those who are significantly obese should receive correct nutritional advice in relevance to their BMI status to reduce under or overgrowth of the fetus.

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