

Diet in pregnancy

3.* Diet in relation to the course and outcome of pregnancy

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In the first paper of this series (Thomson, 1958) I described a survey of the diets of 489 primigravidae living in Aberdeen during the years 1950-3. A straightforward comparison of the nutritive value of the diets with 'recommended allowances' for pregnant women showed that many of the diets were substandard, but a more detailed analysis (Thomson, 1959*a*) led to the conclusion that they were not likely to cause abnormalities in the course and outcome of pregnancy. Yet the range of calorie intake was wide in all social classes, which might have some significance for health during pregnancy.

These conclusions have now been tested by comparing the clinical histories of the subjects with the nutritive values of the diets they took during pregnancy. The results as a whole are discussed in relation to past studies of the influence of diet on the course and outcome of pregnancy.

METHOD

The method by which the nutritive values of the diets were estimated has been described previously (Thomson, 1958). The subjects were grouped according to the occupations of their husbands, thus:

Social class A: 'White-collar' (non-manual) occupations (corresponding to the Registrar-General's classes I and II, with non-manual workers from class III);

Social class B: Skilled manual occupations (Registrar-General's class III, less non-manual occupations);

Social class C: Semi-skilled and unskilled manual occupations (Registrar-General's classes IV and V).

Of the total of 489 subjects, 452 were supervised throughout in the out-patient clinic and hospital ward of a single obstetric unit, in which high and uniform standards of diagnosis and of clinical recording were maintained. The remaining thirty-seven subjects were mostly under private medical supervision and antenatal records were not kept systematically for all of them, but these patients were confined in one private maternity hospital and the matron recorded specially detailed histories for the purposes of the survey.

At the time of the survey, no attempt was being made to regulate the diets or the

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amounts of weight gained during pregnancy. Under the provisions of the National Welfare Foods scheme, all patients were offered concentrates containing ascorbic acid and vitamins A and D, and all were able to buy 1 pint of milk daily at a cheap rate. These 'extras' were by no means always or regularly used (Marr, Hope, Stevenson & Thomson, 1955).

During and after the time that these subjects were being studied, wide-ranging epidemiological investigations were being made on many of the phenomena of maternity. The reviews of Baird (1952*a*) and Baird & Thomson (1954) give general accounts of some of these parallel studies, and further references are given below under the appropriate headings. As a result, it became obvious that the incidence of many of the abnormalities of pregnancy, labour and lactation is greatly affected by factors other than the diet during pregnancy, notably parity, age, physique and standard of medical care. A correlation between the nature of the diet and the incidence of an abnormality is not proof of cause and effect; the association may have a common basis, for example in maternal physique or social circumstances. Inferences as to dietary causation may, therefore, require further support from knowledge of physiology, or from the results of a feeding experiment.

In this investigation, all subjects were pregnant for the first time and had single babies. It can be assumed that standards of medical care were reasonably uniform. Social influences other than diet have been allowed for to some extent by treating the three social classes separately. Other breakdowns, e.g. by age, have been made when it appeared useful.

Many different ways of comparing the dietary with the clinical data have been studied, and it did not appear that any one approach was more fruitful than another. For the sake of simplicity, it was finally decided to classify the records according to the clinical features, and then to examine the composition of the diets taken by women in the several clinical groups.

RESULTS AND DISCUSSION

Normal pregnancy

It is impossible to draw a hard-and-fast line which differentiates 'normal' from 'abnormal' pregnancy. Among those subjects in whom no specific abnormality was diagnosed, some gave a clearer impression of general well-being than others. Some minor disorders, such as nausea in early pregnancy, varicose veins, and tiredness in late pregnancy, were so common that to use them as criteria of abnormality would be to regard most pregnancies as pathological. The group of 'normal' pregnancies used in the analysis was defined as follows. There was no specific abnormality of pregnancy that required treatment; labour was completed spontaneously within 24 h; the baby was in good condition when delivered and weighed 6 lb (2.7 kg) or more at birth; after a normal puerperium, mother and baby were discharged together, the baby being fully breast fed at the time of leaving the hospital.

Table 1 compares the mean nutritive values of the diets taken by 'normal' and 'abnormal' subjects, within each social class as well as for all classes. The differences were small and not statistically significant. The abnormal groups all had a slightly

Table 1. Daily intake of nutrients by 'normal' and 'abnormal' pregnant women, and of subjects with pre-eclamptic toxæmia (P.E.T.) and other hypertensive abnormalities (see p. 512), by social class

Clinical state	Subjects				Mean daily intake							
	No.	Mean age (years)	Mean height (cm)	Mean weight (kg)	Calories (kcal)	Protein (g)	Calcium (g)	Thiamine (mg)	Riboflavin (mg)	Nicotinic acid (mg)	Vitamin A (i.u.)	Ascorbic acid (mg)
Normal	38	25.8	163.3	67.7	2615	79.0	1.16	1.21	2.01	11.9	10 200	79
Abnormal	63	27.0	160.3	64.2	2644	81.2	1.20	1.22	2.07	12.0	9 600	79
P.E.T.	6	29.5	157.0	61.5	2762	81.2	1.30	1.20	1.99	11.1	10 300	70
Hypertensive	23	27.5	162.6	68.5	2758	85.5	1.23	1.34	2.16	12.9	9 900	82
					Social class A							
Normal	38	23.1	158.2	61.7	2503	76.5	0.99	1.16	1.81	11.4	7 700	67
Abnormal	71	24.5	158.3	63.5	2531	79.4	1.10	1.19	1.93	11.3	8 300	65
P.E.T.	7	26.6	156.9	68.6	2642	85.5	1.20	1.22	2.01	11.1	8 000	56
Hypertensive	36	23.6	159.8	65.6	2593	81.2	1.15	1.24	2.00	11.8	9 000	73
					Social class B							
Normal	121	22.0	157.7	61.0	2346	71.2	0.87	1.16	1.76	11.5	7 700	60
Abnormal	158	23.7	157.7	61.7	2361	72.2	0.89	1.15	1.73	11.8	7 400	61
P.E.T.	14	23.8	158.1	61.2	2410	72.1	0.91	1.13	1.73	11.6	7 700	58
Hypertensive	59	23.6	158.8	64.4	2429	75.4	0.94	1.20	1.84	12.3	7 900	64
					Social class C							
					All social classes (means not weighted)							
Normal	197	22.9	158.9	62.4	2428	73.7	0.95	1.17	1.82	11.5	8 200	65
Abnormal	292	24.6	158.4	62.7	2468	75.9	1.01	1.18	1.85	11.7	8 100	66

greater mean calorie intake, associated with which was a slightly greater intake of several nutrients. These small but consistent differences seemed to be attributable to the inclusion of cases of pre-eclampsia in the abnormal group.

Pre-eclampsia

The definition of pre-eclampsia is notoriously difficult; here it was defined in terms of the level of blood pressure in late pregnancy and the occurrence of proteinuria (Nelson, 1955). Though oedema is a common feature it is difficult to define precisely and is not used in the classification. Pre-eclampsia of moderate or severe degree (pre-eclamptic toxæmia, P.E.T.) means a rise of the diastolic blood pressure in late pregnancy to 90 mm Hg or more, together with definite proteinuria (0.25 g/l. or more) not attributable to a urinary infection. Nelson classified cases showing a similar rise of blood pressure without proteinuria as 'mild pre-eclampsia', but I prefer to label them 'hypertensive', which leaves the question open as to whether or not they represent true pre-eclampsia. There were two cases of eclampsia (P.E.T. with convulsions) and these have been included with P.E.T. Nelson's analyses and those of Thomson & Billewicz (1957) show that the incidence of pre-eclampsia in primigravidae is not greatly influenced by maternal social class, stature or age, except that hypertension in late pregnancy, without proteinuria, occurs more commonly in primigravidae aged 30 years or over than in younger primigravidae.

Table 1 gives the mean nutritive values of the diets in the two hypertensive groups and compares them with the means obtained in normal pregnancy, within social classes. The diets of the two groups with hypertension were similar, and both had consistently higher mean calorie values than the diets of normal subjects. Further analysis of the clinical groups confirmed that the 'excess' of calories taken by the subjects with pre-eclampsia was unlikely to be the result of sampling fluctuations, and that it was not accounted for by, for example, an unusually high intake of carbohydrate (Table 2).

Table 2. *Percentage contributions to calorie supply of protein, carbohydrate and fat in three clinical groups of pregnant women*

Group	Protein	Carbohydrate	Fat
Normal pregnancy	12.1	49.6	38.2
P.E.T.	12.2	50.4	37.4
Hypertensive	12.4	49.5	38.0

The relatively high mean calorie intake in pre-eclampsia is consistent with the well-known fact that women with pre-eclampsia tend to gain weight excessively (Chesley, 1944; Thomson & Billewicz, 1957). On the other hand, the findings do not support the idea, which has had wide currency, that pre-eclampsia is associated with high-carbohydrate, low-protein diets (see, for example, Hamlin, 1952). Nor do they support suggestions that diets in pre-eclampsia tend to be low in nicotinic acid (Hobson, 1948; McGanity, Cannon, Bridgforth, Martin, Densen, Newbill, McClellan, Christie, Peterson & Darby, 1954). Not unexpectedly, our pre-eclamptic patients tended to take slightly more of most nutrients than normal patients, because of the positive correlations between calorie intake and intake of nutrients (Thomson, 1959*a*).

Our attention was engaged for a time by the fact that the mean ascorbic-acid intake of subjects who developed P.E.T. was noticeably lower than that of normal patients, at least in social classes A and B. Further breakdown of the data according to the heights and calorie intakes of the subjects failed to confirm that this finding was 'stable'. But it was also found that the differences became slightly greater and more stable when ascorbic acid derived from concentrates was omitted from the analysis and only that derived from food was considered. These curious and inconclusive findings would not be mentioned at all were it not for the suspicion that ascorbic acid may be concerned in the occurrence of pre-eclampsia. Hipsley (1953) attributes a relatively high incidence of this disease in Indians living in Fiji compared with that in native Fijians to a lower fibre intake; but, on his own data, the difference might equally be explained by a lower ascorbic-acid intake. Hipsley further adduces, in support of his fibre hypothesis, the decrease in the incidence of pre-eclampsia which occurred in Holland during the war years, 1940-5; the Dutch diet came to contain more potatoes and turnips, which would increase the supply of ascorbic acid as well as of fibre. McGanity *et al.* (1954) also found the ascorbic-acid intakes of their pre-eclamptic subjects to be slightly lowered (significantly during the second trimester only), which they consider to be a result of treatment rather than a cause of the illness (Martin, Bridgforth, McGanity & Darby, 1957). Pre-eclampsia is not at all likely to be a simple state of ascorbic-acid deficiency; nevertheless, the notion that ascorbic acid may be involved should perhaps be tested experimentally.

Pre-eclampsia and gain of body-weight during pregnancy

It has for long been known that women who develop pre-eclampsia tend to put on more weight during pregnancy than others, and Thomson & Billewicz (1957) have shown that primigravidae who gain not more than 1 lb (0.45 kg)/week have a lower incidence of the disease than those who gain more. Consequently, the finding that women with pre-eclampsia tended to have a high calorie intake is not unexpected. The relation between weight gain, pre-eclampsia and calorie intake was therefore investigated.

Sufficient body-weight records were available for 412 of the 489 subjects. Most of the others were not weighed regularly during pregnancy, and a few were medically examined for the first time late in pregnancy, their previous weights being unknown; women delivered before the 34th week of pregnancy were excluded also. The 412 weight records enabled estimates to be made of the mean weekly gains during the 16-week period from the 20th to the 36th weeks of pregnancy. The means for the three social classes did not differ greatly, being 1.04, 1.08 and 0.99 lb/week (0.47, 0.49 and 0.45 kg/week) for classes A, B and C, respectively; these values are similar to those for the much larger groups described by Thomson & Billewicz (1957).

Table 3 shows, for all subjects, the mean daily calorie intake and the incidence of P.E.T. and other hypertensive complications according to the mean weekly gain of weight. As expected, the calorie intake and the incidence of pre-eclampsia increased with rising rate of gain. Similar patterns were found when each social class was examined separately.

Table 3. *Calorie intakes and incidence of pre-eclampsia in 412 pregnant women arranged according to mean weekly weight gain during the period 20-36 weeks of pregnancy*

Mean weight gain (lb/week)	No. of subjects	Mean daily calorie intake (kcal)	Incidence of P.E.T. (%)	Incidence of hypertension (%)
Under 0.5	26	2228	2.9	13.5
0.5-0.75	78	2273		
0.75-1.00	112	2416	4.4	27.0
1.00-1.25	92	2478		
1.25-1.50	51	2498	13.3	33.7
1.50-1.75	31	2656		
1.75 and over	22	2904		

Table 4. *Weight gain of the women during the period 20-36 weeks of pregnancy, according to calorie intake*

Calorie intake (kcal/day)	No. of subjects	Mean weight gain		Percentage of subjects	
		lb/week	kg/week	With P.E.T.	Hypertensive
Under 1800	38	0.78	0.35	3.8	22.8
1800-2200	94	0.95	0.43		
2200-2600	131	1.00	0.45	6.1	22.9
2600-3000	93	1.08	0.49	8.7	29.5
3000 and over	56	1.24	0.56		

Table 4 presents the data the other way round and shows the mean gain of weight and the incidence of pre-eclampsia at each level of calorie intake.

A regression analysis which took into account the 'initial weight' (the weight at the 20th week of pregnancy) gave the following results. The coefficient of multiple correlation of weight gain (G) on initial weight (W) and calorie intake (C) was 0.33. The partial correlation coefficients were $r_{GC.W} = 0.280$ and $r_{GW.C} = 0.135$. The correlation between the calorie intake and weight gain was 0.30. The regression coefficients for each of the three social classes did not differ significantly from each other. Analysis of variance showed that both calorie intake and initial weight contributed significantly to the variation of weight gain.

A correlation coefficient of 0.3 between calorie intake and gain in weight during pregnancy is probably as large as could be expected in a field study of this kind. There is no reason to believe that total calorie intake should bear any close relation to the proportion available for formation of new tissue; indeed, the variation of total intake is probably determined mainly by activity. Again, differences of weight gained are not wholly due to differences in the amount of new tissue laid down, since much of the weight gained during pregnancy is attributable to storage of water (e.g. increase of plasma volume and of extracellular fluid). Storage of water *per se* probably does not involve the expenditure of much energy. There are, of course, many sources of variation, other than activity and retention of water, which will also tend to diminish the correlation between gain in weight and supply of energy. The regression coefficient of weight gain on calorie intake was small, each increase of 100 kcal/day being associated with a mean weight increase of 0.02 lb/week (0.009 kg/week). This gain is

about one-tenth of the amount that would be expected if 100 kcal were all utilized to form fatty tissue.

It is generally agreed that pre-eclampsia is associated with excess storage of water, which must account for much of the excessive weight gain in that disease. But since water storage, as such, probably does not involve the expenditure of much energy, the fact that an increased intake of energy was associated with an increased incidence of pre-eclampsia (Table 4) may indicate that the mother stores extra 'solid tissue' as well as extra water in pre-eclampsia. Some additional support for this idea has been given in a preliminary communication (Thomson & Billewicz, 1955). Most of the excess water stored during pregnancy appears to be lost within a few days after delivery, since the maternal body-weight in the puerperium usually falls sharply during the 1st week and then much more slowly. Thus, by subtracting the body-weight in early pregnancy from that near the end of the puerperium, an estimate is obtained of the net gain of maternal weight during pregnancy, which, if all excess water has indeed been lost, represents an increase of maternal tissue. A preliminary estimate made in this way indicates that the mean net gain in normal pregnancy is about 4 kg and in pre-eclampsia about 5 kg.

Many obstetricians try to limit the amount of weight that their patients gain by prescribing a low-calorie diet, as a means of preventing pre-eclampsia. Striking results have been claimed, e.g. by Hamlin (1952). But no controlled test of calorie restriction *per se* has been made. A fully controlled trial is probably impossible. In clinical practice, restriction of diet always means alteration of the composition of the diet; indeed, a high-protein, low-calorie diet is usually prescribed. Probably of greater importance, careful antenatal supervision of diet and weight gain almost inevitably connotes intensification of medical supervision in general, which by itself may reduce the incidence and severity of eclampsia and pre-eclampsia (Thomson & Billewicz, 1957). Nevertheless, the present findings indicate that overeating may play some part in the aetiology or the development of pre-eclampsia. But a conclusion that overeating is a major cause would probably not be warranted. The range of calorie intake and of weight gain was wide in both the pre-eclamptic and the normal groups, and there was a large overlap. Many individuals who developed pre-eclampsia gained less weight than the average and took diets of relatively low calorie value. Conversely, many who remained normal took high-calorie diets and gained more than the average amount of weight.

The results reported here refer to primigravidae whose diets and gains of weight were not being controlled. Since regulation of weight is now commonly attempted during antenatal care it would not be easy to repeat these observations. When regulation is practised the picture is altered. Thus, McGanity *et al.* (1954) found the calorie intakes of their pre-eclamptic subjects to be less than average, owing, in their opinion, to the fact that patients who gained weight excessively were advised to eat less.

Duration of gestation

The duration of gestation was estimated in the usual way from the date of the last menstrual period. The accuracy of the estimate was checked against such clinical features as height of the uterine fundus and the time at which foetal movements were first noticed. No 'correction' was made if the birth weight appeared to be unusually low or high for the duration of gestation.

Table 5 gives the mean calorie values by duration of gestation at delivery and by social class. The means give some indication of increased calorie intake with increasing gestation period, but the differences were not striking. The same was true of nutrients.

The correlation coefficient between length of gestation and calorie intake, 0.087, was not statistically significant.

Table 5. *Mean calorie intake of the pregnant women (kcal/day) by duration of gestation and social class*

(Numbers of subjects are shown in parentheses)

Social class	Duration of gestation (weeks)			
	37 and under	38-39	40-41	42 and over
A	2550 (4)	2660 (18)	2690 (59)	2421 (18)
B	2802 (7)	2354 (19)	2478 (60)	2688 (23)
C	2125 (21)	2341 (58)	2351 (143)	2467 (53)
All classes	2333 (32)	2404 (95)	2457 (262)	2512 (94)

Birth weight

As Table 6 shows, birth weight tended to increase with calorie intake. Similar trends were found within each social class, but it appeared that social class had a greater influence upon birth weight than calorie intake.

In the statistical analysis, the duration of the gestation was not included as an independent variable, though it is obviously closely associated with birth weight. Duration of gestation may be regarded as part of the process that gives rise to birth weight rather than as a primary 'factor', and Table 6 indicates clearly that diet does not influence birth weight appreciably through an effect on the gestation period.

Table 6. *Mean birth weight of infant according to calorie intake of the mother*

Calorie intake (kcal/day)	No. of subjects*	Mean birth weight (kg)
Under 1800	47	3.09
1800-2200	106	3.19
2200-2600	156	3.21
2600-3000	104	3.21
3000 and over	66	3.33

* Three macerated stillbirths and seven malformed infants have been excluded.

Birth weight depends to an important extent on maternal size, which is in turn associated with social class. It was found that both maternal height and weight were associated with birth weight, but height added little to the prediction obtained when weight was used alone. The partial correlation of birth weight and calorie intake was

found to be $r = 0.05$, when maternal weight (at the 20th week of pregnancy) was 'held constant', and that of birth weight and maternal weight was $r = 0.29$ when calorie intake was 'held constant'. Obviously, the weight of the mother was much more important in the determination of birth weight than the calorie value of her diet.

A separate analysis was made for subjects delivered in the 40th and 41st weeks of pregnancy, with similar results, and an attempt to obtain better associations between birth weight and calorie intake by the use of non-linear functions was unsuccessful. There was no indication in the data that birth weight is more closely associated with the intake of any nutrient than with calorie intake.

The conclusion must be that, within the range of diets in this survey, the influence of diet on birth weight was small, indeed negligible. The slight association indicated in Table 5 is attributable to common causes, such as body size, which influence both calorie intake and birth weight. Although the range of calorie intake was wide, all the subjects were, presumably, eating to appetite and the context is thus different from that of experiments in which the food supply is forcibly reduced. Smith (1947) found that birth weights in north-west Holland during the famine of 1945 were reduced by about 10%. At the height of the famine, the women were apparently taking less than 1000 kcal/day, and the weight gained by the average woman during pregnancy fell to about 2 kg. There was apparently little increase of foetal or neonatal mortality.

Burke, Harding & Stuart (1943) published evidence suggesting that birth weights increased by about 0.5 lb (0.23 kg) per 10 g increase of protein per day in the maternal diet. An attempt to confirm this finding was unsuccessful (Sontag & Wines, 1947). Platt (1947) and others have suggested that the low birth weights found in some oriental communities are due mainly to poor nutrition. It may be so, if 'nutrition' is interpreted in its widest sense, but there is at present no evidence that the diet taken during pregnancy has any special significance.

It is pertinent to note here that birth weight is of clinical importance in so far as it indicates the capacity of the baby to pass through the birth canal safely and to thrive after birth. Unfortunately, the only objective measures of vitality we have are crude and unsatisfactory, like death rates. An attempt to measure reflexes as indices of vitality was not helpful (Hyttén, 1951).

Other findings

The results do not show that the nature of the diet during pregnancy was associated with the incidence of antepartum haemorrhage, operative delivery, foetal malformation, perinatal death, or failure to establish successful breast feeding. For the sake of completeness, brief notes are given on some of these topics.

Caesarean section. Subjects who were delivered by Caesarean section had lower intakes of energy and of most nutrients than normal subjects, which is undoubtedly attributable mainly to the fact that the Caesarean-section rate rises steeply as maternal stature decreases (Baird, 1952*b*; Thomson, 1959*b*). Small women have a relatively high incidence of contracted pelvis (Bernard, 1952). Contracted pelvis may have been caused by malnutrition in childhood but is not influenced by the diet taken during pregnancy. Many Caesarean sections, especially in elderly primigravidae, are

undertaken for reasons other than disproportion between the foetal head and the maternal pelvis, such as uterine dysfunction and foetal distress in labour. There is no reason to think that abnormal labour due to these causes is affected by the diet taken during pregnancy.

Perinatal mortality. There were, among these 489 subjects, fourteen cases of perinatal death (stillbirth and death in the 1st week of life). The diets of the mothers who lost their babies did not differ appreciably from those of normal subjects. The numbers do not suffice for a satisfactory analysis by cause of death.

This negative result does not mean that there is no association between death rates and diet in pregnancy, but rather that the association is too small to appear in the present data. A study of National statistics suggests strongly that the fall in the stillbirth rate in England and Wales, from 38 per 1000 births in 1940 to 28 in 1945, was due mainly to the improved nutrition of pregnant women (Duncan, Baird & Thomson, 1952). This fall, which was dramatic and unexpected, can be expressed in another less dramatic way: a live-birth rate of 96% in 1940 had risen to 97% in 1945. During this 5-year period, a National food policy was applied with all the special powers available in wartime. If a similar change were expected in a controlled feeding experiment, it would be necessary to use more than 5000 experimental subjects, and a similar number of control subjects, in order to be reasonably sure of obtaining a statistically significant result.

Lactation. No evidence was found that the nature of the diet taken during pregnancy had any influence on the yield or composition of breast milk on the 7th day after delivery (Hyttén, 1954*a*), or on the incidence of breast feeding at the time of discharge from hospital or at 3 months after delivery. The results were also negative in relation to the incidence of failure to breast feed due to an inadequate supply of breast milk.

At the time of the survey, nearly all Aberdeen primiparae attempted to breast feed, and about 85% of them left hospital fully breast feeding, so that epidemiological studies were not hampered by a high rate of refusal to attempt lactation. There was little difference between the social classes in the incidence of breast feeding at the time of leaving hospital. If superior social circumstances, including superior diet, have any beneficial effect on the initiation of lactation, it is counterbalanced by an adverse effect of age. Older primiparae, who predominate in the upper social classes, have a significantly inferior ability to initiate lactation than younger primiparae. But, if lactation is successfully begun, women in the upper social classes are much more likely to persist after leaving hospital than those in the lower classes, so that there is an obvious social gradient in the breast-feeding rate at 3 months *post partum* (Hyttén, 1954*b*; Hyttén & Thomson, 1955). An intensive follow-up study showed that the most important determinant of continued breast feeding is the maternal attitude (Hyttén, Yorston & Thomson, 1958).

These epidemiological findings underline the difficulty of demonstrating the influence of nutrition on human lactation in western civilization. No doubt the difficulties would be even greater in countries, such as the U.S.A., where the great majority of mothers do not attempt to breast feed. The nutritional aspects of human lactation are discussed in detail by Hyttén & Thomson (1960).

GENERAL DISCUSSION

Historical background

Until a few decades ago, obstetricians were concerned mainly with the mechanics of difficult delivery, and, naturally, diet was first invoked in the interests of easy labour. Prochownik (1889, 1901, quoted by Ehrenfest, 1919) stated that if a special diet providing about 2000 kcal/day with a high protein and low carbohydrate content was prescribed during the last few weeks of pregnancy labour would be facilitated. A long controversy ensued, the echoes of which have not yet completely died away. Ehrenfest (1919) has pointed out in a review that Prochownik claimed only that his diet caused the foetal tissues to become more slack, so that the head 'moulded' more easily during delivery; he did not claim that the size of the foetus was reduced, or that the bones of the skull were softer. Nevertheless, his views were soon to be misrepresented by physiologists as well as by obstetricians. Paton (1903) found that litter weight of guinea-pigs whose diets had been severely restricted during pregnancy was reduced as compared with that of guinea-pigs fed freely. This finding, he said, 'confirms Prochownik's conclusions that by dieting the mother the children of rickety women may be so reduced in size as to be viable. . . . To the physiologist the point of chief interest seems to be the demonstration of the limitations in the extent to which the tissues of the mother can be utilised for the construction of the embryo. The nourishment of the maternal tissues seems to take precedence over the nutrition of the foetus.'

This extreme view lost most of its force as a result of studies of metabolism in human pregnancy made during the next 20 years which have, in general, been confirmed by more recent work. Slemons (1919) concluded that pregnancy 'represents for the mother a gain rather than a sacrifice and accordingly her tissues are not deprived of material to supply the new organism'. That would be true while the mother is able to eat to appetite; only if the maternal diet became seriously inadequate would the foetus tend to grow at the expense of maternal tissues. From that point of view 'the quantity of the mother's food is more influential than its quality'. Slemons thought that, clinically, 'there can be no justification for measures intended to restrict the growth of the foetus, for when rigidly carried out they tend to weaken the mother'. Nearly 30 years later, Garry & Wood (1945-6) expressed a similar opinion. Nevertheless, the old argument about maternal-foetal precedence revives from time to time, especially in the light of experiments on animals, from which it is clear that obvious impairment of foetal growth can be produced on subjecting the mothers to much more severe nutritional deprivation than could possibly occur among mothers able to eat to appetite (see, for example, Thomson & Thomson, 1948-9). Hammond (1944) elaborated a theory of 'partitioning' of nutrients in the maternal blood stream between the products of conception and the maternal organism, according to which the tissues with the higher metabolic rate take the first share of the available nutrients. The application of this theory to clinical obstetrics is obscure, and it does not seem to have provoked any research. We still know very little about the physiological, and especially the metabolic, aspects of the maternal-foetal relationship.

There the matter rests. Its origin—the problem of preventing difficult labour—is almost forgotten now, because it has become safe and easy to avoid a difficult vaginal delivery by resort to Caesarean section.

During the 1920's the continuing high rate of maternal mortality, mainly from infection, became the main focus of interest. In these early days of vitamin research, vitamin A was commonly known as the 'anti-infective' vitamin, and Green, Pindar, Davis & Mellanby (1931) reported that administration of a concentrate of vitamins A and D caused a significant reduction of puerperal morbidity. Their work was not confirmed, and, anyway, soon became irrelevant when the dramatic effect of the sulphonamides on 'puerperal fever' had been demonstrated. With the widespread prophylactic use of the antibiotics it would now be impossible to show whether diet has any influence on the incidence of infections during pregnancy.

Until the 1930's obstetricians had been more concerned about the survival and well-being of the mother than of the baby but, with difficult labour and puerperal sepsis both coming under control, increasing attention was paid to the health of the baby. The modern view was taking shape about 25 years ago and was, of course, thrown into relief by a rapidly falling birth rate. In contrast with the view expressed by Slemmons (1919), Mellanby (1933) thought that the quality of the maternal diet was much more important than its quantity, and stressed the importance of minerals and vitamins. He admitted that the evidence was 'somewhat meagre' but predicted that 'when knowledge is more complete, this aspect of the problem will prove to be even more important than appears at the present time'. The events of the next 10 or 12 years seemed to support him. Many dietary and clinical surveys and feeding experiments were conducted in several countries, and nearly all showed, or purported to show, a strong association between the quality of the diet taken by pregnant women and the course and outcome of pregnancy (see, for example, Theobald, 1937; Ebbs, Tisdall & Scott, 1941; Burke, Beal, Kirkwood & Stuart, 1943; Balfour, 1944; Cameron & Graham, 1944; People's League of Health, 1946). Negative findings were exceptional (Williams & Fralin, 1942). Taken together with other dietary studies and the assiduous propaganda of manufacturers of vitamins and 'concentrates', these reports engendered a strong enthusiasm for 'optimum nutrition'. Fortunately, as it happened, the enthusiasm was conveyed to the makers of the National food policy during the war of 1939–45. There is no doubt that their policy was brilliantly successful; for example, the stillbirth rate fell with a rapidity and to an extent unparalleled in peace.

Two symposia of (The) Nutrition Society (1944*a, b*) reflect the confident outlook of the times. But doubts about the technical adequacy of some of the previous investigations were growing. Garry & Wood (1945–6) introduced their review of dietary requirements in pregnancy and lactation by writing of meagre additions to knowledge during the previous decade and of 'the development of a more critical attitude, even of disillusionment...'

The ensuing uncertainty led to several more dietary and clinical studies (Sontag & Wines, 1947; Hobson, 1948; Speert, Graff & Graff, 1951; Macy, Moyer, Kelly, Mack, Di Loreto & Pratt, 1954; McGanity *et al.* 1954; and the study now described). It seems fair to say that the results of all these studies have been substantially negative,

even though some of the authors have made the most of minor and inexplicable correlations, or have argued that their technique must have been inadequate. On the other hand, Jeans, Smith & Stearns (1955) and Woodhill, van den Berg, Burke & Stare (1955) claim to have found striking correlations between reproductive histories and dietary 'ratings'; in both these studies the 'dietary history' method was used to assess the nutritive value of the food intake. Dieckmann, Turner, Meiller, Savage, Hill, Straube, Pottinger & Rynkiewicz (1951) gave protein supplements to a group of women during pregnancy and found a relationship between protein intake and the condition of the babies as graded by a paediatrician, but none between protein intake and duration of labour, toxæmia, prematurity and birth weight. Berry & Wiehl (1952) gave dietary advice to pregnant women and reported a reduced incidence of pre-eclampsia and prematurity.

The disillusionment, of which Garry & Wood wrote 14 years ago, has probably deepened. Unfortunately, there has been little sign of any new approach that will dispel it. It has remained difficult, in the absence of adequate physiological knowledge, to arrive confidently at dietetic conclusions from first principles. Also, little is known about the aetiology of most of the complications of pregnancy, and there is no established body of knowledge of the epidemiology of the phenomena of human pregnancy, on the basis of which plausible deductions as to causation may be made. As already mentioned, we undertook special epidemiological studies in parallel with our investigation of diet in an attempt to clarify the situation.

The importance of perspective

I have recounted the history of research on diet in pregnancy at some length because the current attitude of disillusionment is dangerous. Very few facts have been solidly established. We have to resolve the following dilemma: on the one hand, a belief which seems generally reasonable and is backed by much circumstantial and historical evidence, that mothers can more efficiently undertake the physiological burden of pregnancy and lactation if they are well fed, and, on the other hand, the apparent failure of survey methods to provide a convincing confirmation of this belief. Continuing uncertainty may lead those responsible for the public health to consider, in defiance of the lessons of history, that nutrition in pregnancy is unimportant and may be neglected. The dilemma can, I think, be resolved by putting the facts we have into perspective.

Our attitude towards malnutrition still tends to be conditioned by experience of the classic deficiency diseases, breakdowns of health that can be induced or prevented by relatively small and highly specific changes of diet. It would seem that many workers regard at least some disorders of pregnancy as similar deficiency states. There is no evidence that it is so, but the habit dies hard. For example, the dietary and biochemical data of Mack, Kelly & Macy (1956) failed to support their view that pre-eclampsia is a form of malnutrition. But instead of doubting their original hypothesis, they concluded that their technique must have been inadequate: 'survey methods must be improved and expanded, since not one but perhaps many longstanding nutritional deficits may be involved in the "hidden hunger", and the resultant conditional malnutrition may constitute a predisposing factor to the occurrence of toxæmia.'

In my opinion, the weight of evidence now shows conclusively that few, if any, of the common disorders of pregnancy are likely to be deficiency states of classic type, and that the diets of pregnant women can vary within wide limits, in quantity as well as in quality, without clinically obvious impairment of the reproductive process. If the phenomena of pregnancy and childbirth react sensitively to alterations of diet, reproductive mortality and morbidity in countries where undernutrition and malnutrition are rife would no doubt be much more serious than they are. If, within ordinary limits, diet has an immediate importance for the individual mother and foetus, the key will probably be found in the intimate processes of physiological adaptation to pregnancy, in the idea of impaired efficiency rather than of breakdown.

But even if the importance of diet in pregnancy is usually inconspicuous in terms of the individual, or of small groups of mothers and babies, the same is not necessarily true in terms of large populations. Effects that are too small to be discerned in the study of a few hundred pregnant women may become highly significant on a national scale. Although the National 'feeding experiment' of wartime Britain was uncontrolled, it resulted in the first major reduction of the stillbirth rate, which up to that time had been responding only sluggishly to extension of the maternity services. The wartime result, considered as a nutritional effect, is the more convincing because it was achieved in the context of a society where most of the conditions of living, other than the nutritional, were deteriorating (Duncan *et al.* 1952). It underlines the importance to public health of a sound nutritional policy, and is in no way inconsistent with a wide latitude in the reaction of individuals to dietetic change.

There is also a wider perspective, of even greater importance to the public health. For the individual mother, the diet taken during pregnancy is only the most recent phase of a long history of nutritional experience. There is nothing new in the idea that the main importance of nutrition for pregnancy lies in the past. McCarrison (1937) said: 'The satisfaction of nutritional needs in pregnancy begins with the antenatal lives of the mothers of our race. It must continue during the period of their growth and development up to, during and following the period when they find their fulfilment in motherhood; a fulfilment for which nutrition prepares and makes ready the way.'

Dietary education during pregnancy is by no means wasted even if it often fails to prevent such abnormalities as prematurity and pre-eclampsia. It has a definite and useful part in the management of nausea and vomiting, heartburn, constipation, obesity and oedema. Even more important, it may help to ensure that the next generation has a more favourable experience of good diet, with beneficial effects on the obstetrics of, perhaps, 20 years ahead. I hope, in a subsequent paper, to show that healthy and well-grown mothers have much more favourable obstetric histories than mothers who are stunted and generally unhealthy. The difference in health and physique is probably due, at least in part, to their nutritional experience during growth and adolescence.

Implications for research

Dietary and clinical surveys are tedious and expensive, and it is far from easy to maintain a high standard of technical accuracy in large-scale field work. Controlled feeding experiments among women living at home are doubtless even more difficult to conduct with precision. There seems to be no way of simplifying the procedures without undue sacrifice of accuracy. Since, in a study of several hundred mothers with a fairly wide range of dietary habits, there is no evidence of a dietary level below which reproductive efficiency obviously begins to suffer, it seems unlikely that a survey of a similar population on a larger scale would clarify the problem of nutritional requirements during pregnancy. It is also doubtful if surveys of the levels of nutrients and metabolites in tissues and body fluids would be helpful (Thomson & Duncan, 1954).

Obviously the search for more adequate knowledge of the physiology of pregnancy should be accelerated. There is surprisingly little accurate and reliable information on metabolism during pregnancy. Weight gained during pregnancy has been measured, but not much is known about the nature and quantity of the materials stored. Even less is known of the katabolic processes that take place after delivery. But it is known that there are very large variations from individual to individual, and all of them are unlikely to be equally 'physiological'. Investigation should obviously be directed to the nature and meaning of these variations. Reference to almost any textbook of human physiology will reveal that pregnancy is referred to rather briefly and mainly with reference to endocrine changes. Leitch (1957) has recently shown that Claude Bernard's famous dictum about the constancy of the *milieu intérieur* cannot be applied to pregnancy, since this *milieu* changes continuously during pregnancy. Attempts to apply physiological 'norms' derived from non-pregnant adults may therefore be misleading. Hytten & Duncan (1956), for example, have argued that the physiological haemodilution of pregnancy is not infrequently misconstrued by clinicians as indicative of iron deficiency. There is a world of research in matters such as these, and in them lies the key to understanding of nutrition in pregnancy.

SUMMARY

1. In a preliminary analysis, 489 primigravidae were divided into two groups: 197 with normal clinical history, and 292 in whom there was a clinical abnormality of some kind. The mean nutritive values of the diets taken by these two groups of women did not differ significantly.

2. More detailed analysis confirmed that there was little or no association between the nutritive value of the diet taken during pregnancy and duration of gestation, birth weight, antepartum haemorrhage, operative delivery, foetal malformation, perinatal death, or failure to breast feed.

3. However, pre-eclampsia and a relatively high rate of gain of body-weight were associated with a relatively high calorie intake, and with a raised intake of most nutrients. These correlations are described and discussed. It is emphasized that the findings relate to women whose weights were not being 'regulated' as part of antenatal care.

4. The findings are discussed in relation to previous studies of diet in pregnancy. The weight of evidence shows conclusively that the diets of pregnant women can vary widely, in quantity as well as in quality, without clinically obvious impairment of the reproductive process. But although the importance of diet in pregnancy is usually inconspicuous in terms of the individual mother and her child, the same is not necessarily true in terms of large populations. A dramatic reduction of the National stillbirth rate during the 1939–45 war was almost certainly attributable to improved nutrition.

5. Epidemiological and physiological knowledge of human reproduction is meagre, and the implications of the present results for further research are discussed.

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