AN EVALUATION OF DIETARY INFLUENCES IN THE GENESIS OF TOXAEMIA OF PREGNANCY

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A King that cannot rule him in his diet,
Will hardly rule his Realm in peace and quiet.
—Regimen Sanitatis Salernitanum.

In the seven hundred or more years which have elapsed since this was written, the pleasures of overeating have without difficulty withstood the challenge of moderation which succeeding generations of physicians have pressed upon their usually unwilling patients. War and other major social catastrophes bring with them enforced dietary restriction, and personal dietary discipline almost always seems to fall short of these drastic upheavals in its effectiveness.

The third of July 1954 was " D " day for the gourmet, for on that day all food rationing ceased in Great Britain. No less important was this date for the scientific worker interested in the physiological and pathological processes dependant on, and associated with, diet. An entire national population living under more or less controlled dietetic circumstances offers an excellent scientific control for comparison with circumstances which might arise when dietetic restrictions are removed.

The relationship of diet to certain physiological and pathological processes of pregnancy has enjoyed a great deal of attention in recent years. Much of the evidence produced is conflicting, and even confusing, and it is hoped that these observations may contribute to the solution of some of the problems.

The periods chosen for this survey are covered by the years 1944—1951 and 1954—1958. Before considering any of the obstetrical data, I would like to examine the dietary data in some detail.

The energy value and nutrient content of domestic food consumption is shown in table I. (Taken from *Domestic Food Consumption and Expenditure*—Annual Reports of the National Food Survey

Committee 1944—1951: Her Majesty's Stationery Office.) The years 1944—1949 cover urban working class households. During the course of 1950, however, a change was introduced into the technique of the National Food Survey. In most respects the technique remained the same as that covering 1944—1949, but during that period, as noted above, the main sample covered only urban working-class households. In 1950 the survey was extended to cover a sample representing the whole population in both urban and rural areas.

TABLE I

ENERGY VALUE AND NUTRIENT CONTENT OF DOMESTIC FOOD CONSUMPTION: URBAN WORKING CLASS HOUSEHOLDS 1941—1950 AND ALL HOUSEHOLDS 1950 AND 1951. AVERAGE INTAKE PER HEAD PER DAY

	Urban and working-class households							*All households*	
	1944	1945	1946	1947	1948	1949	1950	1950	1951
Energy value (calories) ..	2387	2375	2307	2308	2387	2425	2441	2474	2466
Total protein (G.)	73	76	78	77	77	76	77	78	77
Animal protein (G.)	35	35	37	36	34	34	37	38	37
Fat (G.) ..	94	92	86	82	88	95	102	101	97
Carbohydrate (G.) ..	311	310	305	315					322
Calcium (mg.)	868	875	912	996	1012	1030	1041	1066	1076
Iron (mg.) ..	13·5	12·7	14·4	14·3	14·2	13·6	13·5	13·6	12·8
Vitamin A (I.U.) ..	3173	2908	3112	3148	3380	3377	3465	3536	3432
Vitamin BI (mg.) ..	1·62	1·47	1·55	1·52	1·57	1·53	1·50	1·51	1·34
Riboflavin (mg.) ..	1·76	1·58	1·65	1·64	1·65	1·64	1·66	1·69	1·60
Nicotinic acid (mg.) ..	13·9	13·2	14·5	12·9	12·8	12·7	12·9	13·0	12·5
Vitamin C (mg.) ..	87	86	89	80	97	91	82	84	68·5
Vitamin D (I.U.) ..	106	143	174	169	198	190	168	172	157

At first sight it might seem that such a fundamental change in the method of sampling might invalidate any conclusions drawn from these figures. However, on closer examination one feels that this is not so. The evidence upon which this argument is based is as follows:

1. The daily dietary intake in 1951 for urban working-class households on the one hand, and all households on the other, is very similar. No standard deviation of the mean figure is given in the official figures, but 2,441 calories differs from 2,474 calories by an insignificant amount.

2. In times of rationing such as those covered by the years under consideration the standard of diet in all households is controlled more by the availability of rationed foods than by the purse. Therefore urban working-class households may without gross inaccuracy be taken as a sample of all households.

3. As a final test an enquiry was made into the breakdown of daily dietary intake in the various classes of society. A non-rationing year—1957—was chosen at random, because if variation were to be expressed at all, it would be expressed after the cessation of rationing. Table II was accordingly constructed (*Domestic Food Consumption and Expenditure*, 1957. Annual Report of the National Food Survey Committee—Her Majesty's Stationery Office.) It will be seen from table II that the working-class groups B and C bear a close resemblance to the "All Household" group, thus supporting a fair comparison between urban working-class households 1944-1949, and all households 1950-1951 and 1954-1958.

Table III shows the energy value and nutrient content of domestic food consumption for all households 1954—1957. (Taken from *Domestic Food Consumption and Expenditure* 1954, 1955, 1956 and 1957. Annual Reports of the National Food Survey Committee—Her Majesty's Stationery Office.)

The Annual Report of the National Food Survey Committee for 1958 has not yet been published (January 1960). It was felt however, that some cases delivered early in 1958 should be included in the survey, as the greater part of the pregnancy concerned would be passed during 1957, and with the rising standard of living, gross dietary deterioration is unlikely to be reported in the report for 1958 when available. For this reason, any case delivered in the first three months of 1958 was included and given a 1957 value for daily dietary intake.

A valid criticism of endeavours to compare the diets of expectant mothers in what may conveniently be called the " Rationed Period " and the " Unrationed Period ", is contained in the observation that the diets which have been discussed so far relate to the whole population. During the rationed period, extra foods and vitamin supplements were allowed to expectant mothers, and in the unrationed period we have no measure of how much more than the general population the expectant mother may eat. On general grounds it is felt that the extras allowed during the rationed period would be at least balanced by the extras taken in the unrationed period. (Attending antenatal clinics through most of both periods, one

TABLE II

ENERGY VALUE AND NUTRIENT CONTENT OF DIETS OF HOUSEHOLDS OF DIFFERENT SOCIAL CLASSES (1957) — PER HEAD PER DAY

	Class								
	A			*B*	*C*	*D*		*OAP*	*All households*
						excluding OAP			
	A1	*A2*	*All*			*with/without earners*			
Energy value (calories) ..	2523	2590	2570	2631	2585	2462	2485	2528	2587
Total protein (G.) ..	79	76	76	76	74	70	70	72	75
Animal protein (G.) ..	53	46	48	44	42	39	40	41	43
Fat (G.) ..	115	114	114	112	108	100	104	105	110
Carbohydrate (G.) ..	292	315	309	331	330	320	317	323	325
Ca (mg.) ..	1122	1080	1091	1039	1009	969	986	1018	1028
Fe (mg.) ..	14·8	14·0	14·2	14·2	14·0	13·1	12·8	12·6	14·1
Vitamin A (I.U.) ..	5145	4675	4800	4421	4130	3704	3912	3867	4289
Thiamin (mg.)	1·33	1·29	1·30	1·31	1·29	1·22	1·20	1·25	1·29
Riboflavine (mg.) ..	1·91	1·76	1·80	1·69	1·61	1·50	1·56	1·59	1·66
Nicotinic acid (mg.) ..	15·0	13·7	14·1	13·7	13·5	12·8	12·8	13·1	13·8
Vitamin C (mg.) ..	73	58	62	53	49	44	44	44	52
Vitamin D (I.U.) ..	140	150	147	147	149	132	121	117	145

Income ranges used to define social classes (1957)

Class	*Gross weekly income of head of household*
A (A1)	£30 or more
(A2)	£18 — £30
B	£10 10s. 0d. — £18
C	£7 — £10 10s. 0d.
D	Under £7.

TABLE III

ENERGY VALUE AND NUTRIENT CONTENT OF DOMESTIC FOOD CONSUMPTION: ALL HOUSEHOLDS 1954—1957. AVERAGE INTAKE PER HEAD PER DAY

	1954	1955	1956	1957
Energy value (calories)	2626	2641	2624	2587
Total protein (G.)	77	77	76	75
Animal protein (G.)	41	42	43	43
Fat (G.)	107	107	108	110
Carbohydrate (G.)	340	342	337	325
Calcium (mg.)	1034	1044	1029	1028
Iron (mg.)	13·4	13·5	13·3	14·1
Vitamin A (I.U.)	3911	4199	4310	4289
Vitamin B1 (mg.)	1·28	1·24	1·21	1·29
Riboflavin (mg.)	1·67	1·65	1·65	1·66
Nicotinic acid (mg.)	13·3	13·1	13·0	13·8
Vitamin C (mg.)	50	51	50	52
Vitamin D (I.U.)	144	144	150	145

has not been impressed by any increase in the frugality of living of one's patients.) Furthermore, the take-up of vitamin supplements during the rationed period fell far short of expectation, thus minimizing the difference between the diet of the general population and expectant mothers. "The average percentage take-up in England for fruit juices did not exceed 45.7 per cent of the potential issue if all expectant mothers had drawn their full allowance; for cod liver oil the corresponding figure is only 21 per cent, and for vitamin A and D tablets, 34.3 per cent, whilst the Welsh averages (covering the current series) have been even lower." (*On the State of the Public Health during Six Years of War* (1946): Her Majesty's Stationery Office.)

While recognizing the adequacy, indeed excellence, of the diet during the rationed period, few would deny that the diet of the unrationed period is more adequate in all departments except, perhaps, in vitamin B1. My impression depends partly upon the experience of having lived through the periods, but, if the factors

already related will bear reasonable statistical analysis, as I think they will, it would be interesting to attempt to show a mathematical relationship between certain dietary factors taken from the rationed and unrationed periods. This will be elaborated shortly.

Material

In the portion of this paper which covers normal pregnancy, the material under consideration relates in the rationed period to 1,000 normal mothers who were delivered of 1,002 babies, and in the unrationed period to 500 normal mothers who were delivered of 500 babies. All cases were booked and delivered in hospital in Crickhowell and Cardiff, and, being delivered in hospital, are not a true cross-section of all the births in the country in the statistical sense. They were, however, a random sample of normal hospital patients delivered between 1944 and 1951 inclusive and January 1954 to March 1958, also inclusive.

The standards of normality were:

1. The mother's first antenatal visit was at or before twelve weeks gestation.
2. There was no pre-existing disease in any major system in the mother. Patients with thyrotoxicosis, tuberculosis, chronic renal disease, diabetes mellitus, cardiac defects, essential hypertension, etc., were all excluded.
3. There was no toxaemia of pregnancy.
4. Delivery took place within twenty days of the expected date. (This may seem a rather liberal variation, though it does only represent two days for each lunar month of pregnancy, a very reasonable variation from the normal cycle of menstruation. If pregnancy is represented by ten missed periods, up to twenty days error might be expected on this basis.)
5. The last antenatal visit took place within a maximum of seven days of delivery.
6. A normal live baby (or babies) was produced.

In the rationed series two caesarean sections at term for simple disproportion were included. In the unrationed series nine caesarean sections were included. The indications for section did not affect the criteria of normality—disproportion, placenta praevia, primiparous breech, and foetal distress.

Following the arguments put forward by the author (1954) the maternal weight at the twelfth week of pregnancy was taken to be equal to the pre-pregnant maternal weight, and the weight gain during pregnancy was taken as the gain from the weight at twelve weeks until the last antenatal visit before delivery. There may well be a minor weight gain in the first trimester, but there is in most women a slight loss of weight in the last few days of pregnancy. It would seem pointless, in view of this, to add a small arbitrary weight at one end of pregnancy, and subtract a similar small arbitrary weight at the other, as patients were not weighed immediately before delivery.

Maternal age is quoted as age on the birthday prior to first antenatal visit.

Maternal weight gain was recorded in pounds and as a percentage of weight at twelve weeks (or pre-pregnant maternal weight).

Infant birth weight was recorded in pounds.

The part of the paper which relates to toxaemic cases covers a random sample over both periods of 1,409 booked patients, 659 from the rationed period and 750 from the unrationed period. As the material was collected in two parts, some of the earlier records had been destroyed when they were required again, which unfortunately reduced the numbers. In the various calculations, due allowance has been made for this.

The criteria upon which a diagnosis of toxaemia of pregnancy should be based are not easy to decide. The point at which normality ends and toxaemia begins is ill defined, and the experience of various investigators betrays obvious difficulties in assessing the starting point of pre-eclampsia. For instance, Nelson (1955) showed that in his series of primigravidae in Aberdeen studied between 1938 and 1953 a fifth had a rise of blood pressure warranting the diagnosis of pre-eclampsia.

In The People's League of Health (1946) investigation the authors state that the incidence of toxaemia varied considerably from hospital to hospital, being as high as 58.9 per cent in one hospital and as low as 5.6 per cent in another.

These variations in the percentage incidence of pre-eclampsia bespeak not so much a variation in patients, but a variation in the yardstick of assessment of pre-eclampsia by the observers concerned.

For the purpose of this investigation I have used the following factors as the diagnostic criteria of pre-eclamptic toxaemia:

1. Patients concerned have no pre-existing renal or other disease, of which hypertension in pregnancy is merely a manifestation.
2. The blood pressure at which toxaemia is deemed to have commenced is, in the absence of other signs, 140/90 mm. Hg.
3. Toxaemia was diagnosed if albuminuria appeared in a formerly normal urine and could not be shown to be due to infection, vaginal discharge, or other non-toxaemic cause.
4. Oedema, of itself, was not regarded as a sign of toxaemia, but if present in association with a diastolic blood pressure of 90 mm. Hg., toxaemia was diagnosed even if the systolic blood pressure was only 130 mm. Hg.

There are shortcomings in this scheme. Hypertension, albuminuria, and oedema are all recognized as the late signs of established toxaemia, but no satisfactory measure of weight gain has yet been established, and no earlier physical sign has yet been recognized as being indicative of toxaemia. Inadequate as the signs recorded above may be, in as much as they were applied to all cases throughout the investigation, they formed a useful basis for comparison

of the incidence of toxaemia in one set of patients in relation to another set.

Weight Changes, Diet, and Toxaemia of Pregnancy

There now seems to be little doubt that excessive maternal weight gain often heralds the onset of pregnancy toxaemia. Chesley and Chesley (1943) measured extracellular water in 1,388 patients and showed that the incidence of pre-eclampsia was six times as great in women who developed excessive extracellular water as in women with normal extracellular water. Zangemeister (1916) pointed out the relationship between oedema formation, weight increase, and incipient toxaemia, and he pointed out that patients with toxaemia lost an average of 12.31 Kg. during delivery and the first ten days of the puerperium compared with a corresponding loss of 7.54 Kg. in normal patients. Using a similar technique, Stander and Pastore (1940) quote 9.36 Kg. as being the loss during delivery and in the first ten days of the puerperium in toxaemic women compared with a loss of 7.65 Kg. in normal women over the same period. Dawson and Borg (1949) regard water retention and undue weight gain as " the predominant and earliest morbid phenomenon of toxaemic pregnancy ". They reviewed 93 cases of definite clinical toxaemia, and taking all ages and parities together they demonstrated a 19 per cent increase over what they regarded as being the normal weight gain in pregnancy as a whole, and 54 per cent increase over normal in the last four weeks.

Evans (1937) studied 52 cases of toxaemia and concluded that " an abnormal increase in weight during any one month is usually an earlier indication of an impending toxaemia than a rise in blood pressure ".

Harding and van Wyck (1934) declared that gains up to 5 lb. per month were normal. Gains of 5—8 lb. per month they regarded with suspicion and gains of 8 lb. or over as indicating potential toxaemia. McIlroy and Rodway (1937) quoted 11 lb. $4\frac{1}{2}$ oz. as being the normal average gain in the last sixteen weeks of normal pregnancy (calculated from 900 patients) and 17 lb. 4 oz. as being the average gain in toxaemic patients (calculated from 75 patients). They thought it of great significance that " . . . throughout pregnancy the average periodic increase was greatest in the toxaemic group of patients ".

Wodon (1935) studied 400 pregnant women. In 306 who had a normal weight gain he found 24 with some degree of toxaemia, but in 94 who gained more than the normal amount he found 72 who showed signs of toxaemia. He regarded 2 Kg. as the maximum

normal weight gain per four weeks in the last twenty-four weeks of pregnancy.

Randall (1925) suggested that women who develop toxaemia lose more weight than normal women early in pregnancy and gain more in late pregnancy. Tompkins and Wiehl (1951) presented a variation of this theme and showed that a normal weight gain in the first two trimesters is associated with a low toxaemia incidence; if, however, a low rate of gain up to the latter part of the second trimester gives way to a sudden gain towards the end of the second trimester, the incidence of toxaemia is greatly increased.

Finnerty, Buchholz, and Tuckman (1958), in an evaluation of chlorothiazide in the treatment of pre-eclampsia, noted the frequent loss of 4-5 lb. in a 24 hour period in a toxaemic patient treated with chlorothiazide showing water retention to be the cause of excessive weight gain in toxaemic patients. Using chlorothiazide in the treatment of toxaemia also, Banfield, Jungck and Greenblatt (1958) demonstrated a loss of weight of 2-6 lb. in all the 47 cases studied in the first 24 hours diuresis following chlorothiazide. Tennant and Leslie (1960) studied 40 cases of pre-eclamptic toxaemia treated with chlorothiazide. They stated:

As the patients were on an adequate diet, weight loss after each course (of chlorothiazide) was taken as evidence of removal of water. In 38 cases weight loss occurred—the maximum weight in one course being 11lb. 4oz. and the minimum 2oz. In two cases no weight was lost and the drug judged ineffective. One of these two cases developed eclampsia. A further noteworthy point is that all cases gained weight rapidly during the rest period.

The principles which have guided the thoughts of most of the authors so far quoted have been those of water retention and its control as being the basic process of pre-eclampsia. However, an additional factor has been introduced by other authors, namely weight increase due to simple overeating. Bingham (1932) considered the control of weight by diet and open air exercise as being all important in the prevention of toxaemia. Hamlin (1952) described how, despite the continuance of pre-eclampsia, eclampsia was abolished in 5,000 cases delivered in Crown Street, Sydney in 1950-51. This success is attributed to various factors, but in the main close antenatal supervision is deemed responsible, including dietary advice and control. He stated that it is normal for a young primipara with low initial blood pressure to increase her weight between the twentieth and thirtieth week by up to 8 lb. only. Less gain usually implies that there will be no toxaemia, and more gain bespeaks impending toxaemia.

" Evidence has been obtained that the disease which appears as eclampsia in late pregnancy can be recognized several months earlier and is probably of metabolic origin. A gain of more than 8 lb. in weight from about 20th—30th week is surely but the warning evidence that pathological processes are already

disturbing the *milieu intérieur*. The damage, I believe, occurs at this stage when there is an imbalance of diet—an excess of carbohydrate and a relative deficit of first class protein and of vitamins."

Stevenson (1952) discussed further the very excellent results obtained at The Women's Hospital, Crown Street, Sydney. He felt that women who are overweight at the beginning of pregnancy are the most prone to develop pre-eclampsia, and that toxaemia is more likely to occur in women whose rate of weight gain is excessive during pregnancy. He goes on " . . . it becomes apparent that dietary control of weight is the only measure available to counteract it (i.e., toxaemia) ". By way of treatment he advises diet, rest and the prevention or treatment of anaemia. The diet advised is 2,100 cal. daily for a mild case of toxaemia, and 1,750 cal. daily if overweight. Stress is laid on the adequacy of the protein content of the diet.

Hughes (1956) also quoted the Crown Street figures. He suggested that the potent weapons against pre-eclampsia are:

(i) A close watch must be kept on blood pressure relative to the blood pressure in early pregnancy rather than on an absolute value.

(ii) A woman who gains more than 1 lb. per week must be examined again in seven days. " The purpose of this is to discover the possible cause of the increase in weight. Is it associated with any other sign such as oedema? Is it dietary excess? Is the patient taking an ill-balanced diet? ".

Nelson (1955) noted that excess weight gain in the 20—30 week period of pregnancy is associated with a higher rate of pre-eclampsia, but only under the age of 30. His main conclusion in relation to the current series was " The relationship of weight gain to pre-eclampsia, and in particular to the severe grade, is too indefinite to be of real practical value in predicting the development of pre-eclampsia; and dietary control is, in fact, unlikely to benefit those who require help most ".

With such a mass of information, one wonders just how much diet, weight gain and pre-eclampsia are related. To refer to purely dietary experimental evidence, de Snoo (1937) showed in his clinic in Utrecht that 70 per cent of all pregnant women suffered from varying degrees of oedema. On a salt free diet this disappeared. In 20,000 pregnancies at his clinic over a period of ten years there was not a death from eclampsia, whereas in 1935 the eclampsia mortality in the whole of Holland was 1 in 3,000 deliveries. Ebbs *et al.* (1942) in the Toronto feeding experiment gave expectant mothers supplements of proteins, fats, fruit, vitamin concentrates and iron. When compared with a control group on a relatively poor diet the women who received the supplementary foods had

fewer miscarriages and premature births and were less liable to toxaemias of pregnancy.

In The People's League of Health experiment (1946), 5,000 expectant mothers attending ten London hospitals between March 1938 and the end of 1939 were studied. A shortage of calcium was found to exist in the diets of 70 per cent of the women and in 90 per cent the diet was deficient in iron. About half were taking inadequate amounts of vitamin A, B complex, or C.

The main investigation consisted of dividing the women into two groups, one group of which received supplements of iron, calcium, iodine, vitamins A, B, C, and D from the 24th week until delivery; the other group was the control group. The results indicated that the women receiving the special diet were protected against the risk of toxaemia in a ratio which was almost 30 per cent.

The balance of opinion is overwhelmingly in favour of the fact that women with toxaemia of pregnancy gain more weight than normally pregnant women. Yet the more one studies the literature the more confused one becomes as to what this actually means. Is this increase in weight which can bespeak early toxaemia:

(*a*) a laying down of fat due to excessive intake of calories as some authors would suggest,
(*b*) a simple fluid retention with or without oedema, or
(*c*) a combination of both (*a*) and (*b*)?

The fact that frank oedema does not exist, does not, of course, vitiate the possibility of (*b*). White (1950) states that the volume of a leg must increase by 8 per cent before oedema becomes clinically manifest.

It is hoped that the current series may cast further light on this problem.

Energy Value of Diet in Periods Studied

Table IV illustrates the mean daily calorie intake over the two periods 1944-51 and 1954-58, and the frequency distribution of these values for the present series.

Reference has already been made to the higher nutritive value in general of the unrationed period compared with the rationed period. It will be seen that the mean daily intake per person is 2,428 Cal. in the rationed period and 2,626 Cal. in the unrationed period. As the difference between these two means is 135.4 times the standard error of the difference, it is very unlikely that it arose by chance. This, therefore, gives us a good starting point for the consideration of the statistical analysis which follows.

Maternal Weight Gain during Pregnancy

Having established by mathematical measure the increase in

TABLE IV

ENERGY VALUE OF DIET, IN CALORIES, FOR CASES STUDIED

Year	*Number of cases*	*Mean energy value (Cal.)*
1944	65	2387
1945	2	2375
1946	0	2307
1947	10	2308
1948	275	2387
1949	283	2425
1950	316	2474
1951	49	2466
1954	37	2626
1955	219	2641
1956	166	2624
1957	53	2587
1958	25	2587 (assumed)
	Total 1500	

Mean daily energy value of diet 1944—1951 = 2428 Cal.
S.D. = 38·25
Mean daily energy value of diet 1954—1958 = 2626 Cal.
S.D. = 18·37

calorific value of the diet in the unrationed, compared with the rationed period, let us now consider maternal weight gain.

Tables V and VI show the frequency distribution of the weight gain during pregnancy (from 12 weeks to term) in pounds. It will be seen in both series that primiparae gained more than multiparae by a statistically significant amount. However, when the rationed and unrationed groups are compared there is no significant difference between either primiparae in each series, multiparae, or all cases considered together as shown in the table VII.

Indeed, the mean gain, considering all cases together, in the rationed group (24.7 $\pm$ 0.27 lb.) might well have come from the same series as the unrationed group (mean 24.57 $\pm$ 0.37 lb.).

Having considered the maternal weight gain in pounds it was then considered as a percentage of pre-pregnant maternal weight (see tables VIII and IX). A comparison of percentage weight gain between the rationed and unrationed groups shows that in primiparae and multiparae there was no significant difference in weight gain during pregnancy. When all cases were considered together there was shown to be a very slightly greater percentage increase in weight in the rationed group than the unrationed, but with a difference only 2.108 times its standard error it is very doubtful if this is really significant.

TABLE V

THE FREQUENCY DISTRIBUTION OF MATERNAL WEIGHT GAIN DURING PREGNANCY—1944 TO 1951

Maternal gain (lb.)	*No. of primiparae*	*No. of multiparae*	*Total*
—10—	0	1	1
— 5—	0	0	0
— 0—	5	0	5
5—	8	15	23
10—	36	50	86
15—	78	95	173
20—	117	118	235
25—	125	111	236
30—	93	52	145
35—	37	17	54
40—	23	7	30
45—	2	3	5
50—	1	4	5
55—	0	0	0
60—65	1	1	2
Total	526	474	1000

Mean total weight gain (primiparae) = 25·77 lb. S.D. = 8·44
Mean total weight gain (multiparae) = 23·5 lb. S.D. = 8·33
Mean total weight gain (all cases) = 24·7 lb. S.D. = 8·46

As the difference between the mean weight gain in primiparae and the mean weight gain in multiparae is 4·277 times the standard error of the difference it may be regarded as significant.

TABLE VI

THE FREQUENCY DISTRIBUTION OF MATERNAL WEIGHT GAIN DURING PREGNANCY—JULY 1954 TO MARCH 1958

Maternal gain (lb.)	*No. of primiparae*	*No. of multiparae*	*Total*
— 5—	1	0	1
0—	1	2	3
5—	5	9	14
10—	15	19	34
15—	35	52	87
20—	62	61	123
25—	56	68	124
30—	42	29	71
35—	17	12	29
40—	5	2	7
45—	4	1	5
50—	0	1	1
55—	0	0	0
60—	0	0	0
65—70	0	1	1
Total	243	257	500

Mean maternal weight gain (primiparae) = 25·42 lb. S.D. = 8·21
Mean maternal weight gain (multiparae) = 23·76 lb. S.D. = 8·2
Mean maternal weight gain (all cases) = 24·57 lb. S.D. = 8·25

As the difference in weight gain between multiparae and primiparae is 2·26 times the standard error of the difference it is just statistically significant.

TABLE VII

MEAN WEIGHT GAIN IN RATIONED AND UNRATIONED GROUPS

	Rationed mean	*Unrationed mean*	*Difference of means* / *St. error of diff.*
Primiparae	25·77 lb.	25·42 lb.	0·55
Multiparae	23·5 lb.	23·76 lb.	0·64
All cases	24·7 lb.	24·57 lb.	0·29

TABLE VIII

THE FREQUENCY DISTRIBUTION OF MATERNAL WEIGHT GAIN (EXPRESSED AS A PERCENTAGE OF PRE-PREGNANT WEIGHT) FOR BIRTHS DURING PERIOD 1944—1951

Percentage maternal weight gain	*Primiparae*	*Multiparae*	*All cases*
— 8—	0	1	1
— 4—	0	0	0
0—	4	0	4
4—	8	21	29
8—	31	46	77
12—	74	93	167
16—	108	90	198
20—	108	92	200
24—	102	75	177
28—	54	26	80
32—	29	19	48
36—	7	6	13
40—	0	3	3
44—	0	0	0
48—	0	0	0
52—	1	1	2
56—60	0	1	1
Total	526	474	1000

Mean maternal weight gain (primiparae) = 21·48 per cent
S.D. = 7·1
Mean maternal weight gain (multiparae) = 19·76 per cent
S.D. = 7·75
Mean maternal weight gain (all cases) = 20·67 per cent
S.D. = 7·46

TABLE IX

THE FREQUENCY DISTRIBUTION OF MATERNAL WEIGHT GAIN (EXPRESSED AS A PERCENTAGE OF PRE-PREGNANT WEIGHT) FOR BIRTHS BETWEEN JULY 1954 AND MARCH 1958 INCLUSIVE

Percentage maternal weight gain	*Primiparae*	*Multiparae*	*All cases*
— 4—	1	0	1
0—	1	3	4
4—	11	8	19
8—	13	32	45
12—	41	47	88
16—	44	53	97
20—	62	47	109
24—	41	45	86
28—	17	11	28
32—	5	8	13
36—	4	1	5
40—	1	0	1
44—48	2	1	3
Total	243	256	499

Gains over 48 *per cent—*1 *multiparae gained* 60·19 *per cent*

Mean maternal weight gain (primiparae) = 20·4 per cent
S.D. = 7·43
Mean maternal weight gain (multiparae) = 19·23 per cent
S.D. = 7·66
Mean maternal weight gain (all cases) = 19·8 per cent
S.D. = 7·57

	Rationed mean	*Unrationed mean*	*Difference of means* / *St. error of diff.*
	per cent	*per cent*	
Primiparae	21·48	20·4	1·9
Multiparae	19·76	19·23	0·89
All cases	20·67	19·8	2·108

Maternal Weight at Three Months Pregnancy

This, as already explained, is taken as being equivalent to the pre-pregnant maternal weight. As toxaemia has been related to pre-pregnant weight by so many authors, it is interesting to consider the pre-pregnant weight of mothers in the rationed and the unrationed series, and compare them.

Tables X, XI, and XII show the frequency distribution, in this sample, of pre-pregnant maternal weight in lbs.

There is, therefore, no significant difference in the pre-pregnant weight of mothers in the rationed and the unrationed series.

TABLE X

THE FREQUENCY DISTRIBUTION OF MATERNAL WEIGHT AT THIRD MONTH OF PREGNANCY FOR DELIVERIES BETWEEN 1944 AND 1951

Maternal weight at 3 months (lb.)	*Primiparae*	*Multiparae*	*All cases*
70—	0	1	1
84—	19	19	38
98—	134	101	235
112—	161	156	317
126—	125	113	238
140—	50	48	98
154—	26	19	45
168—	10	8	18
182—	1	7	8
196—	0	1	1
210—224	0	1	1
Total	526	474	1000

Mean maternal weight at 3 months (primiparae) = 123·69 lb.
S.D. = 18·26

Mean maternal weight at 3 months (multiparae) = 125·02 lb.
S.D. = 19·95

Mean maternal weight at 3 months (all cases) = 124·32 lb.
S.D. = 19·1

TABLE XI

THE FREQUENCY DISTRIBUTION OF MATERNAL WEIGHT AT THIRD MONTH OF PREGNANCY FOR DELIVERIES BETWEEN JULY 1954 AND MARCH 1958 INCLUSIVE

Maternal weight at 3 months (lb.)	*Primiparae*	*Multiparae*	*All cases*
84—	8	11	19
98—	49	54	103
112—	74	85	159
126—	63	59	122
140—	34	29	63
154—	6	12	18
168—	6	5	11
182—	3	1	4
196—210	0	1	1
Total	243	257	500

Mean maternal weight at 3 months (primiparae) = 126·09 lb.
S.D. = 18·89

Mean maternal weight at 3 months (multiparae) = 124·88 lb.
S.D. = 19·06

Mean maternal weight at 3 months (all cases) = 125·47 lb.
S.D. = 19·0

TABLE XII

THE PRE-PREGNANT MATERNAL WEIGHT

	Rationed group	*Unrationed group*	*Difference*
			St. error of diff.
Primiparae	123·69 lb.	126·09 lb.	1·66
Multiparae	125·02 lb.	124·88 lb.	0·093
All cases	124·32 lb.	125·47 lb.	1·104

Foetal Birth Weight

An interesting feature in the current series, though irrelevant to the main argument, is the foetal birth weight.

Tables XIII and XIV show the frequency distribution of foetal birth weight in both series. Boys, it will be seen, in both series

TABLE XIII

THE FREQUENCY DISTRIBUTION OF FOETAL BIRTH WEIGHT FOR BIRTHS IN THE PERIOD 1944—1951

Birth weight (lb.)	*Number of boys*	*Number of girls*	*Number all infants*
4 —	0	1	1
4½—	2	5	7
5 —	7	17	24
5½—	19	35	54
6 —	54	68	122
6½—	70	90	160
7 —	95	109	204
7½—	101	81	182
8 —	62	58	120
8½—	39	30	69
9 —	28	8	36
9½—	15	1	16
10 —	2	0	2
10½—	2	0	2
11 —	1	0	1
11½—12	2	0	2
Total	499	503	1002

Mean weight of boys at birth = 7·56 lb.
S.D. = 1·09
Mean weight of girls at birth = 7·15 lb.
S.D. = 0·95
Mean weight all infants at birth = 7·35 lb.
S.D. = 1·04

As the mean weight of boys at birth is more than the mean weight of girls by an amount which is 6·346 times the standard error of the difference, the difference may be regarded as being statistically significant.

weigh more than girls by an amount which is unlikely to have arisen by chance. There is, however, no significant difference between the birth weight in the rationed and unrationed series.

TABLE XIV

THE FREQUENCY DISTRIBUTION OF FOETAL BIRTH WEIGHT FOR BIRTHS BETWEEN JULY 1954 AND MARCH 1958 INCLUSIVE

Birth weight (lb.)	*Number of boys*	*Number of girls*	*Number all infants*
4 —	0	1	1
4½—	3	2	5
5 —	1	5	6
5½—	5	16	21
6 —	16	28	44
6½—	35	53	88
7 —	52	62	114
7½—	52	38	90
8 —	39	22	61
8½—	19	19	38
9 —	19	5	24
9½—	1	3	4
10 —	1	2	3
10½—	0	0	0
11 —11½.. ..	1	0	1
Total	244	256	500

Mean weight of boys at birth = 7·62 lb.
S.D. = 0·97
Mean weight of girls at birth = 7·24 lb.
S.D. = 0·99
Mean weight all infants at birth = 7·42 lb.
S.D. = 0·99

As the mean weight of boys at birth is more than the mean weight of girls by an amount which is 4·33 times the standard error of the difference, the difference may be regarded as being statistically significant.

Birth weight—both sexes.

The difference in mean birth weight between rationed and unrationed groups is 0·07 lb. As this is only 1·26 times the standard error of the difference, it may not be regarded as being statistically significant.

Incidence of Toxaemia

It will be seen from table XV that the loaded mean percentage of toxaemic cases in the rationed series is 19.58 per cent and in the unrationed series 25.46 per cent, thus illustrating a very significant increase in pre-eclamptic toxaemia in the unrationed series, compared with the rationed. So significant, in fact, is this increase that a correlation coefficient was calculated—see table XVI.

TABLE XV

THE PERCENTAGE INCIDENCE OF PRE-ECLAMPTIC TOXAEMIA IN THE YEARS 1944-51 AND 1954-58

Year	*Number of normal cases*	*Number of toxaemic cases*	*Total*	*Percentage toxaemic cases*
1944	13	4	17	23·54
1945	28	5	33	15·16
1946	46	6	52	11·54
1947	67	16	83	19·28
1948	86	21	107	19·63
1949	80	26	106	24·53
1950	108	21	129	16·28
1951	102	30	132	22·73
1954 July—Dec. inclusive ..	52	21	73	28·77
1955	141	51	192	26·57
1956	173	54	227	23·79
1957	148	50	198	25·25
1958 Jan.—Mar. inclusive ..	45	15	60	25·00

Loaded mean percentage of toxaemic cases 1944—51 = 19·58 per cent
S.D. = 3·82
Loaded mean percentage of toxaemic cases 1954—58 = 25·46 per cent
S.D. = 1·5

As the difference between the means is 37.02 times the standard error of the difference it may be regarded as being statistically significant.

TABLE XVI

MEAN ANNUAL ENERGY VALUE OF DIET AND MEAN ANNUAL PERCENTAGE INCIDENCE OF TOXAEMIA. CORRELATION COEFFICIENT

Percentage incidence toxaemia	*Energy value of diet—calories*							*Total*
	2300–	2350–	2400–	2450–	2500–	2550–	2600–2650	
10—	52	0	0	0	0	0	0	52
12—	0	0	0	0	0	0	0	0
14—	0	33	0	0	0	0	0	33
16—	0	0	0	129	0	0	0	129
18—	83	107	0	0	0	0	0	190
20—	0	0	0	0	0	0	0	0
22—	0	17	0	132	0	0	227	376
24—	0	0	106	0	0	258	0	364
26—	0	0	0	0	0	0	192	192
28—30	0	0	0	0	0	0	73	73
Total	135	157	106	261	0	258	492	1409

Mean incidence of toxaemia = 22·65 per cent S.D. = 4·15
Mean energy value of diet = 2516·4 calories S.D. = 106·9
Correlation coefficient = 0·73

Regression equation and coefficient.

$$\text{Diet in calories} - 2516{\cdot}4 = 0{\cdot}73 \frac{(106{\cdot}9)}{(4{\cdot}15)} (\text{percentage incidence toxaemia} - 22{\cdot}65)$$

Diet in calories = 18·8 (percentage incidence toxaemia) + 2090·4
Regression coefficient = 18·8

The correlation coefficient, 0.73, which is 27.39 times its standard error, and thus highly significant, bespeaks a fairly close relationship between the increase in caloric value of the diet and increase in toxaemia, within the diet range studied.

As a mathematical exercise the Regression Equation and Regression Coefficient are calculated which relates the degree of increase in expected toxaemia rate to unit increase in diet (see table XVI).

Discussion

There seems to be no doubt from the figures presented that within the range of energy values considered, increase in diet is, in some way, related to an increase in toxaemia. One is aware that with statistical techniques the application of a correlation coefficient or the simple relationship of two factors on a *post hoc ergo propter hoc* basis is fraught with possible error. In other words, if daily caloric intake increases in expectant mothers, and the rate of toxaemia increases, are we right in attributing one to the influences of the other, or are there yet other, and unrelated, factors at work? From the obstetrical point of view it would seem that there are many other factors at work but a relationship as close as the one demonstrated between diet and toxaemia, one noted in fact by many workers, is not easy to overlook.

How do these dietary influences operate? This is an even more difficult question to answer. It has been suggested that when diet increases, maternal weight increases, and that this is followed by an increase in toxaemia, of which the weight gain is an early and integral part. I find this very difficult to believe. Many authors, already quoted, have shown that an abnormal increase in maternal weight is an early manifestation of toxaemia, but this weight increase is due to water retention, and is surely unrelated to actual tissue increase in the laying down of extra fat on the part of the mother. Only normal cases have been studied in this series, but the dividing line between normal patients and toxaemic patients is nebulous and an increased weight gain in the unrationed series might be expected to match the increased incidence of toxaemia.

It has been suggested that the increased vascular field offered by maternal fatty deposition robs the placenta of some of its blood supply, thus setting in motion the processes of toxaemia. This series does not bear out that idea. Increased diet does increase the toxaemia rate but it does not, within the range considered, increase maternal weight gain. Thus the influences which operate in the production of toxaemia do so through some agency other than increase in tissue weight or simple adiposity in the mother. Is it possible that the range of dietary increase studied stimulates the endocrine system in some way so that adrenal hyperfunction

precipitates salt and water retention, which is the true basis of weight gain in toxaemia? This is a question which cannot at present be answered, but it seems certain that the simple laying down of fat is not an integral early part of the toxaemic process.

Summary and Conclusions

A study is made of 1,000 normal pregnancies between 1944 and 1951 and 500 normal pregnancies between July 1954 and March 1958. The diet is also considered in the former (rationed) period and the latter (unrationed) period and the diet in the latter period is shown to be of higher energy value than the former.

Comparison is made in the two periods between maternal weight gain during pregnancy, both in pounds and as a percentage of pre-pregnant maternal weight, and no significant difference is demonstrated between the periods.

Similarly no significant difference can be demonstrated between pre-pregnant maternal weight or foetal birth weight in the rationed and unrationed groups.

The incidence of toxaemia in both groups is studied and is shown to be significantly higher in the unrationed series. A correlation coefficient of 0.73 is calculated between the daily mean energy value of diet and percentage incidence of toxaemia, demonstrating a fairly close degree of relationship between increase in diet and increase in incidence of toxaemia.

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