

Effect of Nutrition on Pregnancy and Lactation *

C. GOPALAN, M.D., Ph.D.¹

Pregnancy and lactation constitute states of considerable physiological stress which impose increased nutritional demands. If these demands are not adequately met, it may be expected that not only the nutritional status of the subject will be affected, but also the course of pregnancy and lactation. While a great deal of work with experimental animals has been carried out to elucidate the role of nutrition in pregnancy and lactation, the question arises how far these experimental results are applicable to human subjects. The unfortunate nutritional situation prevalent in certain under-developed countries affords opportunities for the study of the effects of nutritional deficiencies on the course of pregnancy and lactation in the human subject. In this paper, the available literature on the effect of maternal nutrition on the course of pregnancy and the condition of the infant at birth is reviewed, as is the effect of the state of maternal nutrition on the output and chemical composition of milk in nursing mothers. The review reveals many important gaps in our knowledge and highlights the need for further work on this important problem.

Expectant and nursing mothers and infants and children constitute the most vulnerable segments of a population from the nutritional standpoint. In any consideration of the problems of undernutrition, these segments require special consideration. The unfortunate nutritional situation prevalent in many under-developed countries of the world today offers opportunities for studies on the effect of maternal malnutrition on the course of pregnancy, the condition of the infant at birth, and the state of lactation of the mother. Such studies may also be expected to throw light on maternal nutritional requirements during pregnancy and lactation.

NUTRITION AND PREGNANCY

The important questions which require to be considered under this head are:

1. How does the state of maternal nutrition affect the course of pregnancy?
2. How does the state of maternal nutrition affect the condition of the infant at birth and the neonatal period?

3. What are the optimal maternal nutritional requirements in pregnancy which would ensure proper health of the mother, normal course of her pregnancy and satisfactory condition of her infant at birth?

The course of pregnancy

Pregnancy wastage. A great deal of information is available pointing to the effect of nutrition on the course of pregnancy in experimental animals (e.g., Nelson & Evans, 1946, 1953). Nelson & Evans (1953) observed that in rats, when the dietary protein level was reduced to less than 5%, the incidence of foetal resorptions was as high as 70%-100%. The critical need for protein was apparent only during the earliest stages of placental and foetal development. The precise practical significance of these observations from the point of view of the human subject, however, requires cautious appraisal. A survey carried out in South India revealed that among poor women whose dietaries during pregnancy provided 1400-1500 calories and about 40 g of protein daily, nearly 20% of pregnancies had terminated in abortions, miscarriages or stillbirths.² This figure may well be an underestimate and may not include abortions in the early stages of preg-

* Paper submitted to the Joint FAO/WHO Expert Committee on Nutrition, April 1961.

¹ Director, Nutrition Research Laboratories, Hyderabad, India.

² Gopalan, C. Address to the 1960 Annual Meeting of the American Dietetic Association (to be published).

nancy. In a recent study among poor women in Hyderabad, Kalpakam Shankar¹ found that the incidence of pregnancy wastage was about 16%. Such high incidence of pregnancy wastage appears to be the usual finding among undernourished communities in other parts of India also. To what extent this is attributable to lack of proper obstetrical care, is, however, a moot question. The possible role of malnutrition in contributing to the high incidence of pregnancy wastage in undernourished communities would seem to need consideration.

Gain in body-weight during pregnancy. The relatively low gain in body-weight of undernourished women during pregnancy has been commented upon by some workers (Clements & Bocobo;² Venkatachalam et al., 1960; Gopalan³). While the usual weight gain during pregnancy among well-fed communities has been reported to be around 25% of the initial weight, Indian women of the poor socio-economic group weighing initially around 42 kg were found to gain on an average only about 6 kg during pregnancy.

The significance of the comparatively small gain in body-weight of undernourished women during pregnancy requires consideration. In pregnancy there are obviously profound changes in body composition. In a study of the body composition of eighteen undernourished pregnant women in South India (Venkatachalam et al., 1960), it was observed that between the 9th and the 14th weeks of gestation the total body water was about 56% of the body-weight, a value which corresponded to that observed in the non-pregnant women of the same population. Between the 20th and the 28th weeks of gestation, the total body water was about 66% and beyond the 28th week about 70%. The interesting finding was that the increase in body-weight of the subjects which would have been expected from the absolute increment of total body water was much greater than the actual increase in body-weight. Since nitrogen balance studies in these subjects indicated that they were in positive nitrogen balance on their usual protein intake, it was concluded that these women were actually losing body fat during pregnancy. The lower weight gain of these undernourished mothers as compared to well-fed mothers may be a reflection of smaller gain of body protein. Similar studies of

body composition of well-fed pregnant women may serve to decide this question, and may also serve to explain some of the contradictory findings with regard to the relationship between maternal body-weight changes during pregnancy and the birth weight of the infant.

The significance of the loss of body fat during pregnancy observed in the study reported above requires elucidation. The question is whether such loss of body fat is the result of undernutrition. In this connexion, the observations of Scitchik & Alper (1956) are interesting. These workers carried out studies of body composition in well-fed American pregnant women and concluded that the increase in lean body tissue in pregnancy was associated with a depletion of body fat, the fat being probably utilized for the synthesis of lean body tissue.

There is obviously a need for further studies of body composition in pregnancy in different socio-economic groups.

Toxaemias of pregnancy. A high incidence of eclampsia has been observed among pregnant women of the low socio-economic group in certain parts of India. It has been claimed from time to time that malnutrition may play a role in the development of toxæmia of pregnancy. The evidence for such a claim, however, needs careful evaluation.

Summing up an extensive survey of available data regarding the incidence of toxæmia in different parts of the world, Dieckmann (1952) observed: "Eclampsia, toxæmia, hyperemesis and abruptio placentae are either unknown or are very rare among the native women of Kenya, Uganda, Zululand, Tanganyika, Belgian Congo, Ethiopia, Persia, Java, Hawaii, British Malaya, Alaska, Australia and the date oases of Africa, where habits and diet have not been changed by the White race. In contrast, eclampsia is very common in Algiers, Cape Town, Colombo and Puerto Rico, whose natives have adopted many if not all of the diet and other habits of the White race." This may well be an over-simplification of the problem.

The reported incidence of pre-eclampsia and eclampsia in different parts of the world has been set out in Table 1. It will be noted that through the years there has been a reduction in the incidence of eclampsia in Europe, the USA and India, while the incidence of pre-eclampsia has not shown significant variation. The studies of Ebbs et al. (1941) and of the People's League of Health (1942) failed to show any significant effect of diet on the incidence of toxæmia. Nelson (1955) studied the incidence of eclampsia

¹ Data to be published.

² Unpublished report to WHO on nutrition in Ceylon, 1957.

³ Address to the 1960 Annual Meeting of the American Dietetic Association (to be published).

TABLE 1
INCIDENCE OF TOXAEMIAS OF PREGNANCY
IN DIFFERENT COUNTRIES^a

Country	Year	Pre-eclampsia (% incidence)	Eclampsia (% incidence)
British Isles	1938-40	9.3	1.04
	1947-50	11.5	0.632
Australia	1939-41	8.90	0.709
	1947-50	11.00	0.390
New Zealand	1936-40	6.3	0.5
	1947-49	7.0	0.56
USA:			
Boston Lying-in Hospital	1948-49	5.4	0.81
Margaret Hague Hospital, New York	1947-49	8.1	0.09
Chicago Lying-in Hospital	1947-50	8.5	0.84
Jackson Memorial Hospital	1956-57	7.43	0.113
India:			
Women and Children's Hospital, Madras	1936-38	4.4	2.1
	1955-57	7.4	1.7
	1959-60	7.8	1.2

^aData supplied by M. K. Krishna Menon (personal communication).

and pre-eclampsia among different socio-economic groups in Aberdeen. Diet surveys by Thomson (1959) show that the diets of these various socio-economic groups differed in quantity and quality. Despite such differences in the dietaries, Nelson's studies revealed that the incidence of pre-eclampsia was the same in all social classes. Nelson concluded that (a) the incidence of pre-eclampsia had not altered through the years; (b) the incidence of eclampsia had diminished considerably, and (c) the incidence of pre-eclampsia was the same in all classes. Dawson (1953) from his studies in Australia observed that while good antenatal care had almost eliminated eclampsia, it had not reduced the incidence of pre-eclampsia.

A careful appraisal of the available evidence thus fails to provide convincing proof that malnutrition is a significant factor contributing to pre-eclampsia. However, the high incidence of eclampsia in undernourished populations raises two possibilities: (a) that malnutrition may in some manner aggravate the course of pre-eclampsia, or (b) that the higher incidence of eclampsia in undernourished commu-

nities is largely the result of poor obstetric care, as a result of which cases of pre-eclampsia are not spotted early and treated adequately with the consequence that a high proportion of these develop into eclampsia. Obstetricians seem to be almost evenly divided between these two schools of thought. In communities with primitive obstetric care and poor nutrition, it should be possible to devise field experiments which would serve to clarify the position.

The condition of the infant

Birth weight and incidence of immaturity. There is now ample evidence pointing to the effect of maternal nutritional status on the birth weight of the infant. In a survey carried out in South India, the mean birth weight of infants of the low socio-economic group was found to be about 2.8 kg while that of infants of the high socio-economic group was about 3.1 kg.¹ This difference was found to be statistically significant. If all infants including those who were not born at full term had been included, the difference between the two groups would have been even wider.

Using a birth weight of 2.5 kg as the criterion, it was found that the incidence of immaturity among the infants of the low socio-economic group was nearly 30% while the corresponding figure among the high socio-economic group was about 14%. If a birth weight of 2.0 kg was used as the criterion, the incidence of immaturity was less than 2% among infants born to mothers in the high socio-economic group as compared to 10% in the poor socio-economic group.

What is the practical significance of the relatively low birth weight of infants of undernourished mothers? It may be safely conceded that a birth weight of less than 2.5 kg, or even 2.0 kg, need not necessarily indicate functional inadequacy. Indeed, most infants with such low birth weights were observed to thrive normally. It would appear, however, that while birth weight may not be a reliable clinical criterion in individual cases, it may be a useful yardstick when whole communities or population segments are taken into consideration. For example, in Madras City in South India, the infant mortality rate was 136 per 1000 live-births in the year 1954. Nearly 25% of these deaths occurred within seven days after birth and 40% within the first month. This neonatal mortality was found to contribute the bulk of infant mortality in this area. An analysis

¹ Gopalan, C. (1949) Unpublished data.

of the causes of neonatal deaths revealed that nearly 73% of all these deaths occurred in infants with birth weights of less than 2.0 kg. It would thus appear that birth weight in a community is an index of neonatal viability and that maternal nutritional factors which result in low birth weight also contribute to the high infant mortality observed in under-developed regions.

Congenital malformations. It is now well established that congenital malformations can be induced in the offspring of mammals by maternal dietary deficiencies. But congenital anomalies due to spontaneously occurring maternal nutritional deficiency have not been recognized in the human being. The available literature lends no support to the view that the incidence of congenital malformations in infants of malnourished mothers is significantly different from that in infants of well-fed mothers. The strict conditions and devices necessary to produce congenital anomalies in experimental animals have probably no counterpart in human reproduction. In contrast to the rat, the organogenetic period in which most malformations are determined is relatively short in the human subject; and it is followed by a long period of foetal growth in which damaged foetuses can be eliminated. The incidence of pregnancy wastage among malnourished mothers may indeed be a reflection of such elimination of damaged foetuses.

The syndrome of endemic cretinism may be justifiably looked upon as a manifestation of the effect on the offspring of maternal dietary deficiency of iodine, though it is possible that other factors may also contribute to its etiology. The results achieved in Switzerland in the matter of eradication of endemic cretinism through iodization of salt and other dietary improvement may be considered a convincing demonstration of the possible effect of maternal diet on the physical and mental make-up of the infant.

Warkany et al. (1959) have reported congenital anomalies following the ingestion of aminopterin by the mother during early pregnancy. This observation brings to light the possible danger of congenital anomalies resulting from the use of potent anti-metabolites during pregnancy.

Effect of maternal malnutrition on foetal storage of nutrients. The three major nutritional deficiency disorders in infancy and childhood encountered in many under-developed countries are protein malnutrition, hypovitaminosis A and anaemia. It is important to consider to what extent maternal mal-

nutrition during pregnancy may contribute to the development of these deficiencies in infancy and childhood.

It has been found that among poor pregnant women in South India, the vitamin A concentration in the serum declined from 104 IU in the first trimester to 68 IU in the third trimester.¹ After delivery, there was again a rise of serum vitamin A concentration. The serum carotene, however, actually showed a slight increase with advancing pregnancy. These observations are somewhat in line with those of other workers (Lund & Kimble, 1943). The vitamin A of cord blood from infants of these mothers was about 50 IU per 100 ml. On the other hand, cord blood obtained from a group of infants whose mothers had received 10 000 IU of vitamin A orally for a few days in the last trimester returned values averaging 85 IU per 100 ml.

The above observations would indicate that correction of dietary deficiency of vitamin A in the mother during pregnancy might significantly improve the nutritional status of the infant with regard to vitamin A. The available literature with regard to placental transfer of vitamin A and carotene reveals considerable differences in the behaviour of different species. In the rat the amount of vitamin A which passes into the foetal livers appears small and independent of the vitamin A fed to the mothers (Dann, 1932, 1934). In calves, on the other hand, the vitamin A content of the maternal diet has been found to determine the amount of vitamin A transferred to the foetal liver. Thus, while calves of undosed cows had 2.5 IU of vitamin A per g of liver, those of vitamin-A-supplemented cows had values as high as 142 IU (Walker et al., 1949). In pups, kittens, lambs, goats and pigs also it has been demonstrated that the foetal storage of vitamin A could be greatly improved by dosing the pregnant mother with vitamin A (Thomas et al., 1947).

Data with regard to the vitamin A content of livers of newborn infants among malnourished communities are unfortunately rare. Woo & Chu (1940) have reported that full-term infants in China showed values for hepatic vitamin A as low as 10 IU/g, while those of the USA (Lewis et al., 1943) and Finland (Skurnik et al., 1944) had a liver vitamin A content as high as 134 IU/g and 154 IU/g respectively. It is reasonable to expect that maternal malnutrition with respect to vitamin A, because of the resultant poor foetal hepatic storage of vitamin A,

¹ Venkatachalam, P. S., Bhavani Belavady & Gopalan, C. Data to be published.

may predispose the infant to vitamin A deficiency in late infancy and early childhood.

Another factor which may also be expected greatly to influence the hepatic storage of vitamin A in the infant is the pattern of feeding in the immediate neonatal period. It has been found that the vitamin A content of colostrum even among malnourished mothers is as high as 600 IU/100 ml. The vitamin A concentration in milk gradually declines to about 70 IU/100 ml. If the infant is put to breast immediately after birth, as is the case among many underfed communities, better hepatic storage may be expected than if the practice of not feeding the infant at the breast for the first three days—a habit in vogue in some other communities—is followed. Calves not allowed access to colostrum have been found to die within a few days. In such animals, the hepatic vitamin A storage has often been found to be too low to be measurable (Moore, 1957).

Field studies to determine how far the pattern of infant feeding in the early neonatal period influences the onset and course of vitamin A deficiency in children among different poor communities may yield valuable information.

The possible effect of anaemia in pregnancy on the haematological status of the infant at birth and in the neonatal period also deserves consideration. In a survey of the haemoglobin levels of nearly 400 pregnant women in South India, Kalpakam Shankar¹ found a progressive increase in the incidence and severity of anaemia with the advance of pregnancy. Thus, while 20% of subjects had haemoglobin levels less than 10 g% in the first trimester, over 50% had such low values in the later stages of pregnancy. Among the infants, the haemoglobin values decreased from about 18-20 g% at birth to about 10 g% by about the third month. After the sixth month, there was a further deterioration and by the 18th month the average haemoglobin level was around 8.5 g%. The studies of Bhavani Belavady & Gopalan (1959) had revealed that the iron content of milk of these mothers in the earlier stages of lactation was not lower than the values reported elsewhere, and further, that there was no correlation between haemoglobin levels of the nursing mothers and the iron content of their milk samples. In view of this, the high incidence of anaemia within the first six months may be attributable to inadequate foetal stores of iron, probably reflecting the unsatisfactory state of maternal nutrition with regard to

iron. It has been stated that foetal iron stores are deposited in the last trimester of pregnancy and that they are influenced by the mother's nutrition with respect to iron during pregnancy (Darby, 1950). The normal full-term infant born of a non-anaemic mother is believed to possess adequate iron stores at birth, so that a dietary supplement is not required for several months. However, conclusions regarding foetal storage of iron and its adequacy for the formation of new haemoglobin in the full-term infant are not in complete agreement. Early studies indicated that these iron stores compensate for the meagre iron content of milk. Later studies, however, show that the stores of non-haemoglobin iron in the newborn infant's liver are not as large as had been assumed (Stearns & McKinley, 1937; Smith et al., 1950). Gladstone (1932), on the basis of chemical and microscopic studies, saw no evidence of large iron deposits in the liver in the last four months of gestation, the maximum iron storage being reached from 1 to 10 weeks after birth. Langley (1951) confirmed this observation and showed that there was a progressive siderosis in the liver and spleen, starting three days after birth and reaching a maximum during the third week of life. The recession in extramedullary haemopoiesis and red blood cell haemolysis in the neonatal period are believed to be factors which increase neonatal iron stores. If, therefore, the neonatal iron store is derived to a great extent from haemoglobin iron, the question arises whether the content of haemoglobin iron in the newborn infants of undernourished mothers is inadequate. Studies on newborn infants of the poor socio-economic group (Jayalakshmi et al., 1957) had not revealed abnormally low haemoglobin concentration in these infants. But in the absence of data regarding the blood volume of these infants, no precise conclusion about the total haemoglobin iron is possible. The speed with which these infants develop anaemia in the neonatal period, is, however, highly suggestive of defective iron storage in the foetal or neonatal period.

Maternal nutritional requirements in pregnancy

Various national bodies have made recommendations regarding nutrient allowances in pregnancy. The scientific basis underlying some of these recommendations would seem to need careful evaluation. The same observations would also apply to the recommendations regarding nutrient allowances in lactation. The observations discussed above do indicate that the inadequate dietaries of pregnant

¹ Data to be published.

women in some under-developed countries are reflected in the condition of the mother as well as of the infant. While this is understandable, what is intriguing is the fact that a large number of these mothers are able to go through pregnancy apparently normally and to deliver normal infants of average birth weights. Furthermore, most of these mothers are able to breast-feed their infants successfully over long periods. This raises some interesting and important questions connected with the adaptive mechanisms brought into play during pregnancy and lactation to meet the increased nutritional demands of the mother. A great deal of further work is necessary to understand these mechanisms; and unless this is done a rational approach to the problem of maternal nutritional requirements during pregnancy and lactation would not be possible.

NUTRITION AND LACTATION

A descriptive account of the available data on this subject has been presented elsewhere (Gopalan & Bhavani Belavady, 1960). Some of these data may be highlighted here and some further observations may be briefly discussed.

Studies of the output of breast milk at different stages of lactation reveal that, in spite of their inadequate diets, women of the low socio-economic group put out 400-600 g of milk daily for periods extending to over a year. Studies of the chemical composition of milk show that as far as proximate principles are concerned, the milk from these mothers is nearly as satisfactory as those from well-fed mothers. The concentration of the vitamins, however, appears to be rather low in the milk samples from undernourished mothers. Apparently, the inadequate dietaries of these mothers are reflected in the concentrations of vitamins in their milk rather than in that of proximate principles. This conclusion is, however, applicable only if the diets supply at least between 45 g and 60 g of protein daily. It is possible that lower levels of protein intake may be reflected in the low concentration of protein in the milk.

Effect of protein supplementation

Dietary protein supplementation to the mother has not been shown to bring about any significant increase in protein concentration of the milk, where the diets already contained 50 g of protein daily. However, protein supplementation has been found to have an effect on two biochemical features of the

milk; the significance of these effects remains to be elucidated.

The creatine and creatinine concentration of the milk of poor Indian mothers was found to be very high as compared with figures reported from the USA. Thus, Bhavani Belavady (1959) observed that the creatinine N₂ was 2.3 mg/100 ml and creatine N₂ 3.4 mg/100 ml in milk samples from poor South Indian mothers. Karmarkar et al. (1959) in Western India observed values as high as 2.9 mg/100 ml and 8.4 mg/100 ml for creatinine N₂ and creatine N₂ respectively. These values are far higher than those reported by Macy & co-workers (1949), who observed a creatine N₂ concentration of 1.0 mg/100 ml and creatinine N₂ concentration of 1.0 mg/100 ml in milk samples from American women. Gopalan & Bhavani Belavady (1960) found that dietary protein supplementation to the mother brought about a significant reduction in the abnormally high creatine values in milk; the creatinine concentration, however, was apparently unaffected. Creatine in small amounts was excreted in the urine of the subjects. Protein supplementation had, however, no effect on creatine and creatinine output in urine. The serum creatine and creatinine figures in these subjects were normal. The significance of the high creatine content in milk and the effect of protein supplementation in bringing about a reduction thereof require further elucidation.

Another interesting effect of protein supplementation in these mothers was on the xanthine oxidase activity. Bhavani Belavady¹ observed that the xanthine oxidase activity of milk in these mothers was increased significantly after protein supplementation from 12.4 μ l O₂ consumed to 28.5/ μ l O₂ consumed per ml of milk per hour.

Thus, though protein supplementation had no significant effect on protein concentration in milk, it may be noted that it exerted a significant effect on two biochemical constituents of milk. Further, it was found that protein supplementation brought about a significant increase in body-weight and in serum albumin concentration of these mothers. These factors may have to be considered in assessing the adequacy of the dietary protein level in these cases.

Casein: whey protein ratio in milk

Lutz & Platt (1958) had reported that the casein: whey protein ratio in milk obtained from mal-

¹ Data to be published.

nourished Indian mothers was high as compared with the ratio in English mothers. Thus, these authors observed a ratio of 1.30 in Indian mothers and 1.08 in English mothers. On the other hand, studies carried out by Bhavani Belavady & Gopalan (to be published) showed that among poor Indian women in Coonoor, low values of about 0.3 were obtained for the casein: whey ratio. In Hyderabad, however, using the same techniques, values of about 1.08 were observed. The reasons for this striking difference are not clear. Among the women of Hyderabad in whom the effect of protein supplementation was studied, the casein: whey ratio was found to be unaltered by protein supplementation. It must, however, be pointed out that these mothers were not so malnourished as those evidently investigated by Lutz & Platt. There is obviously further scope for work on this interesting aspect.

Vitamins

Bhavani Belavady & Gopalan (1960; and data to be published) found that dietary supplementation with riboflavin, thiamine and ascorbic acid brought about a significant increase in the concentration of these vitamins in milk. On the basis of these supplementation studies, the authors concluded that the minimum dietary intake of riboflavin and ascorbic acid necessary to maintain a maximal concentration of these vitamins in milk was of the order of 3.0 mg and 200 mg respectively.

With regard to vitamin A, however, it was observed that dietary supplementation with vitamin A was not reflected in the concentration of the vitamins in milk even when the supplementation was maintained for 6-9 months. However, it was found that in mothers who became pregnant in the course of lactation, dietary supplementation with vitamin A had a profound effect on vitamin A concentration in milk. Thus, in two such cases it was noticed that the vitamin A concentration in milk rose from 60 IU/100 ml to 530 IU/100 ml and from 75 IU/100 ml to 240 IU/100 ml after four weeks of supplementation with 5000 IU of vitamin A daily. If this interesting observation is confirmed by more extended work, it would mean that the metabolic changes in pregnancy facilitate transfer of vitamin A across the mammary gland. To elucidate the possible role of oestrogens in facilitating the transfer of vitamin A to milk, some studies are currently in progress in the Nutrition Research Laboratories, Hyderabad.

TABLE 2
RESUMPTION OF MENSTRUATION AFTER DELIVERY IN
SOUTH INDIAN MOTHERS

Age of child (months)	Percentage of mothers who had resumed menstruation	
	Poor	Well-to-do
1-3	0	22
4-6	8	84
6-9	25	88
10-12	45	100
13-15	47	100
16-18	70	100

Minerals

The concentration of calcium and iron in the milk samples obtained from women of the poor socio-economic group in South India were found to be around the reported normal range in other parts of the world (Bhavani Belavady & Gopalan, 1959). This is interesting considering the fact that the diets of these mothers supplied hardly 300 mg of calcium daily and the majority of the mothers were anaemic. Supplementation of the diet with calcium and iron did not bring about an increase in the concentration of these nutrients in milk; indeed, paradoxically enough, there was actually a fall in the concentration of these nutrients. This point has been discussed in earlier communications (Bhavani Belavady & Gopalan, 1959, 1960).

Resumption of menstruation during lactation

A recent study¹ has revealed interesting differences between undernourished and well-fed mothers with regard to the time-interval between delivery and resumption of menstruation. The data are set out in Table 2. It will be seen that menstruation was resumed much later in the majority of the poor mothers than in the well-to-do. The significance of this finding, is, however, hard to assess. Whether early resumption of menstruation in the well-fed mothers is the result or the cause of their failure of successful lactation remains to be elucidated.

Discussion

The observations discussed above show that the nutritional status of the mothers is reflected in the

¹ Bhavani Belavady. Data to be published.

TABLE 3
INTAKE OF MILK BY SOUTH INDIAN BABIES ^a

Day after delivery	Milk intake* (ml) in 24 hours by babies with birth weight of:	
	4-5 pounds	8-9 pounds
2nd	80	80
3rd	200	310
4th	260	400
5th	310	430
6th	330	450
7th	360	510
8th	350	540
9th	370	540
10th	360	540

^a Reproduced, by permission, from Athavale (1960).

chemical composition of the milk, at least with regard to the concentration of vitamins. It will be important to know to what extent the growth of infants in these communities could be improved through supplementation of the maternal diet with vitamins. The growth-curve of the infants in these

poor communities is nearly parallel to that of infants from the better-fed groups, at least in the first few weeks, but the initial difference in body-weight is maintained. Perhaps supplementation of the maternal diet with vitamins may actually help to overcome this difference. It may be possible to investigate this question through carefully controlled experiments. The work of Widdowson (quoted by Gyorgy, 1960) has indicated that the initial growth retardation resulting from undernutrition in the neonatal period may be irreversible. If this is so, it will be important to explore the ways by which the growth rate of these infants in the early neonatal period could be improved.

In this connexion, the observations of Athavale (1960) appear interesting; they suggest that the amount of breast milk consumed by the infant in 24 hours may be determined by the body-weight of the infant, as shown in Table 3. This requires to be confirmed by extended studies as its implications appear important.

The foregoing account shows that there are still important areas in which further work is necessary to elucidate the precise role of nutrition in pregnancy and lactation. Such work deserves high priority in view of the widespread maternal and infant malnutrition prevalent in many under-developed regions of the world today.

RÉSUMÉ

Les femmes enceintes et les mères allaitantes, les nourrissons et les jeunes enfants constituent les secteurs les plus vulnérables de la population, du point de vue alimentaire. En effet, des dommages irréparables peuvent survenir si les exigences alimentaires accrues durant ces périodes de la vie ne sont pas satisfaites. La sous-alimentation, dans plusieurs pays insuffisamment développés, permet d'étudier l'effet de la malnutrition maternelle sur le cours de la grossesse, l'état de l'enfant à la naissance, la lactation, et de préciser les exigences alimentaires en rapport avec la physiologie de la femme.

En s'appuyant sur des observations antérieures, sur les expériences faites chez l'animal, et sur ses propres recherches, dans l'Inde, l'auteur aborde les questions suivantes: l'influence de l'alimentation sur le cours de la grossesse et l'état du nouveau-né; les exigences alimentaires qui assureraient une grossesse menée à terme et la naissance d'un enfant ayant de bonnes chances de viabilité. La proportion d'avortements naturels dans les populations sous-alimentées de l'Inde est estimée à 16-20%. La part due au manque de soins obstétriques, dans

ce chiffre, n'est pas connue; celle qui revient à la malnutrition devrait être évaluée. Le gain en poids de la mère pendant la grossesse, qui est de $\frac{1}{4}$ du poids initial dans les collectivités bien nourries, est d'environ $\frac{1}{7}$ dans les collectivités mal nourries; cette différence semble due à une plus grande déperdition de protéines, d'eau et de graisse du corps dans le second groupe.

Quant à la prééclampsie, elle ne paraît pas dépendre directement de la malnutrition. Toutefois, pour expliquer la fréquence élevée de l'éclampsie dans les collectivités mal nourries, on peut avancer que a) la malnutrition aggrave l'évolution de la prééclampsie, ou que b) faute de soins obstétricaux, la prééclampsie n'est pas décelée ou traitée à temps, et qu'elle évolue le plus souvent en éclampsie. Les obstétriciens sont eux-mêmes divisés sur ce point.

Il existe une différence significative entre le poids à la naissance des enfants mis au monde dans les collectivités aisées et dans celles qui sont défavorisées. La différence serait encore plus grande si l'on tenait compte des avortements naturels. L'insuffisance de poids à la naissance

(moins de 2,5 kg) est de l'ordre de 30% des naissances dans les groupes socio-économiquement faibles et de 14% dans les groupes plus favorisés. Cette proportion est respectivement de 10% et 2% si l'on applique le critère de 2 kg de poids initial. Si l'on peut admettre, dans les cas individuels, qu'un poids à la naissance de 2-2,5 kg n'indique pas nécessairement l'inviabilité, ce chiffre donne une indication utile lorsque l'on considère la mortalité néonatale dans l'ensemble d'une population. A Madras, par exemple, la mortalité néonatale intervient pour une large part dans la mortalité infantile, qui était de 136⁰/₁₀₀ en 1954; l'analyse des causes de mortalité néonatale montre que 73% des décès survenaient chez des enfants qui pesaient moins de 2 kg à la naissance. Dans une collectivité, le poids à la naissance est un indice de viabilité néonatale, et les facteurs nutritionnels qui concourent à un poids insuffisant du nouveau-né concourent aussi à élever la mortalité infantile dans les régions sous-développées.

Des malformations congénitales dues à la malnutrition maternelle n'ont pas été signalées dans l'espèce humaine. La fréquence des malformations congénitales ne paraît pas plus élevée dans les collectivités mal nourries. Con-

trairement à ce qui se passe chez le rat, la période d'organogenèse durant laquelle prennent naissance les malformations est relativement courte chez l'homme et, au cours de la longue période fœtale qui suit, les fœtus anormaux peuvent être éliminés. Il se peut qu'une partie des avortements naturels chez les femmes mal nourries correspondent à l'élimination de ces fœtus non viables.

Le rapport entre les trois déficits nutritionnels les plus importants chez le jeune enfant — l'hypovitaminose A, l'anémie ferriprive et la carence protéique —, et le régime alimentaire de la mère durant la grossesse est discuté.

L'état nutritionnel de la mère se reflète dans la composition chimique du lait, du moins sa teneur en vitamines. Il sera intéressant de déterminer dans quelle mesure on peut combler ces déficits par l'administration de vitamines. Selon certaines observations, le retard de croissance durant la période néonatale, dû à l'alimentation déficitaire de la mère, serait irréversible. Il y a lieu d'examiner de quelle façon on pourrait améliorer les conditions de l'enfant au cours de cette période décisive.

Au cours de son exposé, l'auteur signale les nombreux points qui demandent encore à être étudiés, et suggère des sujets de recherches.

REFERENCES

- Athavale, V. B. (1960) *Indian J. Child Hlth*, **9**, 75
 Bhavani Belavady (1959) *Indian J. med. Res.*, **47**, 217
 Bhavani Belavady & Gopalan, C. (1959) *Indian J. med. Res.*, **47**, 234
 Bhavani Belavady & Gopalan, C. (1960) *Indian J. med. Res.*, **48**, 518
 Dann, F. W. (1932) *Biochem. J.*, **26**, 1072
 Dann, F. W. (1934) *Biochem. J.*, **28**, 634
 Darby, W. F. (1950) *J. Amer. med. Ass.*, **142**, 1288
 Dawson, B. (1953) *J. Obstet. Gynaec. Brit. Emp.*, **60**, 80
 Dieckmann, W. J., (1952) *The toxemias of pregnancy*, 2nd ed., St. Louis, Mosby
 Ebbs, J. H., Tisdall, F. F. & Scott, W. A. (1941) *J. Nutr.*, **22**, 515
 Gladstone, S. A. (1932) *Amer. J. Dis. Child.*, **44**, 81
 Gopalan, C. & Bhavani Belavady (1960) *Fed. Proc.*, **20**, Suppl. 7, p. 177
 Gyorgy, P. (1960) *Amer. J. clin. Nutr.*, **8**, 344
 Jayalakshmi, V. T., Ramanathan, M. K. & Gopalan, C. (1957) *Indian J. med. Res.*, **45**, 4
 Karmarkar, M. C., Chokshi, H. R., Shah, V. K. & Ramakrishnan, C. V. (1959) *J. Pediat.*, **55**, 481
 Langley, F. A. (1951) *Arch. Dis. Childh.*, **126**, 64
 Lewis, J. H., Bodansky, O. & Shapiro, L. M. (1943) *Amer. J. Dis. Child.*, **66**, 503
 Lund, C. J. & Kimble, M. S. (1943) *Amer. J. Obstet. Gynec.*, **46**, 486
 Lutz, P. & Platt, B. S. (1958) *Proc. Nutr. Soc.*, **17**, 3
 Macy, I. G. (1949) *Amer. J. Dis. Child.*, **78**, 589
 Moore, T. (1957) *Vitamin A*, Amsterdam, Elsevier, p. 240
 Nelson, M. M. & Evans, H. M. (1946) *J. Nutr.*, **31**, 497
 Nelson, M. M. & Evans, H. M. (1953) *J. Nutr.*, **51**, 71
 Nelson, T. P. (1955) *J. Obstet. Gynaec. Brit. Emp.*, **62**, 49
 People's League of Health (1942) *Lancet*, **2**, 10
 Scitchik, J. & Alper, C. (1956) *Surg. Clin. N. Amer.*, **34**, 1535
 Skurnik, L., Heikel, H. & Westerberg, T. U. (1944) *Z. Vitaminforsch.*, **15**, 68
 Smith, C. H., Sisson, T. R. C., Floyd, W. N. & Siegel, S. (1950) *Pediatrics*, **5**, 799
 Steams, G. & McKinley, J. B. (1937) *J. Nutr.*, **13**, 143
 Thomas, J. W., Loosh, J. K. & William, J. P. (1947) *J. Anim. Sci.*, **6**, 141
 Thomson, A. M. (1959) *Brit. J. Nutr.*, **13**, 190
 Venkatachalam, P. S., Kalpakam Shankar & Gopalan, C. (1960) *Indian J. med. Res.*, **48**, 511
 Walker, D. M., Thomson, S. Y., Bartlett, S. & Kon, S. K. (1949) *The effect of diet during pregnancy on the vitamin A and carotene content of the colostrum of cows and heifers and on the reserves of the calf*. In: *Proceedings of the 12th International Dairy Congress*, Stockholm, p. 83
 Warkany, J., Beaudry, P. H. & Hornstein, S. (1959) *A.M.A. J. Dis. Child.*, **97**, 274
 Woo, T. T. & Chu, F. T. (1940) *Chin. J. Physiol.*, **15**, 83