Chapter 4 Nutrition in Pregnancy and Growth of the Fetus

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Anatomical and physiological changes occur in the body of the mother during pregnancy to create a suitable environment for the growth of the fetus. A complex series of endocrinological and metabolic changes also take place which facilitate the handling of nutrients by the body tissues of the mother as well as their transfer to the fetus. These endocrinological responses provide the basis for the "metabolic economies' described in the pregnant state and evidenced by gain in weight. For example, nitrogen balance studies in healthy primigravidae indicate that a positive nitrogen balance is established after week 12.

In laboratory rats analyses of body composition at different stages of pregnancy have shown that in early and mid-pregnancy considerable storage of protein occurs. At this time, of course, there are only negligible competitive demands from the fetus. During this phase of anabolism, the increase of muscle nitrogen is about 9 per cent, which is equal to half the amount of nitrogen in the products of conception at term. In late pregnancy, when the fetus is growing rapidly, this protein reserve is called upon so that at parturition no net gain of protein is found. This change to the catabolic phase is induced by the hormones of the growing placenta and occurs irrespective of the protein intake. Thus in rats maternal muscle mass acts as an important reservoir for protein during pregnancy.

Similar studies are difficult to conduct in humans, but studies of the amino acid 3-methylhistididine show similar results. This amino acid is liberated during the turnover of protein in muscle, but unlike other amino acids it cannot be utilized and is excreted in urine. The excretion rate rises in late pregnancy, indicating muscle catabolism. These changes in protein metabolism, comprising early conservation followed by utilization, are similar to those in energy metabolism. Together they help to minimize the effects of inadequate diet during pregnancy on fetal growth.

The fetus is comparatively small in the early stages of pregnancy, even though there is rapid cellular multiplication and differentiation. It is not until the third trimester that fetal growth has reached a stage where nutrients are required in appreciable quantities. Studies in healthy and undernourished women show that, up to the last trimester, there is very little difference in fetal weight between the two groups. Significant changes in weight occur mainly between the twenty-fifth and fortieth weeks of pregnancy. (See Fig. 4.1)
EFFECTS OF THE PREVIOUS HEALTH AND NUTRITIONAL STATUS OF THE MOTHER

The growth of the fetus can be regarded as a result of the interaction between its genetic potential and the intrauterine environment. Mothers who enter pregnancy in good health, with sound reproductive physiology and who have not suffered chronic illness or nutritional deprivation in childhood will have larger and healthier infants than mothers who do not have such advantages. Chronic undernutrition in childhood with or without recurrent ill-health is largely responsible for stunting of adult stature. Improvements in health in all the affluent societies of the West are reflected in increase of adult stature. By comparison, the adult female in the developing countries tends to be of short stature and small body build (table 3.1). Thus, there is a significant difference in
average birth weights between babies born in affluent societies and those born in the developing countries. Within a society birth weights tend to be higher in the upper socioeconomic groups compared with the lower, and this is in keeping with the differences in several other health indices in the groups (table 4.2).
Table 4.2 Birth weight and social class

<table>
<thead>
<tr>
<th>Place</th>
<th>Population</th>
<th>Subject</th>
<th>Mean birth weight (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Madras</td>
<td>Indian</td>
<td>Well-to-do</td>
<td>2985</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mostly poor</td>
<td>2736</td>
</tr>
<tr>
<td>South India</td>
<td>Indian</td>
<td>Wealthy</td>
<td>3182</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poor</td>
<td>2810</td>
</tr>
<tr>
<td>Bombay</td>
<td>Indian</td>
<td>upper class</td>
<td>3247</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Upper middle class</td>
<td>2945</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lower middle class</td>
<td>2796</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lower class</td>
<td>2578</td>
</tr>
<tr>
<td>Ghana</td>
<td>African</td>
<td>Prosperous</td>
<td>3188</td>
</tr>
<tr>
<td></td>
<td></td>
<td>General population</td>
<td>2879</td>
</tr>
<tr>
<td>Tanzania</td>
<td>African</td>
<td>upper class</td>
<td>3150</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lower class</td>
<td>2700</td>
</tr>
<tr>
<td>Indonesia</td>
<td>Javanese</td>
<td>Well-to-do</td>
<td>3022</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poor</td>
<td>2816</td>
</tr>
<tr>
<td>Britain</td>
<td>National Cohort1958</td>
<td>Social class I-II</td>
<td>3380</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Social class V</td>
<td>3290</td>
</tr>
</tbody>
</table>


Weight at birth is also influenced by factors operating during pregnancy. Serious illnesses, complications of pregnancy, nutritional deprivation, emotional and psychological stress can all influence the growth of the fetus through their adverse effect on the mother, or by interfering with placental growth and transport of nutrients to the fetus. Fig.4.2 is a summary of these observations.
Our knowledge of the nutritional requirements for growth in the human fetus has developed from a variety of studies. The relationship between food intake in the mother and its effect on the offspring is best seen in animal experiments, especially in those species where the period of gestation is relatively short and a correlation between dietary intake and fetal growth can be readily seen. There have been epidemiological studies in women comparing birth weights in different social groups, and similar observations during famine and war, which show the effects of acute food shortages on fetal growth. More recently there have been several well-documented studies of nutritional intervention in pregnancy. All such studies indicate that most of the growth in the size of the fetus occurs in the latter part of pregnancy. For example, at the end of the third month of gestation, the fetus weighs approximately 30 g. By comparison, towards the end of gestation the fetus is daily laying down 500 mg of nitrogen (equivalent to 3 g of body tissue), over 300 mg of calcium and 200 mg of phosphorus. The maximum rate of fetal growth is during 32-38 weeks of pregnancy when the weight virtually doubles. (See Fig. 4.3)
Figure 4.3  Distribution of pregnancy weight gain between maternal stores, foetus and placenta.

In the healthy mother an increase in body fat accumulation occurs during early two-thirds of pregnancy. Then in late pregnancy there is accelerated breakdown of fat depots and this plays a key role in fetal growth. The foetus benefits not only from fatty acids arising from breakdown of maternal fat depots and transferred across the placenta, but also from glycerol and ketone bodies. Glycerol crosses the placenta in only small amounts but it is a useful substrate for maternal gluconeogenesis, and maternal glucose is quantitatively the main substrate crossing the placenta. Ketone bodies travel across the placenta to the foetus with ease and can be used by the foetus as fuel for oxidative metabolism as well as substrate for lipogenesis. Triglycerides in the maternal blood do not cross the placental barrier, but the presence of lipoprotein receptors in the placenta as well as lipoprotein lipase and other enzymes allows the release of polyunsaturated fatty acids transported as triglycerides in maternal plasma. Free fatty acids in the maternal plasma are also an important source of long chain poly unsaturated fatty acids (LCPUFA) to the foetus. Their placental uptake occurs by a selective process. This selectivity of placental transfer ensures a higher concentration of LCPUFA in foetal circulation compared to maternal. The fatty acid mix delivered to the foetus is largely determined by the fatty acid composition of the maternal blood although as we have seen the placenta is able to preferentially transfer the important PUFA to the foetus as a result of selective uptake by the syncytiotrophoblast and selective export to the foetal circulation. Most of the LCPUFA accumulated by the foetus is stored in the adipose tissue for use in early post natal life.
NUTRITIONAL REQUIREMENTS OF THE FETUS

Proteins
The placenta transports protein primarily as amino acids which are then synthesized by the fetus into tissue proteins. Nitrogen balance studies during pregnancy in healthy English women show an average excess of intake over loss of 92 g; since 50 g are needed for the growth of fetal and placental tissues, there is a margin of safety of 42 g, or 45 per cent. By comparison, dietary studies in undernourished Indian women indicate that they have a safety margin of only 6 g, or 12 per cent.

Fat
Most of the 500 g of fetal body fat is deposited between the thirty-fifth and fortieth weeks of pregnancy, about half of this between the thirty-fifth and thirty-eighth weeks. Close to term fat deposition can reach a rate of 7 g/day. In the early stages of gestation there is no fat laid down apart from essential lipids and phospholipids for the central nervous system and the cell walls. Until the middle of gestation there is only about 0.5 per cent fat in the body of the fetus, after which the amount increases, reaching 7.8 per cent at the thirty-fourth week of gestation and 16 per cent before birth. During the last month of intraterine life as much as 14 g of fat per day is laid down. Of this, placental transport of fatty acids accounts for 40 per cent of fetal fat; the remainder is synthesized by the fetus. Table 2.3 gives the amounts of protein and fat in the normally developing fetus. Both protein and fat increase rapidly in the last 3 months of pregnancy together with the weight of the foetus. Most of the fat is deposited in the subcutaneous tissue so that at term 80 per cent of the body fat is subcutaneous. Fetuses that are small for gestational age have less fat than larger ones.

Table 4.3  Protein and fat in the fetal body

<table>
<thead>
<tr>
<th>Fetal age (weeks)</th>
<th>12</th>
<th>16</th>
<th>20</th>
<th>24</th>
<th>28</th>
<th>32</th>
<th>36</th>
<th>40</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>0.02</td>
<td>0.1</td>
<td>0.3</td>
<td>0.75</td>
<td>1.35</td>
<td>2.0</td>
<td>2.7</td>
<td>3.5</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>1.1</td>
<td>6.3</td>
<td>22.5</td>
<td>65</td>
<td>123</td>
<td>189</td>
<td>227</td>
<td>446</td>
</tr>
<tr>
<td>(derived by N x 6.25)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat (g)</td>
<td>0.1</td>
<td>0.6</td>
<td>2.7</td>
<td>13.1</td>
<td>47.2</td>
<td>120</td>
<td>250</td>
<td>525</td>
</tr>
</tbody>
</table>


Water
Accompanying the accumulation of fat there is a steady fall in the percentage of body water. The very small fetus has a water content of about 94% of body weight in the form of extra cellular fluid which reduces to about 75% of body weight at the end of pregnancy. The distribution of body water between the different fluid compartments also changes over the period with gradual diminution of extra cellular water and expansion in intracellular water associated with increasing total cell mass in the body.
Carbohydrate
The fetus has about 9 g of carbohydrate at the thirty-third week of gestation, and at birth it rises to 34 g. The concentration of glycogen in the liver and skeletal muscles increases during the latter part of gestation, as shown in table 3.4. For all nutrients, the fetus and placenta together contain less than 10 per cent of the amounts present in the non-pregnant woman weighing 60 kg.

Table 4.4 Concentration of carbohydrate (g per 100 g)

<table>
<thead>
<tr>
<th>Maturity (weeks)</th>
<th>Heart</th>
<th>Liver</th>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>31</td>
<td>0.76</td>
<td>0.98</td>
<td>1.63</td>
</tr>
<tr>
<td>40</td>
<td>1.01</td>
<td>3.92</td>
<td>2.67</td>
</tr>
</tbody>
</table>

ROLE OF THE PLACENTA

The placenta plays an important role in the transfer of nutrients from the mother to the fetus. It is not just an organ of simple transport, but is also able to take up nutrients selectively and either process or resynthesize them before they reach the fetus. The supply of nutrients to the growing fetus depends upon both the amount of maternal blood flowing through the placenta and the food substances carried by it. The efficiency with which the placenta can concentrate, synthesis and transport essential nutrients will also determine the food supply to the fetus. (See Fig.4.4)
Factors which regulate fetal growth also regulate the growth of the placenta. Thus the metabolic work of the placenta depends upon the growth capabilities of the fetus and not the other way around. Fetal malnutrition commonly arises either from an insufficient supply from the mother due to some vascular abnormality of the placenta, or from nutritional deficiency in the mother, and only occasionally from reduced placental transport of nutrients. The nature, timing and duration of nutritional deficit is thought to account for the different degrees of intrauterine growth retardation expressed as different forms of fetal malnutrition. It has been estimated that in a third to a half of "small-for-dates' infants (i.e. with birth weight less than 2500 g) the period of gestation is actually more than 37 weeks; thus, the reduction in birth weight is due to growth-retardation rather than immaturity. (See Figs. 4.5 and 4.6)
Figure 4.6  World distribution of low birth weight newborns.
MECHANISM OF PLACENTAL TRANSFER

Fetal blood flows through villi in which the capillaries are covered only by fetal tissue which bathes directly in the intervillous space in a pool of flowing maternal blood. Various parts of the placenta are actively involved in the transfer, processing and synthesis of nutrients under the influence of maternal, fetal and placental hormones. Several modes of transport across the placenta have been described. Relatively lipid soluble molecules such as respiratory gases, anesthetic agents, several drugs and unconjugated bilirubin cross easily by penetrating the cell membrane. Small water soluble molecules such as urea and water also cross easily by diffusion or osmosis. Transfer of glucose is facilitated by a specific carrier molecule but not actively transported. Most amino acids, calcium and potassium as well as phosphorous are transported from mother to fetus by specific carrier mediated processes which consume energy, and carry out active transport leading to higher concentration in fetal than in maternal plasma. IgG, iron and vitamin B₁₂ are transported by means of a receptor mediated mechanism into the fetal circulation. (See Fig. 4.7)

Permeability of lipid insoluble substances is hardly affected by changes in the rate of blood flow. Their passage is ‘membrane limited’ and control of their transfer is largely by a change in placental tissue carrier mechanisms.

Large increases in placental blood flow as gestation proceeds are important so that the transport of respiratory gases can be increased to meet the needs of the growing fetus. Short term modulation of blood flow does not seem to be an important physiological control mechanism, but reduction may reduce gas transfer.

Figure 4.7 Fully developed placenta
The gradient is not directly from maternal to fetal blood, but from maternal blood to the maternal side of the placenta, where proteins, enzymes, nucleic acids, etc. may be synthesized. Further conversion and synthesis occurs in the fetal part of the placenta. A multiplicity of factors affect the transport of amino acids across the placenta. These are activity and location of amino acid transporter systems, changes in the systems as pregnancy advances, changes in the surface area of the placenta, utero-placental blood flows and concentration of the amino acids in maternal blood. (See Figure 4.8).

Carbohydrate is the principal metabolic fuel of the fetus and is provided in continuous supply by transfer of glucose from the mother through the placenta. On the other hand, fat is not a main source of energy in the fetus, so there is only a slow limited transfer of fatty acids across the placenta. Cell growth in the fetus is assumed to result from fetal synthesis of proteins from the amino acids transferred through the placenta.

It has been consistently observed in several mammalian species that placental size at term always correlates with birth weight. This goes to say a great deal about the essential role of the placenta. Besides transfer of nutrients it is also a site of major endocrine activity, including synthesis of a broad range of steroid and peptide hormones, growth factors, cytokines and other biologically active compounds. The best known are chorionic gonadotrophin. And growth hormone (GH) / prolactin like hormones. Placental growth hormones are essential for adapting the maternal metabolism in pregnancy, for normal placental development, and thereby for foetal growth.

Malnourished women, or women in lower socioeconomic groups in developing countries, have a lower mean placental weight compared to well-nourished women or those from higher social groups. In various studies the decrease in mean placental weight has ranged from 14 to 50 per cent.
Placentas from malnourished women have reduced DNA content and protein/DNA ratio. Since DNA is a measure of the number of cells in an organ, this indicates that the number of cells in the placentas of malnourished women is reduced. Morphologically there is a reduced villous surface and thus a diminished area for maternal-fetal exchange. The birth weight of a singleton infant correlates significantly with both the wet and dry weight of the placenta.

**Malarial infection and the placenta.**

In endemic areas malarial infection of the placenta is common and is particularly severe in primigravidae. A subpopulation of *Plasmodium falciparum* has a tendency to adhere and massively sequester in the placenta. Histologically, parasites and leucocytes are found within the intervillous spaces with fibrin deposits. There is proliferation of the cytotrophoblast cells and thickening of the trophoblast basement membrane. (See fig 4.9)

![Figure 4.9 malarial infection of the placenta](image)

The exact mechanism of impaired materno-foetal exchange is not known but it has been postulated that thickening of the trophoblast basement membrane causes mechanical blockage of oxygen and nutrient transport across the placenta. Malarial infection of the placenta is associated with intrauterine growth retardation leading to low birth weight. Antimalarial chemo-prophylaxis during pregnancy significantly reduces placental malaria and prevents low birth weight.

At particular risk of developing severe, life threatening malaria associated complications are women during their first pregnancy. The reported pathologies such as pre term delivery, abortion, intrauterine growth retardation, and death of the mother and the newborn are in large part due to the parasite’s ability to turn infected erythrocytes adhesive and sequester in the intervillous spaces of the placenta. In endemic areas the primigravidae should be considered for antimalarial prophylaxis during the pregnancy.
NUTRITION OF THE MOTHER

During pregnancy maternal metabolism is adjusted by means of a number of hormones that serve as mediators, redirecting nutrients to highly specialized maternal tissues specific to reproduction like placenta and the mammary glands. The increased need for nutrients cannot always be met from the maternal diet, however ample it may be. The requirement for energy providing macronutrients increases modestly compared with several micronutrients that are unevenly distributed among foods. Hence altered nutrient utilization and mobilization of reserves come into play to balance enhanced needs. But this is not always successful, and in communities existing on marginal nutrition nutrient deficiencies are precipitated by repeated reproduction. The transfer of bone forming minerals like calcium, phosphorous, magnesium and zinc across the placenta and later through breast milk place considerable demands on maternal mineral economy. Increase in food consumption, more efficient gastrointestinal absorption decreased mineral excretion and mobilization of tissue stores are the several biological mechanisms that come into play to meet these extra mineral requirements.

The state of the mother’s physiology, especially reproductive physiology, at the time when she commences a pregnancy, has considerable influence on the growth of the fetus. Several studies provide evidence for the relationship between adult size, reproductive efficiency and socioeconomic status. In general, the baby of a short woman is lighter and has less vitality and a lower survival than that of a tall woman. Stunting in the mother cannot be overcome by a good diet in pregnancy, and the same applies to reproductive efficiency. It is axiomatic that preparation for pregnancy should begin with good nutrition and health care in childhood so that women enter motherhood having achieved optimal growth and health. For many years it has been known that the height of the mother is closely related to birth weight and pregnancy outcome, and this was amply demonstrated in surveys of perinatal mortality in the UK in 1958 and 1970. These surveys have also revealed that there are more shod mothers in the lower socioeconomic groups, which means that inadequate nutrition and larger number of illnesses prevent many girls in this social group from achieving optimal physique. They are thus at a disadvantage as regards childbearing. Similar studies in the United States, conducted by the National Institutes of Health, have shown that mothers who weigh more than 150 lb (68 kg) at conception or who gain more than 30 lb (13.6 kg) in weight during pregnancy tend to have larger and healthier babies with a lower perinatal mortality compared to mothers who weigh less or gain less weight than above.

In addition to the above generalizations there are two factors of special significance for the pregnant woman in the developing world. In all traditional societies marriages occur early, usually around the age of menarche. Hence childbearing also commences early. It is now generally agreed that major risk of low birth weight occurs within 2 years of menarche. For example, the risk of delivering a child less than 2500 g in weight is doubled. Moreover, early childbearing and the resultant competition for nutrients between the fetus and a growing mother, as well as the hormonal changes of pregnancy, may be significant factors in the short stature of women in many developing countries. Secondly, in all traditional societies women have an inferior status. This is reflected in the high mortality rates of infant and young girls compared to boys, a shorter life-expectancy at birth for females as well as higher prevalence rates of nutritional deficiencies in women. Thus the nutrition of the mother during pregnancy is often no different from the deplorable state of nutrition in the non-pregnant woman. Hence the high prevalence of low birth weight in several countries. (See Fig. 4.10. and Fig. 4.11)
Daily household chores require much hard work and expenditure of energy. In addition to the processing of food for cooking by pounding or grinding grain to make flour, women also carry the burden of fetching water and firewood, washing of clothes, and disposal of household refuse. (See Figs. 4.12 and 4.13).
A study involving 797 women from six villages in Maharashtra, India, found that maternal activity was inversely related to maternal weight gain up to 28 weeks of gestation. Heavy activity in early and mid gestation was associated with lower mean birth weigh, and smaller neonatal head and mid-arm circumference. The study concluded that in the labouring class excessive maternal activity is associated with smaller foetal size in rural India. As the study points out there occurs a perpetuation of under nutrition and poverty from one generation to another. (See Fig.4.14)
Weight gain in pregnancy

Dietary studies in well-nourished women in Aberdeen showed that the average weight gain during pregnancy was 12.5 kg, including 3.5 kg laid down as fat in the mother, representing an energy store of 30,000 kcal (table 3.5). The gain in weight follows a general pattern:

1. Minimal accumulation (almost all in "maternal compartment") in the first trimester: 1 kg
2. Approximately 0.3 kg per week (of which 60% is in the "maternal compartment") in the second trimester: 3 kg
3. About 0.3-0.5 kg per week (of which 60% is in the "fetal compartment") in the third trimester: 6 kg

Calorie expenditure is not distributed evenly throughout pregnancy, nor does it parallel fetal growth. Rather it is altered minimally during early gestation, increases sharply near the end of the first trimester and remains fairly constant until term. In the second trimester most of the energy costs are attributed to maternal factors like expansion of blood volume, growth of uterus and breasts, as well as fat storage. In the third trimester most energy costs are for the growth of the fetus and placenta.
The total energy cost of pregnancy based on the protein and fat accumulated by the mother and fetus, together with the metabolic cost of accumulation, has been calculated to be 75 000 kcal. Dividing this figure by 250 days yields an increment of 300 kcal/day, equivalent to a 15 per cent increase over non-pregnancy needs.

Table 4.5 Compartments of weight gain in pregnancy

<table>
<thead>
<tr>
<th>Compartment</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetal compartment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fetus</td>
<td>Negligible</td>
<td>1.0</td>
<td>3.4</td>
</tr>
<tr>
<td>Placenta</td>
<td>Negligible</td>
<td>0.3</td>
<td>0.6</td>
</tr>
<tr>
<td>Amniotic fluid</td>
<td>Negligible</td>
<td>0.4</td>
<td>1.0</td>
</tr>
<tr>
<td>Total</td>
<td>1.7</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>Maternal compartment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased size of uterus</td>
<td>0.3</td>
<td>0.8</td>
<td>1.0</td>
</tr>
<tr>
<td>Increased breast size</td>
<td>0.1</td>
<td>0.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Increased blood volume</td>
<td>0.3</td>
<td>1.3</td>
<td>1.5</td>
</tr>
<tr>
<td>Increased extra cellular fluid</td>
<td>0</td>
<td>0</td>
<td>1.5</td>
</tr>
<tr>
<td>Total</td>
<td>0.7</td>
<td>2.4</td>
<td>4.5</td>
</tr>
<tr>
<td>Total gain accounted for</td>
<td>0.7</td>
<td>4.1</td>
<td>9.5</td>
</tr>
<tr>
<td>Usual gain in weight</td>
<td></td>
<td></td>
<td>12.5 kg</td>
</tr>
<tr>
<td>Difference as body fat</td>
<td></td>
<td></td>
<td>3.0 kg</td>
</tr>
</tbody>
</table>

More recently calorimetric and metabolic studies have provided similar results. The mean daily increase in energy intake in a healthy and well-nourished woman is just over 100 calories for the whole period of pregnancy, with a median value of 9 calories for the first trimester, 8 for the second and 216 for the third. In most cases the addition of 340 of milk to a well-balanced diet is sufficient to meet all the extra energy requirements of pregnancy.

Iron and folic acid requirements

Pregnancy imposes a considerable strain on the maternal blood-forming system. In most instances the greatest need is for iron and folic acid. The amount of elemental iron in a fetus at birth is approximately 300 mg and the quantity required for increased red cell formation in the mother to prevent anaemia in the face of increased plasma volume is 500 mg. In other words, the requirement for iron during pregnancy is slightly less than 1 g, concentrated for the most part in the last half of gestation.
In an adequate diet the daily iron content is 10-15 mg, of which 10 -20 per cent is absorbed. Dietary iron will thus provide just a little less than the requirement so that other sources of iron are needed. Body stores of iron, mainly in the bone marrow, are available, but frequently the amount is not sufficient to meet the demand. Iron stores in healthy young American women average 300 mg. However, a significant number of women in developing countries enter pregnancy with depleted or no iron stores because of previous pregnancies or menstrual loss. The relatively small amounts of iron in the diet, and the low stores of iron, are not enough to meet the greatly increased requirements of iron for the synthesis of maternal and fetal haemoglobin. Thus, anaemia is a relatively common complication of pregnancy, even in developed countries.

Anaemia of pregnancy is common in all developing countries. In a collaborative study involving seven Latin American countries, iron deficiency was present in 48 per cent of pregnant women as compared with 21 per cent of non-pregnant women. Similar prevalence studies have shown that 15-50 per cent of women in Africa, and more than 20 per cent of women in Asia, have haemoglobin levels below 10 g/dl, mostly due to iron deficiency.

Maternal anaemia is associated with an increased risk of low birth weight and perinatal death. In one study in East Africa it was found that among mothers whose haemoglobin was 7.4 g/dl or less at the time of delivery, the incidence of low birth weight (< 2.500 g) was 42 per cent and the stillbirth rate was 147.1 per 1000. In mothers with haemoglobin of 8.8 g/dl and above the incidence of low birth weight was 12.7 per cent and stillbirths 51.0 per 1000. Similarly, in Malaysia it has been reported that in mothers with haemoglobin of 6.5 g/dl or less the incidence of low birth weight was 20 per cent compared to 7 per cent in non-anaemic pregnancies and the perinatal loss in the anaemic mothers was more than twice that of the non-anaemic ones. More recently a study of 1000 women in India revealed a close relationship between maternal haemoglobin levels and birth weights. 40 per cent of the mothers who gave birth to light infants had haemoglobin levels below 9g/dl.

Iron sufficiency is also critical for the rapidly growing organ systems of the newborn. Major part of iron in the third trimester foetus and the newborn is found in the red cell mass as haemoglobin, with lesser amounts in the tissues as storage iron such as ferritin, or functional iron like myoglobin, cytochromes, and so on. When supply does not meet demand iron is prioritized to haemoglobin synthesis and non-heme tissues like skeletal muscle, heart and brain will become iron deficient. Maternal iron deficiency is a common cause of depleted iron in the newborn. Very low birth weight and pre-term infants are also at risk of early post natal iron deficiency because they have accumulated less iron during gestation, and experience catch-up growth after birth.

Folic Acid

Animal studies had indicated the possible protective role of vitamins including folic acid against neural tube defects (NTDs), and this had led to small scale intervention studies in pregnant women. In 1991 the results of a clinical trial sponsored by the British Medical Research Council provided evidence that the risk of recurrent NTD was significantly lower among women who took 4000 μg of folic acid daily (without other supplemental vitamins) than among those who did not. More recently a population based intervention study in China has reported on the effectiveness of 400 μg of folic acid in preventing NTD in northern China which suffers a high incidence of the defect.
It has been proposed that closure of the neural tube occurs at several sites and that the clinical types of NTD differ depending on the site at which closure fails. Variations in the cellular mechanisms of closure at various sites might also contribute to clinical variations in NTD. It has also been suggested that a closed tube may reopen in some cases. All these studies indicate that folic acid is just one of the factors influencing closure of the neural tube, and explain why supplementation brings about only 70% reduction in the occurrence of NTD.

As the role of folate in health has come to be better understood public health authorities in several countries are taking steps to improve the intake of folate in the general population. In the United States all grain products like flours and pasta are now fortified with 140 μg of folic acid per 100g of grain. National health services in several countries recommend that at the diagnosis of pregnancy women should be prescribed a daily dose of 400 μg of folic acid to be continued during the early months of pregnancy. The evolving knowledge about the role of folate in health should lead to a global public health policy response in the form of education of the general public, supplementation for the vulnerable groups, and fortification of staple foods.

**Recommended allowances**

As described earlier, studies on the energy costs of pregnancy indicate that about 100 calories per day extra are required for the entire gestation period. These are not evenly distributed but more or less follow the pattern of fetal growth and maternal weight gain. For example, energy balance is maintained by the addition of 10 calories per day in the first trimester, 85 in the second and 220 in the third.

**Other micronutrients**

Maternal micronutrient deficiency which is frequently multiple in developing countries can have serious implication for the growth of the foetus. When maternal nutrient supply is inadequate the delicate balance between maternal and foetal needs is disturbed and a state of biological competition gets established. Hence maternal nutritional status at conception has a bearing on how nutrients are partitioned between the maternal and foetal compartments. In severe deficiency maternal nutrition gets the preference. If the deficiency is marginal the foetal compartment may get favoured. Studies of nutrient partitioning have focused on energy and protein. But it is very likely that the partitioning of micronutrients follows a similar pattern.

Protein requirement is related to energy intake. Most balance studies show a linear relation between calorie intake and nitrogen balance. The average nitrogen retention during pregnancy is 51 ± 40 mg/kg per day at the average intake of 52± 9 calories and 1.7 g protein per kg body weight daily. Based on the above considerations the World Health Organization has recommended an intake of 1.01 g/kg of protein and 46 kcal/kg for the average woman with a body weight of 55kg. These recommendations are general and tend to err on the safe side. Most countries have made specific recommendations in relation to their individual circumstances and national dietary patterns (table 4.6).
Table 4.6  Recommended daily protein and energy allowances for women in several countries

<table>
<thead>
<tr>
<th>Country (year)</th>
<th>Reference body</th>
<th>Weight (kg)</th>
<th>Protein (g/kg)</th>
<th>Energy (kcal/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1974</td>
<td></td>
<td>56</td>
<td>0.70</td>
<td>0.85</td>
</tr>
<tr>
<td>1955</td>
<td></td>
<td>55</td>
<td>1.09</td>
<td>1.31</td>
</tr>
<tr>
<td>1969</td>
<td></td>
<td>55</td>
<td>1.18</td>
<td>2.36</td>
</tr>
<tr>
<td>1968</td>
<td></td>
<td>45</td>
<td>1.00</td>
<td>1.22</td>
</tr>
<tr>
<td>1970</td>
<td></td>
<td>49</td>
<td>1.12</td>
<td>1.32</td>
</tr>
<tr>
<td>1969</td>
<td></td>
<td>55</td>
<td>1.00</td>
<td>1.09</td>
</tr>
<tr>
<td>1968</td>
<td></td>
<td>53</td>
<td>0.95</td>
<td>1.12</td>
</tr>
</tbody>
</table>


As regards iron and folic acid, it has been found that diets which provide the recommended amounts of protein and calories from a mixture of foods will also provide sufficient iron and folic acid. However, to ensure adequate intake it is advisable to take additional iron and folic acid, especially in the last trimester. There is now an overwhelming reason to provide folic acid supplements throughout pregnancy, and preferably before conception if the pregnancy a is planned one.

Effects of nutritional deficiency in pregnancy

Several investigations in laboratory animals show that restricting food in pregnancy can have profound effects on the physiological adjustments in the mother, as well as on the growth and development of the fetus. The effects of restricting only calories cannot be separated from those of restricting proteins, because the body can burn proteins to provide energy; conversely, calories have a protein-sparing effect. Generally speaking, in most laboratory animals food deficiency in pregnancy reduces the size of the litter, the weight of the individual offspring and the survival rate. Subsequent growth of the offspring is also affected, though it is more so when food deficiency extends into the period of lactation.

More recent studies of the cellular mechanisms of growth have measured the DNA content of an organ as an index of cell number and the protein content as an index of cell size. These studies demonstrate that the growth of an organ takes place in phases. There is at first an increase in the number of cells followed by an increase in the size of the individual cell. Food restriction at the time of cell division can significantly affect the size of an organ, and conceivably such a restriction of growth is irreversible.
In the human there are problems in the interpretation of the effects of dietary deficiency on the fetus. In the poorer parts of the world food deficiency during pregnancy is the rule, but comparisons with the more affluent societies are difficult because of the genetic, socioeconomic and other differences. In the case of Western Europe, however, some information is available from "experiments of nature" like famine and war. During the last war, acute food shortage reaching famine proportions occurred on two separate occasions. The siege of Leningrad lasted 18 months (from August 1941 to January 1943) and for a period of 6 months (September 1941 to February 1942) conditions were extremely severe. Two periods of food deficiency can be identified: a time when there was a generally increasing restriction of food, and, later, a period of extreme deficiency when the diet consisted mainly of bread, one half of it made up of defective rye flour and the rest consisting of cellulose, salt and bran. During the early part of the siege the average birth weight decreased significantly. It was found that 49 per cent of those born in the first half of 1942 (thus having suffered nutritional deprivation in the last trimester of pregnancy) weighed less than 2500 g. There were, of course, other potential contributing factors like excessive physical exertion, lack of rest, nervous tension, extremes of cold, and so on.

A similar period of famine was also experienced in Holland in the winter of 1944-45. The period of food shortage was more sharply demarcated than in the case of Leningrad, and overwork and other strains were not so prominent. The famine reached its maximum severity after 6 weeks and lasted for 27 weeks altogether. Thus, pregnant women were exposed to nutritional deprivation for varying lengths of time, but none was exposed for the whole gestation period. Birth weights were lowest in the case of those mothers who had experienced 8-21 weeks of famine, and began to rise immediately after the famine ended. Lowest median birth weights were reached when exposure to famine was in the second half of pregnancy. Exposure very early in pregnancy did not affect the birth weight. Infants born of mothers who had conceived in the latter part of the famine, so that babies experienced 27 weeks of gestation during the famine and an average of 9 weeks after it ended, attained a higher mean birth weight than those born in the early part of the famine.

In the developing world, food shortage is chronic and is often acute during times of poor harvests. In a dietary survey of 352 pregnant women in India, it was found that the mean daily intake was 1402 kcal and 38 g protein. (WHO/FAO recommended allowances are 2200 cal and 55 g protein.) Many women in the lower socioeconomic groups enter pregnancy after a childhood in which undernutrition and recurrent illnesses are common, so that they have not obtained the optimum in growth and physiological development. Thus, the mean body weight in 498 non-pregnant Indian women in the low socioeconomic group was found to be only 42.4 kg; 40 per cent of women in the low and low-middle income groups in India are under 150 cm in height whereas only 15-20 per cent in the upper income groups are of short stature, again indicating the importance of nutrition in early childhood. Moreover, on their marginal nutrition many pregnant women gain very little weight. In one study of 48 pregnant women, the mean gain in body weight from the twelfth to the fortieth week of pregnancy was 6.02 kg.

In another study of 130 pregnant women, almost half failed to gain weight between the thirty-second and thirty-sixth weeks and thereafter. Besides poor weight-gain, many show clinical signs of nutritional deficiency. In a nutritional survey carried out amongst 198 pregnant women during the third trimester of pregnancy, 44 per cent showed clinical signs of vitamin B complex deficiency, 9.5 per cent had oedema of the legs and 14.5 per cent showed signs of lack of vitamin A. The mean birth weight in such women from the lower socioeconomic group was 2778 g as compared to 3055 g in the higher social group.
A survey of dietary intake of women in coastal Tanzania revealed that mean daily energy intake was 1850 kcal and that of protein was 51.5 g. Iron intake in the diet ranged from 10 to 16.2 mg per day. A significant correlation was seen between the energy intake of the mother and the size of the baby. In order to have a baby weighing more than 3.1 kg it was necessary that the mother should consume at least 50 g of protein daily. Forty per cent of the women were eating less protein than this amount. Energy deficiency was a much more serious problem. To have a baby weighing 3.1 kg a mother had to consume 2200 cal per day, and only 20 per cent of the women had as much.

A similar study in four Guatemalan villages showed that the mean intake of calories and protein during pregnancy was 1500 cal and 40 g respectively. The average maternal height in rural Guatemala is 143 cm which is far less than the average height for women in a sample of the white population in the USA. Much of this low maternal height is accounted for by growth retardation during the first 7 years of life. The average weight gain in pregnancy was 7 kg, which is about half of that in well-nourished women in affluent societies. Predictably, the mean birth weight was low at 3 kg and of 39 infants with normal gestational age about a third weighed 2.5 kg or less. Dietary histories again indicated a close association between food intake and the weight of the baby at birth. For example, in 34 women with a daily caloric intake of 700-1800, the mean birth weight was 2.8 kg. In eight women with a daily caloric intake of 1900 to 2,100 calories the mean birth-weight was 3 kg and in 9 women with a daily caloric intake of 2200 - 3100 calories the mean birth weight was 3.2 kg. Supplementation studies showed that the total caloric intake was a more critical variable than protein, and the incidence of lighter babies was highest when the Guatemalan mother consumed less than 1800 cal per day.

In peasant societies the wet season is often the worst time of the year. Food shortages, heavy demands for agricultural work, increased exposure to infection like malaria, diarrhoea and respiratory infection, all coincide to increase stress and poverty. Malnutrition, sickness and indebtedness more commonly occur in the rainy season. Naturally, the weaker groups like mothers and children suffer most. Pregnancies which reach the advanced stage in the wet season are at special risk of resulting in low-birth weight infants. The greatest demand is made on the adult population by the energy expenditure needed for farming. For example, in Upper Volta it was noticed that energy expenditure by women in the wet season was between 2450 and 3600 kcal compared to 2000-2700 in the dry season.

CELLULAR GROWTH IN THE FETUS

The organ sizes and body weights of infants who were either stillborn or died in the neonatal period, were compared in the case of 1002 consecutive necropsies in New York City. In the case of mothers from the low socioeconomic groups, the body size of the infants - as well as the weights of the brain, heart, liver, spleen, thymus, kidneys and adrenals - was all significantly less than in the case of infants born to better-off mothers. The body weight was less by 13-17 per cent of the mean of infants born to well-to-do mothers. Amongst the organs, the thymus, spleen and liver were found to be particularly small.
Measurements of DNA and DNA to protein ratio in kidney, heart and liver of the human fetus indicate that these organs grow in several phases during intrauterine life:

1. Between the fourteenth and twenty-fifth week of intrauterine life, the cells in all the three organs are dividing rapidly. The DNA content approximately doubles each week.

2. Between the thirtieth and fortieth weeks there is a rapid growth in cell size. The cells still increase in number, but more slowly than before.

3. At term all three organs still had less than 20 per cent of the numbers of cells characteristic of the adult, indicating that further increase in cell number occurs after birth.

In the case of babies who were "small-for-dates' the protein/DNA ratio was normal, but the total DNA tended to be low. (See Fig. 4.15)

![Figure 4.15 Total DNA in the brains of small-for-dates infants](image)
WEIGHT AT BIRTH AND BODY COMPOSITION OF THE FETUS

When body composition of stillborn infants in undernourished Indian women was compared with that of similar infants of well-nourished English women, it was found that at 26-28 weeks of gestation the total body weights were similar in both groups, but there were significant qualitative differences. There was a deficit in protein and iron content in the case of the Indian babies (Table 3.7). In the case of Indian fetuses, liver stores of vitamin A, B2 and folate were lower than those of infants of well-nourished mothers.

Table 4.7 Fetal body composition in undernourished Indian and healthy English women at different weights and gestational age.

<table>
<thead>
<tr>
<th>Age (weeks)</th>
<th>Water (g)</th>
<th>Protein (g)</th>
<th>Fat (g)</th>
<th>Cu (g)</th>
<th>Po4 (g)</th>
<th>Mg (mg)</th>
<th>Iron (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight 1000 g</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indian a</td>
<td>27</td>
<td>862</td>
<td>80</td>
<td>26</td>
<td>5.8</td>
<td>3.3</td>
<td>200</td>
</tr>
<tr>
<td>English b</td>
<td>26</td>
<td>860</td>
<td>87.5</td>
<td>10</td>
<td>60</td>
<td>3.4</td>
<td>220</td>
</tr>
<tr>
<td>Weight 1500 g</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indian</td>
<td>34</td>
<td>1248</td>
<td>126</td>
<td>75</td>
<td>10.5</td>
<td>5.0</td>
<td>325</td>
</tr>
<tr>
<td>English</td>
<td>31</td>
<td>1270</td>
<td>156</td>
<td>35</td>
<td>10.0</td>
<td>5.6</td>
<td>350</td>
</tr>
<tr>
<td>Weight 2000 g</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indian</td>
<td>38</td>
<td>1604</td>
<td>176</td>
<td>148</td>
<td>14.6</td>
<td>8.6</td>
<td>425</td>
</tr>
<tr>
<td>English</td>
<td>33</td>
<td>1620</td>
<td>231</td>
<td>100</td>
<td>15.0</td>
<td>8.2</td>
<td>460</td>
</tr>
<tr>
<td>Weight 2500 g</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Indian</td>
<td>40</td>
<td>1935</td>
<td>242</td>
<td>242</td>
<td>19.0</td>
<td>10.8</td>
<td>540</td>
</tr>
<tr>
<td>English</td>
<td>35</td>
<td>1940</td>
<td>306</td>
<td>185</td>
<td>20</td>
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<td>580</td>
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<tr>
<td>Weight 3000 g</td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Indian</td>
<td>40</td>
<td>2238</td>
<td>291</td>
<td>366</td>
<td>22.7</td>
<td>11.4</td>
<td>655</td>
</tr>
<tr>
<td>English</td>
<td>38</td>
<td>2180</td>
<td>344</td>
<td>360</td>
<td>25.0</td>
<td>14.0</td>
<td>700</td>
</tr>
</tbody>
</table>

THE BARKER HYPOTHESIS

In Britain, data collated by the Environmental Epidemiology Unit of the Medical Research Council show that the incidence of ischaemic heart disease, stroke and chronic bronchitis varies with social class and geography, being higher in poorer districts and counties. In the case of some illnesses the variations noted are as large as twofold or more, and cannot be adequately explained by differences in diet or smoking. However, when compared with rates of infant mortality in the same localities half a century or so ago there was seen a marked degree of correlation. The implication is that since rates of infant mortality reflect the general state of health in children such strong correlations indicate the contributory effects of environmental deficiencies suffered during childhood in the form of inadequate feeding, ill health, overcrowding, and poor life-style.

A comparison between infant mortality rates in 1921-25 and ischaemic heart disease in 1968-78 in the same geographical areas of England and Wales also show a close correlation second only to that for bronchitis. The correlation coefficient is of the magnitude of 0.73 compared to 0.82 for bronchitis. This result is intriguing. Increasing rates of ischaemic heart disease in Britain had hitherto been ascribed to changes in life-style including diet. However, the increase has been more among the lower social classes compared to those who are well-off. Such close geographical relation between current mortality from ischaemic heart disease and past infant mortality half a century or so ago throws a new light on the aetiology of ischaemic heart disease. It may possibly be related to events in foetal life influencing growth and development in the foetus as well as growth in early infancy. These observations led to the so-called Barker Hypothesis which links suboptimal growth and development in foetal life to health during adulthood. Evidence suggests that poor growth in utero can lead to a variety of chronic disorders such as cardiovascular disease, non-insulin dependent diabetes and hypertension. Exposures acting in adulthood such as smoking may still influence disease risk in a simple additive manner, but it is argued that foetal exposures permanently alter anatomical structures and a variety of metabolic systems. Relationships of coronary heart disease, high blood pressure and insulin resistance with low birth weight are particularly strong or only observed for subjects who become obese in childhood or later life. The highest risk is typically among adults who were born small and then become overweight during childhood or adulthood, with regard to insulin resistance hypertension and coronary heart disease. These observations provide a life course model for the development of a variety of chronic diseases of adult life where events in foetal life influencing the development of body systems programme the individual for responding to environmental triggers in an adverse manner. To give an example, poor intrauterine development may have an adverse effect on the number of muscle cells that develop in the foetus so that at birth a small growth retarded foetus may have a permanent reduction in the number of muscle cells. Such a child, however, may still compensate by muscle hypertrophy so that in functional terms there may be no evident difference. The same is likely to apply to metabolic and hormonal systems which may be up or down regulated during foetal life but are still capable of modification by other exposures in later life. Studies indicate that in the individual the metabolic pattern is set by the method of feeding in early life. In this respect breastfeeding provides a variety of biological messengers which perform the 'fine tuning' of the developmental time clock in the infant. This matter is further discussed in Chapter 5.

Evidence is strongest concerning the effects of health in childhood on later performance with regard to maternal health. In a number of studies low birth weight rates, foetal and neonatal death rates have been shown to be related to the occupational class of the mother's father and the husband. The implication is that the conditions under which a mother grew up and the conditions under which the pregnancy occurred are both important influences on pregnancy outcome. In some cases the
conditions of mother's upbringing may be more important than those experienced during pregnancy. Furthermore, intrauterine growth of the female foetus may condition her reproductive performance in adult life. For example, mothers of small-for-dates infants have been shown to have been born with low birth weights themselves. Mothers who at birth weighed 2000g have a 2 1/2 times higher risk of abnormal outcome of pregnancy as compared to those who were heavier at birth. These differences can be ascribed to growth disturbance of organ systems including the reproductive and or endocrine systems. Hence ensuring rapid catch-up in growth retarded newborns by feeding on their mothers' milk becomes a priority. This is particularly so with girls in whom achieving optimal growth up to the age of maturity is important, since women tend to have babies of similar birth weights and gestational characteristics in all their pregnancies. This tendency to bear babies of similar birth weight and gestational age adjusted for maternal age, birth order, and complications of pregnancy labour or delivery has been ascribed to a 'programming effect' during intrauterine life.

The concept of 'programming' has been evolved as the underlying biological mechanism with birth size as proxy. Recent findings suggest that fetal programming interacts with post birth environment. The adverse environmental influences that are thought to underlie programming cluster in socially patterned ways leading substantial inequalities in health. Intrauterine growth may suffer on account of a single pathological condition e.g. hypertension. If detected in time early induction of delivery can help to establish an alternative nutrition line through the breast and the infant's development progresses normally. This is illustrated in the figure below. (See Fig.4.15)

![Figure 4.16 Arrested head growth in late pregnancy](image)

On the other hand a constellation of social and biological factors could be operating in disadvantaged mothers causing a slowing of growth early on in pregnancy. This is illustrated in figure 4.15. (See Fig.4.15)
A number of hypotheses have been put forth to explain how programming works. The current consensus based on animal models is that intrauterine growth retardation is associated with fetal exposure to maternal glucocorticoids. Such an exposure at a critical time in fetal development leads to determination of the ‘set point’ of the hypothalamic-pituitary-adrenal axis, and of glucocorticoid receptors in tissues. Another candidate hypothesis is the growth hormone – insulin like growth factor (GH-IGF) axis becomes set to function abnormally leading onto cardiovascular diseases and proneness to type2 diabetes in adulthood.

**EFFECTS OF SUPPLEMENTING MATERNAL DIET**

In well-nourished communities no supplementation is necessary beyond the administration of iron and folic acid in the last trimester and the normal satisfaction of hunger from a mixed diet. In communities where diets are inadequate, and where a large proportion of mothers enter reproduction after a childhood characterized by inadequate growth, supplementation is important to avoid fetal malnutrition. Repeated pregnancies in such a situation lead to depletion of maternal tissues and impaired reproductive efficiency.

Several countries have now evolved national programmes for protecting the diets of pregnant and lactating women through a supply of free subsidized foods, especially milk; through the establishment of fair price retail shops; through fortification of ordinary foods to improve their nutritive value; and sometimes through cash benefits to improve purchasing power.
In developing countries, where health resources are meagre and a large number of families exist on marginal nutrition, it is necessary to identify the minimum supplementation necessary for adequate fetal growth. It is now generally agreed that maternal weight gain in pregnancy is associated with fetal growth. Generally, 1 kg of maternal weight gain leads to an increment of 20-25 g in the birth weight. More specifically, a weekly weight gain of less than 0.2 kg more than doubles the likelihood of the incidence of low birth weight. The effect is even more serious if the pre-pregnancy weight was at the lower end of the scale. The correlation of birth weight with the post-partum weight of the mother is even closer. In several studies mean birth weight has been shown to increase linearly with the post-partum weight until the post-partum weight is 100-110 per cent of ideal maternal weight. At that point birth weight remains constant while post-partum weight continues to increase.

The above studies suggest that fetal growth occurs optimally only when the mother is able to accumulate a critical amount of extra body stores during pregnancy. Thus the metabolic adaptations of pregnancy enable the mother initially to maintain her own body stores of nutrients. If she has adequate quantities of these stores she can succeed in supporting a normal rate of fetal growth even if her own food intake becomes inadequate. If the body stores are small, then the level of food intake seems crucial for maintaining a normal rate of growth in the fetus.

Studies in Guatemala have shown that supplementing the pregnant woman's diet by 233 kcal per day led to a significant increase in fetal birth weight. Heavier babies were born to mothers who consumed more calories during pregnancy, irrespective of whether the calories were derived from the usual diet or from food supplements. The conversion of food calories into fetal tissues was at the rate of 3 g of baby per 1000 kcal of additional food. It was significant that in the same mother there was an average difference of 2.2 g between successive siblings per change of 1000 kcal in the diet. Though there are a host of factors which can influence birth weight, these findings emphasize the importance of adequate food intake during pregnancy. The association between weight gain in pregnancy and birth weight, though statistically significant, is not very strong. Correlation coefficient has been between 0.2 and 0.3 in most studies, and weight gain in pregnancy accounts for about 6 per cent of the variance for birth weights. However, what is more important is the shift of emphasis from protein to calories. In most instances, when the energy requirement is satisfied by adequate food intake, the requirement of protein is also taken care of, unless the diet is bizarre.

More recent work in India adds yet another facet to the whole question of supplementation in pregnancy. When diets of Indian women in low socio-economic groups were supplemented with 50 g protein and 500 cal, the mean gain in weight by the mothers during pregnancy was 2.84 kg, significantly greater than the 1.02 kg in the control group; the mean birth weight of the babies (3000 g) was also significantly greater than that of the controls (2700 g). Interestingly, when the supplement consisted of 30-60 mg of iron and 200-500 mg of folic acid, instead of food, the mean birth weight was 2880 g compared to 2500 g in the controls and the incidence of babies weighing less than 2300 g was 10 per cent as compared to 23 per cent in the control group. The placentas in the supplemented group were heavier and their DNA content was higher. It has been suggested that folic acid has a growth-promoting action besides being a haemopoietic factor. On the basis of these studies the government of India has commenced a national programme of supplementation with iron and folic acid for pregnant women.
INTERVENTION PROGRAMMES

In the past decade several carefully documented studies in pregnant women have demonstrated the importance of adequate dietary intake for optimal fetal growth. For example, in Montreal it was found that pregnant women who were referred for dietary intervention delivered infants with mean birth weights 40 g heavier than in the controls. The frequency of low birth weight was 5.7 per cent among those referred compared to 6.8 per cent in controls. Compared to their matched controls, primigravidae had a 61 g advantage and women who weighed less than 64 kg at conception had a 63 g advantage. Women who were both primigravidae and weighed less than 64 kg had a 73 g advantage. In rural Guatemala where the daily diet consisted of 1500 cal and 40 g protein, the proportion of infants born with low birth weight (<2500g) was 20 per cent. A daily supplement of 70 cal resulted in a gain of 56 g in birth weight. These and similar other studies referred to in the preceding discussion indicate that in women at risk of low birth weight, food supplementation in pregnancy can lead to an increase of between 40 and 60 g in birth weight. The degree of rise depends on the nutritional status of the woman. In thin and undernourished women the rise in birth weight is most evident. In starved women, as for example during the Dutch famine, a deficit of 300-400 g can be made up almost immediately upon adequate feeding.

There is an urgent need to convert the above information into viable health programmes. It is estimated that of the 22 million babies being born with a birth weight less than 2500 g each year, 21 million are in the developing world. A high proportion of neonatal and infant mortality in the developing countries occurs in such infants, so that the effects of fetal malnutrition work well into the first year of life. Moreover, in all countries the incidence of handicaps is also higher in infants with low birth weight compared to normal.

The mean birth weight in the community, and especially the ratio between mean birth weights in the lower and upper socioeconomic groups, is increasingly recognized as a useful measure of the nutrition of the community. This is more so because of the inferior status of the woman in many traditional societies. All intervention programmes have the tendency to follow the Inverse Care Law and benefit the upper strata of the community more than the needy ones. Serial improvements in mean birth weight, and particularly in the proportion of those born with a birth weightless than 2500 g, serve as readily available indices of community nutrition. They also serve as measures of the effectiveness with which nutrition programmes reach the disadvantaged such as, for example, women in the lower socioeconomic groups.

All interventions reach the consumer through services. Hence pre-natal care becomes a priority where nutrition in pregnancy is concerned. In developing countries not more than a third of the pregnant women receive any antenatal care, and the number of women receiving skilled assistance in labour is even smaller. National health programmes in most countries have continued to emphasize institutional care at the cost of coverage with basic services so that improvements in health and nutrition status have been disappointing. For example, in Hyderabad (India) studies undertaken in the 1960s showed that the daily energy intake of pregnant women in low socioeconomic groups was between 1400 and 1600 kcal. A decade later, in spite of several socioeconomic improvements as well as expansion of education and healthcare delivery in urban areas, dietary intakes have remained essentially similar to those of two decades ago. Moreover, the mean body weights during the third trimester of pregnancy (49.5 kg) and mean weight gain in pregnancy (6 kg) have remained unaltered over the past 15 years. Thus, the considerable amount of new scientific knowledge and experience of the past two decades has not
benefited the large majority of the population. Radical new approaches in developing health-care strategy to expand coverage with basic services are needed.

All innovative approaches aimed at extending health coverage have tended to utilize traditional health providers in the community. Several countries, notably Sudan, Niger, Tanzania, Indonesia, Malaysia and the Philippines, have new national programmes for training traditional birth attendants and their subsequent deployment as village midwives. Such health workers may succeed in improving traditional food habits, and can become a local source of nutritional advice as well as distribution of iron and folic acid tablets, and antimalarials where necessary, to the pregnant women. But there is also a need of interventions at other levels through programmes of education, especially female literacy, creation of job opportunities, and availability of subsidized food items for the pregnant woman.

Current thinking based on several intervention studies is that balanced protein and energy supplementation can reduce the risk of small for gestational age by 30%. The benefits of iron and folic acid supplementation are now established by several clinical trials. Programmatic recommendation can be made for intervening with supplements especially in emergencies and in populations with high prevalence of under nutrition.

Greater awareness of the nutritional needs of the pregnant woman in the community must be accompanied by development of local skills and facilities for monitoring adequate fetal growth. Here measurements of sequential fundal height and abdominal girth as measures of fetal growth have been evaluated in several studies and can be developed into a simple tool for use in villages and peri-urban communities. The trained birth attendant can thus help in the primary and secondary selection of pregnant women for more expert care.

FURTHER READING


Centers for Disease Control. Recommendations for the use of folic acid to reduce the number of cases of spina bifida and other neural tube defects.MMWR 1992;41:2-8.


