4. To use this information as a necessary base to develop a predictive model for the expected birth weight using maternal nutrition related variables
The literature was reviewed to cover the following aspects:

1. To define the frontiers of this field
2. To understand the theory in the field that enables placing the question in perspective
3. Learn which procedures and instruments would prove useful and
4. Facilitate interpretation of the significance of the results of this study.

The literature search was accomplished as follows:

An internet search was carried out initially to gather information on journals published since 1990 dealing with:

- Birth Weight
- Child survival and reasons for increased mortality rate
- LBW – Prematurity; IUGR and their determinants
- Nutrition education and its role in improving birth weight
- Predictions of birth weight

A similar search was carried out for review articles cited in Bibliography, over the same period. These were
supplemented by Medline computer search covering the years 1990-2003.

However, no claim is made for the completeness of the literature survey. Many articles containing potential determinants of pre maturity and IUGR and low birth weight and predictions of birth weight have probably not been included, especially those in less frequently cited sources. Within limitations, however, the search method was reasonably comprehensive and relatively objective.

**Birth Weight and its Significance**

From an epidemiological point of view, birth weight of the child is an important index for children’s health and development [Agarwal et.al., 2002]. It also gives us the probability of newborn to survive and experience healthy growth and development. It is also an important indicator of nutritional/health status and reproductive maturity of a pregnant woman, extent of antenatal care she has received and prevailing socio-economic conditions. Low birth weight, with its serious immediate and long-term consequences, deserves greater attention from
pediatricians and policy makers because it is a major contributing factor in two thirds of all infant deaths [Yerushalmy et.al.,1965].

**Defining Low Birth Weight**

Low Birth Weight [LBW] is defined by WHO as a birth weight less than 2500g. Before 1976, the WHO definition was less than or equal to 2500g, since below this value birth-weight-specific infant mortality begins to rise rapidly [Kramer 1987].

Although the causes of LBW deaths, infection and asphyxia were identified several decades back, no organized efforts have been made to prevent LBW, which constitutes 33% of all live births in the country, that has remained unchanged over 50 years. There is an urgent need for national policy on LBW, incorporating new knowledge about the fetal origin of adult diseases –Baker's hypothesis [Bhargava, 2003].

The crux/reason of the high infant mortality rate and the high incidence of low birth weight has been stated by Bhargava [2003] to be less access to health care systems,
with 60-70% births occurring at home where over 60% birth attendants are untrained and only 33.6% births occur in a health care facility.

**How common is Low Birth Weight**

The importance of low birth weight does not lie in its effect on mortality and morbidity but also on its frequency of occurrence in a given population. The best available global estimates of mean birth weight and the prevalence of LBW were produced by WHO in 1979 and updated to 1990. Of the 127 million infants born in the world in 1990, 20 million (16%) were estimated to weigh less than 2500g, and over 90% of these infants were born in developing countries, a function not only of the higher birth rate in these countries but also of their much higher prevalence of LBW (Table 2.1).

**Prevalence of Low Birth Weight in Asia**

National Neonatology Forum Perinatal Database (1994) from 14 large hospitals from nine states spanning 24,410 live births, showed the prevalence of LBW to be 30
percent. The prevalence of low birth weight in Asian
countries is presented in Table 2.2.

The lowest birth weights were reported for Asia, with mean
values ranging from about 2700-2800g in the Indian
subcontinent to 3200-3300g in China and Japan and
corresponding LBW rates of 30%-40% and 5-6%,
respectively.

Table 2.1: World Trends in Prevalence of Low Birth
Weight

<table>
<thead>
<tr>
<th>World Countries</th>
<th>Percent Low Birth Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1979</td>
</tr>
<tr>
<td>Asia</td>
<td>22</td>
</tr>
<tr>
<td>Africa</td>
<td>15</td>
</tr>
<tr>
<td>Latin America</td>
<td>13</td>
</tr>
<tr>
<td>North America</td>
<td>07</td>
</tr>
<tr>
<td>Europe</td>
<td>07</td>
</tr>
<tr>
<td>Oceania*</td>
<td>20</td>
</tr>
</tbody>
</table>
Developing countries 20 19
Developed Countries 07 07
Global 18 17

*(excluding Japan, Australia and New Zealand)

Table 2.2: Prevalence of Low Birth Weight in Asia

<table>
<thead>
<tr>
<th>Asia</th>
<th>Percent Low Birth Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1979</td>
</tr>
<tr>
<td>Eastern</td>
<td>07</td>
</tr>
<tr>
<td>South-Eastern</td>
<td>18</td>
</tr>
<tr>
<td>Southern</td>
<td>34</td>
</tr>
<tr>
<td>Western</td>
<td>12</td>
</tr>
<tr>
<td>Asia</td>
<td>22</td>
</tr>
</tbody>
</table>

WHO (1992)

Prevalence of Low Birth Weights across the Country

According to National Family Health Survey, 1992-93 percentage of LBW in different states is depicted in Table 2.3. The lowest mean birth weights are recorded from Calcutta (Chakrabarty R, 1975; Katua SD 1979) and Baroda (Rajalakshmi, 1978).

In a study by Agarwal et.al., (2002) at Varanasi, India, had low birth weight accounting to 27.4%. Studies from urban
and rural North Arcot District, Tamil Nadu (1972-70) revealed low birth weight prevalence to be 31.9%, Agarwal et.al., (2001). Ghosh et.al., (1979) in 1969-72 from urban Delhi found low birth weight rate of 23%. In urban Ludhiana (Mittal 1976) during 1974-75 the mean birth weight was 2974g with low birth weight rate of 24.6%. These studies indicate that low birth weight deliveries remain a public health problem in India. Table 2.4 presents Mean Birth Weight of Infants in Different Regions of India.

**Trends in Low Birth Weight Prevalence**

Data from global studies indicate that over the last two decades, there has been some reduction in the proportion of LBW deliveries in most developed countries. However in most of the

**Table 2.3: Percentage of Infants with Low Birth Weights**

<table>
<thead>
<tr>
<th>States</th>
<th>Percentage of low birth weights</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mizoram</td>
<td>5.8</td>
</tr>
<tr>
<td>Nagaland</td>
<td>13.3</td>
</tr>
<tr>
<td>Kerala</td>
<td>18.2</td>
</tr>
<tr>
<td>State</td>
<td>Value</td>
</tr>
<tr>
<td>-------------------------</td>
<td>-------</td>
</tr>
<tr>
<td>Meghalaya</td>
<td>19.2</td>
</tr>
<tr>
<td>Arunachala Pradesh</td>
<td>19.6</td>
</tr>
<tr>
<td>Gujarat</td>
<td>21.2</td>
</tr>
<tr>
<td>Karnataka</td>
<td>21.7</td>
</tr>
<tr>
<td>Assam</td>
<td>21.8</td>
</tr>
<tr>
<td>Tamil Nadu</td>
<td>22.6</td>
</tr>
<tr>
<td>Orissa</td>
<td>23.3</td>
</tr>
<tr>
<td>Bihar</td>
<td>25.0</td>
</tr>
<tr>
<td>Goa</td>
<td>25.5</td>
</tr>
<tr>
<td>Haryana</td>
<td>25.9</td>
</tr>
<tr>
<td>Andhra Pradesh</td>
<td>26.3</td>
</tr>
<tr>
<td>Manipur</td>
<td>26.3</td>
</tr>
<tr>
<td>Delhi</td>
<td>27.5</td>
</tr>
<tr>
<td>Tripura</td>
<td>28.0</td>
</tr>
<tr>
<td>Himachal Pradesh</td>
<td>28.3</td>
</tr>
<tr>
<td>Panjab</td>
<td>28.6</td>
</tr>
<tr>
<td>Uttar Pradesh</td>
<td>31.3</td>
</tr>
<tr>
<td>Maharashtra</td>
<td>32.2</td>
</tr>
<tr>
<td>Jammu &amp; Kashmir</td>
<td>32.5</td>
</tr>
<tr>
<td>West Bengal</td>
<td>33.0</td>
</tr>
<tr>
<td>Madhya Pradesh</td>
<td>38.2</td>
</tr>
<tr>
<td>Rajasthan</td>
<td>48.7</td>
</tr>
<tr>
<td>India</td>
<td>33.0</td>
</tr>
</tbody>
</table>

Prema R, M.K. Nutrition Foundation of India. Data from National Family Health Survey, 1992-93
Table 2.4: Mean Birth Weight of Infants in Different Regions of India

<table>
<thead>
<tr>
<th>Study Center</th>
<th>Mean Birth Weight (g)</th>
<th>% below 2500g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delhi**</td>
<td>2,764+545</td>
<td>25.1</td>
</tr>
<tr>
<td>Varanasi</td>
<td>2,628+504</td>
<td>30.6</td>
</tr>
<tr>
<td>Trivandrum</td>
<td>2,881+533</td>
<td>15.3</td>
</tr>
<tr>
<td>Calcutta</td>
<td>2,673+394</td>
<td>20.1</td>
</tr>
<tr>
<td>Jamshedpur</td>
<td>2,693+437</td>
<td>19.0</td>
</tr>
<tr>
<td>Baroda</td>
<td>2,449+520</td>
<td>46.4</td>
</tr>
<tr>
<td>Mumbai</td>
<td>2,597+441</td>
<td>34.9</td>
</tr>
</tbody>
</table>

**ICMR (1990) unpublished data

Developing countries, there has not been any substantial reduction (Table 2.5).

Table 2.5: Mean Birth-Weights in Different Parts of the World
**Region** | **Mean Birth Weight (kg)**
--- | ---
North America, Western Europe and Australia | 3.5-3.6
Eastern Europe | 3.1-3.3
Africa and East Asia | 2.9-3.1
South Asian Countries | 2.7

WHO (1992)

East asian countries and Sri Lanka however have shown some reduction in the LBW incidence in recent years.

Data gathered by WHO (1992) from different countries in the world shows that South Asian infants have the dubious distinction of having the lowest mean birth-weight in the world about 2.7kg. No less than a third of all neonates in this region weigh less than 2,500g.

In Sri lanka the major input during the last two decades has been the improved quality and coverage of health care to pregnant women through the primary health care infrastructure which has resulted in improvement in birth-weights and the improved survival of neonates. This clearly demonstrates that it is possible to achieve substantial reduction in LBW incidence by providing essential minimal
antenatal care as part of the primary health care package. However, additional inputs in the form of dietary improvements during pregnancy could apparently make a further contribution towards lowering LBW incidence, (Prema R 1993).

**Prenatal Effects of Low Birth Weight**

The majority of LBW infants in developed countries are pre-term (premature) infants. On the other hand, in the developing countries, considerable proportions of infants with LBW are full-term infants. LBW in the latter case is a reflection of intra-uterine growth retardation consequent on poor maternal health/nutrition during pregnancy. The majority of LBW deliveries in India are attributable to such intra-uterine growth retardation.

Highest rates of CVD occurred in people who had LBW and whose mothers were thin. Prevalence of CVD fell from 15% in those who weighed 2.5kgs or less at birth to 4% in those who weigh 3.2 kgs or more (Stein et.al., 1996). There is now increasing evidence that LBW babies have
an increased incidence of hypertension later in life than those with a normal birth weight.

Maternal dietary imbalances in critical periods of intra uterine life can trigger a redistribution of fetal resources, affecting a fetus's structure and metabolism in ways that predispose the individual to later cardio vascular and endocrine disorders.

**Post Natal Effects of Low Birth Weight**

Among infants with LBWs, who survive however, the ultimate outcome and quality of growth and development are better in those infants who had not suffered intra-uterine growth retardation. Ghosh's pioneering longitudinal study on growth of LBW infants in India has thrown considerable light on the significance of LBW from the point of view of the future development and growth of the infant. In short, intra-uterine growth retardation reflected in LBW leaves a lasting deleterious imprint on the child’s growth.

Under nutrition, in men and women as evident by small size at birth rates results in Cardio Vascular Disease [CVD], in later life. CVD is also associated with thinness
and stunting at birth with a small head circumference all of which result from reduced fetal growth disease in Western countries. LBW, thinness and stunting are also related to raised blood pressure, increased rates of NIDDM and impaired glucose tolerance (Barker 1998).

A study from Sweden on Indian orphans born in India and adopted by Swedish parents showed that even after two years on good diets in a healthy environment, infants who had started with the initial disadvantage of LBWs continued to grow in a substandard growth trajectory (Proos, 1992). This is in confirmation of the findings of the pioneering longitudinal studies on low birth weight infants carried out by Shanti Ghosh. However, the poor psychomotor development associated with LBWs could be reversed after two years of good diet and good environment.

Data from South India also show a similar association of lower birth weight with cardiovascular diseases originated in fetal life, and higher risk of type 2 diabetes, the insulin resistance syndrome or impaired glucose tolerance in later adult life. These studies suggest that the incidence of low birth weight can be lowered by improving fetal growth in
pregnancies at risk for fetal growth retardation. Bhargava [2003].

Children born with low birth weight are at increased risk of being malnourished at one year of age. By the age of four or five years, such a child suffering a cycle of infection and malnutrition will be seriously stunted and will carry its growth deficiency and often its impaired learning ability, into adult life. As an adult, this growth deficit is translated into reduced work output.

India has the unfortunate distinction of having 75 million malnourished children below the age of five years. The rate of malnutrition among children of this age (63%) is more than double the average for sub-Saharan Africa, and only a little better than Nepal and Bangladesh (Fig 2.1). Even at birth, one-third children have low birth weight, i.e., undernourished in the womb.

According to the NFHS of the undernourished children, 52 percent are stunted, 53 percent are underweight and 18
percent are wasted. The percentage of children who are underweight reaches 63 percent at age one year and declines slightly thereafter. Stunting reaches a peak of 67 percent among three-year-old children. The prevalence of wasting reaches a maximum 28 percent for children who are one year old and declines rapidly thereafter. This portrays a classical picture of chronic malnutrition among children who are basically too small in weight and height for their age. It can be estimated that there are 60 million malnourished children under four of which nearly 60 percent live in the five states of Uttar Pradesh, Bihar, West Bengal, Madhya Pradesh and Maharashtra. Two out of three preschoolers are severely or moderately malnourished. Stunted children grow up as stunted adults. Stunted women are more likely to give birth to low birth weight babies who start life with a handicap.
Fig.2.1: Percentage of children under five who are underweight - moderate and severe

**Low Birth Weight and Mortality Rate**

The cut-off point in birth weight, below which mortality rates in infants are found to show significant increase, is generally considered to be 2,500g. Therefore, the current internationally accepted criterion for LBW is a weight at birth of <2500g [Int. Classfn 1977]

LBW has been found to be the major contributor to the high infant mortality rate of 72/1000 and high neonatal mortality rate of 42/1000 live births in the country. The mortality rates of infants born with a birth weight of <2500g remains higher and decreases continually with birth weight (Fig 2.2.) until an optimum is reached around 3400g followed by a slight increase for the highest weights. [Yerushalmy et.al.,1965]

Overall, it accounts for 800000 infant deaths annually. Among the LBW babies, 7-16% is preterm and 85-90% is with Intra Uterine Retardation, with gestation varying from
26 weeks to 36 weeks and birth weight from 450g to 2500g.

**Fig.2.2: IMR and Birth Weight**

**Source Ghosh et al., 1978**

The Child Survival Revolution, launched by UNICEF in 1982, has targeted major causes of child mortality through dissemination of simple low-cost technologies such as oral rehydration, immunization, breastfeeding and improvement in weaning practices.
The need to reduce the incidence of low birth weight infants is reinforced by the realization that there is a close relationship between LBW and infant mortality and is greatly influenced by socio economic status, education of the mother, age at marriage, spacing between children and birth weight. As a result, reducing the number of low birth weight infants has become a national goal.

India too evolved a National Health Policy in 1983 specifically targeting towards improving child survival in the country and formulated a set of goals to be achieved by the year 2000. The specific goals related to child survival include bringing down all components of child mortality especially, the reduction of neonatal and perinatal mortality to less than 30 per thousand live births (Table 2.6). Progressive improvements in the survival rates of children have followed. Under 5 mortality rate per 1000 births currently stands at a quarter of what it was in 1960.

Although significant progress has been observed in India in reducing mortality rates, analysis of the state level records reveals a rather poor quality of life of the survivors and
insignificant decrease in LBW rates. This observation raises serious concerns and necessitates an approach, which would start addressing qualitative aspects of child health, while continuing to address issues that cause high childhood mortality rates. Some of the common causes of perinatal death are depicted in Table 2.7

**Malnutrition and Child Mortality**

Many studies have pointed out that malnutrition was an underlying or contributing cause of death for approximately half

<table>
<thead>
<tr>
<th>Table 2.6: Child Survival: 2000</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Indicator</strong></td>
</tr>
<tr>
<td>Infant Mortality Rate</td>
</tr>
</tbody>
</table>
Perinatal Mortality Rate <35  43 (1997)
Child (0-4) Mortality Rate <10  23 (1997)

Table 2.7: The common causes of perinatal death
Maternal
Pregnancies at extremes of age - <18 years and >35 years
High Parity
Spacing of less than two years
Poor nutrition, particularly anemia
Toxemia
Prediabetes or diabetes
Fetal
Premature baby
Small but term baby-low birth weight baby (<2500g)
Infants
Birth injuries
Asphyxia neonatorum
Infections including tetanus
Hypothermia
Congenital malformations

of all deaths of children 1 to 4 years old in the countries surveyed. Fifty six per cent of child deaths overall were attributable to malnutrition’s potentiating effect on child mortality with three quarters arising from mild to moderate malnutrition Pelletier et.al.,[1994]. Trends in mild to moderate malnutrition for under five children in rural India have been 71% in 1974-79, 64% in 88-90 and 61% in 93-
94 respectively and do not show as steep decline as Infant Mortality Rate. It is therefore misleading to dismiss mild/moderate forms of malnutrition as being of not much consequence.

In all these studies, plotting logarithms of mortality risks against severity of malnutrition confirmed a correlation between mortality risks and anthropometric deficits, in addition to showing that risks of mortality increase exponentially with severity of malnutrition.

Prevention of malnutrition by all available means is particularly important because once it is established the consequences are grave. In the past five decades case fatality rates from severe malnutrition as reported in the literature have remained unchanged at between 20 and 30 per cent, with the highest levels (50 to 60 per cent) being among those with the presence of edema, which is an indicative of protein deficiency. Women, including adolescent girls and under five-year children, constitute the vulnerable groups because functional consequences of malnutrition in these groups are immense.
Origin of Malnutrition

Malnutrition, primarily caused by an inadequate food, is the result of complex interplay of a number of socio-economic and cultural factors like poverty leading to lack of resources, illiteracy, ignorance and ill health [Fig.2.3]

The effects of malnutrition on human health are felt right from the commencement of life in the womb and are transmitted across the generations. Poor maternal nutrition results in Low Birth Weight (LBW) in infants, who are at risk of growth retardation during childhood. This in turn, affects the nutrition and health of the adolescent girls who are “mothers to be”,


Fig. 2.3: The Multifactorial Aetiology of Malnutrition
leading to perpetuating the Transgenerational cycle of malnutrition. (Fig.2.4).

Breaking this cycle of malnutrition and improving women’s health are major challenges. It obviously depends on reducing low birth weight and improving the health and nutrition of children and of adolescent girls. A cohort study conducted in rural Hyderabad, showed that growth during adolescence is related to nutritional status of the girls at birth, highlighting the perpetuation of childhood malnutrition. This is not just prevalent among girls drawn from the poorer section of the society, even well to do adolescent girls failed to meet the international standards for growth. Therefore the focus should also be on improving the health of women during pregnancy.

As the maternal nutritional reserves of poor urban mothers are far below normal, low birth weights are encountered. To prevent LBW in the deprived population groups, it is imperative that intervention should start before birth (Chada et.al., 1992). Falkner et al. [1994] argues that it could take two or more generations before the effects
Fig. 2.4: Transgenerational Cycle of Malnutrition
of maternal nutrition and low birth weights in poor communities can be reversed.

**Determinants of Low birth weight**

A thorough global scrutiny for the causes of low birth weight reveals that many factors can influence the length of gestation or the rate of intrauterine growth, i.e., that the causality of LBW is “multifactorial”. Nonetheless, there is a considerable confusion and controversy about the factors that have independent effects on LBW as well as the quantitative importance of those effects. LBW in a country where most cases arise from IUGR will probably differ from those of a similar study in another country where most LBW infants are premature. [Kramer 1987]

Because the number of factors that might influence the duration of gestation or intrauterine growth is almost limitless, criteria were required to define the boundaries of the assessment. Kramer [1987] after an extensive review has identified 43 factors/criteria for assessment:

The clinical course of pregnancy and child birth depends on the combined influence of a host of genetic and
environmental factors and thorough knowledge of the natural history and epidemiology of human reproduction is required before one can fully understand the effect of diet during pregnancy upon the outcome [WHO 1965].

Factors influencing the outcome of pregnancy can be divided in the following three categories [Tyagi et.al., 1991] (Table 2.8-2.9)

(i) maternal factors prior to pregnancy
(ii) maternal factors during pregnancy
(iii) during fetal growth period

Recognition of socio-biological maternal factors affecting pregnancy outcome needs due attention to improve fetal well being, to ensure proper development of productive population of the future and to construct a complete data for further studies on fetal outcome among Indian pregnant females. Therefore the factors relevant to the objectives of the topic under research have been briefly reviewed.
Age at Marriage

Maternal age has long been recognized as a risk factor for perinatal mortality, for intrauterine growth retardation and also for preterm delivery [Bekketeig 1984; Golding 1991].

There is an

Table 2.8 : Maternal Factors Prior to Pregnancy
<table>
<thead>
<tr>
<th>Nutritional factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational weight gain</td>
</tr>
<tr>
<td>Caloric intake</td>
</tr>
<tr>
<td>Energy expenditure, work and physical activity</td>
</tr>
<tr>
<td>Protein intake/status</td>
</tr>
<tr>
<td>Iron and anaemia status</td>
</tr>
<tr>
<td>Folic acid and vit.B12</td>
</tr>
<tr>
<td>Zinc and copper</td>
</tr>
<tr>
<td>Calcium, phosphorus, and vit.D</td>
</tr>
<tr>
<td>Vit.B6</td>
</tr>
<tr>
<td>Other vitamins and trace elements</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Maternal Morbidity during pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>General morbidity and episodic illness</td>
</tr>
<tr>
<td>Malaria</td>
</tr>
<tr>
<td>Urinary tract infection</td>
</tr>
<tr>
<td>Genital tract infection</td>
</tr>
<tr>
<td>Table 2.10 Maternal Factors during Fetal Growth Period</td>
</tr>
<tr>
<td>-------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Antenatal care</strong></td>
</tr>
<tr>
<td>First antenatal care visits</td>
</tr>
<tr>
<td>Number of antenatal care visits</td>
</tr>
<tr>
<td>Quality of antenatal care</td>
</tr>
<tr>
<td><strong>Toxic exposures</strong></td>
</tr>
<tr>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Alcohol consumption</td>
</tr>
<tr>
<td>Caffeine and coffee consumption</td>
</tr>
<tr>
<td>Use of marijuana</td>
</tr>
<tr>
<td>Narcotic addiction</td>
</tr>
<tr>
<td>Other toxic exposures</td>
</tr>
<tr>
<td>Shorter duration of full-term gestation</td>
</tr>
<tr>
<td>37-38 weeks as against 40 weeks</td>
</tr>
</tbody>
</table>
increased risk of perinatal mortality associated with young maternal age and with maternal age of 35 years or more [Bekketeig 1984].

Pregnancy outcomes, including birth weight and gestational age are generally less favourable among adolescents and women over 35 years of age [Kramer 1987].

Adolescence has been considered as a risk factor for intrauterine growth retardation [Bolza et.al., 2001] because of physical and psychosocial factors as early marriage, low literacy, inadequate or no knowledge of contraception, very little or no maternal care, widespread malnutrition and anemia in that age group [Ghosh, S 1997]. In a retrospective study conducted by Bozkaya et.al., 1996, showed a 9.9% incidence of preeclampsia, 17.2% incidence of low birth weight and 9.3% of preterm delivery. An adolescent who becomes pregnant within three years of menarche is at special risk for a poor outcome of pregnancy since the nutritional demands of pregnancy are added to the needs of the mother who is still growing and adolescents are more likely to have inadequate diets than
Demographic and psychosocial factors
Maternal age (<18yrs or >35yrs)
Socio-economic status (education, occupation, and/or income)
Marital status
Maternal psychological factors

Genetic and constitutional factors
Infant sex
Racial/ethnic origin
Maternal height
Maternal pre-pregnancy weight
Weight-for-height ratio (Body Mass Index)
Maternal haemodynamics
Paternal height and weight
Additional genetic factors

Obstetric factors
Parity
Birth or pregnancy interval
Sexual activity
Intrauterine growth and gestational duration in prior pregnancies
Prior spontaneous abortion
Prior induced abortion
Prior stillbirth or neonatal death
Prior infertility
In utero exposure to diethylstilbestrol
adults. Their nutritional status appears to worsen also due to such factors as cultural beliefs, taboos and inappropriate food practices [Kraisid T 2000].

High proportion of our women attain motherhood well before they reach adulthood when their anthropometric status is much poorer and when, therefore, they are far more vulnerable to obstetric risks and risks of delivering LBW offspring than would have been the case if they had become mothers in their adulthood.

The average age at marriage of girls in our country, according to the Registrar-General data of 1981, was 16 years. An ICMR study [1990] showed that the average age at marriage of rural girls in six states of India was 13.8 years and their age at consummation of marriage was 15.3 years. Early age at marriage in our country is an important factor leading to high risk of low birth weight and therefore it would appear that a significant impact on the problem of low birth weights could be achieved through a rise in age of marriage of our girls. [Gopalan C, 1994]
Thirty percent of girls are married by 19 years of age. The Sample Registration System (SRS) estimates for 1992 showed that 10.3 percent of the births occurred in women below 20 years of age with a range of 3.3 percent in Punjab to 21.7 percent in Andhra Pradesh.

A WHO report states that children born to adolescent mothers are about 40 percent more likely to die during the first year of life than those born to women in their twenties and are even at greater risk during the second year.

Table 2.11 presents the percentage of teenage mothers (13-19 years) in different states of the country. The median age at marriage for the country as available is 16 years and the median age at first birth is below 20 years (table 2.12).

Under situations of reduced nutrient intake or increased nutrient requirements, competition between the mother and the fetus may limit the availability of the nutrients required for optimal fetal growth (King JC, 2000; Scholl et al., 2000). Hence, this study though registered adolescent, adult and
elderly pregnancies, predominated analysis on the adult pregnancies, between 20-35 years of age.

Table 2.11: Percentage of Teenage Mothers (13-19 years) in Different States of the Country

<table>
<thead>
<tr>
<th>States</th>
<th>Percentage of teenage mothers (13-19 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Madhya Pradesh</td>
<td>26.5</td>
</tr>
<tr>
<td>Nagaland</td>
<td>25.4</td>
</tr>
<tr>
<td>Karnataka</td>
<td>20.8</td>
</tr>
<tr>
<td>Haryana</td>
<td>20.7</td>
</tr>
<tr>
<td>West Bengal</td>
<td>20.4</td>
</tr>
<tr>
<td>Maharashtra</td>
<td>19.9</td>
</tr>
<tr>
<td>Bihar</td>
<td>18.7</td>
</tr>
<tr>
<td>Assam</td>
<td>17.9</td>
</tr>
<tr>
<td>Thirupura</td>
<td>14.9</td>
</tr>
<tr>
<td>Arunachal Pradesh</td>
<td>14.8</td>
</tr>
<tr>
<td>Uttar Pradesh</td>
<td>14.1</td>
</tr>
<tr>
<td>Orissa</td>
<td>13.2</td>
</tr>
<tr>
<td>State</td>
<td>Age at First Birth</td>
</tr>
<tr>
<td>--------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>Rajasthan</td>
<td>13.0</td>
</tr>
<tr>
<td>Tamil Nadu</td>
<td>12.0</td>
</tr>
<tr>
<td>Meghalaya</td>
<td>11.5</td>
</tr>
<tr>
<td>Gujarat</td>
<td>8.9</td>
</tr>
<tr>
<td>Delhi</td>
<td>8.0</td>
</tr>
<tr>
<td>Nagaland</td>
<td>7.3</td>
</tr>
<tr>
<td>Himachal Pradesh</td>
<td>7.1</td>
</tr>
<tr>
<td>Punjab</td>
<td>6.7</td>
</tr>
<tr>
<td>Jammu &amp; Kashmir</td>
<td>6.5</td>
</tr>
<tr>
<td>Kerala</td>
<td>5.9</td>
</tr>
<tr>
<td>Mizoram</td>
<td>5.7</td>
</tr>
<tr>
<td>Manipur</td>
<td>2.9</td>
</tr>
<tr>
<td>Goa</td>
<td>1.6</td>
</tr>
<tr>
<td>India</td>
<td>17.0</td>
</tr>
</tbody>
</table>

National Family Health Survey, 1992-93

Table 2.12: Interstate Differences in Age at Marriage and Age at First Birth
<table>
<thead>
<tr>
<th>State</th>
<th>Median age at marriage (yrs)</th>
<th>Median age at first birth (yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kerala</td>
<td>19.8</td>
<td>21.6</td>
</tr>
<tr>
<td>Tamil Nadu</td>
<td>18.1</td>
<td>20.1</td>
</tr>
<tr>
<td>Uttar Pradesh</td>
<td>15.1</td>
<td>19.5</td>
</tr>
<tr>
<td>Bihar</td>
<td>14.7</td>
<td>19.0</td>
</tr>
<tr>
<td>India</td>
<td>16.1</td>
<td>19.4</td>
</tr>
</tbody>
</table>

NFHS, 1992-93

According to National Nutrition Monitoring Bureau (NNMB), average weight and height of rural women are 42kg and 152cms. About 33 percent of 18 year old women have body weight below 40kg and height less than 145cms (NNMB 1991). This also shows the associated risk of maternal age in the incidence of low birth weight.

**Gravidal Status (number of pregnancies)**

Multiple pregnancies deplete stored nutrients in pregnant women. All studies show that the risk of perinatal loss
decreases after the first delivery, rising again after the fourth or fifth live birth [Bekketeig 1984]. Primi parity is associated with an increased risk of perinatal mortality [Bekketeig 1984; Golding 1991]. First born babies weigh less, and there is an increased risk of low birth weight. Among older women, primiparity has been showed to be associated with an increased intrauterine growth retardation and preterm delivery, even after controlling for maternal education, smoking, maternal diseases and obstetrical complications (Cnattingius 1992). High parity, also referred to as grand multiparity, conveys an increased risk to the fetus and results in higher perinatal mortality rate as well.

**Maternal Literacy Level**

A low level of maternal education increases the risk of perinatal mortality, intrauterine growth retardation and preterm delivery (Cnattingius 1992). Women who are illiterate experience the highest infant mortality rates and women with a primary and higher education, the lowest. Educated women has a better power of understanding the importance of antenatal care and the role of nutrition during
pregnancy and therefore is helpful to modify their behavior during pregnancy and reduce the incidence of low birth weights. Lack of education fails to inculcate in pregnant women the basic understanding of health care practices which is required for women at the time of conception and thereafter. Amin et.al., (1993) also reported that education is a very important factor which influences low birth weight.

It is generally seen that education has its impact on the life style of a person. Higher educational status is associated with better standard of life and positive relationship was found to exist between the nutrient intake and educational status of mothers. Education helps the mothers in understanding the instruction and to follow them. Minimum and maximum nutrient intake were observed among mothers of both the study area who were illiterate and educated upto graduation [Mridula et.al., 2002].

The importance of female education in influencing fertility rate is shown in table 2.13. From the trends in the table it is clear that education is an important way of ensuring durable achievements in fertility rate.
Socio-Economic Status

It has been long recognized that in any community, birth weights follow a socio-economic gradient. Studies conducted by Gopalan (1962) in Chennai were among the first to document the magnitude of these differences in the same ethnic group. Subsequent studies on birth weight in developing and developed countries, including the British Perinatal Mortality Survey, have shown this effect. In India, the magnitude of the

<table>
<thead>
<tr>
<th>State</th>
<th>Female Literacy</th>
<th>Total fertility Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bihar</td>
<td>29.6</td>
<td>04</td>
</tr>
<tr>
<td>Madhya Pradesh</td>
<td>34.3</td>
<td>3.9</td>
</tr>
<tr>
<td>State</td>
<td>Birth Weight</td>
<td>Difference</td>
</tr>
<tr>
<td>-------------</td>
<td>--------------</td>
<td>------------</td>
</tr>
<tr>
<td>Rajasthan</td>
<td>25.4</td>
<td>36.3</td>
</tr>
<tr>
<td>Uttar Pradesh</td>
<td>31.5</td>
<td>4.82</td>
</tr>
<tr>
<td>Kerala</td>
<td>82.5</td>
<td>2.0</td>
</tr>
<tr>
<td>Goa</td>
<td>72.9</td>
<td>1.9</td>
</tr>
</tbody>
</table>

NFHS, (1998)

The difference in birth weights between the high and low-income groups has remained about 400-500g in almost all the regions of the country (Prema R, 1989)
Socioeconomic status is an important determinant of health as well as of morbidity and mortality. Low socioeconomic status may be a social cause of nutritional, toxic, anthropometric or infectious factors that may themselves be causal determinants of low birth weight and therefore call for intervention. It is also independently associated with IUGR in both developed and developing countries (Kramer, 1987). The evidence between low socioeconomic status and preterm labor appears to be less consistent (Kramer, 1987).

In the developed countries women of low socio-economic status are more likely to have shorter birth intervals, make less use of antenatal care and have a higher incidence of systemic and genital tract infection. In developing countries, such women are likely to be shorter and thinner and to consume fewer calories and other nutrients during pregnancy [Kramer, 1987] resulting in dietary energy deficit which underlies most of the observed IUGR in developing countries (Prentice et al., 1983).

Women who marry into a low social class have such risk factors as short inter pregnancy interval, inadequate
nutrition, excessive work during pregnancy and reduced access to health care facilities (WHO 1978; Naeye, 1992;).

In some communities there may even be inequalities in the care provided so that there may be a serious mismatch between health care needs and the efficacy of the care actually provided (Garcia, 1989).

The most easily modifiable aspect of socio-economic status is maternal education [Kramer, 1987] since it is an important factor related to maternal and infant health and low birth weight is noticeable for infants born to parents in less favourable socio-economic circumstances indicated by the mother’s level of education [Khan, ME. 1993; Bhardwaj, et al., 1995; Regi, A et al., 1995; Mavalankar DV, et al., 1991;]

The variables that affect socio economic status are different in case of urban and rural societies. Poor mothers in urban as well as rural areas were found to be chronically undernourished prior to pregnancy. For example, the influence of caste on special status is very strong in rural community but not so much in the cities. Separate scales are hence used for measuring the socio economic status.
Although a variety of methods of classifying the population by Socio Economic Status [SES] have been proposed, the most widely used for urban populations is the one proposed by Kuppuswamy in 1979 [Kuppuswamy, 1981].

The SES scale (urban) developed by Kuppuswamy attempts to measure the socio-economic scale of an individual in an urban community. It is based upon three variables—education, occupation and income. A weightage is assigned for each variable according to a seven point predefined scale. The total of three weightages gives the socio economic score which is graded to five classes. A modification of this scale was suggested by Mahajan [1995] which assess the per capita family income in the place of gross monthly income, and the current price status in 1990. Another modification of the scale proposed by Mahajan [1995] considers the current price levels.

Some studies have shown that the intrauterine growth curves of fetuses of poor socio economic group mothers deviate from those of the better off Indian mothers and the Western mothers during the third trimester of pregnancy,
resulting in the birth of a LBW baby because of intrauterine growth retardation (Fig 2.5).

Fig. 2.5: Intrauterine growth – comparison of Indian and Western Studies

Source: Usher et al., Ghosh et. Al.,

The socio-economic status assessment scale used in this study is presented in Table 2.14.

Poor women gain only about three to five kg weight during pregnancy as opposed to 10 to 12 kg, which better socioeconomic group women do. In a study by Ramachandran (1989) it was found that dietary intake of high-income pregnant women was 2000 to 2500 kcal per
day during pregnancy. Their average pre pregnancy weight was 45 to 55 kg. Weight gain during pregnancy was 11kg and mean birth weight of infants was 3.1 kg. On the other hand low income women consumed 1200 to 1600 kcal, their body weight was 43 kg and they 6kg during pregnancy. Mean birth weight of infants was 2.7kg.

**Anthropometric Measurement**

The pattern of growth and physical status of the body, though genetically determined, are profoundly influenced by diet and nutrition. Hence, anthropometric measurements are useful criteria for assessing nutritional status. Physical measurements such as height, weight and body mass index reflect the total nutritional status over a life time (Taneja et.al., 1998).

**Table 2.14: The categorisation of Socio Economic Status**

<table>
<thead>
<tr>
<th>Particulars</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Education</td>
<td></td>
</tr>
<tr>
<td>1. Professionals/Honors</td>
<td>07</td>
</tr>
<tr>
<td>2. Graduate/Post Gradua</td>
<td>06</td>
</tr>
<tr>
<td>3. Intermediate/Post High</td>
<td>05</td>
</tr>
<tr>
<td>School Dip</td>
<td></td>
</tr>
<tr>
<td>4. High School Certificate</td>
<td>04</td>
</tr>
<tr>
<td>5. Middle School Certificate</td>
<td>03</td>
</tr>
<tr>
<td>6. Primary School</td>
<td>02</td>
</tr>
</tbody>
</table>
7. Illiterate 01
B. Occupation
1. Professional 10
2. Semi professional 06
3. Clerical, shop owner/farm owner 05
4. Skilled Worker 04
5. Semi-skilled worker 03
6. Unskilled worker 02
7. Unemployed 01
C. Family Income/Month (Rs)
1. > 13,500 12
2. 6750-13,499 10
3. 5050-6749 06
4. 4375-5049 04
5. 2025-3374 03
6. 676-2-24 02
7. <675 01

<table>
<thead>
<tr>
<th>Total Score</th>
<th>Socio Economic Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>26-29</td>
<td>upper</td>
</tr>
<tr>
<td>16-25</td>
<td>upper middle</td>
</tr>
<tr>
<td>11-15</td>
<td>lower middle</td>
</tr>
<tr>
<td>5-10</td>
<td>upper lower</td>
</tr>
<tr>
<td>&gt;5</td>
<td>lower</td>
</tr>
</tbody>
</table>

**Maternal Stature**

Height is accepted as a measure of nutritional status throughout life and there is a general agreement that a positive relationship exists between maternal height and mean birth weight. Most of the evidence suggests that maternal short stature/stunting, determined by early

Although factors such as genetics and health history also influence skeletal size, low maternal height and head circumference reflects maternal stunting secondary to nutritional experiences, particularly in early life (Bakketeig et.al., 1984; Golding J 1991). Short stature was associated with low social class, although controlling for social class did not eliminate the effect of stature on perinatal mortality (Baird, et.al., 1969). Short women also tended to have an increased risk of intrauterine growth retardation. Few studies have addressed whether short stature is associated with an increase in preterm births (Berendes 1993). Yadav et.al., (1986) in their study using various cut-off levels of maternal height, also observed height to be a risk factor for LBW.
Observations of Glass et al., 1984 and Lucas et al., 1990 show that stunted mothers are at greater risk of producing a LBW infant and hence there is a vicious cycle of stunted mothers giving birth to girls of low birth weight who because of the permanent damage suffered by them because of IUGR grow and develop in a substandard growth and development category ending up as stunted mothers.

ICMR study (1981-84) in three urban slums in Madras, Delhi, and Calcutta and three rural areas near Chandigarh, Varanasi and Hyderabad (Prem K, 1985) also revealed that maternal height of less than 145 cm is a risk factor for the newborn. Recently Sharma et al (2001) showed that the mean birth weight of women whose height was <150cm was 2.58±0.48kg which is significantly low when compared with the mean birth weight of 2.93±0.26kg in women with a height >165cms. According to the data gathered by the National Nutrition Monitoring Bureau (NNMB 1980) 12 to 25 percent women in our country are with heights less than 145cms.
Maternal short stature leading to shortened gestation by increasing the risk of idiopathic preterm labour was shown by Kramer et al 1995. Maternal height was significantly associated with birth length (Fawzi et al in 1997).

**Maternal Pre pregnancy weight**

Though maternal height has been regarded as a significant independent variable in determining birth weight, height alone is inconsequential with respect to birth weight as this effect is only mediated through weight, which also reflects pre pregnant nutritional status in addition to being closely correlated with height.

Even after correcting for stature, body weight is in part genetically determined and genes that control adiposity or lean body mass could, theoretically, be expressed in the newborn. Even in the absence of such expression, however maternal weight prior to conception reflects nutritional stores potentially available to the growing fetus [Kramer, 1987].
Under weight women have lower plasma volume and rennin-aldosterone response in pregnancy compared with normal weight women [Salas et al., 1998]. It is possible that such lower plasma volume may be associated with uteroplacental insufficiency and the increased prevalence of small for gestational age babies.

A positive and significant relationship between maternal pre-pregnancy weight or pre-pregnancy weight-for-height (BMI) and fetal size has been repeatedly observed in both well-nourished and poorly-nourished populations (Abrams and Laros, 1986; Brown et al., 1981; Committee to study the Prevention of Low Birth Weight, 1985; Eastman and Jackson, 1968; Edwards et al., 1979; Kleinman and Madans, 1985; Kramer, 1987 a; Kramer 1987b; Niswander et al., 1969; Taffel, 1986; Villar and Belizan, 1982).

Pregnant women with preconceptional weight 10% or more below standard for height and age have been found to be at increased risk of delivering a low birth weight. Women who enter pregnancy with 20% or more over standard weight for height and age also exhibit impaired pregnancy outcome [Pitkin et al. 1981]. This risk appears to relate to
both IUGR low birth weight and to preterm low birth weight although the data for IUGR are more consistent [Frank Falkner, 1991; Bakketeig et al., 1984; Villar et al., 1990; Golding J 1991; Berendes 1993].

In addition to higher incidence of low birth weight, pregnancies in underweight women are also associated with several adverse outcomes including birth asphyxia, anemia and increased prenatal mortality rates [Kramer et al., 1995; Spinillo et al., 1998].

WHO world-wide data considers, women with pre-conceptional body weight less than 38kg to be in the “high-risk” category, i.e., likely to suffer obstetric complications and give birth to offspring of low birth weights and the report of the National Nutrition Monitoring Bureau shows that over 15 to 29 percent of adult Indian women in 10 states of India have body weight less than 38 kgs. Sharma et al., [2001] also had recorded a statistically significant risk of delivering low birth weight infants among women who weighed <50kgs in her study group.
Calculated or recalled pre-pregnant weight or that measured at mid-pregnancy has been shown to be a determinant of birth weight. Self reported pre-gravid or pre pregnancy weight was considered to be biased by many. But, that the self-reported weight correlates well with measured weight has been shown by many researchers [Stunkard AJ 1981; Steward AL 1982; Stevens 1986]. Hickey et. al., 1993, also determined a positive and significant correlation (0.96), between the self-reported and weight at the first prenatal visit.

An ICMR study conducted in 1992 showed that only 11 to 38 percent women were having weight between 100 to 90 percent, including pregnant women. Standardized weight for Indian reference women is 50kg.

**Weight Gain During Pregnancy**

Weight gain during pregnancy reflects increases in extra cellular fluid and blood volume, amniotic fluid, maternal fat accretion and the products of conception - the fetus and the placenta and is an important overall indicator of the nutritional status of the mother and fetus (Fig.2.6 and 2.7).
Fig. 2.6: Weight Gain During Pregnancy

Source Ghosh et al., 2000

Fig. 2.7: Pattern and components of Maternal Weight Gain during Pregnancy

Source: Clin Obstet Gynecol 1976;19;489-513

Good nutrition during pregnancy is reflected by satisfactory weight gain of the mother and has a better impact on fetal
growth (Kaur R, et.al., 1997; Nutrition Foundation of India 1988; National Academy of Sciences, 1990). Maternal weight gain is has been shown to be a strong predictor of infant birth weight (Chadha V.k.et.al., 1992; Shayna et.al., 1997) with a correlation coefficient of 0.49 (Karim and Taylor 1997).

Underweight women with a low gestational weight gain are at highest risk of delivering a low birth weight infant and this risk decreases with increasing weight gain (Brown et al., 1981; Edwards et al., 1979; Abrams and Laros, 1986; Amderson et al., 1984). Alternatively excessive weight gain is associated with higher birth weights and increased rate of hypertension and preeclampsia, caesarean delivery labour abnormalities, postdatism and meconium staining. Inappropriate weight gain (adjusted for gestational age) may also be associated with preterm birth, but only limited data address this issue (Hediger, et al., 1989, Abrams, et al., 1989, Berkowitz, 1981; Papiernik et al., 1974; Van den berg and lescgskum 1984). Appropriate gain in weight particularly during the second and third trimester, is an important determinant of fetal growth, [Eastman et.al., 1968; Naeye RL, 1979; Edwards LE,
Good pregnancy outcome occurs within a wide range of maternal weight gains [Abrams 1986; Kramer 1987; Van der Spuy, 1988; Parker 1992] Although an average pregnancy weight gain of 12.5kg has been proposed as optimal, women in developing countries have been reported to gain on an average between 3 and 7kg. (Subcommittee on Maternal and Infant Nutrition in developing Countries, 1983; Collaborative Research Support Program, 1988).

A series of studies in five countries (Scotland, the Netherlands, Thailand, the Philippines and Gambia) estimated that the absolute mean maternal weight gain during pregnancy was low in the less developed compared to more developed countries (7-9kg compared to 10-12 kg). When weight gain was expressed as a proportion of mean maternal prepregnancy weight, women in Scotland, Netherlands, Thailand and the Philippines gained a similar proportion of their pre gravid weight while women from the Gambia gained somewhat less (Durnin 1987)
In a study of 4674 singleton pregnancies, Abrams, observed that only a minority of mothers gained within the recommended range of 9 to 13.6kg and that 80% gained within 10 to 21kg (Abrams. 1994). The results of Abrams study also support the recommendation of a minimum of 9 to 10kgs gestational weight gains for better pregnancy outcome.

Thinner women are at greater risk of delivering small infants and in such cases a maximum pregnancy weight gain may be protective against low birth weight (Allen et.al.,1993). It would therefore, appear that if undernourished women could achieve substantial increase in body weight during pregnancy, low birth weights in their offspring can be avoided.

The observation mentioned above indicated that substantial success with respect to reduction in low birth weight incidence can be achieved even with malnourished mothers through vigorous antenatal care and improved nutrition during pregnancy reflected in satisfactory weight gains.
Kelly et al in 1996 concluded that a single measurement of attained weight at 5 or 7 lunar months is the most practical screening instrument for low birth weight and IUGR in most primary health care settings and provides warning of the need for intervention.

Of greater use than total gain, however, is the pattern by which weight accumulates throughout gestation. A persistent weekly weight gain of less than 0.3kg predicts poor pregnancy outcome revealing that such women require dietary intervention at least during the 3rd trimester (Rosso 1990; Mercy P., et.al., 2002). According to the report of the International Conference on Maternal Anthropometry for prediction of pregnancy outcome, a gain of less than 0.25kg/wk is a danger signal calling for immediate action. Hence, it is suggested that assessment of velocity or increment of weight gain in the second and third trimester of pregnancy is essential for monitoring intra uterine growth (WHO 1991).

Weight gain of the mothers during the gestation period had a direct effect on the birth weight of the infants. The
mothers who gained only 4.69 kgs during gestation delivered low birth weigh neonates with a mean birth weight of 2.29kgs. in contrast to those who gained greater five kgs had delivered neonates with higher birth weight 3.2kgs.

Though maternal heights are related to birth weight of infants studies carried out by the Foundation show that maternal weight and especially maternal weight gain during pregnancy is an important determinant of the birth-weight of the infant. Thus as against the incidence of 30-33 percent low birth weight deliveries in poor communities in general, the incidence of low birth weight deliveries in women with a prepregnancy weight of more than 45 kgs was no more than 17 percent (NFI 1988).

Satisfactory weight gain sin pregnancy can be achieved through nutrition intervention (Prema R,1993). Satisfactory weight gain for Indian reference woman during pregnancy is 10 to 12 kg. In first trimester the weight gain is three kilograms, second trimester 5.5 kilograms and in the third trimester it is four kilogram (ICMR 1992)
**Maternal Body Mass Index**

BMI is being increasingly used as a measure of nutritional adequacy in adults and is considered as a better indicator of chronic energy deficiency (CED) (Reddy et al., 1992). It is also a good index to assess the current forms of malnutrition in a community (Raman et al., 1991). The influence of maternal age, parity and height on fetal outcome vis-à-vis maternal nutritional status and its effect on fetus has been surmounted to some extent by the use of Body Mass Index (BMI; maternal weight for height ratio), which is independent of these factors (Sachdev, 1994).

The time trends in BMI of adult women from the NNMB data are summarized in table 2.15. A distinct shift of the distribution to the right is evident from in the rural population. CED is prevalent even now in 37 to 47 percent of the women, with the severe cases documented at 10 percent. Obesity is also beginning to emerge (7 to 12 percent) now (Sachdev 1997).

Although a linear relationship between maternal weight gain and birth weight has been observed for all categories
of maternal body mass, as pre-pregnancy body mass increases, the influence of gestational weight gain appears to become less important (Abrams and Laros, 1986; Anderson et al., 1984; Eastman and Jackson, 1968; Hytten and Chamberlain, 1980; Kramer, 1987 a & b; Niswander et al., 1969; Rosso, 1985; Taaffer, 1986).

A prepregnancy BMI greater than 29 is more predictive of healthy birth weight than of gestational weight gain and that the predictive value of BMI determined by Spearman correlation, was greater than the predictive value of skin folds, bronchial circumference, adipose and muscular surfaces or fat mass. Wandja et al., (1995). Naidu et al., (1991) reported the incidence of low-birth-weight which is an indicator of “intrauterine malnutrition” is higher among mothers with a low BMI status.

A cross sectional data on women aged 18-74 years, showed that BMI was highest in Egypt and intermediate in Mexico and increased with age in both countries. In contrast, the BMI of Kenyan women was lower and did not increase as they became older. Based on these data it has been suggested that the degree of change in women’s
BMI with age might be a useful indicator of general energy adequacy in population groups (Calloway et al., 1988).

Table 2.15: Trends in Body Mass Index in Adult Women Survey (values in percentage %)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>III (&lt;16)</td>
<td>12.7</td>
<td>11.3</td>
<td>10.4</td>
<td>9.5</td>
</tr>
<tr>
<td>II (16-17)</td>
<td>13.2</td>
<td>12.9</td>
<td>11.2</td>
<td>9.2</td>
</tr>
<tr>
<td>I (17-18.5)</td>
<td>25.9</td>
<td>25.1</td>
<td>25.5</td>
<td>18.0</td>
</tr>
<tr>
<td>All (&lt;18.5)</td>
<td>51.8</td>
<td>49.3</td>
<td>47.1</td>
<td>36.7</td>
</tr>
<tr>
<td>Normal</td>
<td>44.8</td>
<td>46.6</td>
<td>46.3</td>
<td>51.7</td>
</tr>
</tbody>
</table>
Prenatal weight gain among healthy women were reviewed and guidelines published based on pre-pregnancy weight in relation to height, expressed as Body Mass Index (BMI). The recommended gain are presented in Table 2.16.

Parker and Abrams [1992] conducted a study on an heterogenous population of women delivering full term infants to examine the associations between maternal weight gain outside the Institute of Medicine recommendations on the incidence of small for gestational, large for gestational age and cesarean delivery. They had reported that the prenatal weight gain within the Institute of
Medicine ranges reduced the risk of the outcomes. Similar observations were also carried out by Hickey et.al., [1993].

In this study, the association of prenatal weight gain below, within and above the Institute of Medicine recommendations with pregnancy outcomes and birth weight was observed.

### Table 2.16: Recommended Weight gains based on BMI

<table>
<thead>
<tr>
<th>BMI Category</th>
<th>Recommended Weight Gain (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight (BMI &lt;19.8)</td>
<td>12.5-18</td>
</tr>
<tr>
<td>Adequate weight (19.8-26)</td>
<td>11.5-16</td>
</tr>
</tbody>
</table>
Other Maternal Anthropometric Values

Compared to the large amount of data on pre pregnancy weight and gestational weight change, relatively few studies have assessed the relationship between other aspects of maternal body composition and pregnancy outcome. Potentially relevant measures of body composition include maternal arm circumference and skin folds to identify undernourished women during pregnancy.
(Tinker A et al., 1993). However measures of limb circumference avoid the problem of including fetal components and only give information specifically about maternal body composition. Skin folds estimate subcutaneous fat. A combination of the arm circumference and triceps skin fold measurements is required to assess fat and lean tissue reserves. Both methods have methodological and practical limitations during pregnancy.

**Biochemical Assessment**

Of fundamental importance is the concept that many laboratory values are altered by normal gestation in a manner that yields results which, judged by standards derived from non-pregnant individuals, suggest deficiency states. Thus, blood levels of a number of substances such as glucose, calcium, albumin, trace minerals, most amino acids, and nearly all water soluble vitamins, decline with pregnancy. Other blood components, such as lipids and fat-soluble vitamins, rise in concentration during gestation. These changes often are unpredictable on theoretical grounds and failure to recognize them as entirely normal
adjustments in maternal physiology can lead to erroneous interpretations.

Assessment of laboratory measures of nutritional status is limited by methodological problems in the biochemical assay of certain nutrients. Of additional concern are the changes in nutrient serum levels and urinary excretion, which occur as normal physiological adaptations to pregnancy, but which resemble signs of deficiency in non pregnant individuals (Hytten and Chamberlain, 1980; Hytten and Leitch, 1971 ; Winick, 1986). For example, even when maternal iron status is adequate, hemoglobin and hematocrit levels decrease as a result of hemodilution, and decreased levels of vitamins during pregnancy do not necessarily reflect dietary intake and may represent a "normal" response to pregnancy.

Among the numerous maternal factors likely to affect fetal growth (Kramer 1987), while some are modifiable in a positive direction even after conception eg. Maternal nutrition and hemoglobin status by proper antenatal care, others are beyond our control. Hence, it is important to assess the influence of these modifiable factors on
pregnancy and its outcome [Badole et.al., 1991]. Therefore
The only biochemical assessment done in this study was
maternal serum hemoglobin.

**Maternal Hemoglobin**

Maternal anemia is a burning national public health
problem and had been related to poor prenatal and
neonatal mortality. According to the WHO defence
committee on Haematology 1968, a level of HB <11g/dl
during pregnancy has been accepted as indicative of
anaemia holds true till date.

Anemia is found to be far the most widely prevalent
nutritional disorder in the world (sood S.K.1975). It is
particularly severe in developing countries affecting half of
its population. Pregnant women are the most vulnerable
group with the prevalence of anemia being highest among
them. It ranged from 21 to 80 percent in different countries
and India had the highest prevalence with 80 percent of
them suffering from it according to W.H.O quarterly
statistics 1982. An ICMR study (1982) found that over 95
percent of 6 to 14-year-old girls in Calcutta were anemic.
The figure for Hyderabad was 67 percent, for DELHI, 73
percent and for Madras 20 percent. More recently in a study in Gujarat by Kanani, 90 percent adolescent girls were found to be anemic. Anemia results in higher rate of fetal deaths and low birth weight. Since a majority of adolescents girls are anemic, they enter marriage and pregnancy in an anemic state.

Iron deficiency was found to be the most common cause of anemia among pregnant women though deficiency of folate and vitamin B12 were also associated with it (Bothwell T.H. 1972). The physiological changes that take place during pregnancy impose an additional demand of iron on the mother to cater to expanding blood volume and fetus (Fenton V et., al., 1977, Taylor and Lind 1979, Theodore and Ariar 1979). The inability to meet this additional demand of iron through dietary source and consequent exhaustion of iron stores lead to anemia. Most women in developing countries start their pregnancy with absent iron stores (Puolakka et al., 1980). This would possibly explain the high prevalence of anemia in developing countries. In India it is aggravated by the fact that they depend mainly on cereal and vegetable foods which are poor in iron content and as well as it is non-heme based. Besides this
there is also the presence of phytates and phosphates and these are known to inhibit iron absorption (Narasingha Rao et al. 1983)

Several other factors like age, gravidity and gestational age are also known to influence anemia. Young primigravida tend to have lower hemoglobin levels (Hussain et al. 1984). Likewise primigravida and multigravida were found to have lower hemoglobin concentration (Isah H.S. et al., 1985). Hemoglobin concentration was observed to decrease with increasing gestational age. (Shukla M.L. et al. 1982).

Observational studies indicate that anaemia in pregnancy is correlated with poor birth outcomes (Bkack., 2001) and is important risk factor contributing to the high incidence of low birth weight which is three times greater than non-anemic women. Anemia, especially if severe, may impair the oxygen delivery to placenta and fetus, thus interfering in normal intrauterine growth.

Several studies have reported association of anemia with maternal and fetal morbidity (Bakers J 1979) and morbidity as well as prematurity and still birth (Achari and Rani
Likewise studies have also reported on the high incidence of low birth weight babies delivered by anemic mothers (Rozokowski et al. 1966, Verma and Dhar 1976, Kaltreider and Gohnson 1976 and Tyagi N.K. et al. 1985). A significant fall in birth weights has been reported to occur with maternal hemoglobin levels below 8g/dl.

High levels of hemoglobin was found to be significantly associated with high mean birth weight. With every gm% rise in Hb level of mother, birth weight of babies was higher by approximately 100gms. Babies of mothers with Hb level of 11gm% and above were about 450gms heavier than those of mothers with haemoglobin level below 8gm% [Chadha et.al., 1992].

Hemoglobin concentration drops during pregnancy due to hemodilution (a proportionately greater increase in plasma volume as compared to the increase in red cell mass). Hence the course of supplementation as well intake of nutritious foods should be regular in order to achieve a desirable nutritional status.
Mean birth weight was observed to rise with hemoglobin of mother. With every gm% rise in hemoglobin level of mother, birth weight of the babies was higher by approximately 100gms. Babies of mothers with Hb level of 11gm % and above were about 450gms heavier than those of mothers with haemoglobin level below 8gm % (Chadha et.al., 1992).

Based on the hemoglobin status the pregnant women were categorised into normal or anaemic and the anaemic pregnant women were further grouped into mild, moderate or severe according to WHO’s categorisation (Demaeyer, 1989) (Table 2.17)

Anemia is widely prevalent and almost 54 percent (Fig.2.8) of women in Tamil Nadu have been shown to be anemic during the third trimester of pregnancy (Sheshadri, 1995) and have hemoglobin level of less than 11g/dl, the
minimum level of recommended by the WHO.

Fig.2.8: Prevalence of anemia in pregnant women (Hb level less than 11g/dl)

<table>
<thead>
<tr>
<th>Categorisation of Anemia</th>
<th>Hb (g/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&gt;11</td>
</tr>
</tbody>
</table>

Table 2.17: WHO categorisation of Anemia
Mild anemia 11-10
Moderate anemia 10-7
Severe anemia <7

In pregnant women from Hyderabad, possibly from different settings (Prema R, 1981; ICMR 1992), the prevalence of anemia (hemoglobin <11g/dl) has significantly declined from 48.5 percent to 33.2 percent. Estimates based on the ICMR evaluation in 1984-85 of the National Nutritional Anemia Prophylaxis program indicated that 88 percent of pregnant women were anemic and 47 percent (Fig. 2.9) had hemoglobin values below 9g/dl (ICMR, 1989). The latest estimates pertaining to the ICMR multicentric field supplementation trial (published in 1992) on 1,968 pregnant women lowered these estimates to 62 percent and 17 percent respectively (ICMR, 1992).

Fig. 2.9: Percentage of anemia (ICMR 1989)
Observational studies indicate that anemia in pregnancy is correlated with poor birth outcomes (Black 2001) and is an important risk factor contributing to the high incidence of low birth weight which is three times greater than non anaemic women (Allen, 1993).

Several studies have reported association of anemia with maternal and fetal morbidity (Bakers J 1979) and De Maeyer as well as prematurity and still birth (Achari and Rani 1971).

Young primigravida and multigravida tend to have lower hemoglobin levels (Hussain et al., 1984; Isah H.S. et.al., 1985)

An ICMR study (1982) found that over 95 percent of six to 14 year old girls in Calcutta were anemic. The figure for Hyderabad was 67 percent, for Delhi, 73 percent and for Madras 20 percent. Kanani S in Gujarat showed over 90 percent adolescent girls were anemic. Anemia results in higher rate of fetal deaths and low birth weight.
Nutrient Intake

Poor nutritional intake by Indian women as compared to requirements is a well-known fact. The gap further widens during periods of stress such as pregnancy. An expectant mother needs to be provided adequate nutritional intake for maternal and fetal tissue growth and her own usual maintenance requirements. An acute or chronic deprivation of dietary intake may result in a poor pregnancy outcome (Naeye, 1990) and this is reflected in higher prevalence of low birth weight and decrease in mean birth weight.

The total calorie cost of supplying and maintaining the fetus has been estimated to be about 40,000 kcal. Since the greater part of calories will be required mostly during the 2nd and 3rd trimesters, the additional requirements will work out to 200kcal/day. The ICMR Nutrition Expert Group, however, recommended an extra allowance of 300kcal/day.

Amongst the various nutrient needs during pregnancy, adequate energy intake appears to be the most crucial.
This is reflected in decrease in birth weight of 300g-500g in famine, dietary control among well nourished pregnant women and infants born to poor undernourished mothers.

Dietary proteins are essential for fetal growth and metabolism as these proteins provides increased nitrogen and amino acids required for maternal and fetal tissue synthesis [Sachdev et.al., 1994]. Available evidence would indicate about 910g of proteins are deposited in the fetus and maternal tissues during pregnancy. The average daily increment is estimated to be about 5g during the last six months of pregnancy. This will work out to 10g additional proteins in terms of dietary proteins of NPU 50. The ICMR Nutrition Expert Group recommended an extra allowance of 14g/day.

Majority of workers are of opinion that maternal nutrition during pregnancy affects fetal growth [Bhatia et.al., 1983;Kramer, 1987]. Persistantly poor dietary intake throughout gestation results in poor weight gain during pregnancy, which in turn leads to restricted fetal growth.
Nutrition Education

Ample data exist to indicate that under nutrition and poor antenatal care are the major factors responsible for LBWs; improvement in nutritional status when coupled with improved health care has been shown to substantially improve birth-weight. It will obviously not be feasible to undertake massive food supplementation programs targeted at pregnant and lactating women as a public-health policy.

On the other hand it should be possible to achieve improvements in the home diets of pregnant women through a program of nutrition education, designed to help them use inexpensive foods within their economic reach. For example, inexpensive green leafy vegetables when included in the diets of pregnant women, can provide substantial amounts of carotene, vitamin C, iron, zinc and calcium. This will take care of their problem of micro nutrient deficiencies to a considerable extent. Unfortunately, health agencies have not given adequate attention to this practical approach. Good antenatal care must include the component of nutrition education. With
adequate attention towards achieving dietary improvement in poor communities through a sustained program of nutrition education, the results would be far more rewarding. Pregnant women stand in dire need of better diets, not just tablets and capsules only; and such better diets are possible even in the context of poverty with minimal inputs (Prema R, 1990).

Maternal health, nutrition and education are important for the survival and well-being of women in their own right and are key determinants of the health and well-being of the child (Ghosh S, 1994)

Effective utilization of information, education and communication strategies is the core of social marketing. It has been well recognized that one of the weakest links in intervention programs to control malnutrition is absence of proper nutrition education. Scarcity of appropriate location specific educational materials is the weakness of nutrition education endeavors. Therefore preparation of location specific nutrition education material is the need of the hour. [Saibaba et.al., 1999]
Nutrition education is implementation of planned learning process which involves information exchange as well as techniques to motivate and reinforce improved food and nutrition habits in the pursuit of improved health/nutritional status [Alice, M 1982]. Basic objective of nutrition education is to motivate people to adopt healthful nutrition practices.

The role of women in combating malnutrition through changing the social, cultural barriers for good nutrition is vital and unique. Moreover the cost to society of caring for people with diseases such as diabetes and coronary vascular disease, as result of low birth weight, are high and the costs of preventing these diseases by adequate nutrition in utero or during pregnancy would be considerably less [SCN news 1997].

Therefore it is essential that the woman has adequate nutritional knowledge to influence the child and hence the society indirectly. Prenatal Stage represents an ideal opportunity for imparting correct nutritional practices, since at that time, the to be mothers are highly motivated to understand and accept advice (Orstrad C et.al., 1985).
There exits a gap between nutrition knowledge and nutrition practices among pregnant women. They are often mystified and unaware of how to make the wisest food choices from a variety of available foods. They should be made knowledgeable about how to meet nutritional needs without over indulgence in food and wise selection of an appropriate diet from the vast supply of food available. Therefore, Nutrition education becomes necessary to aid them to make economical food choices, that is, to save money while getting optimal nutrition. This is especially important for those from low-income groups.

A significant and favorable impact of regular utilization of antenatal care services by expectant mothers and outcome of pregnancies in terms of Birth Weight and lower incidence of low birth weight, has been reported by showstack et al, (1984) shrivastava et.al., (1982) and Badok et.al.,(1991).

Kaur et.al. (1997) and sachdeva et.al (1993) had reported a better dietary intake and regular intake of iron and folic acid supplements under the influence of nutrition
counseling. Thus, there is a wide scope of imparting nutrition counseling, but there is a need to impart concerted nutrition education with simplified instructions, good rapport with the pregnant women and regular follow-up in diet counseling centers for longer duration to improve the nutritional status of pregnant women and their newborn.

It is also to be noted that mother who attend antenatal clinics regularly are also more likely to have better nutritional status even before the advice due to their better health awareness. Hence, the major emphasis of any antenatal care program should be to identify non attenders or likely dropouts and serve them specifically, Badole et.al., [1991]. Further the authors have concluded that effective modification of both nutrient intake and hemoglobin levels during antenatal visits and regular and efficient antenatal services can significantly influence birth weight and fetal growth.

Nutrition counseling is one of the most effective tools of changing the food habits of people without affecting their sentiments. It is the process by which beliefs, attitudes,
environmental influences and knowledge about foods and health channeled into actual practice which are sound and consistent with individual needs, purchasing power, food availability and socio-cultural background (Orstead, et.al., 1985).

This study used the nutrition counseling strategy to influence the food intake of gestational women as a method to improve the nutritional status of the mothers and their offspring.

In our country the diets of the expectant mothers are poor and their reserves are minimum. To meet the needs of growing fetus, the maternal reserves are exhausted and the later becomes nutritionally deprived leading to high mortality rate of such women in addition to the unborn child. So, continued efforts to develop feasible and effective ways of improving maternal nutrition, during pregnancy are required. Since most ladies are not aware of increase in nutritional requirements during pregnancy, nutrition education during antenatal visits should be considered as an important activity.
Fetal Assessment

Human fetal growth is not uniform and its control is complicated. The first level of control is genetic, the second resides in foeto-placental homeostatic mechanisms and the third in maternal environment acting through the placenta. The overall growth of the fetus in pregnancy is indicated by its body size and anthropometric measurements at birth, particularly weight [Rondo et.al., 1998].

Intrauterine development determines to a certain extent, the future somatic and psychomotor development of an individual and impaired growth in utero has an impact on perinatal morbidity. The evaluation of intrauterine growth is important for rational planning of possible obstetric intervention and for the neonate’s proper management since both over estimation and an under estimation can result in the delivery of a premature infant/fetal death respectively.
Maternal nutrition and fetal growth

Although it may seem obvious that human fetal malnutrition is the result of appropriate delivery of nutrients to or utilization by the growing fetus a specific relationship to the nutritional status of the mother in mid pregnancy has not been identified previously. In developed countries, most mothers who deliver fetally malnourished infants do not show evidence of serious malnutrition. Nonetheless, there is a consensus that maternal nutrition in some way influences human fetal growth. This is based largely on epidemiological studies, some food intervention programs, morphological studies and innumerable animal investigations. Prospective studies attempting to relate quantitative measures of nutrient status of the mother at mid pregnancy or earlier to the growth of her fetus have been limited.

First fetal growth is modulated by maternal nutrition, then some sort of subtle malnutrition, e.g., a nutrient imbalance among nutrients without a deficiency state may affect growth adversely as demonstrated by Harper and
associates in experimental animals. Tews et al., showed that the dispensable amino acids serine and alanine compete for cell transport in vitro. Specialist constructed diets could induce predictable in vitro imbalance sufficient to impair protein synthesis and growth in rats. Thus the imbalance of nutrients, including amino acids, in the maternal microenvironment which are potentially available to the fetus, may modulate fetal growth.

To assess the role of maternal nutrition as regulator of fetal growth, non-nutritional factors contributing to fetal growth must be accounted for. Weight is the most commonly recorded measure of birth size and has received the greatest attention, although it is one of many measures of fetal growth.

◊ Maternal genetic factors account for about 25% of variance in birth weight
◊ The father’s genetic contribution to the variance is only about 1 to 2%.
◊ The proportions of variance in birth weight accounted for by some maternal characteristics and environmental
factors are: maternal age, education, previous delivery of low birthweight infants, 8% ;
◊ her height and prepregnant weight explain another 8%.
◊ Coffee and alcohol consumption have a negative influence on fetal growth and contribute about 2%.
◊ Cigarette smoking has a major deleterious influence and accounts for 3 to 6%; the effect is related to the number of cigarettes smoked per day.
◊ Weight gain from mid pregnancy to term contributes about 3%, and total weight gain during pregnancy contributes about 3 to 6% of the variance in birth weight.

Pattern of Fetal growth

Normal fetal growth is characterized by sequential cellular hyperplasia, followed by hyperplasia plus hypertrophy and lastly by hypertrophy alone thus the fetal growth follows a sigmoid curve (Fig.2.10). Studies of embryology that growth of the fetus can be divided into three periods. The first is the period of blastogenesis in which the fertilized
Fig. 2.10: Stages of Fetal Development

Source: S.R. Williams (1989)
egg cleaves into cells that fold in on one another. These evelove into an inner cell mass, which gives rise to the embryo and an outer coat, the trophoblast, which becomes the placenta. The process of blastogenesis is complete at about two weeks after fertilization.

The second period is the embryonic stage, the critical time when cells diferetiate into three germinal layers. The ectoderm gives rise to the brain, nervous system, hair and skin. The mesoderm produces all the voluntary muscles, bones and components of the cardiovascular and excretory systems. The endoderm differentiates to form the digestive system, respirator system and glandular organs of the body. By 60 days’ gestation all of the major features of the human infant have been achieved.

The fetal stage is the period of most rapid growth. From the third month until term fetal weight increases nearly 500 fold from about six grams (0.2 oz) to 3000g to 3500g (6.5 to 7.6 lb) at birth.
Data obtained from study of induced abortions and spontaneous deliveries indicate that the rate of fetal growth increases from 5g/day at 14-15 weeks of gestation to 10g/day at 20 weeks, and to 30-35 g/day at 32-34 weeks. Fig. 2.11 shows the average weight curve from 10 weeks until term. Thus, the substrate needs of the fetus are relatively small in the first half of pregnancy after which the rate of weight gain rises precipitously. The mean rate of cell replication in the placenta peaks at approximately 230-285g/week at 32/34 weeks of gestation, after which it decreases, reaching zero weight gain, or even weight loss, at 41-42 weeks of gestation (Williamset al., 1982; Alexander et al., 1996). However, if growth rate is expressed as the percentage of increase in weight over the previous week, the maximum percentage of increase occurs in the first trimester and steadily decreases thereafter.
Fetal Malnutrition

At a biological level fetal growth depends upon two components; genomic potential and substrate supply. The genomic potential is derived from both the parents, but can be modified by a number of factors. An adequate substrate supply is essential regardless of the genetic mechanism controlling growth. This supply depends on the placenta, uterus and placental vascular integrity.

The proper balance of quantities of micronutrients for optimal human fetal growth is not known, nor has the contribution of human maternal nutritional status to fetal outcome been determined. Some alterations in the history of dietary intakes of protein, calories and other nutrients have often, but not always been associated with size of the infant at birth. Plasma levels of the trace metals zinc, copper and iron and of various amino acids also have been associated with delivery of small-for-dates, intrauterine growth retarded or fetally malnourished infants.
Human fetal malnutrition may be associated with a decreased number of brain cells as well as impaired mental and physical postnatal development. The incidence of fetal malnutrition is variously estimated to range from 3-10% of all live births in developed societies and 15-20% in less development countries.

Intra Uterine Growth Retardation (IUGR)

Intrauterine Growth Retardation (IUGR) is associated with high pernatal mortality and morbidity as the fetus with IUGR tolerates labour poorly. Intrauterine Growth Retardation (IUGR) is defined as subnormal body weight or mass. Neonates weighing below the 10th percentile for their gestational age or two SD below the mean for the gestation is considered growth retarded.

Accordingly, IUGR can be classified into either intrinsic or symmetrical (Type I – 20%) and asymmetrical or extrinsic (type II – 80%)
Causes of IUGR

Intrinsic IUGR arises from conditions within the fetal compartment itself (Table 2.18). Intrinsic IUGR typically results in a symmetrically small fetus in which the head, abdomen and extremities are all equally affected. These fetuses grow slowly because their growth potential has been permanently affected usually by a severe insult in the first trimester and tend to grow in a low percentile growth lane and stay in it. Medical interventions to improve fetal growth usually have little effect, because the underlying cause is rarely correctable.

Extrinsic IUGR results from restriction in nutrients supply, utero placental insufficiency (Table 2.19). The pattern of growth in extrinsic IUGR is similar to the observed in infants undergoing chronic starvation. Abdominal growth slows, weight gain plateaus or stops, but head and extremity growth are spared. The resulting asymmetry of head and body yield abnormal head abdominal and femur to abdominal measurements on serial sonographic
examination. If allowed to continue, growth disorders of this type lead to progressive intrauterine dehydration, decreased amniotic fluid volume, chorionic hypoxia and fetal death or perinatal asphyxia. It is important to identify

**Table 2.18: Intrinsic Causes of IUGR**

<table>
<thead>
<tr>
<th>Causes</th>
<th>Associated Maternal Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constitutional</td>
<td>small maternal stature</td>
</tr>
<tr>
<td>Genetic</td>
<td>trisomy 18, trisomy 13&lt;sup&gt;5&lt;/sup&gt;</td>
</tr>
<tr>
<td>Toxic</td>
<td>alcohol, nicotine, virus, syphilis</td>
</tr>
<tr>
<td>Teraogenic</td>
<td>radiation, drugs</td>
</tr>
</tbody>
</table>

**Table 2.19: Extrinsic Causes of IUGR**

- Decreased maternal nutritional delivery
- Maternal cardiac diseases
- High altitude severe anemia
- Maternal starvation
**Placental Vasculopathy**
Maternal Hypertension
Autoimmune diseases – lupus erythematosus
Antiphospholipid syndrome
Diabetes mellitus
Placental infections – syphilis, malaria

**In utero constraint**
Mullerian anomalies – uterus: bicornuate, didelphys
Extrinsic mass – uterine fibroid
Multifetal gestation

IUGR antenatally to counsel parents regarding possible causes, to monitor fetal growth and well being.

Small-for-dates infants have congenital anomalies eight times more frequently than normally grown infants.

**Etiology of Intrauterine Growth Restriction**
There are a multiple causes for intra uterine growth restriction:

1. Genetic Factors
2. Congenital Anomalies
3. Infection
4. Multiple gestation
5. Inadequate maternal nutrition

Of the aforesaid factors of intra uterine growth restriction, inadequate maternal nutrition is being discussed in the following section.

**Inadequate Maternal Nutrition**

Numerous animal studies have demonstrated that under nutrition of the mother owing to protein or calorie restriction can adversely affect fetal growth (Dobbing, 1970; Brasel and Winick, 1972). Information from experiments using small animals, in which the fetomaternal mass is much greater than in human pregnancy and the fetal and neonatal growth rate reaches its maximum after birth, must be extrapolated with caution. Such animal studies, however, have engendered important concepts.

Winick (1971) reported three phases of fetal growth: cellular hyperplasia, followed by both hyperplasia and hypertrophy, and then predominantly hypertrophy. Thus, if there is a decrease in available substrate, the timing of the
decrease in available substrate, is reflected in the type of IUGR observed. If the insult occurs early in pregnancy, the fetus is likely to be born with a decrease in cell number and cell size, such as might be observed with severe chronic material under nutrition or an inability to increase utero placental blood flow during gestation, and have symmetric IUGR. However, if the insult occurs late in gestation, such as with twin gestation, the fetus is likely to have a normal cell number but a restriction of cell size, which can be returned to normal with adequate postnatal nutrition, and have asymmetric IUGR.

The importance of maternal nutrition in fetal growth and birth weight has been demonstrated by studies in Russia and Holland, where women suffered inadequate nutrition during World War II. The population in Leningrad underwent a prolonged period of poor nutrition, during which both preconceptual nutritional status and gestational nutrition were poor and birth weights were reduced by 400 to 600g (Antonov, 1947). In Holland, a 6-month famine created conditions that permitted evaluation of the effect of malnutrition during each of the trimesters of pregnancy in a group of women previously well nourished (Stein and
Susser, 1975). Birth weights declined approximately 10%, and placental weights 15%, only when under-nutrition occurred in the third trimester with caloric intake below 1500 g. The difference in severity of the IUGR in these two populations suggests the importance of pre-pregnancy nutritional status, an idea that has been substantiated (Love and Kinch, 1965; Kramer, 1987; Abrams and Newman, 1991). More recent studies show that inadequate weight gain in pregnancy (defined as < 0.27 kg/week, or <10Kg at 40 weeks, or based on suggested weight gain for body mass indices) is associated with an increased risk of IUGR. Weight gain in the second trimester appears to be particularly important (Abrams and Selvin, 1995).

It is still unclear whether generalized calorie intake reduction or specific substrate limitation, such as protein or key mineral restriction or both is important in producing IUGR. Decreases in zinc content of peripheral blood leukocytes correlate positively with IUGR (Meadows, et.al., 1981; Wells et al., 1987), and serum zinc concentrations of less than 60μg/dl in the third trimester are associated with a fivefold increase in low-birth-weight newborns (Neggers et
Similarly, an association between low serum folate levels and IUGR has been reported (Goldenberg et al., 1992a). Although there is no convincing evidence that high protein supplementation is beneficial (Stein et al., 1978; Rush et al., 1980), caloric supplementation can improve birth weights by 50-225g, the largest increase being demonstrated when the net energy increment exceeds 430 kcal/day in an otherwise poorly nourished population (Prentice et al., 1983). However, Warshaw (1985) pointed out that, in a fetus receiving decreased oxygen delivery as a result of decreased uteroplacental perfusion and who has adapted by slowing metabolism and growth, it may not be advisable to increase substrate delivery.

As maternal body mass and plasma volume are correlated, reduced plasma volume or prevention of plasma volume expansion may lead to decreased cardiac output and uterine perfusion and a resultant decrease in fetal growth (Daniel et al., 1989; Rosso et al., 1992; Duvekot et al., 1995).
Another maternal nutrient that may be important to fetal growth is oxygen. The median birth weight of infants of women living more than 10000 feet above sea level is approximately 250g less than that of infants of women living at sea level (Lichty et al., 1957). Pregnancies complicated by maternal cyanotic heart disease usually result in IUGR, but it is unclear whether abnormal maternal hemodynamics or the reduction in oxygen saturation, by approximately 40% in the umbilical vein may account for poor fetal growth (Novy et al., 1968). The association between hemoglobinopathies and IUGR may be due to a decrease in either blood viscosity or fetal oxygenation (Pritchard et al., 1973). Patients with chronic pulmonary disease, such as poorly controlled asthma, cystic fibrosis, or bronchiectasis, or those with severe kyphoscoliosis may be at increased risk (Kopekhager, 1977; Palmer et al., 1983; Thaler et al., 1986).

**Incidence of Intrauterine Growth Restriction (IUGR)**

The incidence of IUGR varies according to the population under examination, The geographic location, and the standard growth curves used as references. In general, approximately one third of all infants weighing less than
2500g at birth have sustained IUGR, and approximately 4-8% of all infants born in developed countries are classified as growth-restricted (Gruenwald, 1963; scott and usher, 1966; Lugo and cassady, 1971; Galbraith et al., 1979; Kramer, 1987).

**Diagnosis of Intra Uterine Growth Restriction (IUGR)**

The diagnosis of IUGR is in part dependent on an accurate evaluation of gestational age. The last menstrual period is a reliable index of gestational age if the mother is seen early in gestation, unless there is a history of irregular menstrual cycles or conception occurred soon after discontinuation of oral contraceptives. Because IUGR, particularly asymmetric IUGR, is rarely detected clinically before 22 to 24 weeks, uterine size should equate with gestational age up to that time (Murphy, 1969). Ultrasonographic evaluation is particularly useful in dating pregnancies if it performed before biological variation begins to have a significant impact (i.e., before 22 weeks of gestation).
History and Physical Examination

Clinical diagnosis of IUGR by physical examination alone is inaccurate, and frequently the diagnosis is not made until after delivery. Most clinical studies demonstrate that using physical examination alone, the diagnosis of IUGR is missed or incorrectly made almost half the time (Campbell and Thomas, 1977; Cnattingius et al., 1984).

Techniques such as tape measurement of the uterine fundus are helpful in documenting continued growth if performed repeatedly by the same observer but are not sensitive enough for accurate detection of most infants with IUGR (Beazley and Underhill, 1970).

Ultrasonography

Currently, ultrasonographic evaluation of the fetus is the preferred and accepted modality for the diagnosis of inadequate fetal growth. It offers the advantages of reasonably precise estimations of fetal weight, determination of interval fetal growth, and measurement of several fetal dimensions to describe the pattern of growth.
abnormality. Use of these ultrasound measurements requires accurate knowledge of gestational age.

• **Estimation of Gestational Age**

Diagnosis of IUGR relies on accurate assessment of gestational age. Routine use of first trimester ultrasound determines the crown rump length, a measurement to accurately predict the gestational age.

• **The Estimation of Fetal Weight**

The sensitivity is considerably lower than abdominal circumference percentile. The positive predictive value of a fetal weight estimate below the tenth percentile is greater. A fetus is labeled as IUGR when both the abdominal circumference and estimated fetal weight percentiles are below their respective norms.

• **Abdominal Circumference [AC]**

Is the single measurement which best reflect fetal nutrition of any growth parameter. The abdominal circumference
percentile has both the highest sensitivity and greatest negative predictive value for the sonographic diagnosis of IUGR. Additional growth studies are performed at 2-3 weeks interval. A closer interval is of no benefit for the evaluation of growth and may be misleading. Abdominal Circumference increase by less than 1cm/2 weeks or a fall of > 1.5 SD in the predictive of IUGR.

• Transverse cerebellar diameter [TCD]

Reece et al., found that the growth of the transverse cerebellar diameter was unaffected by mild to moderate intrauterine insufficiency / dysfunction. Campbell et al. calculated a ratio between the two (TCD/AC ratio) and found that this ratio was gestational age independent and suggested that is may be a method for screening of fetal growth abnormalities.

• Biparietal Diameter [BPD]

Though easy to measure is far from an ideal parameter for the detection of IUGR because the fall in the cranial growth because of insufficient nutrition is late in the process and
the shape of the cranium is readily altered by external forces

- **Head circumference [HC]**

  The HC is not subject to the extreme variability of the BPD and hence more reliable. Pressman et al. (2000) conclude that a single sonogram obtained between 34 and 36.9 weeks’ gestation provided comparable and marginally more accurate prediction of birth weight for all pregnancies, including those of suspected growth abnormality, than sonograms obtained closer to term. They recommend that the strategy of a single sonogram obtained between 34 and 37 weeks’ gestation replace serial sonography or sonograms taken in late pregnancy.

**Gestational Age**

Data from numerous studies in India indicate that there are two main reasons for the high prevalence of LBW. First is
the relatively high pre-term birth rate, ranging from 10-14 percent in different studies. Even among full-term neonates, a significant number are born at 38 and 39 weeks. The entire frequency of distribution of births in relation to gestational age appears to be shifted to the left by about a week. The reasons for this phenomenon are not clear. However, improvement in antenatal care may thus result in the curve shifting to the right and consequent improvement in birth weight (Prema R, 1993)

A significant number of pregnant women deliver their babies in the 37-38th week as against the 39th – 40th week. So though, the deliveries are ‘full term’ the gestation period in many cases is shorter by at least two to three weeks. Preliminary studies carried by the Nutrition Foundation of India, have shown that the incidence of low birth weight deliveries is as high as 33 percent in the deliveries taking place in the 37th week as against 12 percent in cases of babies born in the 40th week. However, the reasons for the shortening of the gestational period are not clear.

The first and foremost common reason to assess gestational age is simply to verify the term status for that
infant after a routine gestation with known obstetric dates. This verification of maturity can provide some assurance that there is decreased risk for problems that are more likely to develop with either pre-maturity or post-maturity. A second reason for establishing a gestational age exists if there has been no prenatal care, there is discrepancy in weight for presumed gestational age, or the dates and the screening examination do not agree. A third need for assessing maturation is to follow progression of the development of an infant as part of routine or high-risk follow-up care.

Assessment of gestational age includes assigning a group designation based on obstetric dates of term, perterm or postterm. Obstetric dating is based on weeks since the last menstrual period with an average cycle length of 28 days. A term infant is any infant whose birth occurs from the beginning of the first day of week 37 through the end of the last day of week 42 after the onset of the last menstrual period (i.e., 260-294 days’ gestation). A reterm infant is one born before the end of the last day of the 37th week (i.e., before 259 days) and a post term infant is one whose birth occurs from the beginning of the first day of the 43rd week
(i.e., after 294 days). Classifying infants born at term, preterm or postterm helps to establish the level of risk for neonatal morbidity and long-term developmental problems (Fig 2.12).

There have been a variety of methods devised for assessing gestational age in the newly born infant. These systems range in complexity from simple visualization of as few as four physical findings to a more highly technical and cumbersome assessment of nerve conduction velocities.

The scoring systems that have been studied and reported the most extensively are those devised by Dunowitz and Ballard. Compared with reliable ultrasound dates, the earlier Ballard system tended to overestimate the age of premature infants and underestimate the age of postterm infants. It was particularly inaccurate in very low birth weight infants with a deviation of over two weeks. However great the inaccuracies, many studies on developmental outcome were based on either the Dubowitz or original Ballard maturational assessments.
Fig.2.12: Definition of gestational age.

Measurement of the biparietal diameter, the head and abdominal circumferences, and femur length allows the clinician to use accepted formulas to estimate fetal weight and to determine whether a fetal growth aberration represents an asymmetric, symmetric, or mixed pattern (Hadlock et al., 1984a 1994b). Intrinsic fetal insults occurring early in pregnancy such as infection, exposure to certain drugs or other chemical agents and chromosomal abnormalities and other congenital malformations are likely to affect fetal growth at a time of development when cell division is the predominant mechanism of growth. Consequently, musculoskeletal dimensions and organ size may be adversely affected, and symmetric growth restriction is observed. Given this set of circumstances,
one might expect to find that femur length and head circumference are low for a given gestational age, as are abdominal circumference and overall fetal weight, characterized as symmetric IUGR.

At the other end of the spectrum, an extrinsic insult occurring later in pregnancy, usually characterized by inadequate fetal nutrition, is more likely to result in asymmetric growth restriction. In this type, femur length and head circumference are spared but abdominal circumference is decreased because of subnormal liver growth and there is a paucity of subcutaneous fat. The most common disorders that limit fetal substrates for metabolism are the hypertensive complications of pregnancy, which are associated with decreased uteroplacental perfusion, and placental infarcts, which limit trophoblastic surface area for substrate transfer. In fact, a falloff in the interval growth of the abdominal circumference is one of the earliest findings in extrinsic or asymmetric IUGR; conversely, the finding of an abdominal circumference in the normal range for gestational age markedly decreases the likelihood.
Different standards for fetal growth throughout gestation have been reported. These standards set the normal range, on the basis of statistical considerations, between two standard deviations of the mean (2.5\textsuperscript{th}-97.5\textsuperscript{th} percentile) or between the 10\textsuperscript{th} and 90\textsuperscript{th} percentiles for fixed gestational ages. The standards most widely used in the 1960s and 1970s where those developed in Denver, Colorado (Lubehenco et al., 1963; battagli and lubchenco, 1967).

**Neonatal Assessment**

For a normal neonate, standard measurements for the assessment of nutritional status include weight, length and head circumference in addition to skin-fold thickness or mid-arm circumference or calculation of ponderal index. This study used the measurements of birth weight, length, head circumference, chest circumference and ponderal index.
Birth Weight

LBW may indicate that the baby did not remain in utero long enough [duration of gestation] or it did not develop well enough [Intra Uterine Growth Retardation (IUGR)]. Two types of LBWs have been identified: 1. Term low birth weights, i.e., born between 37-42 weeks of gestation, but their size may be small for the corresponding gestational age (SGA) and 2. Preterm low birth weights i.e., born before 36 completed weeks of gestation, with appropriate size for corresponding gestational age.

The world health organization (WHO) (1969) classifies all newborn weighing less than 2500g as having low birth weight. Many low birth-weight infants are born near term, having sustained intrauterine growth restriction (IUGR). Low birth weight infants have traditionally been placed in one of the following three classifications (battaglai , 1970; yerushalmy , 1970):

1. Preterm neonate: new born delivered before 37 completed weeks of gestation but are appropriate for gestational age (AGA).
2. Preterm and growth-restricted neonate: new born delivered before 37 completed weeks of gestation who are small for gestational age (SGA).

3. New borns delivered before 37 completed weeks of gestation who are small for gestational age (SGA): term growth-restricted neonate.

Therefore irrespective of the gestational age at birth the neonates are generally categorized by their birth weight as Low Birth Weight (LBW < 2500g), Very Low Birth Weight (VLBW < 1500g), Extremely Low Birth Weight (ELBW < 1000g) and micropremie (<750g) [Reiter et al., 2000]. WHO considers all neonates <2500g to be low birth weights.

The reliance on only gestational age and birth weight neglects the issue of body size and length and the clinical observations that there are two main clinical types of IUGR infants:
1. The infant who is of normal length for gestational age but whose weight is below normal (asymmetrically small)

2. The infant whose length and weight are below normal (symmetrically small)

**Crown Heel Length**

Of all neonatal measurements, crown-heel length is the most subject to variability because it depends on achieving full extension of an infant who is more comfortable in flexion. Measuring the crown-heel length establishes leg and trunk proportionality.

When anomalies of the lower extremities make measuring crown-heel length impossible or meaningless, the crown-rump measurement may still be feasible. The ratio of crown-rump length to total length is relatively consistent throughout gestation.

**Head Circumference**

Measurement of head circumference is important because it is closely related to brain size. Also an indirect way of measuring the growth in utero as well as after birth (Cooke
et.al., 1977; Sankaran et.al.,1983). Maternal nutrition has been shown to influence the function of the circumference in children at a later age (Evans et.al., 1976)

The head circumference is the largest dimension around the head obtained with a tape placed snugly above the ears, the occipitofrontal circumference (OFC). Minor changes in head circumference occur after birth as scalp edema and molding resolve. If there is marked molding due to breech positions, the OFC may be as much as 2cm higher than after molding resolves. Determining the head volume may be more helpful when there are extremes in head shape that affect the OFC. The head circumference may change significantly in the first days after premature birth, particularly when there is relatively large weight loss.

The OFC predictably falls on the same quartile, the cause should be sought because head size in part reflects brain growth. The most frequent reason for a head percentile to exceed that of length is familial. A decreased rate of growth manifested by a flat curve or by decreasing to a lower percentile may indicate poor brain growth. The head circumference exceeds the abdominal circumference until
32 weeks. Between 32 and 36 weeks, the two circumferences are equivalent; after 36 weeks, the abdominal circumference normally is greater. Clearly the abdominal circumference in a neonate is not a stable parameter, being greatly influenced by the quantity of air and feed in the bowel, but in the fetus it is relatively more constant and allows a more consistent base for comparison with biparietal diameter and therefore, head circumference.

**Chest Circumference**

Since birth-weights represent an important method for screening neonates for possible risks, attempts are underway in many countries, including India, to provide facilities for recording birth-weights even in home deliveries. In the pursuit of identifying further nutritional status markers for neonatal nutritional status, several studies were carried out at National Institute of Nutrition (NIN 1984-1995), Indian Council of Medical Research (ICMR) (1990) and WHO (1987). Data from all these studies indicate that the chest circumference is the most suitable parameter for use in identifying the neonates ate
risk. In India, 29cms may be the most appropriate cut-off point for chest circumference.

**Ponderal Index**

Barker et.al., [1994] suggested that ponderal Index is important, so as to distinguish 'growth-restricted' infants from normal constitutively small' with the majority of risk being associated with the growth-restricted group and adult disease.

Infants with a ponderal index of less than the 10<sup>th</sup> percentile for gestational age or a crown-rump length less than the 3<sup>rd</sup> percentile are defined as growth-restricted. This index in term infants is not affected significantly by differences in race or sex. This disadvantage of this index is the potential error introduced by cubing the length. It is clear whether asymmetric IUGR and symmetric IUGR are two distinct entities or are merely reflections of the severity of the growth restriction process (excluding chromosomal aberrations and infections disease).
Neonatal Biochemical Assessment

The value of routine laboratory evaluations on all patients admitted to the hospitals is a subject of debate. Previous recommendation for new borns had included blood type and direct coombs test, haemoglobin and hematocrit, glucose or rapid blood glucose screen, urinalysis. There are no laboratory tests that have been established sufficiently as valuable to be used as a routine procedure in all newborns, [Pitkin, 1981] There is little justification for routine haemoglobin and hematocrit determinants on a single infant with no evidence of anaemia or polycythemia by history or physical examination [Oski, F.A. 1987] & [Oh, W 1986] and hence this study did not attempt creating/insisting on an academic basis, the laboratory investigations of newborns.

Birth Weight Prediction

There is a clinical and nutritional utility in having an accurate method of predicting birth weight in advance of labor. Estimation of fetal birth weight by symphysiofundal height measurement has been reported by various authors including Dare et.al., (1990) Bergstrom and Liljestrand
Labrecge and Boulianne (1987) conducted a study to evaluate the measurement of fundal height in labour as a mean of estimating birth weight in singleton pregnancies. As a diagnostic test they found fundal height useful on an individual basis and recommended that for a mass screening utilization this procedure would have to be integrated to a complete program of maternal and child care.

Several statistical techniques have been recommended to quantify the contribution of these non-nutritional factors to birthweight of infants delivered either prematurely (preterm) or at term, and to predict the outcome of pregnancy. Analysis of variance, multiple regression analysis, linear additive models, and risk factor analysis are most frequently used. Some of the methods were derived from retrospective analyses of large data bases collected many years earlier, e.g., British Perinatal Mortality Survey 1958; Collaborative Perinatal Study, U.S., 1959 to 1966. An effective method of predicting outcome should be sensitive, specific and applicable early enough in pregnancy to permit a suitable intervention, if indicated, and to prevent or modify a predicted adverse outcome.
Serial measurement of fundamental height and a symphysis-fundus (S-F) height graph are being increasingly used for the prediction of birth weight and diagnosis of intra uterine growth retardation. Walrakes et.al., (1995) had tried to develop charts on fundamental height and abdominal girth in relation to gestational age to predict pregnancy outcomes in terms of birth weight.

One recent approach to such birth weight prediction is based upon a quantitative assessment of measurable maternal and pregnancy-specific height, weight, obesity, parity and age in addition to pregnancy specific factors, such fetal sex and length of gestation. Nahun et.al..,(1999) found and reported that term birth weight could be accurately predicted as a function of only six prospectively measurable variables: gestational age, maternal height, maternal weight, third-trimester maternal weight gain rate, parity and fetal sex.

The birth weight prediction equation derived from these six variables could accurately estimate term birth weight to within an average of ± 7.6% of actual birth weight (± 267g).
Maternal anthropometric indicators such as weight gain during pregnancy, upper mid arm circumference useful for screening women at nutritional risks as well as predicting unfavorable pregnancy outcome (mercypaul 2002). Using multiple regression analysis, it was shown that the anthropometric parameters could predict 25% of mothers at risk of giving birth to LBW infants(Raman L, 1989).

Nahum, (2002) used a predictive formula to estimate birth weights for 244 babies using the variables gestational age, maternal height, maternal weight, third trimester weight gain rate, number of prior children and fetal gender. On average, the predictions were accurate to within eight percent. By contrast, ultrasound predictions vary from eight percent to 16 percent. The problem with ultrasound is that it estimates a baby’s birth weight based on only a few measurements, such as the circumference of head or length of femur, while a number of formulas are used to translate the measurements into birth-weight estimates.