CHAPTER 2

REVIEW OF LITERATURE

Nutrition is the key to achieve Millennium Development Goals (MDG’s) (Ann, 2007). The reduction of Low Birth Weight (LBW) forms an important contributory factor to the MDG for reducing child mortality (UNICEF, 2004a). The goal of reducing low birth weight incidence by at least one third between 2000 and 2010 is one of the major goals in, ‘A World Fit for Children’, the declaration and plan of action adopted by United Nations General Assembly Special Session on Children in 2002 (UNICEF, 2004b). One underweight and undernourished child is an individual tragedy. But multiplied by tens of millions, under nutrition becomes a global threat to societies, to economies and generations to come (Ann, 2007). According to Tay (2000) low birth weight infants are at increased risk for growth abnormalities, developmental delays and chronic illness. Hence the present study was conducted to assess the nutritional status of preschool children with low birth weight. In Kerala 11.91 per cent of total population are children under 6 years of age (NRHM, 2008).
A review of literature relating to the present study is given under the following heads:

2.1. Definitions

2.2. Epidemiology of LBW

2.3. Determinants of LBW

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2.4.1. Morbidity and mortality consequences of LBW in neonates and infants

2.4.2. LBW and growth in children

2.4.3. LBW and Immunity

2.4.4. Long term consequences of LBW

2.5. Interventions to prevent LBW and

2.6. Under nutrition in preschool children

2.1. Definitions

**Birth weight** is the first weight of the foetus or newborn obtained after birth, which should be preferably measured within the first hour of life, before significant post natal weight loss has occurred. (WHO, 1992; UNICEF/WHO, 2000).

**Low birth weight** is defined as weight at birth, less than 2500 g (up to and including 2499 g) irrespective of gestational age (WHO, 1976).

Arnold and David (1995) defined low birth weight infants as infants born weighing significantly less than normal who need special formula preparations to meet their nutritional requirements.
SCN (2000) used low birth weight as a proxy for quantifying the magnitude of Intra Uterine Growth Retardation in developing countries because valid assessment of gestational age is generally not available.

Cater and Gill (1994) statistically defined low birth weight, based on an assumption, to which, the distribution of body weight, as a mixture of two components, viz., the predominant component which is normally distributed and included 95 percent of new born and the residual component which is mainly composed of new born under extreme risk for prenatal mortality.

The incidence of low birth weight in a population is defined as the percentage of live births that weigh less than 2500 g. out of the total live births during the same time period.

The low birth weight incidence rate is therefore calculated using the following formula:

\[
\text{Number of live born babies with birth weight } < 2500 \text{ g} \times 100 \\
\text{Number of live births} 
\]

(UNICEF, 2004a)

The terms premature or preterm are used for infants born before 37 weeks gestation (SCN, 2000). Intra Uterine Growth Retardation is a subtype of low birth weight of extra ordinary importance to developing countries. According to de Onis et. al. (1998) a small size of gestational age indicates IUGR or inability of the foetus to reach its growth potential and infants diagnosed with IUGR may be low birth weight at term (≥ 37 weeks gestation and <2500 g) or preterm (<37 weeks gestation and weight less than 10th percentile) or IUGR at 37 weeks gestation and weight less than the 10th percentile with a birth weight ≥ 2500 g.
WHO (1997) defined Small for Gestational Age (SGA) infants as infants with birth weights below a given low percentile cut-off for gestational age. Ashworth (1996) subdivided IUGR infants according to whether they are stunted or wasted or both. Stunted infants have been chronically under nourished *in utero*, resulting in a proportionate reduction in both skeletal and soft tissue growth (Villar *et. al.*, 1989). Wasted infants have linear growth but reduced tissue mass resulting in low ponderal growth, reflecting later onset, primarily in third trimester (Villar and Belizan, 1982 a).

WHO (2002) classified IUGR infants into asymmetric and symmetric. Infants who are less than 10\(^{th}\) percentile for both weight and Ponderal Index (PI) are classified as, ‘asymmetric’ and infants less than 10\(^{th}\) percentile for weight but not Ponderal Index are classified as, ‘symmetric’ IUGR.

### 2.2. Epidemiology of Low Birth Weight (LBW)

According to ACC/SCN (2000) about 30 million infants were born each year in developing countries with low birth weight, representing about 24 percent of all newborns in different countries.

UNICEF (2004b) observed that about 16 percent of children born in the world, 18 percent born in least developed world, 17 percent in developing countries, seven percent in industrialized countries and 30 percent born in South Asia were with low birth weight.

According to NFHS (2007) about 30 percent of children born in India were with low birth weight.
More than 95 percent of low birth weight babies are one born in developing countries (UNICEF 2004a). Kavita (2005) opined that low birth weight is a widespread problem that has seen little improvement in recent decades in developing countries. India alone accounts for 40 percent of low birth weight births in the developing world and more than half of those in Asia (UNICEF, 2004b).

The large proportion of infants not weighed at birth constitutes a significant impediment to reliable monitoring of low birth weight (UNICEF, 2004a). According to WHO (2004b) 58 percent of babies in the developing world are born with a skilled attendant at delivery but only 42 percent are weighed. However, declines in the percentage of births that are not weighed were evident in Asia (excluding China) and appear to be largely driven by improvements in India and Indonesia (WHO, 2004b). UNICEF / WHO (2000) estimated that about 71 percent of births were not weighed in India.

According to SCN (1999), for infants born with low birth weight at the end of a term pregnancy Intra Uterine Growth Retardation has occurred. About 24 percent of new borns in developing countries have IUGR (SCN, 1999). Arifeen (1997) observed that in developing countries IUGR affects about two-thirds of infants born with low birth weight, the remaining one-third of low birth weight infants are born preterm, some of whom are also affected with IUGR.
2.3. **Determinants of LBW**

Identifying the determinants of low birth weight is important because of the health risks associated with low birth weight. Infants born with low birth weight are more likely to die during the neonatal period (Oechsli, 1990) and during the first year of life (Mc Cornick, 1985). Low birth weight is most common in developing countries, where the burden of malnutrition and of infectious diseases is heavy (Michele *et. al.*, 2001).

WHO (1995) identified that many factors affect the duration of gestation and of foetal growth and thus birth weight. These factors are related to the infant, the mother or the physical environment and play an important role in determining the infant’s birth weight and future health (UNICEF, 2004a). Beaten *et. al.* (1990) were of the opinion that at birth, infant weight and length are determined by maternal factors – including nutrition – and gestational age i.e., whether the infant is full term and interpretation of birth weight must take those factors into account.

Prematurity and IUGR are the two main causes of low birth weight (UNICEF, 2004b). According to Villar and Belizan (1982 b), the majority of low birth weight in developing countries is due to IUGR, while most low birth weight in industrialized countries is due to preterm birth.

According to ACC/SCN (2000) the basic causes of the incidence of low birth weight relate to the care of women, access to and quality health services,
environmental hygiene and sanitation, household food security, educational status and poverty. Logerstrom et. al. (1994) opined that malnutrition in women leads to low birth weight, inability to sustain work and reduced capacity to care for the family.

Environmental factors which have an effect on birth weight include seasonal changes, antenatal infections and the general level of socio-economic development (FAO, 1988).

WHO (1995) found that maternal nutritional status before and during pregnancy is an important predictor of poor pregnancy outcomes. Cartetbon et. al. (1999) found that last prenatal weight measurement was significantly associated with higher risk of low birth weight independent of maternal morbidity status. Andersson and Bergstrom (1997) found that maternal pre pregnancy weight, representing a woman’s long term nutritional status, was the most important determinant of birth weight than weight gain during pregnancy, representing a woman’s short term nutritional status.

Cameron and Hofvander (1983) were of the opinion that the causes of prematurity may include high maternal blood pressure, acute infections, hard physical work, multiple births, stress, anxiety and other psychological factors. Kramer (1998) found that in developing countries, the major determinants of growth retardation in utero and low birth weight are inadequate maternal nutritional status before conception, short maternal stature and poor maternal nutrition during
pregnancy, indicated by low gestational weight gain due to inadequate dietary intake.

Prada and Tsang (1998) opined that low pregnancy weight gain may account for more than 14 percent of births with low birth weight of growth retardation in utero and low maternal height accounts for about 18.5 percent. Prevalence rates were high where pre-pregnancy weight for height is low (Miller and Merrit, 1979).

Intrauterine growth retardation and hence low birth weight may also be due to young maternal age, malaria during pregnancy, gastro-intestinal, intestinal parasitosis and / or other infections and cigarette smoking, which are more prevalent in developing countries (Kramer, 1998). Kramer (1987) found that above illnesses and habits were associated with impaired foetal growth. According to Mc Gregor et. al. (1983) infants born to women with placental malaria have a mean deficit in birth weight of about 170 g.

de Onis et. al. (1998) established etiological roles of pre eclampsia, short stature, genetic factors, alcohol and drug use during pregnancy to incidence of low birth weight. Das et. al. (1998) observed that about 54.40 per cent of infants born to women with pre eclampsia were low in birth weight.

The effects of cigarette smoking are becoming a significant factor determining the incidence of low birth weight in some developing countries (Kramer, 1998). Smoking reduces birth weight by 100 to 400 g and increase the risk of incidence of low birth weight baby by about 2.5 times.
Ann and Feachem (1985) found that, a short birth interval of < 12 months is associated with an increased risk of low birth weight.

Michele et. al. (2001) found that infections during pregnancy were significantly associated with lower birth weight and SGA among primiparas and multiparas. It was also found that in utero transmission of HIV may lead to fetal growth retardation (Philip et. al. 2000). According to Steketee et. al. (1996) placental malaria and maternal HIV infection were associated with higher risk of low birth weight and parasite density and infection of the placenta have a negative effect on foetal growth.

2.4. Consequences of LBW

According to ACC/SCN (2000), low birth weight infants suffered intrauterine growth retardation as foetuses are born undernourished with high risk of morbidity and mortality, growth retardation, low immunity and with increased risk of various adult chronic diseases.

2.4.1. Morbidity and mortality consequences of LBW in neonates and infants

Low birth weight is generally associated with increased morbidity and mortality (Bukenya et. al., 1991). Ashworth (1998) found that for preterm infants weighing 2000 – 2500 g at birth, the risk of neonatal death is four times higher than for infants weighting 2500 – 3000 g, and ten times higher than for infants weighting 3000 – 3500 g. Ashworth and Feachem (1985) opined that low birth weight infants during the post neonatal period (> 28 days of age) also have high mortality rates. It was also found that the risk, of low birth weight infants in post neonatal period,
were greater than those for low birth weight infants during the neonatal period (Ashworth and Feachem, 1985). According to Ashworth (1996), term low birth weight infants have increased risks of neonatal and post neonatal death compared with infants of Adequate Birth Weight (ABW) and also reported that term low birth weight infants have risks of neonatal and post neonatal death that are of a broadly similar magnitude to those of preterm infants of the same birth weight.

James et. al. (2001) observed that such mortality rates were greater for male infants than for females.

Low birth weight accounted for 69 percent of Acute Lower Respiratory Infections (ALRI) related and preventable under five deaths in India (Dutta et. al., 1987). According to Arifeen (1997) almost half of the infant deaths from pneumonia or ALRI and diarrhea could be prevented if low birth weight were eliminated.

Lemons et. al. (2001) identified the incidence of chronic lung disease, severe intracranial haemorrhage and necrotizing enterocolitis as major morbidities for the low birth weight infants. Tsao et. al. (1998) observed pulmonary hypertension and severe intraventricular haemorrhage as the factors associated with morbidity and mortality for low birth weight neonates.

Low birth weight is associated with increased risk of mortality from respiratory infections and of hospitalization from pneumonia (Victora et. al., 1990; Victora et. al.; 1994 and Lira et. al. (1996) found an association between low birth weight and the prevalence of cough. However Fonseca et. al. (1996) observed that only infants < 2000 g were at significant risk of pneumonia morbidity. According to
Barros *et. al.* (1992), IUGR infants (preterm + term BW < 10th centile local reference population) were 50 per cent more likely to be hospitalized with pneumonia in the first two year of life than ABW term infants which did not persist into the third year.

Low birth weight is an important determinant of diarrhoea, death and of hospitalization from dehydration (Victora *et. al.*, 1992) and diarrhoea (Victora *et. al.*, 1990; Ittiravivongs *et. al.*, 1991). An association between low birth weight and hospitalization from diarrhoea beyond the first year has been reported by Mertens *et. al.* (1987). Barros *et. al.* (1992) observed that IUGR infants (preterm + term < 10th centile local reference population) were at almost twice the risk of being hospitalized for diarrhoea in the first 2 years of life compared with ABW term infants, while appropriately grown preterm infants experienced only slightly increased risk. Lira *et. al.* (1996) found that term LBW infants aged 0-6 months experienced 33 per cent more days of diarrhoea than ABW term infants. However Bukenya *et. al.* (1991) reported that low birth weight infants aged 0-59 months experienced 60 per cent more days with diarrhoea. An increased risk of diarrhoea morbidity has been reported in India by Saha *et. al.* (1983).

Ashworth (1996) found that wasted newborns were more likely to experience asphyxia at birth than stunted newborns. Kramer *et. al.* (1990) and Caulfield *et. al.* (1991) observed higher morbidity in wasted neonates in the early postnatal period, particularly regarding hypoglycaemia, hypothermia, hyperviscosity, perinatal asphyxia and aspiration syndrome. Wasted IUGR infants often have lower mean
birth weights than stunted IUGR infants which will result in the increased risk of morbidity of wasted neonates (Kramer et. al., 1990). Caulfield et. al. (1991) found that IUGR infants (BW <10th centile of reference population) with a low ponderal index had increased risks of asphyxia and hypoglycaemia during their postnatal hospital stay compared with IUGR infants of adequate ponderal index, even after controlling for birth weight and gestational age. Villar et. al., (1990) observed a negative association between higher ponderal index and neonatal morbidity in term IUGR infants and concluded that wasted low birth weight infants appear more prone to morbidity than stunted infants in early postnatal life.

2.4.2 LBW and growth in children

Martorell et. al. (1996) revealed that males and females of IUGR/LBW underwent partial catch up growth during their first two years of life. After the age of 2 years, there was little further catch-up and the IUGR infants remained stunted during the rest of their childhood, adolescence and adult life.

Early intrauterine growth retardation leads to symmetrical growth retardation in infants with reduced growth in length, weight, head and abdominal circumference when compared to infants of same gestational age. However late intrauterine growth retardation results in asymmetrical growth with normal length and head circumference but with low birth weight (Bakketeig, 1998). Martorell et. al. (1998) reported that neonatal mortality rates are reported to be higher among asymmetrical IUGR infants.
Goldenberg *et. al.* (1998) observed that IUGR boys exhibit neurological under development, than girls and children of lower socio-economic circumstances. It was also found that neurological dysfunction is associated with attention deficit disorders, hyperactivity, clumsiness and poor school performance among children with low birth weight (Hack, 1998). Grantham (1998) reported that deficits in cognitive development of IUGR/LBW children began to appear between 1 and 2 years of age. According to Joachim *et. al.* (2004) the problems of being low birth weight do not resolve even after the first year of survival, by school age these children are more likely to have learning disabilities, attention deficit disorder, developmental impairments and breathing problems (Maureen *et. al.*, 1991; Willerms, *et. al.*, 1992).

Low birth weight infants, who are symmetrical and reduced head growth, the risk of incidence of neurological dysfunction was found to be more and Goldenberg *et. al.* (1998) were doubtful about the outcome of interventions directed towards such infants to improve their status. Harvey *et. al.* (1982) opined that under nutrition affects head circumference before 26 weeks of pregnancy has a greater impact on neurologic function than does under nutrition later in pregnancy. Goldenberg *et. al.* (1998) found that preventing asphyxia reduce the prevalence of major and minor handicaps, especially cerebral palsy and mental impairment observed among low birth weight infants with asymmetrical growth. Low birth weight infants are more likely to experience developmental deficits.
Low birth weight (whether caused by IUGR or prematurity) predicted a 6-point reduction in IQ during school age children from developed countries. The impact is probably stronger in poor environments (Aylward et. al., 1989). A reduction in IQ of as much as 15 points was observed in children who have been severely undernourished in early childhood (Martorell, 1996).

Weight at birth is a strong predictor for size in later life because most IUGR infants do not catch-up to normal size during childhood. Hence the incidence of low birth weight predicts the prevalence of underweight during preschool and subsequent years (Mason et. al., 1999). Martorell et. al. (1998) observed that males and females of 17 to 19 years of age, who were born IUGR-LBW, were about 5 cm shorter and weighed 5 Kg less than those who were not born IUGR-LBW. Such differences are found to be similar in developed and developing countries.

According to Albertson and Karlberg (1994) menarche and maturation are probably not delayed by being born IUGR.

According to Evensen (2004) one in four Very Low Birth Weight (VLBW) children and one in six SGA children had motor problems. It was found that there was no sex differences in motor problems in the VLBW group and for SGA children, the increased risk of motor problems were particularly in manual dexterity in boys (Evensen, 2004).

Grantham (1998) observed that deficits in the growth of anthropometric parameters in early age lead to stunting. Grantham et. al. (2000) found that physical stunting is closely linked to impaired mental and psychomotor development. Low
birth weight with deprived socio-economic and environmental conditions trigger deficient cognitive development among children, however, it was found difficult to isolate the effects of IUGR from the socio-economic and environmental factors in relation to cognitive development (Grantham, 1998).

According to Martorell et al. (1998), adolescent males and females born with IUGR and at an average age of 15 years, performed significantly more poorly on tests of strength, compared to those born weighing at least 2500 g. Haas et al. (1996) observed that adolescents born with low birth weight showed 2 to 3 kg less force to a hand grip dynamometer due to their lower fat-free mass. Hence IUGR has a serious adverse impact on later work productivity and income generating potential (Haas et al., 1996).

2.4.3. LBW and Immunity

Low birth weight is generally an outcome of foetal insult or nutritional insufficiency. According to Rubhana et al. (2007) such exposure will affect immunocompetence and susceptibility to infectious diseases. Ann (1985) observed that immune response of low birth weight infants is severely compromised and is more adversely impaired than that of postnatally malnourished infants. It was also reported that there is reduction in maternal transfer of IgG and impaired synthesis of IgA, IgM and C3 component of complement (Ann, 1985; Chandra, 1999; Godfrey, 1994). Das et al. (1998) observed that blockage of IgG – specific FC receptor site in the placenta due to acute atherosis and reduced uteroplacental perfusion leads to lower IgG in low birth weight infants.
Okoko et al. (2001) found that materno-foetal transfer of antibodies is impaired in premature and low birth weight babies and the reduction in antibody transfer predispose the vulnerable neonates to bacteria and viral infections. There is also reduction in mature fully differentiated T lymphocytes partly due to reduction in serum thymic factor activity (Chandra, 1981; Chandra, 1997).

Chandra (1992) reported that in low birth weight or malnourished infants the Cd4+ antigen on the cell surface and CD8 cells were markedly decreased as a result of inhibitory factors and deficiency of essential nutrients in the plasma leading to reduced immune response.

Phagocytosis and opsonin concentrations and activity of most complement components are decreased along with reduction in C3, C5, factor B and total haemolytic activity (Chandra, 1996).

Rubhana et al. (2007) opined that greater peripheral T cell turnover due to immune activation leading to elevated C reactive protein concentrations and bactericidal activity, result in greater need for replenishment from the thymus leading to lower immune functional reserve in pre school age children born with low birth weight.

2.4.4. Long term consequences of LBW

The causes and effects of low birth weight are complex and have long term implications (ACC/SCN, 2000).
According to WHO (2002), the long term sequelae of preterm birth include neurological complications such as periventricular leucomalacia, cerebral palsy, seizures, delayed development and learning difficulties, pulmonary outcomes such as bronchopulmonary dysplasia, recurrent wheezing with respiratory infections and ophthalmologic complications such as retinopathy and blindness.

The Foetal Origins of Adult Disease (FOAD) hypothesis as proposed by Barker (1998) or Barker hypothesis stated that foetal under nutrition at critical periods of development in utero and during infancy leads to permanent changes in body structure and metabolism. These changes result in increased adult susceptibility to Coronary Heart Disease (CHD) and Non-Insulin Dependent Diabetes Mellitus (NIDDM) (Stein et. al., 1996; Mi et. al., 2000).

According to Barker and Osmond (1986) the adults born with low birth weight suffer an increased risk of high blood pressure, obstructive lung disease, high blood cholesterol and renal damage. Barker (1998) found that adaptations for foetal survival in an inadequate nutritional environment contribute to adult chronic disease, when nutrients are plentiful.

Low birth weight has long term physiological consequences that a woman born as a low birth weight infant may have difficulty in developing a placenta that will provide adequate nutrition to the foetus (Andersson and Bergstrom, 1997).

Mi et. al. (2000) opined that an undernourished foetus conserves or diverts blood flow to the head simultaneously reducing the blood flow to the liver, pancreas
and kidneys leading to reduced secretion of growth hormones, insulin and other endocrine changes which leads to CHD and NIDDM in adulthood.

Hales et. al. (1991) found that more than 20 per cent of men whose birth weights were lower than 2500g had abnormal glucose tolerance, compared with those weighing more than 4000g at birth. Mi et. al. (2000) opined that Type 2 diabetes mellitus and hypertension have a common origin in sub-optimal development in utero, and that syndrome X – Type 2 diabetes mellitus, hypertension and hyperlipidaemia should be renamed, “small baby syndrome”.

Hattersley and Tooke (1999) suggested that presence of gene for insulin resistance in an individual lead to low birth weight and the same genetic predisposition would lead to an increased risk of adult diabetes and CHD.

2.5. Interventions to prevent LBW

The problem of IUGR is multifactorial in nature and hence it is difficult to prevent through one single intervention. WHO (2002) identified nutritional, health care, health related behavior and infection control as possible interventions to prevent low birth weight. According to Judith and Laura (2000) interventions to prevent low birth weight should have the potential to break the cycle of intergenerational under nutrition leading to low birth weight. According to Radha et. al. (2002) attempts to prevent prematurity and low birth weight need long term programmatic interventions addressing the problems of women’s health, nutrition, literacy and overall lifestyle which would require huge input of resources.
WHO (2002) opined that foetal growth is regulated by endocrine processes which may or may not be influenced by interventions as some of the processes are genetically controlled. Thus interventions targeted to modifiable factors occurring during prepregnancy such as maternal stature, BMI, age and birth interval and factors occurring during pregnancy such as maternal weight gain, micronutrient status, energy and protein intake, malaria, smoking/pollution, and violence and stress are seemed to be effective. According to WHO (2002) the effective interventions to prevent IUGR are macronutrient food supplementation, counseling to reduce cigarette smoking, malarial prophylaxis in primiparous women and low dose aspirin in high risk women.

Prentice et. al. (1983) suggested that reduction in workload and cerclage in women with cervical incompetence are effective in reducing preterm birth. Fawsi et. al. (1998) found that protein/energy and multivitamin supplementation during pregnancy significantly reduced preterm births, still birth and miscarriage rates. Mario et. al. (2003) opined that balanced protein energy supplementation can reduce the overall risk of SGA by 30 per cent.

Improving pre pregnancy weight and weight gain during pregnancy are effective strategies to reduce and prevent low birth weight (Andersson and Bergstrom, 1997). Breastfeeding, appropriate complementary feeding (Lucas et. al., 1997) and adequate micronutrient status during infancy, early childhood, adolescence and pregnancy are necessary to reduce and prevent low birth weight (Judith and Laura, 2000). Devinder et. al. (2002) found that improvement in
healthcare system, awareness of values of antenatal care among the general population and better health of the mothers at the onset of reproductive life has helped to improve the birth weight of babies and decrease the perinatal mortality. Such interventions must develop a variety of supportive nutrition communication messages and activities that fit the practicalities of the target group’s lives and their interests (Winichagoon et. al., 1992).

Even though the need for a continued health care across prenatal, antenatal and early childhood periods in order to promote optimal foetal development is increasingly recognized, the transaction of this into delivering the appropriate packages for interventions is still lacking (WHO, 2006).

2.6. Under nutrition in preschool children.

Black and Bryce (2003) opined that malnutrition is a significant contributing factor to more than half of all child deaths. According to WHO (2006 a) one out of four preschool children in developing countries suffer from under nutrition and 10 million children die every year before the age of five and approximately 53 per cent of this is attributable to under nutrition. According to Grant (1994), in developing countries 190 million children under the age of five years are chronically malnourished. Pelletier et. al. (1994) opined that improved child survival was strongly associated with reduced malnutrition in countries characterized by high rates of malnutrition.
According to Reddy (2006) preschool age is one of the most vulnerable periods mainly due to easy susceptibility to malnutrition and infection leading to stunting of physical growth and suboptimal intellectual growth. Thus malnutrition affects the rate of morbidity among the young (Senauer and Gascia, 2001).

UNICEF (2003) reported that malnutrition implied more than half of child deaths worldwide. Malnutrition was directly or indirectly responsible for about 60 per cent of 10.9 million deaths annually among children under five (WHO and UNICEF, 2003). Yegammai et al. (2002) opined that India accounts for 40 per cent of world’s malnourished children and over 40000 children die of malnutrition and related diseases daily.

Grant (1994) reported that globally malnutrition was a stealthy accomplice of poverty and stunts the mental and physical growth of one in three children in the developing world. According to Onis et al. (2000) despite decreasing rates of stunting due to malnutrition in developing countries child malnutrition is still a major public health problem.

Jyothylakshmi and Prakash (2004) reported that a child’s overall development including nutritional status is solely or wholly dependent on the mother’s health and nutritional status from in utero to birth and later on her other characteristics such as literary status, awareness and economic status. The prevention of low birth weight and promotion of adequate growth and development during early childhood result in healthier and more productive adults (Martorell, 1999).
In India most of the children are below acceptable level of nourishment and about half of the death is associated with malnutrition and disease. The variables which contribute to malnutrition included poverty, literacy, lack of knowledge about nutrition and health among the parents, unhygienic environment and infection. As an important strategy to overcome malnutrition, government of India has formulated ICDS programme to increase the survival rate and enhance the health, nutrition and learning opportunities of preschool children and their mothers (Lakshmi et. al., 2003).

A review of several studies indicated that PEM and micronutrient deficiencies, especially those of Vitamin A, iron and iodine, which are of national importance lead to adverse consequences in growth, development, immunity and onset of infections (Annakodi and Premakumari, 2005)

Grant (1994) found that malnourished children become more vulnerable to diseases due to poor resistance to diseases and was observed that nutritional deficiencies and infectious diseases are the major contributors to morbidity and mortality and form a vicious cycle. Pelletier et. al. (1993) estimated that more than half of the deaths among children fewer than five years are due to malnutrition and related incidence of diseases.

According to FAO (1992) malnutrition, the manmade disaster, is an avoidable tragedy with enormous social and economic potential and with deleterious effects on growth and reproduction and undermines health, learning, working capacity and overall quality of life and well being of an individual.