Chapter-II

REVIEW OF LITERATURE

Literature pertaining to this study on “The Effect of Selected Modifiable Maternal Risk Factors during Pregnancy on Early Childhood Obesity.” is reviewed under the following headings:

I. Prevalence and Significance of Childhood Obesity in US and Worldwide Population

II. Importance of Understanding the Underlying Causes of Maternal Risk Factors and Adverse Birth Outcomes Which Leads to Childhood Obesity.
   1. Maternal Smoking
   2. Maternal Overweight And Obese Pregravid Weight
   3. Gestational Diabetes
   4. Effect of dietary intake (Fruits, Vegetables and Dairy products) and daily activity, Screen Time on Nutritional Status

III. Preventive Measures of Childhood Obesity

IV. Importance of Nutritional Counseling on Childhood Obesity.
   1. Improving the Ability of Registered Dietitians through Motivational Interviewing Technique.
   2. Intervention With the trained Same Nutrition Professionals - Taking A Role As A Helper

V. Professional Competency with Current Knowledge for Practicing Nutritionists in Public Health

I. PREVALENCE AND SIGNIFICANCE OF CHILDHOOD OBESITY IN USA AND WORLDWIDE POPULATION.

During the past few decades, levels of childhood obesity worldwide have escalated(Ogden, et.al., 2006). The prevalence of obesity has increased substantially since the mid-20th century. Although there seems to have been an accelerated rate of increase somewhere around 1980, at least in the United States, evidence suggests that obesity has been increasing in prevalence for over one hundred years. Within the United States, this
increase has occurred in every age, race, sex and socioeconomic group. Although recent evidence suggests that the prevalence of obesity may have begun to asymptote within some segments of the U.S. and some other populations, there is no sign of any decreases in U.S. prevalence to date. Obesity has not only increased in the United States but also seems to have increased in virtually every country where detailed data are available. (EmilyJ. McAllister et.al., 2009).

Within Europe, current levels are especially high in southern countries, including Spain and Italy (Lobstein, et.al., 2003). These trends are problematic because childhood overweight is strongly associated with adult obesity and chronic disease risk. Recent evidence suggests growth trajectories leading to overweight and obesity may begin very early in life, with strong associations between rapid growth in early infancy and overweight later in childhood and adolescence. As a result of this data, a growing body of literature has begun to explore whether in utero exposures may influence long-term risk of obesity (Michelle A. Mendez et.al., 2008).

Childhood obesity has more than doubled in children and tripled in adolescents in the past 30 years. The percentage of children aged 6–11 years in the United States who were obese increased from 7 percent in 1980 to nearly 18 percent in 2010. Similarly, the percentage of adolescents aged 12–19 years who were obese increased from percent to 18 percent over the same period. Overweight is defined as having excess body weight for a particular height from fat, muscle, bone, water, or a combination of these factors. Obesity is defined as having excess body fat. Overweight and obesity are the result of “caloric imbalance”—too few calories expended for the amount of calories consumed—and are affected by various genetic, behavioral, and environmental factors (CDC. 2013).

Childhood and adolescent overweight and obesity are particularly detrimental because they often persist in adulthood. The hazards of being overweight or obese in childhood and adolescence have been well researched. The Bogalusa Heart Study found that 60 percent of the overweight children by the time they reach 10 y have at least 1 biochemical or clinical cardiovascular risk factor and 25 percent overweight children have more than two (Freedman, et.al, 1999).
According to National Center for Health Statistics, 2007, the prevalence of both overweight and obesity in the U.S. population has increased dramatically in the last few decades creating an important public health issue. The combined prevalence of overweight and obesity among adults increased from 47 percent in 1976-1980 to 66 percent in 2001-2004. The prevalence of obesity alone among adults during the same period doubled from 15 percent to 32 percent. Among adolescents ages 12 to 19, the prevalence of overweight individuals more than tripled from 5 percent to 17 percent. Six and one-half percent of children ages 6 to 11 years were overweight in the 1976-1980 time period and that rate almost tripled to 17.5 percent in 2001-2004. Several adverse health outcomes have been associated with excessive weight, including increased risks for heart disease, stroke, high blood pressure, type 2 diabetes, and certain cancers (Callaway, et.al, 2006).

II. IMPORTANCE OF UNDERSTANDING THE UNDERLYING CAUSES OF MATERNAL RISK FACTORS AND ADVERSE BIRTH OUTCOMES WHICH LEADS CHILDHOOD OBESITY

There are a number of factors that contribute to the epidemic of childhood overweight and obesity. Body weight is shaped by a combination of genetic, metabolic, behavioral, environmental, socio-cultural, and socioeconomic factors. For a large majority of individuals, overweight and obesity result from excess energy consumption and/or inadequate physical activity (Office of the Surgeon General, 2001).

Unhealthy eating habits and physical inactivity are early to become established and contribute in a major way toward development of childhood overweight and obesity. Commonly suggested modifiable public health strategies to combat childhood obesity are promotion of breast-feeding, limiting television viewing, encouraging physical activity, increasing fruit and vegetable intake, controlling portion sizes, and limiting sweetened drink consumption (Gerberding, JL and Marks, JS. 2004). Thus dietary modification is a very important part of all strategies aimed at combating childhood overweight and obesity.

As it is mentioned before pre-pregnancy weight is a risk factor for obesity during childhood. A study conducted by Robert Whitaker showed that 1 in 4 preschoolers who were born to obese mothers were also overweight. Other risk factors for preschoolers being obese are LGA and being the first-born. (Whitaker, 2004). Into childhood, maternal
pre-pregnancy BMI over 30 is the greatest risk factor for a child in the upper percentile for weight and also for percentage body fat (Catalano, et.al, 2009). The effect is even noted at 10 years of age. When a mother has a high intake of macronutrients, the child also has a high intake of the same nutrients (Brion, et.al, 2010). This shows that the dietary habits of the mother during pregnancy affect the dietary habits of her child. Therefore if the mother exhibits unhealthy eating practices during pregnancy, that the child will also exhibit unhealthy eating habits later (Whitaker, 2004).

Body fat of the infant is measured with tricept, subscapular and flank skinfold measurements, and are found to be significantly increased when born to mothers who are overweight or obese than to mothers who are lean or average. This phenomenon can be attributed to the increased availability of nutrients such as glucose and triglycerides in the mother’s blood supply (Sewell, et.al., 2006).

Another factor contributing to obesity in the child is metabolic syndrome, which is now being associated with LGA births. Metabolic syndrome is defined by the American Heart Association by abdominal obesity, atherogenic dyslipidemia, elevated blood pressure, insulin resistance or glucose intolerance, prothrombic state (high fibrinogen or plasminogen activator inhibitor in the blood), and a proinflammatory state (Whitaker , 2004). Even if the mother did not have diabetes before or during pregnancy, her pre-pregnancy BMI was an independent risk factor for developing metabolic syndrome (Catalano, et.al, 2009). GDM does not always lead to type II diabetes mellitus postpartum, however developing GDM significantly increases the chance of developing type II DM. Pre-pregnancy BMI is the primary predictor of increased birth weight and LGA among women with GDM (Segal, et.al, 2008).

Macrosomia, or LGA (large for gestational age), is defined as exceeding the 90th percentile for gestational age and is also defined as a birth weight greater than 4500g or 10lbs (Brown, 2008). Mothers who are overweight are obese are 1.7 times more likely to deliver an infant weighing more than 4000g; mothers who are morbidly obese are 2.0 times more likely to deliver an infant weighing 4500g than mothers who are lean or average (Weiss, et. al., 2004).
Fetal overgrowth, LGA, related to increased adiposity places the child at risk for developing diabetes, and starts in utero (Catalano, et. al., 2006). Childhood overweight and obesity are increasing problems in developed countries. Obese and overweight children tend to remain so into adult life and consequently, they have an increased risk of suffering from type 2 diabetes and coronary heart disease (Khaodhiar, et.al., 1999).

Fetal growth retardation has been suggested as a risk factor for adult obesity. Several studies have reported that low birth weight is associated with increased risk of obesity, type 2 diabetes and coronary heart disease in adult life (Godfrey, KM, and Barker, DJP, 2000). These associations have led to the ‘fetal origin of adult diseases’ hypothesis.

According to this hypothesis, reduced nutrition in utero leads to adaptive changes in the fetus. These adaptive changes, called programming, may be essential for the survival of the fetus but, in an affluent environment after birth, they may have adverse effects leading to the development of obesity, coronary heart disease and type 2 diabetes (Godfrey, KM, and Barker, DJP, 2000). However, the hypothesis lacks a biological explanation, and confounding factors have not been appropriately accounted for in several studies.

Environmental factors contributing to reduced birthweight are a great concern because of the well-known relation of birthweight to infant mortality and adverse health effects in later life. Recent epidemiological studies have linked maternal tobacco-smoking and other environmental exposures to increased risk of low birth weight, preterm delivery, congenital anomalies, pregnancy loss, foetal growth, birthweight (AstaDanileviciute, et.al., 2012).

Smoking during pregnancy is a principal environmental cause of intrauterine growth retardation in developed countries. Children of smokers have lower weight and length and less fat mass at birth than children of non-smokers. Several investigators have reported an increased risk of obesity in childhood or adulthood after intrauterine exposure to maternal cigarette smoking (Oken, E. Gillman, 2003).
Because parents influence their children’s eating habits (Birch, et.al 2001) and there is evidence to suggest that smokers have less healthy diets, differences in diet may have been responsible for the higher body and fat mass observed among children of smoking mothers. Moreover, mothers who smoke during pregnancy have lower socio-economic status, and they tend to breast feed their children for less time than non-smokers (Hediger, et.al., 2000). Both factors have been related to childhood overweight and obesity and may thus be considered possible confounders (Marius Widerøe et.al., 2003).

1. MATERNAL SMOKING:

Smoking is the second leading risk factor for global mortality (Lopez et.al., 2006). IN THE UNITED STATES, 65 percent of all infant’s deaths occur among low birth-weight (LBW) infants (≤2500g); LBW infants account for 7.6 percent of all live-born infants. The etiology of LBW is largely unknown, but both environmental and genetic factors may play a role. Numerous studies have shown that maternal cigarette smoking during pregnancy is associated with reduced birth weight or increased risk of LBW. Maternal cigarette smoking is identified as the single largest modifiable risk factor for intrauterine growth restriction in developed countries (Wang, et.al., 2002).

Active maternal cigarette smoking has a damaging effect in every trimester of pregnancy, starting with an increase in miscarriage rates during the first trimester and an increased risk of premature delivery, fetal growth restriction, and stillbirth during the second and third trimesters. Maternal smoking is also a risk factor for placental abruption and placenta previa. Epidemiological studies suggest that a similar negative effect can also be observed on fetal growth in cases of passive maternal smoking. Cigarette smoke emits scores of toxins, including cyanide, sulphide, cadmium, carcinogenic hydrocarbons, and nicotine that induce direct cellular damage. Although placental xenobiotic-metabolizing enzymes can detoxify foreign chemicals, tobacco constituents exert a direct effect on the villous cytotrophoblast proliferation and differentiation, which can explain its negative effect on placentation and fetoplacental development. Placental morphological damages related to heavy chronic maternal smoking can be identified as early as the first trimester of pregnancy. Most tobacco toxins have low molecular weights and high water solubility and therefore readily cross the placenta, and chronic maternal smoking is
associated with alterations of protein metabolism and enzyme activity in the fetal blood. Recently it has been shown that pregnant women who smoke have lower folate serum levels than nonsmokers. However, more recent studies on peri-conceptional folic acid supplementation and folic acid food fortification have demonstrated a strong protective effect against the occurrence of fetal neural tube defects (NTD) (Jauniaux, E. et al., 2007).

An association between maternal smoking during pregnancy and excess weight in other phases of life has been reported, with this effect being dose-dependent in childhood and independent of intrauterine growth restriction, and being attributable to the specific effect of cigarettes. Apparently, only mothers who smoke early during pregnancy have children with excess weight. The association between obesity and maternal smoking is observed in early childhood and also during school age and is stronger at later ages.

Parental smoking habits were associated with different dietary patterns among teenagers regardless of whether the teenagers themselves smoked. Dietary differences noted were similar to those observed previously among smokers, with lower intakes of fibre, vitamin C, vitamin E, folates, and magnesium in particular reported among both males and females in households where parents were smokers. These lower intakes were associated with lower intakes of fruit juices, whole meal bread, and some vegetables. Teenagers who lived with parents who smoked had different nutrient and food intakes to those with non-smoking parents, and teenagers exposed to parental smoking appeared to have similar dietary patterns to teenagers who themselves smoked (Crawley, H.F. & While, D. 1996).

Cigarette smoking is an addictive behavior, and is the primary cause of cardiovascular and pulmonary disease, and cancer (among other diseases). Cigarette smoke contains thousands of components that may affect caloric intake and energy expenditure, although nicotine is the major addictive substance present, and has the best described actions. Nicotine exposure from cigarette smoke can change brain feeding regulation to reduce appetite via both energy homeostatic and reward mechanisms, causing a negative energy state which is characterized by reduced energy intake and increased energy expenditure that are linked to low body weight. These findings have led to the public perception that smoking is associated with weight loss. However, its effects
at reducing abdominal fat mass (a predisposing factor for glucose intolerance and insulin resistance) are marginal, and its promotion of lean body mass loss in animal studies suggests a limited potential for treatment in obesity. Smoking during pregnancy puts pressure on the mother’s metabolic system and is a significant contributor to adverse pregnancy outcomes. Smoking is a predictor of future risk for respiratory dysfunction, social behavioral problems, cardiovascular disease, obesity, and type-2 diabetes. Catch-up growth is normally observed in children exposed to intrauterine smoke, which has been linked to subsequent childhood obesity. Nicotine can have a profound impact on the developing fetal brain, via its ability to rapidly and fully pass the placenta. In animal studies this has been linked with abnormal hypothalamic gene expression of appetite regulators such as downregulation of NPY and POMC in the arcuate nucleus of the hypothalamus. Maternal smoking or nicotine replacement leads to unhealthy eating habits (such as junk food addiction) and other behavioral disorders in the offspring (Chen, H. et.al, 2012)

During the past few decades, levels of childhood obesity worldwide have escalated. These trends are problematic because childhood overweight is strongly associated with adult obesity and chronic disease risk (Reilly, 2005). It has been hypothesized that metabolic adaptations linked to maternal smoking may promote prenatal growth retardation through pathways that may increase risk obesogenic growth in the postnatal environment (Blake et.al, 2000).

Because parents influence their children’s eating habits (Birch, et.al, 2001) and there is evidence to suggest that smokers have less healthy diets differences in diet may have been responsible for the higher body and fat mass observed among children of smoking mothers. Moreover, mothers who smoke during pregnancy have lower socio-economic status, and they tend to breast feed their children for less time than non-smokers (Hediger et.al., 2001). Both factors have been related to childhood overweight and obesity and may thus be considered possible confounders. (Von Kries, et.al 1999). Because therapeutic interventions for overweight in children are costly and have far from satisfactory results the development of strategies for prevention of overweight and obesity is a major challenge for health care professionals.
Despite decades of research, press, counter-advertising, and litigation regarding its adverse effects, tobacco use remains a major cause of preventable morbidity and mortality world-wide. Although fewer women in the US and Britain now smoke than in past decades, an increasing number of teenage girls are initiating smoking, and smoking rates are declining less rapidly among women than among men, so cigarette smoking remains common among women who are of childbearing age, pregnant, or breastfeeding (Office on Smoking and Health, GA, 2002). In the developing world, a small but rapidly expanding proportion of women smoke.

As per Institute of Medicine, and National Academy of Sciences (2004), childhood obesity is a leading public health concern in the United States. Fetal growth retardation has been suggested as a risk factor for later obesity (Ogden, et al., 2006). Smoking during pregnancy is a known cause of reduced fetal growth (Guo, et al., 2002). Investigators from several studies have reported a positive association between smoking during pregnancy and subsequent child obesity (Freedman et al., 2005). The studies done by Barker, et al., (1993), and Low et. al., (1992), showed that maternal smoking has been associated with a reduction in newborn birth weight. The reductions in neonatal weight observed with maternal smoking may have life-long consequences. Evidence points to significant pediatric and adult morbidity associated with one’s reduced weight at birth.

Another study found that regardless of race, there are higher LBW occurrences among smokers than among non-smokers (Ventura, et al., 2003). Thus maternal smoking during pregnancy is an established risk factor for miscarriage/perinatal mortality, low birth weight, premature births, and small fetuses. Extensive human and laboratory studies have examined the biological mechanisms of how tobacco smoke affects fetal development. These studies show that many of the 7,000 chemicals in tobacco can cross the placental barrier and have a direct harmful effect on the unborn baby (Allan Hackshaw, et al., 2011).

2. MATERNAL OVERWEIGHT AND OBESE PREGRAVID WEIGHT:

The increasing rate of maternal obesity provides a major challenge to obstetric practice. Maternal obesity can result in negative outcomes for both women and fetuses. The maternal risks during pregnancy include gestational diabetes and preeclampsia. The
fetus is at risk for stillbirth and congenital anomalies. Obesity in pregnancy can also affect health later in life for both mother and child. For women, these risks include heart disease and hypertension. Children have a risk of future obesity and heart disease. Women and their offspring are at increased risk for diabetes. Obstetrician-gynecologists are well positioned to prevent and treat this epidemic (Meaghan A. Leddy et.al., 2008).

Maternal plasma glucose concentrations during pregnancy are a key determinant of fetal growth, and there is evidence that maternal glucose concentrations affect offspring size and adiposity throughout life. The “hyperglycemia-hyperinsulinism” mechanism has been recognized as important since first proposed in the 1950s. It suggests that increased fetal secretion of insulin in response to greater transfer of glucose across the placenta stimulates greater somatic fetal growth and higher levels of subcutaneous fat. In addition, mechanisms acting via cognitive and neuroendocrine development and subsequent offspring appetite and diet might be important. Thus high maternal dietary intakes in pregnancy may lead to increased fetal growth and program neuroendocrine pathways that result in greater appetite, energy intake, and adiposity in offspring later in life (Marie-Jo A Brion et. al. 2010).

Recent national data show that 82.1 percent of Black women and 75.7 percent of Hispanic women are overweight or obese compared to 59.5 percent of White women. In addition, over half of Black women are obese (versus 38.8 percent of Black men and 32.2 percent of White women). Extreme obesity continues to be higher among women (8.1 percent) than men (4.4 percent), especially among Black women who have more than double the rates of extreme obesity as White and Hispanic women (17.8 percent versus 7.1 percent and 6.0 percent). Rates of overweight or obesity are higher for Hispanic men (81.7 percent) compared to Black men (69.9 percent) and White men (74.0 percent), although obesity rates are fairly similar across racial-ethnic groups (Ogden et al., 2012).

The risk of increasing obesity is disproportionate among the races, with the prevalence of obesity increasing most among African American women. The epidemic of obesity is not unique to the USA, as a significant increase in the incidence of obesity in women of reproductive age has been noted over the past decade in Europe, approaching
that reported in the USA insome countries. In this review, unless otherwisnotated, we will
define obesity based on the World HealthOrganization1 and National Institutes of Health
criteria. Normal weight is defined as a BMI of 18.5–24.9, overweightas a BMI 25–29.9
and obesity as a BMI ≥ 30. Obesity canbe further characterised by BMI as class I (30–
34.9), class II(35–39.9) and class III (≥ 40). Among adults aged 20 to 39 years, 28.5
percent were obese while 36.8 percent of adults aged 40 to 59 years and 31 percent of
those aged 60 years or older were obese in 2003-2004 (Flegal, K. M et.al., 2006).

Thus maternal obesity is one of the strongest and most reliable predictors of later
obesity in children. Maternal overweight or obesity entering pregnancy is also a risk factor
for childhood obesity. Infants born to overweight mothers are more likely to be born large
for gestational age, are less likely to be breastfed, and are at higher risk for obesity and
type 2 diabetes in later life (Brittany Dixonet.al., 2012). More than one-third of women of
childbearing age in the U.S. are overweight or obese and this prevalence is increasing.
Therefore, it is of public health importance to study the impact of maternal weight on
adverse pregnancy and birth outcomes.

3. GESTATIONAL DIABETES:

The epidemic of childhood obesity that has occurred in the past 20 years is
associated with an increase in the prevalence of type 2 diabetes mellitus (T2DM) among
children and adolescents. By 1994, 14 percent of children and 12 percent of adolescents were
overweight, as defined by a BMI of 85th percentilefor age, and recent data indicate that the
prevalence of obesity has continued to increase. The increased obesity was accompanied
by an increase in T2DM among adolescents, from 5 percent of new cases of diabetes in
1982 to 45 percent (depending on geographic location) in 1999. There are a number of
risk factors for T2DM among children and adults in addition to obesity. A well-recognized
risk factor among adults is the metabolic syndrome (MS), also called syndrome X, which
was first described in the 1950s and predisposes individuals to diabetes and cardiovascular
disease. MS is defined as the association of obesity, insulin resistance, glucose intolerance,
hypertension, and a characteristic dyslipidemia. There is a growing body of literature on
the prevalence of components of the MS among obese children and adolescents. This
raises great concern about the potential development of not only T2DM but also early
stages of cardiovascular disease in childhood. Longitudinal studies of Pima Indian children demonstrated that birth weight, i.e., either small for gestational age (SGA) or large for gestational age (LGA) exposure to diabetes in utero, and obesity are the major factors in the development of childhood T2DM and hypertension (Charlotte M. Boney, et al., 2005).

India and other countries in Asia are experiencing rapidly escalating epidemics of diabetes and cardiovascular disease. The prevalence of type 2 diabetes in urban Indian adults has increased from <3 percent in the 1970s to >12 percent in 2000 (Ramachandran, et al., 2000).

Hales and Barker proposed that undernutrition at critical periods in intrauterine development causes permanent changes in the structure and/or function of the developing systems of the fetus. This increases susceptibility to disease in later life. Of the many possible insults during the intrauterine life, Hales and Barker favored undernutrition as the most likely cause, though many factors could operate in a similar manner. The original hypothesis of Hales and Barker (1992) overlooked the classic association among maternal diabetes, fetal macrosomia and increased risk of diabetes for the offspring. A recent version of the thrifty phenotype hypothesis allows for this (Hales and Barker, 2001).

However, according to United Nations Children’s Fund (UNICEF) Statistics, World summit for children, the relationships among maternal nutrition, fetal nutrition, neonatal size and later diabetes appear to be more complicated than originally proposed. This may have important implications for preventive strategies. The crucial importance of the intrauterine period in determining health and disease is not difficult to understand if one appreciates that humans begin life as a single cell and that more than three quarters of total cell divisions occur in utero. Substantial growth and development is completed before birth. Intrauterine growth and development involves orchestrated gene expression regulated by the environment of the fetus, which is of course largely regulated by the mother (Parikh, S. J., Yanovski, J. A. 2003).

Surveys show that Gestational Diabetes (GDM) tends to occur more frequently in Asian, Hispanic, and Native American women than in African-American and white women (Ferrara, et al., 2002). Normally, the insulin sensitivity of peripheral tissue decreases approximately 50 percent to 60 percent in late pregnancy in lean women; the
decrease is greater in obese women. Thus hyperinsulinemia is common in all pregnant women, with higher levels seen in overweight and obese women. If pancreatic insulin secretion is adequate, women will remain glucose tolerant throughout gestation and GDM does not develop. It is thought that the shift toward reduced insulin sensitivity (or increased insulin resistance) during late pregnancy occurs to limit maternal glucose utilization and conserve it for diffusion across the placenta to the fetus. Maternal hyperinsulinemia also enhances the rate of maternal adipocyte fat oxidation, which releases more fatty acids into circulation for use as a fuel source by the mother (Hollingsworth, 1986).

The free fatty acids are converted to triglycerides in the liver and returned to circulation as very-low-density lipoproteins, resulting in high very low-density lipoprotein concentrations in late pregnancy (King, J.C., 2006). This dyslipidemia usually disappears after delivery. It is not unusual for a mild inflammatory state to occur in obese pregnant women with glucose intolerance because pro-inflammatory cytokines (i.e., interleukin-6 and tumor necrosis factor-) are produced by the placenta as well as adipose tissue (Hauguel-de Mouzon, et. al., 2006). Research suggests that these pro-inflammatory cytokines may contribute to the decrease in insulin sensitivity seen in obese women with GDM (King, J.C. 2007).

Prenatal factors such as maternal gestational diabetes mellitus (GDM), prepregnant weight, weight gain in pregnancy and glycemia in pregnancy affect the development of the fetus (Vohr, B., and Boney, C. 2008). Risk factors for developing GDM include marked obesity, diabetes in a mother, father sister, or brother, history of glucose intolerance, previous LGA infant and current glucosuria. The infant born to a woman with GDM is at risk for stillbirth, spontaneous abortion, congenital anomalies, macrosomia, neonatal hypoglycemia and death, and increased risk of insulin resistance, type II DM, high blood pressure and obesity later in life (Brown Judy, 2008).

Another factor contributing to obesity in the child is metabolic syndrome, which is now being associated with LGA births. Metabolic syndrome is defined by the American Heart Association by abdominal obesity, atherogenic dyslipidemia, elevated blood pressure, insulin resistance or glucose intolerance, prothrombic state (high fibrinogen or
plasminogen activator inhibitor in the blood), and a pro-inflammatory state (Whitaker, et. al., 2008).

GDM does not always lead to type II diabetes mellitus postpartum, however developing GDM significantly increases the chance of developing type II DM. Pre-pregnancy BMI is the primary predictor of increased birth weight and LGA among women with GDM (Segal, and Hamilton, et. al., 2008).

4. **EFFECT OF DIETARY INTAKE (FRUITS, VEGETABLES AND DAIRY PRODUCTS), DAILY ACTIVITY AND SCREEN TIME ON NUTRITIONAL STATUS.**

   a) **Health Benefits of Fruit and Vegetable Consumption**

   Many epidemiological studies have been conducted to test the beneficial effects of fruit and vegetable intake on many different disease states such as heart disease, cancer, and stroke (Bazzano, L.A. 2006). Recent studies have tested fruit and vegetable intake in relationship to cataracts, diverticulosis, chronic obstructive pulmonary disease (COPD), and hypertension. The results from the research have been positive indicating that fruit and vegetable consumption is protective from various chronic diseases. The nutrients found in fruits and vegetables work synergistically to prevent risk factors for these health conditions. For example, sulfides contained in vegetables may detoxify carcinogens and stimulate anticancer enzymes. Phytoestrogens and phytoesterols found in vegetables are antioxidants, which inhibit growth of cancer cells. Phytoestrogens also play a preventative role in heart disease and hypertension by lowering blood cholesterol levels and platelet aggregation. Antioxidative flavinoids, found in mostly fruits and a couple of vegetables, are protective against cancer, heart disease, stroke, and COPD. Flavinoids may inhibit growth of cancer by reducing cell proliferation. They also inhibit clot formation and inflammatory actions, which decrease risk for heart disease, stroke, and hypertension. Furthermore, flavinoids enhance the function of vitamin C. Vitamin C is an antioxidant that participates in the regeneration of vitamin E, another antioxidant. Other vitamins found in fruits and vegetables are potassium and folate, which both help in the prevention of heart disease and hypertension. Fruits and vegetables also have fiber, which binds to and aids in excretion of cholesterol, fats, and carcinogenic substances. Fiber contributes to motility of substances...
through the digestive tract and prevents diverticulosis from occurring (Van Duyn MAS, Pivonka E. 2000). As per Center for Disease Control, cardiovascular disease is the leading cause of death in the United States and contributes to about 27 percent of deaths each year. Cancer is the second leading cause of death. These leading causes of death have many components that contribute to their formation.

b) Effects of Fruit and Vegetable Consumption on BMI

Lack of adequate fruit and vegetable consumption has become an important public health issue. According to the Canadian Community Health Survey 59 percent of Canadian children 2-17 years of age consume fruit and vegetables less than five times a day (Shields, 2005). These children are significantly more likely to be overweight or obese compared to those who consume fruit and vegetables more frequently. As per Dietary Guidelines Advisory Committee (DGAC) (2010), the conclusion that increased fruit and vegetable intake may protect against increased adiposity in children when consumed as part of a nutrient-rich, energy-balanced diet is based on a full Nutrition Evidence Library (NEL) literature search, supplemented by the findings of prospective studies included in an earlier evidence review conducted by the American Dietetic Association (ADA) (1982 to 2004). Collectively, the evidence review led to the conclusion that increased intake of fruits and vegetables may be associated with reduced adiposity in children.

c) Low Dairy Products Intake and its Negative Impact on Childhood Adiposity:

Milk consumption has declined during the same period. In 1977-78, children age 6-11 drank four times as much milk as any other beverage. In 1994-1996 that decreased to 1.5 times as much milk as sugar sweetened beverages (French, SA. et.al., 2003). In 1977-1978, adolescents drank 1.5 times as much milk as any other beverage and in 1996 they consumed twice as much sugar sweetened beverages as milk. Milk consumption decreased for adolescent boys and girls 37 and 30 percent respectively, between 1965 and 1996 (Cavadini, et.al. 2000).

The hypothesis that dairy consumption may be associated with weight regulation goes back to 1984 when McCarron et al (Lynn et.al., 2006) noted an inverse association between calcium intake and body weight among subjects in the first National Health and
Nutrition Examination Survey cohort. Animal studies have lent additional support to the idea that increasing dietary calcium among those on a low-calcium diet can suppress the lipogenic mechanisms associated with increases in circulating calcitrophic hormone.

A number of studies of calcium intake among adults have concluded that there is an inverse association between calcium intake and adiposity (Teegarden, D. 2003, and Parikh and Yanovski, 2003) although at least one review of randomized trials that were designed originally to study calcium effects on bone density concluded that there is little evidence to support a beneficial effect of calcium or dairy intake on body fat (Barr, 2002). There have been several randomized clinical trials designed to look specifically at calcium and dairy intake and body weight or fat (Zemel et.al., 2005, Zemel et.al., 2004). Those studies suggest that calcium supplementation in the form of dairy foods leads to significantly greater weight and fat loss among obese subjects on a weight loss diet whose levels of calcium were low at baseline. Another randomized trial of dairy calcium among healthy, normal-weight women had neither an adverse, nor a beneficial, effect on body fat (Gunther, et. al., 2005).

Although adult studies have focused on the role of dairy in weight loss, the focus of most studies of children is whether dairy has an etiologic role in the development of obesity. Because milk is an energy-dense food, it is possible that higher intake levels might be associated with increased weight or body fat gain during childhood. Several studies, however, have found no adverse effect of higher milk consumption levels on the amount of stored body fat in children (Phillips et.al., 2003, and Fisher, et.al., 2004). Data from an Italian lifestyle survey found that milk consumption among 5- to 11-year-old children was associated with lower BMI z scores (Barba, et.al., 2005).

A longitudinal study of preschool-aged children found that those consuming more servings per day of dairy, irrespective of total calorie intake, had significantly lower levels of body fat through 8 years of age. (Skinner, J. D. et.al., 2003) In a Kaiser-Permanente study, the amount of calcium in one serving of milk was associated with a 0.78-mm reduction in iliac skinfold (SF) thickness, whereas calcium from non-dairy sources had no effect. (Novotny, R. et.al., 2004) However, a recent review concluded that the majority of studies in children, including clinical trials, to date, do not support a protective effect of calcium and/or dairy on the risk of obesity (Huang, T.T.K. and Mc Crory, M. A. 2005)
d) Physical Inactivity and Sedentary Behaviors and its Negative Health Outcomes:

It is well established that excessive sedentary time, independent of too little exercise, leads to a number of negative health outcomes (Stamatakis, E.et.al., 2011). Collectively, leisure-time screen behaviors, such as television (TV), videos, DVDs, video games, and computers, have been associated with increased inactivity and metabolic risk factors (Healy, G. N.et.al., 2008). Children are accumulating a considerable amount of sedentary screen time, particularly TV viewing, (Rideout, V.J. et.al., 2010, Sisson, S.B et.al, 2009) and some are not getting adequate amounts of physical activity in their leisure time. For children and adolescents, overweight and obesity have been linked to sedentary leisure-time activities (Rey-Lopez, J.P. et.al., 2008)

Research indicates that a decrease in daily energy expenditure without a concomitant decrease in total energy consumption may be the underlying factor for the increase in childhood obesity. Physical activity trend data for children are limited, but cross sectional data indicates that one third of adolescents are not getting recommended levels of moderate or vigorous activity, 10 percent are completely inactive, and physical activity levels fall as adolescents age (IOM, 2004). This situation may actually be worse than these data describe. Activity measured by physical activity monitors tends to be significantly lower than what is reported on surveys (Pate, RR. et.al., 2002).

Watching television, using the computer, and playing video games occupy a large percentage of children’s leisure time, influencing their physical activity levels. It is estimated that children in the United States are spending 25 percent of their waking hours watching television and statistically, children who watch the most hours of television have the highest incidence of obesity(Robinson, T. N. 2001). This trend is apparent not only because little energy is expended while viewing television but also because of the concurrent consumption of high-calorie snacks.

A recent examination of the Department of Education’s Early Childhood Longitudinal Survey (ECLS-K) found that a one-hour increase in physical education per week resulted in a 0.31 point drop (approximately 1.8%) in body mass index among overweight and at-risk first grade girls. There was a smaller decrease for boys. The study
concluded that expanding physical education in kindergarten to at least five hours per week could reduce the percentage of girls classified as overweight from 9.8 to 5.6 percent (Ashlesha Datar, Roland Sturm, 2004).

This rise in childhood obesity has been associated with reduced levels of physical activity (energy expenditure), increased consumption of food (energy intake), or both (Sisson, S.B et.al., 2009). Sedentary screen behaviors, especially TV watching, are hypothesized to contribute to weight gain by reducing opportunities for energy expenditure and increasing energy intake (Williams et.al, 2008). Time spent engaging in TV watching can compete with time spent in other activities that require greater amounts of energy. (Rey-Lopez et.al, 2008). Also, TV watching is often coupled with unhealthy eating behaviors (e.g., increased consumption of soft drinks, fried foods, and snacks) due to influential environmental cues such as food and beverage commercials and easy access to food (Epstein, L.H. et.al., 2005). Thus, sedentary screen behaviors may influence both sides of the energy balance equation.

III. PREVENTIVE MEASURES OF CHILDHOOD OBESITY

Obesity in children is increasing rapidly around the world. The associated major adverse health consequences have been systematically reviewed and the psychological consequences and social marginalization reported. Prevention is clearly the ideal solution to the problem. However for those already overweight or obese, or at an increased risk of becoming so, effective interventions will offer the only chance of reducing the probability of progression to adult obesity, its associated comorbidities, and the increased risk of premature mortality. Essential elements of interventions are likely to include diet and food intake modification, and these have been identified for adult interventions. The efficacy of such interventions in the pediatric population is not known. An examination of successful interventions along with the key dietary changes achieved by participants would inform the optimal dietary treatment for broad dissemination (Reilly, et.al., 2004). Overweight children have a high risk for being overweight in adulthood and to experience typical obesity-related morbidity. Because therapeutic interventions for overweight in children are costly and have far from satisfactory results, the development of strategies for prevention of overweight and obesity is a major challenge for health care professionals.
As per Department of Health and Human Services CiDCaPTobacco Control State Highlights 2002 Despite decades of research, press, counter-advertising, and litigation regarding its adverse effects, tobacco use remains a major cause of preventable morbidity and mortality world-wide. Although fewer women in the US and Britain now smoke than in past decades, an increasing number of teenage girls are initiating smoking, and smoking rates are declining less rapidly among women than among men, so cigarette smoking remains common among women who are of childbearing age, pregnant, or breastfeeding (Hamilton, et.al., 2004).

In the developing world, a small but rapidly expanding proportion of women smoke. Exposure to cigarette smoke in utero puts a fetus at increased risk for a number of adverse health outcomes, including growth restriction (Taylor, et.al., 2006). Whereas smaller size at birth is generally associated with reduced later risk for overweight, recent research suggests that mothers who smoke during pregnancy have children at increased risk for later obesity. The combination of small size at birth and overweight in later life is not only characteristic of the epidemiologic transition from acute to chronic disease, but also confers a high risk of cardiovascular outcomes in adulthood (Oken, et.al., 2003).

The worldwide epidemic of obesity continues unabated. Obesity is notoriously difficult to treat, and, thus, prevention is critical. A new paradigm for prevention, which evolved from the notion that environmental factors in utero may influence lifelong health, has emerged in recent years. A large number of epidemiological studies have demonstrated a direct relationship between birth weight and BMI attained in later life. Although the data are limited by lack of information on potential confounders, these associations seem robust. Possible mechanisms include lasting changes in proportions of fat and lean body mass, central nervous system appetite control, and pancreatic structure and function. Additionally, lower birth weight seems to be associated with later risk for central obesity, which also confers increased cardiovascular risk. This association may be mediated through changes in the hypothalamic pituitary axis, insulin secretion and sensing, and vascular responsiveness. The combination of lower birth weight and higher attained BMI is most strongly associated with later disease risk. We are faced with the seeming paradox of increased adiposity at both ends of the birth weight spectrum-
higher BMI with higher birth weight and increased central obesity with lower birth weight. Future research on molecular genetics, intrauterine growth, growth trajectories after birth, and relationships of fat and lean mass will elucidate relationships between early life experiences and later body proportions. Prevention of obesity starting in childhood is critical and can have lifelong, perhaps multigenerational, impact (Oken, et.al., 2003).

IV. IMPORTANCE OF NUTRITIONAL COUNSELING ON CHILDHOOD OBESITY

a) Improving the Ability Of Registered Dietitians Through Motivational Interviewing Technique

Nutritional counseling is “a supportive process to set priorities, establish goals and create individualized action plans that acknowledge and foster responsibility for self-care” (Curry, and Jaffe, 1998). This process may include assisting the client in recognizing their nutrition-related priorities, establishing patient-stated goals and creating individualized actions plans. Strong evidence supports the combination of behavioral theory and cognitive behavior theory in modifying dietary patterns, weight and health risk factors. This evidence is strongest when applied in intermediate (6-12 months) and long-term (12 month+) durations. Both behavioral and cognitive behavioral theories function under the assumption that all behaviors are learned and influenced by an individual’s internal and external environments. The internal environment may be their physical and mental well-being; where as their external environment may be stress, relationships and availability of unhealthful foods at home (Spahn et. al., 2010).

There are many theories that can help assist a registered dietitian practicing nutritional counseling, including the trans-theoretical model (Prochaska, et.al., 1994) and several strategies, such as motivational interviewing. Registered dietitians (RDs) and dietetics practitioners frequently conduct nutrition counseling with clients to facilitate behavior change. To be effective, dietetics practitioners must be knowledgeable in nutrition and food science, diverse ethnic and regional culinary cuisines, and have practical experience with theory-based behavior change strategies. Theories and models,
validated within the field of dietetics, are frameworks for helping practitioners understand external and internal issues, and the dynamics that lead to behavioral changes (Joanne, M et.al 2010). The Trans-theoretical Model, or 'stages of change' concept recognizes that behaviour change is not stable and has stages of pre-contemplation, contemplation, preparation, action and maintenance (Basler, 1995).

Motivational interviewing is a client-centered strategy designed to elicit behavior change by assisting clients to explore and resolve ambivalence to change. Dietetics practitioners frequently use motivational interviewing when they utilize the trans-theoretical model with clients who are in the pre contemplative, contemplative, and preparation stages and require intervention targeting motivation. When applying this strategy, an RD partners with the client to determine the agenda using empathetic, nonjudgmental, supportive, encouraging, and active listening behaviors. Open-ended questions, reflective listening, affirmations, and summarization are used to help a client explore and resolve ambivalence and barriers to behavior change. Training in motivational interviewing is highly encouraged for working with clients who are not in the action stage of change (Miller, Rollnick, 2008).

b) Intervention With the trained Same Nutrition Professionals - Taking A Role As A Helper.

No matter what theory or behavior change model is providing the greatest influence, the relationship between counselor and client is the guiding force for change. The effect of this relationship is most often cited as the reason for success or failure of a counseling interaction. (Curry, and Jaffe, 1998, Helm and Klawitter, 2007) report that successful clients identify their personal interaction with their therapist as the single most important part of treatment. To set a stage for understanding the basics of an effective counseling relationship, you will investigate the characteristics of effective nutrition counselors explore your own personality and culture, examine the special needs and issues of a person seeking nutrition counseling.
Knowledge of these theories and strategies is important, but even more so is the recognition of our role as helper. The following are five reminders to help promote behavior change and not hurt. A helper:

- Focuses on the positive, not the negative,
- Encourages hope, not fear,
- Respects the potential in the person and the situation,
- Attempts to understand the prospective of the individual, and
- Releases a client to pursue their choice, rather than “our” choice.

Often times, when we as nutritional counselors seek dominance rather than encouragement, exhaustion results. It is best to use our energy on raising awareness of the benefits of the proposed change rather than the negative consequences, or the “right” way to pursue health and creating an environment that promotes change. It is also crucial to care for ourselves, in order to maintain these skills, abilities in focus so, we can truly help others. What are you going to do to care for yourself today? (Cassie Vanderwall, 2011).

V. PROFESSIONAL COMPETENCY WITH CURRENT KNOWLEDGE FOR PRACTICING NUTRITIONISTS IN PUBLIC HEALTH:

Public health nutrition has been a developing field of public health theory and practice for decades. However, it has only relatively recently been seen in the context of workforce development as a strategic component of building capacity for effective action. This focus, initially directed at under-nutrition, has more recently been applied as a gradual response to public health priorities such as non-communicable diseases (particularly cardiovascular disease) and more recently, obesity, diabetes and preventable cancers. This increase in focus in high-income countries, in response to the unsustainable burden of over-nutrition, has grown in parallel with a recognition that workforce capacity is a critical missing link in scaling up nutrition actions in order to accelerate the reduction of maternal and child under-nutrition in lower and middle income countries, many of which face the double burden of disease (Bryce, et. al., 2008 & Heikens, et. al., 2008).
The emphasis on developing designated public health nutritionists as a workforce and professional group, distinct from clinically orientated dietetics or medical nutrition workforce models, recognizes that population-based and promotional-preventive actions are required to address malnutrition in both forms. This requires different work that complements clinical practice and consequently requires additional competencies, the knowledge, skills and attitudes to perform this work (Hughes, R and Somerset, R. 1997).

Competency standards provide the architecture for workforce development by codifying the knowledge, skills and attitudes (ways of thinking) necessary to effectively practice public health nutrition (Hughes, 2004). They have a deliberate focus on effective performance in the workplace, ensuring that workforce preparation and continuing professional development not only enhances what practitioners know, but also that they know how, can show how, and do' (Wass, et. al., 2001).

Competency-based nutritional care is at the forefront of resident education for several reasons. Physicians are increasingly recognizing the importance that diet and exercise play in influencing the prevention, risk, and management of a variety of chronic diseases. Providing good nutritional care to patients is a part of the professional responsibility of every physician. Residency program faculty may seek to integrate nutritional care into their efforts to address the ACGME domains. Demonstration of proficiency in nutritional care can be used to reflect competency in each of the ACGME domains: patient care, medical knowledge, practice-based learning and improvement, interpersonal and communication skills, professionalism, and systems-based practice (Darwin Deen, 2006).

In this framework, the holistic-integrated approach to competency is applied, which outlines the complex combinations of attributes (knowledge, attitudes, ways of thinking, values, skills) required effectively to perform public health nutrition work as defined by public health nutrition core functions. Competence is conceived as a complex structuring of attributes needed for effective performance in specific or varied situations.