

These effects, known as "programming," are a significant risk factor for developing chronic illnesses in adulthood.(1) ? One of the first studies to highlight the fetal origins of disease was a population study in Hertfordshire demonstrating a link between low birth weight and weight at 1 year of age and increased death rate from ischemic heart disease, impaired glucose tolerance and type 2 diabetes.(1) ? This led to the "Thrifty Phenotype" hypothesis, where poor nutrition in utero led to fetal adaptations that produced permanent changes in insulin and glucose metabolism, increasing the risk of developing the metabolic syndrome in adulthood.(2) ? This phenomenon perturbs the established process of fetal fat distribution and contributes to an accumulation of visceral fat, which is linked to insulin resistance, heightened lipolytic activity, decreased levels of adiponectin, resistance to leptin, and elevated inflammatory cytokines. These alterations have been reported as a significant risk factor for adult noncommunicable diseases, including adult-attained height, obesity and metabolic syndrome, type 2 diabetes, cardiovascular disease, and unfavourable reproductive outcomes. Maternal protein restriction during gestation has previously been shown to result in low birth weight of the offspring and impaired development of organs such as the pancreas and kidney leading to impaired glucose tolerance and insulin resistance in peripheral tissues.(3) ? Obesity is a primary pathophysiological mechanism in the development of metabolic syndrome (MetS), a (CVD) risk factors that include dyslipidemia, hypertension, and insulin resistance. Atherosclerosis is the leading cause of CCVD, and numerous studies in recent decades have demonstrated that atherosclerosis begins in childhood. Currently, low birth weight, the reflex of restriction growth intrauterine, is recognized as an independent risk factor for cardiovascular in adulthood. Although the main focus of the field of 'developmental origins of adult health and disease' has been on the effects of poor foetal nutrition, the issue of maternal and hence foetal overnutrition is of growing importance in the context of the current global obesity epidemic. When exposed to inadequate nourishment during the early stages of life, the fetus and young newborns typically exhibit compromised growth in terms of length and weight, while placing a greater emphasis on the development of the brain. It has been argued that an increase in foetal nutrient supply, as a consequence of an increase in maternal energy intake or resulting from maternal glucose intolerance, may result in an increased risk of childhood and later obesity. Fetal malnutrition during the later stages of gestation has been found to potentially increase the likelihood of developing central obesity and associated metabolic alterations. ? During dietary restriction, the growing fetus develops a variety of strategies to boost its chances of survival postnatally in comparable nutritionally deprived conditions. Girls with restricted intrauterine growth showed higher basal levels of gonadotropin-releasing hormone, follicle-stimulating hormone, luteinizing hormone, and estradiol than girls who were born healthy. Furthermore, maternal high fat diet during the preconceptional period and/or throughout pregnancy and lactation has also been shown to result in a similar obesity phenotype in the offspring independent of postnatal nutrition. The precise mechanisms underlying these relationships have yet to be elucidated, exposure to inadequate nutrition during intrauterine development make alteration in gene expression like thrifty phenotype which may result in permanent alteration. Another possibility is alteration in hypothalamic pituitary adrenal axis mediate associations between birth weight and subsequent obesity and adipose tissue distribution. These alterations have an impact on a number of reproductive processes, including oocyte

maturation, ovulation, embryo implantation, placental development, and male fertility. Epidemiological data indicate that maternal obesity is linked to offspring obesity, and a child's body mass index (BMI) correlates with that of the mother. Adult-attained height has been associated with a variety of adult outcomes, including health and disease, cognitive abilities, and earning capacity. Research has demonstrated that people with comparable genetic backgrounds, but exposed to varying dietary environments, may exhibit disparities in adult height. Fetal malnutrition may have an effect on ovarian capacity through its impact on follicle production, possibly causing a decrease in the initial follicle pool or an accelerated follicular loss later. The available research consistently demonstrates a robust association between inadequate nutrition during early life and reduced adult height. Adipose tissue synthesizes and releases peptides, hormones, and adipokines, which have many roles. According to earlier investigations, both animal and human infants that underwent fetal nutrition deficiency showed epigenomic changes, specifically in the form of DNA methylation. After reviewing cohort studies that came to the conclusion that early-life nutritional deprivation was associated with lower birthweight in babies born to mothers who themselves had been stunted as children. Moreover, adult height appears to be an accurate indicator of early-life exposures and is frequently affected by early-life nutrition. In summary, there is strong evidence supporting the benefits of a healthy diet for pregnant women, exclusive and long breastfeeding, and weaning for long-term cardiovascular health. Moreover, recent research has emphasized the significance of elevated liver fat in the development of type 2 diabetes (T2D) among individuals with low birth weight (LBW). The many adaptations that occur within the uterus can provide benefits during fetal development but can have negative consequences when the newborn is exposed to situations that are different from those experienced during gestation. Malnutritional (excess or failure) during this period might cause irreversible structural changes in developing tissues and organs. Early-life nutrition, or the first 1000 days from conception to the age of two, can have a significant and lasting impact on the development of tissues and organs. These recent studies highlight the profound impact that dietary interventions during pregnancy could have on the long-term health of the offspring.(3) ? There is a correlation between short stature in mothers and obesity, hypertension, and stunted offspring. A substantial number of epidemiological research have found a link between birth weight and adult body mass index as a result of intrauterine growth restriction. Recent research has indicated that a healthy diet throughout infancy may be essential for preventing obesity. CVDs are a group of disorders of the heart and blood vessels including mainly coronary heart disease and cerebrovascular disease. LBW is associated with increased mortality from coronary heart disease and a higher risk of hypertension. The first postnatal feeding must be breastfeeding, sustained and exclusive because it has many benefits for health that last throughout life. Diet is a modifiable risk factor and has a large impact on the reduction of CVD morbidity and mortality. Obesity length and age of beginning (early obesity), according to new research, may provide additional insight into the contribution of obesity to T2DM risk. These adaptations include prioritizing brain growth over other body tissues like skeletal muscle and the endocrine pancreas. Another benefit of breastfeeding is that breastfed infants have a distinct growth pattern than formula-fed infants, who are more likely to experience rapid catch-up growth. Early-life malnutrition has the potential to cause fetal developmental modifications, which can lead to changes in the development

of the reproductive system and impairments in reproductive function. Early malnutrition may affect epigenetic mechanisms, specifically the hypothalamus gene expression, which is essential for the development of reproductive function. Studies have found indications that early exposure to starvation may damage females' ability to procreate later in life. According to some reports, spontaneous abortion is more influenced by hereditary variables than stillbirth. These studies imply that early life nutrition may be crucial for human reproduction and may have an impact on future generations. Although the precise mechanisms are still unknown, epigenetic processes have emerged as a possible involvement. Thus both undernutrition, and maternal obesity have been shown to increase the risk of obesity in offspring. (2)

Also, although inconsistently, a correlation has been postulated between adult height and mortality. A substantial number of epidemiological research have found a link between birth weight and adult body mass index as a result of intrauterine growth restriction. Overeating in pregnancy causing excess weight gain in the fetus is also associated with CVD in the future. What is promoted to adults as a healthy diet should start here: fruits and vegetables daily, little salt and sugar, and avoiding processed foods. Type 2 diabetes is considered a pandemic worldwide, and its prevalence is increasing. Diabetes affects or will affect one-tenth of the world's population, according to estimates. Obesity has been highlighted as a significant cause of the rise in type 2 diabetes prevalence. It is widely believed that genes and lifestyle are the key risk factors for diabetes. There is growing evidence that early life nutrition may play an important role in determining diabetes susceptibility in adulthood. Another adaptation is to train the metabolism in such a way that it promotes the storage of nutrients when they are available. All this suggests that the risk for the development of diabetes consequent to early-life nutrition can be broken by improving nutrition during pregnancy and in early childhood. Rapid growth, development, and maturity of tissues and organs characterize the first 1000 days of life. Interventions to avoid adult chronic diseases must prioritize nutrition in early life. According to certain research, early exposure to famine has a long-term effect on an adult's anthropometric measurements. Impact of maternal overnutrition on development of systems regulating body fat composition. Childhood obesity often persists throughout adulthood. Thus, early obesity prevention is crucial. Under steady diets, intrauterine growth restriction favors fat deposition. According to the WHO, more than four out of five CVD deaths are due to heart attacks and strokes. The amount of sodium in the diet of an infant in the first 6 months of life may have an impact on cardiovascular health outcomes in later life and human milk may prevent these effects. The formation of healthy eating habits should begin with maternal nutrition during pregnancy. These factors collectively play a role in the pathophysiology of diabetes. **CONCLUSION** There is substantial evidence that early life nutrition influences several adult health outcomes. **OBESITY AND METABOLIC SYNDROME**

Breastfeeding is associated with lower blood pressure at 3 years of age and this probably has repercussions in adult life. It is well known that dietary habits are established in childhood and that they are difficult to change. A classic study clearly showed this: mothers who had the habit of eating carrots during pregnancy had children who accepted carrot baby food better, when compared to other babies whose mothers did not have the habit of eating this vegetable in pregnancy. Another important phase in forming healthy eating habits in the first 1000 days of life is weaning. Children with a low birth weight are more likely to develop T2DM, particularly if they experience accelerated catch-up growth in early

childhood. Changes to these mechanisms can disrupt metabolism and fat storage. ADULT ATTAINED HEIGHT ? CARDIOVASCULAR DISEASES ? This includes the protective effect of CVD. There are some advantages also for the breastfeeding mother. Recent studies have shown that the foods eaten by the mother during pregnancy influence her child's taste after birth. Nutrition during the first few months of life may also be related to the risk of type 2 diabetes in adults. REPRODUCTIVE OUTCOMES ? Early-life nutrition may have an impact on a person's ability to reproduce. Another effect of poor early nutrition appears to be an earlier menopause. INTRODUCTION ? Obesity begins in childhood for most ? (adults. TYPE 2 DIABETES ???? (4) ???? (6) ???? (7) ??? (8