4 Early Life Nutrition and Non Communicable Disease

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Abstract

The origin of some non communicable disease (NCDs) is in early life. Evidence has shown that early life nutrition is associated with the risk of developing chronic non communicable diseases. Pregnancy and infancy are the most critical stages that influence the risks of NCDs in childhood and adult life. Prenatal maternal undernutrition and low birth weight lead to obesity and increase the risk factors of cardiovascular disease and diabetes later in life. Nutrition is one of the easily modifiable environmental factors that may affect outcome of pregnancy, trajectory of growth, and immune system of the fetus and infant. Healthy eating behaviors associate with prevention of weight disorders in pediatric, non communicable diseases, and deficiencies of micronutrient.

Keywords

Nutrition · Pregnancy · Infancy · Non communicable disease

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4.1 Introduction

Early life nutrition is associated with the risk of developing chronic non communicable diseases (NCDs). Diseases that are non-infectious and non-transmissible are considered as NCDs. According to the World Health Organization (WHO) classification, cardiovascular diseases (CVD), diabetes, chronic respiratory diseases, and malignancy are major disease types of NCDs. The developmental origins of health and disease (DOHaD) hypothesis suggested that nutrition in early life (fetal, neonatal, and infantile periods) and prenatal nutritional environments associated with the risk of developing NCDs in adulthood [1, 2].

Pregnancy and infancy are the most critical stages that influence the risks of NCDs in childhood and adult life. Nutrition is one of the easily modifiable environmental factors that may affect outcome of pregnancy, trajectory of growth, and immune system of the fetus and infant. Nutritional exposures during critical time periods including preconception and the postnatal development correlated to the health of fetus and infant. Thus, nutritional recommendations are important in pregnancy and infancy [3]. Healthy eating behaviors associate with prevention of weight disorders in pediatric, non communicable diseases, and deficiencies of micronutrient [4]. Human studies reported that low birth weight correlated with increased risk of NCDs including type II diabetes,

R. Kelishadi (ed.), *Primordial Prevention of Non Communicable Disease*, Advances in Experimental Medicine and Biology 1121, https://doi.org/10.1007/978-3-030-10616-4_4

obesity, and CVD in later life [5, 6]. Animal studies showed that high fat or low protein maternal diet associated with developing cardio-metabolic disease in offspring [7, 8].

Nutrition during the early months after birth is provided by breastfeeding or formula milk.

Some immune-mediated disorders, obesity and cognitive function disorders are lower in breastfed babies [9, 10].

4.2 Nutrition In Utero and Risk of NCDs in Later Life

Age-associated disease has increased rapidly in developing and developed countries. There is strong correlation between maternal nutrition during pregnancy and increased prevalence of age-associated disease such as CVD, type II diabetes and obesity [11].

Studies showed that offspring of pregnant women during the famine had a low birth weight and were more predisposed to NCDs including CVD, weight disorders, glucose intolerant, hypertension, dyslipidemia, blood coagulation disorders, metabolic and allergic disease in childhood and later life. The time of exposure to the famine associates with the type of disorders in later life. Famine in early gestation increases the risk of CVD, dyslipidemia and obesity. Famine during mid-gestation increases microalbuminurea and renal function disorders. Famine in late-gestation enhances the risk of type II diabetes [12, 13].

Gestational diabetes or maternal obesity has been linked to increased risk of developing the metabolic syndrome and obesity in childhood and later life, premature mortality and coronary heart disease risk in the offspring [14].

Findings showed that Overweight or obese pregnant women had more overweight children than normal weight pregnant women. Body mass index of women at the start of pregnancy is a strong predictor of obesity risk of their offspring in adulthood [15].

Micronutrient deficiencies during infancy and pregnancy are another factor that correlates with healthy outcome in later life. Under-nutrition and vitamin A and zinc deficiencies in infancy associated with deaths of children fewer than 5 years of age. Malnutrition including both under-nutrition and over-nutrition leads to micronutrient deficiencies and NCDs [16].

4.3 Maternal Macronutrient Intake and Its Influence on Appetite and Food Preferences in the Offspring

Appetite and food preferences in the offspring may develop during fetal development. Studies showed that maternal diet during pregnancy was correlated with childhood intake. Protein, fat and carbohydrate intake were assessed during pregnancy and results showed that intake of same nutrients particularly protein and fat of 10 years old children were most strongly related to nutrition in pregnancy. Carbohydrate intake in pregnancy has been related to epigenetic markers changes at birth and childhood [17].

Dutch famine cohort found that the famine during early gestation led to more consumption of high-fat diet by offspring in later life [18].

Evidence has also shown that alterations in the nutritional environment during pregnancy can alter appetite and food preferences in offspring. For example, maternal protein intake reduction can lead to preference for high fat foods in offspring. Maternal under-nutrition can associate with persistent hyperphagia in offspring in later life [19].

4.4 Early Life Nutrition and Allergic Disease

Early life nutrition influences on the developing immune system. Several immune system diseases are inherited. However, genetic factors cannot justify the increase of immune system disorders in recent years. Some maternal nutritional changes including decrease intake of omega 3 polyunsaturated fatty acids, folate and zinc have been correlated with alteration in immune system [20].

A prospective study showed that increased risk of allergies especially eczema in infancy

may be related to highly intake of margarine, vegetable oils and some fruits and vegetables including celery, citrus fruit, and sweet peppers during pregnancy [21].

Other nutrients such as long-chain polyunsaturated fatty acids, prebiotics, probiotics and antioxidants including selenium, zinc, vitamin A, vitamin C, vitamin D and vitamin E have been associated with asthma and allergic disease [22].

Findings showed that antioxidants improved immune system function. According to observational studies, consumption of antioxidant-rich foods including fresh fruit and vegetables and higher antioxidant levels in pregnancy may decrease the risk of wheezing, asthma and eczema in the offspring [23]. However, there are controversial findings [24].

The maternal gut microbial environment is another factor for allergy protection in the offspring. Healthy immune system development needs balance of specific gut microorganisms. Infants with allergic disorders have unhealthy balance of microorganisms in the gut. According to animal studies, gut microorganisms regulate immune system development and decrease allergic disorder, obesity and cardio-metabolic disorders. Soluble prebiotic fiber including oligosaccharides is associated with improvement immune system and metabolic outcome [25].

Omega 3 have anti-inflammatory effects on immune and metabolic outcomes. Some clinical trials showed that fish oil supplementation during pregnancy had immune-modulatory effect and decreased allergic disease outcomes in the offspring. Another, have reported useful effects of omega 3 intake in early life on cardio-metabolic risk factors [26].

Australian cohort study showed significantly association between lower cord blood vitamin D levels and eczema at 1 year of age [27].

4.5 Nutrition in Early Life and Diabetes

One of the most common metabolic diseases is diabetes. Its prevalence is increasing recently. Changes in lifestyle related to urbanization in developing countries lead to increase the risk factors of NCDs including type II diabetes. Genetic and environmental factors are the cause of disease susceptibility. According to human cohorts and experimental animal study, there is association between early life nutritional environment and risk of cardio-metabolic disorders in offspring. Developmental programming is called for this process. It has shown maternal and paternal nutrition play a key role in metabolic programming of the offspring. Potential mechanisms for programming of type II diabetes are not clear. Some components of type II diabetes are correlated to epigenetic dysregulation. Transgenerational transmission of type II diabetes risk is related to eating behavior change and secretion and action of insulin [28].

Human and experimental evidence reported that early life nutrition especially during fetal life and early infancy affect the risk of type II diabetes. Malnourished conditions in fetal life with poor growth in utero (intrauterine growth restriction, IUGR) lead to impaired glucose and energy metabolism including increased peripheral insulin sensitivity, enhanced production of hepatic glucose, decreased insulin sensitivity for muscle protein synthesis and impaired pancreatic development [29].

Several studies showed the U-shaped association between birth weight and type II diabetes. High birth weight (>4000 g) and low birth weight (<2500 g) is associated with an increased type II diabetes risk [30, 31].

The time of solid foods introduction is one of the important nutritional period in infancy. Changes in diet composition including enhance in protein and caloric consumption occur in this period. Findings related to the time of solid foods introduction and weight disorders and glycemic profile in childhood are inconsistent [32]. In addition, there is less studies that investigate the relationship between the time of solid food introduction and glycemic profile in childhood. The difference in the findings is due to various definition of early food introduction; assess outcomes in different stages of childhood and not considering main confounding factors [33].

Study on pregnant women with gestational diabetes mellitus (GDM) showed that prolong breastfeeding was correlated with better glycemic profile and lower A1C levels during childhood. These results highlighted the importance of longer duration of feeding with breast milk for pregnant women with GDM [34].

Breast milk contains high polyunsaturated fatty acids that promote development of brain insulin receptors and lead to lower type II diabetes in later life. Study on Canadian pregnant women with GDM suggested that breastfeeding more than 8 months was associated to lower A1C levels compare to shorter time of breastfeeding [34]. Another study showed that exclusively breastfeeding more than 2 months led to less development of type II diabetes at the age of 10–39 years compare to infants without breastfeeding. The reasons of heterogeneity in findings are various study design and studied populations. Thus, more studies for assessment the role of breastfeeding duration on glycemic profile in childhood and adulthood are needed. In addition, more studies are needed to find new strategies for prevention of childhood obesity in children of pregnant women with GDM [34].

4.6 Nutrition in Early Life and Non-alcoholic Fatty Liver Disease (NAFLD)

According to human study, growth restriction in early life and insufficient nutrient supply for the fetus lead to development of liver disease in later life. Study on women aged 60–79 years showed that there was a relationship between low birth weight and enhanced liver enzymes alanine aminotransferase (ALT), gamma glutamyltransferase (GGT) and hepatic cellular injury [35]. Findings of a case control study reported an association between NAFLD in children and adolescents and IUGR. Low birth weight was associated with high prevalence of nonalcoholic steatohepatitis (NASH). According to evidence, rapid growth pattern after early growth restriction and macronutrient restriction is correlated with NAFLD risk [36].

Animal study showed that restriction of dietary protein during pregnancy and lactation cause to offspring hepatic steatosis and hepatic lipid accumulation in late adulthood. In maternal undernutrition, deposition of hepatic fat happens in fetuses faster than development of offspring adiposity. Thus, it can be concluded that growth restriction, obesity, high fat diets intake, and undernutrition during the critical early life lead to susceptibility and severity of NAFLD [36].

Evidence reports that one way for protection against NAFLD development, progression of NASH and liver fibrosis is early breastfeeding. Longer duration of breastfeeding leads to decrease the risk of obesity and liver disorders in later life [37].

Breast milk is a rich source of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). These long chain polyunsaturated fatty acids (PUFSs) can suppress lipogenesis and liver fibrogenesis. Animal study reported that fish oil that rich in PUFAs reduced hepatic steatosis, lipogenesis and increased lipid oxidation [38].

Several peptides including insulin and leptin are present in breast milk. These peptides affect infant growth and body composition. Findings showed that leptin intervention during the neonatal period led to decrease metabolic disorders and progression of hepatic steatosis [39].

Several fruits including red grapes contain naturally resveratrol. Resveratrol has antioxidant and anti-inflammatory effects. It decreases liver steatosis and dyslipidemia and has useful influence on mitochondrial oxidative stress. Intake of fruits in early life can prevent liver disorders in later life [40].

Another item that has anti-inflammatory properties is taurine. It is a sulfonic amino acid. Studies showed that taurine supplementation during pregnancy and lactation can decrease proinflammatory hepatic profile in children. However, more studies are needed to confirm safety of taurine supplementation during pregnancy and lactation [36].

4.7 Early-Life Nutritional Status and Metabolic Syndrome

Metabolic syndrome (MetS) is defined as some CVD risk factors including obesity, dyslipidaemia, hypertension and high blood glucose. Nutrient restriction in the uterus lead to undesirable changes in organ function and enhanced the risk of CVD in later life and adulthood. Low birth weight is one marker for nutritional deficiency in fetal life. It is associated with MetS in adulthood [41]. Findings showed that nutritional deprivation in utero and incidence of disorders in later life differ according to gender. However, most studies did not stratify analysis by gender [42].

Finding of a meta-analysis demonstrated that low birth weight led to 2.4-fold increase in MetS in adulthood. Previously, this association has been shown only in men [43].

4.8 Early Life Nutrition and Cancer Risk

Evidence shows that some cancers such as breast cancer originate in early life. Epidemiological studies reported that environment factors including nutrition in early life associated with breast cancer risk in later life. According to animal studies, both under-nutrition and over-nutrition influence the risk of cancer susceptibility in children. Early life environmental factors can alter epigenome and affect cancer risk. Nutrition in early life as one of the environmental factor leads to persistent epigenetic changes, alteration in mammary gland development and finally increases susceptibility to breast cancer [44].

Animal study showed that low birth weight and protein restriction during pregnancy and lactation associated with increase in the expression of the insulin and estrogen receptor and more incidences of mammary tumors in later life of children. Risk of mammary tumorigenesis enhance in later life by over-nutrition in early life. High fat diets during pregnancy lead to high birth weight and increase mammary tumors in adulthood [45].

According to animal studies, diet high in n-6 PUFA during the peripubertal period led to more incidence of mammary tumor than eating high n-6 PUFA diet during post puberty. It is suggested that nutrition during peripubertal period associate with susceptibility of cancer risk. However, n-3 PUFA intake during peripubertal period decreases mammary tumourgenesis, mammary cell proliferation and increases apoptosis [44].

According to animal studies, some micronutrient intake in early life influences the cancer risk in later life. Finding of some epidemiological studies showed an inverse association between dietary folate intake and cancer risk [46].

However, another study reported that folic acid supplementation more than 400 μg/day increased the risk of breast cancer. Folic acid consumption is increasing because of food fortification, use of supplement and periconceptional folic acid supplementation for the prevention of neural tube defects. Thus, influence of folic acid supplementation in early life on cancer risk in later life must be assessed [47].

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