

Early-life nutrition may have an impact on a person's ability to reproduce. These findings support the thrifty phenotype theory of reproductive aging and contribute to a better understanding of the causes of early menopause. Early-life malnutrition can have an impact on the methylation pattern of peroxisome proliferator-activated receptors (PPARs), which are highly expressed in reproductive tissues and play a significant role in the transformation from embryo to fetus in developing mammals. After reviewing cohort studies from Brazil, Guatemala, India, the Philippines, and South Africa that involved long-term child monitoring, the Maternal and Child Undernutrition Study Group 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. 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. These alterations have an impact on a number of reproductive processes, including oocyte maturation, ovulation, embryo implantation, placental development, and male fertility. Early stages of development are when primordial germ cells are created, and the fetal era is when follicle reserves are exclusively established. 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. According to a population-based ecological study conducted with 58,601 girls born between 1959 and 1961, the Great Chinese Famine, early exposure to the famine was linked to an increased chance of stillbirth. According to earlier investigations, both animal and human infants that underwent fetal nutrition deficiency showed epigenomic changes, specifically in the form of DNA methylation. According to some reports, spontaneous abortion is more influenced by hereditary variables than stillbirth, which has been shown to be more responsive to environmental circumstances. The malfunctioning of the hypothalamic-pituitary-gonadal axis may also be influenced by early undernutrition. Compared to women who were not exposed, women who were born during the Chinese famine (1956–1964) who were subjected to prenatal starvation had a greater incidence of early menopause. The authors evaluated 11 studies and confirmed that fetal famine exposure was strongly related to a younger age at menopause. 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. The risk of future perinatal death was similarly elevated in women who were exposed to the great Chinese famine during the third trimester of pregnancy. These studies imply that early life nutrition may be crucial for human reproduction and may have an impact on future generations. One of the worst famines in recent memory, the Great Chinese Famine of 1959–1961 is thought to have killed an additional 30 million people and caused 33 million miscarriages. Similar outcomes were noted in Dutch women who had been subjected to starvation during pregnancy. A systematic analysis evaluated whether poor early life factors result in menopause at a younger age. Women go through menopause when their follicular reserve runs out during the course of their lifetime. This could have generational-spanning consequences on the health of the reproductive

.system. Another effect of poor early nutrition appears to be an earlier menopause