DEVELOPMENTAL ORIGIN OF HEALTH AND DISEASE

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This article explores how early-life nutrition, fetal growth, and early developmental conditions influence long-term health outcomes, a framework known as the Developmental Origins of Health and Disease (DOHaD). Historical cohort studies, including those from the Hertfordshire cohort consistently show that poor fetal nutrition and low birth weight increase the risk of chronic conditions such as hypertension, coronary heart disease, impaired cognition, and mental health disorders later in life. Evidence from animal and human studies demonstrates that undernutrition during critical windows of gestation can permanently alter organ structure, metabolism, and hormonal regulation, resulting in lifelong disease susceptibility. It also highlights the burden of chronic diseases—particularly COPD—in low-income regions, where limited awareness, inadequate healthcare resources, and high exposure to indoor air pollution elevate risk. Overall, the findings underscore that birth weight is a strong marker of future health, emphasizing the importance of maternal nutrition, prenatal care, and improved public health systems to reduce chronic disease burden across generations.

Introduction

Human health, often defined as the absence of disease, is determined by a number of factors related to diet, environment, and economics, as well as country of residence and educational attainment. The confluence of these areas highlights the interplay of both

biological and socioeconomic factors that ultimately allow for normal developmental processes to occur and optimal health during the life of a human. Over the past 30 years, substantial attention has been given to the influence of nutrition in utero and during critical periods of growth on health in adulthood, an area termed the “fetal origins hypothesis” or “fetal programming”; it was later modified to the “developmental origins of health and disease” (DOHaD) to better reflect both the gestational and postnatal periods. Beginning with the work of Barker and Osmond in 1986,(1) it was documented that chronic diseases commonly associated with higher income had become more prevalent in lower-income regions of England and Wales. Thereafter, a number of studies were conducted using the Hertfordshire birth cohort, which included more than 15 000 babies for whom birth weight and early feeding practices were documented in the early part of the 20th century.(2) From this cohort, it was shown that children born small are at a higher risk for developing type 2 diabetes (T2D), hypertension, and coronary heart disease.(2–4)Similar results have been published using birth cohorts from South Africa, Finland, the United States, Brazil, and China, among others.(5–9) However, a consistent challenge to human cohort studies has been the elucidation of clear physiological or environmental mechanisms behind the findings.

In China, adults who were exposed in utero to the Chinese famine of 1958 had a 50% increased risk for T2D compared with those not exposed to famine.(12) It was also reported that adults exposed to the famine in utero were almost 4 times more likely to have hypertension than those who had postnatal exposure to the famine.

Finally, in terms of mortality, Utah pioneers whose mothers were pregnant during periods of severe food shortages in the mid-19th century were found to have had a higher risk for mortality compared with those whose mothers were not exposed, but the effect was most consistent for men.(15)

A number of studies have found a link between poor nutrition early in life and mental health and cognitive development. For example, prenatal exposure to the Chinese famine from 1959 to 1961 nearly doubled the risk of schizophrenia for those in the 1960 exposure group compared with those born after the famine (16); these results are strikingly similar to the relative risk of 2.7 for schizophrenia found in the Dutch famine cohort.(19) Studies from different countries have also found that poor nutrition in childhood has a negative impact on cognition. In Peru, stunted children (defined as height-for-age Z score [HAZ] < -2.0) scored significantly lower ($P < 0.05$) on a series of cognitive tests compared with taller children.(17) Children who experienced catch-up growth (indicating a positive change in HAZ) had cognitive scores similar to those of children who remained stunted.(20) However, a study on growth and cognitive performance that included more than 8000 children from 4 developing countries found that children who recovered height from age 1 year to age 8 years performed poorly compared with children who were never stunted but scored better

than children who remained stunted (21); this suggests that timing of nutritional interventions is vital to improving human capital. The point is made even more clear by a study of stunted children in Jamaica who received psychosocial stimulation and scored markedly higher on IQ, verbal, and reading tests compared with stunted children who did not receive such stimulation.(18) Recently, as some of the children from this cohort are now parents, it was reported that offspring of stunted parents scored lower on a battery of cognition tests, independent of birth weight and height-for-age,(22) but it is not clear if the effects are related to social or biological factors. Furthermore, stunted children in India who received nutritional supplementation for 6 months had cognitive test scores similar to those of children who remained stunted, as well as those who recovered height.(23) Although the degree of stunting at age 2 years has been shown to be related to consistent and long-term cognitive deficits, such deficits in a cohort from Cebu, Philippines, declined by age 11 years.(24) The studies reviewed herein indicate that the timing of interventions is clearly critical to the overall impact of nutrition on brain development during gestation and childhood, an area of research that warrants much greater attention to improve human capital throughout the world, but especially in lower-income countries.

Origin of fetal hypothesis

Fetal hypothesis is due to poor nutrition in the middle to late pregnancy and this will lead to poor fetal growth. Gradually, it will slow down the growth of the fetus and later, it will increase the risk of coronary heart disease.

Based on the animal studies, we can say that short periods of undernutrition during pregnancy can permanently change the blood pressure, cholesterol, metabolism and immune function. Due to poor nutrition of mother, babies become so small and they birth with more heart problem. During the first eight weeks after conception (embryonic life), the embryo will form basic structures but it is still tiny due to disproportionate fetal growth. Lack of nutrients and anoxia are the reason forundernutritionand this lead to slow cell division. Undernutrition during critical period will cause permanent effects because different organs grow at different times.

Researchers Widdowson and McCance showed that short periods of poor nutrition can permanently change the number of cells in organs. Earlyundernutrition can lead to change in hormonal secretion, altered metabolism and change in organ structure. These will become memories in our body; that increase disease risklater.

If the baby is birth with small size and disproportionate size of organs. it indicatepoor fetal nourishment. These adaptations may help the fetussurvive during pregnancy, but may increase disease risk later. From epidemiological studies; Hertfordshire found that babies weighing less than 5.5Ib(2500g) had the highest rates of coronary heart disease. Babies weighing 9.5Ib (4310g) had the lowerrisk.

Chronic disease in low income regions

Chronic obstructive pulmonary disease (COPD) is the more common disease in many low and middle income countries. Even though this disease is more common in these regions;yet it remains largely neglecteddisease. In places such as sub-Saharan Africa, especially in rural areas, most of the people don't know what actually COPD is, there is only little awareness among that community.

This lack of knowledge affects their public health planning; because governments and heath systems have not developed any measures to identify, prevent or to manageCOPD.

Many clinics do not have a trainedstaff, inhaled medicines. The main treatment for COPD are either unavailable or it is very expensive for common people. Due to this, patients often remain undiagnosed, untreated or poorly managed.

Tobacco smoking is not only the reason for COPD in Sub-Saharan Africa. Household air pollution is a significant cause. They are using firewood, charcoalor other solid fuels for cooking. This will produce large amount of smoke everywhere. This altogether increases the risk of COPD.

Hypothesis linking birth weight with later disease risk

If you were a slightly heavier baby; your brain will be bit larger and this advantage stay even in the old age. For example; people who had higher birth weight had slightly larger brain volumes at age 73.

Birth weight do notaffect ageing- related brain changes, like cortical thickness, diffusion MRI ageing markers, white matter hyperintensities (WMH). Birth weight reflects fetal development. Better fetal growth will help to acquirebetter cognitive ability in the childhood and less risk of dementia later. So birth weight is a marker of how well the brain developed before birth.

According to HUNT study; low birth weight may be partly genetic which means; families withcertain genes may have both small babies and higher heart disease risk.

According to colarado study; women born premature and with low birth weight are more likely to developpreeclampsia when they become mothers.

Result

- Babies with

a) low birth weight (≤2500g) → 40% had diabetes/glucose problems.

b) High birth weight (>4310g) → Only 14% had diabetes.

- Very low birth weight babies (under 4.5 lb) had 5 times higher risk later, Babies with higher birth weight had larger volumes, better thinking skills in childhoodand lower chance of dementia in old age.
- People who were born with 10w birth weight and higherrisk of heart disease, diabetes, high blood pressure, and poor metabolism as adults.

- In low-income settings, COPD remains common due to lack of awareness, inadequate healthcare, high smoking rates and exposure to indoorair pollution from firewood and solid fuels.
- Women who were born with low birth weight or born premature had a greater chance of developing preeclampsiaduring their own of pregnancies.

Conclusion

I would like to conclude that birth weight is a strongermarker of future health. Therefore it is necessary to focus on prenatal health through regular checkups, a nutritious diet, and avoidingharmful substances like alcohol and tobacco. Fetalundernutrition causes permanent biological changes, including altered organ size, hormone levels and metabolism. Baby with betterfetalgrowth can reduce the risk of chronic diseases.Proper awareness, trained high quality health care staffs, affordable treatments are necessary for low-income regions. Other wise diseases like COPD remains as a neglected disease.

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