Building Community Wellness
The Fetal Experience and Later Health
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It is said that the egg that formed you was at its most vulnerable when your mother was an early fetus. That vulnerability to toxic exposure/irradiation occurred as the primordial cells were traveling to their eventual berth in the ovaries. This is just one indication that the diseases that we may experience as an adult were prompted by events of long ago. Thus, we should examine these long-range effects as we seek to build a healthier community.

The most vulnerable time for brain development is during the embryonic and early fetal period. Brain growth utilizes 70% of fetal energy utilization and an estimated 80-90% of cognitive performance is determined prenatally. Lifelong metabolic and physiologic status is affected by transplacental nutrition. Organ development depends upon a complex sequence of signaling proteins targeting a specific site at a specific time, with little latitude. Any perturbation is likely to have significant effects. There is a substantial fetal loss in very early pregnancy, often before pregnancy is even recognized. More subtle damage may not appear at birth and sometimes, not until adult life. Understanding these processes allows communities and cultures the opportunity to make strategic investments for long-term gains, if there is the will.

The inter-generational effects on health can be categorized into maternal infection and immune response, maternal nutrition, and toxic exposures. These categories will be discussed separately.

Maternal Infection and Immune Response

The fetus can be considered a foreign substance in the mother's body that escapes rejection. The point of contact, the trophoblast, which forms the amniotic membranes, is imbedded in the uterine wall. Since its genetic material differs from the mother, it is vulnerable to conditions that enhance the mother's immune response. Viral infections, bacterial vaginosis and gingivitis are known to increase maternal cytokines, which are the signaling proteins that direct inflammatory cells and also interact with complex mechanisms that determine cell growth and death. There appears to be racial differences in the degree of cytokine response to maternal infection. A study in North Carolina by Beck and associates showed an exaggerated cytokine response to gingivitis and bacterial vaginosis in black, compared with white pregnant women. Increased levels of cytokines have been associated with preterm birth and intrauterine growth retardation. Could this be one factor that determines racial disparities in birth outcomes?

It is also possible that cytokines mediate the effects of maternal viral infection on fetal brain development. In addition, viral infection may unleash an inflammatory process within the fetal brain that can damage neurons and predispose to later

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neurodegenerative disorders. In animal studies, endotoxins, released from bacterial infection in utero, resulted in later neurodegenerative disorders. There is evidence, therefore, that viral infections, bacterial infections, maternal immune response, and fetal immune response can all cause changes in the developing brain that range from minor to severe and scarring to loss of gray matter. Viral infection during early pregnancy has been linked to the development of autism spectrum disorders and schizophrenia in the offspring.

Maternal/Fetal Nutrition

Fetal nutrition may be affected by maternal infection and by placental function. Retrospective and prospective studies suggest that maternal nutritional status at the time of conception is most important to pregnancy outcomes. It is recommended that 20% of energy intact be in the form of fat. Essential fatty acids and docosahexaenoic acid (DHA) are among the nutrients required for development of the fetal brain and retinal. In preterm and low birth weight babies, DHA deficiency has been related to visual impairment and delayed cognitive development. Fetuses accumulate DHA from the mother in the last trimester. Premature infants miss out on this accumulation, as do fetuses of mothers who subsist on a maize-based diet (also deficient in vitamins A, B, and C and zinc, calcium, iron and iodine. Mothers with limited food resources, who have repeated pregnancies with short intervals (Maternal Depletion Syndrome) may be depleted of these nutrients to the degree that the fetus is affected. Dietary supplementation at the time of conception is an important intervention for these populations at risk.

Toxin Exposure

Much attention has been given to prenatal exposure to mercury, thalidomide, dilantin, coumadin, other maternal medications, and now, ACE inhibitors. Perhaps the most pervasive exposure, though, is alcohol. Ingestion of alcohol may affect the early fetus, even before the mother is aware of the pregnancy. Fetal alcohol syndrome (FAS) is associated with a multitude of abnormalities, involving many different organ systems, including:
Learning deficits, behavioral disorders, mood disorders, autism spectrum disorders, auditory processing, hearing, speech, epilepsy, Tourettes Syndrome, craniofacial defects, cardiac defects and asthma. The societal and family costs of (FAS) are high. Each person with FAS costs the taxpayer an estimated $3 million in services such as special education and costs such as incarceration. From 55 to 60% of prisoners may be affected by FAS. In addition, there are substantial costs to the family and loss of earning of the individual. Fetal alcohol syndrome contributes also to homelessness and its attendant societal ills.

Mechanisms of Disease

Barker examined data from 15,000 low birth weight babies born in the UK and found that they were at increased risk for cardiovascular and metabolic disease. Specifically,

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those infants born with low birth weight, smaller head circumference, and decreased ponderal index (skinny, not short) were at increased risk for coronary heart disease, hypertension, stroke, insulin resistance and diabetes. Those low birth weight neonates who became obese infants were at special risk for cardiovascular disease and diabetes. In Barkers study, the primary cause for this was thought to be poor maternal nutrition during WWll. Diminished availability of nutrition was thought to alter gene expression to accumulate fat reserves. Moreover, organs not essential for fetal survival (kidneys, brain) were thought to be at the “end of the lunch line”. Limited nutrition during early development may lead to altered hormone levels and/or changes in tissue sensitivity to these hormones. This phenomenon, known as the Barker Hypothesis, might be additive to genetic predisposition to cardiovascular disease or diabetes in certain populations. Since chronic inflammation may also cause fetal under-nutrition due to poor placental function, there may be a tie-in with the studies by Beck, demonstrating a relationship between maternal bacterial infection and low birth weight. This raises the question of a biologic interaction between poverty, infection, and genetic predisposition that results in health disparities between populations and indicates directions for change. Some of these positive directions include: 1) Improved oral health and nutrition in adolescent and adult women of childbearing age, 2) Education of adolescents about preconception nutrition and avoidance of toxins, and 3) Targeted studies of prenatal toxic exposures in populations at risk.

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