Vitamins for Fetal Development: Conception to Birth

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ARTICLE SUMMARY

- Good maternal nutrition during pregnancy can protect the offspring from diabetes, stroke, heart disease, kidney disease, and memory loss later in life.
- Special preconception and pregnancy diets emphasizing foods dense in particular nutrients were universal among the traditional groups that Weston Price studied.
- Modern science has shown that fat-soluble vitamins are necessary for growth and development; the omega-3 fatty acid DHA is necessary for brain development; the need for biotin during pregnancy increases; folate boosts growth and decreases the risk of birth defects; choline causes a lifelong increase in memory and attention; and the amino acid glycine is required for growth.
- WAPF recommends a dose of high-vitamin cod liver oil per day to yield 20,000 IU of vitamin A, 2,000 IU of vitamin D, and 2 grams of omega-3 fatty acids (about 1 3/4 teaspoon per day).
- Grass-fed animal fats supply vitamins E and K_2; palm oil, fresh fruits and vegetables, nuts, and freshly ground grains are also sources of vitamin E; fermented foods are also sources of vitamin K_2. Leafy greens supply vitamin K_1.
- Biotin can be obtained from liver and egg yolks. Raw egg whites should be strictly avoided and cooked egg whites should be consumed in moderation. Egg yolks can be added to smoothies and ice cream to boost biotin status.
- Folate can be obtained from liver, legumes, beets, and greens. Choline can be obtained from grass-fed dairy, egg yolks, liver, meat, cruciferous vegetables, nuts, and legumes. Figure 7 provides examples of how to meet the folate and choline requirements.
- Muscle meats and eggs should be liberally matched with the above folate-rich foods and with skin, bones, and bone broths to obtain glycine.

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Human life begins, biologically speaking, at conception. This is the moment at which the new organism possesses its own unique combination of some 20,000 genes and the moment at which it becomes capable of growth and cell division. The days and weeks following conception form a critical window within which the nutritional environment of the womb sets the tempo for fetal growth. But nothing more clearly illustrates the continuity between the new life within the womb and the adult he or she will become than the fact that the quality of nutrition during these nine months produces lifelong effects on the brain, kidneys, and the cardiovascular system; determines the risk of degenerative disease; and profoundly influences the quality of life that persists through adulthood and into old age.

Every human cell within a given human being has an identical set of genes, called a genome. These genes come in pairs: one copy from the mother and one from the father. Sperm and egg cells are the exception to the rule and carry only one from each pair. By shuffling the maternal and paternal genes into different combinations to produce each sperm or egg, a single man or woman could theoretically generate an incomprehensibly greater number of combinations than the number of atoms that make up the known universe, thus ensuring that no two human beings on the face of the earth—save identical twins—will carry the exact same genome.[1]

EMBRYONIC AND FETAL GROWTH

During ovulation, an egg is released from the ovary and moves into a tube called the oviduct. At conception, the sperm and egg combine within the oviduct to form a zygote. The genes from each parent combine into pairs and the zygote possesses a new, unique genome that every cell into which it divides will inherit.

Over the course of the first seven days, the zygote divides into a hollow ball of cells as it moves through the oviduct toward the uterus. By the seventh day, it becomes embedded in the uterine wall. At this point it is called an embryo. The heart of the embryo heart by day 23; its brainwaves can be recorded at day 40; in the seventh week it begins sucking, touching its face, hiccupping, frowning and making various other spontaneous movements.

At eight weeks, all organ systems are present and functioning at which point the growing organism is called a fetus. Out of 4,500 body structures present in the adult, 4,000 are present at this eight-week point. The fetus has a firm grip, sucks its thumb, and somersaults by four months; in the beginning of the sixth month it nestles into position to sleep, stretches upon waking and can hear.[2]

At the turn of the third trimester, a dramatic increase in the likelihood that the infant will survive outside the womb if born prematurely takes place—from 15 percent at 23 weeks to 79 percent at 25 weeks.[3] In the last half of this trimester, rapid growth takes place, especially in the skeletal system. Infants born six weeks prematurely have only half the calcium and phosphorus laid down as infants carried to term.[4]

None of this growth and development can take place without nutrients. Fats and carbohydrates fuel the growth. Fats and cholesterol form cell membranes. Amino acids form structural proteins and enzymes. Vitamins and minerals act as cofactors for those enzymes or as regulators of the entire process of growth. These nutrients are uniquely supplied by the mother's diet.

PRIMITIVE PREGNANCY DIETS

The provision of special pregnancy and preconception diets to mothers-to-be was a universal characteristic of the healthy traditional groups studied by Weston Price. In some cases, these groups provided special preconception foods to fathers-to-be as well.

All groups that had access to the sea used fish eggs; milk-drinking groups used high-quality dairy from the season when grass was green and rapidly growing. Some groups used other foods such as moose thyroids or spider crabs, and African groups whose water was low in iodine used the ashes of certain plant foods to supply this element.[5] These foods were added against the backdrop of a diet rich in liver and other organ meats, bones and skin, fats, seafood and the local plant foods.

Fish eggs are especially rich in cholesterol, vitamin B_{12}, choline, selenium, calcium, magnesium, and omega-3 fatty acids. They contain a modest amount of most fat-soluble vitamins but their vitamin K_2 content is unknown (see Figure 1).[6]

The Maasai only allowed men and women to marry after spending several months consuming milk from the wet season when the grass was especially lush and the milk much denser in nutrients. Maasai milk is higher in fat and cholesterol and lower in sugar than commercial American milk. The highest quality Maasai milk used for preconception diets, however, is even richer: compared to commercial American milk, it has over twice the cholesterol, nearly three times the fat, and over five times the quantity of phospholipids (see Figure 2).[7] The phospholipid content is particularly important. Since most of the choline in milk is contained in phospholipids,[8] this means that high-quality Maasai milk is probably about five times richer in choline than the milk you would find in the grocery store.

Compared to grain-fed milk, grass-fed milk is much higher in fat-soluble vitamins, pigments, conjugated linoleic acid (CLA) and omega-3 fatty acids.[9] Price showed that the content of vitamin A, activator X (which we now believe to be vitamin K_2), and essential fatty acids markedly increased in butterfat during the rainy lush season.[10] As the quality of grass increases, we can presume that the content of other grass-related nutrients—such as pigments, vitamin E and CLA—will also markedly increase in the milk.

Although modern science still has much research to accomplish in order to fully elucidate the value of traditional wisdom, it has already confirmed the fact that many of the nutritional factors that we now recognize as the most important to healthy embryonic and fetal development are the same ones emphasized in traditional pregnancy and preconception diets.

VITAMIN E

Vitamin E was originally named “Fertility Factor X” in 1922 because rats could not reproduce without it. Two years later, researchers dubbed it “tocopherol” from the Greek τόκος (tokos), meaning “childbirth,” and φερειν (ferein), meaning “to bring forth.” Its precise role in rat fertility remains unclear and scientists have yet to conclusively demonstrate its essentiality to human reproduction.[11]

Mice lacking the gene for the protein that transports vitamin E across the placenta conceive offspring that die within 11 to 15 days. The nutritional transport system of the placenta is observably malformed by the ninth day. The human placenta makes the same protein, so the role of vitamin E in constructing the nutritional transport system of the human placenta is probably similar. Vitamin E, then—despite the lack of published proof—is almost certainly essential to human reproduction.[11]

Vegetable oils are high in vitamin E, but they are also high in polyunsaturated fatty acids (PUFA), which deplete the body of this nutrient.[12] Palm oil is a much better source, having a vitamin Eto- PUFA ratio that is 8 times higher than that of soybean oil and 13 times higher than that of safflower oil.[13] The vitamin E content of grass-fed animal fats is four times higher than that of grain-fed animal fats.[14] Nuts, seeds, fresh fruits and vegetables and freshly ground grains also contain vitamin E.

VITAMIN A

In the years following the discovery of the role of vitamin E in rat fertility, researchers quickly began to realize that vitamin A was even more important to reproduction. In *Nutrition and Physical Degeneration*, Price described the early work on vitamin A deficiency during pregnancy and the preconception period. In diverse species of laboratory animals, this deficiency produced spontaneous abortion; prolonged labor and death of the mother and her offspring during labor; eye defects including the complete absence of eyes; defects of the snout, dental arches and lips; displacement of internal organs including the kidneys, ovaries and testes; and deafness due to degeneration of the nervous system.[15]

We now know that vitamin A is necessary for the differentiation and patterning of all of the cells, tissues, and organs within the developing body. It is especially important for the development of the communication systems between the sense organs and the brain.[16,17] Even mild vitamin A deficiency compromises the number of functional units called nephrons in the kidneys, which could predispose a person to poor kidney function later in life.[18] Vitamin A is also necessary during fetal development and through adult life to maintain the presence of cells lining the lungs that are covered in hair-like projections called cilia.[19] These hairs sweep away debris and foreign material, protecting the lungs from pollutants and infectious diseases. During and after the formation of all these systems, vitamin A is necessary for their continued growth.

The RDA of vitamin A for pregnant women is 2,600 IU—just 300 IU more than the RDA for women who are not pregnant. There are several problems with this figure, described in the sidebar below. We do not have exact figures for the vitamin A content of the preconception and pregnancy diets used by the groups that Price studied, but they were certainly higher than 2,600 IU per day. These groups prized organ meats, especially liver, and used them on a regular basis. Preconception and pregnancy diets added additional foods rich in fat-soluble vitamins.

The Weston A. Price Foundation recommends 20,000 IU per day from cod liver oil and additional vitamin A from milk, butter, eggs, and three to eight ounces of liver per week. Yet the medical profession warns pregnant women that this quantity of vitamin A increases the risk of birth defects. This belief can be traced to a single study published in 1995 that purportedly found an increase in the risk of birth defects among mothers consuming more than 10,000 IU per day. As discussed in the sidebar below, there are several important flaws in this study. Every other published study on this subject shows this amount of vitamin A to be safe—indeed, one major study of over 25,000 births showed that daily doses of vitamin A up to 40,000 IU cut the risk of birth defects in half.

Considering the ubiquitous role of vitamin A in the development of every organ system of the body and how tightly the body regulates the level of the activated form, we should expect a generous helping of vitamin A-rich organs and animal fats to help perfect fetal development, not to throw it off course. The preponderance of the evidence suggests that this is the case.

VITAMIN D

In the late third trimester, the fetal skeleton enters a period of rapid growth that requires calcium, phosphorus and vitamin D. An infant born six weeks prematurely has laid down only half the calcium into its bones as an infant carried to term.28 There is evidence that vitamin D plays a role in lung development,[29] and it probably plays a much larger role in fetal development in general due to vitamin D's interaction with vitamin A. At birth, the infant's blood level of vitamin D is closely correlated to that of the mother.[30,31] Adequate levels of vitamin D protect the newborn from tetany, convulsions and heart failure.[29]

The rapid skeletal growth that occurs in late pregnancy taxes the vitamin D supply of the mother and her blood levels drop over the course of the third trimester. One study conducted in Britain showed that 36 percent of new mothers and 32 percent of newborn infants had no detectable vitamin D in their blood at all; another showed that 60 percent of infants born to white mothers in the spring and summer had levels under 8 nanograms per milliliter (ng/mL), a level that is overtly deficient.[32]

In 1963, the American Academy of Pediatrics acknowledged the increased need for vitamin D during the third trimester of pregnancy. The Academy lamented the lack of data elucidating the precise amount of this need and suggested that 400 IU per day would cover the requirements of mother and fetus.[28]

In 1997, however, the Institute of Medicine declared that the transfer of vitamin D from the mother to the fetus is so small that the mother's vitamin D status is not affected. Citing a 1978 study showing that the average vitamin D level of pregnant women consuming small amounts of vitamin D at high latitudes was 9.1 ng/mL (25 percent under the level required to protect against overt deficiency) the Institute concluded that "there is no additional need to increase the vitamin D age-related [adequate intake] during pregnancy above that required for non-pregnant women." This conclusion is strange, not only because many of the mothers in this study must have had vitamin D levels below the average, but because the average level itself was already deficient. The Institute set the recommended intake at 200 IU, which it rather dubiously supposed "may actually represent an overestimate of true biological need."[33]

In 2003, the American Academy of Pediatrics' Committee on Nutrition and its Section on Breastfeeding issued a joint statement in which they overturned the 40-year position of the Academy advocating 400 IU in favor of adopting the lower so-called "overestimate" of the Institute of Medicine.[34]

In the second part of this statement, the Academy directed mothers to keep their infants out of the sun, dress them in protective clothing, and liberally cover them in sunblock. In the last part of the statement, it emphasized that breast milk is deficient in vitamin D—making no mention of the fact that the low intake of vitamin D during pregnancy and lactation that it advocates and the practice of keeping infants out of the sun are the precise factors responsible for low vitamin D levels in breast milk and infant vitamin D deficiency.

The Weston A. Price Foundation recommends 2,000 IU per day of vitamin D from cod liver oil, and small additional amounts from fatty fish, shellfish, butter, and lard. Although no studies have directly assessed the use of this dose during pregnancy, a study of over 10,000 infants in Finland conducted between 1966 and 1997 showed that direct supplementation of 2,000 IU per day to infants in the first year of life virtually eradicated the risk of type 1 diabetes over the next 30 years.[35]

VITAMIN K

Compared to vitamins A and D, very little is known about the role of vitamin K in embryonic and fetal development. The enzyme that uses it to activate vitamin K-dependent proteins first shows up in the skeletal and nervous tissue of the embryo.[36] Two vitamin K-dependent proteins, bone Gla protein and matrix Gla protein, are present in the first trimester.[37] These proteins help lay down calcium salts in bone tissue and keep calcium out of the soft tissues where it does not belong.

In 1997, an infant was born to a mother who took Warfarin during pregnancy. This drug interferes with the normal clotting mechanism of the blood by creating an effective vitamin K deficiency. During the early development of the middle third of her face, the cartilage of her septum calcified; at birth, her nose was a stub. Since only twenty percent of the septum protrudes from the face, a mere ten percent reduction in its length can cut the length of the nose in half. She also had cavities and plaques in her spinal cord; she required oxygen due to respiratory distress at birth; and she was quadriplegic by twenty months.[37]

This tragic case of severe deficiency illustrates the essential role of K vitamins in the development of proper facial proportions and the much more important and fundamental development of the nervous system.

Vitamin K_2 has a higher rate of transport across the placenta than vitamin K_1.[38] When mothers receive injections of vitamin K_2, the placenta rapidly accumulates it and then releases it slowly to the fetus over time.[39] Vitamin K_1 is found in leafy greens while vitamin K_2 is found in fermented foods and grass-fed animal fats—especially natto, goose liver, cheese, and to a lesser extent butter and egg yolks.[40]

DHA

The fetus, infant and adult can all convert the omega-3 fatty acid found in plant oils, alpha- linolenic acid (ALA), into docosohexaenoic acid (DHA)—but the rate of this conversion is no more than one percent at all ages and stages of development. DHA may be necessary for the formation of neurons and for the synthesis of the important brain lipid phosphatidylserine; it is also the precursor to an important compound that protects neurons when they are assaulted by oxidative stress. The fetus hoards DHA from the mother and incorporates it into its brain at ten times the rate at which it can synthesize it.41 DHA can be obtained primarily from cod liver oil and fatty fish and in small amounts from grass-fed animal fats.

COD LIVER OIL

The best source of DHA and vitamins A and D is high-vitamin cod liver oil. Cod liver oil also provides another omega-3 fatty acid, eicosapentaenoic acid (EPA). A number of studies have demonstrated the benefits of using cod liver oil during pregnancy and lactation. Rats that are fed cod liver oil during pregnancy give birth to offspring that have higher cognitive performance than controls at six months. If rats are fed a protein-deficient diet during pregnancy, their offspring have disturbed glucose metabolism; if they are also fed cod liver oil, however, the glucose metabolism of their offspring is normal.[42]

The benefits of cod liver oil during pregnancy have been verified in humans as well. Use of cod liver oil during pregnancy is independently associated with birth weight.[43] A double-blind, placebo- controlled study showed the use of cod liver oil during pregnancy and lactation to increase the child's IQ at the age of four years. In this study, the control group received the same amount of fat-soluble vitamins as the cod liver oil group, so the effects are most likely due to the DHA.44 In Norway, use of cod liver oil during pregnancy was associated with a 70 percent reduced risk of type 1 diabetes.[45]

The Weston A. Price Foundation recommends 20,000 IU of vitamin A and 2,000 IU of vitamin D from cod liver oil during pregnancy. This can be obtained from 1¾ teaspoons of the high-vitamin variety. This amount also supplies about two grams of omega-3 fatty acids—the same amount shown in one study to prevent premature delivery.[46]

BIOTIN

Biotin is a B vitamin but has also been called "vitamin H." Researchers have studied its role in pregnancy for decades but only recently have discovered that marginal biotin deficiency during this critical period is the norm.

Several years ago investigators tracked the biotin status of thirteen pregnant women through the course of their pregnancies, measuring a marker of deficiency in their urine. The deficiency marker increased during both early and late pregnancy in all thirteen women; in nine, it increased above the upper limit. These women, however, had none of the traditional symptoms of deficiency such as skin problems or depression. 47

In pregnant rats, a five percent egg white diet produced a marginal biotin deficiency. The activity of biotin-dependent enzymes declined 10 percent in the mother. Yet in the fetus, the activity of these enzymes decreased a full 50 percent. Although the mother had no obvious symptoms herself, her offspring suffered an increased risk of limb and palate defects. These effects were all reversed when biotin was added to the diet in addition to egg whites.[47]

Whether marginal biotin deficiency causes birth defects in humans is an open question, but the results of the rat studies merit attention to increasing one's intake during pregnancy. Most foods contain some of this vitamin, but it is primarily found in liver and egg yolks (see Figure 4).[48]

Egg whites contain a glycoprotein called avidin that strongly binds to biotin and prevents its absorption. Cooking neutralizes avidin, but not completely. Frying destroys 67 percent, boiling the egg white directly for two minutes destroys 60 percent, and poaching only destroys 29 percent.[49] Raw egg whites, then, should be strictly avoided, and cooked egg whites should be consumed in moderation—and never without the yolk. The addition of pure egg yolks to smoothies and ice cream will help boost biotin status.

FOLATE

Folate is probably the vitamin whose essential role in pregnancy is most widely known. It is necessary for the production of new DNA, and new DNA is needed for new cells. The growing life within the womb engages in constant cell division, and the mother must expand her blood supply with the production of new red blood cells as well—these activities demand a generous supply of folate.[50]

Adequate folate intake prevents neural tube defects (defects of the brain and spinal cord) and increases birth weight. It may also prevent spontaneous abortion, mental retardation and deformations of the mouth, face, and heart.[50]

The pregnancy RDA for folate is 600 micrograms (mcg) per day. This figure is based on the amount needed to prevent the folate concentration of the mother's red blood cells from dropping during pregnancy and on urinary markers indicating the amount of folate being used.[51] It assumes that only half of the vitamin is absorbed from food, although this figure is just an average; the rate of folate absorption is dependent on zinc status.

Synthetic "folic acid" is a chemical that is not normally found in foods or the human body. It can be converted into usable forms of folate, but this conversion is limited to about 200 mcg per single dose in healthy volunteers;[52] it may be even more limited during long-term exposure or in certain people. Synthetic "folic acid" does not cross the placenta; folate crosses the placenta as the naturally occurring 5-methyl-tetrahydrofolate.[50] Since the synthetic supplements do prevent neural tube defects, pregnant women should use them if they are not going to eat folate-rich diets; whenever possible, however, it is best to meet the folate requirement from foods. Folate-rich foods include liver, legumes, and greens (see Figure 5 and Figure 7).

CHOLINE

Choline is related to folate because the body can turn it into a compound called betaine that can be substituted for folate in certain chemical reactions. Perhaps for this reason, a low intake is associated with a four-fold increased risk of neural tube defects.[53]

Choline has a much more direct role, however, in the development of the brain. It is especially important for the formation of cholinergic neurons (neurons that use the neurotransmitter acetylcholine), which takes place from day 56 of pregnancy through three months postpartum; and for the formation of the connections between these neurons, called synapses, which occurs at a high rate through the fourth year of life.[53]

Rats fed three times the normal choline requirement during pregnancy give birth to offspring with remarkably resilient nervous systems. These offspring have a lifelong 30 percent increase in visuospatial and auditory memory; they grow old without developing any age-related senility; they are protected against the assaults of neurotoxins; they have an enhanced ability to focus on several things at once; and they have a much lower rate of interference memory. Interference memory is when a past memory interferes with an immediate memory—for example, when a past memory of where you parked your car interferes with your ability to find it when you exit the store.[53]

The RDA for non-pregnant women is 425 milligrams (mg) per day. The RDA for pregnant women is 450 mg per day, only 25 mg more. The increase is based on the typical transfer of choline to and accumulation in the fetus.[54] Rat studies, however, suggest that an amount two to three times this may provide the offspring with lasting benefits. Choline can be obtained from liver, egg yolks, and high-quality grass-fed dairy; it can be obtained to a lesser extent from meats, crucifers, nuts, and legumes (see Figure 6 and Figure 7).

GLYCINE

The amino acid glycine is conditionally essential during pregnancy. Usually, we are able to make enough of it ourselves to meet our basic survival needs; during pregnancy, however, it must be obtained from food. It is the limiting factor for protein synthesis in the fetus, and thus almost certainly a limiting factor for fetal growth.[55]

The fetus can obtain glycine from two sources: the placenta transports glycine from the mother's blood, and it uses folate to manufacture it from another amino acid called serine. The mother can obtain glycine primarily from collagen- rich foods such as skin and bones or bone broths (see Figure 8).[56]

Glycine is depleted in the detoxification of excess methionine, another amino acid. Eggs and meat are the main sources of methionine—it not only constitutes a greater percentage of their total protein but these foods are also higher in total protein than plant foods (see Figure 9).[56] It is important, therefore, for the expectant mother to liberally match her eggs and muscle meats with glycine-rich skin and bones and folate-rich liver, legumes and greens.

THE DEVELOPMENTAL ORIGINS THEORY

The scientific community is showing increasing interest in what is called the "developmental origins theory." This theory postulates that the nutritional environment in the womb affects not only the risk of defects immediately apparent at birth, but also the lifelong risk of degenerative disease.

Weston Price supported an early version of this theory in the 1930s and 1940s. In *Nutrition and Physical Degeneration*, for example, he proposed that an increased risk of tuberculosis was largely determined by a deformation of the chest cavity that began taking shape in the womb and paralleled the deformation of the dental arch that causes crowded teeth. He also demonstrated an association between delinquent behavior and deformities of the dental arch and found the same association with non-delinquent but mentally retarded children as well. In one case, he induced puberty and rapid mental development in a teenager by surgically broadening his maxilla to stimulate his pituitary. The maxilla is the upper jaw bone; it is one of the bones of the middle third of the face, which Price so often found underdeveloped in people who grew up on modern foods.[57]

The modern developmental origins theory (described in greater detail in the sidebar on page 34) observes that birth weight is determined in part by embryonic and fetal nutrition; and low birth weight is in turn associated with an increased risk of heart disease, stroke, high blood pressure, diabetes and kidney disease. To explain these observations, the theory proposes that poor nutrition during pregnancy causes changes in the growth and development of the internal organs, which in turn affects the lifelong risk of degenerative disease. Since poor nutrition during this period can result in lower birth weight, an indirect association between birth weight and the risk of degenerative disease arises.[58]

The ideal birth weight according to these studies appears to be between 8.5 and 9.5 pounds. These figures exclude infants whose birth weights are low because of premature delivery; it is the rate of fetal growth, not the birth weight itself, that counts.[59] The theory does not suggest that the risk of disease is affected only by the rate of growth within the womb—simply that the nutritional environment during this period makes an incomplete yet permanent contribution to that risk.

NUTRITION FOR FETAL GROWTH

Genetics has little if anything to do with birth weight. A 1995 study examined 62 cases of egg donor pregnancies. The birth weight of the baby was not correlated with the donor's weight, the donor's birth weight, or the birth weights of the donor's other children; it was, however, correlated with the recipient's weight.[61] This study shows that birth weight is determined by the environment that the womb provides rather than the genome present at conception.

An intake of meat protein below 25 grams per day during late pregnancy and an intake of carbohydrate above 265 grams per day during early pregnancy are associated with a decrease in birth weight. A low intake of animal protein relative to carbohydrate is also associated with an increase of blood pressure at forty years of age.[62] In order to obtain adequate glycine for growth, meat and egg protein should be balanced with the liberal use of liver, skin, bone broths, legumes and green vegetables.

The use of cod liver oil is independently associated with birth weight.43 Seven out of twelve trials have shown folic acid supplementation to increase birth weight.50 Iron deficiency compromises fetal growth,[46] and a major deficiency in any vitamin or mineral is likely to do the same.

In general, the role of vitamins and minerals in providing for robust fetal growth is understudied and probably much more important than the scarcity of the available literature would suggest.

QUALITY, NOT QUANTITY

Although the quantity of growth is a useful marker for the risk of disease, the determining factor is the quality of growth. Choline may confer remarkable benefits to the developing nervous system without having much of an impact on head circumference, and vitamin A may provide for robust kidney function without having much of an impact on the size of the waist or torso.

We must take the same care in preparing the human womb for the seed of life to be planted therein that we take to prepare the womb of the earth for the seeds of the plant life from which we and our animals will take nourishment. A generous intake of all nutrients—especially the fat-soluble vitamins, essential fatty acids, biotin, folate, choline and glycine—will supply the soil of the womb with everything the life developing within it needs for robust and vigorous growth and a long, healthy life to come.

Figures

FIGURE 1. IMPORTANT NUTRIENTS FOUND IN FISH EGGS

Nutrient Content per 100 grams (% pregnancy RDA)

Cholesterol	588 mg
Vitamin B_{12}	20.0 mcg (770%)

Choline	491 mg (109%)
Selenium	65.5 mcg (109%)
Calcium	275 mg (28%)
Magnesium	300 mg (83%)
EPA (omega-3)	2,741 mg
DHA (omega-3)	3,801 mg
Total omega-3	6,789 mg (485%)
Vitamin A	905 IU (35%)
Vitamin D	232 IU (116%)

FIGURE 2. COMPARISON OF COMMERCIAL U.S. MILK AVAILABLE IN 1970 TO MAASAI MILK IN DRY AND WET SEASONS IN NAROK DISTRICT OF KENYA

Nutrient	American	Maasai – Dry	Maasai – Wet
Fat	3.8 g/100 mL	6.5 g/100 mL	10.4 g/100 mL
Cholesterol	11 mg/100 mL	16.4 mg/100 mL	24.2 mg/100 mL
Phospholipids	21 mg/100 mL	78 mg/100 mL	109 mg/100 mL
Sugar	4.9 g/100 mL	3.4 g/100 mL	3.4 g/100 mL

FIGURE 4. BIOTIN CONTENT OF FOODS

Food	Biotin Content (mcg)
Liver (3 ounces)	27
1 egg yolk	25
Baker’s yeast (1 packet)	14
Whole wheat bread (1 slice)	6
Cheese (1 ounce)	2-6
Avocado	6
Salmon (3 ounces)	4

Chicken (3 ounces)	3
Pork (3 ounces)	2
Artichoke (medium)	2
Raspberries (1 cup)	2

FIGURE 5. MEETING THE DAILY FOLATE REQUIREMENT

The folate requirement for pregnancy can be met by one of any of the following (volume after cooking):

Chicken liver	3.7 ounces
Calf's liver	2.8 – 6.4 ounces
Beef liver	8.2 ounces
Lentils	1.7 cups
Other legumes	2.0-3.0 cups
Spinach	2.3 cups
Asparagus	2.3 cups
Beets	4.4 cups
Most greens	3.0-6.0 cups

FIGURE 6. MEETING THE DAILY CHOLINE REQUIREMENT

The RDA for choline during pregnancy can be met by one of any of the following:

- 2.5 8-ounce glasses high-quality Maasai milk*
- 3.5 egg yolks
- 3.8 ounces beef liver
- 5.5 ounces chicken liver
- 10.6 ounces wheat germ
- 13 ounces bacon
- 1.25 pounds beef
- 1.4 pounds cod, salmon or shrimp
- 1.5 pounds chicken
- 2.2 pounds crucifers, nuts or legumes

* This figure assumes that Maasai milk during the wet season is five times higher in choline than commercial American milk, as discussed above.

FIGURE 7. FOLATE- AND CHOLINE-RICH MEALS AND SNACKS

Any combination of three meals and one snack and most combinations of three meals will exceed the pregnancy RDA for both nutrients. Folate from raw milk is accompanied by a protein that doubles its absorption. Absorption of folate from food in general is dependent on zinc status. High-quality grass-fed milk may be three to five times richer in choline.

Food	Folate mcg (% RDA)	Choline mg (% RDA)
3 eggs	71	378
2 pieces ww toast	43	24
8 ounces milk	12	35
Total	126 (21%)	437 (97%)
1/4 cup dry lentils	80	23
1/2 cup chop onion	15	5
1 medium carrot	12	5
1/2 cup broccoli	84	31
Stock	~	~
8 ounces milk	12	35
Total	203 (34%)	99 (22%)
100 g salmon	29	66
1 cup asparagus	268	47
1 potato	14	23
8 ounces milk	12	35
Total	323 (54%)	171 (38%)
100 g beef liver	253	426
3 ounces bacon	~	35
1 cup onion	30	10
1 potato	14	23
8 ounces milk	12	35

Total	309 (52%)	529 (117%)

Gluten-Free Casein-Free (GFCF)

Food	Folate mcg (%RDA)	Choline mg (% RDA)
3 eggs (omelet)	71	378
1/2 cup onion	15	5
1/2 cup tomato	14	6
1/2 cup broccoli	84	31
4 ounces bacon	~	47
1 potato	14	23
Total	198 (33%)	490 (109%)
100 g chicken liver	560	327
1/2 cup cooked brown rice	4	9
1 cup cooked spinach	263	36
Total	827 (138%)	372 (83%)
SNACKS		
1/2 cup crispy almonds	23 (4%)	36 (8%)
1/2 cup hulled sunflower seeds	159 (27%)	77 (17%)
SMOOTHIE		
3 egg yolks	75	348
1 banana	24	12
8 ounces milk	12	35
Total	111 (19%)	395 (88%)

FIGURE 8. PERCENTAGE OF TOTAL PROTEIN AS GLYCINE

Glycine is found primarily in skin and bones.

- Chicken Breast 5%
- Chicken Skin 16%

- Chicken Stock 31% (estimate)

FIGURE 9. PERCENTAGE OF TOTAL PROTEIN AS METHIONINE

Animal products have a higher percentage of their total protein as methionine than plant products. Although not shown in the chart, they also contain much more protein per unit of weight or volume. The main sources of methionine in the diet, then, are eggs and muscle meats.

Animal Foods	Plant Foods
Bacon 2.3%	Almonds 0.8%
Ground Beef 2.5%	Lentils 0.8%
Chicken 2.8%	Tofu 1.3%
Eggs 3.0%	Walnuts 1.9%

Sidebars

IS THE PREGNANCY RDA FOR VITAMIN A ADEQUATE?

The RDA of vitamin A for pregnant women is only 2,600 IU—just 300 IU more than the RDA for women who are not pregnant. To obtain this figure, the scientists at the Institute of Medicine (IOM) made the following calculation: first, they ascertained from previous reports the amount of vitamin A stored in the livers of fetuses that were spontaneously or voluntarily aborted between 37 and 40 weeks; second, they doubled this figure, assuming that half of the fetal vitamin A stores exist in the liver; and third, they divided this amount over the number of days in the last trimester, during which they presumed this vitamin A would accumulate.[20]

There are several problems with this calculation. Since the fetuses were aborted, we have no idea what their future health would have been like—their visual acuity, their hearing, their intelligence, their facial and dental features, their reproductive health, or their length of life. And the function of vitamin A, of course, is not to be stored but to be used. The fetus does not simply hold on to vitamin A to use it after birth, but rapidly uses and metabolizes it to regulate the entirety of its growth and development. Granted, the IOM acknowledges that it has only used this data because better data do not exist—yet it is important to emphasize just how little the data tell us.

VITAMIN A AND BIRTH DEFECTS

The claim that intakes of vitamin A over 10,000 IU per day can increase the risk of birth defects can be traced back to a 1995 paper published by a group of researchers led by Dr. Kenneth Rothman of Boston University.[21] The researchers followed almost 23,000 women over the course of their pregnancies and found that women who consumed more than 10,000 IU of vitamin A during the first trimester gave birth to offspring with a 2.4-fold greater risk of total birth defects and a 4.8-fold greater risk of cranial-neural-crest defects (a rather broad group of defects whose classification is controversial). Among the 188 women who consumed this amount of vitamin A from “food” alone, there was an 80 percent increase in the risk of total birth defects and two times the risk of cranial-neural-crest defects. Because there were so few women consuming vitamin A from “food” alone, however, the researchers could not conclusively distinguish the association from the effect of chance.

This study has a number of important flaws. Most of the vitamin A came from multivitamins. The authors did not distinguish between various food sources—and most “food” vitamin A comes from fortified breakfast cereals. Three groups of experts wrote to the journal questioning the authors’ classification of cranial-neural-crest defects.22,23,24 Perhaps most important, the authors may have underestimated the rate of certain types of birth defects. The rate of total birth defects among the 20,000 women consuming less than 10,000 IU was only 1.5 percent; by contrast, the generally accepted background rate is 3-4 percent. The rate of defects among the 3,000 women consuming more than 10,000 IU of vitamin A was 3 percent—on the lower end of normal.[24]

The most important objection to this study is the fact that it conflicts with all the other evidence:

- An earlier 1990 study conducted in Spain found that among 25,000 births, doses of vitamin A over 40,000 IU per day carried a 2.7-fold higher risk of birth defects, but doses of vitamin A up to 20,000 IU or between 20,000 and 40,000 IU both carried a 50 percent lower risk of birth defects compared to no supplementation.[25]
- A 1996 study of 522,601 births found that the children of women supplementing with at least 10,000 IU of vitamin A in addition to a multivitamin had a lower risk of birth defects than those of women who did not supplement, although the association could not be distinguished from the effect of chance.[26]
- A 1997 study of 1,508 births found no relationship between birth defects and use of vitamin A supplements, fortified breakfast cereals, organ meats or liver.[27]
- A 1999 prospective study of 311 mothers who consumed between 10,000 and 300,000 IU of vitamin A in the first trimester and a similarly sized group that did not supplement with vitamin A found no evidence of an increased risk of major malformations with increasing dose. The median dose was 50,000 IU. The group as a whole had a 50 percent lower risk of major malformations than those who did not supplement, and there were no major malformations in offspring born to mothers consuming more than 50,000 IU.

The preponderance of the evidence clearly favors the view that 20,000-25,000 IU of vitamin A during pregnancy is safe and may even reduce the risk of birth defects.[63]

THE DEVELOPMENTAL ORIGINS THEORY

The British researcher David J. Barker first proposed the developmental origins theory in the 1980s to explain a puzzling paradox: as British prosperity increased, so did heart disease; yet geographically, the most heart disease was found in the poorest places in Britain. Barker found geographical associations of heart disease with infant mortality, but not with smoking or dietary fat. Yet even infant mortality had declined over the course of the century, just as prosperity had gone up. When he accounted for a time lag between cause and effect of more than 50 years, however, the paradox was resolved—something was determining the risk of disease at or near birth, not late in life when the disease develops.[58]

Barker and his team of researchers then studied the birth weight of individuals born between 1911 and 1930 in Hertfordshire, UK. This allowed them to study the association at the level of individuals rather than local districts. Infants carried to term with birth weights between 8.5 and 9.5 pounds had a 45 percent lower risk of heart disease than infants carried to term weighing less than 5.5 pounds; they had a similarly lower risk of stroke, a nearly 70 percent lower risk of insulin resistance, and a slightly lower blood pressure in the seventh decade of life. The risk declined steadily and evenly between 5.5 and 9.5 pounds and began increasing thereafter. Later, other researchers found similar trends in the United States and southern India.[59]

Data from the three-month Dutch famine that occurred during World War II suggests that specific types of diseases are associated with specific windows of development during pregnancy. Women who were exposed to this famine during their first trimester gave birth to offspring with an increased risk of cardiovascular disease; women exposed during their second trimester gave birth to offspring with an increased risk of kidney disease; women exposed during their third trimester gave birth to offspring with an increased risk of insulin disorders.[60]

Developmental origins theorists have offered several explanations for these associations: poor nutrition could alter the development of the pancreas, which secretes insulin, and the liver, which secretes cholesterol and blood clotting proteins; muscle tissue could program itself for insulin resistance in order to spare glucose and amino acids for the brain when the supply of these materials is limited; overgrowth of the left ventricle of the heart—which itself is independently associated with cardiovascular disease—could be a response to the need to supply a greater volume of blood to the brain at the expense of the other tissues.[59]

Just as Weston Price had associated the skeletal defects that occur because of poor prenatal nutrition with the risk of disease in childhood and adolescence, researchers are now associating the defects of the internal organs that occur due to poor nourishment with the risk of disease in adulthood and old age.

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