Chapter 1 : Fetal origins hypothesis - Wikipedia

David Barker's keen observations have been popularized as the "Barker hypothesis," or "Fetal Origins of Adult Disease" (FOAD). It was his group that noted that low birth weight (LBW) serves as proxy not just for fetal, but also adult health.

Associated risk of lifelong diseases includes cardiovascular disease , type-2 diabetes , obesity , and hypertension. Babies born lighter in weight appear to have an increased rate of mortality than babies born at a heavier weight. Death rate would rise as birth weight increases beyond normal birth weight range. When this theory was first proposed, it was not well accepted and was met with much skepticism. The word "programming" illustrates the idea that during critical periods in early fetal development, there are persisting changes in the body structure and function that are caused by environmental stimuli. This would result in an increased risk of fetal macrosomia and neonatal hypoglycemia. High glucose concentrations in the blood of pregnant women cause an intensified transfer of nutrient to the fetus, increasing fetal growth. Nutrients other than sugar and their linkage to fetal overgrowth in diabetic pregnancy were taken into account, too, but the crucial role of the fetal hyperinsulinism and monitoring of motherly glucose was nevertheless stressed. A tile tribute to the Dutch Famine The dutch famine[edit] Since small birth weight is associated with an increased risk of chronic diseases in later life, and poor maternal nutrition during gestation contributes to restricted fetal development, maternal malnutrition may be a cause of increased disease susceptibility in adulthood. The Dutch famine of or the "Hunger Winter" during World War II serves as an epidemiological study that is used to examine the effects of maternal under-nutrition during different gestational stages. The famine was a period roughly five to six months of extreme food shortage in the west of Netherlands. The daily ration had increased to more than calories in June The period of maternal starvation is shown to have limited intrauterine growth and has been identified as one of the most important contributors to coronary heart disease as well as other chronic diseases later in life. The french paradox[edit] The French paradox regards the seemingly paradoxical fact that people living in France since many generations suffer from a relatively little incidence of heart disease , although the traditional French cuisine is high in saturated fatty acids. One explanation suggested for the paradox is the potential impact of nutritional enhancements during pregnancy and the first months and years of life that would positively influence the health of following generations: After the defeat in the Franco-German War , a nutrition program for pregnant women and small children with the aim of strengthening future generations of soldiers was introduced by the French Government. This might be one explanation for positive health-outcomes in following generations. The fertilized egg or the zygote becomes a blastocyst where the outer layer and the inner cell mass differentiate to form placenta and the fetus respectively. Implantation occurs at this stage where the blastocyst becomes buried in the endometrium. It is also in this stage where the blastocyst develops into an embryo , where all major features of human are present and operational by the end of this stage. During this period of time, the embryo develops rapidly and becomes a fetus. Pregnancy becomes visible at this stage. The pattern and amount of weight gain is closely associated with gestational stages. In the first trimester blastogenesis and early embryonic stages , the mother experiences a minimal weight gain approximately 0. The amount of weight gain depends strongly on their pre-pregnant weight. Gestational weight gain should also be progressive and the recommended weight depends on pre-pregnant body weight. Women having a BMI of This group have the lowest risk of adverse birth outcomes. It is advised that women with a normal weight before pregnancy should gain a total of Participating in aerobic activities such as walking and swimming 3 to 4 times a week is usually adequate. A proper diet is also essential to healthy weight gain. The common saying "a woman is eating for two" often leads to mothers thinking that they should eat twice as much. In reality, only a small increase in caloric intake is needed to provide for the fetus; approximately calories more in the second trimester and calories more in the third trimester. As such, underweight mothers should seek individualized advice tailored especially for themselves. The first column categorizes the type of body weight based on the Body Mass Index. The second column summarizes the total recommended weight gain for each type of body weight, and the third column presents the corresponding weekly weight gain during the period when the fetus undergoes rapid growth

during second and third trimesters.

Chapter 2 : Family History as a Risk Assessment Tool - ACOG

Prenatal origins of adult disease. a cross-sectional study has shown that low birth weight is associated with decreased overall adult health status investigate the mechanism of prenatal.

Advanced Search ABSTRACT Recent research suggests that several of the major diseases of later life, including coronary heart disease, hypertension, and type 2 diabetes, originate in impaired intrauterine growth and development. Evidence that coronary heart disease, hypertension, and diabetes are programmed came from longitudinal studies of UK men and women in which size at birth was related to the occurrence of the disease in middle age. People who were small or disproportionate thin or short at birth had high rates of coronary heart disease, high blood pressure, high cholesterol concentrations, and abnormal glucose-insulin metabolism. These relations were independent of the length of gestation, suggesting that cardiovascular disease is linked to fetal growth restriction rather than to premature birth. Replication of the UK findings has led to wide acceptance that low rates of fetal growth are associated with cardiovascular disease in later life. Impaired growth and development in utero seem to be widespread in the population, affecting many babies whose birth weights are within the normal range. Although the influences that impair fetal development and program adult cardiovascular disease remain to be defined, there are strong pointers to the importance of the fetal adaptations invoked when the maternoplacental nutrient supply fails to match the fetal nutrient demand. The process whereby a stimulus or insult at a sensitive or critical period of development has long-term effects is termed programming 2. In evolutionary terms, the phenomenon is likely to reflect the benefits of plasticity during early development. Consistent with this, it is thought that coronary heart disease may be a consequence of fetal adaptations to undernutrition that are beneficial for short-term survival, even though they are detrimental to health in postreproductive life 3. Experimental studies in animals have documented many examples of fetal programming, with recent studies showing that alterations in maternal nutrition can have long-term effects on the offspring that are relevant to human cardiovascular disease. For example, feeding pregnant rats a low-protein diet results in lifelong elevation of blood pressure in the offspring 4. Rats whose mothers had been fed a diet with a low ratio of protein to energy during pregnancy showed a permanently altered balance between hepatic glucose production and utilization; control rats fed the same diet during postnatal life had no alterations in hepatic glucose metabolism 5. Other notable long-term effects of alterations in maternal nutrition include changes in cholesterol metabolism, insulin secretion, and renal development 3. Although some effects of nutrition may be direct consequences of alterations in substrate availability, several are thought to be mediated by hormonal effects. These may alter the development of specific fetal tissues during sensitive periods of development 6 , 7 , or may lead to long-lasting changes in hormone secretion or tissue hormone sensitivity 8. Experiments in animals have implicated the fetal hypothalamus as a key site that can be programmed by transient changes in prenatal endocrine status 3. In many of these countries, the steep rise was followed by a fall over recent decades that cannot be accounted for by changes in adult lifestyle. The incidence of coronary heart disease is now rising in other parts of the world to which Western influences are extending, including China, India, and Eastern Europe. Although the steep rise in coronary heart disease in Britain and other Western countries was associated with rising prosperity, geographic studies have shown that rates are now twice as high in the poorer areas of the country and in lower-income groups 3. Combined with the limited ability of adult lifestyle risk factors to predict coronary heart disease, this paradox led to the hypothesis that adverse influences in early life might play an important role. Support for this hypothesis came from the observation by Rose 9 that siblings of patients with coronary heart disease had stillbirth and infant mortality rates that were twice as high as those of control subjects. Further support subsequently came from geographic studies reported by Forsdahl 10 , showing that past infant mortality correlated with later arteriosclerotic heart disease in the 20 counties of Norway. Although these studies suggested that a poor standard of living in childhood and adolescence was a risk factor for heart disease, geographic comparisons in England and Wales pointed more strongly to the importance of an adverse environment in intrauterine life and early infancy Areas with high neonatal and postneonatal mortality earlier this century were found to have

markedly elevated coronary heart disease death rates Because low birth weight is strongly associated with elevated neonatal and postneonatal mortality, these observations led to the hypothesis that low-birth-weight babies who survived infancy and childhood might be at increased risk of coronary heart disease as adults. The early epidemiologic studies that pointed to long-term effects of an adverse intrauterine environment were based on the strategy of following up men and women in middle and late life whose body measurements at birth had been recorded. A follow-up study of men and women born in Hertfordshire, United Kingdom, showed for the first time that those who had had low birth weights had relatively high death rates from coronary heart disease in adult life Thus, among men and women born during â€", death rates from coronary heart disease fell progressively with increasing birth weight in both men and women Figure 1 3. A small rise in death rates from coronary heart disease at the highest birth weights in men could relate to the macrosomic infants of women with gestational diabetes. Another study, of men born in Sheffield during â€", showed that it was particularly people who were small at birth as a result of growth retardation, rather than those born prematurely, who were at increased risk of the disease Coronary heart disease death rates, expressed as standardized mortality ratios, in men and women born in Hertfordshire, United Kingdom, from to , according to birth weight. Derived from Osmond et al View large Download slide Coronary heart disease death rates, expressed as standardized mortality ratios, in men and women born in Hertfordshire, United Kingdom, from to , according to birth weight. Replication of the UK findings has led to wide acceptance that low rates of fetal growth are associated with coronary heart disease in later life. For example, confirmation of a link between low birth weight and adult coronary heart disease has come from studies of nurses in the United States 14 ; of men in Caerphilly, South Wales 15 ; and of men and women in Mysore, South India Studies examining the mechanisms underlying these associations have shown that the trends in coronary heart disease with birth weight are paralleled by similar trends between restricted early growth and traditional cardiovascular disease risk factors 3. Subsequent studies have found that a wide range of organs and systems may be programmed by the intrauterine environment. These findings are in keeping with the results of experimental studies in animals and suggest that programming reflects a general principle of developmental biology. Listed in Table 1 are several key tissues and systems for which evidence exists in humans pointing to programming by the nutrient and hormonal milieu of the fetus. Observations linking the intrauterine environment with later hypertension, diabetes, elevated blood cholesterol and fibrinogen concentrations, and polycystic ovary syndrome serve to illustrate some of the principles that underlie fetal programming and are described in more detail below.

Chapter 3 : Chapter 7 - Physical Activity Guidelines - calendrierdelascience.com

FROM FETAL ORIGINS OF ADULT DISEASE TO DEVELOPMENTAL ORIGINS OF HEALTH AND DISEASE. Barker's hypothesis stimulated a great deal of worldwide interest and activity in the area of developmental plasticity, Gillman et al 5 summarized in a report of the meetings of the World Congress on Fetal Origins of Adult Disease that were convened in (Mumbai, India) and (Brighton, United Kingdom.

Chapter 7 Chapter 7: Additional Considerations for Some Adults All Americans should be physically active to improve overall health and fitness and to prevent many adverse health outcomes. Most Americans should follow the Guidelines of the child and adolescent, adult, or older adult chapters, depending upon their age. However, some people have conditions that raise special issues about recommended types and amounts of physical activity. This chapter provides guidance on physical activity for healthy women who are pregnant and for people with disabilities. This chapter also affirms and illustrates how physical activity is generally appropriate for adults with chronic conditions by considering three groups of adults: Adults with osteoarthritis; Adults with type 2 diabetes; and Adults who are cancer survivors. For example, moderate-intensity physical activity by healthy women during pregnancy maintains or increases cardiorespiratory fitness. Strong scientific evidence shows that the risks of moderate-intensity activity done by healthy women during pregnancy are very low, and do not increase risk of low birth weight, preterm delivery, or early pregnancy loss. Some evidence suggests that physical activity reduces the risk of pregnancy complications, such as preeclampsia and gestational diabetes, and reduces the length of labor, but this evidence is not conclusive. Such activity does not appear to have adverse effects on breast milk volume, breast milk composition, or infant growth. Physical activity also helps women achieve and maintain a healthy weight during the postpartum period, and when combined with caloric restriction, helps promote weight loss. Key Guidelines for Women During Pregnancy and the Postpartum Period Healthy women who are not already highly active or doing vigorous-intensity activity should get at least minutes 2 hours and 30 minutes of moderate-intensity aerobic activity per week during pregnancy and the postpartum period. Preferably, this activity should be spread throughout the week. Pregnant women who habitually engage in vigorous-intensity aerobic activity or are highly active can continue physical activity during pregnancy and the postpartum period, provided that they remain healthy and discuss with their health-care provider how and when activity should be adjusted over time. Explaining the Guidelines Women who are pregnant should be under the care of a health-care provider with whom they can discuss how to adjust amounts of physical activity during pregnancy and the postpartum period. Unless a woman has medical reasons to avoid physical activity during pregnancy, she can begin or continue moderate-intensity aerobic physical activity during her pregnancy and after the baby is born. When beginning physical activity during pregnancy, women should increase the amount gradually over time. The effects of vigorous-intensity aerobic activity during pregnancy have not been studied carefully, so there is no basis for recommending that women should begin vigorous-intensity activity during pregnancy. Women who habitually do vigorous-intensity activity or high amounts of activity or strength training should continue to be physically active during pregnancy and after giving birth. They generally do not need to drastically reduce their activity levels, provided that they remain healthy and discuss with their health-care provider how to adjust activity levels during this time. During pregnancy, women should avoid doing exercises involving lying on their back after the first trimester of pregnancy. They should also avoid doing activities that increase the risk of falling or abdominal trauma, including contact or collision sports, such as horseback riding, downhill skiing, soccer, and basketball. Physical Activity for People With Disabilities The benefits of physical activity for people with disabilities have been studied in diverse groups. Overall, the evidence shows that regular physical activity provides important health benefits for people with disabilities. The benefits include improved cardiovascular and muscle fitness, improved mental health, and better ability to do tasks of daily life. Sufficient evidence now exists to recommend that adults with disabilities should get regular physical activity. Physical activity in children and adolescents with disabilities is considered in Chapter 3â€”Active Children and Adolescents. Key Guidelines for Adults With Disabilities Adults with disabilities, who are able to, should get at least minutes

per week 2 hours and 30 minutes of moderate-intensity, or 75 minutes 1 hour and 15 minutes per week of vigorous-intensity aerobic activity, or an equivalent combination of moderate- and vigorous-intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes, and preferably, it should be spread throughout the week. Adults with disabilities, who are able to, should also do muscle-strengthening activities of moderate or high intensity that involve all major muscle groups on 2 or more days per week as these activities provide additional health benefits. When adults with disabilities are not able to meet the above Guidelines, they should engage in regular physical activity according to their abilities and should avoid inactivity. Adults with disabilities should consult their health-care providers about the amounts and types of physical activity that are appropriate for their abilities. Explaining the Guidelines In consultation with their health-care providers, people with disabilities should understand how their disabilities affect their ability to do physical activity. Some may be capable of doing medium to high amounts of physical activity, and they should essentially follow the Guidelines for adults. Some people with disabilities are not able to follow the Guidelines for adults. These people should adapt their physical activity program to match their abilities, in consultation with their health-care providers. For More Information See Chapter 4â€”Active Adults , for details on these Guidelines and how to meet Meeting the Guidelines People with disabilities are encouraged to get advice from professionals with experience in physical activity and disability because matching activity to abilities can require modifying physical activity in many different ways. Some people with disabilities also need help with their exercise program. For example, some people may need supervision when performing muscle-strengthening activities, such as lifting weights. Physical Activity for People With Chronic Medical Conditions Adults with chronic conditions should engage in regular physical activity because it can help promote their quality of life and reduce the risk of developing new conditions. Three examples are provided below to illustrate the benefits of physical activity for persons with chronic conditions. For many chronic conditions, physical activity provides therapeutic benefits and is part of recommended treatment for the condition. However, this chapter does not discuss therapeutic exercise or rehabilitation. Key Messages for People With Chronic Medical Conditions Adults with chronic conditions obtain important health benefits from regular physical activity. When adults with chronic conditions do activity according to their abilities, physical activity is safe. Adults with chronic conditions should be under the care of health-care providers. People with chronic conditions and symptoms should consult their health-care providers about the types and amounts of activity appropriate for them. Osteoarthritis is a common condition in older adults, and people can live many years with osteoarthritis. People with osteoarthritis are commonly concerned that physical activity can make their condition worse. Osteoarthritis can be painful and cause fatigue, making it hard to begin or maintain regular physical activity. Yet people with this condition should get regular physical activity to lower their risk of getting other chronic diseases, such as heart disease or type 2 diabetes, and to help maintain a healthy body weight. Strong scientific evidence indicates that both aerobic activity and muscle-strengthening activity provide therapeutic benefits for persons with osteoarthritis. When done safely, physical activity does not make the disease or the pain worse. Studies show that adults with osteoarthritis can expect improvements in pain, physical function, quality of life, and mental health with regular physical activity. People with osteoarthritis should match the type and amount of physical activity to their abilities and the severity of their condition. Most people can usually do moderate-intensity activity for minutes 2 hours and 30 minutes a week or more, and may choose to be active 3 to 5 days a week for 30 to 60 minutes per episode. Some people with arthritis can safely do more than minutes of moderate-intensity activity each week and may be able to tolerate equivalent amounts of vigorous-intensity activity. Health-care providers typically counsel people with osteoarthritis to do activities that are low impact, not painful, and have low risk of joint injury. Swimming, walking, and strength-training are good examples of this type of activity. Physical Activity for Adults With Type 2 Diabetes Physical activity in adults with type 2 diabetes shows how important it can be for people with a chronic disease to be active. Physical activity has important therapeutic effects in people with diabetes, but it is also routinely recommended to reduce risk of other diseases and help promote a healthy body weight. For example, strong scientific evidence shows that physical activity protects against heart disease in people with diabetes. Moderate-intensity activity for about minutes a week helps to substantially lower the risk of heart

disease. A person who moves toward minutes 5 hours or more of moderate-intensity activity a week gets even greater benefit. Adults with chronic conditions should work with their health-care providers to adapt physical activity so that it is appropriate for their condition. For example, people with diabetes must be careful to monitor their blood glucose and avoid injury to their feet. Physical Activity for Cancer Survivors With modern treatments, many people with cancer can either be cured or survive for many years, living long enough to be at risk of other chronic conditions, such as high blood pressure or type 2 diabetes. Some cancer survivors are at risk of recurrence of the original cancer. Some have experienced side effects of the cancer treatment. Like other adults, cancer survivors should engage in regular physical activity for its preventive benefits. Physical activity in cancer survivors can reduce risk of new chronic diseases. Further, studies suggest physically active adults with breast or colon cancer are less like to die prematurely or have a recurrence of the cancer. Physical activity may also play a role in reducing adverse effects of cancer treatment. Cancer survivors, like other adults with chronic conditions, should consult their health-care providers to match their physical activity plan to their abilities and health status.

Chapter 4 : Family History|Genomics|CDC

In this issue there is a set of papers on various aspects of heart disease, with some cross reference to the origins of adult health in prenatal life and the Barker hypothesis. On the environmental side, congestive heart failure in Quebec is highest during the winter and declines in the summer, with.

Background[edit] The fetus was once believed to be a "perfect parasite", [3] immune to harmful environmental toxins passed from the mother via the placenta. Stemming from this belief, pregnant women of the early to mid 20th century freely drank alcohol, ingested medications, smoked cigarettes, and were largely ignorant of any nutritional needs for a developing fetus. This easy going attitude about pregnancy was challenged, however, by findings relating substances ingested by a mother to tragic outcomes for a fetus. The birth defects crisis due to the medication thalidomide in the s, where thousands of children were born with defects ranging from brain damage to truncated and missing arms and legs is an example of how a seemingly miracle medication supposed to prevent morning sickness instead had disastrous consequences. In , Barker published findings proposing a direct link between prenatal nutrition and late-onset coronary heart disease. His findings were met with criticism, mainly because at the time heart disease was considered to be predominantly determined by lifestyle and genetic factors. Essentially, all transmissions entering the placenta act as "postcards" giving the fetus clues as to the outside world, preparing its physiology appropriately. Thrifty phenotype[edit] The thrifty phenotype hypothesis proposes that a low availability of nutrients during the prenatal stage followed by an improvement in nutritional availability in early childhood causes an increase risk of metabolic disorders, including Type II diabetes, as a result of permanent changes in the metabolic processing of glucose-insulin determined in utero. But, once in the world, the readily accessible processed foods consumed are unable to be processed efficiently by individuals who had their metabolic systems pre-set to expect scarcity. This difference between expected nutritional deficits and actual food surplus results in obesity and eventually Type II Diabetes. Instead, she found that the results hold: Comparisons between the children who were in gestation during the flu pandemic and those in gestation immediately before or after the health crisis show marked differences between the two groups on census data. Across all socioeconomic measures, those who were fetuses during the crisis attained lower educational achievement, income, and socioeconomic status. Even federal welfare payments were higher for the gestational cohort than those born before or after the flu hit. Both prenatally exposed groups suffered lower cognitive abilities and reduced employment levels. Epidemiological and epigenetic support[edit] Epigenetics refers to the study of the behavior of genes, and how gene expression can be altered by the environment without changes made in DNA. This is believed to be particularly possible during prenatal development, and both stress and diet have been known to causes changes to a fetus. Women who are overweight at the time of pregnancy have children that are more likely to be overweight themselves. This could be due to the genetic heritability of genes related to obesity. But, siblings born to these same women after they had weight reduction surgery were no more likely to be overweight than the rest of the general population. The metabolic nature of the children was completely different, despite being born to the same mother, supporting the idea that the gestational environment strongly influences future outcomes. Poor nurture during pregnancy can worsen the hand that nature has dealt. When a significant situation, disaster, or event occurs across a given population, it can be assumed that the entire population is affected, thus generalizing findings across all demographics in a given group. Certain historical events provide epidemiological support for the developmental origins of health and disease, including the Dutch Hunger Winter and the Holocaust. In the United States, the average lifespan dropped by 12 years per person. The study concluded an It is also notable that those who were already born but young between the ages of 1 and 5 during exposure did not have a noticeable increase in coronary heart disease or kidney disease. Being exposed to the pandemic while in utero would lead to an average loss of 0. These effects were much higher or lower depending on the district of Italy. In a recent[when? It has been hypothesized that a definite link exists between influenza-induced stress on the fetus and schizophrenia. Where food was previously plentiful, supplies immediately were cut off in November , resulting in a period of starvation that lasted until

spring of Analyses of the orderly health records from this time period allow for a systematic comparison of the effects of fetal starvation. Individuals who were in utero during the Hunger Winter were subject to different outcomes depending on the period of time in which they were conceived. Those who were in the first trimester during the three-month siege were likely to be born normal size, having caught up with typical development. However, these normal size babies developed high blood pressure, diabetes, and obesity. Contrary to this group, those who were in the third trimester during the siege, who presumably had been well nourished up until the last few months of gestation, were born small. But, these small babies stayed small their entire lives, and did not develop higher rates of obesity or disease. Surprisingly, effects continued to be seen in the offspring of the individuals who were fetuses at the time of the famine. This fasting usually entails abstaining from food or drink for the daylight hours of the month. There are groups that are automatically exempt from having to participate such as the young, sick and old but the list of exemption does not officially include pregnant women though they are most often allowed exemption. The majority of pregnant women however, choose to participate despite the hardship due to cultural and personal pressure. These outcomes were as numerous as a change in birth weight to the long term health of the affected. The studies were conducted primarily in Uganda and Iraq but had some smaller sections in Michigan and other places for control groups or specific studies. The effects on birth weight are negatively correlated with Ramadan fasting. Arab Muslim pregnancies that overlap with the Ramadan fast experienced a lower birth weight of 18 grams per child. The effect was slightly larger at a lower birth weight of grams if Ramadan fell somewhere in the first or second trimester of the pregnancy. Though the measure for disability differs by country the effect is still noticeable. For those born 9 months after Ramadan the likelihood of disability is higher than the surrounding population. The mean rate of disability in Uganda is 3. A similar effect can be observed in Iraq where the mean rate of disability is 1. The effects of exposure to the Ramadan fast can even be observed in mental disorders. In a study conducted in Uganda it was concluded that exposure to the fast, early in a pregnancy effectively doubles the likelihood of a person having a cognitive disorder of some kind. The reported signs of Anemia among the old were higher for those exposed during mid gestation, all other points in the gestation period were found to be insignificant. Anemia is caused by damage to the kidneys so the findings are consistent that the effect is noticeable during mid gestation when the kidneys are being developed. This finding shows that gene expressions can be altered via stressful experiences and then passed down to children through prenatal conditions. While the children of the Holocaust survivors had not themselves experienced Nazi inflicted trauma, they experienced the physiological and emotional trauma as if they had. When compared to Jewish families who were living outside of affected areas of Europe, the findings continued to stand: Please help improve this section by adding citations to reliable sources. Unsourced material may be challenged and removed. April Learn how and when to remove this template message Pollution may affect the health of the mother, or cross over the placenta and enter the developing fetus. Beate Ritz, a professor at UCLA, found significantly higher rates of heart malformations and valve defects in the children born to women living in highly polluted areas of Los Angeles. Maternal stress[edit] Maternal stress has been linked to a number of negative outcomes for the developing fetus. Of the women studied, those who developed PTSD following the attacks had lower basal cortisol levels than a control group. Their children, also, had lower basal cortisol levels than those not exposed to extreme prenatal stressors. Based on the findings that there was a trimester distinction in strength, conclusions can be drawn that the development of a vulnerability to stress was due at least in part to environment in utero. Women in New Orleans at the time who reported enduring multiple severe disaster experiences also had a significantly higher chance of delivering early or low birth weight children. Women who experienced the death of a close family member, friend, or spouse, or were pregnant during a wartime conflict, were more likely to have children prematurely, and the children of these women were significantly more likely than the general population to suffer from schizophrenia in adulthood. Working long hours, having temporarily employment, or reporting physically demanding job tasks showed "significant and strong" associations with poorer later birth outcomes. Out of the workers detained, served sentences and most were deported to primarily Mexico and Guatemala. Latino families feared future deportations and future raids creating psychological stress on Latinos in the area.

Criticism of theory[edit] Criticism of the fetal origins hypothesis can be aimed at the limitations of the research. Confounds abound due to the intertwined nature of environment before and after birth, as well as the correlational factors associated with poverty outcomes. Additionally, the use of historical and longitudinal data raises the question of reliability. Such interventions could instead have increased negative effects, [29] until the specific mechanisms and processes are more deeply understood by which birth and early childhood weight determine development. As stated in "Killing Me Softly: The Fetal Origins Hypothesis", "Such pre-emptive targeting would constitute a radical departure from current policies that steer nearly all healthcare resources to the sick, i. That said, the existing evidence is not sufficient to allow us to rank the cost-effectiveness of interventions targeted at women against more traditional interventions targeted at children, adolescents, or adults. For example, broadening the target population to women who might get pregnant would reduce the set of policies which are cost effective. Because the demonstrated effects range from dramatic to subtle in the wide spread areas of educational achievement, emotional stability, career trajectory, life expectancy, disease prognosis, and psychological disorders, interventions addressing the gestational period could potentially have significant impact on individual and societal levels. Proposed and in effect interventions include the following:

Chapter 5 : Fetal nutrition and adult disease | The American Journal of Clinical Nutrition | Oxford Academic

We conducted a prospective study of prenatal smoke exposure, childhood household smoke exposure, and adult active smoke exposure and mammographic density, a strong intermediate marker of breast cancer risk, in an adult follow-up of existing US birth cohorts.

Delivery of a small for gestational age infant and greater maternal risk of ischemic heart disease [1] "Delivery of a small for gestational age SGA infant has been associated with increased maternal risk of ischemic heart disease IHD. Risk of maternal IHD was evaluated in a population based cross-sectional study of 6, women with a prior live term birth who participated in the National Health and Nutrition Examination Survey , a probability sample of the U. Fetal origins of adult diabetes [2] "According to the fetal origin of adult diseases hypothesis, the intrauterine environment through developmental plasticity may permanently influence long-term health and disease. Therefore, intrauterine growth restriction IUGR , due either to maternal, placental, or genetic factors, may permanently alter the endocrine-metabolic status of the fetus, driving an insulin resistance state that can promote survival at the short term but that facilitates the development of type 2 diabetes mellitus and metabolic syndrome in adult life, especially when the intrauterine nutrient restriction is followed by a postnatal obesogenic environment. Our results suggest that the association between low birth weight and diabetes is due to factors associated with both poor fetal growth and short gestational age. Therefore the list of references do not reflect any editorial selection of material based on content or relevance. References appear in this list based upon the date of the actual page viewing. References listed on the rest of the content page and the associated discussion page listed under the publication year sub-headings do include some editorial selection based upon both relevance and availability. Int J Mol Sci: The association was specific for periconceptional exposure, reinforcing that very early mammalian development is a crucial period for establishing and maintaining epigenetic marks. When size in early life is related to later health outcomes only after adjustment for current size, it is probably the change in size between these points postnatal centile crossing rather than fetal biology that is implicated. Even when birth size is directly related to later outcome, some studies fail to explore whether this is partly or wholly explained by postnatal rather that prenatal factors. These considerations are critical to understanding the biology and timing of "programming," the direction of future research, and future public health interventions. He challenged the idea that chronic disorders such as diabetes and cardiovascular disease are explained only by bad genes and unhealthy adult lifestyles. Initially controversial, his ideas triggered an explosion of research worldwide into the relationship between early development and adult disease. This prize is given to a person who deserved to be a candidate for the Nobel Prize but would probably not be accepted by the Nobel Committee because of the way epidemiologic research is structured and conducted. In epidemiology we can seldom point towards a specific article or even a few articles or a single person who, by himself alone, changes the way we think. It is now clear that the intrauterine milieu is as important as genetic endowment in shaping the future health of the conceptus. Maternal characteristics such as weight, height, parity and ethnic group need to be adjusted for, and pathological factors such as smoking excluded, to establish appropriate standards and improve the distinction between what is normal and abnormal. Currently, the aetiology of growth restriction is not well understood and preventative measures are ineffective. Elective delivery remains the principal management option, which emphasizes the need for better screening techniques for the timely detection of intrauterine growth failure. The altered fuel supply depends on substrate availability, placental transport of nutrients and uteroplacental blood flow from mother to fetus induces alterations in the development of the fetal endocrine pancreas and adaptations of the fetal metabolism to the altered intrauterine environment, resulting in intrauterine growth retardation. The alterations induced by maternal diabetes or maternal malnutrition protein-calorie or protein deprivation have consequences for the offspring, persisting into adulthood and into the next generation. Intelligence is a combination of genetic and environmental influences relative contributions of which are not yet established and may vary over lifespan. Modified Text from [15] Note the commment made by Emeritus Professor P Pharaoh "One caveat that should be borne in mind, concerns the tests that are used to assess

cognitive function. What do these tests actually measure? Ideally they measure innate mental ability, whatever that is, at a point in time.

Chapter 6 : Pregnancy and Vaccination | Vaccines for Pregnant Women | CDC

Purpose of review: Human epidemiological and animal studies show that many chronic adult conditions have their antecedents in compromised fetal and early postnatal development. Developmental programming is defined as the response by the developing mammalian organism to a specific challenge during a.

Nutrition , fetal growth It is now widely accepted that the risks of a number of chronic diseases in adulthood may have their origins before birth. Such diseases include non insulin-dependent diabetes mellitus, hypertension and coronary heart disease. Professor David Barker and colleagues in Southampton have produced a large proportion of the data in this field over the last decade, 1 although the relationship between early life events and adult disease had been raised many years earlier. They have shown that measurements made on babies at birth, including birthweight, length, body proportions and placental weight, are strongly related to either later disease incidence coronary heart disease mortality, non-insulin-dependent diabetes 3 , 4 or risk factors for those diseases hypertension, glucose intolerance, hyperlipidaemia. That is, an event operating at a critical or sensitive period results in a long-term change in the structure or function of the organism. Programming is a well-established biological phenomenon, and there are many common and well-known examples. Female rats given testosterone during the first 4 days of life develop a male pattern of gonatotropin secretion after puberty, and despite normal ovarian and pituitary function, fail to develop normal patterns of female sexual behaviour. Similarly, transient immunization of neonatal rats against growth hormone releasing factor results in permanent impairment of pituitary growth hormone secretion and permanent impairment of growth rate. Thus a programming stimulus in fetal life is proposed to lead both to changes in size at birth and also to altered homeostatic mechanisms such as regulation of blood pressure or insulin sensitivity, which in turn result in susceptibility to disease in later life. Undernutrition was proposed early as a likely programming stimulus, although others such as excessive fetal exposure to glucocorticoids have also been proposed. It will examine the experimental basis for this hypothesis, a number of assumptions and misconceptions surrounding the hypothesis, and the need for caution in applying the results of animal experiments to the human situation. The proposal that nutrition in fetal life is a central stimulus for programming of susceptibility to adult disease is now supported by three main sets of evidence. The first is that manipulation of nutrition during pregnancy in animals can be shown to produce many of the phenomena observed in the epidemiological studies. Publication of the early epidemiological studies led us and many others to attempt to verify experimentally the link between reduced birth size and later disease risk. Size at birth is readily manipulated in experimental animals by altering maternal nutrition in pregnancy. Experimental scientists thus began by using these approaches to investigate the consequences of size at birth for postnatal physiology, producing a rapidly burgeoning literature in the area over recent years. For example, reducing the proportion of protein in the diet of pregnant rats results in offspring which have reduced size at birth and also elevated blood pressure 17 and glucose intolerance 18 â€” 20 in adult life. A variety of experimental approaches to reduce maternal nutrition in pregnancy have lead to similar observations in rats, 21 guinea pigs 22 and sheep. These studies have shown that women exposed during pregnancy to the nutritional limitation imposed by severe famine have offspring with reduced birth size 24 and an increased risk of glucose intolerance 25 and obesity in adult life. The third line of evidence supporting nutrition as a likely programming stimulus is essentially that of biological plausibility, based on current knowledge of the regulation of mammalian fetal growth. There is ample evidence from cross breeding and embryo transplant experiments that size at birth is largely determined by the maternal uterine environment, with relatively little influence of parental genotype. Thus fetal growth in late gestation is normally regulated by fetal nutrient supply. Since nutrition has such a central role in the regulation of fetal growth, it is a good candidate for a programming stimulus, holding a central role in the link between size at birth and subsequent disease risk. Relatively large changes in maternal diet may have little impact on fetal nutrition if the capacity of the fetal supply line allows a large margin of safety for fetal growth. Conversely, common clinical causes of impaired fetal growth such as maternal hypertension associated with reduced uterine blood flow, or placental infarcts

resulting in reduced placental transfer capacity, may severely limit fetal nutrient supply without a corresponding change in maternal nutrition. Much confusion and debate in the literature about the relevance of nutrition to human fetal growth has arisen from failure to make this distinction between maternal nutrition relatively easy to measure but relatively less important and fetal nutrition very difficult to measure but very important. Much of our knowledge of fetal nutrition comes from studies in sheep, which are widely used in studies of fetal physiology because of their large size, long gestation and relative ease of surgical manipulation. These characteristics allow chronic fetal catheterization. Vascular catheters and other monitoring devices such as electrodes and growth measuring devices can be surgically placed in the ewe and fetus, the fetus returned to the uterus and the ewe allowed to recover. Fetal blood can then be sampled and the fetus studied over many days or weeks in the relatively undisturbed conditions of intrauterine life in vivo. Although there are important interspecies differences which must be considered, there are also remarkable similarities in the physiology of fetuses of different species. However, the relative proportions of these fuels varies with the species and with the time in gestation Table 1. For example, the human fetus is very dependent on glucose as a major oxidative substrate, while the sheep fetus derives an increasing proportion of its carbon requirements from lactate as gestation proceeds. Perhaps the most obvious placental influence on fetal nutrition is via its capacity to transport nutrients from the maternal to the fetal circulation. This transfer capacity is influenced by such factors as placental surface area and availability of specific nutrient transporters on the membranes. Recent evidence suggests that these may be influenced in turn by the maternal nutritional environment. In sheep, for example, the placenta converts glucose to lactate which is then released into the fetal circulation where it provides an important fetal oxidative fuel. Similarly, placental metabolism is important in fetal amino acid supply, with virtually all fetal glycine requirements synthesized in the placenta rather than taken up from the maternal circulation in sheep 39 and probably in human pregnancy. The placenta also influences fetal nutrition via its own metabolic demand for nutrients. Similarly the fetus has been shown to export amino acids back to the placenta when supply is limited. Finally, the placenta will influence fetal nutrition because it produces hormones which in turn may influence fetal and maternal nutritional supply. Both placental lactogen and growth hormone are produced by the placenta in large amounts. They contribute to maternal insulin resistance, increasing the availability of glucose and other nutrients in the maternal circulation for transfer to the fetus. However, there are important species differences, particularly in maternal metabolism and placental structure and function, which will determine the effects on the fetus of a given nutritional insult to the mother. The most thoroughly studied animal, the sheep, is a ruminant. One result of this digestive arrangement is that circulating blood glucose is produced endogenously by the maternal liver via gluconeogenesis rather than being derived directly from the products of digestion in the gut. Reduced maternal dietary intake thus leads to reduced production of gluconeogenic substrates from the rumen, largely short chain fatty acids and amino acids. This results in a prompt large fall in maternal blood glucose concentrations. This is less likely in human pregnancy as maternal undernutrition leads to relatively smaller changes in circulating maternal blood glucose concentrations and hence fetal glucose supply. The effect of a change in maternal nutrition will also vary between species according to the growth rate and body composition of the offspring. Thus a very small animal with a short gestation, such as the guinea pig, has fetuses with a relatively high growth rate in late gestation Table 2. Such fetuses must allocate a large proportion of total nutrient supply for growth. Limitations on nutrient supply will therefore have a much bigger effect on growth rate than they would in a larger species with a smaller relative growth rate. Similarly, in species with a large proportion of body fat at birth such as the guinea pig or human infant, a given rate of tissue acquisition will require a higher energy input because of the high energy density of the fat. Once again, this will increase the proportion of available energy supply which must be directed towards tissue growth. Such species would therefore be more vulnerable than other species to restrictions in energy supply in terms of their effects on fetal growth. In the sheep, ketones cross the placenta in only small amounts, 54 but are oxidized by the placenta 55 resulting in increased lactate production, apparently sparing what glucose is available for fetal use. However, the human placenta is permeable to ketones and fatty acids 47 , 56 and fetal tissues may directly oxidize ketones as a substitute for glucose, particularly in organs such as the brain. In sheep maternal fasting reduces fetal glucose

supply but lactate supply is relatively maintained, whereas fasting in pregnant women leads to relative maintenance of fetal glucose supply but increased availability of ketones and fatty acids. However, these examples serve to illustrate how fetal nutrition may be critical in the regulation of fetal growth, while maternal nutrition may have very variable effects depending on such factors as species differences in metabolism and placental function. Distinguishing birthweight from fetal growth The initial epidemiological studies linked birthweight to subsequent disease risk. Later studies examined these risks in relationship to various body proportions at birth such as ponderal index thinness , abdominal circumference, etc. Such apparent post hoc analysis is in practice an attempt to get closer to the origin of the association; that fetal nutrition as a programming stimulus affects fetal growth rather than birthweight. However, the distinction can be readily demonstrated in animals. Fetal sheep growing rapidly in late gestation slow their growth promptly in response to 10 days of maternal undernutrition and resume growth on maternal and hence fetal refeeding. When examined after 10 days of refeeding, these fetuses have the same birthweight and length as control fetuses of well-fed ewes, but have increased heart and kidney size and increased blood pressure. A similar situation can be imagined in human pregnancy where fetuses of similar birthweight may arrive at that point via very different growth trajectories Figure 2. It seems likely that these trajectories would be associated with different patterns of physiological function and likely programming and thus disease risk, although this remains to be demonstrated. If fetal growth is poorly reflected in birthweight, then it seems likely that body proportions would be more informative. Although this seems a reasonable hypothesis, there are few data to assist, and many common assumptions in this area are excessively simplistic. One common assumption is that body proportions provide information about the timing of nutritional insults leading to the limitation of fetal growth. Thus a baby which is proportionately small in weight, length and head circumference at birth is presumed to have suffered from nutrient limitation in early pregnancy, while a baby of similarly low birthweight who is relatively long and thin is presumed to have suffered nutrient limitation in late pregnancy. However, careful examination of large human data sets have failed to find any evidence of two distinct populations in this regard. Furthermore, measurement of fetal growth by ultrasound showed no clear differences in timing or pattern of growth changes in babies found at birth to be either symmetrically or asymmetrically growth restricted. Indeed, studies of maternal undernutrition in sheep have shown that reduced ponderal index thinness is seen in fetuses exposed to undernutrition from early or mid gestation through to term, but not in fetuses exposed only in late gestation. Contrary to expectation, exposure only in early or mid gestation results in increased ponderal index. However, simple limitation of substrates to growing organs leading to reduced size of that organ does not explain the complex effects observed. Maternal protein restriction in pigs results in reduced fetal weight and length at mid-gestation at a time when the fetus is extremely small and fetal protein requirements for growth are most unlikely to have been limiting by this time. Reduced abdominal circumference has been assumed to reflect reduced liver size 63 and this has been used as a possible explanation of the relationship observed between abdominal circumference at birth and lipid metabolism in adulthood. This apparently reasonable assumption has recently been questioned by findings that ultrasound measurements of growth restricted human fetuses show little relationship between liver size and abdominal circumference. There is a need for more extensive pathological studies to determine the true relationship between birth measurements and organ size in human infants. There is certainly good evidence of redistribution of cardiac output in fetuses exposed to hypoxia, with maintenance of blood flow to essential organs such as the brain and heart at the expense of other organs such as the gut and skin. Hypoxaemia appears to occur late in the process of growth restriction in human fetuses, and many IUGR fetuses are not hypoxaemic on direct measurement in utero although head sparing can be demonstrated in these fetuses. Glucose uptake into many tissues is mediated by insulin, and fetal insulin secretion is regulated by glucose and amino acid supply. However glucose uptake into the brain does not require insulin. Thus limitation of glucose and amino acid supply to the fetus will reduce circulating insulin concentrations and glucose uptake into peripheral tissues such as muscle, sparing the available glucose for uptake into the brain which is insulin independent. In addition, as described above, fasting in women increases the supply of ketones to the fetus 47 and the fetal brain has been shown to preferentially take up and oxidize ketones. However, it is likely to reflect

also complex metabolic adaptations to limitations in fetal nutrient supply including an altered hormone environment and altered substrate availability. Timing and balance of nutrients Much of the discussion so far has addressed nutrition in the general terms of overall macro-nutrient supply. However, it is increasingly apparent that the balance of macro- and micro-nutrients reaching the fetus and the timing of any changes in their supply is likely to be important in determining the effects on fetal growth and later physiology. Most information on the balance of nutrients has so far come from human studies. Although the randomized controlled trials of maternal dietary supplements show relatively little effect on birthweight overall, supplements with a relatively high proportion of calories provided as protein actually resulted in reduced mean birthweight. However, amino acids are transported across the placenta to the fetus by a number of amino acid transporters. Increasing availability of some amino acids may therefore result in competition for transporters and reduce the availability of other amino acids to the fetus, potentially limiting growth. Individual amino acids may also have critical roles. Rats fed an isocaloric low protein diet have offspring whose pancreatic islet cells have impaired insulin release. However, supplementation of the maternal low protein diet with taurine alone restored the insulin secretion of the fetal islets. Glycine, for example, appears to be a conditionally essential amino acid for a number of important metabolic compounds including nucleic acids, collagen, haeme and keratin. Fluctuations in nutrient supply to the fetus may also have important effects on fetal growth and the programming of later disease risk.

Chapter 7 : Abnormal Development - Developmental Origins of Health and Disease - Embryology

Fetal origins of adult diabetes "According to the fetal origin of adult diseases hypothesis, the intrauterine environment through developmental plasticity may permanently influence long-term health and disease. Therefore, intrauterine growth restriction (IUGR), due either to maternal, placental, or genetic factors, may permanently alter the.

Reaffirmed Committee on Genetics This document reflects emerging clinical and scientific advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Family history plays a critical role in assessing the risk of inherited medical conditions and single gene disorders. Several methods have been established to obtain family medical histories, including the family history questionnaire or checklist and the pedigree. The screening tool selected should be tailored to the practice setting and patient population. It is recommended that all women receive a family history evaluation as a screening tool for inherited risk. Family history information should be reviewed and updated regularly, especially when there are significant changes to family history. Where appropriate, further evaluation should be considered for positive responses, with referral to genetic testing and counseling as needed. Certain types of cancer, such as breast cancer and colon cancer, appear more frequently in some families, as do some adverse birth outcomes. Coronary artery disease, type 2 diabetes mellitus, depression, and thrombophilias also have familial tendencies. The goal of this initiative is to educate both health care providers and patients about the value of collecting a family history as a screening tool and to increase its use and effectiveness in clinical care by simplifying the collection process and analysis of the family history 1. Over the past 20 years, the Human Genome Project has afforded us a better understanding of the effect of genetic variation on health and disease. This has furthered research in identifying genotypeâ€”phenotype correlations and enhanced the ability to predict those at risk of developing inherited medical conditions. With increased awareness of the importance of using family history as a screening tool and of the value of preventive measures and increased surveillance, there is hope for improved outcomes. Tools for Collecting the Family History Several methods have been established to obtain family medical histories, each with its own advantages and dis-advantages. A common tool used in general practice is the family history questionnaire or checklist. Having the patient complete the questionnaire at home allows extra time for the patient to contact family members and provide more accurate information. Direct patient questioning permits clarification of medical terminology that may be unclear to the patient. Any positive responses on the questionnaire should be followed up by the health care provider to obtain more detail, including the relationship of the affected family member s to the patient, exact diagnosis, age of onset, and severity of disease 2. Another family history assessment tool, commonly used by genetics professionals, is the pedigree. The health care provider may decide to complete a detailed pedigree or refer the patient to a genetics professional for further evaluation. A pedigree ideally shows at least three generations and involves the use of standardized symbols, which clearly mark individuals affected with a specific diagnosis to allow for easy identification see Fig. The pedigree may visibly assist in determining the size of the family and the mode of inheritance of a specific condition, and it may facilitate identification of members at increased risk of developing the condition see Box 1. A pedigree should indicate the age of individuals; if deceased, the age and cause of death; and any relevant health history, illnesses, and age of onset. If any genetic testing has been performed on family members, the results should be indicated on the pedigree. The ethnic background of each grandparent should be listed as well as any known consanguinity 3. A general inquiry about the more distant relatives should be made in case there is a possible X-linked disorder or autosomal dominant disorder with reduced penetrance 4. Red Flags for Genetic Conditions Family history of a known or suspected genetic condition Ethnic predisposition to certain genetic disorders Consanguinity blood relationship of parents Multiple affected family members with the same or related disorders Earlier than expected age of onset of disease Diagnosis in less-often-affected sex Multifocal or bilateral occurrence of disease often cancer in paired organs Disease in the absence of risk factors or after application of preventive measures One or more major malformations Developmental delays or mental retardation Abnormalities in growth growth restriction, asymmetric growth, or excessive growth Recurrent

pregnancy losses two or more Modified from the National Coalition for Health Professional Education in Genetics NCHPEG. Quick Tips for Risk Assessment. Retrieved on September 3, The screening tool selected should be tailored to the practice setting and patient population, taking into consideration patient education level and cultural competence. Whether the pedigree or questionnaire is used, it is important to review and update the family history periodically for new diagnoses within the family and throughout pregnancy as appropriate. A family history screening tool will allow the health care provider to stratify levels of risk 5. The Preconception Period Women often discuss their pregnancy plans with their obstetricianâ€"gynecologist before conception. The preconception period is an ideal time to provide personalized recommendations based on family history. The preconception consultation is also an optimal time to review family history and discuss with a couple the option of undergoing carrier screening for genetic conditions. It is also an opportunity to address any medication concerns before pregnancy eg, the importance of taking a folic acid supplement and avoiding medications such as angiotensin-converting enzyme inhibitors and to ensure that medical conditions are being carefully evaluated. It is important to obtain the family and medical history of both the patient and her partner, including their ethnic backgrounds, any adverse pregnancy outcomes as a couple or with other partners, and any known causes of infertility if applicable. Positive responses will need to be followed up by performance of appropriate risk assessment, testing, and genetic counseling if needed. Any genetic counseling and testing that can be completed before conception is beneficial to the couple, allowing a broader array of options and more time for decision making. Couples may decide not to conceive, or they may consider using a gamete donor or obtaining a preimplantation genetic diagnosis if available. A patient who has had a past adverse pregnancy outcome or has a family history of other adverse pregnancy outcomes, such as miscarriage, preterm birth, a newborn screening test result indicating an abnormality, or birth defects, might be at increased risk of these disorders. Because both genetic and environmental factors may contribute to these outcomes, advising a patient that she is at increased risk of an adverse pregnancy outcome based on family history might motivate her to reduce her environmental risk by, for example, stopping smoking or achieving a healthy weight 8. However, these disorders often have complex geneticâ€"environmental interactions for which environmental modifications can improve the outcome or delay the onset of symptoms 5. For example, diet changes, weight loss, exercise, and glucose monitoring may improve the outcome for an individual with a family history of type 2 diabetes mellitus. Similarly, individuals at risk of cardiovascular disease can benefit from environmental modifications, such as achieving normal blood pressure and cholesterol levels, and those at risk of osteoporosis can undertake calcium supplementation, weight bearing exercise, and bone density screening to improve their long-term bone health. Some family histories show obvious evidence of cancer risk, such as a family in which there are several members with early-onset breast cancer or colon cancer. In assessing family history of cancer risk, it is important to check for evidence of cancer that might be linked to a single underlying genetic cause, such as Lynch syndrome, in which colon, endometrial, ovarian, urinary, or gastrointestinal cancer may be associated with a single familial gene mutation. The proband, or patient noted in generation III-6, is at increased risk of developing type 2 diabetes not only because of her family history of the disease but also because of her development of gestational diabetes in her previous pregnancy. This patient should be counseled not only about maintaining an appropriate diet and exercise routine to lower her risk but also about obtaining an earlier glucose screening in her current pregnancy. Limitations Adoption and limited family size might trigger a lower threshold in detecting family history. Recommendations All women should have a family history evaluation as a screening tool for inherited risk. Where appropriate, further evaluation should be considered for positive responses, with referral to genetic services as needed. Resources The following resources are for information purposes only. Referral to these resources and web sites does not imply the endorsement of the American College of Obstetricians and Gynecologists. Further, the American College of Obstetricians and Gynecologists does not endorse any commercial products that may be advertised or available from these organizations or on these web sites. This list is not meant to be comprehensive. The exclusion of a source or web site does not reflect the quality of that source or web site. Please note that web sites and URLs are subject to change without notice. National Society of Genetic Counselors:

Chapter 8 : Prenatal nutrition - Wikipedia

This article briefly outlines some of the key prenatal and early life influences on the development of adult weight and calendrierdelascience.com the s, intriguing research from British epidemiologist David Barker and colleagues sparked a flutter of research into what was then called the "fetal origins hypothesis" of chronic disease.

Chapter 9 : Pregnancy history linked to risk for Alzheimer's disease - calendrierdelascience.com

More broadly, research has increasingly focused on how a range of factors during pregnancy and gestational development can impact adult health. This work often is placed under the rubric of "fetal origins," where maternal stress, malnutrition, low birth weight, and other factors that impact development in the womb can create long-term.