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Adolescent pregnancy: an overview in developed and developing nations

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ABSTRACT

The purpose of this article is to draw attention to biological risk factors that operate during adolescent pregnancy. One of these, maternal growth, is common among pregnant adolescents in the United States (30-50%) and is likely to be even more prevalent in areas of the world where childhood malnutrition is or was prevalent. Young still-growing women do not mobilize fat stores during the third trimester. Inhibition of maternal fat oxidation leads to reduced infant birth weight. Continued maternal growth has implications for maternal obesity, for early onset insulin resistance and for an increased risk of chronic disease, particularly Type 2 diabetes, in both the mother and her children.

KEY WORDS: *Adolescent pregnancy, pregnancy complications, pregnancy outcome, nutrition, growth, birth weight.*

INTRODUCTION

A secular increase in growth accompanied by decline in age at menarche has occurred throughout the world.[1] Long-term changes in nutrition and public health are held as the causes of secular change as the trend has paralleled improvements in sanitation, food availability and health care, the declining prevalence of childhood infectious diseases and of clinically detectable nutritional deficiency disorders.[2,3] In Europe age at menarche fell by three months per decade during the past century. In the United States secular decline has been approximately two months per decade.[1,2] In Mexico the decline in age at menarche has been estimated to be between 1.3 and 5 months/decade for women from the Yucatan and between 3 to 9.5 months/decade for rural girls and women from Oaxaca.[4-6]

EARLY MENARCHE AND RISK OF ADOLESCENT PREGNANCY

Earlier age at menarche is related to an earlier onset of sexual activity and, since teenagers are unlikely to use contraceptives, an increased risk of adolescent pregnancy.[7,8] The probability of engaging in sexual intercourse before menarche is rare for US adolescents.[9,10] For example at the age of 15, the likelihood of being sexually active was 55% when menarche was experienced at age 11 or less versus 32% for menarche by age 14 or more.[9] Others have confirmed these findings,[11] extended them to US teenagers of Hispanic descent[12] and to young women from the developing world.[13,14]

Rates of adolescent fertility among US teenagers aged 15-19 years are among the highest (49.8 per thousand) in the developed world.[15]

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The recent rate of adolescent childbearing in Mexico, 65.8 per thousand, is slightly above average rates for Latin America and the Caribbean.[15] Adolescent childbearing rates in both the US and Mexico have declined since the year 2000, by 4% (from 51.9 per thousand) for American and 10.6% (from 73.6 per thousand) for Mexican teenagers.[15] However, the age structure of the Mexican population is younger than the US. Thus, even if adolescent fertility rates of the two countries were identical, since there are many more teenagers in Mexico than in the United States, the number as well as the proportion of births to adolescents in Mexico would be substantially higher compared to the United States.[16-17]

ADOLESCENT PREGNANCY COURSE

In the US and elsewhere, the frequency of most complications of pregnancy (placental abruption, placenta previa, premature rupture of the membranes, urinary tract infections and anemia) and characteristics of labor (duration by stage, laceration and episiotomy, use of amniotomy or pitocin augmentation) are similar between adolescent and mature women. Data suggest that Cesarean section is now less frequent among teenagers than mature women and that differences between age groups in anemia and pregnancy induced hypertension have declined. In the past, but not at present, these risks were found in excess among adolescents.[18] Despite this, and although absolute mortality is low, the relative risk of maternal death is increased for the youngest pregnant US teenage women, that is those under the age of 15 years.[19]

Data from Latin America, including Mexico, suggest that pregnancy risk experienced by adolescents is greater than in the US.[20] In comparison to women aged 20-24 years, adolescents are more likely to be anemic, and to have higher risks of operative vaginal delivery but not Caesarean section, and of episiotomy, postpartum sepsis and puerperal endometritis. Although not statistically significant, the youngest mothers (< 16 years) tended to have a higher risk of pre-eclampsia and eclampsia. Consistent with the data on US teenagers, they also experienced higher rate of maternal death; in this instance absolute risk was higher than in the US; relative risk was increased 4-fold compared to mature women aged 20-24 years from Latin America.[20]

ADOLESCENT PREGNANCY OUTCOME

Teenagers from both the United States and Latin America who give birth have an increased risk of poor pregnancy outcomes including preterm delivery, low birth weight and small for gestation births.[20-22] Social factors are often invoked in explanation. Most teenage pregnancies are unplanned and young women often are unaware that they are pregnant and delay entry to prenatal care.[23-24] In the United States rates of adolescent childbearing are highest in disadvantaged at-risk minority women, increasing twofold or more in Blacks and Hispanics compared with Whites.[21] Other risk factors shared by pregnant adolescents in developed and developing countries include poverty, unpartnered status, lack of education, poor diet and nutrition, poor school performance and a family history of early childbearing.[23-25] There are, in addition, inherent biological factors that operate during adolescent pregnancy to impair fetal growth. These factors act even when the mother receives adequate prenatal care and lives under favorable social and economic circumstances.[26]

MATERNAL GROWTH DURING ADOLESCENT PREGNANCY

Adolescent growth and adolescent pregnancy often coincide. In one study 63% of pregnant teenagers had postpartum hand-wrist radiographs with open epiphyses denoting skeletal immaturity and the potential to continue to grow.[27-28] During pregnancy, it is difficult to distinguish maternal growth in stature from background noise caused by pregnancy related changes such as postural lordosis and vertebral compression. In order to detect maternal growth, in Camden (New Jersey) we used the Knee Height Measuring Device (KHMD) to monitor growth and found that 30-50% of pregnant teenagers grew by KHMD.[29-33] Because childhood malnutrition extends the period of growth, in the devel-

oping world maternal growth must occur more frequently than in the United States. In Mexico, Reyna-Samano and colleagues observed a mean increase of 15 mm in the stature of young teenagers aged 13-16 in the first year postpartum.[34]

WEIGHT GAIN AND MATERNAL GROWTH

During pregnancy skinfold thickness increases as subcutaneous fat accumulates through the 1st and 2nd trimester; in the 3rd trimester skinfold thickness decreases, as fat stores are mobilized.[35,36] In contrast, during the 3rd trimester growing adolescents maintain or add to fat stores.[33,37] By the postpartum, body fat and weight are increased. In concert with these increases growing adolescents have significantly larger gestational weight gain and retain a significantly greater proportion of the weight they had gained during pregnancy.[33,37]

BIRTH WEIGHT, PREGNANCY OUTCOME AND MATERNAL GROWTH

Maternal growth is associated with greater gestational weight gain and increased fat stores, which should give rise to greater fetal growth and size. Growing gravidas bear infants who weigh significantly less at birth by 150-200 grams, when compared with infants of non-growing adolescents or with those of mature women.[29-33,38] In Peru, Frisancho used adolescent height below parental height as a rough gauge of growth during adolescent pregnancy.[39-40] Young teenagers (13-15 years) who were still growing had smaller infants than teenagers who had completed growth and lower placental weights.

BIRTH WEIGHT, MATERNAL FAT ACCRUAL AND MATERNAL GROWTH

Continued accrual of fat by the mother during the 3rd trimester is associated with decreased birth weight even when growth is not taken into consideration.[33,36] Pregnant adolescent women gain and retain more fat than do mature women and this tendency is amplified with maternal growth. Gravidas with high fat accumulations (> 75th percentile) who were still-growing had infants with the large decrements in birth weight (-170 g), while growers with a lesser accumulations of subcutaneous fat had smaller decrements (105-135 g) in birth weight. Thus greater accumulation of third trimester fat stores appears to further compromise fetal growth.[33]

MATERNAL GROWTH AND DIET

Maternal diet during pregnancy buffers effects of maternal growth on the fetus. When a still-growing young woman has an energy intake that is at or above the Recommended Dietary Allowance (RDA) for pregnancy, birth weight is modestly diminished (-86 g), but when maternal energy intake is low (< RDA for pregnancy) and maternal growth continues, the deficit in birth weight is quite substantial (-320 g). In the absence of maternal growth, there was no relationship between low maternal energy intake and infant birth weight (-6 g).[33]

MATERNAL GROWTH AND COMPETITION FOR NUTRIENTS

Maternal-fetal competition is theoretically marked by decreased blood flow to the uterus and fetus during the period of maximal fetal growth in the 3rd trimester and reduced transmission of nutrients from mother to fetus. Umbilical artery velocity waveforms reflect placental vascular resistance and thus indirectly measure blood flow velocity. In Doppler velocimetry, the most common index is the systolic/diastolic (S/D) ratio. A high-resistance circuit, which denotes abnormal flow, is characterized by a high S/D ratio. At 30-34 weeks' gestation, S/D ratios of 3 or greater are considered high, and if persistent, are associated with lower infant birth weight. Adolescent growing by KHMD were twice as likely to have a high S/D ratio at 32 weeks' gestation. A high S/D ratio, in turn, was associated with a 285 g reduction in birth weight and maternal growth with a greater than 3-fold risk of small for gestation infants.[38]

MATERNAL GROWTH AND CORD BLOOD NUTRIENTS

In order to examine nutrient transmission during the 3rd trimester, two micronutrients, ferritin and folate, were studied as markers of the competition in cord bloods of growing and non-growing teenagers. Consistent with the hypothesis of maternal-fetal competition there were significantly lower concentrations of the two

marker nutrients in cord bloods of growing gravidas. Thus, based upon two parameters, an inadequate maternal-fetal exchange is associated with maternal growth by KHMD, thus supporting the hypothesis that there is a competition for nutrients between a young, still-growing mother and her fetus.[41]

MATERNAL GROWTH WITH LEPTIN AS A BIOMARKER

We found a biomarker for maternal growth in leptin a polypeptide hormone, the product of the obese gene and the first hormone found to be released by the adipocyte. Leptin levels rise during pregnancy then decline in the postpartum.[42] A portion of the increase with pregnancy is placental in origin with high leptin levels indicating placental insufficiency.[43] By the 3rd trimester leptin levels had surged primarily in the teenagers and by 4-6 weeks postpartum growing gravidas had leptin levels above those taken at entry to care, while the others gravidas had leptin levels that fell below entry levels.[44]

MATERNAL GROWTH, LEPTIN AND PREGNANCY OUTCOME

In growing gravidas, weight and skinfolds typically are lower at entry, and are gained at a greater rate during pregnancy and retained in the postpartum. Likewise, the leptin surge was positively associated with all of the above changes: an increased rate of gestational weight gain, greater increases in skinfolds and increased postpartum weight retention. The leptin surge was also associated with reduced infant birth weight. Gravidas in the highest quartile of the surge had infants weighing approximately 250 grams less than gravidas in the lowest quartile of the leptin surge. The leptin surge was also associated with increased risks of infant low birth weight ($< 2{,}500$ g) and restricted intrauterine growth. Gravidas in the highest quartile had a greater than 5-fold increase in risk of infant low birth weight and approximately a 7-fold increased risk of intrauterine growth restriction.[44]

We suggest that the leptin surge marks the mother's inability to oxidize her fat stores. Reduced lypolysis of maternal fat stores reduces production of free fatty acids, altering the mother's insulin resistant state. Growing teenagers may not develop insulin resistance of pregnancy and make the shift in energy metabolism from carbohydrate to fat. Inhibition of maternal fat oxidation would indirectly reduce glucose for fetal growth and permit the mother to retain fat accrued in early pregnancy. Both conditions characterize maternal growth during pregnancy.[44]

MATERNAL GROWTH AND A REPEAT ADOLESCENT PREGNANCY

Teenagers who grew during two pregnancies had a greater increase in postpartum weight (a 22% increase) compared to teenagers who grow during only one pregnancy (16% increase) or teenagers who did not grow in either pregnancy (9% increase). When compared to pregnant teenagers who never grew, teenagers growing in one pregnancy were 3.4 times more likely to become newly overweight or obese by age 18; those growing in two pregnancies were 5 times more likely to have become overweight or obese.[37,45]

ANIMAL MODELS OF MATERNAL GROWTH

Across species, the drive to tissue synthesis in the still-growing young mother appears to take precedence over the nutrient requirements of the fetus and uterus. Young and growing rat dams, mated at vaginal opening and fed *ad libitum*, had larger gestational weight gains compared to mature pregnant dams. Growing dams delivered significantly smaller pups with retarded skeletal ossification and increased mortality.[46] Adolescent ewes, implanted with ova from superovulated adults inseminated by a single sire, were fed a diet formulated to insure low or rapid maternal growth.[47] Maternal weight was greatly increased among the super-fed young ewes but placental weight was decreased. During late gestation, these pregnancies were characterized by reduced uteroplacental blood flow and nutrient uptake that gave rise to fetal hypoxia, hypoglycemia, and low levels of insulin. During pregnancy, risk of spontaneous abortion was increased, and at delivery fetal growth restriction and pup mortality were increased.[48-50]

Maternal levels of leptin were also increased. Administration of growth hormone during the period of placental growth prompted changes in maternal nutrient deposition from fat to protein. This, in turn was associated with increased fetal growth and size at birth.[48] It is not known if there are changes in the level of placental growth hormone or other hormones of pregnancy in still-growing adolescent girls.

LONG TERM CONSEQUENCES

For the mother

One of the effects of maternal growth during an adolescent pregnancy may be an increased risk of maternal overweight and obesity from an early age. Adolescence is a critical period for the development of obesity and only a fraction (20%) of obese adolescent girls return to a more normal weight within 10 years.[51-52] Adolescent obesity increases risk of chronic disease related to insulin resistance, including type 2 diabetes, coronary heart disease (CHD) and arthritis.[53] Case-control studies show associations between teenage pregnancy, parity or gravidity and cardiovascular disease.[54]

For the infant

One of the effects of exposure to maternal growth during fetal life is restricted fetal growth and lower weight at birth. Infants who are low birth weight (LBW) or who are growth restricted have an increased future risk of chronic diseases linked to insulin resistance. This was first noted by Barker and colleagues in their study of a cohort born in Hertfordshire, England.[55] As adults, low weight infants developed disorders linked to cardiovascular disease through insulin resistance. These included hypertension[56] and pregnancy induced hypertension[57], diabetes mellitus[58], gestational diabetes[59] as well as their precursor states: high cholesterol, high fibrinogen, and the Insulin Resistance Syndrome.[55,60] Alterations in insulin resistance and/or glucose tolerance were noted in low birth weight infants as early as the age of 7 years of age.[61]

CONCLUSIONS

Underlying metabolic factors associated with maternal growth during adolescent pregnancy favor storage over oxidation and promote fat deposition and retention during pregnancy and the postpartum. It is not known if these factors include changes in the pregnancy hormones including placental growth hormone as they do in animal models of growth during adolescent pregnancy.

Childhood malnutrition lengthens the period over which maternal growth occurs, consequently women with poor nutritional status during childhood are likely to be growing more actively and for a longer time than their US counterparts. The competition for nutrients between a young still growing pregnant woman and her fetus increases risk of low birth weight due to intrauterine growth restriction. In a population at high risk of type 2 diabetes,[62] particularly when more than one child is born to a still-growing mother, this could lead to impaired glucose tolerance and advance the onset of obesity, gestational diabetes and type 2 diabetes for both mother and child.

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RESUMEN

El propósito de este artículo es llamar la atención acerca de los factores de riesgo biológicos que operan durante el embarazo en adolescentes. Uno de éstos, el crecimiento materno, es común entre las embarazadas adolescentes de Estados Unidos (30 a 50 por ciento) y es probable que sea más prevalente en áreas del mundo donde la mala nutrición es o ha sido frecuente. Las mujeres jóvenes que aún continúan creciendo no movilizan sus reservas de grasa durante el tercer trimestre. La inhibición de la oxidación de grasas maternas provoca una reducción del peso al nacimiento del infante. La continuación del crecimiento materno tiene implicaciones para la obesidad materna, para el inicio temprano de la resistencia a la insulina, para el incremento en el riesgo de enfermedades crónicas, particularmente la diabetes tipo 2, tanto en la madre como en su hijo.

PALABRAS GUÍA: *Embarazo en la adolescencia, complicaciones del embarazo, consecuencias del embarazo, nutrición, crecimiento, peso al nacer.*

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