Title: PREGNANCY AND FETAL METABOLISM

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Interpretative Summary:

Technical Abstract: This chapter deals with quantitative aspects of macronutrient metabolism and its regulation in maternal and conceptus tissues in vivo, emphasizing data and concepts generated or revised during the decade since publication of a similar chapter in the first edition of this book (see Bell, 1993). Recent findings on the regulation of nutrient partitioning among maternal tissues, the placenta, and fetus(es) are highlighted, as is new information on placental transport mechanisms.
Pregnancy is a diabetogenic state associated with an increase in maternal glucose to supply the fetus and accompanying insulin resistance. Skeletal and bone changes. Pregnancy is associated with reversible bone loss because of increased bone turnover to supply the fetus with calcium. Summary of maternal changes. The heart rate and stroke volume increase to maintain cardiac output and arterial pressure when systemic vascular resistance falls. If red blood cell production cannot keep pace with increasing plasma volume, hematocrit falls during pregnancy, leading to physiological anemia. “Maternal-Fetal Metabolism in Diabetes. If the maternal pancreatic insulin response is inadequate, maternal and, then, fetal hyperglycemia results. This typically manifests as recurrent postprandial hyperglycemic episodes. These postprandial episodes are the most significant source of the accelerated growth exhibited by the fetus. Surging maternal and fetal glucose levels are accompanied by episodic fetal hyperinsulinemia. Fetal hyperinsulinemia promotes excess nutrient storage, resulting in macrosomia. The energy expenditure associated with the conversion of excess glucose into fat causes depletion. The carbohydrate metabolism disorders during pregnancy contribute to the development of DF, which is most frequently characterized by macrosomia or intrauterine growth restriction (IUGR) and is one of the most serious and specific manifestations of maternal DM in the newborn, which increases the risk of birth trauma, perinatal morbidity and mortality [9]. DM in pregnant women affects the fetal heart structurally (cardiac malformations, hypertrophic cardiomyopathy) and functionally (even in the absence of structural changes) with long-term consequences. Later in pregnancy, bone metabolism may be further stimulated by the increased placental transfer of calcium to meet fetal demands for ossification and general metabolism, which far exceeds maternal intestinal absorption. Biochemical markers of maternal bone turnover are elevated in pre-eclampsia. Biochemical markers of bone turnover are reliable indices for measuring changes of bone formation and resorption, reflecting the dynamics of bone metabolism at the cellular level. During normal pregnancy, major changes occur in maternal calcium homeostasis and bone metabolism, in order to fulfill the calcium demand of the fetus for skeletal growth and mineralization.