Endocrine factors involved in intrauterine growth are synthesized both by placenta and by the fetus. Placenta shows very complex hormonal function: progestational, placental, fetal growth and regulatory. **Placental factors with a progestational role** are: progesterone, estrogen, chorionic gonadotropic hormone (hCG), inhibin (A, B) and activin (A, B), CRH. **Progesterone** (1,2,3,4) is necessary to enable the maintenance of pregnancy, by relaxing the uterine muscles. It is produced at the beginning of the pregnancy by ovarian corpus luteum, under the hCG action, then follows a transition period (between gestational weeks 6-12), in which it is synthesized by the placenta (syncytiotrophoblast), after which the placenta becomes the dominant site of synthesis. Levels increase steadily until a few weeks ahead of time in which the placenta becomes the dominant site of synthesis. The production level is increased by 1000 times during pregnancy, up to 100 nmol / L, exceeding 80 mg / day of hormone. The release of the three compounds by the syncytiotrophoblast and require an effort developed by the fetus, mother and the placenta.

**Estrogens** (1,2,3,4,9) prepare and maintain pregnancy and trigger the parturition. They are synthesized by syncytiotrophoblast and require an effort developed by the fetus, mother and the placenta.

Three major estrogens compounds occur in pregnancy estriol (E3) - the major product, estradiol (E2), estrone. The release of the three compounds by maternalplacentar unit is predominantly in the maternal circulation. The production level is increased by 1000 times during pregnancy, up to 100 nmol / L, exceeding 80 mg / day of hormon. By comparison, at the cycle, estrogen levels are 1000 pmol / 1. In the past, measurement of maternal serum or urinary estriol is usually made in the third trimester of pregnancy to assess fetal wellbeing, but now it no longer shows the same value. Fetus and mother are protected by high titres of progesterone by the formation of conjugated steroid compounds. Estrogens prepare and maintain pregnancy, cause vasodilation, with an increased flow to the uterus and miometrial hypertrophy; they determine parturition onset (if placental sulfatase is missing it appears a dysfunctional labor). They are designed to promote tissue growth of the birth canal, in order to allow passage of the fetus without trauma, on the breast, estrogens cause proliferation of the ductal system and glandular tissue with progesterone and inhibition of milk secretion.

**Chorionic gonadotropic hormone** (hCG) (1,2,5,9) has progestational role in maintaining secretion of progesterone.
and oestradiol in the maternal ovarian corpus luteum until the placenta is able to synthesize progesterone. It is secreted by cytotrophoblast, and is detectable in maternal blood 7 to 12 days after conception, and in urine, about 15 days after conception. Urinary concentrations reflect circulating concentrations. HCG levels increase after implantation, reaches a peak (40,000 to 200,000 IU / L) in the weeks 8th and 10th of pregnancy and decreases at nadir. Similar in structure to LH and FSH its role is to stimulate the corpus luteum to continue the production of progesterone until the placenta takes control of progesterone secretion in the 9th gestational week. Other proposed roles are the local stimulation of placental steroidogenesis, secretion of fetal testosterone (it is active early in the fetal testis ). Inhibin (A, B) and activin (A, B) (1,2,3) are secreted by syncytiotrophoblast and cytotrophoblast cells, predominantly in the maternal circulation. Titers increase, especially in the third trimester, after the 20th gestational week for inhibin, while titers for activin at the onset of labor. Their role in maternal circulation, with estradiol and progesterone, is to suppress FSH secretion. At a placental level they are involved in modulating the synthesis of GnRH, HCG, and progesterone.

CRH (corticotropin releasing hormone) (1,2,3,7,8,9) is secreted by fetal syncytiotrophoblast, but also amnios, chorion and maternal decidua. CRH is involved in regulating cortisol synthesis, indirectly through the formation of ACTH propiomelanocortin. In labor it enhances the effects of oxytocin on myometrium, with an increased contractility. CRH levels increase in the second trimester with a maximum level between the 36th week and term. High levels are found in the twins, patients with pregnancy-induced hypertension, patients with fetal IUGR, patients with preterm labor. CRH is considered a placental “clock”.

Placenta has also the ability of synthesing hormones with an important role related to the fetal growth: HPL, HGH-V, growth factors IGF I, II. In the first part of the pregnancy placental lactogen (HPL) CS-A occurs, whereas in the second part variant GH growth hormone (hCG-V). These hormones increase the synthesis of IGF-1,2 maternal factors.

Placental Lactogen (chorionic somatotropin) (HPL / HCS-A, B) (1,7,8,9) regulates fetal growth, sparing glucose for the fetus and mobilizing fatty acids for metabolic needs of the mother. Placenta synthesizes large amounts of HPL (a molecule with similar biological and chemical site like pituitary GH) both in maternal circulation, and in the fetal circulation. More than 99% of HPL is released in the maternal circulation. Detectable in maternal circulation after 6 weeks of gestation, it reaches the maternal circulation, and in the fetal circulation. More than 99% of HPL is released in the maternal circulation. Detectable in maternal circulation after 6 weeks of gestation, it reaches the 20-30mg/ml. It stimulates the anabolism for activin at the 30th week and the values of about 1 g / day at term; its long-term effects are sparing glucose for the fetus, facilitating the use of fatty acids by the mother. HCS quantity secreted is proportional to the size of placenta (normally it is one sixth of fetal weight). It causes retention of Na, K, Ca. In the fetal circulation is secreted in small quantities 1%. At 20 weeks of gestation the concentration is 5 ng / ml and at birth it reaches 20-30mg/ml. It stimulates the anabolism for carbohydrates, lipids and proteins directly by enhancing the synthesis of IGF-1, 2 and the release of insulin. It maintains nutrient availability for a long-term for fetus by providing glucose; HPL has an important role in regulating fetal growth and metabolism of normal breast, but this action is permissive (there are cases with no genes for hGH-V and HPL and the pregnancy was successful).

Placental growth hormone (hGH-V) (7,8,9) is synthesized by placenta growth factor only in maternal circulation and is involved in the mobilization of maternal reserves of glucose, fatty acids and ketones in the second part of the pregnancy (with maximum activity in the third trimester). It temporarily replaces mother GH by suppressing pulsed growth hormone release model. It is secreted by syncytiotrophoblast, similar in structure with GH and prolactin, and it is detectable in maternal circulation around the 5th gestational week, according to some authors at week 8, it’s level increases gradually, reaching the peak at birth (about. 27.5 mU / l). hGH-V activates lipolysis, with the release of fatty acids, which will be used by the mother (there is “shift” from a mother based on carbohydrate metabolism, to the one based on lipids, sparing the amino acids and glucose for the fetus, regulating fetal growth by modulating nutrients and increasing insulin resistance. It also causes mammary epithelial cell proliferation, production and control of maternal IGF-I, II and it induces placental growth.

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