fragments. Normal ranges (mean ± S.D.) after term vaginal delivery were: $H_2O((T))$ 83.9 ± 0.2%, MBV 10.9 ± 0.2%, FBV 7.4 ± 0.9%, EW 57.3 ± 1.3% and IW 11.2 ± 0.6%. $\%H_2O((T))$ was higher after cesarean section; other measurements were not affected. There were no differences between placentas after 33–37 and after 38–42 weeks gestation. Three of eight placentas after rhesus incompatibility had $\%H_2((T))$ above the mean +2 S.D. of term placentas and five of 17 IUGR placentas were below the mean −2 S.D.. The remaining placentas following maternal pre-eclampsia, hypertension, or diabetes had no apparent alteration in $\%H_2O((T))$. A blind histological diagnosis of 'true' edema was associated with both a significantly high %IW and $\%H_2O((T))$. Perhaps this is due to alteration in placental cell volume regulation in certain situations.

Growth and maturation of villi in placentae from well-controlled diabetic women

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Placentae from controls and two groups of diabetic women (one White classes A, B, C and the other classes D, F/R) were collected at 37–42 weeks of gestation. Tissue sections were analyzed using stereological methods in order to quantify the growth and maturational status of villi. Birth and placental weights were recorded and placentae sampled in a systematic manner. Fields of view on formalin-fixed, paraffin-embedded sections were analyzed to obtain estimates of volumes, surface areas, lengths and diffusion (harmonic mean) distances. Comparisons were drawn using three-way analyses of variance with group, mode of delivery and sex of newborn as the principal effects. Mean weights were similar in controls and diabetic groups. Diabetic placentae had a more voluminous fetal capillary bed of greater length, diameter and surface area. In addition, the diffusion distances across fetal plasma (erythrocyte to endothelium) were shorter. Stromal diffusion distance and villous diameter were greater in vaginal deliveries. Interaction effects influenced also villous capillarization, capillary volume, capillary diameter, trophoblast thickness and stromal thickness. Our results emphasize the importance of adaptations on the fetal side of the diabetic placenta. They show that changes can affect the placentae of appropriate-for-age as well as large-for-age babies and provide no evidence that they increase with the severity and duration of diabetes.

Expression of adhesion molecules by endovascular trophoblast and decidual endothelial cells: Implications for vascular invasion during implantation

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During the process of implantation, maternal spiral arteries within the decidua are invaded by trophoblast cells that adhere to and migrate along the luminal surface of the vascular endothelial cells. This phenomenon resembles the events that occur during the migration of neutrophils into an acute inflammatory site, therefore it is possible that similar mechanisms are involved. Indeed, previous observations have shown that endovascular trophoblast expresses the blood group-related antigen sialyl Le(x). In this study, we show, by immunohistology, the expression of both E- and P-selectin by vascular endothelial cells only in the decidua basalis and not in decidua parietalis. In contrast, ICAM-1 is expressed by all vascular endothelium throughout the decidua. Expression of VCAM-1 is variable at the implantation site, and is not expressed by vascular endothelial cells in decidua parietalis. Interestingly, we demonstrate the strong expression of a polysialylated form of NCAM by endovascular trophoblast. Our data suggests that vascular invasion by trophoblast is regulated by the expression of appropriate adhesion molecules which permit interaction between endovascular trophoblast and decidual endothelial cells.

GTP-binding proteins associated with the human placental syncytiotrophoblast plasma membrane

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The nature of GTP-binding components associated with isolated human term placental syncytiotrophoblast microvillous plasma membrane vesicles (SPMV) was determined; these are relevant to elucidation of intracellular signal transduction mechanisms. Four proteins were identified, with molecular weights of 29, 27, 23 and 21 kDa, which specifically bound (α-^{32}P)GTP in the presence of Mg^{2+}. Studies employing anti-p21(c-*ras*) monoclonal antibodies indicated these four GTP-binding components were ras-related and one, the 21 kDa component, may be p21(c-*ras*). In addition, SPMV were also found to express the α subunits of three separate G proteins. A 45 kDa SPMV GTP-binding protein was identified as a substrate for Vibrio cholera toxin and was recognized by a rabbit antibody to the α subunit of the adenylate cyclase stimulating G protein, G(s). A 41 kDa SPMV GTP-binding protein substrate of Bordetella pertussis toxin was also recognized by rabbit antibodies to the α subunits of the adenylate cyclase inhibiting G proteins, G(i)-1 and G(i)-3. No evidence was found to support the presence of the 21 kDa G(p), a G protein previously associated with membranes prepared from whole placental tissue homogenates.

Developmental expression of Glut1 glucose transporter and c-*fos* genes in human placental cells

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Glut1, the brain/erythrocyte glucose transporter is one major isoform of the human placenta and displays an age-specific pattern of expression with mRNA levels five-fold higher in first trimester than in term placenta. By contrast, the mRNA level of the insulin-regulatable glucose transporter Glut4 remains at the limit of detection throughout pregnancy indicating a very low expression of this isoform in the placenta. The nuclear proto-

oncogenes c-*fos* and c-*myc* were also detectable in the human placenta, but c-*fos* only exhibited an age-specific pattern of expression with levels higher in third trimester than in term placenta. Primary cultures of human trophoblast cells from term placenta were used to further study the expression and regulation of Glut1 and c-*fos* genes. Fetal calf serum rapidly and transiently (15 to 60 min) stimulated c-*fos* and Glut1 gene expression suggesting that both genes share similar growth factor-controlled pathways. Glucose inhibited Glut1, but not c-*fos* expression. An eight-fold decrease in Glut1 mRNA was observed when glucose concentration in the medium was increased from 0 to 25 mM, whereas c-*fos* mRNA levels remained very low. These results suggest that in the human placenta, the expression of Glut1 is specifically regulated by glucose concentration. These data demonstrate that (1) Glut1 and c-*fos* mRNA transcripts are expressed in the human placenta exhibiting an age-specific pattern of expression, (2) In cultured trophoblast cells, both genes are stimulatable by fetal calf serum and in contrast to c-*fos*, Glut1 is negatively regulated by glucose. This differential regulation of Glut1 and c-*fos* genes could be relevant to specific metabolic and mitogenic pathways implicated in placental growth and differentiation.

Immunohistochemical localization of urokinase-type plasminogen activator and the plasminogen activator inhibitors 1 and 2 in early human implantation sites

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AM J OBSTET GYNECOL 1994 170/2 (671–676)

Objective — Our purpose was to immunolocalize urokinase-type plasminogen activator and the plasminogen activator inhibitors 1 and 2 in human implantation sites, with emphasis on the types of trophoblast expressing the plasminogen activator and the inhibitors. Study design — Urokinase and the plasminogen activator inhibitors 1 and 2 were localized immunohistochemically in early human implantation sites in unruptured ectopic pregnancies from patients in an in vitro fertilization program. Results — Urokinase kinase and the plasminogen activator inhibitors 1 and 2 were localized in the cytoplasm of cytotrophoblasts and in the cytoplasm and plasma membranes of intermediate and syncytiotrophoblast. Greater staining was noted in nonvillous, relative to villous, cytotrophoblasts for urokinase and both inhibitors. Conclusions — Urokinase-type plasminogen activator and the plasminogen activator inhibitors 1 and 2 were localized in all three forms of trophoblast at the maternal-fetal interface in early human implantation sites, particularly the differentiated and invasive forms of trophoblast. These results support a role for the plasminogen activator or inhibitors in the controlled invasion of the maternal decidua by the trophoblast during human implantation.

Transfer of cytokines through human fetal membranes

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Intact human fetal membranes (amnion, chorion and decidua) were incubated with ^{125}I-labelled cytokines added to the fetal or maternal sides of the membrane. The transfer of ^{125}I-labelled interleukin-6 (IL-6), ^{125}I-labelled tumor necrosis factor α (TNF-α), ^{125}I-labelled interleukin-1α (IL-1α) and ^{125}I-labelled interleukin-1β (IL-1β) was determined by measurement of radioactivity in a gamma counter and the integrity of the cytokines was assessed by acid precipitation and by radioimmunoassay. IL-1α and IL-1β were transferred through human fetal membranes in both feto-maternal and materno-fetal directions at similar rates. Only 2–4% of the cytokine originally added appeared to be intact on the opposing side of the membrane after 24 h of culture. Transfer of intact TNF-α (5–7%) and IL-6 (8–17%) was greater than that of the IL-1 isomers. Low but variable amounts of the four cytokines tested may be transferred through the human fetal membrane. This finding suggested that concentrations of cytokines in amniotic fluid would not reflect those produced by decidua if the fetal membranes are intact.

Transferrin receptor (CD71) expression on circulating mononuclear cells during pregnancy

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AM J OBSTET GYNECOL 1994 170/1 I (202–206)

Objective — We studied transferrin receptor (CD71) expression in peripheral blood mononuclear cells from healthy pregnant women, to determine if a relationship existed between gestational age and circulating CD71$^+$ mononuclear cells. Study design — Cell suspensions were prepared from venous blood from 139 pregnant women (7 to 26 weeks of gestation), incubated with monoclonal anti-CD71 antibody, and analyzed by flow cytometry. Results — When only the first sample from each woman was analyzed, extensive biologic variation between women was shown. An apparent biphasic increase in the percentage of CD71 cells with advancing gestation was suggested. A subgroup of 13 women studied on multiple occasions demonstrated linear increases in CD71$^+$ cells as pregnancy progressed. Conclusions — Pregnant women, when compared with each other, may have differences in the baseline number of circulating CD71$^+$ cells. The increases seen in individuals studied repeatedly are likely to reflect maternal hematopoiesis and current fetomaternal transfusion.

GENETICS

Ultrasonographic measurement of fetal nuchal skin to screen for chromosomal abnormalities

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Objective — The purpose of this prospective investigation was to determine the utility of ultrasonographic measurement of the fetal nuchal skin in screening for chromosomal abnormalities. Study design — In 1510 patients undergoing genetic amniocentesis at 14 to 21 weeks' gestation, the fetal nuchal skin fold was measured. A measurement of $\geq$ 6 mm was considered abnormal. Results — In fetuses with normal karyotype the