with normal R-Gln. At protein concentrations at which R-Gln was readily apparent, PGE, DEAE cellulose chromatography and immunodiffusion demonstrated R-Thr in apoHDL but *not* in apoHDL$_T$. However, antiHDL$_T$ reacted weakly with R-Thr suggesting the presence of traces of R-Thr in apoHDL$_T$. Using antiR-Thr sera and increasing the amount of apoHDL$_T$ 10 to 100-fold, an antigen was seen that migrated on PGE and eluted from DEAE the same as normal R-Thr and was immunochemically dentical with R-Thr.

These results indicate a markedly disproportionate decrease in R-Thr compared to R-Gln in Tangier disease and suggest the hereditary defect is one of control of R-Thr synthesis.

19 *Lp System of Plasma in Individuals at Risk of Developing Coronary Disease.* EVERETT W. LOVRIEN and LYLE J. FAGNAN, Crippled Children's Div., Univ. of Oregon Med. Sch., Portland, Oreg. (introduced by Martin Lees).

The Lp system of human plasma represents a genetic polymorphism of beta lipoprotein (BLp). KAHLICH-KOENNER showed an increased BLp in Lp^a(+) individuals compared to Lp^a(−) persons. We have produced an antiserum to Lp^a in order to investigate the possibility that Lp^a(+) children are more likely to develop coronary artery disease as adults than Lp^a(−) children.

Lp^a factor was isolated from a single normal Lp^a(+) donor, an Lp^a(−) donor and from an Lp^a(+) donor who has type II hyperbetalipoproteinemia (II BLp). The procedure for Lp^a isolation included: (1) a modification of the method of Berg using 0.65 $MKPO_4$ elution from hydroxalapatite and (2) ultracentrifugation flotation gradient at a density of 1.100 in KBR modified from Schultz. Anti-Lp^a was produced by immunizing rabbits followed by absorption with Lp^a(−) serum. Immunelectrophoresis revealed a pure antiserum. Anti-BLp was also prepared in order to quantitate BLp by radial immunodiffusion. Sera were typed for Lp^a by agarose immunodiffusion.

Investigation of 240 members of a family with II BLp revealed that segregation of Lp^a was independent of the segregation of II BLp. This indicates that the BLp associated with Lp^a(+) factor and the BLp associated with II BLp was controlled by different genetic loci. The presence of Lp^a(+) was less of a determinant of coronary disease than II BLp. The production of anti Lp^a now makes it possible to investigate other populations with various risks of coronary disease.

20 *Intrauterine Diagnosis: Comparative Enzymology of Fibroblasts Cultivated From Maternal Skin, Fetal Skin, and Amniotic Fluid Cells.* MICHAEL M. KABAEK, CLAIRE O. LEONARD and TIM H. PARMLEY, Depts. Ped. and Ob.-Gyn., Johns Hopkins Univ. Sch. Med., Baltimore (introduced by R. Rodney Howell).

We have compared the specific activities of several lysozomal enzymes in skin fibroblasts cultured from 7 normal women (MAT), their fetuses (FET), and fibroblasts developed from corresponding amniotic fluid cells (AMN). All samples were obtained between the 17th–21st week of pregnancy. Highly significant differences in the mean specific activities of β-galactosidase, β-D-N-acetylglucosaminidase, and arylsulfatase A are evident between fibroblasts developed from each of these sources. In addition, the pattern of differences in the 3 fibroblast types is dissimilar for each enzyme:

β-galactosidase:	AMN > MAT > FET
β-D-N-acetylglucosaminidase:	AMN = MAT > FET
Arylsulfatase A:	MAT > AMN > FET

Both the pattern and relative differences in enzyme specific activities are consistent in each of the 7 groups of cultures studied. If quantitative enzymologic data from cultured amniotic fluid cells is to be used for the antenatal diagnosis of specific inherited genetic disorders, it is essential that relative specific activities of the enzymes in question be established in fibroblasts developed from each of the pertinent cell types. These relationships are also critical if heterozygous fetuses are to be correctly identified *in utero* and if fetal tissue is to be used to corroborate intrauterine diagnoses based on amniotic cell analysis.

21 *Dependence of Epithelial Morphogenesis on Extracellular Acid Mucopolysaccharide (MPS).* MERTON BERNFIELD, Dept. of Ped., Stanford Med. Center, Palo Alto, Calif.

The control of morphogenesis is poorly understood, but may involve extracellular substances associated with cell surfaces (e.g. collagen, MPS). Epithelio-mesenchymal interactions provide a system for investigating the role of extracellular materials in organogenesis. Since epithelial morphogenesis is not exclusively dependent upon collagen [BERNFIELD, Dev. Biol., in press, 1970], the localization and morphogenetic significance of MPS was studied. The embryonic salivary epithelial-mesenchymal interface contains MPS as shown by (1) specific staining (Alcian blue at various Mg++ concentrations and Schiff reactivity after two-step periodate oxidation), (2) autoradiography (^{3}H-glucosamine and $^{35}SO_4$) and (3) removal of stain and radioactivity with MPSase. The MPS is localized almost exclusively at the surface of the growing and branching adenomeres. The role of MPS in epithelial morphogenesis was studied by autoradiography and organ culture after treating the rudiments with various enzymes (collagenases, proteases, sialidases and MPSases) to remove mesenchyme and MPS. Untreated rudiments undergo progressive branching and characteristic epithelial morphogenesis in organ culture. Single-step removal of mesenchyme and surface-associated MPS, followed by recombination with fresh mesenchyme, results in loss of adenomeres and a delay in epithelial morphogenesis. Removal of mesenchyme, but not surface-associated MPS, yields epithelia which maintain a characteristic contour and undergo continued morphogenesis when recombined with fresh mesenchyme. Removal of MPS from these epithelia causes a loss of contour and a delay in morphogenesis. The data indicate that epithelial surface-associated MPS (or MPS-protein complexes) are required for normal epithelial morphogenesis, and that this system may serve as a model for studies of morphogenetically active materials.

22 *A New Growth Disorder With Marked Acceleration of Skeletal Maturation and Relative Failure to Thrive.* RICHARD E. MARSHALL, DAVID W. SMITH, C. RONALD SCOTT, C. BENJAMIN GRAHAM, Dept. of Ped., Univ. of Washington Sch. of Med., Seattle, Wash.

Two infants are described with a new syndrome characterized by increased early linear growth, relativ failure to thrive, and the most marked early acceleration of osseous maturation of any condition described to date: Bone age for both patients at 6 months of age