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THE DEVELOPMENT OF CELLULAR IMMUNITY TO STREPTOCOCCUS Haya R. Rubin and Ricardo U. Sorensen, Case Western Reserve University, Rainbow Babies & Childrens Hospital, Departments of Pediatrics and Pathology, Cleveland.

The role of antigenic exposure in the development of cellular immunity to bacteria was evaluated analyzing the responses of neonatal and adult lymphocytes to two streptococcal antigen preparations. Responses to the soluble antigen SK-SD were analyzed in diluted whole blood cultures (WB) and in ficoll-hypaque separated mononuclear cells (MNC) from peripheral blood in adults (PBL) or cord blood (CBL). Responses to whole cell, killed streptococcus group A, and group B I and B III strains were analyzed with MNC only. Results of proliferative responses were assessed by ³H-thymidine incorporation, and expressed as mean counts per minute over background for triplicate cultures (net cpm).

	Strep-A	SK-SD	
Neonates	1922	0	Results in the table show that both
Adults	4099	6917	newborns and adults responded to killed strep A. (Similar results were found for strep. B I and B III.)

In contrast, only adults responded to SK-SD in WB (shown in table) or MNC. Normal newborns had no responses over background to this antigen. Yet, lymphocytes from a newborn with strep A sepsis had a response of 7152 net cpm to SK-SD. This indicates that newborns can develop a strong SK-SD response with exposure, but otherwise do not respond.

Neonatal lymphocytes do respond to all strains of killed strep. tested, without known prior exposure. This may reflect a pathway of cellular immunity to non-specific bacterial components, active before antigen-dependent responses develop.

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FETAL HEPATIC VENOUS OXYGEN SATURATION AND LIVER OXYGENATION. Abraham M. Rudolph and James Bristow. Univ. Calif., Cardiovasc. Res. Inst., San Francisco.

Previously we showed that the left lobe of the fetal liver in fetal lambs receives highly saturated O₂ umbilical venous (UV) but no poorly saturated portal venous (PV) blood. Total left lobe flow is 350 ml/min/100g, whilst the right lobe flow is 500ml/min/100g and it receives PV and UV blood. To assess whether the differing blood O₂ sats entering the two lobes affect oxygenation, we developed methods for chronic cannulation of the hepatic venous veins (HV) in fetal lambs. The left and right HV join the inferior vena cava (IVC) anteriorly just below the diaphragm; they were cannulated through pursestring sutures in the thoracic IVC through right thoracotomy. In 24 fetal lambs (116-122 d) we placed catheters in L or R HV, PV, IVC, and descending aorta (AO). The O₂ sat in the LHV was higher than that in RHV; it was close to UV O₂ sat, but RHV O₂ sat was similar to that in AO.

O₂ sats (percent) were:

	AO	UV	HV	IVC
Left HV (n5)	55.2±6.4	79.6±9.9	67.4±8.0	32.2±6.6
Right HV (n13)	57.9±10.8	80.8±8.1	60.0±10.5	36.0±8.3

Blood flows to the R and L lobes of the liver from hepatic artery, UV, and PV were measured with radioactive microspheres and, together with O₂ content in various vessels, we showed that oxygen consumption in ml/100g/min were similar in left (3.4) and right (3.9) lobes of the liver. The different O₂ sats in L and R HV could be important in determining oxygen supply to various fetal organs, depending on distribution of L and R HV blood. (Supported by NIH grants HL 06285 and HL 24056.)

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ADIPOSE TISSUE COMPOSITION - RELATIONSHIP TO DIET. David Schiff, George Chan, University of Alberta, Edmonton.

Human breast milk (EBM), Similac (SIM), SMA, and Intralipid (IL) vary in their fatty acid composition. This study reports on the effect of dietary fat on the composition of adipose tissue in infants during the first 3 months of life. 18 infants ranging in age from 1 day to 3 months and requiring surgery were studied. Subcutaneous adipose tissue was obtained from the incision site and analyzed for fatty acids. Fatty acid composition of the diet and adipose tissue are as follows:

		Formula Fatty Acid Composition % Total					
		C12:0	C14:0	C16:0	C18:0	C18:1	C18:2
	EBM	7.7	12.7	25.0	9.6	33.0	5.0
	SIM	11.3	11.0	12.5	4.5	21.3	38.4
	SMA	7.4	6.1	14.4	8.1	43.9	18.2
	IL			11.0	4.5	26.0	49.6
		Adipose Tissue Fatty Acids % Total					
		C14:0	C16:0	C16:1	C18:0	C18:1	C18:2
NO.	Diet	Duration					
6	NPO	1 d	3.2	45.9	12.8	3.5	29.7
2	EBM	12 d	4.1	31.0	9.6	5.7	42.0
3	SIM	16 d	5.1	30.8	10.7	3.1	32.4
1	SMA	3 M	3.2	14.7	2.7	4.7	60.1
2	IL	20 d	3.0	39.0	10.0	4.1	32.9
4	IL	27 d	4.4	22.0	5.0	3.8	33.6
	SIM	2 M					

This study shows that diet can alter an infant's adipose tissue composition in a short time.

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THE EFFECTS OF EXOGENOUS GLUCAGON ON FETAL METABOLISM IN THE SHEEP. Richard L. Schreiner, James A. Lemons, Helen Moorehead, Rick Bohnke, Debra Reyman. (Spon. by Jerry Bergstein), Indiana University School of Medicine, Indiana University Hospitals, Department of Pediatrics, Indianapolis.

The effect of exogenous glucagon on fetal and maternal glucose, fructose, insulin and glucagon concentrations was studied in the chronic fetal sheep model. Eight near-term fetuses were infused with glucagon (40 ng/min) for 60 min via a fetal femoral vein catheter. Fetal plasma glucagon increased from 37.66±7.86 (SEM) to 420.50±38.59 pg/ml (p<.001) within 15 min of beginning the infusion, reaching 531.80±117.54 pg/ml (p<.001) by 60 min and returning to the baseline level 60 min after stopping the infusion. Fetal arterial glucose increased from 12.06±0.84 to 15.50±0.68 mg % (p<.0001) after 15 min of infusion (p<.001), and reached a maximum level of 17.18±0.94 mg % by 60 min (p<.001). Fetal arterial plasma insulin was significantly increased within 60 min of infusion (16.45±3.73 to 29.38±4.41 uU/ml, p<.001). Fetal fructose did not change. During the 60 min infusion and postinfusion period, there were no changes in the maternal concentrations.

These results, in addition to our previous studies demonstrating an increase in fetal glucagon concentration with prolonged maternal fasting, provide further evidence that glucagon is an important regulator of fetal metabolism.

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DEVELOPMENT OF BETA-ADRENERGIC RECEPTORS IN FETAL RAT LUNG, HEART, AND BRAIN TISSUE. Donald L. Shapiro, U. of Roch School of Med., Strong Mem. Hosp., Dept. of Peds. Roch. NY

Beta-adrenergic cell membrane receptors were studied in membrane fractions prepared from developing rat lung, heart, and brain tissues. Receptors were identified using radiolabeled (³H) dihydroalprenolol (DHA), a beta-adrenergic antagonist with high affinity for receptor sites. Developmental profiles from 19 days gestation to 24 hours after birth (TABLE) showed no significant increase in the amounts of beta-adrenergic receptors in brain tissue. In heart tissue, an increase occurred between 19 and 20 days gestation. In lung tissue, there was a marked increase in receptors between 20 and 21 days gestation. This coincides with maturation of the pulmonary surfactant system, and it is speculated that the increase in lung receptors is associated with type II pneumocytes which are known to have beta-adrenergic receptors.

pmoles DHA bound/mg tissue (± S.E.)

DAYS GESTATION	19	20	21	BIRTH + 24 hours
BRAIN	538 ± 197	483 ± 219	481 ± 93	461 ± 33
HEART	1038 ± 62	1843 ± 65	1494 ± 70	1845 ± 323
LUNG	1896 ± 182	2084 ± 29	4578 ± 440	3254 ± 727

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EFFECTS OF ACUTE SALT LOADING ON BLOOD PRESSURE REGULATION IN THE NEWBORN LAMB. Sharon R. Siegel, UCLA Div. of Nephrology, Los Angeles, California.

The normal newborn has an impaired conservation of sodium; high renin-angiotensin system (RAS) levels; and a decreased pressor responsiveness to angiotensin II (A-II). The purpose of this study was to determine whether increasing the newborn salt intake would suppress the high RAS levels and alter blood pressure (BP) responsiveness. In Expt 1, 8 newborn lambs were infused with NaCl 10 mEq/kg in 20 ml/kg 5% D/W over 60 min, followed by serial doses of A-II (0.025, 0.5, 0.1, and 0.25 ug/kg/min), each for 15 min. Mean aortic BP was monitored continuously. In Expt 2 (0.25 ug/kg/min) was infused into 5 newborn lambs after a similar NaCl loading. Plasma renin activity (PRA) was measured before and at 5, 15, and 30 min of the A-II infusion. In Expt 1, A-II (0.025 ug/kg/min) increased BP 30 mm Hg in the ewe, caused no change in the control newborn, and increased BP 15 mm Hg in the volume expanded newborn; (0.25 ug/kg/min) increased BP 30 mm Hg in the control newborn and 40 mm Hg in the volume expanded newborn. In Expt 2, PRA decreased from 22.1 ± 2.1 ng/ml/hr (M and SEM) to 10.8 ± 2.1 (p<.05) after the volume expansion alone, and to 5.2 ± 1.0 (p<.05) after 30 min of the A-II infusion. BP decreased 10 mm Hg after the salt loading alone. In conclusion: Acute salt loading, 1) increases the pressor responsiveness to A-II; 2) suppresses the renin-angiotensin system; and 3) decreases basal BP in the newborn lamb. Therefore, the renin-angiotensin system control of BP and A-II responsiveness may be related to the newborn sodium state.