1840 CORRELATION OF MORPHOLOGIC AND CLINICAL RADIOLOGIC ASSESSMENT OF LUNG DISEASE IN NEONATES. Jon R. Wispé and Robert J. Roberts. University of Iowa College of Medicine, University of Iowa Hospitals and Clinics, Departments of Pediatrics and Pharmacology, Iowa City, IA.

We compared the morphologic appearance of the lung with the clinical and radiologic diagnosis in 6 infants with RDS (BW 0.7-1.6 kg), 6 with non-RDS lung disease > 35 wks gestation (BW 2.4-4.9 kg), and 2 infants born without respiratory disease (BW 2.9-3.5 kg). Morphologic assessment involved scanning electron microscopy (SEM) of whole lung and of methyl methacrylate corrosion casts of the pulmonary vascular bed.

In pre-term infants, a good correlation was observed between the clinical and radiologic diagnosis of RDS, the clinical course, and the morphologic appearance of the lung. Those infants with the most severe RDS had the most dramatic reduction in number of alveoli. In contrast, in infants with non-RDS lung disease, interpretation of the disease process by clinical and radiologic means lacked consistent correlation with morphologic features, particularly of the vascular bed. Abnormalities in lung parenchyma (septal wall thickness and alveolar development) appeared to vary independently from vascular abnormalities (extent of microvascular development and organization). The insight provided by SEM of the lung and its associated vascular bed explains why similar therapeutic manipulations, whether respiratory or pharmacologic, fail to result in similar clinical responses in all infants with a common clinical or radiological diagnosis. (Supported in part by NIH GM12675 and a PMA Fellowship.)

1841 PHOSPHATIDYLGLYCEROL (PG) STIMULATES SYNTHESIS OF PHOSPHATIDYLCHOLINE (PC) IN CULTURED TYPE II PNEUMOCYTES. Alasdair M. Gilfillan, Arthur J. Chu and Seamus A. Rooney. Yale Univ., Dept. of Pediatrics, New Haven, CT.

We examined the effect of exogenous phospholipid (PL) on PL synthesis in type II cells. Cells isolated from adult rats were cultured for 18-20 h in medium containing fetal bovine serum and antibiotics. After washing, the cells were further incubated for 1-6 h in serum free medium containing PL - dispersed by sonication - and radiolabeled precursor. PL increased the rate of choline incorporation into PC. PG and cardiolipin stimulated the most but phosphatidic acid, phosphatidylethanolamine (PE), phosphatidylinositol and phosphatidylserine - but not PC or sphingomyelin - were also stimulatory. The effect of PG was concentration dependent in the range 10^{-9} to 10^{-4}M and was apparent at all times examined. Incubation with 10^{-5}M PG for 2 h increased the rate of [^{3}H]choline incorporation into PC by 72% from 5200±700 (mean ± SE, n=7) cpm/10^6 cells (no PG) to 8600±900 (P<0.001, paired t test). Similarly, PG increased the rate of [^{3}H]glycerol incorporation into PC by 50% from 720±100 cpm/10^6 cells to 1080±140 (P<0.002, n=6). The effect was specifically on PC; rates of glycerol incorporation into other PL or of [^{14}C]ethanolamine into PE were not increased. It is unlikely that the effect of PG is due to increased substrate as it was not mimicked by glycerol, glycerophosphate or palmitate. Neither does it appear to be due to enhanced cell growth: rates of [^{14}C]leucine and [^{3}H]thymidine incorporation into protein and DNA, respectively, were not increased. The relevance, if any, of these findings to surfactant turnover and reutilization remains to be elucidated. (Supported by HD-10192.)

1842 MONITORING STUDIES IN OLDER PRETERM INFANTS WITH PERSISTENT APNEA. Carol Lynn Rosen, Daniel G. Glaze, James D. Frost, Jr. (Houston)

Fifteen preterm infants (PC ages 36-42 weeks), with persistent apnea, bradycardia, and/or cyanosis, but otherwise normal, were monitored within a 2 week period using 3 methods: 1) pneumogram (PG), 12-hour recording of chest impedance changes and EKG; 2) oxycardiorespirogram (OCR), 4-hour recording of tcO_2, impedance pneumography and EKG; 3) polygraphic sleep studies (PS), 6-12 hour recording of 11 parameters including EEG, tcO_2, respiratory movements, $ETCO_2$, and EKG. Abnormalities were identified in all 15 infants: 13 by PS, 10 by PG, 9 by OCR. The following abnormalities were recorded: prolonged apnea (PA), excessive periodic breathing (PB), bradycardia (B), disorganized breathing (DB), increased mixed and obstructive pauses (M/O), and hypoxemia during feeding (HF):

	PA	PB	B	DB	M/O	HF
PG (# infants)	0	0	10	4	--	--
OCR (# infants)	3	--	4	1	--	4
PS (# infants)	6	1	5	--	8	9

During PS elevated CO_2 occurred in 10 infants. All were discharged on home monitors: 9 had subsequent apnea or bradycardia alarms that self-corrected; 3 had events requiring stimulation but not CPR. Only one infant with prolonged apnea by PS had an alarm requiring intervention. No single finding predicted risk for subsequent serious episodes. Our data suggest that monitoring studies are helpful in characterizing cardiorespiratory abnormalities in these infants and that PS may document abnormalities not recognized during PG or OCR.

1843 PERSISTENT APNEA IN OLDER PRETERM INFANTS: POLYGRAPHIC STUDIES AND HOME MONITOR FOLLOW-UP Carol Lynn Rosen, M.D., Daniel G. Glaze, M.D., James D. Frost, Jr., M.D. (Houston)

Fifty-four preterm patients (postconceptional age 36 to 44 weeks), otherwise well and ready for discharge, were identified with persistent symptoms of apnea, bradycardia, and/or cyanosis. At the time of referral, symptoms were primarily sleep-related in 22 (41%), feeding-related in 16 (30%), and both sleep and feed-related in 16 (30%). Polygraphic recordings documented cardiorespiratory abnormalities including: prolonged apnea in 12 (22%), excessive periodic breathing in 4 (7%), bradycardia in 21 (52%), disorganized breathing or increased number of mixed and obstructive respiratory pause in 21 (39%), and elevated CO_2 values in 24 (44%). All infants were discharged on home cardiorespiratory monitors. Nineteen (35%) were discharged on xanthines. Twenty-eight (52%) infants had subsequent apnea or bradycardia alarms that self-corrected. Seven (13%) had subsequent serious alarms at home requiring stimulation, but none required CPR. These alarm conditions occurred between 39 and 54 weeks postconceptional age. Only one of the infants with documented prolonged apnea had subsequent alarms requiring intervention. Our data suggest that some preterm infants with persistent symptoms of apnea, bradycardia, and cyanosis are at risk for subsequent serious episodes, and that home monitor observation is a safe alternative to prolonged hospitalization in appropriate families.

1844 α_1 ANTITRYPSIN (AAT) ACTIVITY IN DEVELOPMENT OF BRONCHOPULMONARY DYSPLASIA (BPD). W. Rosenfeld, L. Concepcion, H. Evans, R. Jhaveri, V. Brunot. Interfaith Medical Center and Downstate Medical Center, Brooklyn, NY.

Some of the toxic side effects of oxygen therapy are indirectly attributable to the release of proteases including elastases from PMNs. These may contribute to the pathogenesis of BPD. AAT inhibits elastases and if deficient may lead to an imbalance favoring proteolytic digestion of pulmonary connective tissue. We studied the possible role of AAT activity by measuring the trypsin inhibitory capacity (TIC) in prematures with IRDS who received oxygen.

Twenty prematures with IRDS ($\bar{m}$ wt 1052 gms; $\bar{m}$ GA 29 wks; M/F = 12/8) had serial TICs drawn on days 1,3,7 and weeks 2-10. They were analyzed for severity of IRDS (mild: IMV$\leq$3 days, severe: IMV$\geq$4 days) and for the development of BPD using standard clinical and xray signs. Higher TICs were observed in 9 patients with mild IRDS (0.91 mg/ml) than in 10 with severe IRDS (0.36 mg/ml) ($p<0.02$) on day 1 but were comparable thereafter. In the 11 patients who did not develop BPD, TICs (1.05 mg/ml) were higher than in the 9 who did (0.31 mg/ml) ($p<0.001$) on day 1, week 4 (0.62 vs 0.33 mg/ml) ($p<0.03$), and week 8 (0.78 vs 0.47 mg/ml) ($p<0.03$).

Decreased AAT activity was associated with the increased severity of IRDS on day 1 and with the occurrence of BPD on day 1 and weeks 4&8. However AAT activity was also directly related to gestational age ($r = .6589$; $p<0.05$). Hence AAT activity may play a pathogenetic role in the development of BPD, or may be a non specific marker, or both.

1845 BETAMETHASONE (Bt) STIMULATES WHILE DIHYDROTESTOSTERONE (DHT) DOES NOT INFLUENCE THE DEVELOPMENT OF FETAL (F) PULMONARY EPIDERMAL GROWTH FACTOR (EGF) RECEPTORS (R) F.H.Sadiq, V.Chechani, S.Harris and U.Devaskar (Spons. W.J.Keenan) Dept. of Peds., St. Louis Univ. St. Louis, MO

Cellular mechanisms of glucocorticoids induced acceleration and DHT mediated inhibition of F pulmonary surfactant production remain unclear. Plasma membrane R mediated EGF has an important role in F lung maturation. The influence of Bt or DHT on the development of F lung plasma membrane(LPM) EGF R was investigated. Bt (.085mg/kg or saline (Control I) was administered on 25d and 26d of gestation to the rabbit doe. Daily DHT (10mg in ETOH+25mg in demethyl sulfoxide) or the vehicle (Control II) was begun on 15 day. (plasma androgen* 1.29±.11 vs .54ng/ml). LPM were characterized by protein, DNA, % recovery and 5'-nucleotidase (NTD) activity on d 27. Number of EGF R sites were estimated by ^{125}I-EGF binding assays. Scatchard plots were linear. All data X±SEM, *p<.05 vs sex group control (C-I).

	N	WT (gm)	LPM NTD L. Homo.	% Sp. Bind .1mg LPM Prot.	EGF R #X10^{-10} mg LPM Prot.	Kdx10^9
Bt ♂	5	*22±1	3.5±.5	*2.98±.5	*22±3	5.1±1.1
Bt ♀	5		4.5±.5	*2.23±.4	*21±3	5.6±1.0
CI ♂	6	28±2	3.4±.5	.98±.1	6±1	3.7±.5
CI ♀	6		4.0±.6	.99±.2	7±2	3.9±.6
DHT	4	33±2	3.4±.5	1.28±.2	8±3	4.0±.7
CII	3	27±3	4.1±.8	1.31±.1	6±2	4.1±1.0

Conclusions: 1)Maternal Bt retards F body wt, 2)F pulmonary EGF R did not differ by sex at 27 day, 3)Bt stimulates F pulmonary EGF R in ♂ and ♀, 4)DHT does not influence F pulmonary EGF R.