878 RECATEGORIZING CHILDHOOD ACUTE LYMPHOBLASTIC LEUKEMIA WITH MONOCLONAL ANTIBODIES TO HUMAN T CELLS. P.M. Sondel, W. Borcherding, N.T. Shahidi, J.C. Schultz, D. J. Ganick, R. Hong. Depts. of Pediatrics, and Human Oncology, University of Wisconsin, Madison, Wisconsin.

The immunologic surface markers of lymphoblasts from three patients with childhood acute lymphoblastic leukemia (ALL) were analyzed. Blasts from two of these patients did not form rosettes with sheep erythrocytes and the third did so marginally, suggesting these patients had non-T cell leukemia. These blasts were also tested with commercially available monoclonal antibodies (OKT 3, 4, 6, 8,) that detect thymocyte differentiation markers, and all three patients were highly reactive with at least 2 of these reagents.

We anticipate the availability of multiple standardized monoclonal reagents will necessitate a recategorization of ALL phenotypes. Furthermore, some of these leukemic phenotypes may not correspond to normal stages of lymphoid differentiation. Therefore, it may be inappropriate to attempt to identify and categorize leukemic cells by the pathways of normal differentiation. We suggest that leukemic cells be identified by the presence or absence of standardized immunologic and enzymatic markers without attempting to classify them by prior labels of "T, null, B, pre-B or pre T."

879 ABNORMAL CONCANAVALIN A CAPPING - ANOTHER DEFECT OF NEUTROPHILS FROM HUMAN INFANTS. Ronald G. Strauss and Michael J. Hart, University of Iowa College of Medicine, Department of Pediatrics, Iowa City, Iowa.

Concanavalin A (con A) binds to neutrophil (N) plasma membranes and induces granule exocytosis, chemotaxis and increased oxidative metabolism. Con A binds diffusely to the N surface, but caps form on N exposed to colchicine or to oxidants (a process dependent on interactions of plasma membrane, cytoskeleton and several biochemical systems). To test the competency of these interactions, we studied fluorescein con A capping of N isolated from venous blood of newborns, their mothers and adult controls. Diffuse con A binding occurred readily, but capping of infant and maternal N was decreased ($p < .05$). Colchicine induced capping in 38 ± 3, 51 ± 3 and 69 ± 2 (mean % ± SEM) of N from 20 infants, mothers and controls. The oxidant, diamide, induced caps in 45 ± 8, 59 ± 11 and 82 ± 6% respectively. The decreased response was striking because spontaneous capping (without induction) was higher in infants (23% ± 3) and mothers (38% ± 4) than in controls (14% ± 3). Thus, the mean increment in capped N induced by colchicine was 15, 13 and 55% respectively. Spontaneous capping seemed related to oxidation since values were reduced ≅ 70% ($p < .05$) when N were exposed to superoxide dismutase and catalase. Thus, spontaneous and induced con A capping of infant and maternal N are abnormal, perhaps due to autooxidation. Many cellular processes are involved, and study of this abnormality may identify mechanisms responsible for the other dysfunctions of infant N.

● **880** AUTOOXIDATION AND DECREASED VIABILITY OF NEUTROPHILS OBTAINED FROM HUMAN INFANTS. Ronald G. Strauss and Esther L. Snyder, University of Iowa College of Medicine, Department of Pediatrics, Iowa City, Iowa.

Infant neutrophils (N) exhibit decreased viability in vitro. We investigated autooxidation as a cause since increased oxidative metabolism and decreased oxidant protective enzymes (glutathione peroxidase and catalase) coexist in infant N. N were isolated from blood of newborns, their mothers and controls and were stored in buffer at 37°C as either resting (nonphagocytic) or phagocytic N. Viability was determined by cell count and dye exclusion after 0, 20, 42 and 66 hours. Viability of both resting and phagocytic infant N was less ($p < .01$) than was viability of maternal or control N at all times (e.g., mean % ± SEM of resting N viable after 20 hours in 19 studies = 49 ± 3, 75 ± 3 and 78 ± 2, respectively). Viability of maternal and control N was decreased by phagocytosis to that of resting infant N; possibly an oxidant effect as it was prevented when phagocytic N were exposed to superoxide dismutase and catalase. Resting infant N released more ($p < .02$) H_2O_2 than did resting maternal or control N (0.25, 0.16 and 0.14 nM/2.5 x 10^6/minute, respectively) to suggest oxidant damage even in resting infant N. Further, resting infant N lost viability more rapidly than did control N when exposed to the H_2O_2 generator, glucose-glucose oxidase, indicating increased susceptibility to oxidation. Thus, the decreased viability of infant N in vitro seems related to autooxidation. If this occurs in vivo, it is one mechanism for the dysfunctions of infant N that might be approached pharmacologically.

● **881** ABNORMALITIES IN ARACHIDONIC ACID (A.A.) METABOLISM IN NEONATAL PLATELETS AND THEIR RELEVANCE TO NEONATAL PATHOPHYSIOLOGY. Marie J. Stuart, Judith B. Allen, SUNY, Upstate Medical Center, Dept. Peds., Syracuse, N.Y.

Since A.A. is the precursor for vasoconstrictive prostaglandins that are elevated in a variety of neonatal pathophysiologic states (eg. RDS), we assessed platelet A.A. release and subsequent metabolism through the cyclooxygenase and lipoxygenase pathways in platelets (plts.) from 8 term neonates and compared them to 13 adult controls. Evaluation revealed a significant increase in the chemotactic lipoxygenase product HETE in the neonate (41.5 ± 2; 1SE) when compared to the adult (31.2 ± 2.1). The cyclooxygenase product TXB_2 was decreased in neonatal plts. (11.1 ± 1.7 vs 19 ± 1.7; $p<0.01$. Uptake of ^{14}C A.A. into plts. of both neonates and adults was similar. Neonatal plts. however released a greater amount ($p<0.001$) of AA (24.7 ± 2.8%) compared to adults (14.6 ± 0.8). Enhanced release of AA from the plt. of the neonate suggests that differences exist between neonatal and adult phospholipases A_2, C, and diglyceride lipase (enzymes involved in the release of AA from cell membranes). Our findings provide proof for the presence of an "aspirin-like" defect in the plt. of the newborn i.e. inhibition of the plt. enzyme cyclooxygenase, and also demonstrates an increase in the release of AA from the neonatal cell membrane. This finding may play an important role in a variety of pathologic states in the neonate mediated via cellular prostaglandins.

882 DECREASED PROSTACYCLIN OR PGI_2 PRODUCTION IN THE INFANT OF THE DIABETIC MOTHER (IDM). CORRELATION WITH MATERNAL HbA_{1c}. M.J. Stuart, S.G. Sunderji, J.B. Allen SUNY, Upst. Med. Ctr., Depts of Peds and Perinatology, Syracuse.

Platelet (plt) and vascular prostaglandins are important regulators of normal hemostasis. Plts produce endoperoxides and thromboxanes which are proaggregatory and prothrombotic, whereas vessels produce PGI_2 which is antiaggregatory and antithrombotic. Since the IDM has an increased predisposition to thrombosis, we evaluated arachidonic acid (AA) metabolism and PGI_2 production in the umbilical vessels of 12 control infants and 9 IDM of similar gestational age. Mean uptake of ^{14}CAA into vascular tissue of controls and IDM were similar at 9157 ± 1096 (1SE) and 12036 ± 1691 cpm per 30 mgm vascular tissue resp. Thrombin stimulated release of ^{14}CAA was similar in the controls (5.0 ± 0.7) when compared to IDM (5.5 ± 1.3). However in the IDM, the production of vascular 6-Keto-$PGF_{1\alpha}$ (the stable end product of PGI_2) was decreased ($p<0.02$). Controls incorporated 5003 ± 533 cpm (5.2 ± 0.4%) into 6-Keto-$PGF_{1\alpha}$ when compared to 3033 ± 490 cpm (3.2 ± 0.6%) in the IDM. A significant inverse correlation ($r=0.87$; $p<0.02$) was observed between maternal HbA_{1c} levels and the conversion of AA to 6-Keto-$PGF_{1\alpha}$ in the IDM. Rigorous control of maternal diabetes appears to be the best prophylaxis against the decrease in PGI_2 in the IDM. We have previously shown that plt. endoperoxides are increased in the IDM. Thus, the normal balance between proaggregatory (plt) and antiaggregatory (vascular) prostaglandins is disrupted in the IDM, a factor which favors thrombosis.

883 DECREASED CHEMOTACTIC ACTIVITY IN NEONATAL PLASMA: ROLE OF CHEMOTACTIC FACTOR INACTIVATOR AND COMPLEMENT LEVELS. Raymond Tannous and Roger Spitzer . Sponsored by Ronald G. Strauss. University of Iowa, Department of Pediatrics, Iowa City; and Upstate Medical Center, Department of Pediatrics, Syracuse.

Compared to adult plasma, the chemotactic activity of neonatal zymosan activated plasma (ZAP) is decreased. Consequently, we evaluated the role of chemotactic factor inactivator (CFI) and complement levels in this defect. Plasma of 15 neonates and 15 adults were studied. The chemotactic activity of ZAP was measured under agarose, using adult neutrophils as target cells. CFI levels were determined by incubating 0.1 µl plasma with 50 µl C5-derived chemotactic fractions and then measuring the percent decrease in glucosaminidase release from cytochalasin B-treated neutrophils. C3, C5, factor B, properdin and properdin convertase were determined immunochemically; and C1, C2, C4, C3-9 and CH50 by hemolytic assays in 10 of these infants and adults.

Compared to adult plasma, the chemotactic activity induced by infant plasma was decreased (80 ± 7% of control [mean ± SD], vs. 95 ± 3%, $p < 0.001$), CFI levels were increased (19 ± 6 vs. 7.5 ± 3, $p < 0.001$), and complement component levels were decreased ($p < 0.05$), except for C2. The chemotactic activity correlated inversely with CFI levels ($r = -0.958$, $p < 0.001$). After adjusting for the effects of CFI, there was no significant correlation between the chemotactic activity and any of the complement components tested. Thus, differences in CFI, rather than in complement levels, accounted for the differences in chemotactic activity between infant and adult plasma.