▲ 103

Vitamin E Nutritional Status in the Neonate - Evaluation of α-Tocopherol Blood Cells and Buccal Mucosal Cells

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The aim of this study was an evaluation of vitamin E nutritional status in neonates by determination of α-tocopherol in plasma (PL), red blood cells (RBC), platelets (PLT), monocytes (MN), polymorphnuclear leucoytes (PMN) and buccal mucosal cells (BMC). Neonates and children up to 16 years of age were evaluated.

Patients, Methods: 65 children and 81 neonates were enrolled in this study. α-tocopherol was measured by HPLC-technique with electrochemical detector.

Results: (a:mean±SD, n=number of patients)

	Neonates	Children
PL μg/dl	402±155[a] (81)[n]	769±99 (65)
RBC, μg/dl packed cells	203±48 (29)	241±47 (55)
PLT, μg/mg protein	0,03±0,02 (29)	0,12±0,05 (52)
MN, $\mu g/10^9$ cells	4,06±1,02 (40)	8,47±2,66 (51)
PMN, $\mu g/10^9$ cells	2,59±1,33 (24)	4,35±1,23 (25)
BMC, ng/mg protein	25,4±7,6 (25)	47,8±15,8 (30)

Conclusion: During the neonatal period, the lowest levels were documented in all blood fractions excluding RBC, α-tocopherol in PL, PLT, MN, PMN and BMC in newborns amounted to only about one-half or one-third of older child values, implicating a relative vitamin E deficiency in the newborn.

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▲ 104

NEONATAL SCREENING FOR GLUCOSE-6-PHOSPHATE DEHYDROGENASE (G-6-PD) DEFICIENCY: Sex Distribution and Role of Paternity

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G-6-PD deficiency is a common condition associated with neonatal jaundice and even kernicterus. As it is an X-linked condition, many population surveys and studies of neonatal jaundice have been confined to males. Classically, both parents should be affected in order for their female offspring to be similarly affected; however, under certain circumstances the mother only need be enzyme deficient. We screened male and female infants of high risk mothers in our Sephardic Jewish population to determine the prevalence of the condition in both sexes, and determined the marriage patterns of the parents of the female enzyme deficient infants. A qualitative color reduction screening method was used (Sigma) capable of differentiating between those normal for G-6-PD or grossly deficient in the enzyme. A total of 806 infants was screened between 1989 and 1992: 127 of 420 males (30.2%) and 40 of 386 females (9.9%) had G-6-PD deficiency. The predicted female incidence (9.1%) according to the Hardy Weinberg equation was not significantly different from the actual incidence (P=.58). The results were further analysed according to parental marriage patterns:

PARENTAL GROUPS	% OF POPULATION	% OF DEFICIENT FEMALES
A.Identical high risk	35.4	53.6
B.Non-identical high risk	23.3	32.1
C.High risk mother, low risk father	41.3	14.3

As expected, G-6-PD deficient females were more common in situations where both parents were of high risk ethnic background, but surprisingly, were also detected (14%) when the father was from a low risk ethnic group, including Ashkenazi Jews in whom the incidence of G-6-PD deficiency is close to zero. We conclude that severe G-6-PD deficiency is common in females of high risk families, and that it can occur not infrequently in females even if the father is not of a high risk family background. Female infants in high risk populations should be included in screening programs for G-6-PD deficiency for the prediction and evaluation of neonatal hyperbilirubinemia, regardless of paternal status.

▲ 105

GLUCOCORTICOID SENSITIVITY OF HUMAN NEONATAL LEUKOCYTES

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Glucocorticoids (GC) are widely used as immunosuppressive and anti-inflammatory drugs. They are known to inhibit the proliferative response of leukocytes after activation with mitogen, possibly by interfering with the interleukin-2 (IL-2) mRNA synthesis. Apart from IL-2 mRNA, GC also inhibit the transcription of genes encoding for IL-1 and IL-6. Until now, the effects of GC have predominantly been studied in adults. Therefore we studied the in vitro effects of the GC dexamethasone on the proliferative capacity of T cells in 15 human newborns. Our data show that neonatal leukocytes are more sensitive for inhibition of the proliferative response by dexamethasone than adult cells (EC 50 neonatal cells: 10^{-9} M; adults 3.10^{-8}M $p<0.01$). This difference in sensitivity is not related to differences in GC receptor expression in neonatal and adult cells. Dexamethasone inhibits the expression of the IL-2 receptor on neonatal cells to a larger extent than on adult cells (70% versus 25% inhibition). However, this difference can not explain the difference in sensitivity between adult and neonatal cells. We show that the increased sensitivity of neonatal cells for GC is due to their diminished capacity to produce IL-2. Addition of interleukin-2 can restore the proliferative capacity after dexamethasone inhibition of neonatal cell proliferation.

▲ 106

INFLUENCE OF PARITY ON MOTHER-TO-CHILD TRANSMISSION OF HIV

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Background: Risk factors for mother-to-child transmission of HIV are still incompletely defined. An effect of parity has hitherto not been described.
Design: In a national prospective study, 316 children of HIV-positive mothers were followed up for at least 6 months. Infection status was determined in 254 of them according to the criteria of the Working Group on Mother-to-Child Transmission of HIV (AIDS 1993, 7:1139-1148) or, in children under 15 months, by concordant (positive or negative) results of virus detection tests in at least 2 samples by at least 2 methods (PCR, culture, antigen after immune complex disruption). Transmission rate was 18.1%. The effect of potential risk factors on transmission rate was analysed.
Results: Univariate analysis showed an association between primiparity and increased transmission rate: odds ratio (OR) 2.2, 95%-confidence interval (95%-CI) 1.1-4.6, $p<0.05$. Logistic regression confirmed this association (adjusted OR 2.4) and showed, in addition, a negative association between transmission rate and elective cesarean section (OR 0.36, 95%-CI 0.13-0.97, $p<0.05$). The effect of primiparity was less pronounced in combination with elective cesarean section (OR 1.7) than with other delivery modes (OR 2.5, difference not significant). HIV-infected children older than two years were less likely to get a younger sibling during the observation period than their uninfected counterparts (0 of 22 vs. 10 of 101, $p<0.05$ by logrank test).
Conclusions: Primiparous women appear to transmit HIV to their children at a higher rate. This could be explained by increased intrapartum transmission because of longer and more complicated labour in primiparas and/or by a self-selection of women with lower risk of transmission among those deciding to have additional children.

● 107

MOTOR TESTS OF INTERHEMISPHERIC CONTROL AND COGNITIVE FUNCTION IN VERY PRETERM INFANTS AT EIGHT YEARS. Vincent Kirkbride, Jenny Baudin, Ann Lorek, Judith Meek, Juliet Penrice, Jan Townsend, Simon Roth, David Edwards, David McCormick, Osmund Reynolds, Ann Stewart, Dept of Paediatrics, University College London Medical School, London,UK.

We have previously reported that damage to the posterior corpus callosum might explain some of the subtle cognitive processing deficits found later in many preterm infants. To test this hypothesis we carried out motor tests of interhemispheric control in 197 consecutively examined very preterm infants(<33 weeks gestation) at eight years. In addition to the Kaufman Assessment Battery for Children (K-ABC) and other studies, two tests of mirrorwise(Tm) and alternate(Ta) forearm diadochokinetic (pronation-supination) movements were performed. The alternate movement represents the most complicated form of interhemispheric control and reciprocal inhibition. The time difference between the two movements (Ta-Tm) is considered to give an indication of the efficiency of transfer of motor information between the hemispheres (particularly the posterior corpus callosum). There was a significant and linear relation between this time difference and the Kaufman Mental Processing Composite (K-MPC) score ($p<0.002$, DF 2). We conclude that the corpus callosum has an important role in cognitive development and posterior corpus callosum efficiency is related to cognitive outcome.

▲ 108

SURFACTANT DOWNREGULATES HUMAN MONOCYTE AND NEUTROPHIL RESPONSES TO HYPEROXIA. Haresh M. Kirpalani, Peter Conlon, Manel Jordana. Dept. of Pediatrics McMaster Univ., Canada.

Monocytes (M) and neutrophils (N) are implicated in hyperoxic lung injury. We investigated effects of surfactant (ST) on N and M in hyperoxia (HO2). METHODS: N and M from healthy adults were plated at $1x10^6$-$4x10^6$/ml. Cells were incubated with (+ST) or without (-ST) surfactant (100μg/ml;Exosurf) and exposed to either HO2 (95% O_2+5% CO_2) or air, for 2 to 18 hours. Supernatants was examined for TNF-α and IL-8 (ELISA). Cells were examined for PMA stimulated Oxygen Radical (OR) production by luminol dependent chemiluminescence. RESULTS: i)By 2 hours HO2 increased OR by N; ST reduced OR, both in air and HO2 (3 subjects). Peak OR (mV) as (Mean(SD).

Air - ST 4.17 (2.21) vs HO2 - ST 6.67 (3.43)
Air + ST 1.10 (0.66) vs HO2 + ST 3.13 (1.28)($p<0.05$)

ii) At 18hrs: TNF-α was expressed as % of air control.

M:Air - ST HO2 - ST HO2 + ST (6 subjects)
100% 156.2(86.1) 77.9(40.3) $p<0.05$
N:Air - ST HO2 - SF HO2 + ST (6 subjects)
100% 122.3(71.8) 27.5(32.5) $p<0.05$

In N and M, TNF-α release was markedly diminished by ST.

iii) At 18 hrs: IL-8 was expressed as % of air control.

N:Air - ST HO - ST HO + ST (6 subjects)
100% 92.9(46.3) 68.5(48.4) $p>0.05$.

In N IL-8 release was not signficantly affected by ST.

CONCLUSIONS:ST significantly downregulates TNF-α and OR release by N and M. This may be of potential importance.