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THYROID FUNCTION IN CHILDREN LIVING IN THE VICINITY OF ZINC SMELTING WORKS-MIASTECZKO ŚLĄSKIE

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Heavy metals influence on thyroid,particularly that of Pb can be one of many goiter causing factors. The aim of the study was the assesment in 239 children /4-15 years/ living in the proximity of Smelting Works,Miasteczko Śląskie, T_3,T_4,TSH and Pb,Zn,Mg,Ca of blood concentrations and determination of goiter frequency. 52,4% of those children showed an elevated blood Pb concentration and decreased concentrations were found: Zn 20% and Mg 29,4%. Pubescent children had more frequent occurence of thyroid goiter. In addition to that,goiter was found in youngest children. T_4 concentrations were within the normal values,and showed positive correlation with Pb. Only in 2 cases with Pb concentration exceeding 40µg/dl low T_4 concentrations were found. No relation was noted,between Pb concentrations and those of T_3 and TSH. In the youngest group a negative correlation was discovered between Ca and T_3 concentrations,and between those of TSH and Mg.The increased goiter frequency in children inhabiting the polluted area can't be explained by, Pb intoxication only.This problem is more complex than that and is a result of a multifactor influence of a polluted environment,among the others,heavy metals interaction with other elements.

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HEAVY METALS AIRBORNE POLLUTION INFLUENCE ON SELECTED CONGENITAL MALFORMATIONS IN THE KRAKÓW DISTRICT AREA

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Since 1986 airborne dustfall has been recorded over the Kraków District area (KDA) by a 53 test sites located on the grid basis. Dust samples were analyzed quarterly by an Atomic Absorption Spectrophotometry for **cadmium, nickel, copper, lead** and **chromium** concentration in a fallout. Congenital malformations (1300 cases) were ascertained by multiple source registration during the period of 4 years (1986 - 1989) over KDA. Neural tube defects - NTD (68 cases), facial clefts - FC (66 cases), heart defects - HD (279 cases) and limb malformations - LM (429 cases) were assigned to the measure grid areas according to the place of inhabitance. The number of inhabitants in grid areas were calculated using a planimetric method on the basis of the City Council population density records. The number of congenital malformations correlated well with the number of inhabitants in analyzed subareas ($R = 0.64$ to $R = 0.78$ $p < 0.01$, checked for each class of malformation). For further calculations a standarized malformation distribution was used. Simple regression tests were performed for the malformation distribution (dependent variable) and the listed metals means distribution in the air fallout (independent variable).
Results and discussion: Most of regression tests revealed no relationship between the variables. The correlation coeficient values were insignificant for Pb, Cu and Cr ($-0.2 < R < 0.2$). A weak negative correlation between LM and NI ($R = -0.32$ $p = 0.1$) was found. There was correlation between NTD and Cd ($R = 0.47$ $p = 0.07$). The results reject the hypothesis about the influence of airborne heavy metal pollution on selected congenital malformations incidence. A positive correlation between the cadmium fallout and the incidence of NTD needs further elucidation.

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PLASMA CERULOPLASMIN (CP) LEVELS IN NEWBORN INFANTS WITH SEVERE JAUNDICE (J) OF UNKNOWN ETIOLOGY AND WITH J DUE TO GLUCOSE-6-PHOSPHATE-DEHYDROGENASE (G6PD) DEFICIENCY.

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The role of liver immaturity in the pathogenesis of severe J in G6PD deficient newborns is debated.Plasma levels of CP,a protein synthesized by the liver,could provide an assessment of hepatic immaturity.CP was measured by nephelometry in cord-blood in 4 Groups of newborns either without J (maximum serum bilirubin <8 mg/dl) or with J (max.bilirubin >15 mg/dl):Group 1),20 full-term without J; Group 2),20 full-term with J of unknown etiology;Group 3),12 G6PD deficient without J;Group 4),12 G6PD deficient with J. Mean CP levels in Groups 2) and 4) were significantly lower than in 1) and 3) ($p<0.05$).CP was also measured on day 2-3 in 3 Groups of infants: Group 5),26 full-term without J;Group 6),36 full-term with J;Group 7),31 G6PD deficient with J.Mean CP levels in Groups 6) and 7) were significantly lower than in 5),($p<0.05$). These results,and our previous observations of an elevated α-Fetoprotein level in cord-blood of newborns with J (*),suggest that liver immaturity plays an important role in the genesis of J in both normal and G6PD deficient newborns.They also suggest the possibility of identifying at birth those infants who are at risk of severe J requiring treatment. (*) Ped.Res.22:225,1987 and 24:268,1988.

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CEREBRAL FLUID NEOPTERIN LEVELS IN CHILDREN WITH MENINGEAL RELAPSE OF MALIGNANT HEMOPATHY.

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Cerebrospinal fluid (CSF) neopterin concentrations were measured by HPLC in 44 normal children and 15 children affected by meningeal relapse of malignant hemopathy (13 acute lymphoblastic leukemia and 2 high grade lymphoma). When meningeal relapse was diagnosed, all the patients had CSF neopterin levels higher than the mean normal value + 2 SD, but no correlation was observed between CSF neopterin and blast count. In 10 children, CSF data before relapse were available : CSF neopterin at time of initial diagnosis of the hemopathy was 29.4 ± 12 nmol/l (median = 32.8) and 104.2 ± 56 (median = 85.6) when meningeal relapse ($p < 0.01$, Wilcoxon test). In 3 patients, elevation of CSF neopterin preceded (15 to 30 days) the occurrence of neurological signs or presence of blast cells in CSF. In absence of infectious context, a rise of CSF neopterin is an indicator of cerebral active phase of the malignant hemopathy. Such increase can reflect a cell mediated immunologic process induced by leukemic cells. The measure of CSF neopterin appears helpful in the monitoring of patients with malignant hemopathy to detect early meningeal relapse.

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ELECTRON PARAMAGNETIC RESONANCE(EPR)MONITORING OF ASCORBATE FREE RADICAL(A•)AS MARKER OF INCREASED IRON-MEDIATED OXIDATIVE DAMAGE(IMOD)IN PLASMA OF NEONATES.

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In common neonatal disorders (hemolytic disease, asphyxia, hypoperfusion) as well as in red cell transfusion the oxidative risk can increase, as free iron is released from damaged tissues and promotes conversion of hydroperoxides into the highly reactive hydroxyl/alkoxyl radicals. In this reaction ascorbate is concurrently oxidized to A•. To investigate the capability of neonatal plasma to counteract IMOD, samples of pre-term (11 subjects, GA 28-35 wks) and term infants (11 normals, GA 38-42 wks) were challenged with scalar amounts of Fe^{++}, as well as plasma of 7 adult controls. The reaction was evaluated by EPR monitoring of the A• produced.

RESULTS: 1) althought A• production was slighly lower than in adults, the Fe^{++} µM which triggered the reaction were significantly smaller in pre-term and term infants (21.1 ± 6.0 and 31.1 ± 10.3 vs. 43.7 ± 4.6; $P<0.001$ and <0.05). This difference was not referable to a different saturation of transferrin since the total and latent iron binding capacity of plasma was similar both in neonatal and adult samples. 2) in contrast with adults, in pre-term and term infants ($P<0.001$) the EPR segnal persisted for a longer time and after 15 min. A• values were still 69.2 ± 8.9 and 63.5 ± 23.3 per cent of initial peak. CONCLUSIONS: 1) Neonates have a reduced tolerance to IMOD; 2) In this condition ascorbate behaves as a pro-oxidant, instead of antioxidant compound and its administration can be dangerous; 3) Prevention of IMOD should be directed to the search for effective iron chelators.

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MODIFICATION OF TUMOUR GROWTH BY HERPES SIMPLEX VIRUS AND OTHER VIRUSES

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The tumourigenicity of neoplastic hamster and mouse cell lines and tumour explants was reduced by infection with herpes simplex virus (HSV-1), a thymidine kinaseless mutant of herpes simplex virus (MDK), encephalomyocarditis virus (EMC) and bovine mammillitis virus (BMV). There was an approximate relationship between duration of virus infection in vitro and reduction in the rate of tumour development. The rate of tumour development was also reduced by 'site inoculation' of virus (HSV-1) at various time intervals following inoculation of tumourigenic BHK-21 cells indicating that virus was capable of reducing the rate of tumour development in a situation where the neoplastic cells were already transplanted into the susceptible host species. Finally, inoculation of herpesviruses and encephalomyocarditis virus into established subcutaneous tumours in hamsters and mice reduced the rate of tumour growth.

It is suggested that the therapeutic role of wild type, mutant or recombinant viruses merits further exploration towards prevention and treatment of human cancer.