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Liver enzyme induction caused by antipyrine. Malaka-Zafiriou, K., Kolioukas, D., Kontopoulos, E. and Cassimos, Chr. Pediatric Clinic, University of Thessaloniki, "Aghia Sophia" Hospital, Thessaloniki, Greece. Among the substances which have an inducing effect on the enzymatic activity of the liver, antipyrine has been lately used therapeutically, in pregnant women in order to prevent neonatal hyperbilirubinemia. In the present paper we determined liver microsomal enzyme activity caused by antipyrine, by studying various hepatic enzyme systems responsible for bilirubin as well as drug metabolism. Twelve (12) volunteers participated in the study, aged 29-36 years. Three hundred (300) mg of antipyrine per day were administered p.o., to each one of them for a period of 10 days. Urinary D-glucuronic acid (D-GA) excretion, serum gamma-glutamyltranspeptidase ( $\gamma$ -GT), urinary salicylamide glucuronide formation rate and saliva half-life of antipyrine. No statistically significant difference was observed in serum  $\gamma$ -GT and saliva half-life of antipyrine. On the contrary, a very significant statistical difference ( $P < 0,001$ ) was found in urinary D-GA excretion and salicylamide glucuronide formation. The above results show that the administration of antipyrine caused an increase of the hepatic enzyme activity, as far as the enzymes involved in the metabolism of glucuronic acid, as well as those involved in the formation of salicylamide glucuronide are concerned. It is well known these enzyme systems take part in the metabolism of bilirubin.

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LENTZE\*M.J. and J.SCHNEIDER\* (Intr.by H.T.VERSMOLD)  
Universitätskinderklinik, D-8 München, Federal Republic of Germany.

Lysosomal Enzymes in Human Fetal Gastrointestinal Tract. Distribution and development of lysosomal enzymes (LE) in human fetuses have previously been studied only in small intestinal mucosa. In this study we examined for the first time the distribution and development of  $\alpha$ - and  $\beta$ -glucosidase,  $\alpha$ - and  $\beta$  galactosidase, N-acetyl- $\beta$ -glucosaminidase, acid phosphatase and alk.phosphatase in gastric, duodenal, jejunal, ileal and colonic mucosa and lam.muscularis of 23 fetuses 12-36 weeks old. Enzyme activities were measured using a fluorometric method with methylumbiliferone coupled compounds as substrates. Activity was expressed as hydrolyzed substrate in nmoles/mg protein/min. At the 12<sup>th</sup> week of gestation in all parts of the G.I. tract LE activity could be detected. Activities were low in oesophageal, gastric and colonic mucosa; in jejunal and ileal mucosa they were tenfold increased ( $p < 0.001$ ); those found in lam.muscularis were similarly distributed. During gestation LE develop with lower activities in gastric, duodenal and colonic as they do in jejunal and ileal mucosa: we found a fivefold increasing activity from the 12<sup>th</sup> to 36<sup>th</sup> week for  $\alpha$ - and  $\beta$ -glucosidase, alkphosphatase and a sixfold decreasing activity in  $\beta$ -galactosidase and acid phosphatase. We conclude: 1) The topographical distribution of LE shows characteristic pattern with peak activities in small intestinal mucosa and lam.muscularis. 2) Lysosomal enzymes in the G.I. tract are already measurable at the 12<sup>th</sup> week of gestation. 3) During gestation LE activities in gastric, duodenal and colonic mucosa develop similarly to those found in the small intestinal mucosa of human fetuses.

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DEL PRINCIPE D., MENICHELLI A., MANCUSO G., PERSIANI M., D'ARCANGELO C. Dept. of Pediatrics and CNR Center for Respiratory Viruses. Univ. of Rome, Italy

Cyanide insensitive  $O_2$  consumption in zymosan stimulated platelets. We investigated the  $O_2$  consumption of human platelets, stimulated by zymosan, an inducer of release reaction (Zucker and Grant, 1974). The platelet suspensions (in PBS, pH 7.4) were prepared from fresh blood samples, by separating the leukocytes from PRP (contamination  $< 1$  cell/ $10^8$  platelets) through a layer of Ficoll (23% w/v). Zymosan was incubated with autologous citrated plasma for 30 min and then washed three times in saline. The  $O_2$  consumption was measured polarographically by a Clark electrode. The KCN (1mM) treated platelets, after addition of NADH (1mM) showed a  $O_2$  uptake of  $1 \pm 0.3$  nmol/min/ $10^9$  platelets even in the presence of ASA (100mM) and TYA (225  $\mu$ g/ml) as the mean  $\pm$  SD of 15 experiments. There was no increased  $O_2$  consumption in non opsonized zymosan stimulated control platelets. The reaction failed to occur by zymosan incubated with hydrazine treated plasma. Our results suggest that an activation of a NADH CN-insensitive oxidase by an immunological stimulus, occurs in platelets like that polymorphonuclear leukocytes.

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Duffy blood group system/Fy/phosphoglucutase/PGM/ and gutamate-pyruvate transaminase red blood cell enzyme/GPT/type examinations in homo- and heterozygous

cases of mucopolysaccharidosis/CF/. Aranka Laszlo<sup>x</sup>, L. Szabo<sup>x</sup>, and K. Gyurkovits/intr. by D. Boda/. Dept. of Pediat. Univ. Med. School, Szeged, and Dept. of Forensic Medicine, Semmelweis Univ. Med. School, Budapest. The phenotype and genotype distributions and the gene frequencies of the Fy blood group system and the PGM and GPT red blood cell-enzyme systems were examined in homo- $n=18$ /, and heterozygotes  $n=38$ / with CF, and the results were compared with the data for the average Hungarian population. The Fy gene frequencies in Hungary are normally  $Fy^a=0,451$ , and  $Fy^b=0,549$ . In the CF homozygote and heterozygote groups, the Fy phenotype distribution and gene frequency did not differ appreciably from the values for the average population. In the CF homozygote group  $Fy^a=0,464$ , and  $Fy^b=0,535$ ; in CF heterozygotes:  $Fy^a=0,381$ , and  $Fy^b=0,618$ . As regards the Fy blood group/since this is the neighbouring gene locus of the amylase/, we investigated whether the CF homo- or heterozygosity varies synchronously with the Fy homo- or heterozygosity. A close correlation was not found and thus the hypothesis of a common gene regulation was rejected. The distribution of the PGM<sub>1</sub> phenotypes in the Hungarian population was as follows: PGM<sub>1</sub> 1=58,18%, PGM<sub>1</sub> 2=36,96%, PGM<sub>1</sub> 2=4,86%. The gene frequencies were: PGM<sub>1</sub> 1=0,766, PGM<sub>1</sub> 2=0,234. PGM and GPT phenotype in CF groups conformed with the overall population.