Human monitoring for genetic effects // Ed. By A.Cebulska-Wasilewska. IOS Press. NATO Science Series. Series I. Life and Behavioural Sciences – Vol. 351. 2003. P.151

CHEMICAL CLASTOGENICITY AND ANTICLASTOGENICITY IN MOUSE MICRONUCLEUS TEST

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Chemical mutagens ethyl methanesulfonate (EMS) and dimethyl terephthalate (DMtP) were compared in mouse micronucleus (MN) test. Simultaneously, anticlastogenic activity of two derivatives of 1,4-dihydropyridine (DHP and GP) was studied. *CBAxC57Bl/6$_j$* males and pregnant females were exposed to chemicals by i.p. Frequencies of micronucleated polychromatic erythrocytes (MN PCEs) in bone marrow of adults and in fetal liver were analyzed 6, 12, 18, 24, 30, 36, 48 or 24, 48 and 72 h after injections.

EMS (300 mg/kg) induced the highest MN PCE frequency in bone marrow 36 h after treatment of adults. The clastogenic effects were the same in both males and females. In fetuses, peak of MN PCEs was observed 24 h after female exposure to EMS, i.e. earlier than in a maternal organism.

On contrary, DMtP at the dose of 1/40 LD_{50} induced 4–7,3‰ of MN PCEs in males and was inefficient in pregnant females slightly increasing MN level in fetal cells. Thus, chemicals studied induced different effects depending on physiological status of animals but the both penetrated the placental barrier.

In males, the anticlastogenic effect of DHP (1/10 LD_{50} or 340 mg/kg) was observed at a peak of MN production reaching 30%. GP inhibited EMS clastogenicity too. In pregnant females, the anticlastogenic effect of DHP was prolonged and higher than in males reaching 70 % at the MN peak. In foetuses, this antimutagen was not efficient. Both antimutagens did not change the polychromatic/normochromatic erythrocyte (PCE/NCE) ratio as compared with EMS action. DHP (1/50 and 1/10 LD_{50}) affect

MN PCE frequency induced DMtP neither in females nor foetuses.

Thus, antimutagens studied inhibited EMS clastogenicity in males and females. Anticlastogenic activity was higher in females and was not revealed in foetal cells. Antimutagens of 1,4-dihydropyridine series were inefficient against DMtP in transplacental test. Data obtained allow the supposition that AM effects are mediated through protective systems of the organism.