carcinogen which caused lung, liver and cervical cancers. It is also reported as an endocrine disruptor that affects on the reproductive system and sperm counts. DBCP causes DNA damage and genotoxicity in animal cells. However, at the present time, the exact mechanism of the action of DBCP with respect to mutagenicity and carcinogenicity is unknown. For this reasons, we studied mutation spectrum induced by DBCP using transgenic Big Blue mutation assay which can investigate both the mutagenic potential and the mechanism of action of suspected mutagen and carcinogen. Big Blue Rat2 cells were treated with 0.748, 0.387 and 0.207 mM of DBCP, resulted in 50, 70 and 80% survival, respectively. The mutant frequency (MF) of *lacI* in control was 4.22×10^{-5}, whereas MF value in DBCP-treated groups showed statistically significant and dose-dependent increase. The DBCP-induced and spontaneous mutation spectrum will be represented at this report in detail.

[PA4-22] [04/21/2000 (Fri) 10:30 - 11:30 / [1st Fl, Bldg 3]]

THE ROLE OF HEPATIC METABOLISM IN THE ACUTE TOXICITY OF PARATHION

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Effects of phenobarbital and/or ketoconazole pretreatment on the acute toxicity of parathion were examined in male Sprague-Dawley rats. Phenobarbital pretreatment (50mg/kg/day, ip) for 4 consecutive days resulted in a significant decrease in inhibition of acetylcholinesterase activity in rats challenged by parathion (2mg/kg, ip). In contrast, ketoconazole (50mg/kg, ip) pretreatment potentiated the toxicity of parathion in brain. The protective effect of phenobarbital was markedly reduced by additional ketoconazole treatment. Phenobarbital treatment at the dosage regimen used in this study increased cytochrome P450 contents, protein contents and hepatic microsomal enzyme activities; p-nitrophenol hydroxylase, p-nitroanisole hydroxylase, aminopyrine N-demethylase and erythromycin N-demethylase. Hepatic aliesterase activity responsible for the detoxication of parathion was increased by phenobarbital. Ketoconazole treatment decreased aminopyrine N-demethylase and erythromycin N-demethylase. Induction of erythromycin N-demethylase by phenobarbital was blocked by ketoconazole treatment. Either phenobarbital or ketoconazole itself did not affect the acetylcholinesterase activity or the total hepatic glutathione contents. The results indicate that hepatic metabolism plays a significant role in the toxicity of parathion, although the target organs of organophosphorus compounds are primarily extrahepatic tissues such as brain and lung.

[PA4-23] [04/21/2000 (Fri) 10:30 - 11:30 / [1st Fl, Bldg 3]]

Monitoring Studies on Endocrine Disruptors(Cd, Pb, Hg) in Humans

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ABSTRACT

Human and vital systems were exposed to a variety of endocrine disruptors and accumulated by them which acted as many kinds of hormones. So they affected the number of germ and undermined male reproductive systems, immune systems and endocrine systems. This research was