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Anti-apoptotic mechanism of mouse testicular Leydig cell TM3 after Benzo[a]pyrene exposure

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Benzo[a]pyrene (BaP) has been known to be associated with mutagenesis, carcinogenesis and apoptosis of many mammalian cells. In the present study, we investigated the potential anti-apoptotic mechanism of testicular Leydig cell TM3 after BaP treatment (0.01-100 uM for 72 hours). BaP treatment did not significantly evoke the increase of apoptosis in TM3 cells compared to vehicle-treated control. BaP treatment in TM3 cells increased both Ah receptor and Arnt protein contents. Both transcriptionally and translationally, the CYP1A1 levels were elevated significantly by the BaP treatment, but the mesurement of CYP1A1 activity by ethoxyresorufin-o-deethylase assay revealed that BaP (10uM) is not able to elevate CYP1A1 activity in TM3 cells. Procaspase-3 protein content obviously increased after BaP, but its activation was not detectable. Importantly, X-chormosome linked inhibitor of apoptosis protien (XIAP) content increased and maintained during the treatment. Finally, the BaP treatment to TM3 cells did not evoke the decrease of mitochondrial membrane potential as well as release of cytochrome c. In conclusion, the anti-apoptotic mechanism in TM3 cells after BaP might be strongly related to the increased status of XIAP level as well as the maintenance of mitochondrial integrity.

Keyword : Benzo[a]pyrene, Leydig cell, apoptosis