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Benzo[a]pyrene induces apoptosis in endometrial RL95-2 cells

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Benzo[a]pyrene (BaP), one of the polycyclic aromatic hydrocarbons, can disturb cellular signal transduction pathways and also induce apoptosis in some cells. In the present study, BaP (10 uM) has been treated to RL95-2 endometrial carcinoma cell line. BaP treatment resulted in inhibition of cell proliferation. BaP increased both AhR and Arnt protein contents, and the translocation of these proteins to nucleus were evident. BaP treatment also significantly elevated the CYP1A1 protein expression. Measurement of mitochondrial membrane potential (MMP) by JC-1 dye revealed that BaP significantly decreased MMP. In addition, immunocytochemical study showed that both cytochrome c and AIF proteins were released from mitochondria after treatment, which indicates mitochondrial apoptotic pathway might be involved in BaP-induced RL95-2 cell apoptosis. Western blot analysis showed that while caspase-9 and caspase-3 were activated at 48 hrs after BaP, caspase-8 activation was not detected, again indicating that mitochondria-mediated intrinsic apoptotic pathway might be dominantly operating in RL95-2 cells than extrinsic apoptotic pathway. Bap treatment decreased X-linked inhibitor of apoptosis protein. Finally, caspase-3 activity was confirmed by PARP cleavage. Taken together, we demonstrated that BaP induced-RL95-2 cell apoptosis is occurred by mitochondria-mediated intrinsic pathway.

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