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POLYCHLORINATED BIPHENYLS INDUCE ARYL HYDROCARBON RECEPTOR-INDEPENDENT APOPTOSIS OF MOUSE SPLEEN CELLS

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Polychlorinated biphenyls (PCBs) are ubiquitous environmental contaminants, and many of their toxic effects, including their immunotoxicities, are mediated by the activation of aryl hydrocarbon receptor (AhR). We previously reported that Aroclor 1254, one of the most widely used PCB mixtures, increased DNA fragmentation in mouse spleen cells, suggesting that apoptosis was correlated with the immunotoxicity of PCB (Yoo et al., 1997). In the present study we investigated the mechanism by which PCB induces apoptosis, and the involvement of AhR in the PCB-mediated apoptosis of mouse spleen cells. Aroclor 1254 induced DNA fragmentation without AhR activation, and the apoptosis was unaffected by α-NF, a well-known antagonist of AhR. Moreover, the PCB congeners (PCB 47, 52, 128, and 153), which have little affinity for AhR, induced DNA fragmentation, whereas congeners (PCB 77, 126, and 169), which have high affinity for AhR, did not induce fragmentation. The di-ortho form of PCB (PCB 153) and Aroclor 1254 induced DNA fragmentation in the spleen cells of both AhR knockout mice (AhR-/-) and Ah low-response mice (DBA/2), whereas the non-ortho form of PCB (PCB 126) did not induce DNA fragmentation. In the light of these findings, it is evident that AhR is not involved in PCB-mediated apoptosis.

keyword : polychlorinated biphenyl, aryl hydrocarbon receptor, apoptosis