KOSMOS Vol. 67, 2, 361–373, 2018

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PEROXISOME PROLIFERATOR-ACTIVATED RECEPTORS IN CARCINOGENESIS – FACTS AND CONTROVERSIES

Summary

Peroxisome proliferator-activated receptors (PPARs) belong to the nuclear receptor family. So far, three isoforms of PPARs: alpha, beta and gamma have been described. As ligand-dependent transcription factors, they participate in the regulation of diverse physiological processes. PPARs are involved in the regulation of lipid and glucose metabolism. They also control inflammatory processes or cell proliferation and differentiation. PPARs are also implicated in the regulation of reproductive functions. Furthermore, results of several studies clearly indicate, that PPARs are involved in carcinogenesis. PPARα mediates in hepatocellular tumor growth in rodents, but its role in human hepatocytes is not so obvious as in rodents. The role of PPARβ/δ in carcinogenesis still remains unclear. It is believed, that PPARβ/δ has important function in colorectal tumor growth. In turn, the expression of PPARγ has been demonstrated in different types of tumor cells and its role in carcinogenesis seems the most complex. There are reports that indicate antiproliferative and proapoptotic effects of PPARγ activation. It has been also demonstrated that PPARγ ligands inhibit angiogenesis and induce terminal differentiation. In this review, we summarize current findings regarding the involvement of the three PPAR isoforms in carcinogenesis.

Key words: cancer, carcinogenesis, nuclear receptors, PPAR ligands, transcription factors