

## *Abstract*

Many currently produced chemicals reveal specific properties which allow them to be referred to as endocrine disruptors (ED). These substances exhibit an exogenic hormone activity and usually act as antagonists or agonists of endogenous hormones. The exogenic EDs studied in this work were 17 $\alpha$ -ethinylestradiol (EE2) and benzo[a]pyrene (BaP). 17 $\beta$ -estradiol (E2), a typical endogenous hormone, was also included to the study.

In the presented work, the effect of these EDs and their combinations on the expression and specific activities of cytochromes P450 (CYP) 1A1 and 2C was determined. First, the microsomal fraction (MF) of liver, kidney and lung of rats premedicated with these compounds or without premedication was isolated. CYP expression was assessed by the Western blot analyses in these MF samples. Moreover, CYP1A1 and CYP2C specific activities were evaluated. It was found that premedication of rats with BaP increased CYP1A1 expression in all above mentioned organs. Whereas BaP strongly induced rat CYP1A1, EE2 and E2 were almost without this effect. But, when these disruptors were administered to rats with BaP, they supported its potency to induce CYP1A1. Further, CYP2C11 expression and its specific activity were gently increased by premedication of rat with EE2 and its combination with BaP. Premedication of rat with BaP also significantly increased the expression of CYP2C11 in kidney. On the contrary, premedication of rat with BaP and its combination with EE2 decreased the CYP2C6 specific activity in liver. E2, EE2 and its combination lead to increasing of this specific activity.

Different expression of the CYP1A and 2C affected by the studied EDs and their combinations may modulate their metabolic pathways and genotoxic activity in organism, as well as their environment effects.

(In Czech)

**Keywords:** 17 $\alpha$ -ethinylestradiol, benzo[a]pyren, 17 $\beta$ -estradiol, endocrine disruptors, cytochrome P450, microsomal fraction, expression, specific activity