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five papers accepted for presentation at
the 59th Meeting of the Serbian Chemical Society.

BH-2

# Ekspresija JNK kinaza u THP-1 celijama tretiranim Ru(II) kompleksima

Milena P. Krstić[1], Juan Francisco Santibanez[2], Jelena Poljarević[3], Sanja R. Grgurić-Šipka[3], Sunčica Z. Borozan[1]

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C-Jun N-terminalne kinaze (JNK) spadaju u mitogen-aktivirane protein kinaze (MAPK) i imaju važnu ulogu u kontroli niza ćelijskih procesa uključujući proliferaciju, kancerogenezu i apoptozu. Kompleksi rutenijuma pokazali su izuzetan potencijal kao mogući citostatici. Upravo iz ovih razloga ispitivan je mehanizam delovanja kompleksa Ru(II) sa *N*-alkilfenotiazinima (hlorpromazinom, trifluoperazinom i tioridazinom) na signalne parametre (t-JNK, p-JNK i β-aktin) u THP-1 ćelijama humane leukemije. U ćelijama tretiranim kompleksom sa fluoperazinom u koncentraciji od 10 μM ($IC^{50}$ je 10,5 μM) ekspresija t-JNK povećana je za 47,45%, dok je ekspresija p-JNK dvostruko veća u poređenju sa netretiranim ćelijama, što sugeriše da su t-JNK i p-JNK uključeni u apoptozu ovih ćelija.

# Expression of JNK kinases in THP-1 cells treated with Ru(II) complexes

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The c-Jun N-terminal kinase (JNK) belongs to the family of mitogen-activated protein kinases (MAPKs) that play an important role in the control of a number of celular processes, including proliferation, cancerogenesis and apoptosis. Ruthenium complexes have shown remarkable potential as possible cytostatics. Precisely for these reasons, the mechanism of action of Ru(II) complexes with *N*-alkylphenothiazines (chlorpromazine, trifluoperazine, and thioridazine) on signalling parameters (t-JNK, p-JNK and β-actin) in THP-1 human leucemic cells was investigated. In cells treated with fluoperazine complex at a concentration of 10 μM ($IC^{50}$ is 10.5 μM), an increased expression of t-JNK by 47.45% was found while the expression of p-JNK was twice higher compared to untreated cells, suggesting that t-JNK and p-JNK are involved in the apoptosis of these cells.

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