Apoptotic mechanisms of octreotide on HepG2

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A research team from Greece examined the effect of octreotide on cellular proliferation, apoptosis and caspase activation in HepG2 hepatocellular carcinoma (HCC) cells. The results supported the induction of caspase-mediated signaling pathways of octreotide antitumor activity in HepG2 cells, and indicated that measurements of serum octreotide levels may be important, at least in clinical trials, to verify optimal therapeutic drug concentrations.

Hepatocellular carcinoma (HCC) is the fifth most common malignancy in the world and is estimated to cause approximately half a million deaths annually. Undoubtedly, the best available treatment for all liver tumors is complete surgical resection. However, the synthetic somatostatin analogue octreotide has been found effective in inhibiting tumor growth in a variety of experimental models. It has been reported that octreotide inhibits the proliferation and induces apoptosis of different HCC cell lines in vitro. The mechanisms of apoptosis induction however are not well understood.

A research article published on January 21, 2011 in the World Journal of Gastroenterology addresses this question. The authors confirmed that octreotide inhibited HepG2 proliferation at a concentration of 10-8 mol/L. Interestingly, lower concentrations of octreotide increased proliferation and this is possibly an additional reason for divergent results in both clinical trials and in vitro studies of octreotide in HCC. Also, their results support the induction of a caspase-mediated apoptotic pathway by octreotide in HepG2 cells, implicating both a receptor-

mediated and mitochondrial-apoptotic pathway.

The findings of the present study indicate that measurements of serum octreotide levels may be important, at least in clinical trials, to verify optimal therapeutic drug concentrations.

More information: Tsagarakis NJ, Drygiannakis I, Batistakis AG, Kolios G, Kouroumalis EA. Octreotide induces caspase activation and apoptosis in human hepatoma HepG2 cells. World J Gastroenterol 2011;17(3): 313-321. www.wjgnet.com/1007-9327/full/v17/i3/313.htm

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