

Galangin and its emerging anti-neoplastic effects

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To the Editor: I read with great interest the recent article by Zhang et al. (2012a). Interestingly recent data suggest that galangin may influence carcinogenesis in a number of systemic tumors besides melanomas.

For instance, galangin activates the caspase 8/t-Bid mitochondrial pathway in hepatocellular carcinomas (Zhang et al. 2012b). Simultaneously there is an increase in Bid disintegration resulting in increased tBid. Simultaneously, galangin administration results in increased release of cytochrome-*c* into the cytoplasm by virtue of enhanced translocation of Bax to mitochondria in the neoplastic cells (Zhang et al. 2010). As a result, galangin results in accentuated apoptosis in these tumors. This enhanced galangin mediated apoptosis is inhibited by up regulation of Bcl-2.

Similarly, galangin modulates glutathione S-transferase P function. As a consequence, it enhances apoptosis in gastric malignancies. Simultaneously, it also affects ubiquitin carboxy-terminal hydrolase isozyme function thus further accentuating apoptosis in gastric neoplastic cells (Kim et al. 2012). Modulation of the above enzymes results in accentuated

expression of JNK as well as ERK 1/2. Simultaneously, there is an increase in the cleavage of poly (ADP-ribose) polymerase.

Similar, when administered to imatinib sensitive leukemias, galangin increases the sensitivities of these hematological malignancies. It also helps overcome imatinib resistance in imatinib resistant Bcr-Abl expressing leukemias (Tolomeo et al. 2008). A decline in Bcl-2 level is seen following galangin administration. Similarly, a decline in cdk1 and cdk4 levels accompanies galangin administration. As a result of this enhanced sensitivity there is an increase in G0/G1 phase cells. Similarly, tumor growth is attenuated in animal models with Ehrlich ascites carcinoma following the administration of galangin (Jaiswal et al. 2012). The above examples clearly illustrate the significant role of galangin in the need for oncology and further studies in this regard.

References

- Jaiswal JV, Wadegaonkar PA, Hajare SW (2012) The bioflavonoid galangin suppresses the growth of ehrlich ascites carcinoma in Swiss Albino mice: a molecular insight. *Appl Biochem Biotechnol* 167:1325–1339
- Kim DA, Jeon YK, Nam MJ (2012) Galangin induces apoptosis in gastric cancer cells via regulation of ubiquitin carboxy-terminal hydrolase isozyme L1 and glutathione S-transferase P. *Food Chem Toxicol* 50:684–688
- Tolomeo M, Grimaudo S, Di Cristina A, Piptone RM, Dusonchot L, Meli M, Crosta L, Gebbia N, Invidiata FP, Titone L,

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- Simoni D (2008) Galangin increases the cytotoxic activity of imatinib mesylate in imatinib-sensitive and imatinib-resistant Bcr-Abl expressing leukemia cells. *Cancer Lett* 265:289–297
- Zhang HT, Luo H, Wu J, Lan LB, Fan DH, Zhu KD, Chen XY, Wen M, Liu HM (2010) Galangin induces apoptosis of hepatocellular carcinoma cells via the mitochondrial pathway. *World J Gastroenterol* 16:3377–3384
- Zhang W, Lan Y, Huang Q, Hua Z (2012a) Galangin induces B16F10 melanoma cell apoptosis via mitochondrial pathway and sustained activation of p38 MAPK. *Cytotechnology*. Sep 22 [Epub ahead of print]
- Zhang HT, Wu J, Wen M, Su LJ, Luo H (2012b) Galangin induces apoptosis in hepatocellular carcinoma cells through the caspase 8/t-Bid mitochondrial pathway. *J Asian Nat Prod Res* 14:626–633