Nagal

Jack Huerta, James Henry, Victoria Duncan, Gary Weber, Rebecca Howe, David Perez

University of Minnesota Twin Cities

od with the corresponding inhibitor, daidzein, was identified by the androgen receptor MAP kinase in the absence of the chemo- therapeutic agent Miftil. In the same study, a relatively small I.D. increase in proteinuria was observed in the rat oral route of treatment with Miftil, but this finding was not confirmed by a further breast examination. Miftil, in its common conjugation with the iron receptor, was detected in the oral route of treatment with the same drug, but this finding was not confirmed by a further breast examination. In summary, our data demonstrate that Miftil inhibits the cell growth and motility of rats with a hepatoprotective effect and blocks hepatic liver damage. Miftil has been shown to be well-suited for the treatment of hepatic conditions such as liver cancer and sepsis, and its use has been used as an therapeutic agent for cancer in the human breast and prostate [13–16]. However, its potential against drug-induced liver damage and its effect against aspirin are not known. In conclusion, Miftil inhibits the hepatoprotective androgen-sensitive hepatic proliferation. In the present study, our data demonstrated that Miftil inhibits the hepatic growth by inhibiting the cell growth and motility of rats with a hepatoprotective effect. Acknowledgements This work was supported in part by grants from the European Union of Clinical Research Treatment Research, EU14-02-01/02, EU14-02-01/02, NCT01-01/02, and U29-JED03/02 from the National Natural Science Council (no. FR3-1995-06-01). References [1] P. Knaus, J. A. McManus, J. H. Gordon, J. H. Gonzalez, S. M. Ritchie, T. S. Smith, R. A. Cook, and G. C. Russell. 1999. Human therapeutic agent: aspirin and its role in hepatic carcinogenesis. Nat. Rev. [2] S. M. Harris, C. C. Adam, N. C. Gregory, M. A. Bannan, D. E. Brown, and P. M. Vogel. 2004. In vivo: Determination of the oxidative stress-induced extracellular expression of the critical enzyme, P3-oxidase. Int. J. [3] S. E. Eller, T. R. Ottman, S. K. Rehle, J. D. Hays, S. S. Black, and S. M. Harris. 1996. Erythrocyte activity of aspirin in humans: relevance to the development of hepatic carcinogenesis. [4] S. F. Jilawian, W. M. Bracht, M. B. Yu, and J. J. D. Brown. 1999. Hormone-induced inhibition of human canonical endothelin-beta 1-induced production of apoptotic proteins: role of specific nuclear receptor kinases. J. Biol. Chem. 271:8331–8341. [5] G. C. Russell, J. H. Gordon, J. H. Madden, and P. M. Vogel. 2002. Robust prognosis of patients with hepatic carcinogenesis: a systematic review. [6] J. H. Gordon, M. E. Russell, J. H. Madden, and P. M. Vogel. 1989. The role of nuclear factor-kappa B in the development of hepatic carcinogenesis. J. Clin. [7] A. B. Butler, B. Heidrich, R. J. Bont, S. M. King, and E. H. Ellis. 1998. The mechanisms of aspirin-induced apoptosis of human hepatocellular carcinoma. Cell. Mol. Pharmacol. Rev. 14:443–449. [8] S. M. Harris, M. A. Baker, A. F. Young, P. M. Vogel, and D. E. Brown. 1996. Capillary regulation of hepatic apoptosis by aspirin and its role in human carcinogenesis. Int. J. Cancer 40:1159–1164. [9] N. C. Dutcher, T. D. Carney, C. T. Oliver, and R. A. Smith. 2000. Acetylation of N