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Meeting abstract

Cancer and nutrition: role of amino acids (AA) for the regulation of hepatocellular apoptosis

Miriam Mayer[1], Adolf Ellinger[2], Anneliese Karwan[1], Rolf Schulte-Hermann[1] and Wilfried Bursch*[1]

Address: [1]Department of Medicine I, Institute of Cancer Research, Research Unit Toxicology and Prevention, Medical University of Vienna, Austria and [2]Center for Anatomy and Cell Biology, Department of Cell Biology and Ultrastructure Research, Medical University of Vienna, Austria

Email: Wilfried Bursch* - wilfried.bursch@meduniwien.ac.at

* Corresponding author

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Since decades nutrition is known to play a central role in development of cardiac diseases, cancer and diabetes. However, the causative nutritional factors and their mode of action are not well understood. Accumulating evidence suggest that certain nutrients (including glucose and AA) may regulate cell signalling events/gene expression independently of hormones, possibly acting through specific nutrient "sensor" mechanisms [1]. Previous in vivo studies by ourselves on the regulation of rat hepatocyte proliferation and apoptosis revealed that the lack of glucose and/or AA blocks the initiation of DNA synthesis (G_1/S-transition). Furthermore, feed restriction favoured apoptosis in rat liver. Here we report on the role of AA for apoptosis of human hepatoma cells (HCC-1.2). The following changes in AA composition – as compared to standard RPMI 1640 medium – exerted a pro-apoptotic action, without and with TGF-β1 treatment: (1) deprivation of branched chain AA, with Val > Leu > Ile when tested individually; (2) deprivation of Phe, Trp, Lys, Met, Thr, either as group or, with the exception of Met, when tested individually; (3) deprivation of His and Gln seemed less effective than the other individual AA tested; (4) occurrence of autophagy was demonstrated light- and electron-microscopically. In summary, this first series of experiments revealed HCC-1.2 cells sensitive to the AA deprivation, rendering this model suitable for studying pharmacological actions of AA.

References

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