Nephroprotective effect of cystathionine is due to its diverse action on the kidney and Ehrlich ascites tumor cells

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Abstract:
Tumor cells, unlike normal cells, are characterized by trace cystathionase (CST) activity and sulfane sulfur levels. The present studies aimed to establish whether cystathionine (CT), a substrate of cystathionase, can selectively influence the thiol-dependent antioxidant power of the kidney and Ehrlich ascites tumor (EAT). CT treatment reversed the changes in renal concentrations of non-protein thiols (NPSH), reactive oxygen species (ROS), sulfane sulfur and activities of rhodanese, cystathionase and glutathione S-transferase (GST) in tumor-bearing mice, which returned to the level observed in healthy animals. The results demonstrated that CT corrected all harmful changes in the mouse kidney induced by EAT. In contrast, CT did not elicit such effect in EAT cells, in which it only increased ROS level. It indicates that CT can selectively protect the kidney of tumor-bearing mice against nephrotoxicity of drugs as well as restore biological function of sulfane sulfur. On the other hand, cisplatin (CP) did not affect any of the parameters under study in the kidney of tumor-bearing mice. Interestingly, cisplatin markedly lowered glutathione S-transferase activity and increased sulfane sulfur level and rhodanese activity in tumor cells. It is also worth noting that CP doses devoid of nephrotoxic effect in tumor-bearing mice could enhance cystathionine action on the kidney, causing an additional increase in NPSH and CST and rhodanese activity.

Key words:
cisplatinum, cystathionine, Ehrlich ascites tumor, glutathione, kidney, nephrotoxicity