Selenium prevents tumor development in a rat model for chemical carcinogenesis

Previous studies in animals and humans have shown that selenium compounds can prevent cancer development. In this work we studied the tumor preventive effect of selenium supplementation, administrated as selenite, in the initiation, promotion and progression phases in a synchronized rat model for chemically induced hepatocarcinogenesis, the resistant hepatocyte model. Selenite in supra-nutritional but subtoxic doses (1 and 5 p.p.m.) was administrated to the animals through the drinking water. Such supplementation during the initiation phase did not have a tumor preventive effect. However, selenite treatment during the promotion phase decreased the volume fraction of pre-neoplastic liver nodules from 38% in control animals to 25 (1 p.p.m.) and 14% (5 p.p.m.) in the selenium-supplemented groups. In addition the cell proliferation within the nodules decreased from 42% in the control to 22 (1 p.p.m.) and 17% (5 p.p.m.).

Immunohistochemical staining for the selenoenzyme thioredoxin reductase 1 revealed an increased expression of the enzyme in liver nodules compared with the surrounding tissue. The activity was reduced to 50% in liver homogenates from selenium-treated animals but the activity of the selenoenzyme glutathione peroxidase was essentially unaltered. Selenite treatment (5 p.p.m.) during the progression phase resulted in a significantly lower volume fraction of liver tumors (14 compared with 26%) along with a decrease in cell proliferation within the tumors (34 compared with 63%). Taken together our data indicate that the carcinogenetic process may be prevented by selenium supplementation both during the promotion and the progression phase.

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