Selenium and anticarcinogenesis: underlying mechanisms.

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PURPOSE OF REVIEW: To discuss recent research related to anticarcinogenic mechanisms of selenium action in light of the underlying chemical/biochemical functions of the selenium species, likely to be executors of those effects.

RECENT FINDINGS: Recent studies in a variety of model systems have increased the understanding of the anticarcinogenic mechanisms of selenium compounds. These include effects on gene expression, DNA damage and repair, signaling pathways, regulation of cell cycle and apoptosis, metastasis and angiogenesis. These effects would appear to be related to the production of reactive oxygen species produced by the redox cycling, modification of protein-thiols and methionine mimicry. Three principle selenium metabolites appear to execute these effects: hydrogen selenide, methylselenol and selenomethionine. The fact that various selenium compounds can be metabolized to one or more of these species but differ in anticarcinogenic activity indicates competing pathways of their metabolic and chemical/biochemical disposition. Increasing knowledge of selenoprotein polymorphisms has shown that at least some are related to cancer risk and may affect carcinogenesis indirectly by influencing selenium metabolism.

SUMMARY: The anticarcinogenic effects of selenium compounds constitute intermediate mechanisms with several underlying chemical/biochemical mechanisms such as redox cycling, alteration of protein-thiol redox status and methionine mimicry.

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