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The Chemopreventive Mechanism of Selenium: Selenomethionine Regulation of P53 Activation

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Recently, selenium has been reported to have cancer-preventive properties, although the mechanism is little known. To elucidate the mechanism, we examined the effect of selenomethionine (SeMet) on the functional status of p53. Here we show the activation of p53 tumor suppressor in the presence of SeMet without DNA damage. In addition, p53-dependent response in SeMet treatment was blocked by a dominant-negative Ref1. This experiment provides that p53 activation might be modulated by redox signaling. Furthermore DNA repair was significantly activated in SeMet-treated cells on p53-dependent pathway. This study suggests that SeMet may play an important role to DNA repair by p53 activation.

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