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A radical approach to cancer.

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Abstract

Reactive oxygen species are known to be potentially dangerous, but are also needed for signal-transduction pathways. Tumor cells have relatively low amounts of superoxide dismutase (SOD), which quenches superoxide anion ($O_2^{(-)}$), and as a result of a higher level of aerobic metabolism, higher concentrations of $O_2^{(-)}$, compared to normal cells. But this may not be true of all tumor cells. Some tumor cells have relatively higher amounts of vitamin E, a potent anti-oxidant, and a higher level of anaerobic metabolism, resulting in a balance that is tilted more towards higher anti-oxidant capacity. In both instances of higher aerobic and anaerobic metabolism methods designed to augment free radical generation in tumor cells can cause their death. It is suggested that free radicals and lipid peroxides suppress the expression of Bcl-2, activate caspases and shorten telomere, and thus inducing apoptosis of tumor cells. Ionizing radiation, anthracyclines, bleomycin and cytokines produce free radicals and thus are useful as anti-cancer agents. But they also produce many side-effects. 2-methoxyoestradiol and polyunsaturated fatty acids (PUFAs) inhibit SODs and cause an increase of $O_2^{(-)}$ in tumor cells leading to their death. In addition, PUFAs (especially gamma-linolenic acid), 2-methoxyoestradiol and thalidomide may possess anti-angiogenic activity. This suggests that free radicals can suppress angiogenesis. Limited clinical studies done with gamma-linolenic acid showed that it can regress human brain gliomas without any significant side-effects. Thus, PUFAs, thalidomide and 2-methoxyoestradiol or their derivatives may offer a new radical approach to the treatment of cancer.

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