

Re: Breast cancer – what to eat

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- *From:* ironjustice <teamtanner@xxxxxxxxxxxxx>
 - *Date:* Sat, 17 Nov 2007 12:44:25 –0800 (PST)
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On Nov 12, 6:32 am, "BASTARDO 65346354354" <n...@xxxxx> wrote: What kind of food and food supplements should she eat? <<

Anticancer Res 2002 Sep–Oct;22(5):2685–92

Induction of apoptosis by iron depletion in the human breast cancer MCF–7 cell line and the 13762NF rat mammary adenocarcinoma in vivo.

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[Medline record in process]

It is known that the interruption of normal iron metabolism with chelators of iron, toxic metals, toxic metals bound to transferrin, or anti–transferrin receptor antibodies leads to significant inhibition of tumor cell growth in cell culture systems and animal models. In the present study, we found that iron depletion was produced by the iron chelator deferoxamine mesylate, the free toxic metals gallium or indium, and the toxic metals gallium or indium bound to transferrin in the MCF–7 human breast cancer cell line, and this induced the condensation and fragmentation of chromatin, and the formation of DNA fragments characteristic of apoptosis. The induction of apoptosis

was quantitated with acridine orange and ethidium bromide staining of apoptotic cells, separation of fragmented DNA from radiolabeled cells, and in situ terminal deoxynucleotidyl transferase–mediated dUTP–digoxigenin nick end labeling (TUNEL) assays. The apoptosis, caused by deferoxamine mesylate, and gallium or indium bound to transferrin in the MCF–7 cells, can be completely inhibited by excess ferric chloride or equimolar iron–loaded transferrin. Gallium–transferrin and indium–transferrin complexes induced more apoptosis than their respective salts in the MCF–7 cells. Deferoxamine mesylate induced a small increase in the endogenous expression of both the bcl–2 and bax genes in the MCF–7 cells and this can be prevented by ferric chloride. In the 13762NF rat mammary adenocarcinoma model, in situ TUNEL assays showed that the iron–deficiency following a low iron diet or intravenous injection of deferoxamine mesylate produced 5.32 +/- 3.90% and 6.46 +/- 3.58% of apoptotic cells, respectively, compared to 2.01 +/- 1.20% of apoptotic cells in the control rats maintained on a normal diet ($p < 0.05$ and $p < 0.01$, respectively, Student's t–test). This