COPPER AND CANCER

The copper activated enzyme, lysyl oxidase, has been mentioned in regard to its effect on collagen and its cross-linking functions. One avenue of this cross-linking that has yet to be mentioned is its connection with cancer. Breast cancer invasiveness was found to be directly correlated with the presence of lysyl oxidase. When higher levels of lysyl oxidase were present, a scar-like barrier was formed around the tumor and the spread of the tumor was prevented. When there were low levels of lysyl oxidase, a poor grade collagen barrier was formed around the tumor and the spread of the tumor and normal tissues became susceptible to degradation and breakdown by the spreading tumor. The high levels of lysyl oxidase were found to be a primary host defense mechanism forming and early and strong barrier preventing the spread of a tumor.

There is a cytotoxic action of ascorbate in the presence of copper ions on malignant melanoma cells.

Copper is necessary for the synthesis of an enzyme called *catalase*. This enzyme may be the most powerful defensive mechanism a cell has to prevent its breakdown by hydrogen peroxide. We can think of it as a cell's anti-artillery. An invading cell such a bacteria or cancer uses hydrogen peroxide to destroy man's normal cells. It's a very powerful weapon at the cellular level. The normal host cell uses catalase to break down hydrogen peroxide into its components of water and oxygen, leaving the invader's weapon useless. If there's a lack of catalase, the hydrogen peroxide can quickly alter and destroy a normal cell. In a tumor, the catalase is used to prevent normal cell destruction, and in hindering the tumor to increase in size.

Catalase depends on a regulator, as do all enzymes. The regulator in this case is an iron compound in the form of heme. Copper is absolutely essential in the utilization by man to form heme. Without copper, normal heme cannot be made and the heme cannot be utilized to turn on catalase. Low catalase in the liver is not due to the lack of heme's components—iron and protein—but due to a defect in the formation of the heme molecule by copper. The liver is the center of catalase formation, and every cancer sufferer has lower catalase levels than normal. It has been shown that if a tumor is removed in the liver, or in another part of the body surgically, the catalase activity returns to normal. If the tumor redevelops, the catalase level drops again.

Catalase is directly and indirectly inhibited by fluoride. This has been shown as far back as 1946 and later in 1961. The direct mode of action is by binding the iron (heme) **and indirectly by binding the copper and preventing the hemes normal formation. Fluoride makes one more susceptible to microbial, and cancerous attacks on cells by reducing the normal cells primary defenses, its armor called catalase.**

The reduction of catalase in the liver of cancer patients is due to the breakdown of heme synthesis. The catalase and cytochrome oxidase deficiency is not due to the lack of iron, but to a defect in the formation of the heme molecule. Copper regulates this formation. The amounts present have been correlated to the amount of copper found in the diet.

Back in 1939, Shultze focused on the levels of cytochrome oxidase in the liver of rats when fed a diet that was first normal and then deficient in copper. After several weeks on low copper diet, the cytochrome oxidase activity of the liver fell to 1/7 of the normal level. When the rats were given just a small supplement of copper, (.1 mg cu/day)**, the enzyme activity rose to near normal levels after only five days. This liver enzyme is quite sensitive to the dietary levels of copper.**

The copper activated enzyme, *lysyl oxidase*, has already been mentioned in regard to its effect on collagen and its cross-linking functions. One avenue of this cross-linking that has yet to be mentioned is its connection with cancer. At the Pasteur Institute in France, breast cancer invasiveness was found to be directly correlated with the presence of lysyl oxidase. When higher levels of lysyl oxidase were present, a scar-like barrier was formed around the tumor and the spread of the tumor was prevented. When there were low levels of lysyl oxidase, a poor-grade collagen barrier was found around the tumor and normal tissues became susceptible to degradation and breakdown by the spreading tumor. The high levels of lysyl oxidase were found to be a primary host defense mechanism forming an early and strong barrier preventing the spread of a tumor.

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