Ray Peat's Newsletter

A New Approach to Detoxifying

Ernest Krebs, the inventor of the laetrile cancer treatment, compiled some interesting facts about cancer metabolism. Noting that cancer cells are typically fairly deficient in the enzyme rhodanese, which protects cells against cyanide poisoning if the dose isn't too high, he theorized that cyanide might selectively kill the relatively unprotected cancer cells, especially if the dose could be concentrated near the cancer.[1]

While amygdalin releases a large proportion of its cyanide in the bowel, his original laetrile, the glucuronide form, should liberate its cyanide largely in areas of inflammation or injury, including cancers.

Although cancer cells have a low level of rhodanese, they typically have intense glycolytic metabolism which compensates for their respiratory defect. Otto Warburg and Dean Burk showed pretty decisively that "defective respiration" exists in all of the cancers studied.

Maybe we can see a different meaning in the same facts.

What if the respiratory defect that was so carefully documented by Warburg and Burk is the result of damage to the detoxifying rhodanese enzyme? If cyanide is a general threat to respiration, a deficiency of rhodanese would allow it to damage respiration, and this, according to Warburg, should lead either to cell death or, if the cell can adapt adequately, to the production of cancer.

Rhodanese occurs in all animals, and seems to be localized inside the mitochondria. Although it does handle some other sulfur reactions, its basic function is thought to be the elimination of cyanide, by the addition of sulfur, forming thiocyanate. Thiocyanate is fairly non-toxic, though it can be broken down into cyanide again, and it does have a significant anti-thyroid effect.

Herbivores eat many plants that are rich in cyanide-releasing compounds, so it is logical that they should be equipped with rhodanese. But carnivores are not exposed to these toxic vegetable products, so it is hard to explain why they have the enzyme. The fact that serum albumin, which has many detoxifying functions, carries sulfane sulfur, the form which is used by rhodanese to react with cyanide, supports the idea that rhodanese is involved in the elimination of cyanide.

People who smoke or eat significant amounts of plant material are exposed to cyanide, but the fact that the rhodanese enzyme exists in all animals, whether their diet includes "cyanophoric" compounds or not, suggests that we might want to consider other possible sources of cyanide.

People studying lipid peroxidation in liver cell extracts noticed that carbon monoxide was being produced. Many people have observed that stressed cells emit ammonia.[2] There are industrial processes which use ammonia and carbon monoxide to produce cyanide, using a metal as catalyst.[3] Since carbon monoxide binds to metal atoms, it might be held in a form which reacts easily with ammonia. Then during stress, which causes both lipid peroxidation and ammonia formation, rhodanese would be needed to protect the respiratory cytochromes from the cyanide, which would otherwise inhibit respiratory energy production, and other processes involving the cytochromes. Within the mitochondria, a cytochrome P-450 converts cholesterol to pregnenolone. The loss of both energy and steroid hormones would have major consequences. Outside the mitochondria, many other cytochrome functions will be inhibited; other types of enzymes would also be inhibited.

Another possible source of cyanide would be bacteria in the intestine; methane and ammonia are other possible starting materials for making cyanide. The urea cycle itself, inside the mitochondria, is a conceivable source.

Even if dietary plant material turns out to be the main source of cyanide, it is important to be able to eliminate it efficiently.

When flowers of sulfur is taken orally, some of it becomes available for use in the rhodanese system. It might be less irritating and more effective to use a form of sulfur which is chemically more available, namely thiosulfate. (My thoughts on light removing carbon monoxide from cytochromes led me to think about photography; sodium thiosulfate is used to clear the photographic emulsion, removing the silver which was not exposed to light. In that sense, its activity as a reductant is equivalent to light's action).

In acute cyanide poisoning, a large dose of sodium thiosulfate (12 grams) is injected rapidly; it is poorly absorbed when taken orally. I decided to try small oral doses, about 50 mg. a few times per day. It has improved my tolerance for chocolate and wheat, which I still reacted to in spite of using pregnenolone, which was permitting me to eat many other foods that earlier would have caused migraines and asthma. Pregnenolone seems to stabilize the cytochromes physically, while rhodanese (with sulfur) restores their chemical activity. Some druggists still sell sodium thiosulfate as a treatment for skin diseases. It has been used to treat arthritis, which would be logical if it is able to increase formation of pregnenolone and the various steroid hormones.

Since very high levels of cortisone destroy the detoxifying cytochrome enzymes,[4] the use of thiosulfate – to restore any of the remaining cytochromes which might be blocked by cyanide – would seem to be appropriate as part of an anti-stress approach to disease. As far as I know, it hasn't been tried as part of cancer therapy; improving a cancer cell's viability might, as many people believe, reduce its virulence, but it might just make it a more energetic cancer cell.

References

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3. *Chemical Abstracts 101* (1984), P9548 (NH_3 and CO); 93656 (NH_3 and CH_4); 115982 (propylene and nitrogen dioxide).
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For subscriptions write to:
Ray Peat's Newsletter
P.O. Box 3427
Eugene, OR 97403