

Selenium, Cancer and Mercury

Selenium, is a reddish-brown solid Metalloid, somewhat translucent, and of dull metallic glance, insoluble in water, and alcohol. It exists crystalline, and vitreous; at water's boiling heat it melts and boils, evolving odour like stale horse-radish.

Selenium is a potent immune stimulator – the most potent immune stimulator of all some think. Selenium is an essential component of thyroid metabolism and antioxidant defense, as well as immune function. It may improve activation and proliferation of B-lymphocytes and enhance T-cell function. Selenium is essential for our immune system to function at optimal performance. Thus we should not be surprised to find out those cancer patients with low selenium levels tend to have a wider spread of the disease, more recurrences and die sooner.

Blood selenium levels often indicate the presence of cancer and even the severity of cancer in a patient.

Selenium influences both the innate, 'non-adaptive' and the acquired, 'adaptive' immune system. The innate immune system includes barriers to infection and nonspecific effector cells such as macrophages. Both the T and B lymphocytes form the major effector cells of the acquired system that mature with exposure to immune challenges. Selenium-deficient lymphocytes are less able to proliferate in response to mitogen, and in macrophages, leukotriene B₄ synthesis, which is essential for neutrophil chemotaxis, is impaired by this deficiency. These processes can be improved by selenium supplementation. The humoral system is also affected by selenium deficiency; for example, IgM, IgG and IgA titers are decreased in rats, and IgG and IgM titers are decreased in humans. In endothelial cells from asthmatics, there is a marked selenium deficiency that results in an increase in expression of adhesion molecules, which causes greater adhesion of neutrophils.

Selenium is also involved in several key metabolic activities through its selenoprotein enzymes that protect against oxidative damage. Further, selenium deficiency may allow invading viruses to mutate and cause longer-lasting, more severe illness. Animal research has shown selenium and vitamin E have synergistic effects, enhancing the body's response to bacterial and parasitic infections.

Proving the point that selenium is a potent immune stimulator is an 18-month study of 262 patients with AIDS found those who took a daily capsule containing 200 micrograms of selenium ended up with lower levels of the AIDS virus and more health-giving CD4 immune system cells in their bloodstreams than those taking dummy pills. These AIDS patients who took selenium were able to suppress the deadly virus in their bodies and boost their fragile immune systems, adding to evidence that selenium has healing powers we need to pay attention to in treating cancer patients. Those with severely compromised immune systems due to AIDS had dramatically better immune system response with selenium supplementation and this finding is consistent with the information presented by the NIH.

Selenium is an important weapon against cancer.

As an antioxidant nutrient, selenium prevents the action of free radicals which are believed to be causative agents behind degenerative diseases such as premature ageing, cancer and atherosclerosis. Clinical trials have also indicated that selenium can have a role to play in combating oxidative diseases, enhancing the immune response, increasing male fertility, improving psychological mood scores and reducing the pain and stiffness in arthritis sufferers.

The implicit importance of selenium to human health is recognized universally. Selenium is incorporated as selenocysteine at the active site of a wide range of selenoproteins.

Dr. Emanuel Revici, a Romanian-born physician, scientist, author, and humanitarian had five major papers on lipids, pain, and cancer deposited by the Pasteur Institute into the eminent National

Academy of Sciences during the Second World War. By 1948, Revici had begun exploring the use of selenium in treating cancer and as a means for rendering radiation less harmful. Dr. Revici's use of selenium in the treatment of cancer predates mainstream interest in this mineral by more than twenty years. Selenium is one of the major trace elements always found deficient in cancer-prone populations. Research has shown that it is of value not only in preventing cancer but also in treating it.

Revici uses a special molecular form of selenium (bivalent-negative selenium) incorporated in a molecule of fatty acid. In this form, he can administer up to 1 gram of selenium per day, which corresponds to 1 million micrograms per day, reportedly with no toxic side effects. In contrast, too much selenite (hexavalent-positive selenium) has toxic effects on animals, so human intake of commercial selenite is limited to a dosage of only 100 to 150 micrograms by mouth. Dr. Revici often administered his nontoxic form of selenium by injection, usually considered to be four times more powerful than the form given orally.

Dr. Gerhard Schrauzer, professor of biochemistry at the University of California at La Jolla, publicly credited Revici for "having discovered pharmacologically active selenium compounds." Dr. Gerhard Schrauzer noted almost 30 years ago if every woman in America took 200 micrograms of supplementary selenium daily that breast cancer rates would rapidly decline in the space of a few short years. Dr. Schrauzer is professor emeritus from the University of California, San Diego School of Medicine and has chaired two world conferences on selenium and cancer.

Dr. Richard Donaldson of the St. Louis Veterans' Administration Hospital conducted a clinical trial with terminally ill cancer patients. He found that when he could rise the patients' blood levels of selenium into the normal range, their pain and tumor sizes were often reduced. In a 140 patient study of cancer victims treated with selenium, Dr. Donaldson reported in 1983 that some patients

deemed terminal with only weeks to live were completely free of all signs of cancer after four years; all the patients showed a reduction in tumor size and in pain.

The amount of selenium needed to obtain normal blood levels varied from person to person. Normal healthy people usually were seen to have normal blood selenium levels on normal diets however it seemed that cancer patients had lower selenium levels on similar diets. Apparently they could not get enough without supplements. Dr. Donaldson found that he had to supplement the cancer patients with at least 200 to 600 micrograms of selenium per day and in some cases 2,000 micrograms of selenium per day were required to obtain normal blood selenium levels.

There are now seven population studies in the past six years that examined the possible connection between selenium and prostate cancer. All but one of them has found selenium protective.

In one recent study, men with the highest levels of selenium in their blood were about half as likely to develop advanced prostate cancer as the men with the lowest blood selenium. The “Nutritional Prevention of Cancer Project” (NPC) was a controlled, randomized cancer prevention trial in which 1,312 patients received a daily 200 mcg dose of selenium or a placebo for up to 10 years.

A 1996 study by Dr. Larry Clark of the University of Arizona showed just how effective selenium can be in protecting against cancer. In the study of 1,300 older people, the occurrence of cancer among those who took 200 micrograms of selenium daily for about seven years was reduced by 42 percent compared to those given a placebo. Cancer deaths for those taking the selenium were cut almost in half, according to the study that was published in the Journal of the American Medical Association on December 25, 1996. In addition, the people who had taken selenium had 63 percent fewer prostate cancers, 58 percent fewer colorectal cancers, 46 percent fewer lung cancers and overall 37% fewer cancers. Selenium was found to

reduce the risk of lung cancer to a greater degree than stopping smoking.

It is noteworthy, that the Food and Drug Administration has determined that there is sufficient evidence to warrant a qualified health claim for Se and cancer. Furthermore, the recent discovery that defects in the SECIS-binding protein 2 (SBP2), which is an indispensable protein for the incorporation of Se into the selenoproteins, result in thyroid dysfunction.

Much of what selenium does you cannot feel while it is doing it, but if you do not have it, then you will feel it later and you will not like the feeling at all – especially if the feeling of dying is not a turn-on to you.

One important study found that high blood levels of selenium are associated with a four- to fivefold decrease in the risk of prostate cancer. Scientists at Stanford University studied 52 men who had prostate cancer and compared them to 96 men who did not. One surprising finding was that blood levels of selenium generally decreased with age. It is well known that the risk of prostate cancer increases dramatically as one age.

Those who have studied geographical differences have seen that in low-selenium regions, higher death rates occurred from malignant lymphomas and cancers of the tongue, esophagus, stomach, colon, rectum, liver, pancreas, larynx, lung, kidneys and bladder. Dr. Harold Foster has stated that death rates in the USA for breast, colon, rectal and lung cancer are lower when blood selenium levels are high. Dr. Foster is the one to have reported that cancer patients with low selenium levels tend to have a wider spread of the disease, more recurrences and die sooner. This is critical information that fits rationally into the entire picture of selenium being compiled by medical science.

The West African country of Senegal is dominated by high concentrations of selenium in the soil and thus in their foods and as

expected we find that Senegalese males had the world's lowest rates for cancer of the trachea, bronchus and lung; stomach and colon; the fourth lowest for prostate cancer and sixth lowest for esophageal cancer. Senegalese women had the lowest incidence of cancers of the trachea, bronchus, lung, esophagus, stomach and colon and second lowest for breast cancer and fifth lowest for cancer of the uterus.

In China, where the selenium levels in the soils varies much more dramatically than in the United States and the population is less mobile, an ecological study in 1985 showed dramatic results in linking cancer with selenium deficiencies. Dr. Shu-Yu Yu measured the selenium content of blood stored in blood banks in 30 different regions in China, and classified the regions as high selenium, medium selenium, and low selenium. They then compared death rates from cancer to the selenium rates and found there was an exact correlation. In the low selenium classification, three times as many people died from cancer as in the high selenium classification.

There is no doubt that selenium is essential for human health and that these elements may protect against cancer and other diseases. For this reason people in regions which are naturally rich in selenium tend to live longer. Selenium, especially when used in conjunction with vitamin C, vitamin E and beta-carotene, works to block chemical reactions that create free radicals in the body (which can damage DNA and cause degenerative change in cells, leading to cancer). Selenium also binds strongly with mercury protecting us from its damaging effects.

Selenium and the Rising Tide of Mercury

Tuna is uniformly rich in selenium. Nearly 300 scientific studies have demonstrated that this essential element protects against mercury exposure. Any group carping about mercury in fish without also talking about selenium is hiding half the story.

One of the main concerns is about mercury and its toxicity. Although the majority of attention has been given to fish in the media as

posing the great health threat in regards to mercury toxicity we have to entertain the possibility that because of selenium, which is an antidote for mercury, fish could be not be as much of a problem as dental amalgam, mercury injected via vaccines, or direct absorption through the air, water and other foods which are becoming increasingly contaminated.

The way things are now public attention is focused mostly on fish consumption as the main danger from mercury and this is actually a red herring removing us from focusing on the total threat that mercury has become. It moves our attention away from the combined effects from all sources put together.

The first report on the protective effect of selenium against mercury toxicity appeared in 1967. Since then, numerous studies have shown selenium supplementation counteracts the negative impacts of exposure to mercury, particularly in regard to neuro-toxicity, fetotoxicity, and developmental toxicity. The ability of selenium compounds to decrease the toxic action of mercury has been established in many species of mammals, birds, and fish. The detoxifying effect of selenium on mercury toxicity is due to a formation of a biologically inactive complex containing the elements in an equimolar ratio. The complex is unable to pass biological barriers, placenta and choroid plexus and is stored in the liver and the spleen, even in the brain in a non toxic form.

It is well recognized that mercury and sulphur bind together to form complexes. This binding property is the basis of chelating therapy used as a treatment in cases of acute and chronic mercury poisoning. The complexes between mercury and selenium are less generally known but of much higher affinity. Physiologically, sulphur is far more abundant than selenium, yet because of selenium's higher affinity, mercury selectively binds with selenium to form insoluble mercury selenides. This interaction has been assumed to be a 'protective' effect whereby supplemental selenium complexes the

mercury and prevents negative effects in animals fed otherwise toxic amounts of mercury.

When selenium and mercury are found together, they connect forming a new compound making it difficult for the body to absorb the mercury separately. Scientists have also tagged cysteine in fish binding with mercury also making it safer to eat. When mercury 'binds' to selenium or cysteine it is no longer free to 'bind' to anything else — like brain or kidney tissue.

Selenium deficiency results not only in a decrease of GSHPx activity, but also in a decrease of GSHPx protein.

Dr. Laura Raymond and Dr. Nicholas Ralston of the University of North Dakota tell us that, “Measuring the amount of mercury present in the environment or food sources may provide an inadequate reflection of the potential for health risks if the protective effects of selenium are not also considered. Owing to the extremely high affinity between mercury and selenium, selenium sequesters mercury and reduces its biological availability. It is obvious that the converse is also true; as a result of the high affinity complexes formed, mercury sequesters selenium. This is important because selenium is required for normal activity of numerous selenium dependent enzymes.”

Selenium's involvement is apparent throughout the mercury cycle, influencing its transport, biogeochemical exposure, bioavailability, toxicological consequences, and remediation.

Glutathione happens to be the most important of these selenium dependent enzymes. Mercury is highly toxic but mercury's toxic ruin varies greatly with selenium and glutathione levels. These are the key variables that determine the harm done or the power each individual has to escape the poisonous effect of mercury and other dangerous toxins in the environment. Our defensive shields against both acute and chronic exposure to mercury depend very much on selenium and

glutathione. Selenium is useful as a controlling agent for mercury, which attacks insulin and its binding sites.

Selenium is a hugely important subject for more reasons than easily meets the eye. Mercury binds with selenium reducing its availability for other functions i.e., for glutathione production in the cells. Thus it is not unreasonable reasoning to see a chain of events starting with mercury contamination passing from mother to child in utero (via mercury vaccines for mother and mothers dental amalgams and fish consumption) stripping the yet to be born of selenium. Newborns receiving more contamination through mother's milk add to the profile of babies having their selenium levels depleted and thus their glutathione levels set too low to resist childhood vaccines containing thimerosal (fifty percent ethyl mercury) and other toxic elements.

The last 25 years the average daily selenium intake has fallen from 60µg/day to 35µg/day. The UK government has established a Reference Nutrient Intake (RNI) level of selenium at 75µg/day.[xxxix] Therefore a nutritional gap now exists between the actual recommended level of daily selenium and what people are actually achieving through their diets. When we calculate in the 'Rising Tide of Mercury' and the extra demands that makes on our selenium stores/nutritional intake we can now see the disaster that has been in the making for decades.

Studies have implicated reactive oxygen species (ROS) and depletion of intracellular glutathione as major contributors to mercury-induced cytotoxicity. Selenium is absolutely essential in the age of mercury toxicity for it is the perfect antidote for mercury exposure. It is literally raining mercury all over the world but especially in the northern hemisphere. And of course with the dentists poisoning a world of patients with mercury dental amalgam and the doctors doing the same with their mercury laden vaccines, selenium is more important than most of us imagine.

Selenium offers online in real defense time against mercury. As mercury enters our bodies, if there is sufficient selenium it will mop up the mercury before it can bind to its favorite sulfur sites or pass through the blood brain barrier. Taking more selenium reduces the level of 'free' mercury doing damage. Minerals and trace elements, the basic building blocks of our bodies, are just not as readily available in our diet as they once were and in the case of selenium this is compounded by the fact that certain vast areas of the world have low selenium contents in the soil and thus the food.

An excess of a toxic metal and/or a relative deficiency of a nutritional element can be found as significant contributors to every disease.

General Information on Selenium

High doses of vitamin C (over 1 gram) may reduce the absorption of selenium. This mineral is best taken one hour before or 20 minutes after taking vitamin C supplements.

Selenium deficiency impairs thyroid hormone metabolism by inhibiting the synthesis and activity of the iodothyronine deiodinases, which convert thyroxine (T4) to the more metabolically active 3,3'-5 triiodothyronine (T3). In rats, concurrent selenium and iodine deficiency produces greater increases in thyroid weight and plasma thyrotrophin than iodine deficiency alone, indicating that a concurrent selenium deficiency could be a major determinant of the severity of iodine deficiency.

Later studies showed that serum T4 was maintained at control levels when both dietary iodine and selenium were low, but not when iodine alone, or selenium alone, was low. Activity of thyroidal GSH-Px (erythrocyte glutathione peroxidase) was lowest in rats fed a diet containing high iodine and low selenium. The results suggested that high iodine intake, when selenium is deficient, may permit thyroid tissue damage as a result of low thyroidal GSH-Px activity during thyroid stimulation. A moderately low selenium intake normalized circulating T4 concentration in the presence of iodine deficiency.

Adequate selenium nutritional status may help protect against some of the neurological effects of iodine deficiency. Researchers involved in the Supplementation study in France, which was designed to assess the effect of vitamin and mineral supplements on chronic disease risk, evaluated the relationship between goiter and selenium in a subset of this research population. Their findings suggest that selenium supplements may be protective against goiter. Selenium (Se) in the form of selenocysteine is an essential component of the family of the detoxifying enzymes glutathione peroxidase (Gpx) and of the iodothyronine selenodeiodinases that catalyze the extrathyroidal production of tri-iodothyronine (T₃). Thus, Se deficiency may seriously influence the generation of free radicals, the conversion of thyroxine (T₄) to T₃ and a thyroidal autoimmune process.

Recent studies concluded that a positive effect of Se on thyroidal autoimmune process was shown and indicated that high serum Se levels (>120 ug/l) may also influence the outcome of GD. (Graves disease). A recent study testing the various dosages of selenium confirmed that doses greater than 100mcg of selenium (as L-selenomethionine) were required to maximize glutathione peroxidase activities in autoimmune thyroiditis.

Selenium is also essential for the production of estrogen sulfotransferase which is the enzyme which breaks down estrogen. A deficiency of selenium can thus lead to excessive amounts of estrogen, which may depress thyroid function, and also upset the progesterone-estrogen balance. Animal studies have shown that the addition of selenium supplementation will alleviate the effects of excess iodine intake. Iodine and selenium deficiencies must both be resolved for iodine treatment to be effective.

Selenium (Se), one of the essential trace elements, plays a major part in many metabolic functions.

For magnesium to be retained inside cells you need good antioxidant status. Selenium is the main mineral antioxidant. Foods are unreliable

because food content is dependent on soil levels of selenium. Foods rich in selenium include whole grains, organ meats, butter, garlic and onion. Sea foods are rich in selenium and obviously not dependent on soil levels.

Ironically, until approximately 40 years ago, selenium was known only as a poison. It is now known that selenium is essential for the normal function of many of the systems of the body and selenium deficiency can have adverse consequences on these systems. Selenium can act as a growth factor; has powerful antioxidant and anticancer properties; and supports normal thyroid hormone homeostasis, immunity, and fertility.

Two of the 22 primary amino acids are distinguished by their possession of selenium: selenomethionine and selenocysteine. Selenomethionine is biochemically equivalent to methionine and is chiefly regarded as an unregulated storage compartment for selenium. In contrast, selenocysteine is tightly regulated and specifically incorporated into numerous proteins that perform essential biological functions.

Selenium, Chromium and Heart Disease

Dr. Majid Ali and Dr. Omar Ali write, “Deficiency of selenium and chromium are established risk factors of IHD. Selenium-dependent antioxidant systems are important parts of human antioxidant enzyme systems, especially in the regeneration of glutathione and other thiol antioxidants. An association between low serum selenium levels and atherogenesis, lipid peroxidation in vivo, and progression of carotid atherosclerosis has been reported. Salonen et al. observed that selenium deficiency was associated with an excess risk of myocardial infarction as well as morbidity and mortality from other expressions of coronary artery disease and other variants of cardiovascular disease in Eastern Finland. In this study, cardiovascular death and myocardial infarction were associated with low serum selenium levels in a matched-pair longitudinal study.

Chromium supplementation in patients with type II diabetes results in improved glucose tolerance, lower total cholesterol and triglycerides levels and higher HDL cholesterol levels.”

Intake

The standard of recommended intake levels of selenium is under debate. The UK reference nutrient intake (RNI) is 75 µg per day for men and 60 µg per day for women. The American recommended dietary allowance (RDA), set at 55 µg per day for both men and women. These numbers should be looked at as the bare minimum and do not take into account the increased need for selenium because of the rising tide of mercury in the environment and thus our bodies. Also dosage would be in part dependent on the type of source of selenium used since absorption rates would vary widely.

Forms of Selenium

Back in 1998 Dr. Stephen B. Strum said, “We recommend selenium supplements be given as an organic, rather than an inorganic form. Organic sources of selenium such as selenomethionine, selenocysteine or mixtures of organic forms found in brewer’s yeast have a better safety profile. Recent research indicates higher doses of selenium can be safely given and may possess additional anticancer activity. We currently use daily selenium doses in the 400-800 mcg range in our patients. Other investigators are studying the effects of selenium at much higher doses (1,000-3,000 mcg/day) for prostate cancer and claim to have had little or no toxicity. Clearly, this area is controversial and requires further study.”

Getting better forms of selenium because of the difference in absorption and bioavailability in the various forms of selenium is a good idea. The University of Miami study utilized selenomethionine which has 3 times the bioavailability of the sodium selenite form that is less expensive and more commonly used.

Selenium in its inorganic form is poorly absorbed by the body. Most of the body's selenium comes from organic sources, where selenium is bonded with sulphur-containing amino acids, the commonest being L-selenomethionine. Many nutritional supplements contain the poorly absorbed inorganic selenium. Selenium formulations containing L-selenomethionine are good choices but the ideal delivery system is provided by spirulina and perhaps by yeasts and even now by probiotics. When spirulina is grown in ponds with selenium added, the spirulina absorbs the inorganic selenium transforming it into organic selenium. The selenium becomes protein bonded to the amino acids in spirulina, which are present in abundance.

Selenium is a vital component of the metallo-protein enzyme glutathione peroxidase. This is a major component in the body's free radical defence system. Thus the availability of selenium is the limiting factor in the production of glutathione peroxidase.

Remarks

Your citation of the mercury and selenium research by Dr. Laura Raymond and Dr. Nicholas Ralston of the University of North Dakota is a very important part of this discussion. However, your discussion implies that selenium protects against mercury toxicity. While this is in a way true, you have the discussion backwards. Selenoproteins are absolutely crucial for many cell functions, especially cells involved in nervous system development and function. What Drs. Raymond and Ralston have actually found is that too much mercury robs the cells of these critical selenium-containing proteins, by tightly binding the selenium, as you have described. Thus, it is not direct mercury poisoning or mercury toxicity that is the problem, but rather selenium DEFICIENCY that can result from excess (above the reserve amount of selenium) mercury ingestion/absorption by the body. Fortunately, most common sea foods and ocean fish, including several varieties of tuna, salmon, etc., have far more selenium than mercury. This makes the mercury

content of these species a moot point, and as such, these fish provide valuable selenium for development and function of the nervous system