CHAPTER 1

Is Calcium Really a Killer?

Impartial Science Delivers a "Guilty" Verdict

Blowing the whistle on the toxic effects of excess calcium will undoubtedly provoke a firestorm of deafening propaganda. The dairy industry, the medical establishment, and supplement companies have invested untold time, money, and talent in promoting the necessity of increased calcium consumption along with calcium supplementation in the prevention and treatment of osteoporosis. To implicate calcium — a universally-accepted nutritional "good guy" — as a cause of disease and death will incite retaliation from challenged egos and threatened bank accounts. However, once the evidence gets widespread exposure the fear of class action lawsuits will muster spin-doctors and lawyers to fast and furious action in an effort to limit damage and protect many self-serving interests.

Déjà vu here we come again… During the 50's most of America watched the tobacco industry battle efforts to debunk their claim that smoking was actually a healthy habit. Once they lost that skirmish the fight moved to years of vehement "not true" and "you can't prove it" claims against any attempt to link tobacco and cancer.

Defeated once again, they finally tried to convince the state attorneys general and the public that they were completely ignorant of ties between smoking and lung cancer. At massive cost to their reputation and capital they finally lost! Today no one doubts the veracity of the initial indictments against tobacco. Yet how many unfortunate smokers suffered enormously and had their lives shortened because of an industry's greed?

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In like fashion recent scientific data provides an overwhelming case against calcium. What follows is based on research studies published in peer-reviewed medical journals. When considered as a whole the evidence delivers a resounding "guilty" verdict on the common existence and enormous toxicity of calcium excess in the adult population today.

At the time of this writing little consideration is given to the possibility that anyone, especially the elderly, may be suffering from a toxic calcium excess, even as they continue to dose, and overdose on it. Deeply etched and often-parroted warnings from doctors, the popular press, the dairy industry, and supplement suppliers assert that calcium deficiency is a common and nearly universal problem in postmenopausal women and aging men.

The evidence provided in this chapter shows that the opposite is true. Osteoporosis, by definition, is a degenerative condition of the bone associated with a significant calcium deficiency in the bony structural matrix. Somehow the observation of a calcium-deficient state in osteoporotic bone is promoted as proof of a general, body-wide, calcium deficiency.

The scientific evidence, however, paints a much different picture: the degree of calcium deficiency in osteoporotic bone is actually an indicator of the amount of excess calcium that has taken up residence in non-bone tissues. The real problem is not a lack of calcium in the diet, but rather a "relocation" of calcium from the bones to other areas of the body.[1,2,3]

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The body-wide distribution of excess calcium is of far greater concern to the longevity and well-being of an older person than any of the problems associated with osteoporosis. Not only does increasing calcium intake fail to improve bone strength, it fuels calcium excess everywhere in the body.

This excess calcium in non-bone tissues been shown to increase mortality from all causes. Not only that, it has specifically been linked to a substantially greater risk of death from America's two deadliest diseases: coronary heart disease and cancer. Consider this sobering evidence…

Excess Calcium Promotes Heart Disease

You are 30% more likely to have a heart attack and up to 20% more likely to have a stroke if you take an extra 500 mg of calcium or more per day — that's the consensus derived from a comprehensive review of 15 independent clinical investigations. The reviewing researchers reported that subjects taking calcium supplements (500 mg or more per day) had a 27 to 31% higher risk of heart attack and a 12 to 20% greater risk of stroke.[4-6]

Dump more calcium into the caldron and the brew becomes even more deadly. A study of over 61,000

participants viewed over a 19-year period concludes that those with calcium intakes over 1,400 mg/day had an alarming 40% increased risk of death from cardiovascular disease in general and a 114% increase in risk of death from a reduced flow of blood to the heart muscle (ischemic heart disease).[7]

Over one-third of Americans over the age of 45 have evidence of arterial calcification.

Another recent clinical trial compared individuals who regularly supplemented with calcium with individuals who took no supplements at all. These researchers also concluded that those who take supplements had a significantly increased risk of heart attack.[8] The same study further found that calcium supplementation significantly increased total cholesterol levels in postmenopausal women. Total cholesterol is a factor generally considered to be an important measure of coronary artery disease risk.

Healthy postmenopausal women taking calcium supplements were the subjects of a large, 5-year population study. Investigators reported a substantial increase in vascular event rates, such as heart attacks and strokes. Higher calcium intake was further implicated because these vascular events were even more pronounced in women who claimed to be highly compliant in taking their supplements, which would suggest a larger total ingestion of calcium.[9,10]

Perhaps even more conclusive evidence is found through the use of computed tomography (CT Scans). This powerful diagnostic tool uses computer technology and x-rays to produce cross-sectional "slices" or images of bodily structures. CT Scans reveal that over one-third of Americans over the age of 45 have evidence of arterial calcification.[11] This percentage rises drastically with greater

age, literally skyrocketing in postmenopausal women as well as in testosterone-deficient males.

The calcification of arteries is so intrinsically related to coronary disease that the measurement of calcification in these vessels is used to assess the development and progress of the disease.[12,13] A 1990 study demonstrated that calcium content in arterial plaque increases as the plaque develops. The earliest clearly visible evidence of atherosclerosis appears as fatty streaks in the arterial walls. Investigators reported that these fatty streaks had 13 times more calcium than healthy arterial tissues. Moderately evolved plaques had 25 times more calcium than normal, and fully developed plaques had 80 times more calcium. Roughly 50% of the dry weight of the advanced plaques was comprised of calcium salts. Early stages of atherosclerosis appeared cholesterol-laden, while the advanced plaques were very calcium-rich.[14]

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Other investigators also report that older patients had increased calcium content in their atherosclerotic plaques when compared to younger patients.[15] This is clearly consistent with the increasing degrees of excess calcium ingestion observed in older patients.

There is also evidence that calcium is playing an important role in the early development of atherosclerosis, even when the coronary artery calcium score is zero. Plaques are not only present in individuals with zero scores,[16] the distribution of the plaques is similar to that seen in patients with calcified plaques.[17,18]

This strongly suggests that a younger person with plaque should still have the same concerns of calcium excess since calcium likely plays just as important a role

in the early evolution of plaque, before it finally becomes detectable by a CT Scan.

Plaque does not just appear one day with an easily detectable calcium load without an earlier stage of development when it was not detectable. A coronary calcium score of zero would be spectacular in an 80-year old, but in a 40-year old it needs to be correlated with other laboratory data and clinical observations. In the case of the younger person with a minimal calcium load and a coronary artery calcium score of zero it should never be assumed that lifestyle does not need modifications because the stage for detectable calcium accumulation might already be set. Lifestyle and risk factor modification is best begun when calcium has not yet accumulated to a detectable level.

Calcifications in the thyroid gland were found to increase the incidence of malignancy.

Excess Calcium Promotes Cancer

Calcium and cancer are frequent bedfellows. That fact has been evident for decades. However, medical researchers not wanting to implicate this near-sacred nutrient in the initiation and development of cancer have been reluctant to connect the dots.

Both microscopic and easily visible calcifications are frequently seen in malignant tissues. Such deposits tend to occur at points of inflammation and antioxidant deficiency. However, if the calcium/phosphorus metabolism is sufficiently out of balance, deposition can occur without inflammation. Once calcium begins to accumulate — with or without an initiating point of inflammation — the presence of calcification often initiates or increases inflammation that facilitates further deposition.

These tissue calcifications are commonly seen in cancer patients, and evidence suggests that there is a significant causal link between the two. For example, calcifications in the thyroid gland were found to increase the incidence of malignancy.[19]

Advanced MRI techniques detected calcification in 95% of malignant prostate glands.

Several studies clearly demonstrate the friendly relationship between excess calcium and cancer. In one such study, investigators used advanced magnetic resonance imaging (MRI) techniques to detect calcification in 22 of 23 (95%) malignant prostate glands.[20]

Granted, just because calcium and cancer often occupy the same space doesn't prove calcium's causative role. But we have a smoking gun: a mechanism scientists call "oxidative stress." The following two facts establish an indisputable link between excess calcium and cancer:

1) Oxidative stress is a well-known causal factor of all degenerative diseases, especially cancer.
2) Increased intracellular calcium always increases oxidative stress, even in the absence of detectable calcification.

It also appears that the aggressiveness of some cancers is directly related to the concentration of calcium in the cells. In small cell lung carcinoma — a highly malignant cancer — the cancer cells actually multiply through a chemical process that increases the calcium concentration inside the cells. Several researchers have demonstrated that as the concentration of intracellular calcium

increases, the invasive nature of cancer also increases, resulting in a metastatic spread.[21-24]

Other research shows a strong correlation between bone mass and risk of malignancy. Women with the highest bone mass were found to have an increased risk of developing breast cancer.[25-28] Interestingly none of these authors could satisfactorily explain how higher bone mass could ever be undesirable, and furthermore how it could have anything to do with the development of breast cancer. An understandable and probable mechanism emerges as three other relationships are considered.

1) High bone mass readings do not necessarily mean healthier bone but rather suggest that women with the highest bone mass are those who ingest and supplement the most calcium and therefore are prone to the highest levels of excess calcium.
2) Continued exposure to excess calcium results in calcium deposition in non-bone tissues including the breasts. Women with breast cancer frequently have macro- and microcalcifications on mammography.[29,30] In fact many breast biopsies are performed because of the presence of such calcifications.[31]
3) And finally, calcium deposits in breast tissue allow and promote calcium migration into individual breast cells (increased intracellular calcium) increasing cancer-promoting oxidative stress.

In addition, studies show that breast cancer patients with calcifications are less likely to survive their battle with the disease.[32]

There is however, a cancer/calcium partnership that provides even more solid proof of calcium having a

cancer-promoting activity. Cancer cells seem to need, or at least want to acquire, increased intracellular calcium. Many cancer cells actually develop an increased number of calcium channels. These channels facilitate and promote the flow of calcium into cells, helping to cause a state of increased intracellular calcium.[33] This influx increases oxidative stress and promotes the spread of cancer to other glands and organs.

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Conversely, inhibiting calcium uptake appears to make cancers less invasive and less prone to grow new blood vessels.[34] When calcium is actually pulled out of metastasizing melanoma cells in the brain, the metastatic nature of those cancer cells is reduced. Furthermore, reduction of calcium content in such a cell resulted in a reduced degree of intracellular oxidative stress. The reduction of oxidative stress actually makes these malignant cells more resistant to chemotherapy since the chemo (toxic chemicals) attempts to kill cancerous cells by increasing intracellular oxidative stress to the point of cell death are less effective in accomplishing this goal.[35]

While excess calcium in a cell does not always result in cancer it always produces decreased cellular health through an increase in oxidative stress that can manifest itself in a number of disease states including heart disease and cancer.

Excess Calcium & Toxins Promote Increased Cellular Dysfunction and Death

Poisons are toxic primarily because they start flameless molecular fires that consume whatever they touch. In the process they generate more toxins through a chain

reaction that spreads like a blaze through a forest. Scientists call these pro-oxidant "fires" oxidative stress. Antioxidants are the body's frontline defense against pro-oxidants and the oxidative stress they generate. They are able to extinguish oxidative stress and the initiating toxins — without becoming toxic themselves — as long as an unspent supply remains available.

If calcium levels remain too high a cascade of reactions push cells toward programmed cell death.

The amount of calcium outside cells [extracellular calcium], depending on the type of cell, can be 1,000 to 10,000 times higher than levels inside the cell [intracellular calcium]. This difference in concentrations means that there is always calcium on the outside that wants to get inside. Through various mechanisms, calcium is able to enter the cell. Once in, if calcium levels remain too high a cascade of reactions push cells toward programmed cell death [apoptosis] or complete cell destruction [necrosis].[36]

Clear evidence shows that several known toxins greatly facilitate calcium's passage through cell membranes. Formaldehyde, for example, induces escalated intracellular calcium levels.[37] Exposure to methylmercury, an especially toxic form of mercury, also facilitates calcium's passage into cells, ultimately leading to increased cell death.[38,39] Arsenic induces an influx of calcium into cells.[40] Elevated glucose levels, as seen in diabetes, cause damage on their own, but they appear to instigate cell death by increasing intracellular levels of calcium as well.[41]

As mentioned previously, the increase in intracellular calcium in many cancer cells is fed by an increased number of calcium channels in those cells.[42] Calcium channel blockers are agents that impede calcium's entrance into

cells through these channels, and in so doing they can prevent the manifestation of certain disease states. For example, calcium channel blockers prevent the appearance of neurological disorders in rats exposed to a powerful toxin [methylmercury].[43]

And even in the absence of toxins high intracellular calcium levels still initiate cell injury and death in neurons.[44] Even in the absence of known toxin exposure, chronically elevated calcium levels in the cell appear to be a common denominator for most, if not all, forms of oxidant-induced cell damage and death.[45-48] It is through this very process that sustained elevations of intracellular calcium play a significant part in degenerative neurological diseases such as Lou Gehrig's disease [amyotrophic lateral sclerosis or ALS],[49] Parkinson's disease,[50] and Alzheimer's disease.[51]

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Excess Calcium Increases Death Rate from All Diseases

As already demonstrated, coronary artery calcium scores have been shown to reliably predict cardiac events and increased risk of death from heart attack. These same scores, however, also accurately predict the risk of death from all causes (all-cause mortality).[52,53] As calcium scores rose — starting at undetectable levels of calcium — all-cause mortality also increased.[54]

Other studies have demonstrated the great significance of calcium accumulation as an independent factor that increases all-cause mortality. A recently published study conducted over a period of 8 years found a strong association between increased calcium levels and death

from any cause.[55] Another study demonstrated that individuals with increasing coronary artery calcium scores — in the absence of other traditional coronary artery disease risk factors — had a substantially higher all-cause mortality rate than individuals with three or more significant risk factors but a coronary calcium score of zero.[56]

Coronary artery calcification is also associated with a greater chance of death from all causes.

Even when calcium is not being tracked in the coronary arteries its accumulation still appears to predict all-cause mortality. Calcification around the bases of the aortic and mitral valves is also associated with increased death from all causes. When compared to individuals with no calcification in either valve, calcification around one valve increased all-cause mortality risk. When both valves had calcification the risk of all-cause mortality was higher still. These valve calcifications were found to be independently associated with mortality risk,[57] indicating that the calcification process is strongly related to dying from anything, not just heart disease.

It is very important to note that coronary artery calcification is also associated with a greater chance of death from all causes. That means that even though calcium accumulation may be more easily detected in the coronary arteries than elsewhere in the body, this particular calcification demonstrates the universal role calcium has in accelerating the course of all chronic degenerative diseases. Not only is the coronary calcium score a good indicator of coronary artery disease and plaque burden,[58] it is also a good indicator of the severity of chronic degenerative diseases in general.

Additionally, other studies have found that higher serum parathyroid hormone levels were associated with increased all-cause mortality.[59-64] This is consistent with the fact that parathyroid hormone serves to increase calcium concentration in the blood by a number of mechanisms.

Identification of the milk-alkali syndrome established the toxicity of calcium supplementation long ago.

And finally, in the large study of over 61,000 women mentioned above, it was demonstrated that those who ingested 1,400 mg of calcium/day or more not only were much more likely to die from a vascular event, but they also had an increased mortality from all causes. Alarmingly, those with the highest calcium consumption, whether from dietary and/or supplemental sources, posted a death rate two and one-half times (257%) higher than the groups who ingested less.[65]

The Toxicity of Calcium Supplementation

Identification of the milk-alkali syndrome established the toxicity of calcium supplementation long ago. This condition was first identified in 1923 when peptic ulcer disease was commonly treated with milk and sodium bicarbonate.[66] Although milk and sodium bicarbonate alone were sufficient to induce the syndrome it evolved from a relatively infrequent condition to almost disappearing when proton-pump inhibitors came into widespread use by the mid-1980's for ulcers and effective treatment no longer required an intake of calcium.[67] However, when calcium carbonate, an over-the-counter antacid, largely replaced milk as the predominant source of calcium for individuals trying to self-treat their peptic ulcer diseases the incidence of the milk-alkali syndrome

increased substantially.[68,69] The "current" milk-alkali syndrome, now caused by calcium carbonate and little or no milk, has become the third leading cause of hypercalcemia [excess calcium] of any degree and the second leading cause of striking hypercalcemia in patients with end stage renal disease.[70]

Of particular interest in the current version of the milk-alkali syndrome is that it has been seen with a supplemental amount of roughly 2,000 mg of calcium, along with milk ingestion and some vitamin D supplementation.[71] One woman with the syndrome deliberately supplemented only 1,000 mg of calcium carbonate daily, but was found to be taking additional calcium carbonate antacids along with 800 IU of vitamin D daily — not extraordinary amounts for many individuals.[72] This is especially important since many current recommendations for routine calcium intake/supplementation are as much as 1,500 mg daily in patients.

If the assumption is made that some calcium must be supplemented this provides little margin for error between a recommended amount of calcium and an amount that can potentially result in hypercalcemia, metabolic alkalosis, and renal insufficiency, the classical triad of the milk-alkali syndrome.[73,74] It also strongly supports the assertion that many people are ingesting far too much calcium on a regular basis. Even if they do not push themselves into a classical milk-alkali syndrome it is very clear that individuals can fuel states of calcium excess by pairing a relatively modest dose of vitamin D with "ordinary" calcium supplementation.