Calcium, Aging, and Chronic Degenerative Disease

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Death By Calcium

Calcium, Aging, and Chronic Degenerative Disease

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Calcium:
Background Information

Calcium is prominently featured in the popular press and medical literature primarily because of its still nearly universally-recommended usage in the treatment of osteoporosis, and even for good health in general.

While calcium is essential for normal cellular function, elevations of calcium both inside and outside the cells are part of the ***final common denominator*** for nearly all chronic degenerative diseases, for the development and aggressiveness of any given cancer, and for premature cell death. The evidence also indicates excess calcium ***causes*** many chronic diseases, and is not just incidentally associated with them.

Calcium:
Background Information

This presentation will give the scientific basis for the still little-appreciated toxicity of calcium in virtually all chronic degenerative diseases. Most importantly, it will be shown that although there is clearly a decreased content, or deficiency, of calcium in osteoporotic bone, there is a corresponding ***excess*** of calcium outside of the bone, more so the worse and more long-standing the osteoporosis has been. The degree of deficiency of calcium in the bone is actually the major ***measure*** of the degree of calcium excess throughout the rest of the body.

In other words, it will be shown that the vast majority of the adult population in the developed parts of the world suffer from body-wide excesses of calcium, ***not*** deficiencies, as so many in medicine today continue to assert.

Calcium:
Facts and Fictions

Fiction #1:

"Calcium supplementation prevents fractures."

Many studies involving calcium supplementation that showed decreased fracture incidence also supplemented vitamin D, typically 800 IU daily, as 400 IU of D along with calcium did not consistently produce a benefit. (Jackson et al., 2006 [16481635]; Bischoff-Ferrari et al., 2005 [15886381]).

Adequate vitamin D alone reliably decreases fracture incidence. (Bischoff-Ferrari et al., 2012 [22762317]; Rizzoli et al., 2013 [23320612])

Calcium supplementation alone ***does not*** prevent fractures (Kurabayashi, 2012 [22549199])

Calcium:
Facts and Fictions

Fiction #1:

"Calcium supplementation prevents fractures."

Calcium supplementation can ***minimally improve*** the results of the bone mineral density tests. However, this is a cosmetic change only, as the increased calcium content from such supplementation is really no different from a fresh coat of paint on a fence with rotting wood. It looks better, but it does not restore any structural integrity to the fence, and the fence will continue to deteriorate. The healthy structural matrix of the bone requires vastly more than calcium for repair and regeneration. While the test can show a better "number," it is not reflective of better bone.

Calcium:
Facts and Fictions

Fiction #2:

"Calcium supplementation and increased dietary calcium are good for you."

This is a case of marketing completely trumping science. The truth: a prospective study over a 19-year period followed 61,433 women. Women with the highest intake of calcium and who supplemented calcium as well had an ***all-cause mortality*** risk 2.5 times higher than those women who did not supplement calcium. (Michaelsson et al., 2013 [23403980])

Calcium:
Facts and Fictions

Fiction #3:

"You cannot get enough calcium in your diet without dairy products."

More marketing. Enjoy your dairy if you wish, but do not delude yourself into thinking that anything beyond a minimal amount of it in your diet is good for you. Body-wide states of calcium deficiency are very strongly entrenched myths, nothing more. As is too often the case with a number of strongly-promoted scientific/medical assertions and beliefs, ***the exact opposite is true***.

Calcium:
Facts and Fictions

Fiction #4:

"Everyone with osteoporosis has a calcium deficiency."

In the bones, yes; in the body, no. Fully 99% of the body's normal content of calcium is in the bone, and as osteopenia and osteoporosis relentlessly break down the integrity of the bone, a large amount of this calcium ends up where only ***0.1%*** of it should be: in the rest of the body.

Calcium:
Facts and Fictions

Fiction #5:

"The biggest danger faced by the osteoporosis patient is disability or death following a fracture."

Another completely wrong but commonly held view.

In 10,000 postmenopausal women with low bone mineral density, a 60% increased chance of death was seen in the lowest quintile of BMD compared to the highest quintile (aka, heaviest supplementers). However, most of the deaths did not relate to a fracture. (Browner et al., 1991 [1677708]

Significance: while an osteoporotic fracture can be deadly, the consequences of its commonly accepted treatments are ***deadlier***.

Calcium, Vitamin C, and Oxidative Stress

It is the biochemistry of vitamin C and redox chemistry that is the major key in understanding both the physiology of health and the pathophysiology of disease in man and animals.

The effects of all toxins and all infections are pro-oxidant in nature, and it is their ability to deplete (oxidize) vital biomolecules that allow them to cause and promote chronic diseases.

It is the ability of vitamin C as the body's premier antioxidant to replete (reduce) both pro-oxidant toxins directly or to reduce back to normal status the biomolecules they have already oxidized.

Osteoporosis is a Focal Scurvy of the Bones

Bone physiology involves three important interactive factors:

1. Formation (minerals, collagen, proteins, calcium)
2. Destruction (the dissolution and release of minerals and calcium, which degradation of collagen and proteins, along with less cross-linking of these structural molecules
3. Ongoing oxidative stress levels, with low levels favoring bone formation and high levels favoring bone destruction

Osteoporosis is a Focal Scurvy of the Bones

1. Increased oxidative stress (aka increased inflammation) in the bones, as tracked by higher levels of C-reactive protein, reliably predicts increased chances of osteoporotic fracture in elderly women (Nakamura et al., 2011 [20936400])
2. Increased levels of many other inflammatory parameters relate to increased fracture risk (Lacativa and Farias, 2010 [20485900])
3. C-reactive protein levels and multiple other inflammatory parameters are significantly reduced by high doses of vitamin C (Mikirova et al., 2012 [22963460])

Osteoporosis is a Focal Scurvy of the Bones

4. Osteoblasts, the bone-forming cells, are stimulated to develop from precursor cells by vitamin C (Carinci et al., 2005 [15777530])
5. The precursor cells from which osteoblasts develop are stimulated to proliferate by vitamin C (Choi et al., 2008 [18640597])
6. Vitamin C is essential for the type III collagen required for the accelerated growth of osteoblasts (Maehata et al., 2007 [17306970])
7. Osteoclasts, the bone-destroying cells, are inhibited by vitamin C (Gabbay et al., 2010 [20410296])

Osteoporosis is a Focal Scurvy of the Bones

8. A vitamin C deficiency results in osteoclast proliferation, with resulting increased bone resorption (Hie and Tsukamoto, 2011 [20444587])
9. Net bone loss, with demineralization and loss of integrated calcium, is accelerated when vitamin C levels get sufficiently depleted (Park et al., 2012 [22974214])
10. The cross-linking of bone collagen (comprising 90% of the bone matrix protein) required for structural strength, is strongly supported by vitamin C (Munday et al., 2005 [15946412])

Osteoporosis is a Focal Scurvy of the Bones

11. Supplemental vitamin C alone lowers the risk of osteoporotic fractures in elderly individuals (Leveille et al., 1997 [9425455])
12. Dietary vitamin C intake (always vastly lower in amount than achieved with any significant supplementation) had no association with fracture risk (Sahni et al., 2009 [19347239])
13. Elderly patients who already sustained an osteoporotic fracture had significantly lower vitamin C blood levels than those who were fracture-free (Martinez-Ramirez et al., 2007 [18622945])

Osteoporosis is a Focal Scurvy of the Bones

14. Vitamin C supplementation, without calcium supplementation, resulted in significantly greater bone mineral density at all bone sites tested (Morton et al., 2001 [11149477])
15. In mice with ovaries removed, vitamin C prevented the bone loss otherwise seen (Zhu et al., 2012 [23056580])
16. Vitamin C substantially accelerates the healing of experimental fractures (Yilmaz et al., 2001 [11510911])
17. Vitamin C significantly improves the strength of healed fractures (Alcantara-Martos et al., 2007 [17356161])

Calcium
and Oxidative Stress

Calcium, which is concentrated up to 10,000 times more outside the cells of the body than inside, needs to be maintained within a fairly narrow range of normal inside the cells to permit optimal cellular function. Any intracellular calcium concentrations above this normal range are always accompanied by ***increased intracellular oxidative stress***, and are probably the primary cause of this state. The very large gradient of calcium from extracellular to intracellular requires a significant consumption of energy in order to be properly maintained, and any significant compromise in energy availability invariably leads to progressively increasing and toxic intracellular calcium concentrations.

Calcium and Oxidative Stress

The bones contain up to 99% of the calcium present in the body. The calcium in solution in the blood, the extracellular fluids, and the cytoplasm inside the cells is only approximately 0.1% of the total calcium content in the body. As osteoporosis and the osteopenia of aging evolve over time, an enormous amount of calcium floods the extracellular fluids, and calcium deposition invariably results. The intracellular calcium levels also rise sufficiently to substantially increase intracellular oxidative stress, promoting all chronic degenerative diseases, while also being a required factor for malignant transformation.

Calcium, Intracellular Oxidative Stress, and Malignancy

Levels of Intracellular Oxidative Stress

1. None, or not readily detectable (as in dormant and non-replicating cells)
2. Minimal (baseline physiological level of oxidative stress, as in viable but non-replicating cells of less metabolically active organs)
3. Minimal to moderate (physiological oxidative stress in viable but non-replicating cells of organs with a high level of physiological activity, like heart) functions (Santos et al., 2011 [21236334]; Bogeski et al., 2011 [21930299]
4. Moderate (can be normal or abnormal, depending on what the cell is doing; when normal, the moderate state is usually transient; if calcium and iron levels are elevated, moderate almost always represents the arrival or imminent arrival of the malignant state)

Calcium, Intracellular Oxidative Stress, and Malignancy

Levels of Intracellular Oxidative Stress

5. Moderate to elevated (characteristic of established and replicating cancer cells; never seen in normal cells unless transient, as in proceeding to programmed cell death) Parri and Chiarugi, 2013 [23146119] Many cancer cells have increased expression of calcium channels (Embi et al., 2012 [23216811])
6. Elevated (most metabolically active of cancer cells, anaplastic or metastatic) Shen et al., 2013 [23373752]
7. Greatly elevated (in cancer cells with upregulated Fenton activity, as provoked by chemotherapy)
8. Maximal (very transient, proceeding promptly to apoptosis or frank necrosis)

Regulation of Intracellular Calcium Levels

1. Changes in cell permeability
2. The buffering and/or binding of calcium inside the cells (Yanez et al., 2012 [22453954])
3. The sequestration or compartmentalization of calcium in intracellular organelles (Galva et al., 2012 [23077175])
4. Regulation of energy-requiring active calcium extrusion from the cell [calcium pump] (Kurnellas et al., 2007 [17956247]))
5. Modulation of calcium channels (membrane proteins); of greatest importance in the control of intracellular calcium concentration (use of CCBs, natural and drug)

Calcium, Intracellular Oxidative Stress, and Disease

It is the highest elevations of intracellular calcium that ultimately result in cell death (Garcia-Prieto et al., 2013 [23250754]; Schwartz et al., 2013 [23220009])

Toxins routinely increase intracellular levels of calcium, with resulting increased intracellular oxidative stress, and ultimately cell death (Chi et al., 2012 [23160928]; Roos et al., 2012 [22927718]; Li et al., 2012 23049237])

Elevated intracellular calcium levels have been consistently seen in ALS, Parkinson's, and Alzheimer's (Kawamata and Manfredi, 2010 [20493207]; Surmeier et al., 2011 [21884755]; Corona et al., 2011 [21697951])

Calcium and Calcium Channel Blockers

Calcium channels refer to proteins in the cell membrane that facilitate selective permeability to calcium ions.

Calcium channel blockers (CCBs), or calcium antagonists, comprise a class of drugs that prevents the ability of calcium to utilize these calcium channels and gain entry into the cells of the body.

Although widely used for different reasons today, CCBs were developed as antihypertensive agents, as they have vasodilating effects.

The only significant side effect of CCBs is that of excessive calcium-blocking effect, which translates to impaired vascular contraction and hypotension.

Calcium and Calcium Channel Blockers

For CCBs to work as antihypertensives and to have their many other positive clinical effects, the direct implication is that a state of calcium excess inside the cells of the body must exist in the first place. While the different CCBs can have additional effects, their most profound effect remains blocking calcium entry into the cells. Therefore, a logical conclusion of the effects of CCBs throughout the body is that:

The positive effects of CCBs in any disease process occur because the diseased cells have increased intracellular levels of calcium.

Calcium and Calcium Channel Blockers

In addition to high blood pressure, many diseases are known to benefit from CCB administration:

1. Coronary artery spasm (Kusama et al., 2011 [21389642]); angina pectoris (Siama et al., 2013 [23016717])
2. Anti-atherosclerosis (Ishii et al., 2012 [22653165])
3. Pulmonary hypertension (Montani et al., 2010 [20543192])
4. Raynaud's phenomenon (Huisstede et al., 2011 [21704799]
5. Acute head trauma (Aslan et al., 2012 [22854593])

Calcium and Calcium Channel Blockers

Diseases known to benefit from CCB administration:

6. Epilepsy (Ianneti et al., 2009 [19303743])
7. Chemotherapy-induced peripheral neuropathy (Tatsushima et al., 2013 [23206755])
8. Alzheimer's disease (Anekonda and Quinn, 2011 [21925266])
9. Parkinson's disease (Pasternak et al., 2012 [22387374])
10. Osteoporosis (rat study, Shimizu et al., 2012 [21881574])

Calcium, Intracellular Oxidative Stress, and Disease

Further support for the concept that increased intracellular calcium leads to intracellular oxidative stress (toxicity):

1. Calcium channel blockers prevent the neurological damage from methylmercury in rats (Sakamoto et al., 1996 [8882354])
2. Calcium channel blockers decrease the chances of death from all causes (Gillman et al., 1999 [10323641]; Gibson et al., 2000 [10922432]; Lubsen et al., 2005 [15716708]; Costanzo et al., 2009 [19451836])
3. Calcium channel blocker use is inversely related to prostate cancer incidence (Poch et al., 2012 [23280547])
4. Calcium channel blockers can decrease cytoplasmic iron accumulation, an important factor in malignant transformation and the further elevation of intracellular oxidative stress (Chattipakorn et al., 2011 [21860702])

Chronic Calcium Excess: The Rule, Not the Exception

Five major causes of bodywide calcium excess:

1. Excess calcium ***intake*** (supplementation and/or diet)
2. Excess calcium ***mobilization*** from the bones
3. ***Deficient vitamin C and antioxidant stores*** in the bones and elsewhere
4. Sites of ***chronic infection and inflammation*** in the body (especially root canals and tonsils)
5. Chronic ***deficiencies of important regulatory hormones*** (testosterone, estrogen, thyroid)

Chronic Calcium Excess: The Rule, Not the Exception

Extracellular Calcium Excess

One third of American over the age of ***45*** have arterial calcification when assessed by computed tomography (Guzman, 2007 [17544025]. Anytime overt calcification is seen outside of the bones, a state of excess calcification exists by definition. Logically, a significant degree of calcium excess will be present well before calcium deposition finally occurs.

Chronic Calcium Excess

Extracellular Calcium Excess

Ectopic calcifications very commonly seen in patients with cancer.

Using advanced magnetic resonance imaging, 22 of 23 prostate cancer patients were found to have prostatic calcifications (Bai et al., 2013 [23308170])

Women with the highest scores on bone density testing had an increased risk of developing breast cancer (Zhang et al., 1997 [9032046]) Mammography on women with breast cancer often have macro- and microcalcifications (Holmberg et al., 2013 [23370209])

Chronic Calcium Excess

Extracellular Calcium Excess

Coronary artery calcium (CAC) scores reliably predict not only death from heart disease, but also death from all causes (Jacobs et al., 2012 [22357989]; Kiramijyan et al., 2013 [23206921]) This indicates that the main indicator of extracellular excess, ectopic calcium deposition, reliably predicts increased risk of death from any chronic degenerative disease. This further indicates that a presence of a chronic extracellular calcium excess will reliably indicate secondary chronic intracellular calcium excesses.

Chronic Calcium Excess

Intracellular Calcium Excess and Cancer

Well-established relation to the malignant state. Also, even further increases in intracellular calcium increase the degree of cancer cell proliferative and invasiveness/metastatic capacity (Gudermann and Roelle, 2006 [17158754]; Kaufmann and Hollenberg, 2012 [22453980]; Ryu et al., 2013 [23328481)

Conversely, the ***removal*** of calcium from the intracellular space ***decreases*** metastatic capacity (Lin et al., 2010 [20824051])

Osteoporosis Reversal Agents

1. Magnesium
   - A. A natural calcium channel blocker (Fawcett et al., 1999 [10618948]); magnesium and calcium are biological antagonists (Anghileri, 2009 [20228002])
   - B. Dissolves calcium deposits (Steidl and Ditmar, 1990 [2133625])
   - C. Mg deficiency increases intracellular calcium (Fox et al., 2001 [11811859])
   - D. Increases bone density and decreases fracture incidence (Ryder et al., 2005 [16274367])
   - E. Decreases all-cause mortality (Woods and Fletcher, 1994 [7908076]; Shechter et al., 2003 [12845247])
   - F. Non-toxic at supplemented doses

Osteoporosis Reversal Agents

2. Vitamin K

A. Activator of proteins known to inhibit ectopic calcification, like osteocalcin and matrix Gla protein (MGP) (Theuwissen et al., 2012 [22516724])

B. Helps dissolve existing calcifications (Schurgers et al., 2007 [17138823])

C. Neutralizes warfarin (an agent that causes ectopic calcification) (Price et al., 1998 [9743228])

D. Decreases risk of fracture (Shiraki et al., 2000 [10750566]) and improves bone quality (Saito, 2009 [19949271])

E. Decreases cardiac and all-cause mortality (Geleijnse et al., 2004 [15514282])

F. No definable toxicity at any dose level (Pucaj et al., 2011 [21781006])

Osteoporosis
Reversal Agents

3. Vitamin C

A. Osteoporosis is bone scurvy, with a generalized deficiency of antioxidants as well. The other agents discussed compensate for this scurvy to the degree that some antioxidant capacity is restored to the bone. However, the rebuilding of new bone requires adequate vitamin C

B. Decreases bone resorption and increases bone synthesis; supplemental vitamin C results in greater bone density; accelerates fracture healing and is essential for normal bone integrity

C. Highest VC levels, least fractures.

D. Decreases all-cause mortality.

E. Virtually non-toxic at any dose; no definable toxic level ever defined

Osteoporosis
Reversal Agents

4. Vitamin D

A. Normal vitamin D blood levels with most dietary regimens assures adequate calcium intake

B. Critical for far more than bone and calcium metabolism, regulating up to 2000 genes (Wacker and Holick, 2013 [23306192])

C. Deficiency causes osteoporosis (Bolland et al., 2010 [19906799])

D. Excess worsens osteoporosis (Masterjohn, 2007 [17145139])

E. A key determinant of bone mineral density while growing up (Pekkinen et al., 2012 [22768331])

F. In therapeutic range, decreases all-cause mortality (Semba et al., 2010 [19953106]; Schottker et al., 2013 [23446902])

Osteoporosis
Reversal Agents

5. Essential fatty acids

A. Some with calcium channel blocking activity (Ye et al., 2010 [20206488]; Pages et al., 2011 [21664114])

B. Highest levels afford protection against loss of bone mineral density (Farina et al., 2012 [22392875]; Moon et al., 2012 [22507833])

C. Blood levels inversely related to total mortality (Pottala et al., 2010 [20551373])

D. No clear toxic effects; some GI intolerance at high intakes

Osteoporosis
Reversal Agents

6. Estrogen

A. Decreases ectopic calcification in the coronary arteries (Weinberg et al., 2012 [22747181]; Higher blood levels, lower CAC scores (Jeon et al., 2010 [20512078])

B. Inhibits a protein that promotes calcification (Osako et al., 2010 [20595654])

C. Deficiency increases proinflammatory cytokines (Das, 2002 [11815671])

D. Lessens incidence of osteoporotic fractures (de Villiers and Stevenson, 2012 [22612613])

E. Deficiency increases all-cause mortality (Mansur et al., 2012 [22701354]) and promotes metabolic syndrome (Mauvais-Jarvis et al., 2013 [23460719])

Osteoporosis Reversal Agents

7. Testosterone

A. Deficiency a clear risk fracture for fractures (Torremade-Barrera et al., 2013 [23246104])

B. Has calcium channel blocking effects (Oloyo et al., 2011 [21439799])

C. Prostate cancer frequently associated with low testosterone levels (Mearini et al., 2013 [22068548])

D. Testosterone levels inversely related to CAC (Park et al., 2012 [22522505])

E. Deficiency increases all-cause mortality (Fukai et al., 2011 [21143567]; Grossman et al., 2012 [22280063])

Osteoporosis
Reversal Agents

7. Testosterone

Factors in restoration of deficiency (similar to restoration of estrogen deficiency)

Dose, type, formulations, routes, duration, timing, accompanying antioxidants, serial clinical correlation and laboratory testing, target levels

Also, PSA monitoring:

Testosterone promotes preexisting prostatic cancer cell growth, but does not induce malignant transformation in the prostate. Testosterone therapy actually offers the benefit of exposing earlier the presence of preexisting prostate cancer with follow-up PSA testing.

Osteoporosis Reversal Agents

8. Thyroid hormone

A. Substantial effects of the metabolism of all the cells in the body (Boelaert and Franklin, 2005 [16214936])

B. Necessary for early skeletal development and the establishment of peak bone mass (Williams, 2009 [19885809])

C. High and low thyroid function increases fracture risk (Wojcicka et al., 2013 [22634735])

D. TSH has a direct bone-protecting effect, not involving the thyroid gland (Ma et al., 2011 [21745106]; Sun et al., 2013 [23716650])

Osteoporosis
Reversal Agents

8. Thyroid hormone

E. High and low thyroid hormone levels are independent risk factors for death from all causes. This includes subclinical hypothyroidism (T4 normal, TSH elevated) and subclinical hyperthyroidism (Tseng et al., 2012 [22726629]; Ceresini et al., 2013 [23647402])

F. Thyroid status must be a routine part of all general medical evaluations, and needs to be rechecked every few years in the older population.

Reversing Osteoporosis and Chronic Degenerative Disease

Primary goals:

1. Minimize new toxin exposure
2. Eradicate acute and chronic infections
3. Eliminate accumulated toxins
4. Normalize or improve deficient regulatory hormones (estrogen, testosterone, thyroid)
5. Optimize antioxidant and nutrient levels, especially vitamin C
6. Selectively and appropriately utilize prescription medications

Reversing Osteoporosis and Chronic Degenerative Disease

1. Minimize new toxins

   A. No calcium, iron, or copper supplementation

   B. Address dental toxins/infections

      a. Root canals

      b. Gums

      c. Cavitations

      d. Dental implants

      e. Toxic dental materials (mercury, nickel)

Reversing Osteoporosis and Chronic Degenerative Disease

1. Minimize new toxins

C. Dietary/digestive (optimize bowel transit time)

Food combining, chewing, liquids, enzymes, low glycemic, minimize meat quantities, minimize dairy (less calcium and poorer digestion)

Poor foods digested perfectly produce less toxicity than optimal foods digested poorly.

Reversing Osteoporosis and Chronic Degenerative Disease

2. Eradicate infections

Most infections significantly lowering antioxidant stores through the body will be found in dental sources. However, for many individuals, chronically infected tonsils will significantly impair or even block the recovery from many different chronic diseases. Usually tonsils that need extracting will be on the same side of the body as an existing or even previously extracted root canal-treated tooth. Generally, once a tonsil has drained a root canal for any significant length of time, it cannot recover even if the root canal is extracted. Rather, it becomes a source of chronic focal infection, seeding pathogens and toxins throughout the body.

Reversing Osteoporosis and Chronic Degenerative Disease

3. Eliminate old toxins

Remember that ***detoxification*** is ***retoxification***.

A. Traditional agents (DMSA, DMPS, BAL, EDTA, penicillamine, deferasirox)

B. Nutrient agents (alpha lipoic acid, inositol hexaphosphate, any agent supporting and increasing intracellular GSH levels, such as NAC, whey protein, and liposome encapsulated GSH)

C. Sweating (far infrared sauna, aerobic exercise)

D. Always protect with adequate antioxidant coverage

Reversing Osteoporosis and Chronic Degenerative Disease

4. Correct critical hormone deficiencies

Testosterone, estrogen, and thyroid deficiencies affect all the cells of the body. All three play prominent roles in calcium metabolism, in the bone and elsewhere

Replacement must be low dose, slow to increase dose, and target lab should be low to mid-range normal, no more, and even a lower target for the person over age 70. Other critical laboratory tests, such as metabolic syndrome parameters, should be stable or improving during the treatment period.

Reversing Osteoporosis and Chronic Degenerative Disease

5. Optimize antioxidant levels

A. Vitamin C, regular form as tolerated; liposome-encapsulated, 1 to 2 grams daily; ascorbyl palmitate, 500 to 1,000 mg daily

B. Lysine, 1,000 to 2,500 mg daily and proline, 250 to 500 mg daily (2x more for known CAD)

C. Vitamin D3: aim for blood level of 50 to 80 ng/cc

D. Vitamin K2, 3 to 6 mg daily

E. Magnesium glycinate, 200 to 400 mg daily

F. Omega-3 EFA, fish oil source, 2,000 to 4,000 mg daily

G. Vitamin E as mixed tocopherols, 400 IU twice daily

H. Beta carotene, 25,000 to 50,000 IU daily

I. Vitamin B complex, one daily

Reversing Osteoporosis and Chronic Degenerative Disease

6. Appropriate prescription medications

Only really to be utilized when a satisfactory clinical response is not seen with the rest of the protocol. The side effects of selective estrogen receptor modulators (SERMs), bisphosphonates, calcitonin, strontium ranelate, and parathyroid hormone are substantial. This needs to be considered in the context that vitamin C, vitamin D, vitamin K, magnesium, EFAs, and properly adjusted hormone levels all decrease the chances of death from all causes. The prescription meds have side effects and only rarely positively impact longetivity.

Reversing Osteoporosis and Chronic Degenerative Disease

6. Appropriate prescription medicines

Whenever clinical circumstances allow, consider using a calcium channel blocker, which also decreases all-cause mortality.

While not a recognized medical indication, it might be wise to consider calcium channel blocker therapy in any older patients for the known positive effects of this drug class on basic disease pathology and longevity.

Tracking Toxicity

Many different treatment protocols exist for many different conditions, both in mainstream medicine and in complementary or alternative medicine.

Many different protocols can produce a ***net*** positive result, but still fall far short of an ***optimally*** positive result.

Tracking Toxicity

Parameters of calcium accumulation, along with routine laboratory testing and serial clinical evaluation, offer the optimal way to know that the patient is being treated optimally.

Infrequently, and mostly in older patients who have long-standing pathology and a large amount of secondary tissue and organ damage, the optimal protocol can only achieve a slowing of the ongoing disease pathology and a slowing of calcium accumulation. Taking the measures needed to mobilize calcium is much more effective at slowing aging to physiological levels, not reversing it. However, in younger individuals, some reversal of the parameters of aging is clearly achievable.

Calcium Toxicity: Recap

1. ***All*** chronic degenerative diseases feature increased extracellular ***and*** intracellular levels of calcium. Calcium deposition often results in the extracellular space, where calcium concentrations are vastly higher than inside the cells. The increased extracellular calcium always leads to increased intracellular calcium, which always results in increased intracellular oxidative stress.

Calcium Toxicity: Recap

2. While a calcium deficiency is always present in the bones of individuals with chronic disease and is especially severe in those with osteoporosis, it is precisely the chronic loss of calcium from its large bony reservoir that continually feeds the excess presence of calcium elsewhere in the body. Calcium supplementation and excess dairy intake only further fuel this excess extraskeletal calcium presence.

Calcium Toxicity: Recap

3. While vitamin D is essential for good health, it is essential that supplementation is monitored by blood testing, as both vitamin D deficiency and vitamin D excess promote osteoporosis and the redistribution of calcium from the bones to the rest of the body.

Calcium Toxicity: Recap

4. While many different factors play a role in calcium intake, the most important factors are:

    A. Calcium content in the diet (especially dairy, as non-dairy sources of calcium do not significantly factor into causing a state of excess calcium)

    B. Calcium supplementation

    C. Vitamin D status

Calcium Toxicity: Recap

5. Death from ***all*** causes is increased by increased calcium intake, whether from calcium supplementation, or an increased dietary calcium intake, or both.

For Contact and Further Information

For questions or comments:

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I cannot offer personal consultations, but I will take any questions regarding my vitamin C-centered protocols that an inquiring caregiver might wish to ask me by email.