The Importance of Bioactive Silicates in Human Health

by Guy E. Abraham, MD

Introduction

The element silicon does not exist in nature in its pure form but rather is always combined with oxygen. Next to oxygen, silicon is the most abundant element in the earth's crust. Silicon dioxide or silica, SiO_2, is formed by covalent binding of two atoms of oxygen to one atom of silicon. Free silica is found both in the amorphous state such as opal and in the crystalline state such as quartz. Biological systems are capable of forming amorphous microparticles of hydrated silica, but they cannot form the crystalline state of silica. Under proper conditions, one molecule of silica will react covalently with one molecule of water to form monometasilicic acid, H_2SiO_3; combined with two molecules of water, it becomes monoorthosilicic acid, H_4SiO_4. In both plants and animals, monoorthosilicic acid is the biologically active form of silicon and silica. Up to saturation at 100 parts per million (100 PPM), aqueous solution of monoorthosilicic acid exists in its free form, below a pH of 9. Above saturation, monoorthosilicic acid loses one molecule of water to form monometasilicic acid which polymerizes and precipitates out of solution to form opal. Plant tissues contain this form of opal, with extremely small particle sizes. The loss of two molecules of water from silicic acid results in the formation of silicic anhydride, more commonly known as silica or silicon dioxide, SiO_2. When two molecules of monoorthosilicic acid combine by condensation (loss of on molecule of water), they generate disilicic acid. When three molecules of monoorthosilicic acid combine through condensation, with the loss of four molecules of water, trisilicic acid is formed:

$$2\ H_4SiO_4 = H_2O + H_6Si_2O_7$$

$$3\ H_4SiO_4 = 4\ H_2O + H_4Si_3O_8$$

The natural silicates found in rocks correspond to these silicic acids, combined with transition metal oxides and as salts of alkali metals (silica-mineral complexes). Sixty percent of all rocks are made of silica, either in its pure form or as silica-mineral complexes. Erosion of those rocks releases into the soil, streams, and ground water all these forms of silica and silicates. Silica is often the principal solute in natural fresh water, where it occurs entirely as monoorthosilicic acid. The reported concentrations of monoorthosilicic acid, expressed as silicon, ranged from 0.8-5.0 PPM in streams and 3.5-28.0 PPM in ground water compared to 1.0-40.0 PPM in soil solution.[1-4] In an aqueous environment, hydrated silica releases silicic acid in solution by hydrolysis. The amount of silicic acid released is dependent on the surface/volume ratio of the silica particles. The smaller the particle sizes, the greater the surface/volume ratio and the greater the release of silicic acid.[5] Increasing the zeta potential (electrical charge) of the aqueous suspension of silica by the presence of alkali metal salts of polyacidic organic compounds enhances the hydrolysis and release of silicic acid. Humic acid in the moist soil enhances the release of free silicic acid from the soil silica-mineral complexes. Coating of the hydrated silica particles with organic compounds stabilizes the silica particles and inhibits the release of silicic acid. These two opposing factors control hydrolysis and release of free silicic acid and play an important role in the availability of silicic acid in the moist soil.[1-5]

The Silica Cycle

The silica cycle begins with the uptake of bioactive silica, that is monoorthosilicic acid, by roots of plants. Below a pH of 9, monoorthosilicic acid, hereafter called silicic acid, exists in its free form in water up to saturation (100 PPM). After translocation, condensation, and precipitation in plant tissues, silicic acid performs several important functions.[6]

Structural: Silica contributes compression-resistance and rigidity to the cell walls which aids in photosynthesis by improving lights interception. It also renders the plant drought-resistant.

Physiological: The presence of silica reduces evaporation and transpiration, therefore conserving tissue water. It also promotes oxygen availability via the roots through increased rigidity of the air canals.

Protective: Silica increases the resistance to pathogens, insects, and mollucs. It also protects the plants from toxicity of excess metals, such as manganese and iron, by distributing these oxides evenly in plants tissues, increasing their solubility. Silicic acid also forms a silicate coating around microparticles of the oxides of these metals, increasing their stability, preventing aggregation and precipitation. These microscopic amorphous particles of silica, and the other silica-containing plant tissues, return

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to the soil for recycling after the death and decay of plants.

The next step in the silica cycle involves the assimilation of plant silica by herbivorous animals. Although plants and animals can combine silicic acid into organic substances, it is most likely via Si-O-C silanolate bonding through condensation of a silanol group with a hydroxyl group of an organic compound as proposed by Schwartz.[7] The Si-C bond, that is true organic silicon, does not exist in the earth crust, only man-made.

Plants in the early stage of growth contain a small fraction of silica in the form of silicic acid. The proportion of silicic acid decreases with the increasing age of the plant and approaches zero in mature plants, probably due to coating of the silica particles with organic polyals and polyphenols. Drying of plant tissues causes condensation of silicic acid, so that silicic acid concentrations in dried plants of any age are reduced markedly. Therefore, a large proportion of the ingested silica from feed (greater than 95%) by herbivorous animals is not bioavailable.[8] Silicic acid in water is much more bioavailable, and is either absorbed rapidly after a short transit time in blood, excreted in the urine, or diffused passively in extracellular fluid compartments. Plant tissues contain much larger amounts of silica than animal tissues. Plants assimilate silica from the soil more efficiently than animals absorb and retain silica from plants. As a result, herbivorous animals excrete in their urine 10 -30 times more silica than carnivorous animals.[1]

Essentiality of Bioactive Silicates in Higher Animals

The essentiality of the element silicon in the form of bioactive silicates in higher animals was demonstrated 30 years ago by the research performed on chicks by E.M. Carlisle[9] and on rats by K. Schwartz[10] from the University of California at Los Angeles. These researchers used sodium monomethasilicate (Na_2SiO_3) to supplement the diet of chicks and rats made deficient in silicon by a low silicon diet. The addition of one molecule of water to metasilicic acid generates silicic acid in the intestinal tract. Silicon, in the form of bioactive silicates, was found to be essential for normal growth, bone formation and production of connective tissue in chicks and rats. In young growing bones, the concentrations of silicon were in the same ranges as the concentrations of calcium, magnesium, and phosphorus. As the bones progress to maturity, silicon is progressively replaced with calcium, suggesting that silicon plays a role in the formation of bone matrix prior to calcium deposition.[11]

Carlisle investigated further the mechanism of action of silicon in growth and health. The highest silicon concentrations generally occur in connective tissues, and organs with the highest amounts of connective tissue have the highest concentrations of silicon. When several tissues from rats were analyzed for silicon content, the aorta, trachea, and tendon were 4-5 times richer in silicon than liver, heart, and muscle.[12] The skin also was found to contain high levels of silicon. Aging causes a decrease in silicon concentrations only in the aorta and the skin. In 12-week-old rabbits, the mean concentrations of silicon were respectively in the aorta and the skin 80 µg Si/gm tissue (dry weight) and 45 µg Si/gm tissue. At 18-24 months of age, the levels dropped to 15 and 9 µg/gm respectively. The skin of fetal pigs averaged 95 µg Si/gm dry weight, compared to 10 µg/gm in mature pigs. No change was observed with aging in silicon concentrations of the heart, liver, and muscle.

Further research by Carlisle revealed that the high silicon content of connective tissue arises mainly from its presence as an integral component of the glycosaminoglycans (mucopolysaccharides) and their protein complexes. Extraction and fractionation of connective tissue showed the glycosaminoglycan-protein complexes to be high in silicon. Through further purification, the silicon was recovered in glycosaminoglycan fraction. Enzymatic hydrolysis of glycosaminoglycans did not release free silicic acid but rather yielded products of lower molecular weight, still containing silicon in bound form. Even disaccharides of chondroitin sulfate A contained significant amounts of silicon in bound form.[10] Silicon also increases the hexosamine content of articular cartilage.

Collagen, which is present in connective tissue, is the most abundant of all proteins in higher vertebrates, accounting for up to one-third or more of the total body protein. Carlisle demonstrated that silica in its bioactive forms, plays an important role in collagen synthesis.[13,14] Collagen contains 12% of the aminoacid proline and 9% of 4-hydroxyproline. Silica increases synthesis of proline from glutamic acid. In the presence of iron and vitamin C, silica enhances markedly hydroxylation of proline residues of procollagen to form collagen. Silica also promoted the formation of extracellular matrix (ground substance)[15] with a concomitant increase in interstitial fluid, due to hydration of the extracellular substances.

Metabolism of Silicon in Man

In most studies of bioactive silicates performed on man, the values reported are expressed as concentrations of the element silicon (Si). The compounds measured, however, were obviously not pure Si but derivatives of

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silica and silicic acid. To convert values expressed as Si to values expressed as silica, multiply by 2.14. To convert values from Si to silicic acid, multiply by 3.4. When values reported in publications are expressed in molar concentrations, these conversion factors do not apply.

Balance Studies: To this point, the only silicon balance study on humans that has been published was performed at the US Department of Agriculture in Beltsville, Maryland.[16] Eleven male subjects, ranging from 37 to 58 years of age were evaluated over two periods of 26 days. The subjects alternated between two different diets: a low-fiber diet consisting of juice from fruits and vegetables and a high-fiber diet consisting of fruits and vegetables. During the last seven days, balance studies were performed daily, and the mean values reported. The low -fiber diet contained 4.6 gm of fiber and 21 mg Si/day; the high-fiber diet contained 23.8 gm of fiber and 45.8 mg Si/day. The mean ±SEM of the daily silicon levels in urine and fecal excretions were 12.2±1.1 mg Si (urine) and 12.3±0.9 mg Si (fecal excretion) for the low-fiber diet. For the high fiber diet, those values were 16±2.6 mg Si and 44.4±2.6 mg Si. The mean percentage of Si intake recovered in fecal excretions were 59.0±5.3% for the low-fiber diet and 96.5±4.3% for the high-fiber diet. With both diets, a negative silicon balance was obtained, with mean daily losses of 3.5±1.6 mg and 14.6±3.5 mg Si for the low- and high-fiber diets, respectively.

When balance studies are performed on essential trace elements, a negative balance means that, under the conditions of the experiment, more nutrients would be required to achieve balance. Surprisingly, the high-fiber diet, with intake of Si twice as high as the low-fiber diet, resulted in a daily loss of Si four times higher than the low-fiber diet. The percentage of Si in the diet recovered in fecal excretions on the high fiber diet (96.5±4.3%) is similar to those observed in farm animals.[8] Since the bioactive and bioavailable form of silicon is silicic acid, the above results could be explained by the greater solubility of silicic acid in the liquid diet of the low fiber regimen. It is likely that food fiber does not contribute significantly to the bioavailable pool of silicic acid in the intestinal tract and may even interfere with the absorption of free silicic acid.

Silicon Levels in Biological Fluids: Over a wide range of intakes, serum silicon levels are maintained within a narrow range because of a very efficient renal clearance system. Renal clearance of silicon is similar to glomerular filtration rate (GFR) with the implication that silicon as silicic acid is passively cleared by the kidney and is dependant on the GFR. In patients with decreased GFR, serum silicic acid increases in proportion with the degree of renal impairment.

Silicon exists in serum exclusively as silicic acid and is diffused passively into the extravascular extracellular fluid compartments where silicon concentrations are maintained in the same range as those in serum.[17] In herbivorous animals like sheep and cattle, serum silicic acid levels are twice as high as in man. Urine silicon excretion depends on bioavailable silicon in the diet and renal function.

Dobbie and Smith measured the levels of silicon in serum and urine in normal subjects of both sexes on different diets and in patients with renal impairment.[18] In one woman on water only, the intake of silicon was 16 micromoles/day, and the urinary excretion was 16 micromoles/day; that is 100% of the silicon intake. In nine male subjects on a dairy diet consuming an average of 3.4 liters of milk/day, the mean silicon daily intake was 96 micromoles and 24-hr urinary excretion was 130.0±0.13 micromoles, a negative balance of 34 micromoles, or 0.95 mg Si per day, which is a minimum since fecal excretion of Si was not measured. In subjects on a "normal diet" (73 male and 67 female subjects), the main silicon excretion in the 24-hr urine collections ranged from 200-310 micromoles, or 5.6-8.7 mg Si/day. In five male and nine female subjects consuming 5 gm of magnesium trisilicate (an antacid) containing 1.6 gm of silicon (57 millimoles), the mean ± SD of silicon level in the 24-hr urine collections was 3.1±1.02 millimoles, that is 5% of the amount ingested.

In patients with renal impairment on a normal diet, the amount of silicon excreted was proportional to the GFR, with 0.288±0.114 millimole/24-hr in 42 patients with GFR above 100 ml/min and 0.136±0.057 millimoles in 57 patients with GFR below 20 ml/min. Serum silicon levels in 10 men and 11 women with chronic renal failure were about twice as high as levels observed in normal subjects.

In random blood samples obtained from normal subjects, the level of serum silicon (X±SD) was 21.3±5 micromoles/L for 95 male subjects and 21.7±5 for 78 female subjects. No significant difference was observed between the sexes. The authors did not specify the menstrual status of the female subjects, that is, whether they were pre- or post-menopausal. As we will discuss later, post-menopausal women were found to have lower levels of serum silicic acid compared to pre-menopausal women.

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Review of Some Pertinent Research Studies on Silicon with Potential Clinical Applications

Silicon, in the form of bioactive mineral silicates, has been recognized for 30 years as an essential element participating in the normal metabolism of higher animals. So far, these finding have not been translated into practical recommendations for human health. There are no recommended daily allowances, and no cases of silicon deficiency have been reported. However, there is convincing evidence in favor of bioactive silicates playing important roles in the maintenance of optimal levels of mental and physical health.

Mental Health: An association between silicon deficiency and Alzheimer's disease (AD) was proposed by Carlisle in 1987.[19] Five percent of the population over 70 years of age suffers from AD. Another important fact is that AD is twice as common in women as in men.[20] In an attempt to reproduce in old rats the neurotoxicity of aluminum observed in AD and to evaluate the protective effect of silicon, Carlisle studied the effects of silicon and aluminum supplementation in the diet of female rats on silicon and aluminum levels in 12 brain regions. Two ages of female rats were studied: 22 days old (weanling rats) and 10 months old (Carlisle called them "retired female breeders"). We will call them young and old female rats. Groups of rats were assigned to four dietary regiments:

1) Low silicon diet.
2) Silicon supplementation using sodium metasilicate at 600 PPM in the diet.
3) Low silicon diet, plus aluminum supplementation, with aluminum oxide or chloride at 5,000 PPM in the diet.
4) Silicon supplementation + aluminum supplementation.

The experiment was carried out for 22 months in the four groups of young rats; and 18 months in the four groups of old rats.

At the end of the experiment, silicon and aluminum were measured in 12 different brain regions. There was no significant difference between the aluminum levels of the 12 brain regions in the low silicon diet of young and old rats. On the low silicon diet, aluminum supplementation showed no effect on brain aluminum levels in the young rats but increased brain aluminum levels significantly in the hippocampus and posterior cortex of old rats. Aluminum supplementation had no effect on brain aluminum levels in silicon supplemented old rats, suggesting a protective role of bioactive silicates, in adequate amounts, against aluminum neurotoxicity in old age.

Regarding brain silicon levels, there were differences between the 12 brain regions examined, with the highest levels observed in the hippocampus, caudate, lentiform nucleus, posterior cortex, olfactory bulbs and mid brain. Silicon supplementation increased brain silicon levels in the caudate, hippocampus and lenticular nucleus, the areas involved in AD. Aluminum supplementation decreased brain silicon levels, mainly in the caudate, hippocampus and lentiform nucleus. In the discussion section of her publication, Carlisle compared her results in rats with data available in human subjects. The age of her old rats approximates the age of 70 years in humans. In both rats and humans, the brain becomes more permeable to aluminum with aging. In both old rats and men, the highest concentrations of aluminum were found in the hippocampus. The protective role of silicon against aluminum neurotoxicity in old age is reflected by the observation that in the old rats supplemented with silicon, aluminum supplementation did not increase brain aluminum levels, whereas in the low silicon group of old rats, brain aluminum increased following aluminum supplementation.

Carlisle commented, "The finding that aluminum supplementation of the diet results in a significant decrease in brain silicon content in selected regions of the brain, including those regions thought to be involved in Alzheimer's disease, may be important … If silicon performs a role in brain metabolism, it is possible that aluminum may interfere with some specific silicon function. The variations in regional brain silicon concentrations support the possibility that silicon may be a functional trace element in the brain. The significantly higher levels of silicon in brain compared to plasma ($p < 0.05$), especially in certain regions (e.g., the hippocampus, caudate, and lentiform nucleus), even on very low dietary levels of silicon, also support this possibility … silicon appears to protect rats against aluminum induced abnormal behavior."

In support of Carlisle's postulate, Charnot has reported decreased levels of serum silicic acid following menopause in women, and following castration in women and female rats.[21] Serum silicic acid levels in castrated female rats were about 50% of the serum levels observed in control female rats. No effect was observed on serum silicic acid in castrated male rats. This could explain the higher incidence of AD in post-menopausal women versus old men. In the 15th Edition of *Harrison's Principle of Internal Medicine* published in 2001,[21] a recent pro-

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spective study is quoted, on the effect of estrogen replacement therapy on the prevention of AD in women. "Estrogen replacement therapy appeared to protect by about 50% against the development of AD in women." The effect of estrogen could be via increased peripheral silicic acid levels and, subsequently, increased brain bioactive silicate and silanolate levels. The response to estrogen in those women would probably be better if supplemented with bioactive silicates.

No published study, so far, has attempted to evaluate the effect of bioactive silica on either prevention or cure of AD. Since there are no recommended daily allowances, the daily amount administered in such studies would be tentative and depend on the form of bioactive silicates supplemented. Carlisle's study in rats used 600 PPM silicon as sodium metasilicates in the diet consumed. A 200-gm rat consumes an average of 20 gm of dry food/ day. Therefore, those rats would consume the equivalent of 60 mg Si/kg body weight. For a 70-kg human, that computes to 4.2 gm Si, which seems an excessive amount. However, the antacid, magnesium trisilicate, is consumed at those levels of Si daily by some individuals. We do not know the bioavailability of sodium metasilicate under the conditions of Carlisle's experiments.

In 115-lb sheep, balance studies showed that the fraction of silicon absorbed is dependant of the amount ingested.[8] On three different diets containing a daily average of 0.85 gm, 8.5 gm, and 22 gm of silica (measured as silica), the percentage of silica intake recovered in 24-hr urine collections (measured as silica) were 3.5, 1.7 and 0.8. The ingested amounts expressed as mg silica/kg BW/day would be 18, 180, and 490 (8.4, 84, and 220 if expressed as silicon). Since Carlisle's rats consumed 60 mg Si/kg BW/day, the bioavailability would be about 2%, or 1.2 mg Si/kg BW/day. For a 70-kg human, the amount of bioavailable and bioactive silicon required to neutralize the neurotoxic effect of aluminum, would be 84 mg Si/day. The amount of aluminum consumed by human subjects from an average diet is much lower[22] than the levels present in Carlisle's diet of aluminum supplementation. However, aluminum contamination of food and drink and the amount of aluminum in OTC medications may contribute to such high levels of aluminum in some individuals.

The protective role of silicic acid against aluminum toxicity has been demonstrated *in vitro* by Birchall and Espie.[23] The enzyme prolylhydroxylase is required for collagen synthesis. Silicon, in the presence of iron and vitamin C, enhances the activity of this enzyme *in vitro*.[14] Aluminum competes with iron for binding sites on prolylhydroxylase and inhibits this enzyme in the proper ratio with iron. The inhibition of this enzyme by aluminum at 100 micromolar concentration could be prevented with silicic acid added at 600 micromolar concentration. It is possible that the right amount of the right bioactive silicates may not only prevent AD but also reverse it. Since the anatomical lesions, together with high, localized aluminum concentrations, observed in certain regions of the brain of AD patients[24] were also found in patients with amyotrophic lateral sclerosis,[25] bioactive silicates may also be effective in non-AD related neurotoxicity of aluminum.

Physical Health: As previously mentioned, high levels of silicon occur in the organs with the highest amount of connective tissue such as the aorta, trachea, skin, tendons, and bones. The finding by Carlisle[12] that silicon deficiency is manifested by abnormalities involving articular cartilage and connective tissue has potential clinical applications in arthritic conditions.

In studies performed in animals and men, aging causes a progressive decrease of silicon concentrations only in the aorta and the skin. Loss of skin silicon in old age is associated with loss of interstitial water with decreased formation of collagen, glycosaminoglycans, and hexosamine. Bioactive silica supplementation may improve the texture of the skin in older individuals by increasing synthesis of these substances, resulting in increased hydration of the extracellular matrix.

Of greater clinical implication, however, is the loss of silicon in the aorta with aging. Loeper, *et al*,[26] observed that in man and rabbits high concentrations of silicon were found in the aorta, which is very rich in collagen, elastin, and glycosaminoglycans. In the rabbits, the aorta contained the highest levels of silicon, 5-10 times higher than levels in the pancreas, liver, heart, and kidneys. Loeper measured silicon levels in human aortas with different degrees of atheromatous lesions. With values expressed as µg Si/100 mg nitrogen, he observed the following:

1) In 19 cases with a normal aorta (i.e., no atheromatous lesion), 180±21 µg Si/100 mg nitrogen.
2) In 28 cases with moderate aorta damage (i.e., one or two plaques), 105±12 µg Si/100 mg nitrogen.
3) In 22 cases with serious aorta damage (i.e., numerous atheromatous plaques), 63±8 µg Si/100 mg nitrogen.

Loeper then studied the effect of bioactive silicates in rabbits on a high cholesterol diet for two months. Out of

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31 rabbits that received cholesterol, 24 suffered from atherosclerotic lesions of the aorta. Out of 28 rabbits who received the cholesterol diet plus intravenous silicates (sodium silicate, lysine silicate, or monomethyltrisilanol) at 10 mg every other day, only six aortas showed atheromatous lesions. The other 22 samples were perfectly healthy with smooth and shiny walls. Among 10 rabbits who received the cholesterol diet plus oral silicate (sodium silicate) at 10 mg/day, three out of the 10 showed atheromatous lesions. Overall, 77% of the rabbits on the cholesterol diet developed aortic lesions, compared to a combined rate in both groups receiving silicon (IV or oral) of 23%. Loeper further observed that elastin fibers of the aorta contain a high level of silicon. Silicate supplementation increased the thickness and the integrity of elastin.

In another publication by Loeper,[27] three groups of rabbits followed for two months were studied for atheromatous lesions of the aorta. In the control group of 15 rabbits, 14 had no lesions, and one rabbit had two atheromatous plaques. In 18 rabbits fed a high cholesterol diet for two months, only two were without lesions. Out of the 16 with lesions, eight were moderate, and eight were severe with multiple plaques. In the third group of 18 rabbits fed the high cholesterol diet for two months but with supplementation concurrently, using 10 mg monomethytrisilanol IV every other day, 14 rabbits had normal aortas without any lesions, three presented with one or two lesions, and one had several plaques. The male rabbits used by Loeper weighted 2.5 kg and received 10 mg IV every other day. Since the IV mode of administration is 100% bioavailable, 10 mg every other day would give a daily amount of 5 mg. Since the molecular weight of monomethytrisilanol is 110, 5 mg of this product would be equivalent to 1.25 mg Si/day. Corrected for body weight and expressed per kg/BW/day, the amount would be 0.5 mg Si. For a 70-kg person, 35 mg Si/day in the form of bioavailable and bioactive mineral silicates would be required.

The protective role of bioactive silicates against tumors in general, and brain tumors in particular, was reported by Carlisle.[28] When she kept several generations of rats on a low-silicon diet, the incidence of tumors, overall, was higher than rats supplemented with sodium metasilicate. In a longitudinal study lasting two years in the same generation of rats, equivalent to a 70-year longitudinal study in human subjects, the low silicon diet resulted in brain tumors in 60% of the rats but no tumor was observed in the group on the silicon supplemented diet. The clinical implications of such findings in cancer prevention, in general, and brain tumor, in particular, are obvious. Since reported 17 years ago, no follow-up study has appeared in the scientific literature, due to Carlisle's untimely death.

Discussion

The problems with silicon breast implants have given a bad name to silicon. Pulmonary complications of silicosis, an industrial lung pathology, due to inhalation of crystalline silica particles, such as asbestos (fibrous silicates of complex composition), has given a bad name to silica. Proper education would be required in order to make a distinction between the forms of silica and silicon mentioned above and the very important bioactive forms of silica, that is silicic acid and amorphous silica-mineral complexes, which could play an important role in brain function with application to AD and old age dementia. Besides AD, attention deficit disorders (ADD) may be another area worth investigating with bioactive silicate supplementation.

One approach that has been suggested to lower the incidence of AD and dementia due to aluminum neurotoxicity is to increase fluoride levels in drinking water above the EPA limits of 1.4-2.4 mg/L.[29 and vide infra] Fluoride blocks the absorption of aluminum from the intestinal tract by forming an aluminum-fluoride complex. One study in three counties of South Carolina revealed that two counties with fluoride levels within the EPA limits experienced a high annual incidence of primary degenerative dementia (PDD) in the population above 55 years of age: 17.1/100,00 and 20.8/100,000. However, in the county with high fluoride levels (4.18 mg/L) in the water supply, the incidence of PPD was only 3.6/100,000. Using high levels of fluoride to prevent aluminum neurotoxicity is not a good idea. To start with, activated alumina is used in water treatment plants to remove fluoride from water apparently in order to prevent mottling of tooth enamel. This procedure is the cause of elevated aluminum in drinking water to start with. It does not make sense to put fluoride back into municipal water in order to neutralize the neurotoxicity of aluminum after removing fluoride with alumina gel in water treatment plants. Besides, fluoride has its own problems — adverse effects on bone integrity, thyroid function, and cognition,[30-32] just to name three problems. There must be a better way, and it may include bioactive silicates. Future research would dictate the form of bioactive silicates most appropriate for replacement and supplementation. If Carlisle's postulate regarding an essential role of silicates in brain function is valid, and if silicon deficiency in the form of bioactive silicates is involved in AD, ADD, atherosclerosis, and arthritis, a good starting point would be to titrate the amount of bioavailable and bioactive silicates required to reverse these pathologies.

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The formulation of a silica-mineral complex should take into consideration the available data on the factors involved in the availability of silicic acid in the moist soil and the bioavailability of silicic acid from silica in its different forms in animals and men. The smaller the particle sizes of silica, the greater the hydrolysis of silica and the greater the release of silicic acid. Next to an aqueous solution of silicic acid, which is the most bioavailable form, microparticles of silica-mineral complexes combined with organic polyacids in a solid dosage form may be the next best alternative.

About the Author

Guy E. Abraham, MD, is a former Professor of Obstetrics, Gynecology, and Endocrinology at the UCLA School of Medicine. Some 35 years ago, he pioneered the development of assays to measure minute quantities of steroid hormones in biological fluids. He has been honored as follows: General Diagnostic Award from the Canadian Association of Clinical Chemists, 1974; the Medaille d'Honneur from the University of Liege, Belgium, 1976; the Senior Investigator Award of Pharmacia, Sweden, 1980. The applications of Dr. Abraham's techniques to a variety of female disorders have brought a notable improvement to the understanding and management of these disorders.

Twenty-five years ago, Dr. Abraham developed nutritional programs for women with premenstrual tension syndrome and post-menopausal osteoporosis. They are now the most commonly used dietary programs by American obstetricians and gynecologists. Dr. Abraham's current research interests include the development of assays for the measurement of iodide and the other halides in biological fluids and their applications to the implementation of orthoiodosupplementation in medical practice.

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