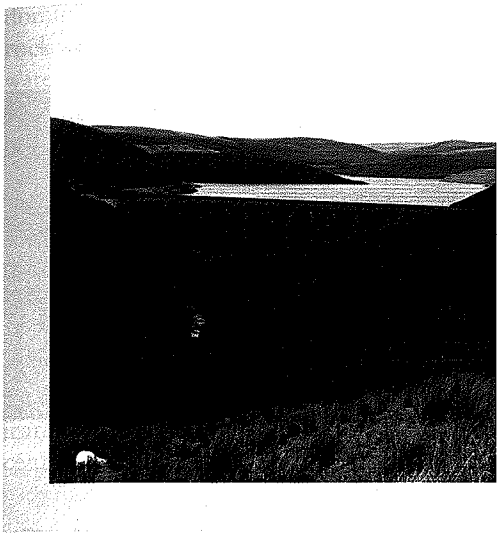

CHAPTER 13

WATER HARDNESS AND HEALTH EFFECTS

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I. DEFINITION OF WATER HARDNESS

Water hardness is the traditional measure of the capacity of water to react with soap and describes the ability of water to bind soap to form lather, which is a chemical reaction detrimental to the washing process. Hardness has little significance in terms of hydrochemical studies, but it is an important parameter for water users. Today, the technical significance of water hardness is more concerned with the corrosive effects on water pipes that carry soft water.

Despite the wide usage of the term, the property of hardness is difficult to define exactly. Water hardness is not caused by a single substance but by a variety of dissolved polyvalent metallic ions—predominantly calcium and magnesium—although other ions, for example, aluminum, barium, iron, manganese, strontium, and zinc, also contribute. The source of the metallic ions are typically sedimentary rocks, and the most common are limestone ($CaCO_3$) and dolomite ($CaMg(CO_3)_2$).

Hardness is normally expressed as the total concentration of calcium and magnesium ions in water in units of $mg\,L^{-1}$ as equivalent $CaCO_3$. Hardness can be determined by substituting the concentration of calcium and magnesium, expressed in $mg\,L^{-1}$, in the following equation:

$$\text{Total hardness} = 2.5(Ca^{2+}) + 4.1(Mg^{2+}) \qquad (1)$$

Each concentration is multiplied by the ratio of the formula weight of $CaCO_3$ to the atomic weight of the ion; hence, the factors 2.5 and 4.1 are included in the hardness relation (Freeze & Cherry, 1979).

In Europe, water hardness is often expressed in terms of degrees of hardness. One French degree is equivalent to $10\,mg\,L^{-1}$ as $CaCO_3$, one German degree to $17.8\,mg\,L^{-1}$ as $CaCO_3$, and one English of Clark degree to $14.3\,mg\,L^{-1}$ as $CaCO_3$. One German degree of hardness (dH) is equal to 1 mg of calcium oxide (CaO) or 0.72 mg of magnesium oxide (MgO) per 100 mL of water.

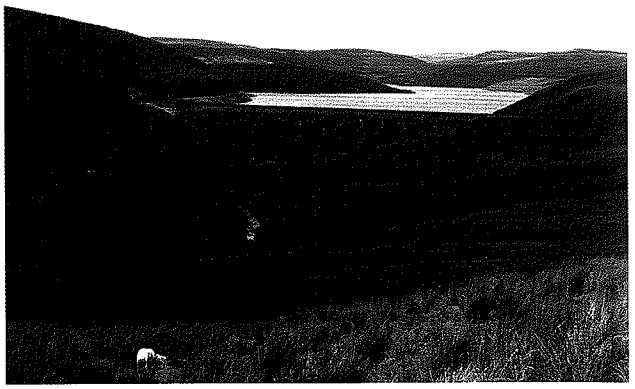
Where reported, carbonate hardness includes that part of the total hardness equivalent to the bicarbonate and carbonate (or alkalinity). If the hardness exceeds the alkalinity, the excess is called the non-carbonate hardness, and is a measure of the calcium and magnesium sulfates. In older publications, the terms "temporary" and "permanent" are used in place of carbonate and non-carbonate. Temporary hardness reflects the fact that the ions responsible may be precipitated by boiling, such that:

$$Ca^{2+} + 2HCO_3^- \xrightarrow{\text{Heat}} \underset{\text{"scale"}}{CaCO_3\downarrow} + H_2O + CO_2\uparrow \quad (2)$$

A number of attempts have been made to classify water hardness. Water with hardness values greater than $150\,mg\,L^{-1}$ is designated as very hard. Soft water has values of less than $60\,mg\,L^{-1}$. Groundwaters in contact with limestone or gypsum ($CaSO_4.2H_2O$) rocks can commonly attain levels of $200–300\,mg\,L^{-1}$. In water from gypsiferous formations, $1000\,mg\,L^{-1}$ or more of total hardness may be present (Hem, 1985).

Hardness in water used for domestic purposes does not become particularly troublesome until a level of $100\,mg\,L^{-1}$ is exceeded. Depending on pH and alkalinity, hardness of about $200\,mg\,L^{-1}$ can result in scale deposition, particularly on heating, and increased soap consumption. Soft waters with a hardness of less than about $100\,mg\,L^{-1}$ have a low buffering capacity and may be more corrosive to water pipes resulting in the presence of heavy metals such as cadmium, copper, lead, and zinc in drinking water. This depends on the pH, alkalinity, and dissolved oxygen concentration of the water.

No health-based guideline value is proposed for hardness because it is considered that the available data on the inverse relationship between the hardness of drinking water and cerebrovascular disease (CVD) are inadequate to permit the conclusion that the association is causal (WHO, 2002). However, a concentration of $500\,mg\,L^{-1}$ is at the upper limit of aesthetic acceptability.



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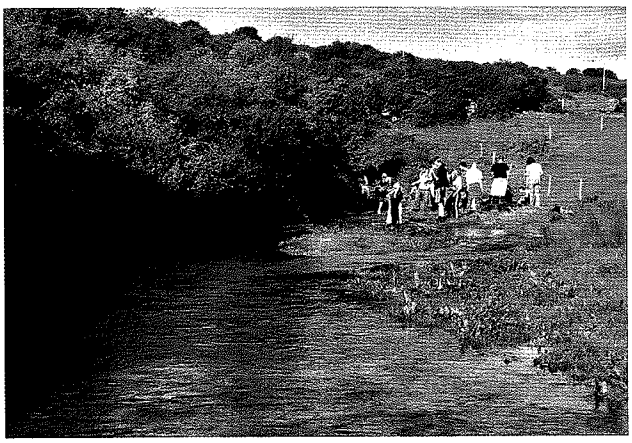

B

FIGURE 1 Illustration of two types of water resources and the influence of catchment geology on water hardness. In (A) surface water runoff from the impermeable Silurian mudstones and siltstones to the Dinas reservoir in west Wales is a soft water with a hardness of $19\,mg\,L^{-1}$ as $CaCO_3$, pH of 7.0, and electrical conductivity of $73\,\mu S\,cm^{-1}$ (sample date 1998; Hiscock & Paci, 2000). In (B) groundwater discharge from the Fergus River Cave springs developed in Carboniferous limestone in County Clare, Ireland, is a very hard water with a hardness of $256\,mg\,L^{-1}$ as $CaCO_3$, pH of 7.7, and electrical conductivity of $440\,\mu S\,cm^{-1}$ (sample date 2002).

II. NATURAL HYDROCHEMICAL EVOLUTION OF GROUNDWATER

The combination of geology and hydrology of a river catchment is important in determining the hardness of water. As illustrated in Figure 1A, catchments underlain by impermeable rocks that are resistant to erosion generate surface runoff with little time for weathering to occur. As a result, the surface water has a chemical composition similar to dilute rainfall and is characterized as soft water. In contrast, and as illustrated in Figure 1B, catchments underlain by permeable rocks allow water to infiltrate below the ground surface such that groundwater in contact with the rock mass promotes solutional weathering, which potentially leads to the development of fissures and conduits. As a result, the groundwater attains a high concentration of dissolved constituents and is characterized as hard water.

In natural aqueous systems, the chemical composition of water is continually changing and is controlled pre-

dominantly by geological factors. The geochemistry of rock weathering determines the release of elements into water. In groundwaters, a natural evolution of chemical composition is recognized. Dilute rainwater with a sodium chloride water type and containing CO_2 enters the soil zone whereupon further CO_2, produced by the decay of organic matter, is dissolved in the infiltrating water to form carbonic acid. Within the soil and unsaturated zone of sedimentary rocks, this weak acid dissolves soluble calcium and magnesium carbonates such as calcite and dolomite to give high concentrations of calcium, magnesium, and bicarbonate. In crystalline igneous and metamorphic rocks, slow weathering of silicates by the attack of carbonic acid releases low concentrations of calcium, magnesium, potassium, and sodium in the infiltrating soil water or groundwater and also produces bicarbonate.

Away from the supply of oxygen in the soil and unsaturated zone, infiltrating water becomes increasingly anoxic as a result of progressive bacterial reduction of oxygen. Below the water table and with increasing reducing conditions, iron and manganese become mobilized and then later precipitated as metal sulfides. In the presence of disseminated clay material within an aquifer, ion exchange replaces calcium for sodium as the water evolves to a sodium bicarbonate water type. Hence, the groundwater is naturally softened by ion exchange reactions. In the deeper, confined section of aquifers, mixing with saline water may occur to produce a sodium chloride water type before a region of static water and aquifer diagenesis is reached. Either part or all of this classic sequence of hydrochemical change is identified in a number of aquifers including the Floridan aquifer system.

The Floridan aquifer system occurs in the southeast United States (Figure 2) and is one of the most productive aquifers in the world. The aquifer system is a vertically continuous sequence of Tertiary carbonate rocks of generally high permeability. Limestones and dolomites are the principal rock types, although in southwestern and northeastern Georgia and in South Carolina the limestones grade into lime-rich sands and clays. The Floridan aquifer is composed primarily of calcite and dolomite with minor gypsum, apatite, glauconite, quartz, clay minerals, and trace amounts of metallic oxides and sulfides.

The total hardness of water in the Upper Floridan aquifer varies from <50 to >5000 mg L^{-1} as $CaCO_3$. Generally, where the system is composed only of limestone, the total hardness is equivalent to carbonate hardness and is <120 mg L^{-1}. Groundwater with higher total hardness usually results from (1) dissolution of other aquifer minerals, primarily dolomite and gypsum; (2) mixing of fresh water with residual saline groundwater; (3) encroachment and mixing of modern seawater; or (4) contamination. Natural softening of the groundwater by cation exchange is thought to be responsible for the low hardness in Escambia and Santa Rosa Counties, Florida (Sprinkle, 1989).

A sequence of hydrochemical evolution is identified by Sprinkle (1989), which starts with calcite dissolution in recharge areas that produces a calcium-bicarbonate dominated water type with a total dissolved solids (TDS) concentration of generally less than 250 mg L^{-1}. Downgradient, dissolution of dolomite leads to a calcium-magnesium-bicarbonate water type. Where gypsum is abundant, sulfate becomes the predominant anion. In coastal areas, as shown in Figure 3, seawater increases the TDS concentration and the water type changes to sodium-chloride. In the western panhandle of Florida, cation exchange leads to the development of a sodium-bicarbonate water type and a less hard groundwater.

For additional reading on groundwater modeling, see Modeling Groundwater Flow and Quality, this volume.

III. THE HARD-WATER STORY

The history behind what is considered today to be a commonly accepted fact—that hard water protects against CVD—is often referred to as the "hard-water story." The hard-water story started in 1957 with Jun Kobayashi, a Japanese agricultural chemist. He had for many years been engaged in studies of the nature of irrigation water from an agricultural point of view. In these studies he found a close relation between the chemical composition of river water and the death rate from "apoplexy" (CVD). The death rate of apoplexy in Japan was extraordinarily high compared to other countries and the biggest cause of death in Japan. Kobayashi found that it was the ratio of sulfur to carbonate (SO_4/CaO_3) that was related to the death rate from apoplexy. He suggested that inorganic acid $CaCO_3$ might induce or prevent apoplexy (Kobayashi, 1957).

Three years later a study was presented by Schroeder (1960), comprising the 163 largest cities in the United States. He found inverse correlations between water hardness and CVD in both men and women. He also studied the relation between different water constituents and coronary heart disease (CHD) among 45- to 64-year-old men. He found significant correlations

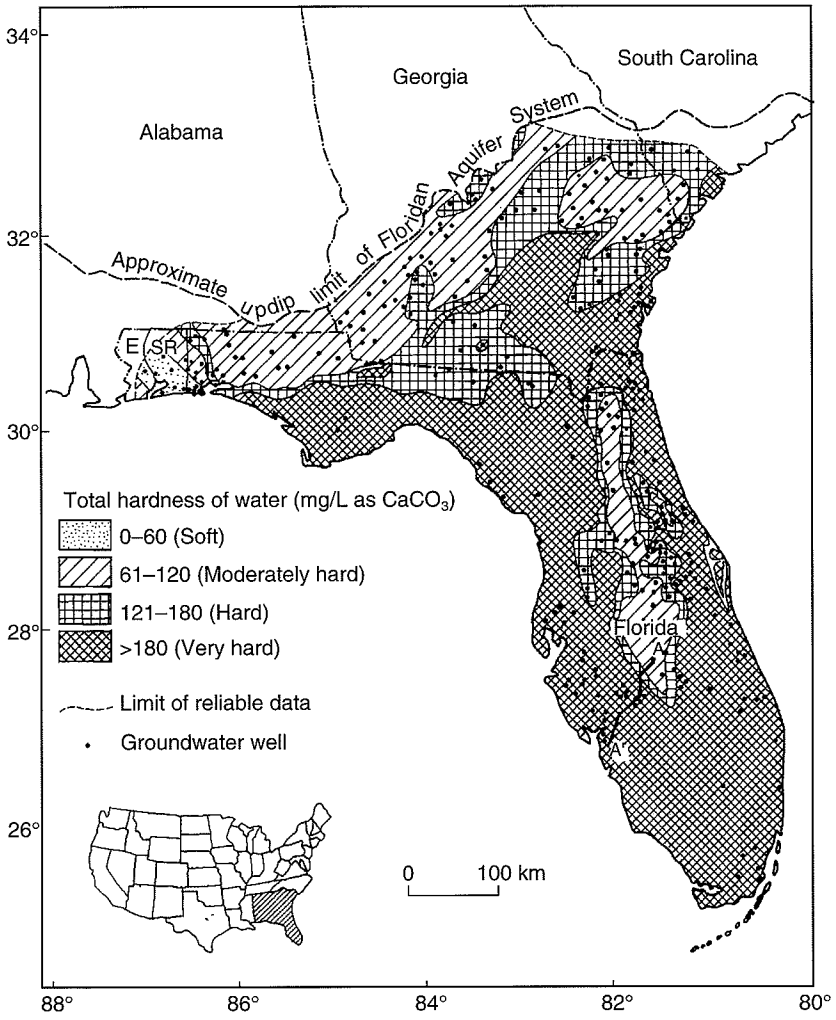

FIGURE 2 Map of the extent of the Upper Floridan Aquifer showing the spatial distribution of total hardness of water. Escambia and Santa Rosa Counties, Florida, are located at positions E and SR, respectively. (After Sprinkle, 1989.)

between death rate from CHD and sulfate and bicarbonate, respectively, but not for the ratio of the agents. He also found significant negative correlations between deaths from CHD and magnesium, calcium, fluoride, and pH. The coefficient of correlation for magnesium in drinking water was $r = -0.30$, $p < 0.01$, and for calcium $r = -0.27$, $p < 0.01$ (Schroeder, 1960).

During the following decades several studies on the relation between water hardness and CVD were presented. Initially the studies dealt with the question of whether there was a toxic effect in soft water or a protective effect in hard water. As described above, soft waters usually have low buffering capacity and are more corrosive, which leads to higher amounts of toxic trace elements. It has been proposed that this could account for the observed increased mortality. However, the results from previous studies have not supported this hypothesis (Marier, 1986a). For example, in a nationwide survey of more than 500 tap waters in Canada, no significant correlation was found between mortality and trace elements like lead, cadmium, cobalt, lithium, mercury, molybdenum, nickel, or vanadium (Neri et al., 1975). Gradually it became obvious that it was a protective effect from hard water that was responsible for the relations seen. In different parts of the world, studies have been made on the relation between magnesium and calcium in the local drinking water and CVD mortality. These studies were generally based upon death registers and water data at regional or municipality levels. The results of these studies are summarized in Table I.

Even with these studies, the results were not conclusive as to the role of magnesium and calcium in drinking water for CVD. Most of the studies showed a relation

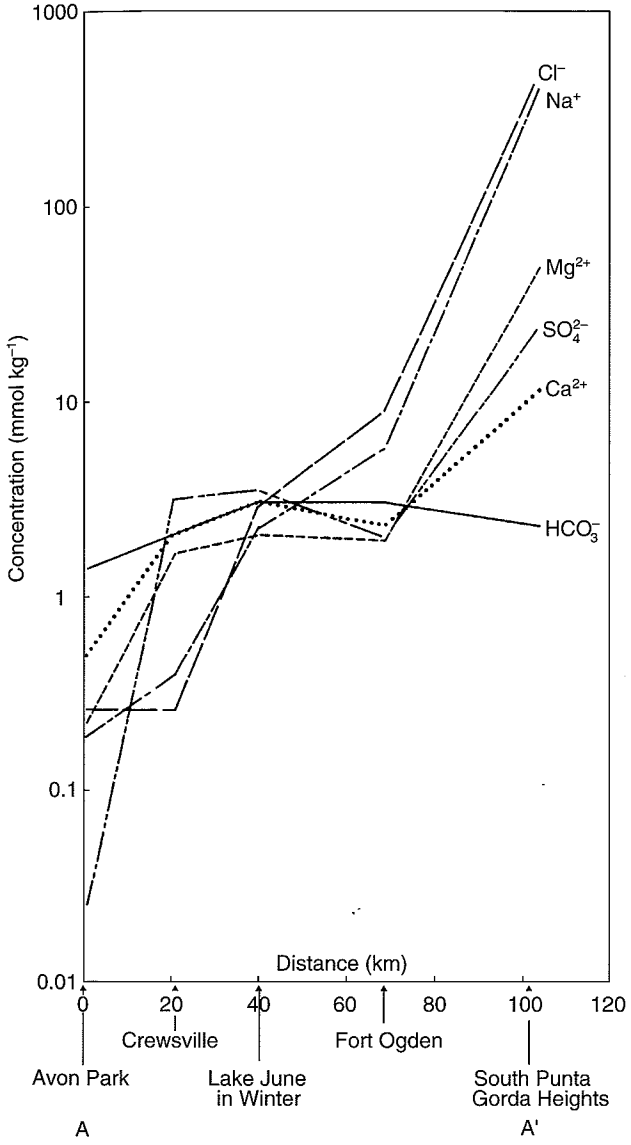

FIGURE 3 Hydrochemical section along line A-A′ (for location see Figure 2) showing the variation in concentrations of major ions downgradient in the direction of groundwater flow. Relative to calcium and magnesium, the groundwater experiences an increasing concentration of sodium as a result of ion exchange and mixing with saline water in the coastal zone. (After Sprinkle, 1989.)

between different cardiovascular deaths and either magnesium or calcium, or both, but some studies showed no relation at all. However, most of these studies were ecological meaning that the exposure to water constituents was determined at group levels with a high risk of misclassification. Often, very large groups, for example, all inhabitants in large cities or areas, were assigned the same value of water magnesium and calcium, despite the presence of several waterworks or private wells.

TABLE 1. The Effect of High Magnesium (Mg) or Calcium (Ca) Levels in Drinking Water on the Mortality from Cardiovascular Disease (CVD), Coronary Heart Disease (CHD), or Cerebrovascular Disease (CD) in Different Studies Published since 1975

	Mg			*Ca*		
Studies	*CVD*	*CHD*	*CD*	*CVD*	*CHD*	*CD*
North America						
Dawson et al., 1978	↓			↓		
Neri et al., 1975		↓			—	
Great Britain						
Shaper et al., 1980	—			↓		
Maheswaran et al., 1999		—			—	
Germany						
Teitge, 1990		↓				
Sweden						
Nerbrand et al., 1992	—			↓		
Rylander et al., 1991		↓	—		↓	↓
South Africa						
Leary et al., 1983		↓				

↓ = lower mortality, — = no difference.

In addition, the disease diagnoses studied were sometimes unspecific, with wide definitions that included both cardiac and cerebrovascular diseases. In some studies, it is also unclear whether the range of magnesium and calcium in drinking water was large enough to allow for appropriate analyses.

One of the most comprehensive studies of the geographic variations in cardiovascular mortality was the British Regional Heart Study. The first phase of this study (Pocock et al., 1980) applied multiple regression analysis to the geographical variations in CVD for men and women aged 35–74 in 253 urban areas in England, Wales, and Scotland from 1969 to 1973. The investigation showed that the effect of water hardness was nonlinear; much greater in the range from very soft to medium-hard water than from medium to very hard water. The geometric mean for the standardized mortality ratio (SMR) for CVD for towns grouped according to water hardness both with and without adjustments (by analysis of covariance) for the effects of four climatic and socioeconomic variables (percentage of days with rain, mean daily maximum temperature, per-

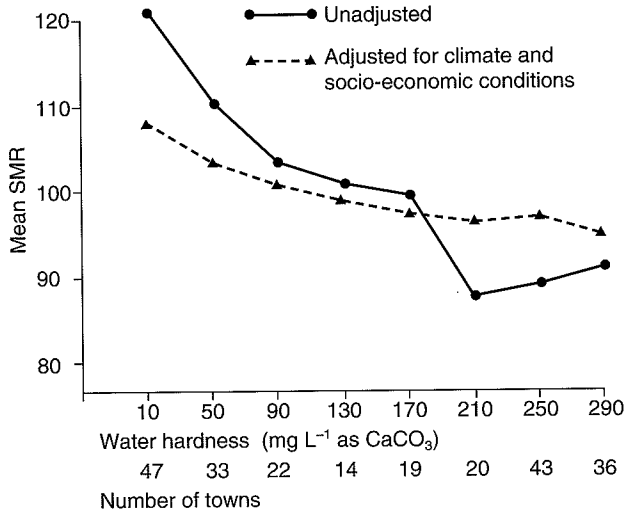

FIGURE 4 Geometric means of the standardized mortality ratio (SMR) (for all men and women aged 35–74 with CVD) for towns in England, Wales, and Scotland grouped according to water hardness (in concentration units of $mg L^{-1}$ as $CaCO_3$ equivalent). (After Pocock et al., 1980.)

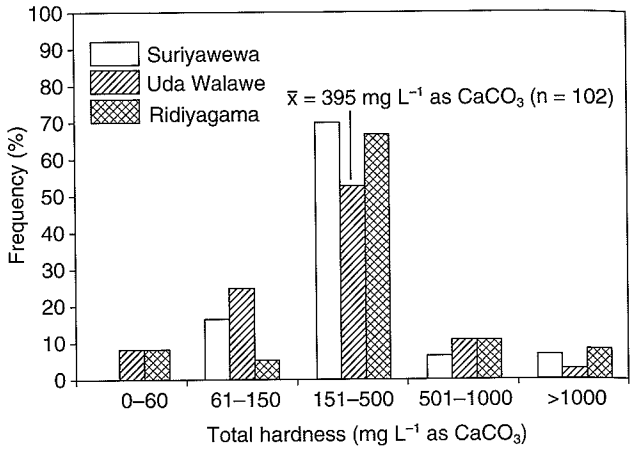

FIGURE 5 Histogram of total hardness values recorded for groundwaters sampled during the wet season (January and February 2001) from dug wells and tubewells in three subcatchments of the Uda Walawe basin of Sri Lanka. (Data courtesy of L. Rajasooriyar.)

centage of manual workers, car ownership) is shown in Figure 4. The adjusted SMR decreased steadily in moving from a hardness of 10 to 170 $mg L^{-1}$ but changed little between 170 to 290 $mg L^{-1}$ or greater. After adjustment, CVD in areas with very soft water, around 25 mg L^{-1}, was estimated to be 10–15% higher than in areas with medium-hard water, around 170 $mg L^{-1}$, whereas any further increase in hardness beyond 170 $mg L^{-1}$ did not additionally lower CVD mortality. Hence, it appeared that the maximum effect on CVD was principally between the very soft and medium-hard waters. Adjusting for climatic and socioeconomic differences considerably reduced the apparent magnitude of the effect of water hardness (Pocock et al., 1980).

A problem with correlation studies such as the British Regional Heart Study, as argued by Jones and Moon (1987), is the failure of much of the research to consider the causal mechanism that links independent variables to the disease outcome. Also, many of the calibrated models presented in the literature are socially blind in including only variables pertaining to the physical environment, which often contributes a large number of water quality elements. Even in those better analyses that have included social variables, as in the case of the British Regional Heart Study, the relatively strong correlation found for calcium in England and Wales may be a result of calcium acting as a very good surrogate for social variables. The soft water areas of the north and west of the British Isles equate to the areas of early industrialization, and today these areas house a disproportionate percentage of the socially disadvantaged (Jones & Moon 1987). Therefore, it is important that further studies undertake the challenge of quantitatively analyzing the separate effects of social variables from those of water hardness.

Fewer studies have been carried out in developing countries but Dissanayake et al. (1982), for example, found a negative correlation between water hardness and various forms of CVD and leukemia in Sri Lanka. More recent studies (Dissanayake, 1991; Rajasooriyar, 2003) have highlighted the problem of high fluoride concentrations and associated dental fluorosis in areas of hard water abstracted from crystalline bedrock aquifers in Sri Lanka.

Rajasooriyar (2003) measured total hardness in dug wells and tubewells in the Uda Walawe basin of southern Sri Lanka in the wide range of 7–3579 $mg L^{-1}$ as $CaCO_3$ with an average of 395 $mg L^{-1}$ as $CaCO_3$ (Figure 5). Compared with the government water quality limit of 600 $mg L^{-1}$ as $CaCO_3$, 12% of the 102 samples collected during the wet season in 2001 were in excess of the limit and are considered too hard to drink (values above 100–150 $mg L^{-1}$ as $CaCO_3$ are locally considered too hard as a water supply). Soft waters are found in areas with a dense irrigation network supplied by rain-fed surface reservoirs. Irrigation canal waters in the Suriyawewa and Uda Walawe subcatchments were measured in the dry season to have a total hardness in the range 40–90 $mg L^{-1}$ as $CaCO_3$ (Rajasooriyar, 2003), and it is leakage of this water source that leads to the soften-

ing of shallow groundwater. The majority of the groundwaters (63%) in the fractured aquifer represent very hard water with carbonate hardness in the range 151–500 mg L^{-1} as $CaCO_3$ contributed by the weathering of ferromagnesian minerals, anorthite, calcite, and dolomite. The products of this weathering lead to high concentrations of dissolved calcium, magnesium, and bicarbonate in groundwaters. Exceptionally high values of hardness (>1000 mg L^{-1} as $CaCO_3$) typically occur in non-irrigated areas with additional non-carbonate hardness contributed by pyrite oxidation and, in the case of the coastal Ridiyagama coastal catchment, by salt water inputs.

IV. STUDIES AT AN INDIVIDUAL LEVEL

In recent years some studies have been made with a higher precision as regards exposure classification than the previously cited ecological studies. Most of them have been conducted with a case-control design. This means that the exposure to water magnesium and calcium has been estimated for individuals who have suffered from the disease as well as for healthy control persons, and the difference in risk calculated. A similar type of study design is the prospective cohort study, where the subjects are followed over time.

A. Studies in Finland

Punsar and Karvonen made a cohort study in 1979. They compared the death rate from CHD in two rural areas in western and eastern Finland. The cohort comprised men 40–59 years old in 1959, and they were followed for 15 years. The water data were not truly individual but were median values in ten subareas in the western area and 33 subareas in the eastern area. The ranges of subarea medians were 6.9–27.8 mg L^{-1} of water magnesium in the western area and 0.6–7.3 mg L^{-1} in the eastern area. The cohort in eastern Finland had a death rate from CHD which was 1.7 times higher than the western cohort. Calcium was not investigated.

A few years later, Luoma et al. (1983) published a case-control study conducted in the southeastern region of Finland. Cases were men 30–64 years of age with a first acute myocardial infarction (AMI; alive or deceased), who were pair-matched with hospital controls for age and region (rural vs. urban). In addition, population controls were selected and matched for age and municipality. All subjects submitted a sample of their drinking water. The range of magnesium in the drinking water was 1.0–57.5 mg L^{-1} for the cases and 0.75–30.0 mg L^{-1} and 1.0–16.0 mg L^{-1} for the hospital controls and population controls, respectively. For case-population control comparisons, the relative risk (RR) with 95% confidence limits was 4.7 (95% confidence interval [CI] 1.3–25.3) for magnesium levels lower than 1.2 mg L^{-1}. This means that those with the lowest magnesium levels had a risk of AMI almost five times higher than those with higher magnesium levels. For the case-hospital control comparisons, the RR was 2.0 (95% CI 0.7–6.5), that is double the risk. They also found inverse relations with fluoride levels but no relation to calcium.

B. Studies in Sweden

In Sweden, three case-control studies have been conducted during the last decade. First, using mortality registers, the relation between death from AMI and the level of magnesium and calcium in drinking water was examined among men. A few years later a study with a similar design was made comprising women. The studies were conducted in southern Sweden, in a relatively small geographic area, where there was a great difference between as well as within the municipalities regarding magnesium and calcium content in drinking water. The advantage with this limited study area was that the possible risk of such confounding factors as climate, geographical, cultural, and socioeconomic differences was minimized.

Seventeen municipalities were identified whose water quality with respect to water hardness, acidity, and treatment procedures had been basically unchanged (change of hardness <10% and pH <5%) during the most recent ten years. Figure 6 shows the location of the 17 municipalities that comprised the study area.

Cases were men (n = 854) and women (n = 378) in 17 municipalities in southern Sweden who had died of AMI between ages 50 and 69 years. Controls were men (n = 989) and women (n = 1368) of the same age group who had died of cancer. Individual water data were collected. Table II shows the number of waterworks, the range of magnesium in drinking water, and the amount of magnesium in water supplied to the most densely populated area in each municipality.

The subjects were divided into quartiles according to the levels of magnesium. Odds ratios were calculated in relation to the group with the lowest exposure. Adjustments for age were made in all analyses. The results show odds ratios of 0.65 for men and 0.70 for women in the quartile with highest magnesium levels in the

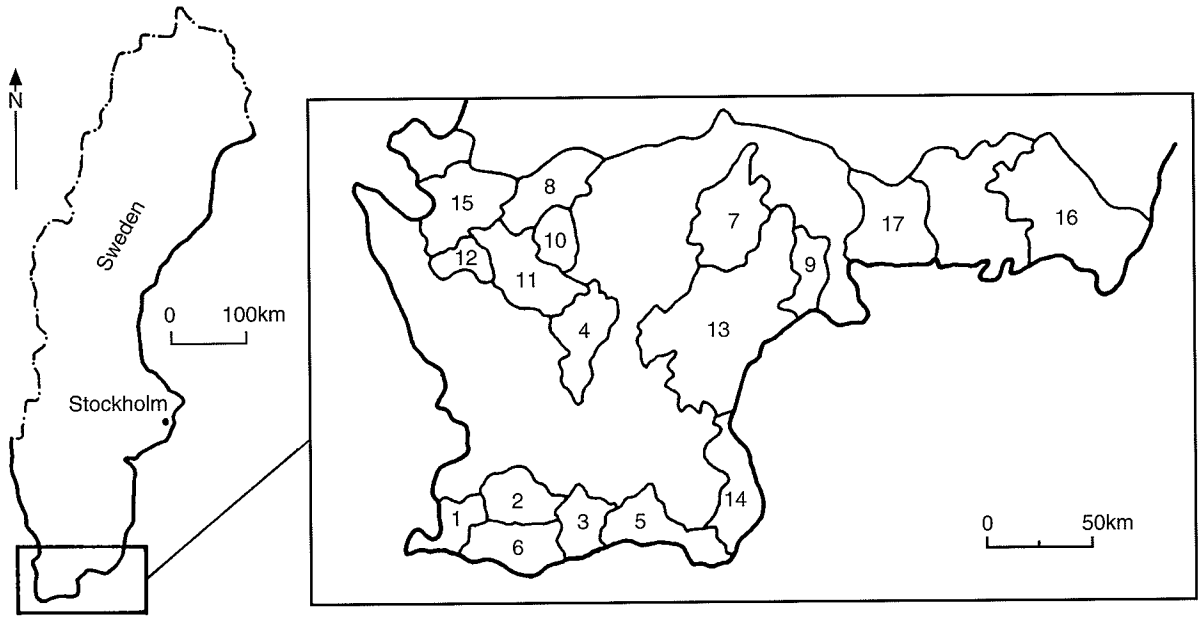

FIGURE 6 The country of Sweden with Skåne and Blekinge counties enlarged. The 17 municipalities in the studies are numbered corresponding to Table II, i.e., 1 Vellinge, 2 Svedala, 3 Skurup, 4 Höör, 5 Ystad, 6 Trelleborg, 7 Ö Göinge, 8 Örkelljunga, 9 Bromölla, 10 Perstorp, 11 Klippan, 12 Åstorp, 13 Kristianstad, 14 Simrishamn, 15 Ängelholm, 16 Karlskrona, and 17 Karlshamn.

TABLE II. Number of Waterworks, Range of Magnesium (Mg) Concentrations in Drinking Water ($mg L^{-1}$), and Amount of Mg in Water Supplied to the Most Densely Populated Areas ($mg L^{-1}$)

Municipality no.[a]	*Number of waterworks in the study*	*Range of water Mg* ($mg L^{-1}$)	*Water Mg in the most densely populated area* ($mg L^{-1}$)
1	2	5.9–10.0	10.0
2	2	6.8–20.0	20.0
3	4	2.6–10.0	9.0
4	2	8.8–13.0	8.8
5	5	5.1–11.9	7.5
6	4	6.5–18.0	16.0
7	9	3.3–13.5	5.0
8	3	3.0–6.9	6.9
9	5	1.3–7.6	1.3
10	2	4.0–9.0	9.0
11	2	5.5–9.0	8.0
12	1	10.0	10.0
13	15	1.3–14.4	6.7
14	9	3.8–13.4	9.7
15	2	7.0–11.0	11.0
16	9	2.0–13.0	2.0
17	2	1.9–5.0	2.0

[a]Municipality numbers correspond to Figure 6.

drinking water (≥9.8–9.9 $mg L^{-1}$) (Figure 7). This means that the risk of dying from AMI was about 30% lower compared with the risk for those who used drinking water with the lowest levels of magnesium (Rubenowitz et al., 1996, 1999).

A few years later a prospective interview study was conducted in the same area where men and women who suffered from AMI from 1994 to 1996 were compared with population controls (Rubenowitz et al., 2000). The results showed that magnesium in drinking water protected against death from AMI, but the total incidence was not affected. In particular, the number of deaths outside hospitals was lower in the quartile with high magnesium levels. This supports the hypothesis that magnesium prevents sudden death from AMI rather than all CHD deaths. The mechanisms that could explain these findings are discussed below.

V. Physiological Importance of Magnesium

A. Physiological Properties of Magnesium in Humans

Magnesium is involved in several important enzymatic reactions. All reactions that involve ATP (adenosine triphosphate), that is energy demanding reactions, have an absolute magnesium requirement (Reinhardt, 1988).

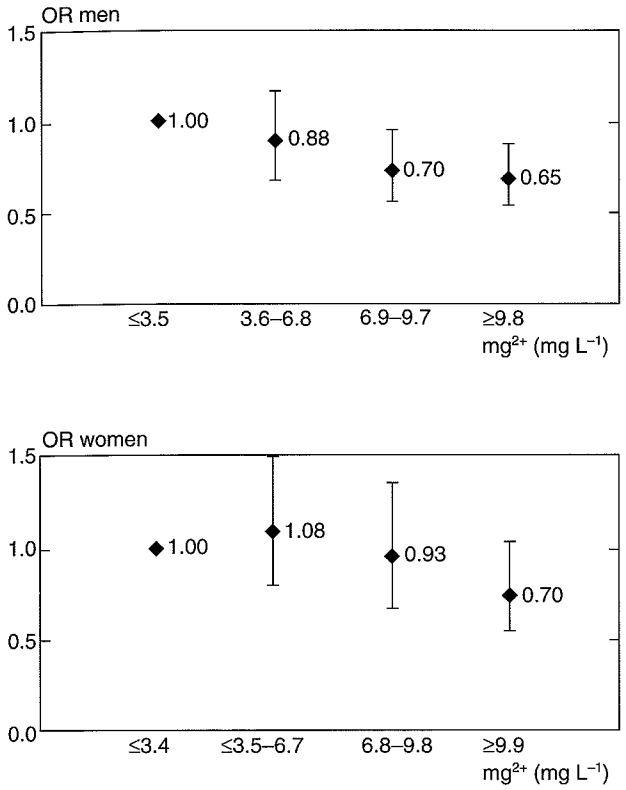

FIGURE 7 Odds ratios (OR) with 95% confidence intervals for death from AMI in relation to magnesium in drinking water (adjusted for age and calcium) in men and women.

Important processes in the body mediated by magnesium are, for example, synthesis of protein, nucleic acid and fat, glucose use, neuromuscular transmission, muscular contraction, and transport over cell membranes (Altura & Altura, 1996) (see also Chapter 30, this volume).

Magnesium is essential to the cardiovascular system, and two properties are especially important: stabilizing the cardiac electric system (preventing cardiac arrhythmia) and regulating vascular tone (Reinhardt, 1991). There are multiple mechanisms behind these properties.

Magnesium is needed to maintain the normal gradient of potassium and calcium over cell membranes (Altura et al., 1981). It is well known that magnesium is necessary to maintain intracellular levels of potassium. This is done by blocking the outward passage of potassium through the cell membrane and by activating the enzyme Na/K-ATPase. It has a similar function in Ca-ATPase. Magnesium also has a direct effect on potassium and calcium channels in the cell membranes (Reinhardt, 1991).

Furthermore, as regards impact on vascular tone, magnesium is a necessary activator for the synthesis of cyclic adenosine monophosphate (c-AMP), which is a vasodilator. It also acts as a natural calcium antagonist by competing for calcium binding sites in the vascular smooth muscle and thus reducing the constrictive effect of calcium in the blood vessels (Reinhardt, 1991). In addition, the vasoconstrictive actions of hormones such as angiotensin, serotonin, and acetylcholine are enhanced in the case of magnesium deficiency (Altura et al., 1981).

VI. MAGNESIUM INTAKE

A. Magnesium Deficiency

Marginal magnesium deficiency probably affects a large proportion of the population whose dietary intake does not reach the recommended amount. Although severe magnesium deficiency is not common in the population, hypomagnesemia is often present among hospitalized patients (Altura, 1994). A survey showed that hypomagnesemia was the most common electrolyte abnormality in patients entering the intensive care unit and was present in 20% of the patients (Reinhardt, 1988) (see also Chapter 8, this volume).

The causes of hypomagnesemia include reduced intake caused by starvation or intravenous therapy without magnesium supplement; impaired absorption owing to chronic diarrhea or malabsorption syndromes; increased renal loss caused by diseases such as diabetes or renal diseases or by the use of alcohol or such drugs as diuretics or antibiotics (Reinhardt, 1988).

B. Magnesium Intake From Food

The largest part of the total magnesium intake is from foodstuffs. Magnesium is present in many foodstuffs and in large amounts in nuts, beans, green leafy vegetables, and whole grain cereals. Mainly because of increased industrial treatment, which decreases magnesium levels by 80 to 95% (Marier, 1986b), it has been suggested that the majority of people today have a lower magnesium intake than the recommended dietary amount (RDA) of 6 mg kg^{-1} day^{-1} (Marier, 1986b; Seelig, 1986; Durlach, 1989). Table III shows the magnesium content in some foodstuffs and beverages.

Approximately 40% of magnesium ingested in food is normally absorbed (Hardwick et al., 1990). The proportion absorbed is inversely related to the amount ingested, however, and has been shown to range between approximately 10 and 70% (Fine et al., 1991).

TABLE III. Magnesium (Mg) Content in Food and Beverages

Food items and beverages	*Mg (mg 100 g^{-1})*	*Food items and beverages*	*Mg (mg 100 g^{-1})*
Food items		**Food items**	
Bread, white	23	Bananas	33
Bread, fiber-rich	50	Fish	25
Muesli	100	Shrimp	42
Corn flakes	16	Meat	25
Cheese	34	Eggs	13
Common beans, white, dried	184	Almonds	280
Common beans, brown, dried	131	Peanuts	188
Corn	23	Dark chocolate	130
Spinach	79	Milk chocolate	60
Avocado	39		
Broccoli	23	**Beverages**	
Tomato	10	Milk	12
Cucumber	10	Coffee[a]	5
Mushrooms	13	Tea[a]	3
Brown rice	110	Beer, low alcohol content	8
White rice	34	Beer, high alcohol content	11
Potatoes	24	Red wine	12
Apples	5	White wine	3
Oranges	10	Spirits	0

[a]Magnesium content in the water not included.

The absorption rate also depends on the intake of other foodstuffs. Elements such as calcium, phosphorus, fibers, and phytic acids are known to diminish the absorption rate of magnesium (Seelig, 1986). Most magnesium is absorbed in the distal small intestine, mainly by passive diffusion through the paracellular pathway, but also to a smaller extent by solvent drag and active transport (Hardwick et al., 1990).

VII. WATER MAGNESIUM AND BODY MAGNESIUM STATUS

A. Magnesium Intake From Water—Importance for Body Magnesium Status

Magnesium has a natural source in water from the weathering of a range of rock types. In igneous rock, magnesium is typically a constituent of the dark colored ferromagnesian minerals, which include olivine, pyroxenes, amphiboles, and dark colored micas. In altered rocks, magnesian mineral species also occur such as chlorite, montmorillonite, and serpentine. Sedimentary forms of magnesium include magnesite ($MgCO_3$) and dolomite ($CaMg(CO_3)_2$). Magnesium is substantially less abundant than calcium in all rock types and so in most natural waters the magnesium concentration is much lower, usually by 5–10 times, than the calcium concentration. Concentrations of magnesium in fresh waters are controlled by solution and precipitation reactions involving magnesium-bearing silicate and carbonate minerals with concentrations typically less than 50 mg L^{-1}, although values above 100 mg L^{-1} are recorded.

Magnesium intake via water depends on the level in drinking water. An important issue in discussions about the relation between water magnesium and AMI is whether magnesium in drinking water can be critical for the body magnesium status, as the main part of the magnesium intake derives from food (Neutra, 1999). It has been suggested that the quantitative contribution of water magnesium may be crucial for body magnesium status for those who have a low dietary intake and use water with high magnesium levels (Durlach et al., 1989). In addition, cooking food in magnesium-poor water leaches out magnesium, while cooking in magnesium-rich water diminishes this loss (Haring & van Delft, 1981).

Furthermore, it has been suggested that magnesium in water, appearing as hydrated ions, has a higher bioavailability than magnesium in food, which is bound in different compounds that are less easily absorbed (Durlach et al., 1989; Theophanides et al., 1990). However, simultaneous intake of other agents could diminish the absorption rate, as discussed above.

Plants cultivated in areas with magnesium-rich water may have higher magnesium content, especially if the soil is magnesium-rich and the land is irrigated with magnesium-rich water. People living in such areas who eat locally grown vegetables and fruits may also benefit from an addition to the total magnesium intake, especially during summer months. However, genetic factors appear to have a greater effect on plant magnesium composition than do soil and environmental factors (Wilkinson et al., 1987).

Some previous studies have shown relations between water magnesium and body magnesium content. In a study of baboons, tap water was more effective than dietary supplementation in increasing serum levels of magnesium and zinc (Robbins & Sly, 1981). Anderson

et al. (1975) found relations between water magnesium and magnesium content in the heart muscle. The myocardial magnesium was approximately 7% lower among residents of cities with soft water. There was, however, no difference in magnesium content in the diaphragm or the pectoralis major muscle. Other studies have shown relations between water magnesium levels and the magnesium content in skeletal muscle and in coronary arteries (Landin et al., 1989).

In a loading test, 10 subjects who normally used water with a magnesium level of $1.6\,mg\,L^{-1}$, instead were given drinking water with $20\,mg\,L^{-1}$ magnesium. After six weeks of supplementation with magnesium-enriched water, the excretion of magnesium was increased, which indicated improved body magnesium status (Rubenowitz et al., 1998).

Another study demonstrated that the ionized serum magnesium level was raised after only six days of an increased dietary load of magnesium, but not the total body magnesium. However, a correlation between ionized serum and ionized intracellular magnesium was shown (Altura & Altura, 1996).

From the available data it is apparent that the quantitative contribution from water may be crucial. The magnesium levels in drinking water in the Swedish case-control studies ranged from 0 to $44\,mg\,L^{-1}$. With a total daily intake of drinking water of two liters, the proportional magnesium contribution from water thus ranges from 0 to $88\,mg\,day^{-1}$. This is a percentage contribution between 0 and 25% of the RDA of $350\,mg\,day^{-1}$. For those who have a daily intake lower than the RDA and use magnesium-rich water, the contribution would be even more important.

In the prospective study, the calculated intake of magnesium from food ranged from 157 to $658\,mg\,day^{-1}$, median $356\,mg\,day^{-1}$. This means that a large number of the subjects had a lower intake than the RDA. One subject with an intake of 157 mg magnesium per day used drinking water with $3.5\,mg\,L^{-1}$, which means a 4.5% addition to the magnesium intake from food. If she instead had used water with $40\,mg\,L^{-1}$, the addition would have been 50%, and the total daily intake $240\,mg\,day^{-1}$.

VIII. CALCIUM IN DRINKING WATER AND CARDIOVASCULAR DISEASE

The majority of the previous ecological studies showed an inverse relation between calcium in drinking water and CVD, as reported above. The physiological mechanisms that could explain the relationship are not clear. There is, however, evidence that calcium deficiency can cause hypertension, which is a well-known risk factor for both stroke and AMI (Lau & Eby, 1985; Moore, 1989; Waeber & Brunner, 1994).

A. Calcium Deficiency

Calcium deficiency is common among the elderly, especially women. The absorption and renal conservation of calcium decreases with age. The absorption of calcium from food varies between 15 and 75% (Schaafsma, 1992), but in menopausal women the absorption is only about 20–30% (Heany & Recker, 1985). Furthermore, calcium intake is often decreased among the elderly (Harlan et al., 1984). The RDA for calcium in Sweden is $800\,mg\,day^{-1}$ for adult women and $600\,mg\,day^{-1}$ for men. In the United States, the RDA is 1000–1500 mg day^{-1} for adults.

A study comprising 61,000 women in Sweden aged 40–76 showed that calcium intake decreased with age and that a majority of postmenopausal women had a deficient calcium intake (Michaelsson, 1996). Several studies conducted in the United States have also shown an intake lower than recommended, especially among women (Fleming & Heimbach, 1994). For individuals with a deficiency, the additional calcium from water could be crucial to prevent this. Along with the contribution of drinking water, cooking food in calcium-rich water has been shown not only to prevent leaching, but even to increase calcium levels in the food (Haring & van Delft, 1981).

B. Calcium and Blood Pressure

Several studies have shown an inverse relation between dietary calcium intake and blood pressure. Meta-analysis comprising nearly 40,000 people has shown that a high calcium intake lowered both systolic and diastolic blood pressure (Cappucio et al., 1995). Low serum concentrations of ionized calcium have been measured in patients with hypertension (McCarron, 1982). There are several possible mechanisms that could explain how calcium lowers blood pressure.

One mechanism may be that hypocalcemia inhibits Ca-ATPase activity, which leads to an increase in free intracellular calcium and contraction of vascular smooth muscles (McCarron, 1985). Calcium supplementation has been shown to be efficacious, especially among salt-

sensitive hypertensives with a deficient basal calcium intake (Sowers et al., 1991). These individuals often have increased levels of the calcium regulatory hormones parathyroid hormone and active vitamin D ($1,25\text{-}(OH)_2\text{-}D$), which can cause increased peripheral resistance. Dietary calcium suppresses these hormones which causes the blood pressure to decrease. Calcium and calcium regulatory hormones may also influence blood pressure regulation via the central nervous system. Calcium also induces natriuresis, which has been shown to lower blood pressure in postmenopausal hypertensive women (Johnson et al., 1985).

The case-control studies reported above showed no clear relation between calcium and AMI.

IX. WATER HARDNESS AND OTHER HEALTH EFFECTS

The results of several studies have suggested that a variety of other diseases are also correlated with water hardness, which include various types of cancer. Incidences of a correlation between cancer and water hardness have been reported from studies in Finland and Taiwan. In northern Finland, correlations of age-adjusted incidences of various forms of cancer with the geochemical composition of groundwater were undertaken by comparing geochemical maps showing the hardness, uranium, iron, and nitrate content of water and maps showing the areal distribution of the incidences of ten forms of cancer (Piispanen, 1991). A statistically significant positive correlation was identified between water hardness and several forms of cancer, especially for all forms of cancer combined in the female population ($r = 0.66$). Piispanen (1991) suggested that drinking hard water may be an initiator and promoter of cancer, although it is admitted that a positive correlation between the geochemical and medical variables does not necessarily prove a cause-and-effect relationship between these variables. More recent research in Taiwan also proposes a possible association between water hardness in drinking water and several types of cancer. In a study of esophageal cancer, Yang et al. (1999) set up control groups that consisted of people who had died from causes other than cancer, and the controls were pair-matched to the cancer cases by sex, year of birth, and year of death. For esophageal cancer, the results showed that there was a 42% excess risk of mortality from esophageal cancer in relation to the use of soft water.

In a Japanese study of water hardness, regional geological features, and the incidence of struvite stones, Kohri et al. (1993) found a positive correlation between the magnesium-calcium ratio of tap water and the incidence of struvite stones. The incidence of struvite stones was both high in regions of basalt and sedimentary rock and low in granite and limestone areas.

In an ecological study of the relation between domestic water hardness and the prevalence of eczema among primary-school-age children in Nottingham, England, McNally et al. (1998) found a significant direct relation between a one-year period and lifetime prevalence of eczema and water hardness, both before and after adjustment for confounding factors (sex, age, socioeconomic status, access to health care). Eczema prevalence trends in the secondary-school population were not significant.

X. CONCLUSIONS AND CONSEQUENCES

The significance of earlier studies of the link between water hardness and CVD is unclear, and it is suggested that the reported associations may reflect disease patterns that can be explained by social, climatological, and environmental factors rather than by the hardness of the water. However, from the results of epidemiological and experimental studies, the most significant conclusion is that magnesium in drinking water prevents death from AMI, either by preventing arrhythmia or spasm in coronary blood vessels. It is not difficult to imagine the importance of these findings for public health.

A. A Public Health Perspective

Marier and Neri (1985) attempted to quantify the importance of water magnesium using a number of the abovementioned epidemiological studies. They estimated that an increase in water magnesium level of 6 $mg\,L^{-1}$ would decrease CHD mortality by approximately 10%.

The data collected in the Swedish case-control studies can be used to estimate the impact of water magnesium on the incidence of myocardial infarction in the study population. If everyone in the male study base were to drink water from the highest quartile

($\geq 9.8\,mg\,L^{-1}$), the decrease in mortality from AMI would be about 19%. This means that the age-specific incidence of death from myocardial infarction in the study area would change from about 350/100,000 $year^{-1}$ to 285/100,000 $year^{-1}$. The decrease of the incidence per $mg\,L^{-1}$ magnesium can be calculated to be approximately 10/100,000 $year^{-1}$, which is an even larger decrease than Marier and Neri (1985) estimated.

For women, the corresponding decrease in mortality from AMI would be about 25% if everyone were to drink water from the highest quartile ($\geq 9.9\,mg\,L^{-1}$). The age-specific mortality among women in the study area would change from about 92/100,000 $year^{-1}$ to 69/100,000 $year^{-1}$.

B. Consequences

What should be the consequences of today's knowledge of a recommended intake of magnesium? Is there sufficient evidence to recommend an increased intake of magnesium? If so, should the recommendation be applicable to the whole population or only to certain risk groups? Furthermore, what would be the best way to increase magnesium intake?

The consumption of magnesium-rich food and water can be encouraged, and the use of water softeners in areas with hard water discouraged. At least one tap with unfiltered water for drinking should always be left. Although such recommendations are hardly controversial, it is difficult to make them effective, especially if the target is the population as a whole.

Theoretically, a possible way of increasing magnesium intake would be to add magnesium to drinking water, especially in areas with naturally soft water. This is already done in some waterworks in order to reduce corrosiveness. To do so on a larger scale would, however, be expensive and politically difficult.

Recommendations to use oral magnesium supplementation would not be practically feasible for the whole population, but could be considered to be directed toward certain risk groups.

Before a general prevention program can be accepted, large-scale intervention studies must be conducted to accurately evaluate the preventive effect of magnesium. This would require several thousands of subjects, however, and would thus be cumbersome and expensive. Nevertheless, in view of the significant implications for public health, such studies should be conducted. The possibility of a simple and harmless way of reducing AMI mortality rate must not be overlooked.

SEE ALSO THE FOLLOWING CHAPTERS

Chapter 5 (Uptake of Elements from a Biological Point of View) · Chapter 8 (Biological Responses of Elements) · Chapter 22 (Environmental Medicine) · Chapter 23 (Environmental Pathology) · Chapter 31 (Modeling Groundwater Flow and Quality)

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