Role of magnesium in the "hard-water story"

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Summary

The "water story" is briefly reviewed in light of 3 decades of research with input from South Africa, Japan, the U.S., Britain, and Canada. It is shown that there are 2 "water factors" (i.e., corrosivity and magnesium) that can influence human mortality.

Résumé

Le thème «histoire eau» est examiné en relation avec 30 années d'étude sciéntifique provennant de l'Afrique-du-sud, le Japon, les Etats-Unis, la Grande Bretagne, et le Canada. On démontre qu'il-y-a 2 «facteurs eau» (la corrosiveté et le magnesium) qui peuvent influencer la mortalité du genre humain.

Zusammenfassung

Es werden Untersuchungsbefunde über die Bedeutung der Wasserhärte vorgestellt, die in 3 Jahrzehnten in Südafrika, Japan, den Vereinigten Staaten, in England und Kanada erhoben wurden. Es scheint, daß 2 Faktoren im Trinkwasser, nämlich die Korrosivität und das Magnesium, die Mortalität der Bevölkerung beeinflussen können.

It was 30 years ago that researchers in South Africa first reported that magnesium supplementation had been remarkably effective in the treatment of degenerative heart disease in more than 200 white South Africans who — unlike Bantu tribesmen — had a low dietary intake of magnesium and a high incidence of coronary ailments [14].

One year later, a Japanese report announced an interrelation between human cerebrovascular mortality and the composition of river water [12], and this was the origin

Tab. 1: Types of water hardness

Total hardness	= Ca + Mg
Carbonate hardness	$= HCO_3 + CO_3$
Non-carbonate hardness	$= SO_4 + CI$

of what has since been termed the "water factor" or "water story". However, this Japanese observation had nothing to do with magnesium (or calcium), but was based on other water-related considerations. The explanation requires a few definitions (see Tab. 1).

Total hardness: This is a measurement of the sum of the molecular concentration of calcium and magnesium, although it is not usually expressed as such. For example, a hardness expressed as "100 ppm CaC0₃" denotes a water containing 1×10^{-3} M of Ca+Mg, and this concentration seems to be intermediate between "hard" and "soft" designations [16]. A disadvantage of the "total hardness" measurement is that the Ca-vs-Mg proportionality is extremely variable among waters from different localities. Thus, the weightbasis Ca/Mg ratio can vary between 0.2 and 15.0, and this means that magnesium (or calcium) can account for as little as 10% - and as much as 90% — of the "total hardness" measured [17].

Carbonate hardness: This denotes the proportion of hardness chemically equivalent to the concentration of carbonate-bicarbonate, and is thus an index of water's alkalinity and buffer capacity [11]. The preponderance of the ion species is bicarbonate, although the measurement is often based on total carbonate analysis; therefore, the designations CO₃ and HCO₃ are often used interchangeably. Waters of low "carbonate hardness" have only limited buffer capacity, and therefore have a tendency to become acidic when exposed to airborne CO₂ [4, 9, 11].

Non-carbonate hardness: This denotes the proportion of hardness attributable to anions such as sulfate and chloride which do not confer any appreciable buffer capacity. Soft waters high in sulfate are considered to be especially corrosive [7, 9, 27].

In the initial Japanese study [12], cerebrovascular mortality was directly related to high waterborne SO₄/CO₃ ratios (weight basis). Sulfate was the dominant anion (i.e. $SO_4/CO_3 > 1.0$) in 21% of the rivers surveyed, and this was attributed to Japan's volcanic geology, especially in northern Honshu where some-acid waters had a low pH between 3 and 4. It must be emphasized that these were soft waters (Ca = 12.5, and Mg = 2.7 mg/l) whose total hardness averaged 42 ppm CaCO₃ Epidemiologically, the situation in Japan was "roughly correlated to acidity, and especially the sulfate content of river water"; it was also noted that acid waters high in sulfate or chloride could be expected to solubilize trace metals from geological strata [25]. However, the SO₄/CO₃ (or SO₄/HCO₃) factor

was not detected in a U.S. epidemiological assessment [26]. The possible importance of total hardness was first emphasized in the U.S., where an inverse correlation between State averages for water hardness and metropolitan "all causes" mortality was attributable primarily to an effect on cardiovascular diseases [26]. This inverse correlation between total hardness of drinking-water and cardiac-related diseases was soon confirmed by British researchers [21], who obtained a strong correlation with waterborne calcium but not magnesium. This is in sharp contrast with observations in the U.S. [27] and Canada [22] where the correlation coefficient for calcium was only 60% of that for magnesium. However, it has been pointed out that British hard waters are preponderantly calcic, but contain relatively little magnesium [15, 17]. It is also important to emphasize that the British data showed that total hardness, carbonate hardness, and waterborne calcium produced very similar correlation coefficients [21]. Moreover, there has been the emergence of detailed critiques emphasis whose is that epidemiological data in Britain can best be explained on the basis of toxic trace-element contamination in corrosive soft waters of low carbonate content [4, 5] and thus somewhat analogous to the Japanese situation. In this regard, it must be admitted that — although the British studies did not find a meaningful correlation with waterborne sulfate nor with non-carbonate hardness - they did not pursue the SO₄/HCO₃ aspect [21].

There is, nevertheless, evidence for this "corrosivity" factor in the U.S. coal-mining Appalachian "triangle" of eastern Ohio, Pennsylvania, and northern West Virginia [7, 9]. In this general region, a pH as low as 3.9 has been found in some waters [9, 26]. Furthermore, such corrosive waters are hard waters (total hard-150-450 ppm CaCO₃)

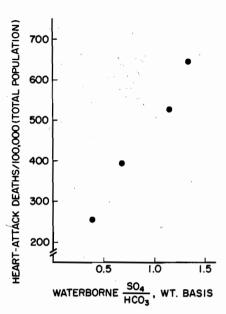


Fig. 1: Heart-attack mortality as a function of waterborne SO₄/HCO₃ ratio (plotted from the data of Bain, 1979) Linear regression: $y = 119 + 388 \times$ r = 0.99

which are known as "calcium sulfate" types [7, 9]. One of the studies revealed what was termed "slight" correlations with heart-attack deaths, positively related to sulfate and negatively related to bicarbonate content of surface waters used for drinking [7]; these opposite trends are here combined into SO₄ HCO₃ ratios, and the result is rather striking (Fig. 1), and definitely reminiscent of the Japanese observations. It must be emphasized that the waters relating to Fig. 1 are calcium-rich hard waters with a pH range of 6.5 to 8.0 [consult ref. 7], and thus only resemble the Japanese waters because of the interrelation between mortality and waterborne SO₄/HCO₃ ratios.

The one consistent observation in the aforementioned epidemiological observations in Japan, Britain, and the U.S. Appalachian region, is that mortality is an inverse function of the waterborne carbonatebicarbonate content. Because carbonate-poor waters lack buffer capacity and are thus likely to become acidic and/or corrosive, it has been proposed that such waters (usually soft) solubilize toxic trace elements from geological strata [7, 9, 12] or from plumbing conduits [4, 5, 23, 27] and that this "corrosivity" factor therefore accounts for the increased mortality. This is certainly a plausible hypothesis [8], although it remains nebulous because there has been no Dose/Response identification of the toxic substance(s) in health-related studies of potentially-harmful waters. In-a nationwide survey of more than 500 tap waters in Canada, no meaningful correlation was found between mortality and waterborne trace elements such as cadmium, cobalt, lead, lithium, mercury, molybdenum, nickel, or vanadium [22].

At this juncture, it is relevant to recall a statement in a 1962 report about the U.S. situation [24]:

"The high-rate (mortality) areas ... have practically no magnesium in the drinking-water ... A majority of the cities in the low-rate areas have some magnesium in the drinking-water, i. e., more 10 ppm."

The U.S. region of highest mortalis an Atlantic seaboard strip whose extremities include Massachusetts and Georgia [24]. In comparison, there is a U.S. zone where mortality (i. e., "all causes", cardiovascular, coronary) is about 40% lower; this lowest-mortality region is in the midwest prairies, as delineated by the dotted lines in Figure 2. It can be seen that this lowest-mortality zone extends from within Texas to the Canadian border. Note that, on the map, the U.S. zone has been extended in a semi-circular arc encompassing southerly portions of Canada's 3 prairie Provinces. To the east, the map also highlights a relatively small region of southern Ontario where there is an oval-shaped area measuring only about 150 × 100 km, but which contains the



Fig. 2: North American regions having high concentrations of magnesium in drinking-water, and/or, having the lowest mortality rates (see text)

preponderance of Ontario's magnesium-rich hard drinking-waters and where the maximum water-borne magnesium concentration is 49 mg/L [23].

It is in Ontario that Anderson found that — in comparison with residents of hard-water localities (Mg = 30 mg/l) — residents of softwater communities (Mg = 2 mg/l) have a myocardial magnesium content that is 6% lower [2], and ex-

perience about 40% more suddendeath ischemic heart disease [3]. At the southern end of the U.S. lowest-mortality zone (Figure 2), inscribed dots pin-point 6 Texas communities whose waterborne magnesium concentration is 30-ormore mg/l [10]. This "west Texas" region has the lowest mortality rates in the U.S. [10, 24]; moreover, an epidemiological study of the role of local drinking-water led

to the conclusion that "... the most mortality protection would seem to be the ingestion of magnesium..." [10]. Further north, the points shown in the Canadian prairie region denote 31 communities whose drinking-water has revealed a magnesium concentration of 30-ormore mg/l [23]; moreover, it is in this region that Canada's lowest mortality rates are seen (Tab. 2), especially in Saskatchewan and

Tab. 2: Provincial averages for waterborne magnesium, and standardized "all causes" mortality per 100,000 males, age 35-64 (adapted from Neri et al., 1977)

Province	Magnesium mg/L	Mortality rate
Saskatchewan	31	809
Manitoba	23	744
Alberta	13	969
Ontario	12	975
Prince Edward Island	7	924
Quebec	4	958
New Brunswick	3	929
Nova Scotia	2	1026
British Columbia	2 .	1070

Manitoba. Note that the mortality trend is inversely proportional to the average waterborne magnesium concentration in each Province [23].

Thus, both extremities of the North American lowest-mortality zone are associated with high water-borne magnesium (Fig. 2). Incidentally, the highest drinking-water Mg concentration was found to be 100 mg/l in Texas [10], and 96 mg/L in Canada's prairie region [23]. In this presentation, the "criterion" of 30 mg/l was selected because waters with that level of magnesium

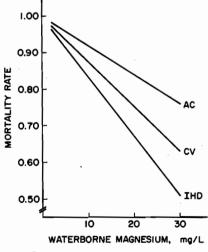


Fig. 3: Illustration of how waterborne magnesium relates to mortality from ischemic heart disease (IHD), cardiovascular disease (CV), and "all causes" mortality (AC)

are numerous enough to allow graphic illustration of the geographic distribution. However, as previously stated, it remains true that "40 mg of magnesium per liter ... is unusual in public water supplies" of North America [28].

The Canadian mortality-vs-magnesium trend (Tab. 2) can be expressed as a linear regression equation for which r = 0.82; and thus, ",all causes" male mortality/100,000 $= 1021 - (8.1 \times Mg)$ in which "Mg" is the waterborne magnesium concentration in mg/l. With this equation, it can be calculated that a 10% reduction in "all causes" mortality can be expected with each waterborne magnesium increment of 12.6 mg/l. As reported in a recent discussion of regression analysis [18], a 10% reduction of cardiovascular and ischemic heart disease mortalities can be expected with a waterborne magnesium increment of 8.2 and 6.1 mg/l, respectively. The three trends are intercompared in Fig. 3 which illustrates that waterborne magnesium is most effective against ischemic heart disease (IHD), less effective against cardiovascular (CV), and least effective against "all causes" (AC) mortality. According to Anderson's thesis that sudden-death myocardial infarction is the process most likely to be influenced by the "water factor" [1, 3], the pattern in Fig. 3 is to be expected.

It is surprising that the overall trend in Fig. 3 is so clear-cut, because the IHD data is from a 1983 South African report, and the CV data is from a 1962 U. S. report [see ref. 18], whereas the AC data is from a 1977 Canadian monograph [23]. Because of these disparities, the pattern shown in Fig. 3 is intended merely for illustrative purposes. But nevertheless, the trends in Fig. 3 reinforce the fact that "the waterborne magnesium factor (is) a global phenomenon" [18].

Experimental studies conducted during the late 1950s provided information [20, 30] that remains per-

tinent in today's discussion. Thus, heat stress, cold stress, or a high cholesterol diet each increased the dietary magnesium requirement. Under such conditions, failure to increase the magnesium intake caused a decreased oxidative phosphorylation efficiency of mitochondria, particularly in the heart. This caused a pre-necrosis reduction in the number of heart mitochondria (i.e., down to 65% of normal). along with "wash-out" of magnesium from the intracellular compartments. As stated in one of the reports, this "clearly demonstrates the protective value of dietary magnesium against the stresses to which one may be exposed in daily life" [20]. Moreover, it also illustrates the need to allow a considerable safety-factor when recommending an RDA (Recommended Dietary Allowance) for magnesium.

Another stressor that must be considered is exercise stress, because recent experimentation has shown that activities involving endurancestress are impaired by inadequate magnesium intake, but enhanced by magnesium supplementation [6, 29]. The aforementioned depletion of oxidative phosphorylation which accompanies an inadequate dietary magnesium intake is a reminder that intracellular Mg: ATP complex is the reserve storage of highenergy phosphate that is vital for cardiac energetics. If the Mg: ATP reserve becomes depleted, a sudden adrenergic/cholinergic surge for increased cardiac output may not be met, and thus could result in a "sudden death" infarctation [15, 16, 17].

The role of magnesium in the "water story" consists of providing an extra supply of magnesium. For example, daily ingestion of 2 liters of water (in coffee, tea, soup etc.) containing 30 mg/l of magnesium would provide 60 mg of magnesium, and this represents 17 and 20% of the U. S. RDA for men and women, respectively. And, in a recent review [19], it was calculated

that the dietary intake of magnesium by U.S. adults averages 76% of the RDA. It can therefore be appreciated that the extra magnesium supplied by water can go a long way toward replenishing an otherwise-inadequate magnesium intake. It has recently been demonstrated that effective clinical metabolic improvement can be achieved with supplemental magnesium dosages that approximate the "shortfall" in intake, as assessed by the U.S. RDA, and that this is therefore "an indication that the U.S. RDA is a reasonably accurate index of metabolic magnesium requirements" [19].

Thus, there are 2 parts to the "water story" and both have plausibility [8]. Furthermore, both factors might conceivably "pull in opposite directions". In one region, a 10% increase in mortality could bé associated with low-carbonate corrosive waters; in a different region, waterborne magnesium might result in a 10% decrease in mortality. The overall difference in mortality between the 2 regions would be 20%, but due to two different water-related phenomena. One also wonders what would happen if a corrosive low-carbonate water is also rich in magnesium - would the adverse effect of a high SO₄/ HCO₃ ratio tend to override any beneficial effect of magnesium? In this "acid rain" era, we may soon know the answer.

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