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CARDIO-PROTECTIVE CONTRIBUTION OF HARD WATERS TO MAGNESIUM IN-TAKE

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The inverse correlation between water hardness and incidence of cardiac fatalities was first reported in 1957, and has since been observed in many regions of the world (Masironi, 1972). It is therefore reasonable to expect that a «common factor» in drinking-water would largely explain this global trend, and several researchers have suggested that the magnesium present in hard waters has a cardioprotective influence (Parsons et al., 1961; Marier et al., 1963; Bajusz, 1967; Marier, 1968; Holtmeier, 1969; Anderson, 1972).

Recent Canadian studies have reinforced the hypothesis that hard-water magnesium may play a crucial role in the prevention of cardiac ailments. In a nation-wide survey of 15 elements present in 575 drinking-waters (Neri et al., 1975, 1977), magnesium emerged as the most likely «candidate element» on the basis of several criteria, i.e., it was present in more than 10% of the sampled waters; magnesium is a consistent function of the softness-hardness gradient; magnesium represents a significantly-high proportion of the daily intake from other sources; the known metabolic effects of magnesium are consistent with the hardness-mortality trend. Furthermore, analysis of 350 tissue samples from 161 autopsy cases (Anderson et al., 1973, 1975) revealed that myocardial magnesium was 6% lower in «cardiac death» patients from soft-water localities, in comparison with hard-water regions; also, myocardial magnesium in all «cardiac death» tissues averaged 22% lower than in the group of non-cardiac fatalities. A similar 12-to-15% lower heart-muscle magnesium content in sudden-death cardiac cases has also been reported in Britain (Chipperfield and Chipperfield, 1973; Behr and Burton, 1973), as well as a 33% lower magnesium content in coronary arteries of sudden-death cardiac cases in soft-water regions (Crawford and Crawford, 1967).

Thus, there is compelling evidence to support the involvement of waterborne magnesium in a cardio-protective role. The beneficial effects of magnesium on the heart, along with the adverse effects of an inadequate magnesium intake, are well documented (Simon, 1963; Marier, 1968; Holtmeier, 1969; Szelenyi, 1973) and make it a prime candidate to explain the «water factor» (Seelig and Heggveit, 1974; Marier, 1976; Chipperfield and Chipperfield, 1977; Anderson, 1977; Burch and Giles, 1977). Clinically, magnesium has been found to have a vasodilator effect in hypertensive

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patients, and also increases cardiac output in all patients (Mroczek et al., 1977; Szelenyi, 1973), and is used in the stabilization of cardiac rhythmity (Chadda et al., 1972, 1973, 1976, 1977; Singh et al., 1975, 1976a, 1976b, 1976c; Babaknia and Niebyl, 1978).

But, in spite of the above-cited documentation, the magnesium hypothesis has yet to gain widespread acceptance among those studying the water-hardness interrelations. The objections focus on two main points:

(i) waterborne magnesium does not make a significant contribution to daily total magnesium intake;
(ii) the absence of overt signs of magnesium deficiency among the general population is a contra-indication that waterborne magnesium serves as an important means of supplementation.

This presentation will attempt to shed further light on the overall situation.

* * *

To assess the impact of any waterborne component, some idea of its contribution to total daily intake is required. Figure 1 presents the type of data that is very difficult to find in the scientific literature: it plots daily magnesium intake as a function of water hardness in various regions. Data for 3 of the 4 regions were obtained from the study by Leverton et al (1961), and represent magnesium intake from self-selected diets in Alabama (6 subjects), Minnesota (7 subjects), and Nebraska (7 subjects); the other value was obtained from Greger’s (1977) survey of (34) subjects in Indiana. All of the subjects were women, aged 19 to 24 in the study by Leverton et al, and aged 70 to 81 in the study by Greger. The water hardness data are taken from Schroeder’s (1960) compilation, and represent State-wide averages; this was the only data readily available.

Although the day-to-day variability in intake of magnesium from self-selected diets is large (± 23%), a linear regression line was drawn through the various points that represent «average magnesium intake», and this allows an estimation of the contribution made by waterborne magnesium (Fig. 1). Note that, at «zero» water hardness, the trend indicates a daily magnesium intake of 238 mg, but that the intake can be 306 mg/day at a water hardness of 400 (i.e., where hardness is expressed as mg CaCO₃/L); on this basis, a water with a hardness of 400 contributes 68 mg of additional magnesium per day.

In Table I, calculations obtained from the regression equation (Fig. 1) are intercompared with calculations based on water composition per se. Note the good agreement between the values obtained by both methods of calculation. Note also that each 100 units of hardness contributes 6-to-7% of the estimated total magnesium intake per day; thus, a water with a total hardness of 400 contributes 24.5% of the magnesium intake, and this is in fairly good agreement with the 27% contribution that Hankin et al. (1970) reported for a water hardness of 347. In Table I, the factor of 0.33 (i.e., for the Molar contribution of magnesium to total hardness) is close to the average of 0.34 that Schroeder (1960) reported for 163 U.S. metropolitan areas, and this indicates that the waters of the 4 regions depicted in Figure 1 are of this general compositional type.
Figure 1: Daily magnesium intake as a function of drinking-water hardness.

Table II illustrates the diversity that can be encountered in hard waters from different regions. In particular, note that the 25 U.S. cities with the lowest death-rates from coronary disease (Schroeder, 1966) are proportionately richer-than-average in their magnesium content, and therefore contribute significantly more magnesium per unit of water hardness; in fact, these waters resemble the California hard waters studied by Hankin.
### TABLE I

Comparison of two calculations for estimation of daily magnesium intake including the contribution by waterborne Mg

<table>
<thead>
<tr>
<th>Total Hardness of Water</th>
<th>Estimated* Mg intake mg/day</th>
<th>Estimated Mg hardness ppm Mg</th>
<th>Mg hardness from 2.4 L** water/day (mg)</th>
<th>Total daily Mg intake (water + Baseline) mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>238</td>
<td>—</td>
<td>0</td>
<td>238</td>
</tr>
<tr>
<td>100</td>
<td>255</td>
<td>33</td>
<td>8.0</td>
<td>19 (= 7.4%)</td>
</tr>
<tr>
<td>200</td>
<td>272</td>
<td>66</td>
<td>16.0</td>
<td>38 (= 13.9%)</td>
</tr>
<tr>
<td>300</td>
<td>289</td>
<td>99</td>
<td>24.0</td>
<td>57 (= 19.6%)</td>
</tr>
<tr>
<td>400</td>
<td>306</td>
<td>133</td>
<td>32.0</td>
<td>76 (= 24.5%)</td>
</tr>
</tbody>
</table>

* Mg intake, mg/day = (0.17 x Hardness) + 238.
** A daily water intake of 2.4 L (in all forms) is in agreement with the findings of ICRP (1970), Spencer et al. (1970), and Schroeder et al. (1969, p. 830).

### TABLE II

Compositional diversity of waters from different regions

<table>
<thead>
<tr>
<th>Authors and Year</th>
<th>Locale</th>
<th>Total Hardness (i.e. T.H.)</th>
<th>Ca (mg/L)</th>
<th>Mg (mg/L)</th>
<th>Molar Mg relative Total Hardness</th>
<th>Mg intake from 2.4 L water mg/day... /100 T.H.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schroeder (1960)</td>
<td>163 U.S. metropolitan areas</td>
<td>118</td>
<td>31.1</td>
<td>9.75</td>
<td>0.34</td>
<td>23.4</td>
</tr>
<tr>
<td>Schroeder (1966)</td>
<td>25 U.S. cities with lowest death rates from coronary disease</td>
<td>125</td>
<td>22.5</td>
<td>16.5</td>
<td>0.55</td>
<td>39.6</td>
</tr>
<tr>
<td>Hankin et al. (1970)</td>
<td>18 California hard waters</td>
<td>312</td>
<td>58.5</td>
<td>40.0</td>
<td>0.55</td>
<td>96.0</td>
</tr>
<tr>
<td>Godwin and Brown (1973)</td>
<td>California hard water</td>
<td>72.0</td>
<td>58.5</td>
<td>40.0</td>
<td>0.55</td>
<td>96.0</td>
</tr>
<tr>
<td>Anderson et al. (1975)</td>
<td>3 Ontario hard waters</td>
<td>421</td>
<td>119.4</td>
<td>29.4</td>
<td>0.29</td>
<td>70.6</td>
</tr>
<tr>
<td>Crawford et al. (1968)</td>
<td>5 Ontario soft waters</td>
<td>33</td>
<td>8.9</td>
<td>2.8</td>
<td>0.33</td>
<td>5.7</td>
</tr>
<tr>
<td></td>
<td>9 British soft waters</td>
<td>31</td>
<td>8.5</td>
<td>2.5</td>
<td>0.32</td>
<td>6.0</td>
</tr>
<tr>
<td></td>
<td>6 British hard waters</td>
<td>293</td>
<td>102.0</td>
<td>9.25</td>
<td>0.13</td>
<td>22.2</td>
</tr>
</tbody>
</table>


et al. (1970). In comparison, the Ontario waters studied by Anderson et al. (1975) are comparable to the average for U.S. metropolitan areas (Schroeder, 1960), as are the soft British waters studied by Crawford et al. (1968). However, note that Crawford et al.'s hard waters are relatively poor in magnesium; thus, these very hard British waters contribute the same low amount of magnesium calculated for the 163 U.S. metropolitan areas (Schroeder, 1960). This may explain why the British have been unable to find any significant correlation with waterborne magnesium in their water-hardness surveys; however, the lack of information on (non-water) intake of dietary magnesium in Britain leaves this entire question open. In summary, the contribution that waterborne magnesium makes to total magnesium intake is dependent on the characteristics of the waters in a particular region. Nevertheless, it is obvious that North American hard waters (i.e., hardness > 100) generally contribute about 23 mg of dietary magnesium per day, but that this waterborne contribution can be about 2, 3, or 4 times (or more) greater, depending on the degree of water hardness and/or the compositional makeup of waters containing a higher-than-normal proportion of magnesium (Table II).

In a comprehensive review of human magnesium requirements Seelig (1964) recommended an intake of 6 mg/kg/day, and this is equivalent to a daily intake of 360 or 420 mg by persons weighing 60 or 70 kg, respectively. However, Manalo et al. (1967) have calculated that the daily magnesium requirement for a 70 kg man ranges from 265 to 350 mg. In short-term balance studies, Leverton et al.'s (1961) data reveal a daily magnesium requirement of 320 mg for young women, whereas the data of Jones et al. (1967) indicate a requirement of 280 mg/day in a mixed population of adults. Overall, it would seem that a reasonable estimate of magnesium requirement in adults is about 300 mg/day, or somewhat higher.*

Schroeder et al. (1969) have suggested that a magnesium intake as low as 200 mg/day might be adequate for adults; however, in a recent East German review, Fehlinger and Seidel (1977) conclude that such a low intake should be considered «minimal», i.e., it probably represents a mere subsistence level.

In Table III, it can be seen that the magnesium intake observed in the U.S.A. and Europe falls considerably short of the daily requirements cited by the various authors. As discussed by Seelig (1964), Caddell (1972), Marier (1975), and Fehlinger and Seidel (1977), and as illustrated by Schroeder et al. (1969), Schroeder (1971), and Hamilton and Minski (1972/3), the reasons for the widespread dietary magnesium inadequacy are largely attributable to the loss of magnesium during processing of food staples, e.g.,

<table>
<thead>
<tr>
<th>Process</th>
<th>% magnesium lost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refining of flour from wheat</td>
<td>80</td>
</tr>
<tr>
<td>Polishing of rice</td>
<td>83</td>
</tr>
<tr>
<td>Production of starch from corn</td>
<td>97</td>
</tr>
<tr>
<td>Extraction of white sugar from molasses</td>
<td>99</td>
</tr>
</tbody>
</table>

* Jones et al. (1967) and Schroeder et al. (1969) have cautioned against the practice of expressing magnesium requirement on a «per kg body weight» basis, because the magnesium required by normal and obese persons is quasi-identical.
### TABLE III
Surveys of the dietary magnesium intake in various regions of the world

<table>
<thead>
<tr>
<th>Authors and Year</th>
<th>Country</th>
<th>Person surveyed</th>
<th>Mg requirement* mg/day</th>
<th>Mg intake mg/day</th>
<th>Intake Requirement %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leverton et al. (1961)</td>
<td>U.S.A.</td>
<td>30 young women</td>
<td>320 5.3</td>
<td>279 (average)</td>
<td>87</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>174 (lowest)</td>
<td></td>
</tr>
<tr>
<td>Holtmeier and Kuhn (1972)</td>
<td>West Germany</td>
<td>Dietary survey of 1852 persons</td>
<td>360 5.1</td>
<td>235 (average)</td>
<td>65</td>
</tr>
<tr>
<td></td>
<td></td>
<td>34 Elderly women</td>
<td></td>
<td>283 (average)</td>
<td></td>
</tr>
<tr>
<td>Greger (1977)</td>
<td>U.S.A.</td>
<td></td>
<td>300 5.0</td>
<td>162 (lowest)</td>
<td>94</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>251 (average)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>190 (lowest)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>31 Elderly men</td>
<td>350 5.0</td>
<td>4.2</td>
<td>54</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>Mohamed (1976) Sweden</td>
<td></td>
<td>The daily intake of magnesium is remarkably low, especially in women pensioners, as compared to the internationally accepted dietary daily allowance.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* As cited by the various authors.

Note that, in Table III, magnesium intakes as low as 162 mg/day have been observed, and average intakes range from 235 to 283 mg/day. In Figure 1, the linear regression indicated a «baseline» magnesium intake (at «zero» water hardness) of 238 mg/day, and this is very close to the average intake of 235 mg/day that Holtmeier and Kuhn (1972) reported in West Germany. In Table II, it has been shown that hard waters can contribute from 39 to 96 (or more) mg of magnesium per day, and it can therefore be appreciated that waterborne magnesium can «make the difference» between inadequacy and sufficiency.

Three further comments are warranted, concerning waterborne magnesium, i.e.,

(i) Arnaud’s (1977) autoradiographic study of magnesium uptake from sulfated mineral water has indicated that 40 to 50% of the waterborne magnesium is absorbed. In comparison, only 33% of the magnesium in a normal diet is absorbed (cf Burch and Giles, 1977). It is therefore possible that hard-water magnesium is «more available» than an equivalent amount of dietary magnesium; if so, this would imply a greater metabolic significance for the proportion of magnesium contributed by water.
(ii) Epidemiological studies should include consideration of the extent to which water-softening devices are used in a given locality. Failure to allow for this factor may obscure an otherwise-detectable interrelation.

(iii) A beneficial effect of waterborne magnesium care only be expected where the magnesium intake from other sources is inadequate.

* * *

The lack of obvious evidence of widespread magnesium deficiency in human populations has led some authors (Jones et al., 1967; Schroeder et al., 1969) to conclude that the dietary magnesium intake is generally adequate, and they have therefore seen no need for supplementation. These authors were referring to acute magnesium deficiency (see Fehlinger and Seidel, 1977); however, it is likely that a much more subtle form of magnesium insufficiency is prevalent in the modern-day world.

The current dietary intake of magnesium has been termed «marginally inadequate» (Anderson, 1977), whereas the degree of magnesium deficiency has been called «chronic low-grade» (Burch and Giles, 1977) and «sub-acute» (Neri et al., 1975). It is therefore not surprising that there have been no overt signs of magnesium insufficiency among the general population.

As illustrated by Masironi (1972) and Anderson (1978), today's world is confronted by an «epidemic» of sudden-death ischemic heart disease which afflicts people in the 25-to-50 yr. age-group and accounts for approximately one-half of all deaths in middle-aged men (Anderson, 1977). In 1969, Anderson et al proposed that the «water factor» relates entirely to sudden-death cardiac seizures which occur because the myocardium itself can become overly vulnerable to infarction; this proposal elicited favorable comment from Schroeder (1969), and there is now considerable evidence to support it, i.e.,

(i) Sudden-death cardiac subjects were found to have lower magnesium levels in heart muscle, in comparison with those who had died from other causes (Chipperfield and Chipperfield, 1973; Behr and Burton, 1973);

(ii) Sudden-death cardiac cases in a soft-water locality had a lower magnesium level in coronary arteries, in comparison with similar subjects from a hard-water region (Crawford and Crawford, 1967);

(iii) Myocardial magnesium concentrations were lower in soft-water areas than in hard-water regions, whether the cause-of-death was cardiac-related or not (Anderson et al., 1975).

(iv) Myocardial tissue can become selectively depleted of magnesium, whereas the magnesium content of skeletal, diaphragm, and pectoralis muscles remains unchanged (Behr and Burton, 1973; Seelig and Heggtveit, 1974; Anderson et al., 1975), and no change may be seen in serum and/or erythrocyte magnesium concentration (Chaparwal et al., 1971; Zieve, 1975; Anderson, 1977).

The consequences of magnesium depletion (especially at the cardiac site) are an increased likelihood of cardiac arrhythmia, calcification, necrosis, and infarction (Hoitmeier, 1969; Szelenyi, 1973; Seelig and Heggtveit,
1974; Marier, 1976; Burch and Giles, 1977). It is relevant to note that, of the two main intracellular cations, magnesium — but not potassium — has been effective in the stabilization of cardiac arrhythmia (Chadda et al., 1972), in the prevention of adrenalin-induced cardiopathy (Savoie, 1971), and in maintaining myocardial integrity during cardiac arrest (Kalmar et al., 1975; Tyers, 1975). The beneficial role of magnesium has also been discussed in the enhancement of thiamin utilization (Zieve, 1975), and in the prevention of histamine shock during the «sudden unexpected death» syndrome (Caddell, 1972).

As for an underlying mechanism that might explain the role of magnesium insufficiency in sudden-death cardiac seizures, it can be suggested that the earliest site-of-action (and probably the most critical) is within the heart mitochondria (Heggtevit et al., 1964; Seelig and Heggtevit, 1974; Jacobus et al., 1975; Crompton et al., 1976; Ryan and Ryan, 1977). It is possible that the heart mitochondria are the organelles that respond most readily to an inadequate magnesium intake, thereby tending to become selectively depleted of magnesium, with consequent impairment of the heart’s ability to respond to a sudden surge of stimulus (e.g., adrenalin). Future work may show that dietary magnesium requirements should be based on the maintenance of mitochondrial integrity. Meanwhile, these words by Héroux et al. (1977) can be used to summarize the current situation, i.e.

«There is a level of magnesium deficiency which is characterized by few overt signs of the deficiency, but which — over the long term — results in a decline in homeostatic potential.»

* * *

ABSTRACT — The reported inverse correlation between water hardness and incidence of cardiac fatalities has led to tentative hypotheses concerning the role of magnesium intake in this phenomenon. The present article reviews the evidence in favor of the magnesium hypothesis, with emphasis on differentiating between diet-derived and water-borne magnesium intake, and their respective cardio-protective potentials.


REFERENCES


SCHROEDER, H. A. (1971): "Losses of Vitamins and Trace Minerals, resulting from


