Quantifying the Role of Magnesium in the Interrelationship between Human Mortality/Morbidity and Water Hardness

J.R. Marier\textsuperscript{a}, L.C. Neri\textsuperscript{b}

\textsuperscript{a}Biological Sciences Division, National Research Council of Canada, and
\textsuperscript{b}Epidemiology Division, University of Ottawa, Ottawa, Canada

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**Abstract.** An attempt has been made to quantify the effect of waterborne magnesium on human mortality/morbidity, based on epidemiological and clinical observations reported in several regions of the modern-day world. A consistent pattern has emerged, indicative of a global phenomenon, which illustrates the importance of waterborne magnesium in protecting against cardiovascular trauma and other ailments. These findings attest to the inadequate metabolic magnesium status among modern-day humans, especially those who reside in ultra-soft-water localities.

The vital role of magnesium for optimal cardiac function has recently been reviewed [5, 11]. Similarly, the correlation between human health and drinking water hardness has been the subject of a comprehensive report issued by the National Research Council of Canada [15], in which it was concluded that the intake of dietary magnesium is suboptimal in the modern-day world, and that waterborne magnesium can make a sizeable contribution to the total daily intake of magnesium in some hard-water regions. The purpose of this presentation is to attempt to quantify the effect of waterborne magnesium on human mortality/morbidity, based on observations reported in several regions of the modern-day world.

\textit{Muss} [17] used the data of \textit{Schroeder} [20] to conduct a state-by-state intercomparison throughout the continental United States, and thereby derived the following equation for the linear interrelation between total cardiovascular (CV) mortality and the total hardness (TH) of drinking water:

\[
\text{CV deaths/100,000} = 905.5 - (0.916 \times \text{TH}).
\]

On this basis, \textit{Muss} [17] concluded that a TH of 100 would reduce CV mortality by 10.1% among white males, age 45–64 years.
This interrelation can be interpreted in terms of waterborne magnesium, because Schroeder [20] reported the average chemical composition of drinking water in 163 metropolitan centers throughout the continental USA. From this, it can be calculated that water having a TH of 100 contains 8.3 mg/l of magnesium. And, bearing in mind that Mun [17] had associated this with a 10.1% decrease in CV mortality, it can be further calculated that a 10% reduction in CV mortality would be conferred by a waterborne magnesium concentration of 8.2 mg/l.

In South Africa, Leary et al. [10] have recently assessed mortality from ischemic heart disease (IHD) as a direct function of waterborne magnesium concentrations. As illustrated in figure 1, there appear to be two distinct linear trends, and no ready explanation can be offered for this disparity. Linear regression equations were calculated for each trend, and also for the aggregate of all the points shown in figure 1. On this basis, it can be calculated that a 10% reduction in IHD mortality would be conferred by, respectively, a waterborne magnesium concentration of 5.8, 6.2, and 6.1 mg/l.

Note: It has been suggested [19] that exponential curves would be a more realistic manner of presenting the data derived from the US and South African surveys. The point is well taken. However, curvilinear plots do not readily lend themselves to a quantitative assessment of the respective trends. (An exponential
Fig. 2. Approximation of the trend observed in USSR localities, indicating increased prevalence of ECG changes and high BP at lower waterborne Mg concentrations [plotted from the data of ref. 12]. Note: all subjects were females, aged 20-49 years.

This approach is illustrated in figure 2, although the data contained therein will be discussed later in this presentation.

When the linear-regression approach is used, it can be seen that there is a surprising degree of agreement between the US and South African data, i.e., a 10% reduction in CV or IHD mortality can be expected with a waterborne magnesium increment of approximately 8 or 6 mg/l, respectively. Thus, there is an indication that waterborne magnesium may be more effective in protecting against IHD mortality than against total CV mortality. This is compatible with observation made in Canada, where Anderson [3] intercompared soft water (2 mg/l of Mg) and hard-water (30 mg/l) localities in Ontario and found that there were 15-30% more sudden-death IHD fatalities in the soft-water region, most likely attributable to fatal arrhythmic seizures induced by an inadequate magnesium intake.

Further light has been shed on this topic by Levin et al. [12] who studied the influence of drinking water hardness on blood pressure (BP) and electrocardiographic (ECG) changes in USSR populations. The strongest correlations were with waterborne magnesium. Although there was considerable variability in the clinical data found among various population subgroups, it is possible to present a graphic illustration of the overall trend (fig. 2). Note that there was a 3-fold increased prevalence of high arterial BP at the lowest waterborne magnesium level (2.4 mg/l) in comparison with the 'control' locality (26.2 mg/l). Also, at progressively lower levels of waterborne magnesium, there was an increasing frequency of ECG abnormalities until - at very low waterborne magnesium levels - more sinus arrhythmia was also observed among the population. Thus, the data in figure 2 suggest that moderate increments of waterborne magnesium can have a noticeable effect on BP and ECG patterns among long-term residents, and this magnesium-related trend is supported by other recent studies. For example, there is now experimental evidence [1] demonstrating that increasing the severity of magnesium deficiency produces graded elevations of arterial BP. Also, clinical supplementation of magnesium (360 mg/day, equivalent to the RDA requirement) reduced both the systolic and diastolic BP by an average of 13 mm Hg in
hypertensive patients during a 6-month period [4]. Concerning ECG patterns, it has been reported [9] that a bolus oral supplement of 600 mg of magnesium per day for 4 days prevented postsurgical ECG abnormalities; in the same study, it was suggested that ECG patterns might be a means of providing an accurate index of the intracellular myocardial magnesium status in humans. In this connection, recent work in China [21] has shown that the early ECG changes in mild magnesium deficiency are limited to T wave abnormalities, and it was stated that: ‘When neuromuscular irritability exists ... the ECG may serve as an important clue to pointing out the possibility of magnesium deficiency.’

Here, it is appropriate to point out that magnesium-rich mineral waters have recently been used for therapeutic purposes (table I), and that one of the findings involved magnesium-mediated abatement of neuromuscular hyperexcitability. In table I, it can also be noted that the magnesium dosages were in the neighborhood of 100 mg/day, and that clinical improvement was seen within the relatively short timespan of 15 days to 3 months.

All of the aforementioned observations present a consistent pattern, illustrating the importance of magnesium in protecting against cardiovascular trauma and other metabolic aberrations.

In Canada, there has been emphasis on selective depletion of myocardial magnesium which averaged 6% lower in subjects residing in soft-water areas than in individuals residing in hard-water areas [2]. Such a depletion, even if not fatal, represents a considerable proportion of the 12–27% magnesium loss found in non-necrosed myocardial tissue of ischemic and/or sudden-death cardiac fatalities [5, 16], and is felt to be a predisposing factor in ‘sensitizing’ the heart and making it more vulnerable to stress-induced fatal seizures [3, 14, 18]. An approximation of the enhanced risk has been calculated as a function of the myocardial magnesium content at autopsy [8], and this is shown in figure 3. Note that, when the myocardial magnesium decreases from 1,060 to 550 μg/g, the apparent relative risk (solid line) increases by a factor of >100 fold. In figure 3, the dotted line represents an attempt to correct for loss of myocardial magnesium caused by the ischemic/necrotic process per se; on this basis, a myocardial magnesium decrease from 1,220 to 680 μg/g enhances the risk >10-fold.

In the softest water regions of Ontario, Canada, the average 6% shortfall in myocardial magnesium content occurs in both cardiac and noncardiac subjects [2]. Furthermore, it has been estimated that the hardest Ontario waters provide 53 mg more magnesium per day than do the extremely soft waters [3, 16]. Thus, a 53-mg magnesium

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**Table I. Use of magnesium-rich mineral waters for therapeutic purposes**

<table>
<thead>
<tr>
<th>Mg dosage, mg/day</th>
<th>Duration</th>
<th>Result</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>90</td>
<td>15 days</td>
<td>abated neuromuscular hyperexcitability</td>
<td>6</td>
</tr>
<tr>
<td>67–134</td>
<td>3 months</td>
<td>improved condition of asthmatics</td>
<td>7</td>
</tr>
</tbody>
</table>
supplement per day seems to make a 6% difference in the magnesium status of the heart; and, bearing in mind that cardiac fatalities have been associated with an average 18% shortfall of myocardial magnesium [5, 16], then prevention of myocardial magnesium loss might be expected by providing $18/6 \times 53 = 159$ mg of supplementary magnesium per day. This is very close to the daily supplement dosages previously advocated [15], although the question arises: 'What would this mean in terms of waterborne magnesium?'

An assessment of dietary magnesium intake in 4 diverse regions of the continental USA has indicated [14] that drinking water having a TH of 100 would contribute 17 mg of magnesium per day. At the outbreak of this presentation, we have already seen that such waters tend to contain 8.3 mg/l of magnesium. Therefore, in this context, the intake of water (in all forms, e.g., coffee, tea, soups, reconstituted beverages etc.) is $17/8.3 = 2$ liters per day. From this, it follows that the aforementioned 159 mg/day magnesium supplement would require a communal water supply containing $159/2 = 80$ mg/l of magnesium.

Note: Such water suppliers are uncommon, and this is why alternate modes of supplementation have also been discussed [16].

In the previously discussed epidemiological data from the USA and South Africa, a 10% decrease in cardiac-related mortality was conferred by a waterborne magnesium increment of 8 or 6 mg/l, respectively. With an aggregate water intake of 2 liters per day, this is equivalent to a waterborne magnesium contribution of 16 or 12 mg per day. A simi-
lar calculation can be applied to other water-related observations discussed in this presentation.

Thus, the fact that the waterborne magnesium factor has been observed in the USA, South Africa, the USSR, and Canada, is indicative of a global phenomenon which reflects the inadequate metabolic magnesium status in several regions of the modern-day world. As previously discussed [15, 16], factors that contribute to an impoverished magnesium status include an over-dependency on refined dietary stables, alcohol abuse, chronic digitalis usage, and long-term diuretic therapy.

One further comment can be made about waterborne magnesium. It had been suggested [14] that waterborne magnesium may be approximately one third better absorbed than dietary magnesium, and recent research in Holland indicates that this may indeed be the case [13]. If this finding receives extensive corroboration, this would mean an enhanced metabolic effectiveness of waterborne magnesium, i.e., greater than an equivalent dosage of dietary magnesium. However, this aspect must await clarification by future research.

L’évaluation quantitative du rôle du magnésium dans l’interrelation entre la mortalité/la morbidité humaine et la dureté de l’eau


References


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J.R. Marier,
Biological Sciences Division,
National Research Council of Canada,
Ottawa, K1A OR6 (Canada)