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Scientific Advisory Committee on Nutrition

SACN STATEMENT ON HARD WATER AND CARDIOVASCULAR DISEASE

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Introduction

1. Softened or desalinated spring and bottled drinking waters in the UK must meet a minimum statutory requirement for hardness which is based on epidemiological evidence suggesting that hard water may reduce cardiovascular disease (CVD) risk. Calcium (Ca) and magnesium (Mg) are the main minerals that give water its hardness and the suggested protective effect of hard water on CVD risk has been attributed to its Ca and Mg content.

2. This paper reviews the evidence for an association between water hardness and cardiovascular health. It follows a request from the Food Standards Agency for advice on whether there is sufficient scientific evidence to support the continued provision for a statutory minimum hardness for spring and bottled drinking water which has been softened or desalinated.

Background

Hard water

3. Water hardness is a measure of the concentration of minerals dissolved in water, primarily Ca and Mg, as bicarbonates, sulphates and chlorides. Hard water is formed when water passes through limestone or chalk and Ca and Mg ions dissolve in the water. Ca concentration is an indicator of the hardness level which is usually expressed in terms of the equivalent quantity of calcium carbonate ($CaCO_3$) in milligrams per litre or parts per million (British Water, 2001; United Utilities, 2007).

4. The World Health Organization (WHO) (2006) defines water hardness as "*a measure of the concentration of divalent cations that contribute to scale formation and soap curd. In most water supplies, Ca and Mg are the predominant contributors to hardness. Water considered to be moderately hard contains about 60-120 mg/L as calcium carbonate*".

Regulatory requirements for bottled water

5. There are three categories of bottled water: natural mineral water[1], spring water[2] and bottled drinking water[3]. Under Directive 2009/54/EC, natural mineral water may not be

[1] Natural mineral water is water originating in an underground water table, deposit or aquifer, which emerges or is extracted from a source tapped at one or more natural or bore exits. It must come from an officially recognised spring, be microbiologically wholesome

subject to any treatment that changes the essential characteristics of the water. In the UK, spring and bottled drinking water may undergo treatment in order to meet the chemical and microbiological limits.

6. The original requirement for a minimum hardness of water for human consumption was controlled by European legislation (Directive 80/778/EEC). In 1998, this Directive was replaced by Directive 98/83/EC which removed the requirement for minimum hardness from EU legislation. Consequently, several Member States do not have equivalent national provisions to the UK.

7. The UK retained the requirement for minimum hardness as a national provision in SI 1999 No. 1540 (as amended), as well as its replacement SI 2007 No. 2785 (as amended), on health grounds, based on advice from COMA[4] and Department of Health officials (see paragraphs 11-13). The existing wording for the minimum hardness provision was retained in the latter UK legislation, i.e., the status quo (*in the case of water prepared from water which has been softened or desalinated, its hardness is not below a minimum concentration of 60 mg Ca/L),* which, in effect, permits remineralisation. Parallel regulations have applied to Scotland, Wales and Northern Ireland.

8. In 2003, the European Commission organised a public consultation, the Drinking Water Seminar[5], to allow stakeholders to give their views on the revision of Directive 98/83/EC. The following comments were made regarding calcium (and magnesium)*: "a minimum limit for calcium in water that has been softened or desalinated is supported, but this could also be done on a case by case basis according to some. Setting a minimum level for naturally soft waters is another matter. Health benefits need further attention".*

9. The Food Standards Agency requested an updated opinion from SACN on the available epidemiological evidence regarding the role of water hardness, with reference to Ca and Mg, so that it can review the existing provisions and contribute to discussions within the Community. The Commission has indicated that the issue of water hardness may be considered during the review of the Drinking Water Directive.

Scientific basis for regulatory requirement

10. In 1974, the COMA panel on Diet and Cardiovascular Disease advised caution on proposals to soften water supplies (Department of Health and Social Security, 1974). In 1984, in its report on '*Diet and Coronary Heart Disease'* (Department of Health [DH], 1984*),* COMA noted that an EEC directive due to be implemented in 1985, would require a minimum hardness for water which had been artificially softened and advised that *'individual members of the public who soften water in their own homes may wish to take the precaution of drinking unsoftened water'*.

(i.e. free of parasitic, pathogenic harmful micro-organisms) and have been protected from all risk of pollution. The composition and characteristics of the water must remain stable.

[2] Water can only be labelled with the description "spring water" if it originates in an underground water source, bottled at source) and satisfies most of the exploitation conditions, microbiological criteria and some of the labelling requirements that apply to natural mineral water. Spring water must also comply with limits laid down in Schedule 3 for physical, chemical and microbiological parameters. Unlike natural mineral water, water that is labelled as spring water does not have to be officially recognised.

[3] Bottled drinking water means drinking water which is bottled, and is neither described as 'spring water' or a recognised natural mineral water. It can come from a variety of sources, including municipal supplies.

[4] Committee on Medical Aspects of Food Policy.

[5] Scientific synthesis report. Drinking water seminar. 27 and 28 October 2003. Available at http://circa.europa.eu/Public/irc/env/drinking_water_rev/library?l=/drinking_seminar_27-28/dws_final_report/_EN_1.0_&a=d

11. In 1994, as part of their review on '*Nutritional Aspects of Cardiovascular Disease*' (DH, 1994), COMA noted that studies since 1981 '*have not altered the balance of the evidence and the explanation for the association remains unknown'*. COMA concluded that in view of the consistency of evidence it remained prudent not to soften drinking water supplies.

12. Subsequent advice (Natural Mineral Water**,** Spring Water and Bottled Drinking Water Regulations 1999, 2003, 2004) from DH officials was that, because the properties of water hardness that provide the benefits to cardiovascular health are not known, the aim should be to retain rather than reintroduce minerals. The intention of this advice was to take a precautionary approach by preventing the supply of water which has been excessively artificially softened, whether or not it is then treated to "re-harden" it.

13. In 2005 the WHO published a report, '*Nutrients in Drinking Water',* which considered the relationship between hard water and CVD risk (WHO, 2005). The report concluded that: *'on balance, the hypothesis that consumption of hard water is associated with a somewhat lowered risk of CVD was probably valid, and that Mg was the more likely contributor of those benefits*'.

14. In 2006, the WHO convened a meeting of experts on the possible protective effect of hard water against CVD. A report of this meeting, '*Calcium and Magnesium in Drinking Water: Public Health Significance',* was published in 2009 (WHO, 2009). The report concluded that *"the epidemiological evidence for the water hardness-cardiovascular disease hypothesis is still not proven. However, at present, the balance of epidemiological evidence supports the link between magnesium and cardiovascular mortality…..not removing magnesium from drinking water, or in certain situations increasing the magnesium intake from water, may be beneficial, especially for populations with an insufficient dietary intake of the mineral".*

15. The meeting identified key knowledge gaps and made a number of research recommendations. Knowledge of local dietary intake of Ca and Mg from both food and water sources relative to needs was considered to be fundamental to decisions about whether water may be a useful source of these nutrients. The research recommendations included the need for well designed analytical epidemiological studies (case-control or cohort) to elucidate the health implications of waterborne Ca and Mg. It was recommended that such studies should assess consumption of Ca and Mg both from the diet and from water, collect data on cardiovascular risk factors, and that study populations should include a range of drinking water concentrations to provide a range of Ca and Mg exposures.

Physiological role of magnesium and calcium

16. Mg is closely involved with Ca in metabolism. Ca homeostasis is controlled in part by an Mg requiring mechanism which releases parathyroid hormone (DH, 1991).

Magnesium

17. Mg is as a cofactor for over 300 enzyme reactions, many of which are involved in energy metabolism. It is also involved in protein and nucleic acid synthesis, is essential for the mineralisation and development of the skeleton, and plays a role in cellular permeability and neuromuscular excitability (Saris *et al*, 2000).

18. Mg deficiency is characterised by progressive muscle weakness, failure to thrive, neuromuscular dysfunction, tachycardia, ventricular fibrillation, coma and death (DH, 1991). Because of these cardiac effects, low dietary intake of Mg has been postulated as a risk factor for coronary heart disease (CHD). Deficiency of Mg has also been implicated in hypertension and type 2 diabetes (Saris *et al,* 2000).

Calcium

19. The main function of Ca is structural and approximately 99% is found in bones and teeth. The remaining 1% is found in tissues and fluids where it is required for the regulation of a number of enzymes and hormonal responses, blood clotting, nerve transmission, vascular contraction and vasodilatation, muscle contraction, and glandular secretion. Ca deficiency leads to a decrease in bone mineral content and mass that results in a weaker bone structure, leading to increased risk of fractures.

Absorption and bioavailability of calcium and magnesium from mineral water

20. The bioavailability of Ca and Mg is reported to be higher from drinking water than from food (Couzy *et al,* 1995; Durlach *et al,* 1985). This is because Ca and Mg are usually present in water as free ions and are therefore more readily absorbed compared to food, where they are mostly bound to other substances (WHO, 2005)**.**

21. Absorption of Ca from mineral water was found to be comparable to that of Ca from milk (Couzy *et al,* 1995; Heaney, 2006) which has been reported to be about 30% (Heaney *et al*, 1988). Verhas *et al* (2002) reported a bioavailability rate of 59% for Mg which lies in the upper reported range for absorption from foods. Sabatier *et al* (2002) reported that Mg absorption from mineral water was 46% when consumed alone and was significantly greater when consumed with a meal (52%).

Dietary sources of calcium and magnesium in the UK

Dietary sources of calcium

22. The National Diet and Nutrition Survey (NDNS) of adults aged 19-64 years (Henderson *et al,* 2003) reports that the main source of Ca in the diet is milk and milk products which contribute 43% of total intake. As white flour is fortified with calcium, cereal and cereal products (including bread and breakfast cereals) are another important source, contributing 30% of total intake. Dietary supplements make a very small contribution to total Ca intake (about 4% for women; negligible for men).

23. For adults aged 65 years and over (Finch *et al,* 1998), the main source of Ca in the diet is also milk and milk products (providing about 50% of total intake for free living adults and institutionalised adults). Cereal and cereal products contributed 25% and 31% towards total intake for free-living and institutionalised adults respectively. In free-living individuals, the contribution of dietary supplements to total Ca intake was 1% for women and negligible for men. For institutionalised individuals, dietary supplements contributed to 1% of total Ca intake for women aged 85 years and over but only made a negligible contribution for other women and men.

Dietary sources of magnesium

24. The main dietary source of Mg for adults aged 19-64 years are cereals and cereal products providing 27% of total intake and drinks[6] which contributed 20% to the total intake for men and 13% for women. For men, half the contribution made by drinks was from beer and lager (10% compared to 2% for women) and coffee contributed 4% of Mg intake for men and women. The contribution from milk and milk products was 11%. Dietary supplements contributed to no more than 2.5% of mean intakes.

25. For free-living and institutionalised adults aged 65 years and over, cereals and cereal products are the main source of Mg in the diet providing 35% of total intake for free living adults and 37% for institutionalised adults. Milk and milk products contributed 15% of total intake for free-living adults and 21% of total intake for institutionalised adults. The contribution made by drinks was 11% for free-living adults and 7% for institutionalised adults.

Dietary intakes of calcium and magnesium in the UK

26. Information on Ca and Mg intakes of British adults has been obtained from the NDNS series (Henderson *et al,* 2003; Finch *et al,* 1998) and the Low Income Diet and Nutrition Survey (LIDNS) (Nelson *et al,* 2007). Information on Ca and Mg status is not available from the NDNS or LIDNS.

Current recommendations for calcium and magnesium intake

27. For adults, the reference nutrient intake (RNI[7]) for Ca is 700 mg/d and the lower reference nutrient intake (LRNI[8]) is 400 mg/d (DH, 1991).

28. The RNI for Mg is 300 mg/d for men and 270 mg/d for women; the LRNI is 190 mg/d for men and 150 mg/d for women (DH, 1991).

Calcium intake in the UK

Adults aged 19-64 years

29. In the NDNS, mean daily intakes were 1007 mg for men and 777mg for women; 2% of men and 5% of women had intakes below the LRNI.
30. In the LIDNS, mean daily intakes were 906 mg for men and 711 mg for women; 8% of men and 13% of women had mean daily intakes below the LRNI.

Adults aged 65 and over

31. In the NDNS, mean daily intakes of free living adults were 836 mg for men and 690 mg for women; 5% of men and 9% of women had intakes below the LRNI. For institutionalised individuals, mean daily intakes were 953 mg for men and 861 mg for women; 1% of women and less than 0.5% of men had intakes below the LRNI.

32. In the LIDNS, mean daily intakes were 834 mg for men and 725 mg for women; 4% of men and 8% of women had intakes below the LRNI.

[6] Includes soft drinks, alcoholic drinks, tea, coffee and water.
[7] The RNI represents the amount of a nutrient likely to meet the needs of 97.5% of the population.
[8] The LRNI represents the amount of a nutrient likely to meet the needs of 2.5 % of the population.

Magnesium intake

Adults aged 19 to 64 years

33. In the NDNS, mean daily intakes were 308 mg for men and 229 mg for women; 9% of men and 13% of women had an intake below the LRNI. A significantly higher proportion (about 20%) of women aged 19-34 years had intakes below the LRNI.

34. In the LIDNS, mean daily intakes were 262 mg for men and 196 mg for women; 25% of men and 26% of women had intakes below the LRNI.

Adults aged 65 years and over

35. In the NDNS, mean daily intakes of free-living adults were 254 mg for men and 197 mg for women; 21% of men and 23% of women had intakes below the LRNI. Mean daily intakes of institutionalised individuals were 215 mg for men and 188 mg for women; 39% of men and 22% of women had intakes below the LRNI.

36. In the LIDNS, mean daily intakes were 229 mg for men and 188 mg for women; 36% of men and 26% of women had intakes below the LRNI.

Current intakes of bottled water

37. Information on the consumption of bottled drinking water of the British population is provided in the NDNS; however, information on the contribution of Ca and Mg from bottled drinking water to total Ca and Mg intakes is not available. In the LIDNS, bottled water consumption is not separated from tap water consumption.

38. The NDNS shows that mean daily intakes of bottled drinking water[9] for adults aged 19-64y were 61 g for women and 54 g for men. In free living adults aged 65y and over, mean daily intakes of bottled water were 2 g for men and 8 g for women. Bottled drinking water was not consumed by institutionalised adults aged 65y and over.

39. In 2006, natural mineral water accounted for 66% of the UK bottled water market with spring water and bottled drinking waters making up 21% and 13% respectively (Zenith 2006).

40. In 2004, the proportion of UK adults drinking bottled water was reported to be 51% (Zenith, 2006). Young adults aged 15-24 years were the highest consumers of bottled still water (61%). The trend continued in 2005, with 55% of adult aged 15 and over consuming bottled water (Zenith 2006).

Overview of the evidence on water hardness and CVD

41. A number of dietary and lifestyle factors can affect the risk of developing CVD. The major risk factors include smoking, obesity, hypertension, physical inactivity, raised LDL cholesterol, and alcohol consumption (WHO, 2003).

42. There is a large body of epidemiological evidence (mainly ecological and case-control studies carried out in the USA, Europe, and Japan) suggesting a protective association between drinking water hardness and CVD mortality.

[9] Includes still and carbonated mineral water, but excludes sweetened drinks and tonic water.

43. The protective effects have been attributed to Ca and Mg which are both found in hard water and may play a role in the aetiology of CVD. Although the evidence is inconsistent, some studies have suggested that Ca and Mg may have significant effects on reducing blood pressure (Dickinson *et al*, 2006a; Dickinson *et al*, 2006b) which is a major risk factor for CVD (Prospective Studies Collaboration, 2002); so it is possible that the postulated effects of hard water in reducing CVD mortality are mediated through lowering of blood pressure. Animal studies have shown that Mg deficiency may be associated with cardiac irritability, atherosclerosis, and cardiac arrhythmias (Marx and Neutra, 1997).

44. It has also been suggested that the apparent benefits associated with consumption of hard water could be due to the absence of a harmful constituent in soft water, since soft waters are corrosive and could contain toxic metals such as cadmium and lead leached from plumbing materials (WHO, 2005).

Meta-analyses and reviews

45. The evidence for an association between hard water and CVD risk was reviewed by the WHO in 2005 and updated in 2009 (WHO, 2005, 2009). Over 80 epidemiological studies (from 1957 to 2005) were considered. Based on the studies between 1979 and 2004 (19 ecological, 7 case-control, and 2 cohort) it was concluded that there was little evidence to support an association between water hardness or Ca concentration in drinking water and CVD, however, a lower than recommended intake of Mg could increase the risk of dying from, and possibly developing, CVD, stoke or hypertension. The 2009 review, which updated the epidemiological evidence after 1978 (20 ecological, 7 case-control, and 2 cohort studies), concluded that *"the epidemiological evidence for the water hardness-cardiovascular disease hypothesis is still not proven. However, at present, the balance of epidemiological evidence supports the link between magnesium and cardiovascular mortality"*.

46. A review by Monarca *et al* (2006) concluded that there was little evidence that CVD risks are associated with water hardness or Ca concentration; however, there was some evidence suggesting low intakes of Mg may increase the risk of dying from and possibly developing CVD.

47. A systematic review of analytical observational studies (Catling *et al*, 2008), which included 9 case-control studies and 3 cohort studies, concluded that: there was significant evidence of a protective effect of increasing magnesium concentrations in drinking water and CVD mortality based on a meta-analysis of 5 of the case-control studies; no association between water hardness and CVD mortality; and that the evidence for calcium was unclear. There was insufficient evidence to examine drinking water hardness, Ca, or Mg concentrations and CVD morbidity.

48. The studies considered in the WHO reports and systematic reviews (Monarca *et al*, 2006; Catling *et al*, 2008) are summarised below.

Ecological studies

49. From 1957 to 1978, more than 60 ecological studies on water hardness and CVD mortality were published worldwide. A protective association of water hardness with CVD mortality was reported in many, but not all, studies. Most studies did not consider

confounding factors and, those that did, provided insufficient information to evaluate possible confounding.

50. Out of the 20 ecological studies published after 1978, which were reviewed in the WHO report (2009), only 2 (Nerbrand *et al,* 1992; 2003) considered some of the major risk factors for CVD. Ten studies found a significant protective association between water hardness and CVD mortality. In studies where Ca and Mg were evaluated separately, similar associations with CVD mortality were found for each. Six studies found a very small or no association with CVD mortality.

51. One study (Nerbrand *et al,* 2003) which reported higher CVD mortality rates in areas with soft water, found that total Ca and Mg intakes at an individual level (n=207) were actually higher in the soft water areas due to greater intakes of foods containing Ca and Mg. Of the investigated risk factors for CVD, the low density lipoprotein concentrations were higher for individuals in the soft water areas which could explain the higher CVD mortality rates.

Case control studies *(Table 1, Appendix)*

52. The review by Catling *et al* (2008) (see paragraph 47) included 9 case-control studies. Eight of these studies investigated the association of Ca and Mg concentrations in drinking water with CVD mortality (Luoma *et al*, 1983; Rubenowitz *et al*, 1996, 1999, 2000; Yang, 1998; Yang & Chiu, 1999; Rosenlund *et al*, 2005; Yang *et al*, 2006); 1 study (Luoma *et al*, 1983) included cases that were either alive or deceased following acute myocardial infarction (AMI) so it was not possible to consider differential effects on morbidity and mortality risk. Five of the remaining 7 studies found a significant protective effect of Mg levels in drinking water on mortality risk from AMI (Rubenowitz *et al*, 1996, 1999, 2000), stroke (Yang, 1998) or hypertension (Yang & Chiu, 1999). Five studies found no significant association between Ca concentration and CVD mortality with no consistent direction of association (Rubenowitz *et al*, 1996; 2000; Yang, 1998; Yang & Chiu, 1999, Rosenlund *et al*, 2005); 2 studies found a significant protective effect of Ca on AMI mortality risk (Rubenowitz *et al*, 1999; Yang *et al*, 2006). Only 1 study considered drinking water hardness and CVD mortality and reported no significant association (Comstock, 1971).

53. Two studies considered CVD morbidity (Luoma *et al*, 1983; Rosenlund *et al*, 2005) and reported no significant association between Mg or Ca in the drinking water and AMI (Luoma *et al*, 1983; Rosenlund *et al*, 2005) or water hardness (Rosenlund *et al*, 2005).

54. A meta-analysis of 5 of the 7[10] case-control studies which had examined CVD mortality and drinking water Mg or Ca (Catling *et al,* 2008), found that drinking water Mg concentrations in the highest exposure range (8.3-19.4 mg/L) compared with the lowest exposure range (2.5-8.2 mg/L) were significantly associated with a decreased CVD mortality risk (odds ratio, 0.75; 95% CI, 0.68-0.82; $p<0.001$); heterogeneity between studies was moderate. A summary estimate was not calculated for drinking water Ca concentration and CVD mortality as there was a high degree of heterogeneity between studies.

[10] The study by Rosenlund et al (2005) was not included as the exposure data (mg/d) was not comparable with the other studies (mg/L); the study by Rubenowitz *et al* (2000) was not included as data were not presented for drinking water Ca concentration.

Cohort studies *(Table 2, Appendix)*

55. The review by Catling *et al* (2008) identified 3 cohort studies in 5 publications (Punsar *et al*, 1975; Punsar & Karvonen, 1979; Comstock 1979; Comstock *et al*, 1980; Morris *et al*, 2001). Punsar & Karvonen (1979) followed men (n=1711) from 2 rural areas of Finland for 15 years and reported proportionally higher mortality from CHD (14.7% vs 8.7%; p<0.001) in areas with lower Mg concentrations in drinking water (3.1 mg/L vs 13.1 mg/L). Comstock (1979) reported stroke mortality and Comstock *et al* (1980) reported arteriosclerotic heart disease mortality in a cohort of men and women in the USA (n=30,534) followed over 12 years; no association was found between water hardness (0 vs 200 mg/L $CaCO_3$) and stroke mortality or arteriosclerotic heart disease mortality. The studies by Punsar & Karvonen (1979), Comstock (1979), and Comstock *et al* (1980) did not take account of major CVD risk factors.

56. Morris *et al* (2008), updating their previous analysis (Morris *et al*, 2001), reported findings from 25 years follow-up of a cohort of men (n=5796) from 24 towns in the UK with wide variations in levels of hardness in drinking water (0.27-5.28 mmol/L $CaCO_3$). Ca and Mg intakes at an individual level were estimated from a subsample of participants (n=947).

57. At town level, after adjustment for age, water hardness was found to have a significant protective effect on CVD incidence (hazard ratio [HR] for twofold increase in hardness, 0.94; 95% CI, 0.89-0.98); p=0.007); after further adjustment for potential confounding variables (cholesterol, body mass index, smoking, physical activity, alcohol, social class, and height) the association became weaker (HR, 0.96; 95% CI, 0.91-1.01; p=0.08). No association was found with CHD incidence (HR, 0.99; 95% CI, 0.94-1.04; p=0.62) or CHD mortality (HR, 0.96; 95% CI, 0.90-1.02; p=0.18).

58. At an individual level, after adjustment for age and 7 confounding variables (see previous paragraph), the HR for a twofold increase in water hardness was not statistically significant for CHD incidence (HR, 0.99, 95% CI, 0.94-1.04; p=0.62), CVD incidence (HR, 0.96; 95% CI, 0.91-1.01; p=0.08) or CHD mortality (HR not provided). No significant association was found between individual Ca intake from water and CHD incidence (HR, 1.06; 95% CI, 0.97-1.17; p=0.19), CVD incidence (HR, 1.04; 95% CI, 0.97-1.12; p=0.26), or CHD mortality (HR, 1.01; 95% CI, 0.84-1.22; p=0.89). Increased Mg intake from water was associated with an increase in CHD incidence (HR, 1.10 per twofold increase; 95% CI, 1.01-1.20; p=0.045) and CVD incidence (HR, 1.06 per twofold increase; 95% CI, 0.99-1.14; p=0.087) but not with CHD mortality (HR, 1.09; 95% CI, 0.91-1.31; p=0.33).

Intervention studies

59. No intervention studies have investigated the effect of hard water on CVD outcomes.

60. One intervention study (Rylander *et al*, 2004) examined the effect of 3 different waters containing varying levels of minerals (including Mg, Ca, sodium, potassium, sulphate, bicarbonate, chloride, fluoride, and silica) on blood pressure, which is an important risk factor for CVD (Prospective Studies Collaboration, 2002). The three different waters were: (i) water low in minerals (containing 2 mg/L Mg and 68 mg/L Ca); (ii) Mg enriched water (containing 82 mg/L Mg and 4 mg/L Ca); and natural mineral water (containing 84 mg/L Mg and 486 mg/L Ca as well as higher quantities of the other minerals). Subjects (n=70; aged 45-64 years) consumed at least 1 L/day of their allocated water for a

period of 4 weeks. No differences in blood pressure were found between groups consuming the different types of waters.

61. Subsequent analyses excluded individuals (n=15) with serum or urine values for Mg or Ca above the 75th percentile on the basis that they had sufficient intakes of these minerals and would not be influenced by the intervention. No association was found between blood pressure and consumption of the water low in minerals or the Mg enriched water. However, there was a significant decrease in both systolic and diastolic blood pressure in the group consuming the natural mineral water (containing higher quantities of Ca and several other minerals) from 156.8/91.7 mm Hg at baseline to 150.1/88 mm Hg at 2 weeks and 150.4/89.1 mm Hg at 4 weeks. The authors advised caution in the interpretation of the study due to the small number of participants.

Summary

62. Most ecological studies suggest a protective effect of hard water on CVD mortality risk.

63. Evidence from case-control studies suggests a protective association between CVD mortality and concentration of Mg but not Ca in drinking water.

64. Prospective epidemiological data are limited. The available data from these studies do not provide consistent evidence of a protective effect of hard water, Mg, or Ca on CVD morbidity or mortality.

Levels of magnesium and calcium associated with benefits

65. One of the problems in comparing studies is the various ways in which water exposures are reported. Water hardness is expressed in various units, including mg/L $CaCO_3$ and parts per million (ppm). Definition of water hardness units can also differ by country; for example, French degrees (Fd^o; 1Fd^o = 10 $CaCO_3$ml/L) or German degrees (^{o}dH; 1 ^{o}dH = 7.1 mg Ca or 4.3 mg Mg). Some studies assessed water exposure in terms of hardness units whilst others measured concentrations of Ca and Mg.

66. COMA reported that the size of the protective effect was small, most clearly observed at water hardness levels below 170 mg/L ($CaCo_3$), and that increases in water hardness above this level were not associated with further reductions in mortality risk (DH, 1994).

67. The WHO report (2005) noted that exposure-response information was limited but that several authors had suggested that reduced CVD mortality and other health benefits may be associated with minimum levels of approximately 20-30 mg/L Ca and 10 mg/L Mg.

68. A meta-analysis of case-control studies (Catling *et al,* 2008) (see paragraph 54) reported that Mg concentrations between 8.3-19.4 mg/L were associated with a decreased risk of CVD mortality.

Conclusions

69. The available data do not provide consistent evidence for a beneficial effect of hard water, or drinking water Ca or Mg concentration, on CVD risk.

70. Epidemiological evidence suggesting that hard water protects against CVD risk is largely based on ecological studies that considered population exposures to hard water and

mortality statistics. Most of these studies had a number of limitations e.g., lack of exposure at the individual level and risk of exposure misclassification. They also do not take account of possible variations in water over time or within an area.

71. Evidence from epidemiological studies investigating the association between hard water and CVD risk at an individual level is limited.

72. The majority of case-control studies have generally indicated that increased Mg concentration has a protective effect on CVD mortality and that there is no association between Ca concentration and CVD mortality. There is insufficient evidence from case-control studies to assess the effect of hard water on CVD mortality or the effect of hard water, Ca or Mg concentration on CVD morbidity risk.

73. Very few cohort studies have investigated the association between hard water and CVD risk. Results from these studies do not provide consistent evidence for any benefit of water hardness or calcium or magnesium concentration on CVD risk. There are insufficient data from cohort studies to reach clear conclusions on the association between drinking water quality and CVD risk.

74. An important limitation in most case-control and cohort studies is measurement of hard water consumption. Most studies used an ecological measure of drinking water quality and did not quantify individual consumption. This means that the exposure variable assigned to individuals in the study is not a reliable reflection of their exposure. Another important limitation is that most studies did not consider the major risk factors for CVD.

75. Data from the NDNS and LIDNS indicate low calcium intakes for free-living women aged 65 years and over (9%) and low income men and women aged 19-65+ years (8-13%). A relatively high proportion of all age, sex, and income groups have low magnesium intakes (20% of women aged 19-34 years; 25% of low income men and women aged 19-64 years; 21%-39% of free-living and institutionalised adults aged 65 years and over; 36% and 26% of low income men and women aged over 65 years respectively).

76. Food is the principal source of Mg and Ca in the UK. Information is not available on the contribution of bottled water to total dietary intakes of Mg and Ca or whether the amounts obtained from bottled waters make a significant contribution to total intakes.

Recommendations

77. There is currently insufficient evidence suggesting beneficial effects of hard water on CVD risk to support the retention of a statutory minimum hardness for bottled water which has been softened or desalinated.

References

British Water. Water Hardness: Description and Measurement. *Information on water treatment in the home.* 2001. Fact sheet 10.

Catling LA, Abubakar I, Lake IR, Swift L, Hunter PR. A systematic review of analytical observational studies investigating the association between cardiovascular disease and drinking water hardness. *J Water Health*. 2008; (4):433-42.

Comstock GW. Water hardness and cardiovascular diseases. *Am J Epidemiol.* 1979; 10(4):375-400.

Comstock GW, Cauthen GM, Helsing KJ. Water hardness at home and deaths from arteriosclerotic heart disease in Washington County, Maryland. *Am J Epidemiol.* 1980; 112:209-16.

Couzy F, Kastenmayer P, Vigo M, Clough J, Munoz-Box R, Barclay DV. Calcium bioavailability from a calcium- and sulfate-rich mineral water, compared with milk, in young adult women. *American Journal of Clinical Nutrition*. 1995; 62(6):1239-44.

Department of Health and Social Security. *Diet and Coronary Heart Disease. Report of the panel on Diet in relation to Cardiovascular and Cerebrovascular Disease.* Report on Health and Social Subjects *7*. London: HMSO, 1974.

Department of Health and Social Security. *Diet and Cardiovascular Disease. Report of the Panel on Diet in relation to Cardiovascular Disease.* Report on Health and Social Subjects 28. London: HMSO, 1984.

Department of Health. Dietary References Values for Food Energy and Nutrients for the United Kingdom. Report on Health and Social Subjects 41. London: HMSO, 1991.

Department of Health. *Nutritional Aspects of Cardiovascular Disease.* Report on Health and Social Subjects *46*. London: HMSO, 1994.

Dickinson HO, Nicolson DJ, Cook JV, Campbell F, Beyer FR, Ford GA, Mason J. Calcium supplementation for the management of primary hypertension in adults. *Cochrane Database Syst Rev.* 2006a April 19; (2):CD004639.

Dickinson HO, Nicolson DJ, Campbell F, Cook JV, Beyer FR, Ford GA, Mason J. Magnesium supplementation for the management of essential hypertension in adults. *Cochrane Database Syst Rev.* 2006b July 19; 3:CD004640.

Durlach J, Bara M, Guiet-Bara A. Magnesium level in drinking water and cardiovascular risk factor: a hypothesis. *Magnesium.* 1985; 4(1):5-15.

Finch S, Doyle W, Lowe C, Bates CJ, Prentice A, Smithers G, Clarke PC. *The National Diet and Nutrition Survey: People Aged 65 Years and Over. Vol. 1: Report of the Diet and Nutrition Survey.* London: TSO, 1998.

Heaney RP, Weaver CM, Recker RR. Calcium absorbability from spinch. *Am J Clin Nutr.* 1988; 47(4):707-9.

Heaney RP. Absorbability and utility of calcium in mineral waters. *Am J Clin Nutr.* 2006; 84(2):371-4.

Henderson L, Irving K, Gregory J, Bates CJ, Prentice A, Perks J, Swan G, Farron M. *The National Diet and Nutrition Survey: adults aged 19 to 64 years. Volume 3: Vitamin and mineral intake and urinary analytes*. London: TSO, 2003.

Luoma H, Aromaa A, Helminen S, Murtomaa H, Kiviluoto L, Punsar S, Knekt P. Risk of myocardial infarction in Finnish men in relation to fluoride, magnesium and calcium concentration in drinking water. *Acta Med Scand.* 1983; 213(3); 171-6.

Marx A, Neutra RR. Magnesium in drinking water and ischemic heart disease. *Epidemiol Rev.* 1997; 19(2):258-72.

Monarca S, Donato F, Zerbini I, Calderon RL, Craun GF. Review of epidemiological studies on drinking water hardness and cardiovascular diseases. *Eur J Cardiovasc Prev Rehabil.* 2006; 13(4):495-506.

Morris RW, Whincup PH, Lampe FC, Walker M, Wannamethee SG, Shaper AG. Geographic variation in incidence of coronary heart disease in Britain: the contribution of established risk factors. *Heart.* 2001; 86(3):277-83.

Morris RW, Walker M, Lennon LT, Shaper AG, Whincup PH. Hard drinking water does not protect against cardiovascular disease: new evidence from the British Regional Heart Study. *Eur J Cardiovasc Prev Rehabil.* 2008; 15(2):185-9.

Natural Mineral Water, Spring Water and Bottled Drinking Water Regulations (SI No. 1540). London: HMSO, 1999.

Natural Mineral Water, Spring Water and Bottled Drinking Water (Amendment) (England) Regulations (SI No. 666) London: HMSO, 2003.

Natural Mineral Water, Spring Water and Bottled Drinking Water (Amendment) (England) Regulations (SI No 656) London: HMSO, 2004.

Nelson M, Erens B, Bates B, Church S, Boshier T. *Low Income Diet and Nutrition Survey. Volume 2: Food consumption and nutrient intake*. London: TSO, 2007.

Nerbrand C, Svärdsudd K, Ek J, Tibblin G. Cardiovascular mortality and morbidity in seven counties in Sweden in relation to water hardness and geological settings. The project: myocardial infarction in mid-Sweden. *Eur Heart J.* 1992; 13(6):721-7.

Nerbrand C, Agréus L, Lenner RA, Nyberg P, Svärdsudd K. The influence of calcium and magnesium in drinking water and diet on cardiovascular risk factors in individuals living in hard and soft water areas with differences in cardiovascular mortality. *BMC Public Health.* 2003; 3:21.

Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet.* 2002; 360:1903–13.

Punsar S, Erämetsä O, Karvonen MJ, Ryhänen A, Hilska P, Vornamo H. Coronary heart disease and drinking water. A search in two Finnish male cohorts for epidemiologic evidence of a water factor. *J Chronic Dis*. 1975; 28(5-6):259-87.

Punsar S, Karvonen MJ. Drinking water quality and sudden death: observations from West and East Finland. *Cardiology.* 1979; 64(1):24-34.

Rosenlund M, Berglind N, Hallqvist J, Bellander T, Bluhm G. Daily intake of magnesium and calcium from drinking water in relation to myocardial infarction. *Epidemiology.* 2005; 16(4):570-6.

Rubenowitz E, Axelsson G, Rylander R. Magnesium in drinking water and death from acute myocardial infarction. *Am J Epidemiol.* 1996; 143(5):456-62.

Rubenowitz E, Axelsson G, Rylander R. Magnesium and calcium in drinking water and death from acute myocardial infarction in women. *Epidemiology.* 1999; 10(1):31-6.

Rubenowitz E, Molin I, Axelsson G, Rylander R. Magnesium in drinking water in relation to morbidity and mortality from acute myocardial infarction. *Epidemiology.* 2000; 11(4):416-21.

Rylander R, Arnaud MJ. Mineral water intake reduces blood pressure among subjects with low urinary magnesium and calcium levels. *BMC Public Health.* 2004; 4:56.

Sabatier M, Arnaud MJ, Kastenmayer P, Rytz A, Barclay DV. Meal effect on magnesium bioavailability from mineral water in healthy women. *Am J Clin Nutr.* 2002; 75(1):65-71.

Saris NE, Mervaala E, Karppanen H, Khawaja JA, Lewenstam A. Magnesium. An update on physiological, clinical and analytical aspects. *Clin Chim Acta.* 2000; 294(1-2):1-26.

United Utilities (2007). Water Hardness. *Key facts on water quality.*

Verhas M, de la Guéronnière V, Grognet JM, Paternot J, Hermanne A, Van den Winkel P, Gheldof R, Martin P, Fantino M, Rayssiguier Y. Magnesium bioavailability from mineral water. A study in adult men. *Eur J Clin Nutr.* 2002; 56(5):442-7.

World Health Organization. *Guidelines for Drinking Water Quality* 3rd ed. International Programme of Chemical Safety. Geneva. WHO, 2003.

World Health Organization. *Nutrients in Drinking Water. Water Sanitation and Health Protection and the Human Environment. The World Health Report*. Geneva: WHO, 2005.

World Health Organization. Meeting of Experts on the Possible Protective Effect of Hard Water against Cardiovascular Disease. Washington DC. WHO, 2006.

World Health Organization. *Calcium and magnesium in drinking water. Public health significance.* Geneva: WHO, 2009.

Yang CY. Calcium and magnesium in drinking water and risk of death from cerebrovascular disease. *Stroke.* 1998; 29:411-14.

Yang CY, Chiu HF. Calcium and magnesium in drinking water and the risk of death from hypertension. *Am J Hypertens.* 1999; 12(9):894-9.

Yang CY, Chang CC, Tsai SS, Chiu HF. Calcium and magnesium in drinking water and risk of death from acute myocardial infarction in Taiwan. *Environ Res*. 2006; 101(3):407-11.

Zenith International Ltd. *Zenith Report on UK Bottled Waters*. Zenith, 2006.

APPENDIX

Table 1: Case-control studies on the relationship between CVD and hardness and/or calcium/magnesium concentrations of drinking water *(adapted from WHO, 2009)*

Reference	Country, area, year	Cases	Controls	Age (years)	Drinking water parameters	Odds Ratio (95% CI)
Comstock (1971)	USA, Maryland; 1967-1968	189 M deaths from arteriosclerotic heart disease deaths	378 M population controls *Matched by age*	45-65	Water hardness range 0-450 ppm $CaCO_3$ Mean water hardness 0-99 ppm Mean water hardness 150+ ppm	RR unadjusted 0.78 1.35
Luoma *et al* (1983)	Finland; south-eastern region; 1974-1975	58 M with AMI alive or dead	50 M (hospital controls) *Matched by age & type of community* 50 M (population controls) *Matched by age & municipality*	37-64	 Ca concentration <16 mg/L 16-18 mg/L 19-20 mg/L >20 mg/L Mg concentration <1.2 mg/L 1.2-1.5 mg/L 1.6-3.0 mg/L >3.0 mg/L	OR unadjusted Hosp controls / OR unadjusted Population controls 0.73 (0.22-1.99) 0.56 (0.25-1.28) 0.77 (0.30-1.91) 1.07 (0.48-2.42) 0.91 (0.35-2.36) 1.64 (0.73-3.85) Reference Reference 2.00 (0.69-6.52) 4.67 (1.30-25.32)* 1.11 (0.41-3.10) 2.29 (0.88-6.58) 1.00 (0.36-3.08) 1.63 (0.62-4.52) Reference Reference
Rubenowitz *et al* (1996)	Southern Sweden, 17 municipalities; 1982-1989	854 M dead from AMI	989 M dead from cancer	50-69	 Ca concentration <34 mg/L 34-45 mg/L 46-81 mg/L ≥82 mg/L Mg concentration <3.6 mg/L 3.6-6.8 mg/L 6.9-9.7 mg/L ≥9.8 mg/L	OR age-adjusted Reference 0.88 (0.65-1.19) 0.84 (0.64-1.10) 1.06 (0.82-1.38) Reference 0.88 (0.66-1.16) 0.70 (0.53-0.93)* 0.65 (0.50-0.84)*

Reference	Country, area, year	Cases	Controls	Age (years)	Drinking water parameters	Odds Ratio (95% CI)
Yang (1998)	Taiwan, China, 252 municipalities with single water source; 1989-1993	17,133 M + F dead from stroke	17,133 M + F dead from other causes, excluding CVD *Matched by sex, year of birth, year of death*	50-69	 Ca concentration <24.4 mg/L 24.4-42.3 mg/L 42.4-81.0 mg/L Mg concentration <7.3 mg/L 7.4-13.4 mg/L 13.5-41.3 mg/L	OR adjusted for age, sex, urbanisation level, Mg concentration Reference 1.5 (0.99-1.11) 0.95 (0.88-1.01) Adjusted as above & for Ca concentration Reference 0.75 (0.65-0.85)* 0.60 (0.52-0.70)*
Rubenowitz *et al* (1999)	Southern Sweden, 16 municipalities; 1982-1983	378 F dead from AMI	1368 F dead from cancer	50-69	 Ca concentration ≤31 mg/L 32-45 mg/L 46-69 mg/L ≥70 mg/L Mg concentration ≤3.4 mg/L 3.5-6.7 mg/L 6.8-9.8 mg/L ≥9.9 mg/L	OR adjusted for age and Mg Reference 0.61 (0.39-0.94)* 0.71 (0.49-1.02) 0.66 (0.47-0.94)* OR adjusted for age and Ca Reference 1.08 (0.78-1.49) 0.93 0.64-1.34) 0.70 (0.50-0.99)*
Yang and Chiu (1999)	Taiwan, China, , 252 municipalities with single water source; 1990-1994	2336 M + F dead from hypertension	2336 M + F dead from other causes, excluding CVD *Matched by sex, year of birth, year of death*	50-69	 Ca concentration 4.0-11.3 mg/L 11.4-30.0 mg/L 30.1-37.7 mg/L 37.8-53.5 mg/L 53.5-81.0 mg/L Mg concentration 1.5-3.8 mg/L 3.9-8.2 mg/L 8.3-11.1 mg/L 11.2-16.3 mg/L 16.4-41.3 mg/L	OR adjusted for age, sex, urbanisation & Mg Reference 1.23 (0.94-1.62) 1.32 (0.98-1.78) 1.12 (0.83-1.51) 1.26 (0.92-2.02) OR adjusted for age, sex, urbanisation & Ca Reference 0.73 (0.57-0.93)*** 0.66 (0.50-0.87)*** 0.67 (0.50-0.89)*** 0.63 (0.47-0.84)***

Reference	Country, area, year	Cases	Controls	Age (years)	Drinking water parameters	Odds Ratio (95% CI)
Rubenowitz *et al* (2000)	Southern Sweden, 18 municipalities; 1994-1996	263 M + F dead from AMI	258 M + F dead from other causes	50-74	Ca concentration,0-235 mg/L	OR adjusted for age & Mg (highest vs lowest quartile) 0.89(0.59-1.33)
					Mg concentration, 0-44 mg/L	OR adjusted for age & Ca (highest vs lowest quartile) 0.64 (0.42-0.97)*
		823 M + F surviving after AMI	853 M + F without AMI		Ca concentration, 0-235 mg/L	OR adjusted for age & Mg (highest vs lowest quartile) 0.97(0.78-1.21)
					Mg concentration, 0-44 mg/L	OR adjusted for age & Ca (highest vs lowest quartile) 1.16 (0.93-1.45)*
Rosenlund *et al* (2005)	Sweden (1992-1994)	497 M + F with AMI 677 M + F without AMI (controls)	677 M + F without AMI	45-70		OR adjusted for age, sex, smoking, hypertension, DM, SES, physical activity, job stress (95% CI)
					Ca intake from tap water (mg/d) <42.4 >42.3	 1.00 1.07 (0.62-1.85)
					Mg intake from tap water (mg/d) <6.9 >6.9	 1.00 1.07 (0.63-1.82)
Yang *et al* (2006)	Taiwan, China, , 252 municipalities with single water source; 1994-2003	10,094 M + F deaths from AMI	10,094 M + F deaths from other causes Matched by sex, year of birth, year of death	50-69	Calcium concentration (mg/L) ≤24.4 25.1-42.4 42.6-81.0	OR adjusted for age, sex, urbanisation level of residence, and Mg concentration of water 1.00 0.79 (0.73-0.86) 0.71 (0.65-0.77)
					Mg concentration (mg/L) ≤7.7 7.8-13.5 14.1-41.3	OR adjusted for age, sex, urbanisation level of residence, and Ca concentration of water 1.00 1.00 (0.93-1.08) 1.09 (0.99-1.19)

AMI, acute myocardial infarction; BMI, body mass index; Ca, calcium; CI, confidence interval; CVD, cardiovascular diseases; DM, diabetes mellitus; F, females; M, males; Mg, magnesium; OR, odds ratio; SES, socioeconomic status

* P<0.05; *** P<0.001; all others not statistically significant

Table 2: Cohort studies of the relationship between cardiovascular diseases and drinking water hardness and calcium and magnesium levels
(adapted from WHO, 2009)

Study (year)	Country	Population (n)	Follow-up (years)	Cause of death	Adjustments	Drinking water parameters	Outcome measure
Punsar and Karvonen (1979)	FINLAND	504 M west Finland 622 M east Finland 49-59 years	15	CHD		Mg concentration West Finland: 12.7 mg/L East Finland: 3.3 mg/L (Sub-area median concentrations of water assigned to residents)	49/504 (9.7%) CHD deaths 95/622 (15.3%) CHD deaths Relative risk: east vs west = 1.6 (p<0.01)
Comstock *et al* (1980)	USA	30, 534 M + F >25 years	12	AHD	Smoking, socioeconomic status, duration of residence, marital status, church attendance, education	Water hardness 0 ppm vs 200 ppm (median water hardness for each district assigned to residents)	Relative risk: M:7+ y residence 0.69 (NS) M:<7 y residence 0.86 (NS) F: 7+y residence 1.06 (NS) F<7 y residence 1.73 (NS) Total: 1.02 (NS)
Morris *et al* (2008)	UK	5796 M from 24 towns 40-59 years	25	CHD	Age, cholesterol,BMI, smoking, physical activity, alcohol, social class, height	Water hardness at town level (0.27 -5.28 mmol/L) Water intake of Ca at individual level (n=705) *(concentration not given)* Water intake of Mg at individual level (n=705) *(concentration not given)*	Hazard ratio per 2-fold increase: 0.96 (0.90-1.02); p=0.89 1.01 (0.84-1.22); p=0.89 1.09 (0.91-1.31); p=0.33

AHD, arteriosclerotic heart disease; CHD, coronary heart disease, NS, not significant