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ORIGINAL ARTICLE

# Dietary magnesium intake and blood pressure: a qualitative overview of the observational studies

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**Published reports of 30 separate sets of analyses from 29 observational studies relating dietary intake of magnesium to blood pressure (BP) were identified through a comprehensive search using MEDLINE and BIDS-EMBASE. Three studies were prospective, 24 cross-sectional (25 reports), of which four also contained a longitudinal component, and two were obtained from baseline data in a trial. Various dietary methodologies were used: 24-h dietary recall ($n = 12$), food-frequency questionnaire (8), food record (7), and duplicate diet (2). Twelve reports compared magnesium intake or BP level between subgroups. Seven showed a negative association between magnesium intake and BP level, and five reported no association. From 18 of the 30 sets of analyses either a regression estimate or a Pearson correlation coefficient was reported. Many reports also allowed identification of subgroups by sex, age and race. Ninety population samples and subgroups could thus be identified from the 30 reports. All 11 Pearson-*r* correlation coefficients reported for systolic BP (SBP) (three significant, $P < 0.05$) and 10 (out of 12) Pearson-*r* correlation coefficients reported for diastolic BP (DBP) (four significant) were negative. Seven reports (13 subgroups for SBP, 11 subgroups for DBP) gave partial regression coefficients after adjustment; 10 (seven significant) and eight (six significant) were negative for SBP and DBP, respectively. For 13 subgroups in five papers, Pearson-*r* correlation coefficients were reported after adjustment for confounding factors. Eight (out of 13) showed a negative relationship for SBP and DBP. This review points to a negative association between dietary magnesium intake and BP. A systematic quantitative overview is needed to reconcile the inconsistencies of the results of individual studies and to quantify the size of such relationship.**

**Keywords:** blood pressure; magnesium; observational study; review

## Introduction

The possible relationship between dietary divalent cations such as calcium and magnesium and blood pressure (BP) follows reports in the 1950s and 1960s of a negative ecological association between drinking water hardness and both cardiovascular and cerebrovascular mortalities.[1–5] However, since intake of these cations from drinking water generally represents only a small proportion of total intake, the role of dietary intake in relation to cardiovascular disease has become the focus of more recent investigation. Epidemiological and clinical research of the association of dietary calcium intake and BP has been extensively reviewed in recent years[6–11] including obtaining pooled quantitative estimates of effect.[7,11] However, less is known of the possible role of dietary magnesium on BP.[9,12,13]

This review was undertaken to re-assess the evidence from observational (mainly cross-sectional and prospective) studies relating dietary magnesium intake to BP in the light of recent reports on this question.

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## Materials and methods

Published reports of observational studies relating dietary intake of magnesium to BP were identified through a comprehensive computerised search using MEDLINE (January 1966 to December 1995) and BIDS-EMBASE (January 1980 to December 1995) searching for the keywords 'magnesium', 'blood pressure' and 'hypertension', through examination of cited reference sources and by personal contact with several investigators who are experts in the field. Papers written in languages other than English were included. A copy of each paper was obtained and relevant data were abstracted independently by two of the authors (SM, FPC). Any discrepancies were reconciled.

### Description of selected papers and population subgroups

Thirty-one published papers from observational studies carried out in 29 different populations worldwide were identified[1a–31a] (Tables 1 and 2). Two papers reporting findings from the Yi people study used the same dataset and are combined for the purpose of this review,[16a–17a] whereas two papers from the Australian children study were treated as independent reports as they contained different analyses and results.[22a,24a] The 30 reports came from the USA ($n = 11$), China (4), Australia (4), Canada (2), Belgium (2), Finland (1), France (1), Greece (1), Japan (1), Netherlands (1), Spain (1), and Sweden (1). Three reports published in languages other than English, ie, Chinese, French and Japanese, were included.[14]

Twenty-three of the reports involved adults,[1a–7a,9a,11a–19a,21a,23a–31a] while five focused on children and adolescents,[10a,20a,22a,24a,26a] one on infants,[19a] and in one the study population was not stated.[8a] Nineteen included males and females,[5a–12a,21a–31a] six males only[1a,13a–18a] and five females only.[2a–4a,19a,20a]

Three studies were prospective, 24 cross-sectional, of which four also contained a longitudinal component, and two were obtained from baseline data in a trial. Sample size of these 30 studies ranged widely from 40 in the study among US elderly[25a] to 58 218 in the US Nurses Study.[3a] The median sample size was 253. Various dietary methodologies were used: 24-h dietary recall ($n = 12$) including single (9), 3 (2) and 7 days (1); food-frequency questionnaire (9) including semiquantitative food-frequency (6); food record (7) and duplicate diet (2).

Most studies controlled for age and body mass index, with varied control for other confounders. Many reports also allowed identification of subgroups by sex, age and race. Ninety population samples and subgroups could thus be identified. Three reports included subgroups stratified by BP level (normotensive/hypertensive)[21a] or by the use of anti-hypertensive medication (including/excluding those who were on anti-hypertensive medication).[23a,30a]

In 12 of the 30 papers, magnesium intake or BP level were compared between subgroups[1a–12a] (Table 1). The remaining 18 studies reported either a regression estimate or a Pearson-*r* correlation coefficient[13a–31a] (Table 2).

## Results

### Analysis of reports which compared subgroups without reporting a regression or correlation estimate

Twelve papers reported some comparison between magnesium intake and BP subgroups without a regression or correlation estimate[1a–12a] (Table 1). Four reports compared dietary magnesium in hypertensive and normotensive persons,[1a,4a,8a,9a] and two compared magnesium intake among people in the upper and lower percentiles of the BP distribution.[2a,10a] One study reported dietary magnesium intake for students with and without a family history of hypertension;[6a] another reported relative risk of hypertension among quintiles of magnesium intake.[3a] Two studies compared BP levels among groups with different dietary intakes,[5a,7a] one report gave mean magnesium intake and BP levels for males and females,[11a] and one for different age groups.[12a] Seven of these 12 studies reported a negative association between magnesium intake and BP and five reported no association (Table 1).

### Analysis of reports which reported a regression or correlation estimate

Forty-six subgroup analyses from 18 studies reported either a regression or a Pearson-*r* correlation coefficient of magnesium intake to BP[13a–31a] (Table 2).

In five subgroups from two papers, simple regression coefficients were reported.[17a,20a] Median regression coefficients were −0.08 mm Hg/100 mg Mg (range −17.0 to +3.5, $n = 5$) for systolic BP (SBP) and −1.68 (−17.8 to +10.1, $n = 5$) for diastolic BP (DBP).

All 11 simple Pearson-*r* correlation coefficients for SBP in 11 subgroups of six reports were negative (three significant, $P < 0.05$).[13a–15a,19a,21a,25a] Ten out of 12 simple correlation coefficients for DBP in 12 subgroups of seven reports were negative (four significant).[13a–15a,19a,20a,24a,25a]

Ten reports (22 subgroups for SBP, 21 subgroups for DBP) applied multivariate analyses to control for covariates[15a–19a,22a–23a,26a–31a] (Table 2).

Seven reports (13 subgroups for SBP, 11 subgroups for DBP) gave partial regression coefficients after adjustment for confounding factors; 10 (seven significant) and eight (six significant) were negative for SBP and DBP, respectively.[16a–18a,22a,23a,26a,28a,29a] One report indicated only significant negative direction of the association between magnesium intake and DBP in young males without reporting a regression or a correlation estimate.[10a]

Thirteen subgroups in five reports gave Pearson-*r* correlation coefficients after adjustment for confounding factors.[15a,19a,27a,30a,31a] Eight of 13 were negative for both SBP and DBP.

These quantitative associations between dietary magnesium and BP are summarised in Table 3. Negative association between magnesium intake and BP was reported from simple regression-correlation analysis in 15 (93.8%) of 16 subgroups for SBP and 14 (82.4%) of 17 for DBP, and significant negative association in four (25%) and six (35.3%), respectively. When adjustment for confounders was considered, negative association was reported in 18 (69.2%) of 26 subgroups for SBP and 16 (66.7%) of 24 for DBP, and significant negative association in nine (34.6%) and eight (33.3%), respectively. One positive association was reported for SBP (6.3%) and three (17.6%) for DBP on simple regression-correlation analysis, and eight for SBP (30.8%) and eight for DBP (33.3%) on multiple regression-correlation analysis. One significant positive association was reported.

**Table 1** Summary of reports which did not present regression or correlation estimates

| *First author (reference)* | *Year* | *Country* | *Population* | *Design* | *Sample size* | *Age (mean or range)* | *Dietary method* | *No. of sub-groups* | *Subgroups* | *Main results* |
|---|---|---|---|---|---|---|---|---|---|---|
| *Males only* | | | | | | | | | | |
| 1. Zemel (1a) | 1988 | US | Blacks and whites | XS | 66 | 49.5 | 4d Record | 4 | Race, NT *vs* HT | Mg in HT < Mg in NT |
| *Females only* | | | | | | | | | | |
| 2. Thulin (2a) | 1980 | Sweden | Females | XSL | 60 | 52 | Duplicate | 2 | BP <30% *vs* 95% < BP | NS |
| 3. Witteman (3a) | 1989 | US | US nurses | Prosp | 58218 | 34–59 | SFFQ | 5 | Mg intake quintiles | HT RR lower with higher Mg |
| 4. Overloop (4a) | 1994 | France | Pregnant females | XSL | 125 | 26.5 | 24h Recall | 4 | 1st & 3rd trimester, NT *vs* HT | in 1st trimester Mg in HT < Mg in NT |
| *Both males and females* | | | | | | | | | | |
| 5. Rouse (5a) | 1983 | Australia | Religious population | XS | 293 | 33.2 | 24h Recall | 6 | Sex, vegetarian groups | BP in high Mg < BP in low Mg |
| 6. Lehtonen (6a) | 1985 | Finland | Students | XS | 147 | 25.1 | 7d Record | 4 | Sex, FH(+) *vs* FH(−) | NS |
| 7. Margetts (7a) | 1986 | Australia | Mild hypertensive | Trial | 58 | 30–64 | 24h Recall | 3 | Diet groups | Mg in high BP < Mg in low BP |
| 8. Fodor (8a) | 1987 | Canada | Newfoundland study | XS | 173 | NA | Duplicate | 2 | NT *vs* HT | Mg in HT < Mg in NT |
| 9. Karanja (9a) | 1987 | US | Normotensive | Trial | 70 | 21–70 | 24h Recall | 4 | Sex, NT *vs* HT | in F, Mg in HT < Mg in NT |
| 10. Martell-Claros (10a) | 1989 | Spain | Terrejon study | XS | 1109 | 14–18 | 7×24h Recall | 2 | BP < 50% *vs* 90% < BP | NS |
| 11. Kesteloot (11a) | 1990 | Belgium | Interuniversity research | XS | 4055 | 50.4 | 1d Record | 2 | Sex | NS |
| 12. Kafatos (12a) | 1993 | Greece | Elderly | XS | 164 | 65–91 | 24h Recall | 6 | Sex, age groups | NS |

XS, cross-sectional; XSL, cross-sectional with longitudinal component; Prosp, prospective; NA, not available; SFFQ, semi-quantitative food-frequency questionnaire; NT, normotensive; HT, hypertensive; FH, family history of hypertension; Mg, magnesium; BP, blood pressure; F, females; RR, relative risk; NS, not significant.

**Table 2** Summary of reports which did present regression or correlation estimates

| *First author (reference)* | *Year* | *Country* | *Population* | *Design* | *Sample* | *Age (mean or range)* | *Dietary method* | *No. of sub-groups* | *Subgroups* | *Reporting* | *Covariates* | *Main results* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Males only* | | | | | | | | | | | | |
| 1. Joffres (13a) | 1987 | US | Honolulu heart study | Prosp | 615 | 46–65 | 24h Recall | 1 | – | r (uni) | – | –SBP, –DBP |
| 2. Zhang (14a) | 1989 | China | Yi and Han farmers | XS | 55 | 48.3 | 24h Recall | 2 | Race | r (uni) | – | NS |
| 3. Ascherio (15a) | 1991 | US | Normotensive | XS | 805 | 40–69 | SFFQ | 1 | – | r (uni, co) | age, BMI, alc, kcal | NS |
| 4. He (16a, 17a) | 1991 | China | Yi people study | XS | 419 | 15–78 | 3×24h Recall | 4 | Race, immigrant | b (uni, co) | age, BMI, HR, alc, kcal | –SBP, –DBP |
| 5. Ascherio (18a) | 1992 | US | White health professionals | Prosp | 26187 | 40–75 | SFFQ | 2 | Entry phase, 4yrs later | b (co) | age, BMI, alc,Ca, K, Fibre | –SBP, –DBP |
| *Females only* | | | | | | | | | | | | |
| 6. McGarvey (19a) | 1991 | US | Maternal offspring study | XSL | 212 | Mothers | SFFQ | 4 | Age | r (uni, co) | K, Ca | –SBP, –DBP |
| 7. Simon (20a) | 1994 | US | Children | XS | 2030 | 9–10 | 3rd Record | 2 | Race | b (uni) | – | in White, –DBP |
| *Both males and females* | | | | | | | | | | | | |
| 8. Johnson (21a) | 1987 | US | Elderly | XS | 62 | 54–81 | FFQ | 6 | Sex, NT, HT | r (uni) | – | NS |
| 9. Jenner (22a) | 1988 | Australia | Children | XS | 884 | 9 | FFQ | 2 | Sex | b (co) | age, weight, height, socio, month of examination, kcal | in M, –DBP |
| 10. Kesteloot (23a) | 1988 | Belgium | Interuniversity research | XS | 8058 | 49 | 24h Recall | 4 | Sex, HT medication | b (co) | age, BMI, HR, alc, kcal | in F, –SBP |
| 11. Vandongen (24a) | 1989 | Australia | Children | XS | 884 | 9 | FFQ | 2 | Sex | p (b)(uni) | – | in M, –DBP |
| 12. Ideno (25a) | 1989 | US | Elderly | XS | 40 | 65–86 | 24h Recall | 2 | Sex | r (uni) | – | –DBP |
| 13. Wu (26a) | 1991 | China | Students | XS | 181 | 12–16 | 3×24h Recall | 2 | FH(+), FH(–) | b (co) | kcal | in FH(+), –DBP, –DBP |
| 14. Hamet (27a) | 1992 | Canada | Montreal study | XS | 182 | 38 | 3d Record | 2 | Sex | r (co) | kcal | NS |
| 15. Tian (28a) | 1995 | China | Tianjin population | XS | 663 | 20–64 | 7d Record | 2 | Sex | b (co) | age, BMI, alc, kcal, Na, K, Ca | NS |
| 16. Van Leer (29a) | 1995 | Netherlands | Dutch population | XSL | 20921 | 20–59 | SFFQ | 2 | Sex | b (co) | age, town, survey year | –SBP, –DBP |
| 17. Itoh (30a) | 1995 | Japan | Islander | XS | 147 | 52.5 | 3d Record | 2 | HT medication | r (co) | age | NS |
| 18. Ma (31a) | 1995 | US | Aric study | XS | 14882 | 45–64 | SFFQ | 4 | Sex, race | r (co) | age, BMI, kcal | in F, –SBP, –DBP |

Prosp, prospective; XS, cross-sectional; XSL, cross-sectional with longitudinal component; SFFQ, semi-quantitative food-frequency questionnaire; FFQ, food-frequency questionnaire; NT, normotensive; HT, hypertensive; FH, family history of hypertension; r, Pearson-*r* correlation coefficient; b, regression coefficient; p(b), *P* value based on regression analysis; uni, univariate; co, covariate; BMI, body mass index; alc, alcohol intake; kcal, energy intake; HR, heart rate; Na, sodium intake; Ca, calcium intake; K, potassium intake; fibre, fibre intake; socio, socioeconomic strata; M, males; F, females; SBP, systolic blood pressure; DBP, diastolic blood pressure; –, significant negative association; NS, not sigificant.

**Table 3** Summary of quantitative associations between dietary magnesium and blood pressure

| | *SBP* | | *DBP* | |
|---|---|---|---|---|
| | *Simple* | *Adjusted* | *Simple* | *Adjusted* |
| Numbers of subgroups | 16 | 26 | 17 | 24 |
| Negative association (*n*) | 15 | 18 | 14 | 16 |
| Proportion (%) | 93.8 | 69.2 | 82.4 | 66.7 |
| Significant negative association (*n*) | 4 | 9 | 6 | 8 |
| Proportion (%) | 25.0 | 34.6 | 35.3 | 33.3 |
| Positive association (*n*) | 1 | 8 | 3 | 8 |
| Proportion (%) | 6.3 | 30.8 | 17.6 | 33.3 |
| Significant positive association (*n*) | 0 | 0 | 0 | 1 |
| Proportion (%) | 0 | 0 | 0 | 4.2 |

## Discussion

This review of observational epidemiological studies is suggestive of a negative association between dietary magnesium intake and BP. This negative association appears to be consistent within and across different studies using a range of methodologies. However, as the majority of studies do not appear to have been designed specifically to examine the association of dietary magnesium with BP, inconsistencies of design, analysis and reporting between studies complicate their interpretation.

First, the methods of dietary data collection and assessment varied across the studies. Precision in the assessment of usual or habitual diet is dependent on the number of days of dietary data collection. In many of the studies reviewed methods were inadequate to classify individuals with any precision within the population distribution of true (habitual) intake, due to the large day-to-day variation within person in dietary consumption including magnesium.[15–19] High intra-individual variation can attenuate the absolute values of regression and correlation coefficients.[20] Thus, any potential association between magnesium intake and BP would tend to be underestimated with bias in the estimates of the regression of correlation coefficient toward a zero value (regression dilution).[20–23]

Second, the high degree of intercorrelation among several nutrients (multicolinearity) makes the interpretation of independent dietary associations with BP difficult, because people eat food, not isolated nutrients.[24–26] Furthermore, the variable precision with which highly correlated nutrients are measured represents an additional potential problem. The interpretation becomes difficult when more than one such nutrient is entered into the multivariate analyses.[8,22,23] There was considerable variation across studies in the control for important potential confounders for any dietary magnesium and BP association. Most studies controlled for sex, age and body mass index in the design or analysis. Control for calorie intake, potassium, calcium and alcohol intake varied. Only one study included sodium as a potential confounder estimated by 24-h urinary sodium excretion.[28a] No study considered protein intake which has been suggested to be negatively related to BP, in both animal experiments[27,28] and in epidemiological studies.[29,30] The analysis of association in different subgroups showed the extent to which adjustment for confounders varied the consistency of the findings.

A related methodological problem is the possible statistical overadjustment of dietary nutrient-BP association for covariates (such as body mass index) that are much more precisely measured, with much less day-to-day variation than dietary nutrients.[31–34] The estimates of the association with BP appeared to be strongly influenced by the inclusion of body mass index in multiple regression models.[33,34]

Third, the use of anti-hypertensive medication may have modified the distribution of BP in the population and may have led to biased estimates of the magnesium-BP relationship, especially as hypertensive persons may have changed their dietary intake as a consequence of the diagnosis of high BP.[35] Only three reports addressed this issue by considering subgroups according to BP status (normotensive/hypertensive)[21a] or anti-hypertensive medication (including/excluding those who were on anti-hypertensive medication).[23a,30a]

Fourth, potential publication bias is inherent to population studies on the association between BP and dietary factors, including magnesium.[36] Twelve of 30 reports identified in our comprehensive search of the literature did not present an estimate of the association but only some unquantified differences between various subgroups. Is it possible that more studies reporting a negative, statistically significant, association might have been published in comparison with those reporting either no relation or a positive association.

In conclusion, the present review points to a negative association between dietary magnesium intake and BP. However, an overall estimate of this association is difficult to obtain because of methodological problems including methods of dietary data collection and assessment, regression-dilution, multicolinearity, biases such as publication bias, and inconsistency of design, analysis, and reporting. A systematic quantitative overview (meta-analysis) would help to reconcile the inconsistencies of the results of individual studies, and to quantify the size of such relationship.

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