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# Hypomagnesaemia is associated with diabetes: Not pre-diabetes, obesity or the metabolic syndrome

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ABSTRACT

*Aims:* The mechanism for the association between diabetes and hypomagnesaemia remains uncertain. This study aimed to test whether hypomagnesaemia is present in pre-diabetes, obesity and the metabolic syndrome.

*Methods:* 1453 adults from randomly selected households from rural Victoria, Australia, attended for biomedical assessment. Serum magnesium concentrations, hypomagnesaemia defined using local laboratory criteria (<0.70 mmol/l), and defined by the bottom quintile of serum magnesium concentrations, were compared in different diabetes pre-cursor states including metabolic syndrome using ATP III criteria.

*Results:* The mean serum magnesium was $0.84 \pm 0.06$ mmol/l and 25 (1.7%) had a low magnesium. Mean magnesium was lower among those with known diabetes than those with new diabetes, impaired glucose tolerance (IGT), impaired fasting glucose (IFG) and normal subjects (0.79 (0.78–0.81) vs 0.83 (0.81–0.86); 0.84 (0.82–0.85); 0.84 (0.82–0.86); 0.85 (0.84–0.85) mmol/l). After adjusting for confounders, and compared with those without diabetes, hypomagnesaemia was 10.51 (1.37–80.60)-fold more common with new diabetes, 8.63 (2.20–33.90)-fold more common with known diabetes, 6.77 (1.75–26.17)-fold more common among those taking anti-hypertensive medication but with no difference to those with IGT/IFG (0.90 (0.10–8.10)).

*Conclusion:* Diabetes is associated with hypomagnesaemia, but not its pre-cursor states.



The prevalence of obesity in Australia has more than doubled in the last 20 years with approximately 60% of the population estimated to be overweight or obese [1]. Obesity, impaired fasting glucose (IFG), impaired glucose tolerance (IGT), the metabolic syndrome and past gestational diabetes (GDM) are known to be risk factors for the development of type 2 diabetes (T2DM). Several authors have demonstrated an association between T2DM and hypomagnesaemia although whether the relationship is causal remains uncertain [2–6].

With its widespread role in ATP metabolism [7], it is conceivable that hypomagnesaemia could contribute to the development of diabetes, but there remains a relative scarcity of data examining the prevalence of hypomagnesaemia in association with diabetes pre-cursor states. We hypothesized that if a low serum magnesium was associated with the risk of developing diabetes, rather than with diabetes itself, or its treatment, we would find relative hypomagnesaemia in one or more of obesity, the metabolic

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**Table 1 – Characteristics of subjects with and without laboratory defined hypomagnesaemia.**

| | Serum magnesium ≥0.70 mmol/l | Serum magnesium <0.70 mmol/l | Significance |
|---|---|---|---|
| N | 1428 | 25 | |
| Age (years) | 53 ± 16 | 63 ± 16 | 0.001 |
| %Female | 56.0% | 76.0% | 0.045 |
| Magnesium (mmol/l) | 0.84 ± 0.06 | 0.65 ± 0.04 | <0.001 |
| Creatinine (μmol/l) | 79 ± 29 | 84 ± 5 | 0.381 |
| Sodium (mmol/l) | 140 ± 2 | 139 ± 3 | 0.012 |
| Potassium (mmol/l) | 4.0 ± 0.3 | 4.1 ± 0.5 | 0.240 |
| Calcium (mmol/l) | 2.33 ± 0.94 | 2.35 ± 0.97 | 0.172 |
| Uric acid (mmol/l) | 33 ± 9 | 35 ± 10 | 0.479 |
| Body Mass Index (mg/m$^2$) | 27.9 ± 5.2 | 27.9 ± 6.9 | 0.972 |
| Blood pressure (mmHg) | | | |
| Systolic | 132 ± 22 | 137 ± 23 | 0.269 |
| Diastolic | 72 ± 10 | 70 ± 10 | 0.385 |
| Total cholesterol (mmol/l) | 5.3 ± 1.0 | 5.2 ± 1.2 | 0.704 |
| LDL-cholesterol (mmol/l) | 3.2 ± 0.9 | 2.9 ± 0.9 | 0.103 |
| HDL-cholesterol (mmol/l) | 1.4 ± 0.4 | 1.5 ± 0.4 | 0.284 |
| Triglyceride (mmol/l) | 1.5 ± 1.1 | 1.8±1.2 | 0.111 |
| Fasting glucose (mmol/l) | 5.4 ± 1.2 | 6.5 ± 2.9 | <0.001 |
| HbA1c (%) | 5.3 ± 0.5 | 6.0 ± 1.4 | <0.001 |
| Aboriginal/Torres Strait Is | 1.1% | 4.0% | 0.184 |
| Metabolic Syndrome by ATPIII criteria | 24.7% | 40.0% | 0.080 |
| High blood pressure | 56.6% | 80.0% | 0.019 |
| Treated hypertension (includes diuretic use) | 23.1% | 72.0% | <0.001 |
| Treated heart disease | 7.1% | 8.0% | 0.869 |
| Gestational diabetes ever | 3.2% | 0% | 0.367 |
| Current smoker | 16.3% | 20.0% | 0.622 |
| Any alcohol | 83.6% | 64.0% | 0.009 |
| Activity (≥150 min/week) | 43.5% | 32.0% | 0.250 |
| Regional centre vs small town | 52.0% | 52.0% | 0.998 |
| Use full fat milk | 41.5% | 44.0% | 0.798 |
| Cut fat off the meat | 31.0% | 25.0% | 0.821 |
| Cut skin off chicken | 44.2% | 48.0% | 0.702 |
| Dairy items ≥twice/day | 61.7% | 60.0% | 0.866 |
| Fruit eaten ≥twice/day | 55.6% | 76.0% | 0.042 |
| Vegetables eaten ≥4 times/day | 32.5% | 52.0% | 0.040 |

syndrome and/or IGT/IFG when compared with the wider population.

## 1. Research design and methods

The study was carried out between June 2001 and March 2003 across the seven main towns in the Goulburn Valley, Victoria, Australia (populations 2094–35,828) as previously described [8]. The district includes major horticulture, viticulture, dairy and food manufacturing industries. The study included a 2-step approach: interviews of all residents in randomly selected households (a ''census'') and then invitation of all usual residents (resident for at least 6 months) aged ≥25 years to attend for a ''clinic''. The initial census of 2376 randomly selected households (half in the regional centre, a 12th in each of the six smaller towns) was undertaken. Houses were revisited until a response was received, including evenings and weekends. Visits were preceded by media releases and information leaflets were dropped off at each house. The approach was based on similar surveys in the United Kingdom and New Zealand [9,10]. Questionnaires relating to personal health were completed for all occupants in face-to-face interviews wherever possible. Occasionally, questionnaires were self-completed, with a follow-up interview to address any gaps or queries. All usual residents (resident for at least 6 months) aged ≥25 years who completed the household survey were then invited to attend, fasting, at a ''clinic'' at a nearby site between 07.00 and 10.00 a.m. Demographic, health and socioeconomic data were collected. Previously validated questions were used to assess physical activity [11], fat item intake [12], smoking, alcohol, vegetable, fruit and dairy food consumption [13] as previously reported [14].

Blood pressure was performed using a Dinamap semiautomatic oscillometric recorder (Critikon, Tampa, FL, USA) in a seated position after participants had rested for at least 5 min. An appropriate cuff size was used. The arm was supported by a table at the level of the heart. Three readings were taken at 1 min intervals. The mean of the first two recordings was recorded. If the difference between the three readings was

**Table 2 – Mean serum magnesium and prevalence of hypomagnesaemia in different, metabolic states.**

| | N | Unadj mean magnesium (Mmol/l) | With hypomagnesaemia (%) | Significance | Mantel Haenszel odds ratio (adjusted for age, sex) |
|---|---|---|---|---|---|
| Normal | 1168 | 0.84 ± 0.06 | 0.9 | <0.001 | 1.00 |
| Impaired glucose tolerance | 98 | 0.84 ± 0.06 | 1.0 | | 0.88 (0.11–7.17) |
| Impaired fasting glucose | 51 | 0.85 ± 0.05 | 0 | | 0 |
| New diabetes | 31 | 0.84 ± 0.09 | 6.5 | | 5.71 (1.18–27.7) |
| Known diabetes | 105 | 0.80 ± 0.08 | 10.5 | | 11.1 (4.27–28.7) |
| Past gestational diabetes | 45 | 0.83 ± 0.06 | 0 | 0.367 | 0 |
| Treated hypertension | 348 | 0.83 ± 0.08 | 5.2 | <0.001 | 7.69 (2.59–22.9) |
| Metabolic syndrome | 363 | 0.84 ± 0.07 | 2.8 | 0.080 | 1.69 (0.73–3.91) |
| Low weight (BMI < 19 kg/m$^2$) | 18 | 0.85 ± 0.06 | 5.6 | 0.065 | 3.16 (0.36–28.6) |
| Normal weight (BMI 19–24.9 kg/m$^2$) | 439 | 0.84 ± 0.06 | 2.5 | | 1.42 (0.56–3.64) |
| Overweight (BMI 25–29.9 kg/m$^2$) | 584 | 0.85 ± 0.06 | 0.7 | | 0.35 (0.10–1.21) |
| Obese (BMI 30+, kg/m$^2$) | 398 | 0.83 ± 0.06 | 2.0 | | 1.00 |

BMI: body mass index.

>10 mmHg, the mean of the two closest measurements was used. Height was measured to the nearest 0.5 cm without shoes using a stadiometer. Weight was measured to the nearest 0.1 kg using a mechanical beam balance after removal of shoes and excess clothing. Body mass index (BMI) was calculated in kg/m$^2$ and overweight defined as 25.0–29.9 kg/m$^2$ and obese as ≥30.0 kg/m$^2$ [15]. Waist circumference to the nearest 0.5 cm was measured halfway between the lower border of the ribs and the iliac crest in the horizontal plane. Two measurements were recorded and if different by ≥2 cm, a third measurement was taken. The mean of the two closest measurements was used in the analyses. Glucose was collected and measured as previously described [8]. Serum magnesium was measured using the Hitachi 917 R (Hitachi, Tokyo, Japan; reference range 0.70–1.15 mmol/l) i.e. local laboratory definition of hypomagnesaemia was <0.70 mmol/l. Glucose tolerance status including diabetes was defined by World Health Organisation criteria [8], and metabolic syndrome (MS) by ATPIII criteria [8]. Hypertension was considered present if reported as having been diagnosed by a doctor or nurse. The study was approved by the Goulburn Valley Ethics Committee and signed individual consent was obtained.

Data were analysed using SPSS v15 (SPSS Inc., Il, USA). Only 15 subjects were of non-European descent and 45 of Southern European descent and hence analyses do not adjust for ethnicity. Results shown are either mean (95% confidence intervals), mean ± standard deviation, median (range) or frequencies (percentages). Statistical significance was taken at the 5% level and all tests are two tailed. Unadjusted discrete variables were compared using Chi squared test and continuous variables using analysis of variance.

The association between a low serum magnesium and different characteristics was assessed using logistic regression with hypomagnesaemia defined by a serum magnesium concentration <reference range (i.e. <0.70 mmol/l) compared with all other subjects, or by comparing the lowest and highest quintile of serum magnesium concentrations (≤0.79 mmol/l vs ≥0.89 mmol/l, respectively). The logistic regression was undertaken using simultaneous, forward and backward entry of variables with little change in the result: only simultaneous entry is shown.

## 2. Results

The response rate was 61.3% overall. Of the 1453 subjects (56.3% women, 1.2% non-European) with magnesium results (one missing), the mean ± standard deviation age was 53 ± 16 years and BMI was 27.9 ± 5.2 kg/m$^2$. Those refusing to attend were younger (49 ± 18 years vs 53 ± 16 years, $p < 0.001$), less

**Table 3 – Odds ratios of having local laboratory defined hypomagnesaemia according to glucose tolerance and hypertension status.**

| | Odds ratio | Significance |
|---|---|---|
| Not taking anti-hypertensive | 1.00 | 0.006 |
| Taking anti-hypertensive medication | 6.77 (1.75–26.17) | |
| Normal | 1.00 | 0.026 |
| Impaired glucose tolerance/impaired fasting glucose | 0.90 (0.10–8.10) | |
| New diabetes | 10.51 (1.37–80.60) | |
| Known diabetes | 8.63 (2.20–33.90) | |

Other variables entered include age, sex, smoking, alcohol, fruit/vegetable consumption, obesity by Body Mass Index, metabolic syndrome, education status, rural town.

**Table 4 – Odds ratios of being in the lower quintile ($n$ = 298) vs upper quintile ($n$ = 331) for magnesium according to glucose tolerance, hypertension and obesity status.**

| | Odds ratio | Significance |
|---|---|---|
| Including those taking anti-hypertensive medications | | |
| Not taking anti-hypertensive | 1.00 | 0.011 |
| Taking anti-hypertensive medication | 1.82 (1.15–2.88) | |
| Normal | 1.00 | <0.001 |
| Impaired glucose tolerance/impaired fasting glucose | 1.45 (0.77–2.72) | |
| New diabetes | 1.46 (0.47–4.57) | |
| Known diabetes | 4.93 (2.33–10.42) | |
| Normal weight (BMI 19–24.9 kg/m$^2$) | 1.01 (0.58–1.75) | 0.001 |
| Overweight (BMI 25–29.9 kg/m$^2$) | 0.51 (0.32–0.81) | |
| Obese (BMI 30+, kg/m$^2$) | 1.00 | |
| Excluding those taking anti-hypertensive medications: lower ($n$ = 202) vs upper quintile ($n$ = 248) | | |
| Normal | 1.00 | <0.001 |
| Impaired glucose tolerance/impaired fasting glucose | 1.48 (0.80–2.75) | |
| New diabetes | 1.44 (0.46–4.46) | |
| Known diabetes | 5.52 (2.65–11.49) | |
| Normal weight (BMI 19–24.9 kg/m$^2$) | 0.93 (0.54–1.61) | 0.001 |
| Overweight (BMI 25–29.9 kg/m$^2$) | 0.48 (0.30–0.76) | |
| Obese (BMI 30+, kg/m$^2$) | 1.00 | |

Other variables entered include age, sex, smoking, alcohol, fruit/vegetable consumption, metabolic syndrome, education status, rural town. BMI: body mass index.

likely to own their own home (67% vs 79%, $p < 0.001$), have health insurance (41% vs 48%, $p = 0.001$), have completed university (13% vs 17%, $p = .006$) or to be in full time employment (34% vs 38%, $p = 0.037$), than those who attended, but were similar with regard to gender and ethnicity.

Mean serum magnesium was $0.84 \pm 0.06$ mmol/l and 25 (1.7%) participants had a low serum magnesium. Table 1 compares those with the low serum magnesium with other participants.

Those with hypomagnesaemia were older, more likely to be female, had a higher plasma fasting glucose and HbA1c, were more likely to have hypertension diagnosed (but comparable blood pressure), more likely to eat sufficient fruit and/or vegetables but less likely to drink alcohol. There was no difference in the proportion with health insurance or having undertaken higher education as measures of socioeconomic status.

Table 2 shows the mean serum magnesium, proportion with, and odds ratio for, hypomagnesaemia in different metabolic states. Those with new and known diabetes had an excess risk of hypomagnesaemia. Treated hypertension had a high risk of hypomagnesaemia. However, none of IGT, IFG, past GDM, the metabolic syndrome or obesity were associated with hypomagnesaemia. Table 3 shows the results of a logistic regression confirming the independent excess risk of hypomagnesaemia among those with treated hypertension and those with known and new diabetes. A multivariate analysis of variance with serum magnesium as the dependent variable shows a lower mean value (0.79 (0.78–0.81)) mmol/l among those with known diabetes than normal subjects (0.85 (0.84–0.85)) mmol/l. However, those with new diabetes had an adjusted mean serum magnesium of 0.83 (0.81–0.86) mmol/l, those with IGT 0.84 (0.82–0.85) mmol/l and those with IFG 0.84 (0.82–0.86) mmol/l.

Among the 25 with hypomagnesaemia, nine (36%) had treated hypertension alone, 11 (44%) had diabetes and treated hypertension, two had diabetes alone (8%) and one had a short bowel syndrome (BMI 18.7 kg/m$^2$).

Table 4 shows logistic regressions comparing those in the lower ($\leq$0.79 mmol/l) and upper ($\geq$0.89 mmol/l) quintile of magnesaemia. Those with known diabetes and treated hypertension were more likely, and those who were overweight less likely to be in the lower quintile. These findings were not affected by excluding those with treated hypertension (including those on diuretics). Past GDM remained a non-significant entrant when repeating the analysis among women alone.

## 3. Conclusions

Our study is in agreement with various other studies in concluding that there is an association between T2DM and hypomagnesaemia [2–6]. We have, however, failed to demonstrate a cross-sectional relationship between serum magnesium and obesity, IFG, IGT or past GDM, suggesting that the hypomagnesaemia–diabetes relationship is most likely associated with diabetes itself rather than its development (unless occurring immediately before the transition between the high risk states and diabetes). Furthermore, the relationship between hypomagnesaemia and new diabetes appears to be due to a small number of individuals with hypomagnesaemia (in association with treated hypertension) rather than across all of those with new diabetes (as shown by the mean serum magnesium and the logistic regression comparing top and bottom quintiles).

The relationship between treated hypertension and diabetes may help explain some of the findings elsewhere where

hypomagnesaemia and pre-diabetes have been found to be associated. A high proportion of those with diabetes have hypertension [16] and treatment with a diuretic is recommended in the management of hypertension [17], a graded, inverse, independent relationship between serum magnesium level and the subsequent development of incident type 2 diabetes has been shown in white middle-aged adults after adjusting for diuretic use [4]. Others have recently shown an association between hypomagnesaemia and impaired glucose metabolism (excluding diabetes) in a high risk Mexican population, however they did not adjust for treatment of hypertension [5]. Previous studies have demonstrated an association between hypomagnesaemia and dyslipidemia [3,6], an association that we have not been able to confirm. Some studies found a relationship with diabetes but did not undertake a full OGTT [6].

The relationship between dietary intake of magnesium and components of the metabolic syndrome is conflicting [5,18–22]. Song et al. [18] were able to demonstrate a statistically significant inverse correlation between serum magnesium and the risk of T2DM in overweight women only. Lopez-Ridaura et al. [19] also stated that the inverse relationship between dietary magnesium intake and the risk of T2DM was attenuated when corrected for BMI. The evidence for association of hypomagnesaemia with obesity as defined by BMI is unclear [6,23], our study now suggesting that there is no independent relationship between obesity and hypomagnesaemia.

Whole grains and dietary fibre, which are a rich source of magnesium have also been shown to have a favourable effect on the risk of T2DM [20,21]. However, some studies have not corrected for this dietary variable [18]. Most patients with higher intakes of magnesium in these studies were also shown to be leaner and more physically active. The ARIC study on the other hand, failed to demonstrate an association between dietary magnesium intake and the risk of T2DM [4]. Our nutritional measures were validated, but crude and hence unlikely to detect major dietary differences between those with and without hypomagnesaemia. The greater fruit and vegetable intake among those with hypomagnesaemia in Table 1, dropped out of the analyses in the logistic regression and probably reflects the greater proportion with diabetes (and therefore more likely to have received dietary advice). Those with diabetes do eat more fruit in some parts of rural Australia [24], a feature likely to be enhanced in a major fruit producing are where access is easy and inexpensive. We find it hard to explain why those who were overweight were less likely to have a low magnesium (in the logistic regression comparing top and bottom quintiles), although this may reflect dietary choices.

Our study has a number of strengths beyond its population-based sampling including the use of oral glucose tolerance test to assess the glycemic status and consideration of other medical conditions which might be associated with hypomagnesaemia. There were several limitations however. First, this is a cross-sectional study rather than prospective, and therefore a temporal relation between hypomagnesaemia and diabetes cannot be inferred from our data. Second, we have used serum magnesium as our indicator of magnesium status. As magnesium is a predominantly intracellular cation, intracellular magnesium deficiency and total body magnesium deficiency may exist in spite of normal serum magnesium levels. We have used only a single measurement of serum magnesium without measuring ionized free magnesium, calcium, potassium and phosphate, and hypomagnesaemia in isolation can often be transient. However serum magnesium is the most widely used measure of magnesium status, and has been commonly used in studies attempting to ascertain the relationship between magnesium and diabetes/metabolic syndrome.

In conclusion, we have shown that hypomagnesaemia is associated with new and previously diagnosed diabetes, but not its pre-cursor states. The data do not support a causal role for hypomagnesaemia in the development of diabetes.

## Conflict of interest

There are no conflicts of interest.

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