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Association between serum magnesium and blood count: influence of type 2 diabetes and central obesity

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Abstract

The relationship between serum Mg and blood cell counts in Chinese adult diabetes or central obesity was assessed by investigating 8163 subjects with China Health and Nutrition Survey (mean age 59·6 years, 54·9 % men). Participants were classified according to blood Mg (below 0·65 mmol/l, or 0·66–0·94 mmol/l or above 0·95 mmol/l), type 2 diabetes (yes/no) and central obesity (yes/no). Leucocytes, erythrocytes, platelets (PLT), Hb and glycated Hb (HbA1c) were determined using standardised methods and conditions. HbAc1, leucocytes and PLT were significantly higher among subjects with central obesity than without central obesity ($P < 0·05$). A significant increase for Hb, erythrocytes, PLT, but not leucocytes, across progressive Mg groups was observed in subjects without diabetes ($P < 0·05$). Hb, erythrocytes and HbAc1 were significantly higher among subjects with higher Mg than in subjects with lower Mg with diabetes ($P < 0·05$). Central obesity disturbed the positive association between PLT count and serum Mg. Type 2 diabetes caused metabolism disorder in serum Mg, blood sugar and blood cell count. Hb, erythrocytes and PLT, but not leucocytes, are positively correlated with serum Mg, but this association is somehow disturbed by type 2 diabetes or central obesity.

Key words: **Central obesity syndrome: Leucocytes: Blood platelets: Magnesium metabolism: Hb assays**

Mg is one of the important ions that maintain nerve, muscle and heart stress. Mg plays a key role in a wide range of cellular functions known to affect many aspects of the endocrine system[1–4]. Many studies showed that lower serum or urinary Mg was associated with increased risk of ischaemic stroke[5], CHD and cardiovascular mortality[6,7]. The main cause of hypomagnesaemia in humans is usually inadequate dietary intake. Dietary Mg is mainly absorbed by the small intestine via passive cell-side transport, which is driven by an electrochemical gradient and solvent drag[8]. Low Mg intakes raise inflammatory and CVD risks and increasing dietary Mg intake is associated with a reduced risk of stroke, heart failure, diabetes and all-cause mortality[9–11]. Some studies have shown that serum Mg concentrations was negatively associated with type 2 diabetes and indices of glycaemic control and insulin resistance[12]. There is also a literature view that serum Mg concentration is positively correlated with diabetes and glycaemic control index and insulin resistance[13–15].

Blood cell count is a commonly used and widely available test method and is also associated with type 2 diabetes or obesity to some extent[16–19]. In addition, in many studies, the use of Mg and serum Mg for disease treatment has been explored based on the relationship between Mg and blood cells[20–23]. There is data suggesting that magnesium valproate is beneficial as an anti-diabetic agent in type 2 diabetes mellitus and also prevents its cardiac complications[24]. Study indicates that type 2 diabetic patients have intracellular Mg defieiency and that oral Mg supplementation can increase the intracellular levels towards normal. In addition, Mg supplementation markedly reduces platelet (PLT) aggregation in response to known agonists of PLT aggregation[25]. There was no significant correlation

Abbreviations: CHNS, China Health and Nutrition Survey; HbA1c, glycated Hb; PLT, platelets; WC, waist circumference.

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between serum Mg and PLT counts in maintenance haemodialysis patients[26]. However, studies on the relationship between serum Mg and human blood cell counts, especially for type 2 diabetes and central obesity, are rare.

The aim of our study was to assess changes in the relationship between serum Mg and blood cell count in China adults with type 2 diabetes or central obesity. Using the Metabolic, Life-style and Nutrition Assessment in adults survey – China Health and Nutrition Survey (CHNS), we report that an elevated Hb, erythroctye and PLT count increased across progressive Mg groups in some subgroups.

Research design and methods

Study design

The CHNS is a large-scale, national cross-sectional survey that was designed to investigate the health and nutritional status among Chinese residents. Currently, data are available for 2009. A stratified multistage cluster random process was used to draw samples from nine provinces of China, which included Shandong, Henan, Liaoning, Hunan, Heilongjiang, Jiangsu, Hubei, Guangxi and Guizhou. All participants voluntarily joined the present study with informed consent and the study was approved by institutional review board from the University of North Carolina at Chapel Hill and Chinese Center for Disease Control and Prevention.

Study population

Participants included in the present study were aged 18 years or older. Information on age, sex, region, activity level, dietary behaviour was collected. There were 8163 participants included in the survey organised in 2009. This sample is varied, with variation found in a wide range of related biochemical markers, healthy factors, nutritional and demographic measures[27]. In addition, blood counts included leucocyte count, erythrocyte count and PLT count.

The present study is a cross-sectional analysis with complete information for the determination of type 2 diabetes, central obesity, blood cell count and blood Mg, as described later. Every subject underwent same examinations during this survey.

Measurements and definitions

Testing of blood cell counts was completed in local laboratories of each site in accordance with consensus guidelines of the CHNS. Testing of glycosylated Hb (HbA1c) and blood Mg was completed only in the provincial laboratories, which meets all requirements for accurate measurement and testing. The method of routine blood testing was the 3 or 5 classification automated haematology analyser. (Blood samples were analysed using the Sysmex XE-2100D automated haematology analyser. For the method of the specific machine, follow its standard operating procedure.)

Weight was measured to the nearest 0·1 kg with lightweight clothing on a calibrated beam scale and height was measured to the nearest 0·1 cm without shoes using a portable stadiometer. BMI was calculated as weight in kilograms divided by the square of height in metres. Waist circumference (WC) was measured at a point midway between the lowest rib and the iliac crest in a horizontal plane using non-elastic tape. Height, weight and WC were measured by trained examiners following a standard protocol from the WHO[28]. Height, weight and WC measurements were made at the same location and followed the same protocol at each study visit.

Central obesity was defined by WC >90 cm for men and >80 cm for women[28]. Laboratory data were obtained within 2 months of the questionnaire visit.

Subjects were initially classified with type 2 diabetes (yes/no) and central obesity, and then further classified according to the blood Mg (below 0·65 mmol/l, or 0·66–0·94 mmol/l or above 0·95 mmol/l) in accordance with classification of the CHNS.

Additional information based on interviews and the physical examination of patients at the time of the visit and data from clinical records included age, sex, weight, height, BMI, dyslipidemia, dietary factors and use of anti-diabetic drugs.

Statistical methods

Differences between Mg groups in socio-demographic and clinical characteristics were evaluated by ANOVA for continuous variables and the χ^2 test for categorical variables. In particular, we calculated and tested the differences in blood cell count among the three Mg groups: in the total sample, type 2 diabetes (yes/no) and central obesity (yes/no) subgroups.

A generalised linear model of the association between blood cell count (leucocytes, erythrocytes, PLT) and Mg (three groups) was built adjusting for age (continuous), sex, BMI (continuous), WC (continuous), type 2 diabetes (yes/no), anti-diabetic drugs treatment (yes/no), insulin injection (yes/no), blood pressure (continuous), energy intake (continuous), fat intake (continuous), protein intake (continuous), carbohydrate intake (continuous), HbA1c (%), urea (continuous), uric acid (continuous), apo A-1 (continuous), apo B (continuous), lipoprotein (continuous), creatinine (continuous), HDL-cholesterol (continuous), LDL-cholesterol (continuous) and insulin (continuous).

Separate adjusted models were also built for subjects with and without type 2 diabetes. As a sensitivity analysis, we performed more parsimonious models, excluding adjustment for some variables dealing with possible collinearity among some covariates. $P < 0{\cdot}05$ were considered to be statistically significant.

Results

A total of 8163 subjects (with mean age of 50·9 years and 46·7 % males) were included in this analysis. Most of them (91·7 %) were without type 2 diabetes and 38·4 % had central obesity. The mean clinic blood cell count (leucocytes, erythrocytes, PLT) were 6·3 ($\times 10^9$/l), 4·7 ($\times 10^9$/ml) and 213·0 ($\times 10^9$/l) and the mean blood Mg was 0·94 mmol/l.

In participants with type 2 diabetes, shown in Table 1, 52·2 % had normal Mg, 1 % had low Mg (≤0·65 mmol/l) and 46.8 % had high Mg (≥0·95 mmol/l). Subjects with high Mg were characterised by significantly older age ($P < 0{\cdot}0001$), higher blood pressure ($P < 0{\cdot}0001$) and higher WC (in male) ($P < 0{\cdot}0001$), but these differences were not clinically meaningful ($P > 0{\cdot}05$).

The presence of Mg was accompanied by a significantly higher prevalence in various biochemical indicators ($P < 0{\cdot}01$).

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Table 1. Sample characteristics according to magnesium status
(Mean values and standard deviations; numbers of participants and percentages)

	Total (*n* 8163)		Low Mg ≤ 0·65 mmol/l (*n* 84, 1 %)		Normal Mg 0·66–0·94 mmol/l (*n* 4261, 46·8 %)		High Mg ≥ 0·95 mmol/l (*n* 3818, 52·2 %)		
Variable	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P*
Age (years)	50·9	15·1	48·3	14·7	49·8	15·2	52·2	14·9	<0·0001
≥60 years									<0·0001
n	2275		18		1074		1183		
%	27·9		21·4		25·2		31·0		
Risk factors									
Systolic pressure (mmHg)	124·9	19·0	121·0	18·5	124·1	19·2	126·0	18·7	<0·0001
Diastolic pressure (mmHg)	80·3	11·2	76·6	13·2	79·5	11·3	81·2	11·0	<0·0001
BMI (kg/m^2)	23·4	3·5	23·3	3·4	23·2	3·5	23·6	3·5	<0·0001
Obesity (BMI ≥ 30 kg/m^2)									
n	338		2		168		168		0·2223
%	4·14		2·38		3·94		4·4		
Waist circumference (cm)									
Men	84·4	10·2	82·6	8·3	83·6	10·1	85·2	10·3	<0·0001
Women	81·3	10·3	84·6	11·4	80·5	10·3	82·2	10·2	<0·0001
Central obesity									<0·0001
n	3136		42		1530		1564		
%	38·4		50·0		35·9		41·0		
Diabetes									0·0069
n	680		4		326		350		
%	8·33		4·8		7·7		9·2		
Dietary factors									
Energy intake (kcal/d)*	2141·3	658·7	2198·8	587·7	2121·7	663·6	2155·2	654·5	0·1618
Fat intake (g/d)	75·4	39·7	82·7	31·8	75·3	40·9	75·3	38·5	0·2347
Protein intake (g/d)	65·8	22·9	65·4	26·4	64·7	22·5	67·0	23·2	<0·0001
Carbohydrate intake (g/d)	295·1	101·4	291·3	108·5	293·2	102·0	297·3	100·6	0·2376
Analytical values									
HbA1c (%)	5·6	0·9	5·3	0·5	5·6	1·0	5·7	0·8	<0·0001
Urea (mmol/)	5·5	1·6	5·3	1·5	5·3	1·5	5·6	1·7	<0·0001
Uric acid (mg/dl)*	308·3	106·3	268·2	83·1	294·1	90·0	325·2	120·1	<0·0001
Apo A-1 (g/l)	1·2	0·4	1·0	0·2	1·2	0·4	1·1	0·4	<0·0001
Apo B (g/l)	0·9	0·3	0·8	0·2	0·9	0·3	0·9	0·3	<0·0001
Lipoprotein (mg/dl)*	15·6	22·9	13·1	12·8	15·8	22·7	15·4	23·2	0·4193
Creatinine (μmol/l)	87·4	22·5	85·2	13·2	85·4	17·8	89·7	26·7	<0·0001
HDL-cholesterol (mmol/l)	1·4	0·5	1·4	0·4	1·4	0·5	1·4	0·5	0·3340
LDL-cholesterol (mmol/l)	3·0	1·0	2·8	0·8	2·9	0·9	3·1	1·0	<0·0001
Insulin (μIU/ml)*	14·3	22·1	8·4	8·6	13·7	20·1	15·2	24·3	0·0003
Antidiabetic drugs									
Oral drugs									0·4467
n	182		1		102		79		
%	2·2		1·2		2·4		2·1		
Insulin injection									0·3672
n	51		0		31		20		
%	0·62		0·0		0·73		0·52		

HbA1c, glycated Hb.
* To convert energy in kcal to kJ, multiply by 4·184. To convert uric acid in mg/dl to μmol/l, multiply by 59·48. To convert lipoprotein in mg/dl to μmol/l, multiply by 0·0357. To convert insulin in μIU/ml to pmol/l, multiply by 6·945.

These biochemical indicators include all the analytical values in Table 1 except for lipoprotein and HDL-cholesterol values.

Table 2 shows blood cell count values in the whole group according to the central obesity category and Mg status. A significant increase for blood cell counts, with the exception of leucocyte and PLT count, across progressive Mg groups (from low Mg ≤ 0·65 mmol/l, normal Mg 0·66–0·94 mmol/l to high Mg ≥ 0·95 mmol/l) in all subjects were noted ($P < 0·05$). In addition, when compared with the group without central obesity, the mean increment in PLT among subjects with central obesity was 211·1 (×10^9/l) in the group with Mg ≤ 0·65 mmol/l, 216·8 (×10^9/l) in the group with Mg 0·66–0·94 mmol/l and 217·2 (×10^9/l) in the group with Mg ≥ 0·95 mmol/l. Unlike the whole subjects and central obesity groups, a significant increase for PLT across progressive Mg groups in the subjects without central obesity group was noted ($P < 0·01$). HbAc1, leucocytes and PLT were significantly higher among subjects with central obesity than without central obesity ($P < 0·05$). HbAc1, leucocytes and erythrocytes were significantly higher among subjects with type 2 diabetes than without ($P < 0·05$).

Hb, erythrocytes, but not PLT and leucocytes, were significantly higher among subjects with higher Mg than in subjects with lower Mg ($P < 0·05$) both in central obesity (yes/no) subgroups.

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Table 2. Blood cell count and Hb values according to central obesity and magnesium status (Mean values and standard deviations)

	Total							Without central obesity							With central obesity						
	Low Mg ≤0·65 mmol/l		Normal Mg 0·66–0·94 mmol/l		High Mg ≥0·95 mmol/l			Low Mg ≤0·65 mmol/l		Normal Mg 0·66–0·94 mmol/l		High Mg ≥0·95 mmol/l			Low Mg ≤0·65 mmol/l		Normal Mg 0·66–0·94 mmol/l		High Mg ≥0·95 mmol/l		
	Mean	SD	Mean	SD	Mean	SD	*P*	Mean	SD	Mean	SD	Mean	SD	*P*	Mean	SD	Mean	SD	Mean	SD	*P*
Hb (g/l)	131·1	16·4	138·7	20·6	144·0	19·9	<0·0001	134·7	15·8	139·0	21·1	144·4	20·0	<0·0001	127·6	16·3	138·2	19·7	143·4	19·9	<0·0001
Leucocytes (×10^9/l)	6·1	1·8	6·3	2·1	6·3	1·8	0·4904	6·1	1·5	6·2	2·1	6·3	1·9	0·1678	6·2	2·1	6·5	2·1	6·4	1·7	0·2804
Erythrocytes (×10^9/ml)	4·4	0·5	4·6	0·7	4·8	0·7	<0·0001	4·4	0·5	4·7	0·7	4·8	0·7	<0·0001	4·3	0·6	4·6	0·7	4·8	0·7	<0·0001
PLT (×10^9/l)	202·3	49·1	210·9	67·1	215·7	69·5	0·0559	193·6	41·4	207·6	66·9	214·6	68·8	0·0071	211·1	54·7	216·8	67·2	217·2	70·5	0·5868
HbA1c (%)	5·3	0·5	5·6	1·0	5·7	0·8	<0·0001	5·3	0·5	5·5	0·9	5·5	0·8	0·0022	5·4	0·5	5·8	1·1	5·9	0·9	0·0080

PLT, platelets; HbA1c, glycated Hb.

Table 3. Blood cell count, Hb and glycated Hb (HbA1c) values according to central obesity and magnesium status in patients without type 2 diabetes and in patients with diabetes (Mean values and standard deviations)

	Total							Without central obesity							With central obesity						
	Low Mg ≤0·65 mmol/l		Normal Mg 0·66–0·94 mmol/l		High Mg ≥0·95 mmol/l			Low Mg ≤0·65 mmol/l		Normal Mg 0·66–0·94 mmol/l		High Mg ≥0·95 mmol/l			Low Mg ≤0·65 mmol/l		Normal Mg 0·66–0·94 mmol/l		High Mg ≥0·95 mmol/l		
	Mean	SD	Mean	SD	Mean	SD	*P*	Mean	SD	Mean	SD	Mean	SD	*P*	Mean	SD	Mean	SD	Mean	SD	*P*
Patients without diabetes																					
Hb (g/l)	131·1	16·6	138·6	20·5	143·8	19·9	<0·0001	135·2	16·0	138·8	21·0	144·2	19·8	<0·0001	127·0	16·4	138·2	19·5	143·2	20·0	<0·0001
Leucocytes (×10^9/l)	6·1	1·8	6·2	2·0	6·3	1·8	0·6355	6·0	1·5	6·2	2·0	6·3	1·9	0·9754	6·1	2·1	6·4	2·1	6·3	1·7	0·2431
Erythrocytes (×10^9/ml)	4·4	0·5	4·6	0·7	4·8	0·7	<0·0001	4·4	0·5	4·6	0·7	4·8	0·7	0·0255	4·3	0·6	4·6	0·6	4·8	0·7	<0·0001
PLT (×10^9/l)	203·8	49·7	210·7	67·2	215·5	69·2	0·0412	195·2	41·7	207·5	66·5	214·4	68·6	0·0154	212·5	55·7	217·1	68·2	217·3	70·1	0·6794
HbA1c (%)	5·3	0·4	5·4	0·5	5·5	0·4	<0·0001	5·3	0·4	5·4	0·4	5·4	0·4	<0·0001	5·3	0·4	5·5	0·5	5·6	0·4	<0·0001
Patients with diabetes																					
Hb (g/l)	132·5	10·4	139·6	21·8	145·3	20·2	0·0006	125·0	1·4	142·7	22·4	146·8	22·3	0·3143	140·0	9·9	137·8	21·3	144·5	19·0	0·0016
Leucocytes (×10^9/l)	6·9	1·2	7·0	2·8	6·7	1·8	0·2870	7·0	2·0	7·0	3·8	6·7	1·8	0·4537	6·7	0·0	7·0	2·0	6·7	1·9	0·4355
Erythrocytes (×10^9/ml)	4·1	0·7	4·7	0·7	4·9	0·7	0·0016	3·7	0·1	4·9	0·7	4·9	0·7	0·1832	4·9	0·6	4·6	0·7	4·8	0·7	0·0021
PLT (×10^9/l)	172·0	17·0	212·8	66·1	217·1	72·6	0·2766	160·5	14·8	209·3	74·6	218·3	72·5	0·4986	183·5	10·6	214·9	60·7	216·5	72·8	0·3025
HbA1c (%)	5·4	1·5	7·9	2·2	7·4	1·5	0·0002	4·2	0·2	8·0	2·9	7·5	1·8	0·0173	6·7	0·2	7·8	1·8	7·4	1·4	0·0107

PLT, platelets.

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Table 4. Generalised linear model of the association between blood cell count, Hb and magnesium according to diabetes status* (Mean values and 95 % confidence intervals)

	Mg groups						
	≤0·65 mmol/l		0·66–0·94 mmol/l		≥0·95 mmol/l		
	Mean	95 % CI	Mean	95 % CI	Mean	95 % CI	*P*
Full model							
Total							
Hb (g/l)	135·9	131·8, 140·0	139·5	138·5, 140·5	142·7	141·7, 143·8	<0·0001
Leucocytes (×10^9/l)	6·3	5·9, 6·7	6·4	6·2, 6·5	6·3	6·2, 6·4	0·3764
Erythrocytes (×10^9/ml)	4·5	4·3, 4·6	4·7	4·6, 4·7	4·8	4·7, 4·8	<0·0001
PLT (×10^9/l)	197·0	181·7, 212·4	206·2	202·2, 210·1	210·5	206·5, 214·5	0·0116
Subjects without diabetes							
Hb (g/l)	136·6	132·6, 140·6	140·4	139·8, 141·1	143·4	142·7, 144·1	<0·0001
Leucocytes (×10^9/l)	6·3	5·8, 6·7	6·3	6·2, 6·3	6·2	6·1, 6·3	0·4987
Erythrocytes (×10^9/ml)	4·5	4·3, 4·6	4·7	4·6, 4·7	4·8	4·7, 4·8	<0·0001
PLT (×10^9/l)	202·9	187·5, 218·3	209·6	207·1, 212·0	213·5	210·9, 216·2	0·0412
Subjects with diabetes							
Hb (g/l)	140·0	118·6, 161·4	139·4	136·8, 142·0	145·9	143·4, 148·4	0·0006
Leucocytes (×10^9/l)	5·8	3·7, 7·9	6·9	6·6, 7·1	6·6	6·4, 6·9	0·2870
Erythrocytes (×10^9/ml)	4·4	3·5, 5·3	4·7	4·6, 4·8	4·9	4·8, 5·0	0·0016
PLT (×10^9/l)	149·7	74·1, 225·2	211·8	202·7, 220·9	210·9	202·0, 219·8	0·2766
Parsimonious model†							
Total							
Hb (g/l)	135·9	131·9, 139·9	139·5	138·5, 140·5	142·8	141·8, 143·8	<0·0001
Leucocytes (×10^9/l)	6·3	5·9, 6·7	6·3	6·3, 6·4	6·3	6·2, 6·3	0·4730
Erythrocytes (×10^9/ml)	4·5	4·3, 4·6	4·7	4·7, 4·7	4·8	4·8, 4·8	<0·0001
PLT (×109/l)	196·0	181·5, 210·5	207·2	203·6, 210·9	213·0	209·3, 216·7	<0·0001
Subjects without diabetes							
Hb (g/l)	136·7	132·7, 140·7	140·5	139·9, 141·1	143·5	142·8, 144·1	<0·0001
Leucocytes (×10^9/l)	6·2	5·8, 6·6	6·3	6·2, 6·3	6·2	6·2, 6·3	0·5704
Erythrocytes (×10^9/ml)	4·5	4·3, 4·6	4·7	4·7, 4·7	4·8	4·7, 4·8	<0·0001
PLT (×10^9/l)	201·2	186·7	209·8	207·5, 212·1	214·9	212·5, 217·4	0·0016
Subjects with diabetes							
Hb (g/l)	138·8	117·8, 159·8	139·1	137·0, 141·3	145·7	143·6, 147·8	<0·0001
Leucocytes (×10^9/l)	7·2	5·4, 9·0	6·8	6·6, 7·0	6·7	6·5, 6·9	0·7349
Erythrocytes (×10^9/ml)	4·5	3·6, 5·3	4·7	4·6, 4·8	4·9	4·8, 5·0	0·0003
PLT (×10^9/l)	158·3	91·9, 224·7	212·0	203·4, 220·5	216·1	207·8, 224·5	0·1888

PLT, platelets.
* Models were adjusted for demographic characteristics, lifestyles, dietary factors and clinic variables (including diabetes, blood glucose level, antidiabetic drugs treatment, insulin injection, blood pressure, urea, uric acid, apo A-1, apo B, lipoprotein, creatinine, HDL-cholesterol, LDL-cholesterol, insulin).
† The parsimonious model adjusted for the same variables as the full model except for some variables dealing with possible collinearity among some covariates.

Data for subjects with and without type 2 diabetes are presented in Table 3. As depicted in Table 3, a significant increase for Hb, erythrocytes, PLT across progressive Mg groups was observed in subjects without type 2 diabetes ($P < 0·05$). However, no significant changes for Hb, leucocytes, erythrocytes, PLT across progressive Mg groups were observed in subjects with type 2 diabetes and without central obesity ($P > 0·05$).

Multivariable analysis of blood cell count with magnesium

The generalised linear model showed that after full adjustment for demographic characteristics, lifestyles, dietary factors and clinic variables (including diabetes, blood glucose, level, anti-diabetic drugs treatment, insulin injection, blood pressure, urea, uric acid, apo A-1, apo B, lipoprotein, creatinine, HDL-cholesterol, LDL-cholesterol, insulin), Hb, erythrocytes, PLT, but not leucocytes, were significantly higher among subjects with higher Mg than in subjects with lower Mg ($P < 0·05$). Multivariable models for leucocytes failed to attain statistical significance. When the parsimonious model was used, the aforementioned general pattern was quite similar (Table 4). And also, this pattern was noted as quite similar among subjects without type 2 diabetes by using a generalised linear model or parsimonious model. However, both multivariable models for PLT failed to attain statistical significance among subjects with type 2 diabetes, different from subjects without type 2 diabetes.

Discussion

The previous study shows that central obesity increased the risk of type 2 diabetes in Chinese people and was associated with increasing glucose. In central obesity, diet-induced weight loss reduces PLT activation[29]. In our study, central obesity disturbed the positive association between PLT count and serum Mg. However, this positive association was still noted in subjects without central obesity.

These data demonstrate that the presence of higher serum Mg is not associated with significant and substantial increases or decrease in leucocyte count, irrespective of central obesity and existence of type 2 diabetes. Erythrocytes, PLT and Hb are significantly and substantially higher in the presence of higher serum Mg in patients without type 2 diabetes and without central obesity. On the contrary, PLT is not associated with serum Mg among patients with type 2 diabetes or central obesity. Even

after full adjustment for demographic characteristics, lifestyles, dietary factors and biochemical markers, increasing trends in Hb and erythrocytes were noted along with the increase of serum Mg in all subjects and PLT in subjects without type 2 diabetes and without central obesity.

There was no significant correlation between serum Mg and blood count and Hb in type 2 diabetes without central obesity. In the study of Scibior *et al.*[30], a statistically significant increase in Hb level along with the increase of serum Mg concentration was demonstrated. Therefore, metabolism between Hb and serum Mg, in subjects with diabetes but without central obesity, was disturbed and somewhat special.

Significant increase ($P < 0{\cdot}05$) in total leucocyte count was observed in diabetic untreated rats compared with diabetic rats with oral Mg treatment. Erythrocyte count was increased ($P < 0{\cdot}05$) in diabetic rats (with oral Mg treatment) compared with the diabetic untreated group[31]. But in our study, there was no correlation between serum Mg and leucocyte count in adults without oral Mg treatment in any subgroup. On the other hand, increased level of leucocyte count was associated with incidence of type 2 diabetes[32]. Our study supported this view in every subgroup ($P < 0{\cdot}05$), according to Table 3.

Recent evidence attests that erythropoiesis disorder is commonplace in human body disorders such as CVD, venous thromboembolism, cancer, type 2 diabetes, community-acquired pneumonia, chronic obstructive pulmonary disease, liver and kidney failure as well as in other acute or chronic conditions[33]. Among the biological Mg surrogates, an association was found between serum Mg and erythrocyte Mg. Serum Mg was involved in the erythrocyte metabolism[34].

In the paper by Sanchez-Morito *et al.*[35], Mg deficiency led to increased intestinal absorption of Fe and decreased erythrocyte counts. The structural and functional changes in the erythrocyte caused by Mg deficiency probably account for the decrease in erythrocyte and Hb concentrations[35]. In our study, subjects' erythrocyte counts increased with increasing serum Mg, except for type 2 diabetes. Thus, serum Mg increase might be a positive factor in raising erythrocyte count. However, diabetes may disrupt the metabolism between Mg and erythrocytes.

In the previous study, serum Mg was found to be positively correlated with PLT count ($P < 0{\cdot}001$)[30]. Also, there was significant reduction in HBA1c in groups with given Mg supplementation[36]. Similar results can be found in our study: serum HbA1c of subjects with type 2 diabetes in high Mg group was lower than serum HbA1c of subjects in normal Mg group. But in the low Mg group, there was a strange phenomenon where subjects showed lower level of HbA1c. Compared with subjects without type 2 diabetes, type 2 diabetes caused metabolism disorder in blood sugar and serum Mg.

Immature PLT fraction is elevated in patients with type 2 diabetes and associated with poor glycaemic control[37]. The Mg level was inversely correlated with leucocyte ($P = 0{\cdot}028$) and PLT ($P = 0{\cdot}016$) counts on patients with haemodialysis[38]. However, serum Mg was inversely associated with thrombocytopenia in healthy adults[39]. Our study partly supported this view in subjects without type 2 diabetes and central obesity. But in subjects with type 2 diabetes or central obesity, there was no correlation between PLT and serum Mg, accounting for disturbed metabolism by diseases. And there was no significant difference between subjects with type 2 diabetes and without type 2 diabetes in the PLT count. The low numbers in the low Mg group is one limitation of the study. We will pay attention to this issue in future research.

In summary, blood cell count, with the exception of leucocytes, was associated with serum Mg, but this association is somehow disturbed by type 2 diabetes or central obesity.

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All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent was obtained from all individual participants included in the study.

D. L., L. Y. and S. L. contributed to the study concept, design and supervision; the analysis and interpretation of the data; and the drafting and critical revision of the manuscript for important intellectual content. Q. Z., L. Z. and Q. L. contributed to the analysis and interpretation of the data and to critical revision of the manuscript for important intellectual content. H. L. and J. Z. contributed to administrative, technical and material support for the study; to the analysis and interpretation of the data; and to critical revision of the manuscript for important intellectual content.

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