

Magnesium in Metabolic Syndrome: Review of Studies

Melina Duffoo*

Department of Medicine, European Miguel de Cervantes University, Spain

*Corresponding author: Melina Duffoo, Department of Medicine, European Miguel de Cervantes University, Spain, Tel: 051949674708; E-mail: melina.duffoo@gmail.com

Rec Date: Dec 28, 2015, Acc Date: 28 Dec, 2015, Pub Date: 06 Jan, 2016

Copyright: © 2016 Duffoo M. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Introduction

Magnesium is an element that is widely and its major route for human beings is presented through water and food. Magnesium absorption is inversely proportional to the intake and occurs primarily from the ileum to the colon. Magnesium is a mineral that is present in the diet including whole grains, green leafy vegetables, legumes and nuts [1]. Emerging evidence indicates that a high intake of magnesium from diet or supplements can affect favorably to a cluster of metabolic abnormalities including insulin resistance, hypertension and dyslipidemia, which is known as metabolic syndrome.

The reference dietary intake for magnesium is 420 mg per day for adult women and 320 mg for men. Magnesium balance is regulated by the interaction between magnesium intake through the diet, intestinal absorption and renal excretion of magnesium exchange this mineral in bone [2]. Although the serum magnesium may not reflect the total deposits of body magnesium, magnesium levels in serum is commonly used as a standard to define the deficiency of this mineral [3]. Magnesium is a cofactor of hundreds of enzymes, particularly those cellular reactions involved in the transfer, storage and use of energy [4].

The beneficial effects of intake of magnesium can be explained by various mechanisms, including the improvement of glucose homeostasis and insulin [5], oxidative stress [4], lipid metabolism [6], vascular or myocardial contractility [4], vasodilation [7], endothelium dependent antiarrhythmic effects [4], anticoagulants or antiplatelet effects and anti-inflammatory effects [8].

Observational studies of prospective cohort design are optimal for the study of dietary intake of long-term primary prevention of chronic diseases. However, prospective data on magnesium intake are relatively limited. In human intervention studies, a randomized, double-blind, placebo-controlled study is considered the best approach to examine a cause-effect. However, short-term controlled studies are usually conducted in the situation of secondary prevention of chronic disease due to cost considerations.

Obesity and Insulin Resistance

Obesity, particularly abdominal or visceral adiposity has been demonstrated as a root cause of insulin resistance and type 2 diabetes [9]. Some epidemiological studies have directly examined the effects of whole grains on body weight and changes weight.

Epidemiological evidence suggests an important role of magnesium in insulin sensitivity. Some cross-sectional studies have shown an inverse association between magnesium levels in plasma and erythrocytes and fasting insulin levels in both diabetic patients and in apparently healthy individuals [10]. Several studies have also found an association between magnesium intake and insulin homeostasis

quantified by insulin CLAMP technique [11]. Similarly, a significant inverse association between magnesium intake from diet and fasting insulin concentrations in several cross-sectional studies based on population was observed [10]. There was no clutch as in any cross-sectional study, the observed associations cannot be established as causal. Rosolova et al have reported a relation between the magnesium concentration in plasma and the disposition of glucose by insulin.

Dyslipidemia

Magnesium may decrease the activity of lecithin and HMG-CoA, and increased lipoprotein lipase activity. The HMG-CoA reductase is the rate limiting enzyme in cholesterol biosynthesis. Lipoprotein lipase is responsible for the conversion of triglycerides to HDL-C and thus leads to a decrease in hepatic synthesis and secretion of VLDL-triglycerides.

Several studies have evaluated the effect of magnesium supplementation on blood lipids and normal people or hyperlipidemic patients. In 1960, a clinical study reported that a combination of magnesium and potassium chloride reduced lipoprotein α γ β in 10%. Davis et al reported in 1984 that oral magnesium chloride (18 mmol/day) for 118 days it decreased the total cholesterol, LDL and VLDL and increasing HDL in 16 patients with hyperlipidemia significantly. In a controlled Rasmussen et al study, 47 patients with ischemic heart disease and myocardial infarction were randomly supplemented with magnesium hydroxide (15mmol/day) or placebo for 3 months. Magnesium supplementation led to a significant decrease Apo B (15%), a small increase of no difference in LDL Apo A and LDL. It was also observed that magnesium supplementation decreased total cholesterol and triglycerides in a study of 30 patients with chronic renal failure [12].

Hypertension

For decades it has accumulated a substantial amount of research that involve the crucial role of magnesium intake in regulating blood pressure [13]. Invitro studies have shown that magnesium has multiple functions which can contribute to their antihypertensive effects [13]. Background proposed mechanisms including inhibition of intracellular calcium mobilization as a calcium antagonist, attenuation sodium adverse effect by stimulating the ATPase activity of sodium - potassium or increasing urinary sodium excretion, decreased release of catecholamines, improving myocardial contractility and smooth muscle tone vascular endothelium-dependent vasodilatation, systemic inflammation and secretion of insulin action [4].

Prospective data on the ratio of magnesium intake with the development of hypertension is very limited. In the study of women's health, Song et al reported that high magnesium intake at baseline was

associated with a modestly lower risk to develop hypertension in apparently healthy women of middle age and older [13,14].

Similar effects were observed in nonsmoking women with no history of diabetes or high cholesterol levels were less likely to change the diet, these data are similar to the follow-up study of health professionals has refueled one significant inverse relationship between intake magnesium through diet and blood pressure [15].

In summary, the evidence suggests that magnesium may be an important nutrient required for human health. A lot of evidence has shown that high magnesium intake from diet or supplements can favorably affect a cluster of metabolic abnormalities including insulin resistance, hypertension, and dyslipidemia, known as metabolic syndrome. However there are still many important questions.

Inflammatory Syndrome

A history of an association between magnesium and immune function derives from the findings showing early onset of clinical signs of inflammation in rat's deficient magnesium, activation of immune cells during experimental magnesium deficiency and elevated of acute phase proteins in the state of magnesium deficiency [16].

References

1. Cleveland LE, Goldman JD, Borrund LG (1994) Data tables: Results from USDA's 1994 continuing survey of food intakes by individuals and 1994 diet and health knowledge survey. Beltsville, MD: Agricultural Research Service, U.S. Department of Agriculture.
2. Ford ES, Mokdad AH (2003) Dietary magnesium intake in a national sample of US adults. *J Nutr* 133: 2879-2882.
3. Innerarity S (2000) Hypomagnesemia in acute and chronic illness. *Crit Care Nurs Q* 23: 1-19.
4. Barbagallo M, Dominguez LJ, Galioto A, Ferlisi A, Cani C, et al. (2003) Role of magnesium in insulin action, diabetes and cardio-metabolic syndrome X. *Mol Aspects Med* 24: 39-52.
5. Nadler JL, Buchanan T, Natarajan R, Antonipillai I, Bergman R, et al. (1993) Magnesium deficiency produces insulin resistance and increased thromboxane synthesis. *Hypertension* 21: 1024-1029.
6. Altura BT, Brust M, Bloom S, Barbour RL, Stempak JG, et al. (1990) Magnesium dietary intake modulates blood lipid levels and atherogenesis. *Proc Natl Acad Sci U S A* 87: 1840-1844.
7. vitale JJ, Nakamura M, Hegsted DM (1957) The effect of magnesium deficiency on oxidative phosphorylation. *J Biol Chem* 228: 573-576.
8. Seelig MS, Heggveit HA (1974) Magnesium interrelationships in ischemic heart disease: a review. *Am J Clin Nutr* 27: 59-79.
9. Wilson PW, Grundy SM (2003) The metabolic syndrome: practical guide to origins and treatment: Part I. *Circulation* 108: 1422-1424.
10. Ma J, Folsom AR, Melnick SL, Eckfeldt JH, Sharrett AR, et al. (1995) Associations of serum and dietary magnesium with cardiovascular disease, hypertension, diabetes, insulin, and carotid arterial wall thickness: the ARIC study. *Atherosclerosis Risk in Communities Study. J. Clin Epidemiol*, 48:927-940.
11. Humphries S, Kushner H, Falkner B (1999) Low dietary magnesium is associated with insulin resistance in a sample of young, nondiabetic Black Americans. *Am J Hypertens* 12: 747-756.
12. Kirsten R, Heintz B, Nelson K, Sieberth HG, Oremek G, et al. (1988) Magnesium pyridoxal 5-phosphate glutamate reduces hyperlipidaemia in patients with chronic renal insufficiency. *Eur J Clin Pharmacol* 34: 133-137.
13. Touyz RM (2003) Role of magnesium in the pathogenesis of hypertension. *Mol Aspects Med* 24: 107-136.
14. Song Y, Sesso HD, Manson JE, Cook NR, Buring JE, et al. (2006) Dietary magnesium intake and risk of incident hypertension among middle-aged and older US women in a 10-year follow-up study. *Am J Cardiol* 98: 1616-1621.
15. Ascherio A, Rimm EB, Giovannucci EL, Colditz GA, Rosner B, et al. (1992) A prospective study of nutritional factors and hypertension among US men. *Circulation* 86: 1475-1484.
16. Purvis JR, Cummings DM, Landsman P, Carroll R, Barakat H, et al. (1994) Effect of oral magnesium supplementation on selected cardiovascular risk factors in non-insulin-dependent diabetics. *Arch Farm Med*, 3: 503-508.