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## Reply to MM Joosten et al.

Dear Editor:

In their letter, Joosten et al. discuss an important aspect of our trial (1), i.e., that 24-h urinary magnesium excretion was lower than expected. We are very grateful for their positive comments on our work and would like to further discuss this relevant topic.

In our study, we found a difference in mean urinary magnesium of 1.36 mmol/24 h, equivalent to 1.11 mmol/L, between baseline and follow-up after 12 wk of supplementation. These values were similar to those reported in a meta-analysis of interventional studies (2), where the mean difference in urinary magnesium between treated and control groups was 1.74 mmol/L (95% CI: 1.01, 2.47 mmol/L), and in a trial in a group of hypertensive middle-aged women, in whom 6 mo of 485 mg magnesium aspartate–HCL supplementation led to a mean increase of 1.80 mmol/24 h (3). Given the differences in the characteristics of the samples, the duration of the trials, the laboratory assessment of urinary magnesium, the dietary magnesium intake, and the types of magnesium used for supplementation, we felt that our findings were consistent with much of the literature.

We nonetheless recognized that the fact that 300 mg of bioavailable magnesium corresponded to only 33 mg of excreted magnesium warrants further investigation. Joosten et al. suggested 3 plausible hypotheses to explain these findings. With regard to their first hypothesis, we agree that poor compliance is unlikely because our participants were fit and motivated and received biweekly phone call reminders (1). As for the completeness of the 24-h urinary collections, taking baseline urinary output as an example, participants reported a significantly higher than estimated mean urinary production (1861 $\pm$ 579 vs. 1510 $\pm$ 519 mL) (4), and the urinary excreted magnesium should paradoxically be considered in excess rather than in deficit. At the same time, we need to remember the known problems encountered in older people with formulas for assessing renal function (5), so the differences between their reported and estimated diuresis should be considered very cautiously. Poor gastrointestinal absorption of the form of magnesium administered might help to explain our findings. The gastrointestinal absorption of magnesium is known to decline with aging (6), and magnesium oxide might be absorbed less than other forms of magnesium (7), leading to a less marked increase in urinary magnesium concentrations. But because magnesium oxide can be given in a single daily dose (1), and this can improve older people's compliance with the treatment (8), we felt that this choice would provide a good balance between compliance and absorption.

In addition to these considerations, we believe that the possibility of a subclinical form of magnesium deficiency in older people is an important factor to consider. Intracellular magnesium tends to decline linearly with aging (6), so supplementation with oral magnesium first fills intracellular, and only after the extracellular compartment, finally leading to a lower urinary magnesium excretion.

Finally, it may be that other forms of magnesium could prompt a more evident magnesium excretion, and possibly different results in physical performance variables. We therefore agree with Joosten et al. that further studies in older people should be encouraged with the use of urinary magnesium as a marker of magnesium status.

None of the authors had any conflicts of interest to declare.

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