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The "Calcium Antihypertension Theory"

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The current issue of the *American Journal of Hypertension* continues the saga of the "calcium antihypertension theory" by publishing an "updated" metaanalysis of the randomized controlled trials of calcium supplementation on blood pressure.[1] Unfortunately, the paper does not extend our knowledge compared to previously published metaanalyses[2,3] and, in particular, on large randomized trials such as the Trials of Hypertension Prevention (TOHP).[4] The sole purpose of this "new" analysis appears to be to support the need for cumulative metaanalyses ". . . to ensure that clinicians know the best current estimates of treatment effects"[1] and to compare effect size between nondietary and dietary trials. The authors' conclusions are that calcium supplementation leads to a small reduction in blood pressure—this was already known from previous overviews—and the effect of food rich in calcium is as great as supplementation, a totally unjustified conclusion.

Does the present metaanalysis improve the estimate of effect size? Figure 1 shows the average estimate of effect (and 95% confidence intervals) of the three most recent metaanalyses[1–3] and of the TOHP study.[4] As already argued in response to a previous metaanalysis,[5] all estimates are compatible with each other. However, this argument is conveniently ignored by Griffith et al.[1] The trivial increase in 'average' effect size is statistically significant due to the larger sample size, but it is not significantly different from previous results.

Why have the authors now decided to exclude studies previously included and, on the contrary, include studies previously regarded as unsuitable? For instance, studies giving <1 g of supplementary calcium are now excluded, although there is no evidence of a dose-dependent effect in any of the metaanalyses. Yet, when including dietary studies, Diet Approaches to Stop Hypertension Study (DASH)[6] was included even though it only achieved an estimated increase in dietary calcium of 800 mg with the combined diet as compared to the control diet. Moreover, this is not the only nutrient that changed in the experimental diet. Griffith et al fail to justify these criteria for including and excluding studies.[1]

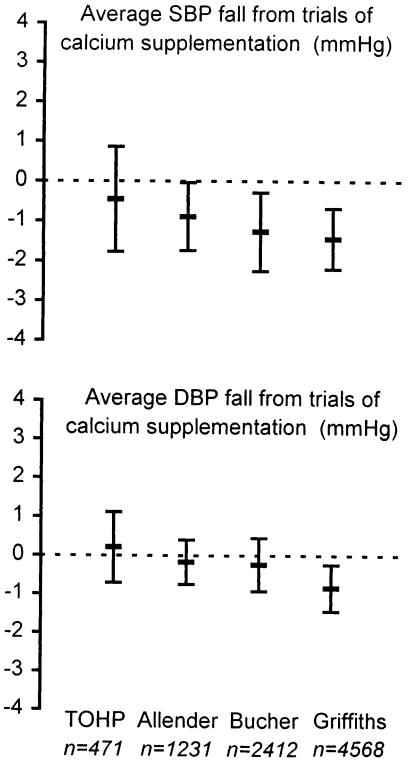

FIGURE 1. *Summary of the results of the three most recent quantitative overviews of controlled trials of calcium supplementation and changes in blood pressure[1–3] and the results of a large trial (TOHP).[4] Bars indicate 95% confidence intervals.*

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TABLE 1. CHANGES IN NUTRIENTS FROM CONTROL (OR BASELINE) IN THE DASH AND IN THE CCNW DIETS

Nutrient	DASH	CCNW—Men	CCNW—Women
Calcium (mg/day)	+822 (+285%)	+1239 (+140%)	+939 (+119%)
Total fat (g)		−50 (−57%)	−36 (−55%)
SFA (% of kcal)	−7.1 (−50%)		
PUFA (% of kcal)	+0.6 (+10%)		
MUFA (% of kcal)	−2.5 (−20%)		
CHO (% of kcal)	+6.0 (+12%)		
Protein (% of kcal)	+4.1 (+30%)		
Cholesterol (mg/day)	−82 (−35%)	−196 (−63%)	−115 (−52%)
Fiber (g/day)	+22 (+240%)	+14 (+65%)	+11 (+58%)
Potassium (mg/day)	+2663 (+150%)	+1532 (+47%)	+1232 (+45%)
Magnesium (mg/day)	+304 (+173%)	+347 (+100%)	+281 (+96%)
Iron (mg/day)		+17 (+100%)	+14 (+97%)
Sodium (mg/day)		−1254 (−32%)	−836 (−27%)
Zinc (mg/day)		+16 (+123%)	+14 (+140%)
Copper (mg/day)		+3 (+175%)	+2 (+185%)
Vitamin B_6 (mg/day)		+2 (+100%)	+2 (+123%)
Vitamin B_{12} (μg/day)		+8 (+120%)	+7 (+147%)
Vitamin C (mg/day)		+222 (+200%)	+189 (+175%)
Vitamin A (IU/day)		+9145 (+231%)	+7762 (+210%)
Vitamin E (IU/day)		+14 (+47%)	+14 (+58%)
Vitamin D (IU/day)		+151 (+62%)	+134 (+69%)
Folate (mg/day)		+0.44 (+142%)	+0.37 (+132%)
Niacin (mg/day)		+18 (+69%)	+18 (+86%)
Pantothenic acid (mg/day)		+14 (+264%)	+12 (+279%)

DASH, Diet Approaches to Stop Hypertension[6]*; CCNW, Campbell's Center for Nutrition and Wellness*[7]*; SFA, saturated fatty acids; PUFA, polyunsaturated fatty acids; MUFA, monounsaturated fatty acids; CHO, carbohydrates.*

Griffiths et al also claim that the new aspect of their overview is the inclusion of trials where calcium-rich diets have been given in comparison to calcium supplementation. Both DASH[6] and the Campbell's Center for Nutrition and Wellness (CCNW)[7] diets were dietary interventions in which the 'experimental' diet differed from the 'control' diet for a variety of nutrients only one of which was calcium. For instance, the DASH combined diet contained 240% higher fiber, 150% higher potassium, 173% higher magnesium, 30% higher protein, and substantially reduced fat content compared to the control diet. Likewise, the CCNW diet also contained higher fiber, potassium, magnesium, and lower sodium and cholesterol and varied from the control diet for all vitamins and trace elements (Table 1). Therefore, to attribute any change in blood pressure to calcium *only* in these dietary studies is incorrect.

The paper therefore does not add to our knowledge of the role of calcium in the control of blood pressure but does add further confusion. This publication will allow further misquotes and misrepresentations to be made. For instance, the paper of Griffith et al has already been quoted in a recent editorial[8] as showing that ". . . calcium derived from food sources may have as much as twice the beneficial effect of supplements . . .," an incorrect conclusion.

Why is the evidence about calcium subject to such distortion in comparison to other divalent cations, such as magnesium? In 1982, a campaign to release the pressure on the sodium-blood pressure issue was undertaken by a snack food giant. As part of their strategy there was the idea that they should encourage and support the "calcium antihypertension theory" despite their own admission that an increase in dietary calcium would be unlikely to have an effect on hypertension. Since then, the campaign has progressed and many of the studies purporting the wisdom of the "calcium antihypertension theory" have been funded by the National Dairy Council (like the one by Griffith et al) and the food industry. Presumably, one objective of the "calcium antihypertension" campaign is to keep the argument alive by increasing the quantity of manuscripts in a scientific journal. A continuous repetitious republication of metaanalyses primarily serves this aim given the lack of original new scientific supportive evidence and will allow further misrepresentation to be made.

It is ironic for the food industry that all the evidence so far suggests that large increases in calcium intake have little or no effect on blood pressure fulfilling their own prophecy back in 1982. But at least it relieved the pressure on salt.

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