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Dairy Consumption and Hypertension: Arterial Stiffness and Pulse Pressure

Merrill F. Elias University of Maine - Main, mfelias@maine.edu

Gregory A. Dore

Georgina E. Crichton

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Dairy Consumption and Hypertension: Arterial Stiffness and Pulse Pressure Merrill F. Elias, Gregory A. Dore and Georgina E. Crichton

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Letter to the Editor

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Dairy Consumption and Hypertension: Arterial Stiffness and Pulse Pressure

To the Editor:

We read with interest the meta-analysis of dairy consumption and hypertension by Soedamah-Muthu et al¹ in the November 2012 issue of Hypertension. Using a stringent set of exclusionary criteria, a literature consisting of 1709 unique prospective studies of dairy and milk consumption was reduced to 9 studies yielding an analysis sample size of 52756 subjects with 15367 cases of incident hypertension. This well-controlled meta-analysis confirmed hypotheses generated by previous cross-sectional work, that is, total and low-fat dairy were modestly protective with respect to incident hypertension; relative risk=0.97 (95% confidence interval, 0.95-0.99) and 0.96 (95% confidence interval, 0.94-0.98). Pulse wave velocity (PWV), the current gold standard measure of arterial stiffness, was not an outcome measure in this meta-analysis. Not only is PWV now recognized as an important tool in the diagnosis and management of hypertension, it is also an independent predictor of stroke and cardiovascular mortality and morbidity.²

PWV was not excluded from the Soedamah-Muthu et al metaanalysis, nor was it omitted. Our most recent search of the literature (November 27, 2012) indicated no cross-sectional or prospective examination of dairy consumption in relation to this important outcome variable, other than our recent study.³

In a sample of 587 community-based participants of the Maine-Syracuse Study (58% men, mean age 65, SD=12), stroke and dementia excluded, we reported a statistically significant linear cross-sectional relation between self-reported dairy intake, PWV, systolic blood pressure, and pulse pressure, but found no significant association between dairy intake and diastolic blood pressure, total cholesterol, low-density lipoprotein-cholesterol, or high-density lipoprotein-cholesterol. This was true for a basic model adjusting for age, sex, education, and race, as well as an extended model additionally adjusted for height and weight (or waist circumference), heart rate, antihypertensive drug treatment, mean arterial pressure, the lipid values, depressed mood, grains per day, vegetables per day, sweets per day, protein per day, and total food servings per day. Mean PWV values and 95% confidence intervals associated with reported consumption levels of never or seldom, 1 time per week, 2 to 4 times per week, 5 to 6 times per week, and ≥ 7 times per week were as follows: 11.00 (10.3–11.7), 10.8 (10.1–11.5), 10.6 (10.3–11.00), 10.0 (9.6–10.3), and 10.1 (9.8– 10.4) for the fully adjusted model and also when, in a final step, selfreport of physical activity was added to the model.

Limitations of our study were absence of information on change in blood pressure over time after our self-report dairy consumption survey, and inability to separate high- and low-fat dairy because of characteristics of our dietary questionnaire. Nevertheless, our study suggests a potentially important next step, that is, longitudinal studies of high- and low-fat dairy and PWV, and inclusion of PWV in future trials. There are 2 important questions: (1) will slowing in the rate of rise over 9 years, now seen for systolic blood pressure in the Alonso et al study, 4 be paralleled by a slowing in rise in PWV; and (2) does dairy consumption protect against incident-elevated PWV? Recent normative data make it possible to use one or more criteria of incident PWV as an outcome. 5

Disclosures

None.

Merrill F. Elias

Department of Psychology and Graduate School of Biomedical Science University of Maine Orono, ME

Gregory A. Dore

Department of Psychology University of Maine Orono, ME

Georgina E. Crichton

Nutritional Physiology Research Centre University of South Australia Adelaide, Australia

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