Correspondence

Letter by Bryan Regarding Article, “Red and Processed Meat Consumption and Risk of Incident Coronary Heart Disease, Stroke, and Diabetes Mellitus: A Systematic Review and Meta-Analysis”

To the Editor:

I read with interest the article by Micha et al reporting a weak association between processed meats but not red meats and coronary heart disease and diabetes mellitus. This type of systematic review of epidemiological data is very important, but in my opinion this particular report lacks the proper physiological perspective and context for accurate interpretation of the data. The authors suggests that the increased risk may be due to nitrates and nitrates used as preservatives, but their Table 2 indicates minimal difference in the nitrile and nitrate content of red versus processed meats, which reflects the fact that the amount of endogenous nitrogen oxides in meat or muscle exceeds that added in meat processing. Furthermore, their conclusions appear to contrast the emerging cardiovascular benefits of nitrite and nitrate. In fact, dietary nitrite and nitrate have been shown to reduce inflammation, restore endothelial function, reduce C-reactive protein, protect from heart attack and stroke, and even improve exercise performance.

Although modern epidemiology is an essential discipline in helping us identify associations between certain events and disease, a proper perspective and context is often missing. The authors report a relative risk of 1.42 for processed meat intake and coronary heart disease, suggesting a 42% higher risk of developing coronary heart disease if you eat processed meats. However, the processed meat/coronary heart disease portion of this study relies on only 5 studies, 2 of which were not statistically significant. Most problematic, however, is that 83% weight was given to a single study, Sinha et al, which ascribes mortality to various diet/lifestyle factors. Sinha et al also discuss their difficulty in adjusting risk for important cardiovascular confounding factors such as smoking, diabetes mellitus, and blood pressure. Furthermore, the Micha analysis extrapolates beyond the dietary intake ranges described in the Sinha study. Thus, one is left with a meta-analysis that is overweighted by a single study and has many questions related to confounding factors.

Studies such as this and others leave scientists and consumers alike confused as to what we should or should not eat. We have been told for decades to eat our vegetables. However, the large European Prospective Investigation into Cancer and Nutrition (EPIC-Oxford) study found that vegetarians had increased colon cancer risk compared with nonvegetarians (relative risk in vegetarians compared with meat eaters was 1.39 (95% confidence interval, 1.01 to 1.91), raising the specter that some dietary component in vegetarians increases risk, or that meat eating conferred decreased risk of this type of cancer. Does this mean that we should not eat our vegetables at the risk of getting colon cancer by the same argument as Micha et al put forward? My point is not to discredit important epidemiological data, but rather to put it in proper perspective. Epidemiology as a discipline is important and critical to public health protection and understanding disease associations, but it, alone, cannot establish causation. This extensive review by Micha et al is an important area of research, but we clearly need more research to clarify mechanisms and/or appropriate dietary recommendations.

Disclosures

Dr Bryan has a financial interest in Neogenis Inc.

Nathan S. Bryan, PhD
Brown Foundation Institute of Molecular Medicine
University of Texas Health Science Center at Houston
Houston, TX

References

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Nathan S. Bryan

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