COMMENTS AND RESPONSES

Associations Between Dietary Fiber and Inflammation, Hepatic Function, and Risk of Type 2 Diabetes in Older Men: Potential Mechanisms for the Benefits of Fiber on Diabetes Risk

Response to Wannamethee et al.

In their recent article reporting on the beneficial preventive effect of the intake of fiber on the occurrence of type 2 diabetes, Wannamethee et al. (1) did not provide any mechanisms to explain this phenomenon. They also reported an association of low concentrations of C-reactive protein and interleukin-6 with high fiber consumption.

Some of our recent work provides a mechanistic explanation for these observations:

1) While a high-fat, high-carbohydrate (HFHC) meal induces oxidative stress and inflammation, including an increase in the intranuclear nuclear factor-κB binding, and sets up the transcription of proinflammatory genes like interleukin-6, tumor necrosis factor-α, and C-reactive protein, a meal rich in fiber and fruit does not (2,3).

2) An HFHC meal induces a concomitant increase in plasma concentration of endotoxin (lipopolysaccharide) and the expression of its specific receptor, toll-like receptor-4 (TLR-4). This combination provides a further proinflammatory mechanism that is relevant to subsequent meals because both of these effects last for more than 5 h. In contrast, a meal rich in fiber and fruit does not induce these effects.

3) An HFHC meal induces an increase in the suppressor of cytokine signaling-3, which interferes with both insulin and leptin signal transduction. In addition, because TLR-4 deletion protects mice from insulin resistance induced by diet-induced obesity, the induction of TLR-4 would also potentially contribute to the induction of insulin resistance. In contrast, a meal rich in fiber and fruit does not induce cytokine signaling-3 or TLR-4 (2).

Since our meals and eating patterns in today's modern society are partial to HFHC-type proinflammatory meals, which are low in fiber, the effects of diets rich in fiber appear beneficial from the point of view of the prevention of diabetes as well as inflammation.

HUSAM GHANIM, PHD
AJAY CHAUDHURI, MD
PARESH DANDONA, MBBS, DPHIL, FRCP

From the Division of Endocrinology, Diabetes and Metabolism, State University of New York at Buffalo, and Kaleida Health, Buffalo, New York.
Corresponding author: Husam Ghanim, hghanim@kaleidahealth.org.
DOI: 10.2337/dc09-2127
© 2010 by the American Diabetes Association. Readers may use this article as long as the work is properly cited, the use is educational and not for profit, and the work is not altered. See http://creativecommons.org/licenses/by-nc-nd/3.0/ for details.

Acknowledgments— No potential conflicts of interest relevant to this article were reported.

References

1. Wannamethee SG, Whincup PH, Thomas MC, Sattar N. Associations between dietary fiber and inflammation, hepatic function, and risk of type 2 diabetes in older men: potential mechanisms for the benefits of fiber on diabetes risk. Diabetes Care 2009;32:1823–1825
2. Ghanim H, Abuaysheh S, Sia CL, Korzeniewski K, Chaudhuri A, Fernandez-Real JM, Dandona P. Increase in plasma endotoxin concentrations and the expression of toll-like receptors and suppressor of cytokine signaling-3 in mononuclear cells after a high-fat, high-carbohydrate meal: implications for insulin resistance. Diabetes Care 2009;32:2281–2287
3. Aljada A, Mohanty P, Ghanim H, Abdo T, Tripathy D, Chaudhuri A, Dandona P. Increase in intranuclear nuclear factor kappaB and decrease in inhibitor kappaB in mononuclear cells after a mixed meal: evidence for a proinflammatory effect. Am J Clin Nutr 2004;79:682–690

Downloaded from http://diabetesjournals.org/care/article-pdf/33/3/e43/605254/zdc00310000e43.pdf by guest on 09 August 2022