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Michael Keenan, PhD

Michael Keenan earned his PhD from the University of Illinois-Urbana in Nutritional Sciences in 1984. He remained at University of Illinois for two years as a post-doc before moving to Louisiana State University in 1986. After ten years of investigation of the interaction between vitamin D and the ultratrace mineral boron, Michael Keenan changed to obesity research. In 2001 Michael Keenan teamed up with Roy Martin who had recently moved to LSU from Georgia and began doing mechanistic, proof-of-concept rodent studies with resistant starch. This partnership has been very fruitful as far as publications and grant funding. Initially their research focused on the mechanism of body fat reduction in rodents with diets with resistant starch using isocaloric control diets. In recent years their focus has moved into the effects of resistant starch on the microbiota.

"Review of Dietary Resistant Starch Mechanisms - Findings from Animal Models"

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Animal models of diabetes and obesity of different etiologies have been used to demonstrate that dietary resistant starch has powerful effects on multiple mechanisms important in improving “metabolic health.” For example, dietary resistant starch improves maternal glycemic control in Goto-Kakizaki rat, a model of diabetic pregnancy. Resistant starch from high amylose maize (HAM-RS2) reduces body fat and increases gut bacteria in ovariectomized (OVX) rats, an animal model of menopausal weight gain. A Caenorhabditis elegans model of obesity was used to demonstrate that aqueous extract of cecal contents of rats fed resistant starch reduced body lipid staining in worms. Proposed mechanisms include the following: 1. enhanced gut microbial fermentation, 2. increased plasma levels of gut peptides, 3. elevated detoxification pathways, 4. improved bioavailabity of dietary polyphenols. The ability of dietary resistant starch to improve “metabolic health” consistently in different animal models provides strong support for positive outcomes in clinical studies. Two obese models that did not respond to resistant starch with reduced body fat and improved insulin sensitivity were GLP-1 receptor knockout mice and Zucker Diabetic fatty (ZDF) rats. Thus, it appears that functional GLP-1 and leptin receptors are necessary for beneficial health effects of resistant starch. Funding sources: Ingredion Incorporated and Louisiana State University AgCenter.

Learning objectives:

1. Several animal models of obesity and/or diabetes have fermented resistant starch and have had reduced body fat and improved glycemic control.
2. Two animal models of obesity and diabetes have fermented resistant starch and have not responded with reduced body fat (Zucker Diabetic Fatty rats and GLP-1 receptor knockout mice) and improved glycemic control (Zucker Diabetic Fatty rats).
3. Rodent studies demonstrate that functional GLP-1 and leptin receptors are necessary for responding to fermentation of resistant starch.
4. Human studies demonstrate that production of GLP-1 is not required for improved glycemic control when fed resistant starch, thus, other mechanisms exist for the benefits of consumption of resistant starch.