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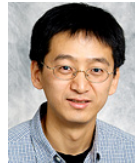
Cornell researcher strives to break the link between obesity and diabetes

By Metta Winter

Obesity and type 2 diabetes are inextricably linked, but biochemist and geneticist Ling Qi is working to break that connection.

Finding just the right gene could do it, says Qi, an assistant professor of nutritional sciences in the College of Human Ecology.

As a postdoctoral fellow at the Salk Institute for Biological Studies in La Jolla, Calif., before he joined the Cornell faculty last summer, Qi found that some of the mice in his lab became obese on a Western-diet regimen while others did not. In other cases, some developed diabetes after gaining weight on a Western diet but some didn't. The question is: Why?



Qi is trying to find out. In his Cornell laboratory, he is looking at two mechanisms that could potentially impact obesity and diabetes: the endoplasmic-reticulum (ER) stress response, which affects the expression of proteins, and the inflammation status of fat tissues.

Diabetes occurs when certain molecules malfunction in the signaling pathway, an event Qi finds fascinating.

"In the case of ER stress response, there's a DNA-binding protein that drives gene expression in cells; it's a key element for cells to respond to environmental cues -- in this case to glucose changes," explains Qi, who just won the 2008 Rosalinde and Arthur Gilbert Foundation/American Federation for Aging Research (AFAR) New Investigator Awards in Alzheimer's Disease as well as a Junior Faculty Award from the American Diabetes Association..

Little is known about this protein, so Qi and his research associate, postdoctoral fellow, graduate student, technician and an undergraduate student in his lab are seeking to learn more. Their studies in inflammation status also are proving to be promising, because it is now recognized that fat is a lot more than a storage depot for energy; it is an active organ that secretes hormones, many of which affect obesity and insulin sensitivity.

In a 2006 Science paper he published and in a manuscript he recently submitted, Qi describes how he demonstrated that altering fat-cell function changes obesity and insulin.

"Fat tissue has become the center of the metabolic control. If you change the fat mass, you will likely see the changes in insulin sensitivity of the whole system," says Qi, who has also published in the journal Nature.

When fat cells become bigger, immune cells infiltrate into the fat tissues. Qi wants to know what the functions of these macrophages are and what signals the recruitment of these macrophages. Such studies are so fundamental that they could one day reveal the origins of obesity and diabetes.

"It is crucial to understand the mechanism underlying the etiology of obesity and diabetes to develop well-targeted, efficient pharmacological interventions," Qi explains. "Working on genetic control of obesity and diabetes is very challenging but quite rewarding."

This article has been adapted from Human Ecology Magazine. Metta Winter is a writer with the Office of Publications and Marketing.

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