

Bees with an impaired insulin partner gene prefer proteins over carbs

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A new study of food-choice behavior in honey bees, published April 1 in the open-access journal *PLoS Genetics*, has identified a gene involved in bees' decisions to bring protein or nectar back to the colony. By taking control of the Insulin Receptor Substrate gene (IRS), an insulin partner gene in the bees' fat cells, researchers at Arizona State University and the Norwegian University of Life Sciences made the insects forego carbohydrates (sugar-containing nectar) and favor protein (pollen).

Food decisions are responsible, in part, for the epidemic of metabolic disorders in humans, such as diabetes and obesity. IRS partner [genes](#) are found in people where deficiencies in the [insulin](#) pathway have caused metabolic disease. Insulin is thought to change eating behavior by signaling to its IRS partner in the brain. In contrast, the results from Dr. Gro Amdam and colleagues show that IRS outside of the brain can also

modify food-choice behavior.

Most fat cells in honey bees are located in the abdomen. Researchers found that the experimental bees with normal IRS in the brain, but artificially reduced IRS in abdominal fat, returned to their colonies with less nectar than control bees. These bees' increased attraction to pollen and diminished interest in carbohydrates suggested an alteration in sensitivity to sugar. However, further testing determined that these bees gave up nectar without losing their taste for sugar.

While IRS affects the food choices of bees, it is not the only gene involved. Previous studies identified vitellogenin, a gene that also is active in fat cells. Its effect on the bees' loading of protein and carbohydrates is opposite to that of IRS. The researchers could not, in the present study, find a direct connection between the two genes.

More information: Wang Y, Mutti NS, Ihle KE, Siegel A, Dolezal AG, et al. (2010) Down-Regulation of Honey Bee IRS Gene Biases Behavior toward Food Rich in Protein. PLoS Genet 6(4): e1000896. [doi:10.1371/journal.pgen.1000896](https://doi.org/10.1371/journal.pgen.1000896)

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