Mice with fewer insulin-signaling receptors don't live longer

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Scientists studying longevity thought it might be good to lack a copy of a gene, called IGF1 receptor, that is important in insulin signaling. Previous studies showed invertebrates that lacked the copy lived longer, even if their bodies were less responsive to insulin, the hormone that lowers blood sugar.

A new study from The University of Texas Health Science Center San Antonio challenges this. Knocking out one copy of the gene failed to increase the life span of male mice, and it only modestly increased the life span of female littermates.

Martin Adamo, Ph.D., professor of biochemistry, and Arlan Richardson, Ph.D., professor of cellular and structural biology, lead the laboratories that conducted the study. "Our data show insufficiency of this insulin-signaling gene does not produce a robust increase in life span as previously reported in invertebrates," Dr. Richardson said.

Dr. Adamo said: "This demonstrates that reducing insulin signaling through the IGF1 pathway in mammals does not play the same role in aging that is observed in invertebrates."

A receptor is a molecule on a cell's membrane that receives chemical signals. Knocking down the genetic instructions that make IGF1 receptors results in reduced insulin signaling.

The study is described Nov. 23 in the journal PLoS ONE.

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