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The MRL mouse strain is naturally resistant to high fat diet-induced hyperglycemia

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The MRL mouse strain has long been studied for its autoimmune phenotypes, its super healing abilities and now we are investigating the mouse strain because it proves to be resistant to high fat diet-induced type 2 diabetes (T2D) pathology. Amongst the T2D characteristics that the control C57Bl/6J mice display, after HFD feeding are; hyperglycemia, insulin resistance, and cardiac hypertrophy. The HFD-fed MRL mice display none of these phenotypes. They remain sensitive to insulin and glucose and their cardiac function remains normal. In the preliminary steps of identifying the causative molecular mechanisms behind this HFD resistance we have identified increased skeletal muscle pAMPK and glycolysis. Both of these features are well known to be indicative of T2D resistance. We also assessed the levels of various hormones implicated in the pathogenesis of T2D. Surprisingly, we identified decreases in adiponectin levels in both HFD MRL and control diet (CD) MRL mice compared to their diet matched controls. As expected, insulin levels were increased in the control C57Bl/6J strain with HFD feeding. However, in the MRL mice insulin was decreased in the HFD mice. We also assessed food intake and grams gained for the four mouse groups. Both of the MRL mouse groups consumed significantly fewer food calories but gained significantly more weight. We are now attempting to combine the data for a model which explains the metabolic resilience of the MRL mice.

Biography

Ahlke Heydemann completed her PhD training in 1998 from the University of Chicago. In 2011 she joined The University of Illinois at Chicago to investigate pathologic differences in muscular dystrophy and metabolism caused by underlying genetic modifiers.

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