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Original Research

CEACAM1: A Link Between Insulin and Lipid Metabolism

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Objective: Liver-specific inactivation of CEACAM1 by a dominant-negative transgene (L-SACC1 mice) impaired insulin clearance and caused insulin resistance and increased hepatic lipogenesis. To discern whether this phenotype reflects a physiologic function of CEACAM1 rather than the effect of the dominant-negative transgene, we characterized the metabolic phenotype of mice with null mutation of *Cc1* gene (*Cc1*^{-/-}).

Research Design and Methods: Mice were originally generated on a mixed C57BL/6x129sv genetic background and then backcrossed twelve times onto the C57BL/6 background. More than 70 male mice of each of *Cc1*^{-/-} and wild-type *Cc1*^{+/+} groups were subjected to metabolic analyses, including insulin tolerance, hyperinsulinemic-euglycemic clamp studies, insulin secretion in response to glucose, and determination of fasting serum insulin, C-peptide, triglyceride and free fatty acid levels.

Results: Like L-SACC1, *Cc1*^{-/-} mice exhibited impairment of insulin clearance and hyperinsulinemia, which caused insulin resistance beginning at 2 months of age when the mutation was maintained on a mixed C57BL/6x129sv background, but not until 5-6 months of age on a homogeneous inbred C57BL/6 genetic background. Hyperinsulinemic-euglycemic clamp studies revealed that the inbred *Cc1*^{-/-} mice developed insulin resistance primarily in liver. Despite substantial expression of CEACAM1 in pancreatic β -cells, insulin secretion in response to glucose in vivo and isolated islets was normal in *Cc1*^{-/-} mice (inbred and outbred strains).

Conclusions: Intact insulin secretion in response to glucose and impairment of insulin clearance in L-SACC1 and *Cc1*^{-/-} mice suggest that the principal role of CEACAM1 in insulin action is to mediate insulin clearance in liver.

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Key Words: Obesity • insulin clearance • insulin action • insulin secretion • CEACAM1

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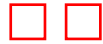
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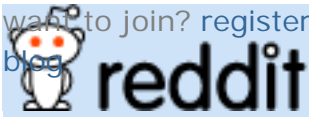
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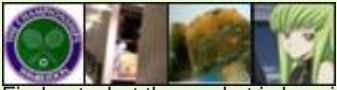
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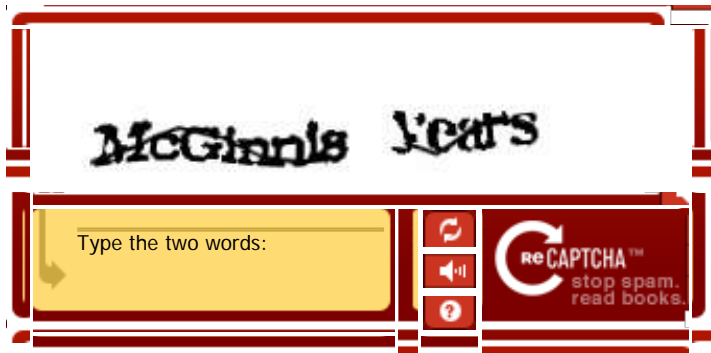
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