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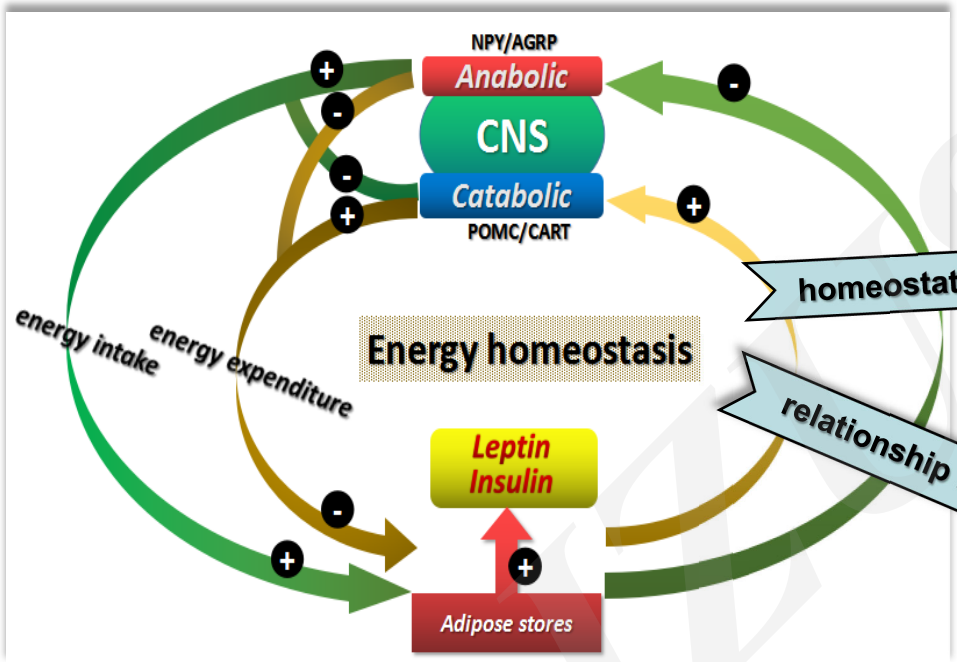
A disputed evidence on obesity: comparison of the effects of *Rcan2*$^{-/-}$ and *Rps6kb1*$^{-/-}$ mutations on growth and body weight in C57BL/6J mice

Key words: *Rcan2* gene, *Rps6kb1* gene, growth, body weight regulation, obesity

Backgrounds

Body weight is thought to be tightly controlled by homeostatic mechanisms, in which leptin plays a critical role through hypothalamic pathways. However, this popular hypothesis is hotly debated.



homeostatic disorder→obesity

?



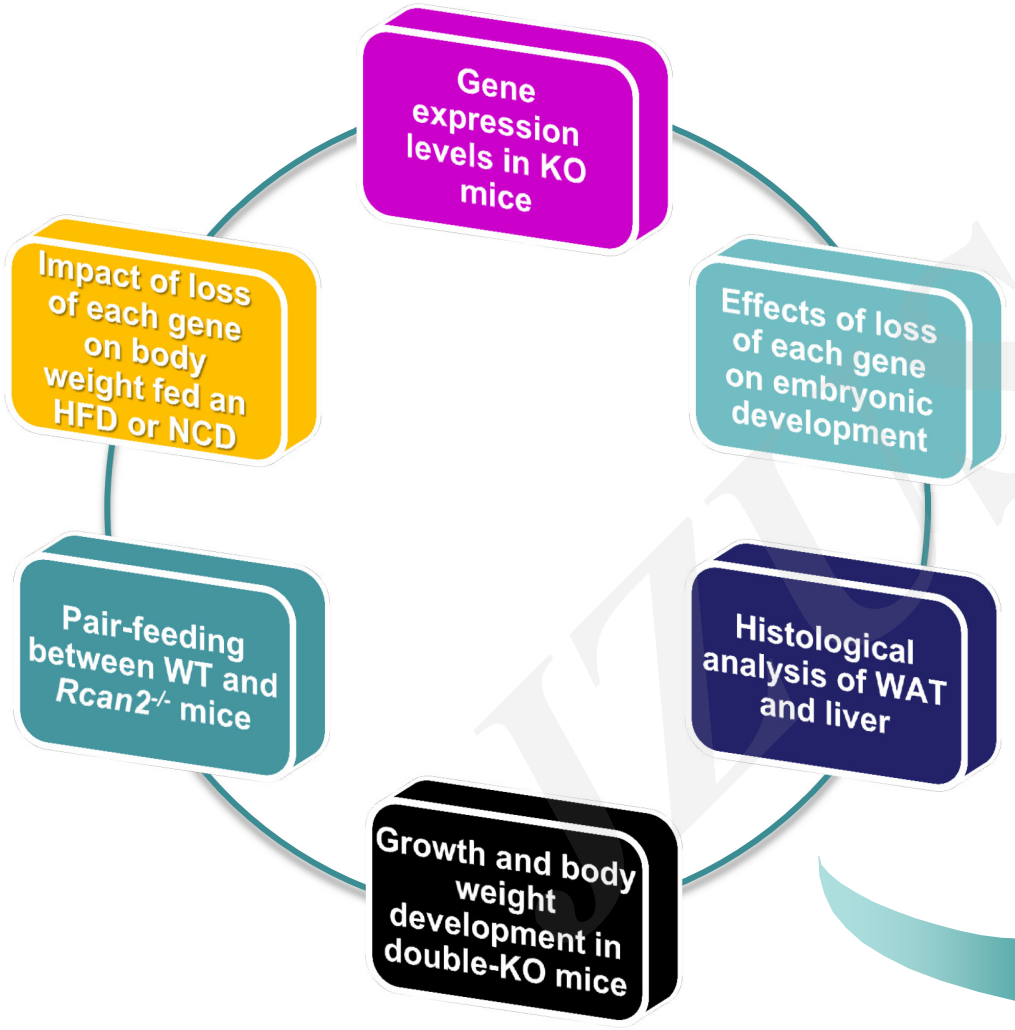
relationship between obesity and HFD

?



We found that *Rcan2* gene increases food intake and promotes obesity development through a leptin-independent mechanism.

Research Summary



This study mainly focused on the comparison of the effects of *Rcan2*$^{-/-}$ and *Rps6kb1*$^{-/-}$ mutations on growth and body weight in C57BL/6J mice.

*The relationship between Rcan2 and Rps6kb1 on body weight regulation

*Further evidence for the function of Rcan2 on the development of obesity

*The association of HFD and Rcan2 with obesity

Innovation points

- **Determination** of the fact that *Rcan2*$^{-/-}$ and *Rps6kb1*$^{-/-}$ mutations both affect growth and body weight, but through different mechanisms.

- **Further Confirmation** of the important role of *Rcan2* gene in the development of obesity.

- **Finding** that HFD is likely to promote weight gain through a gene-independent mechanism.

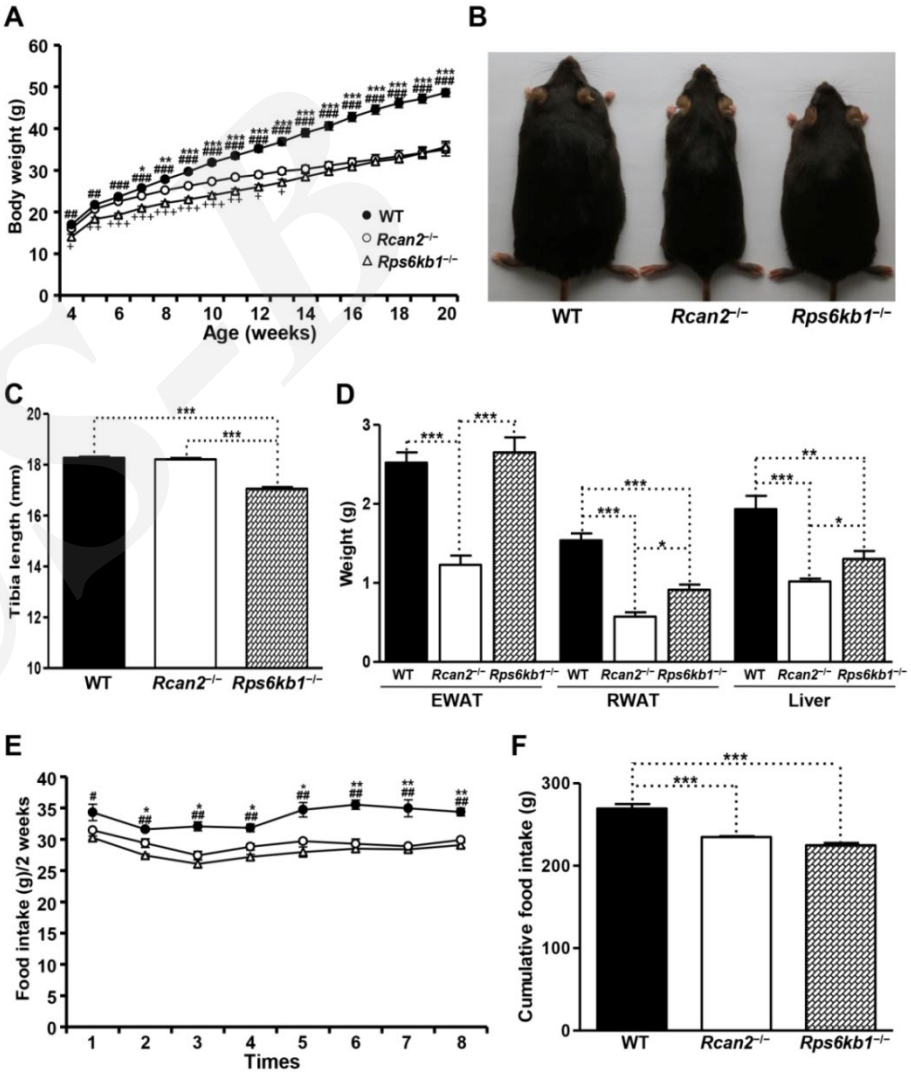


Fig.3 Phenotypes of WT, *Rcan2*$^{-/-}$ and *Rps6kb1*$^{-/-}$ mice fed on an HFD.