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Novel roles of miR-199b in regulating fat and bone metabolism

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i. Public Health Problem:

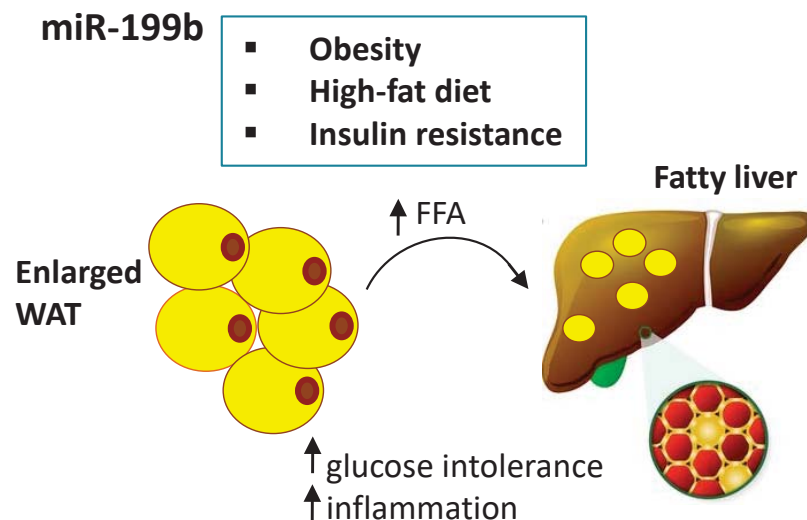
- The incidence of obesity has reached epidemic proportions worldwide and has contributed to an increase in the risk of numerous chronic disorders → type 2 diabetes, liver pathologies, dyslipidemia, and cardiovascular diseases.
- Obesity can have negative effects on bone remodeling → reduced mineral density → osteoporosis.
- Imbalance between food intake and energy expenditure → obesity → accumulation of fat mass and energy storage in white adipose tissue (WAT).

ii. Objectives:

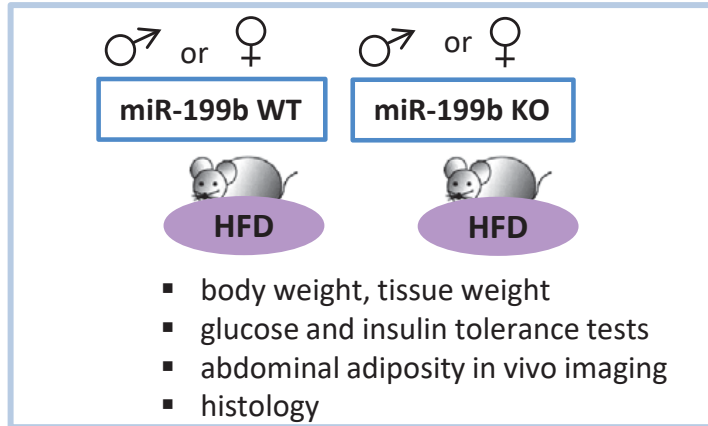
- During obesity overworked fat cells reach their storage capacity; this leads to the deleterious accumulation of lipids in the liver, skeletal muscle, pancreas, and heart.
- MicroRNA 199b is strongly elevated in low calorie diet-responders in obese individuals, which correlated with weight loss → gap in our understanding whether miR-199b regulates the components of metabolic homeostasis.

iii. Hypothesis:

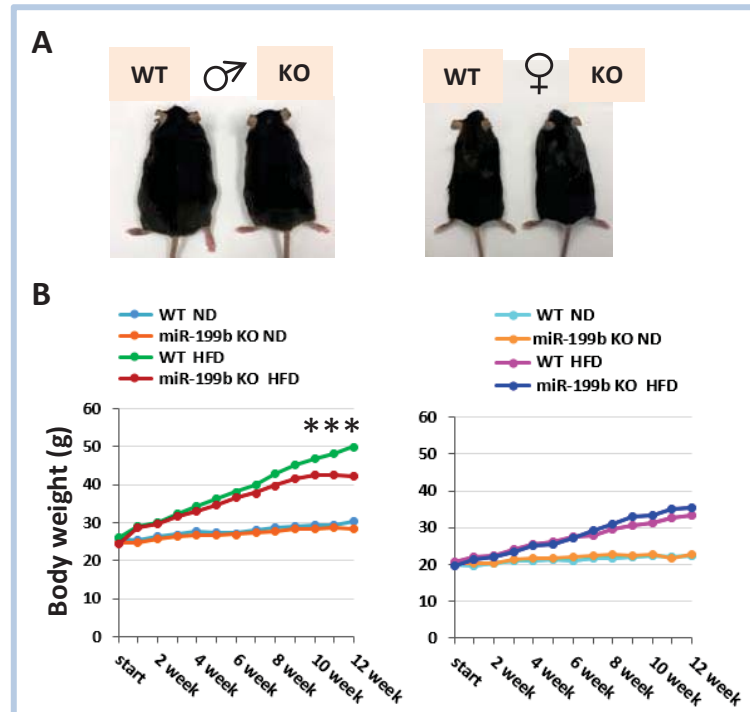
- MicroRNA 199b controls susceptibility of abdominal adipose tissue accumulation and development of obesity



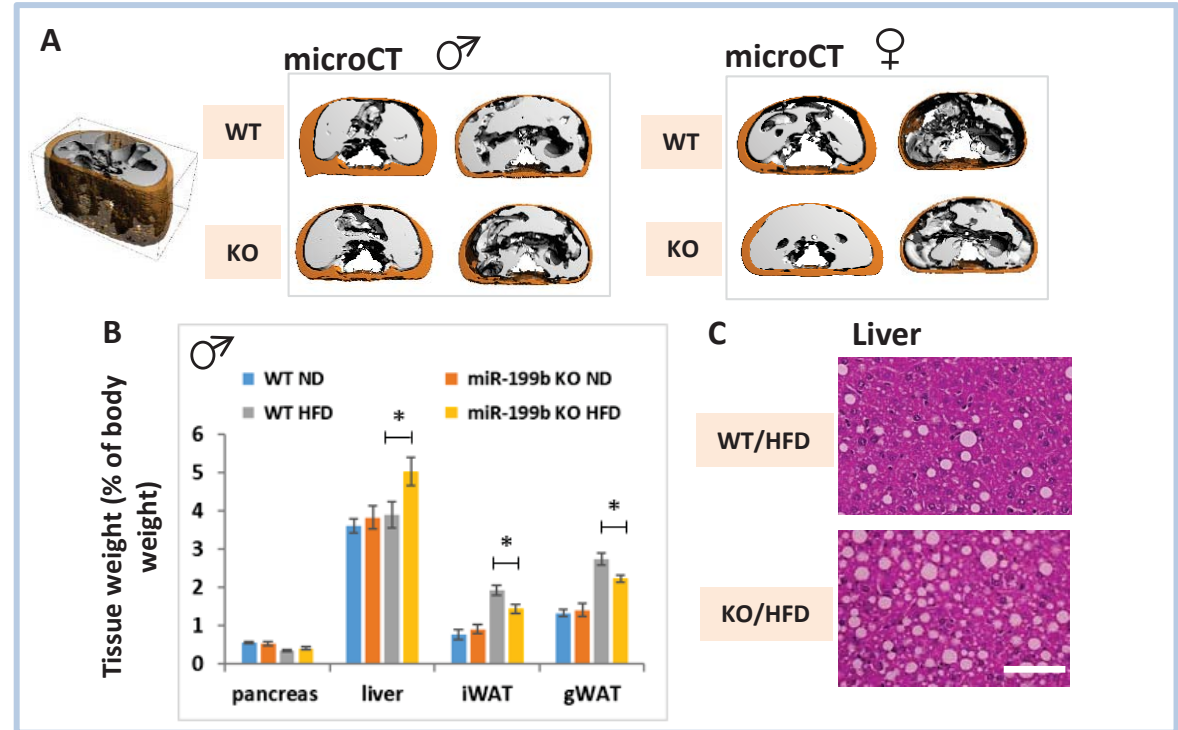
1. Study model and strategy



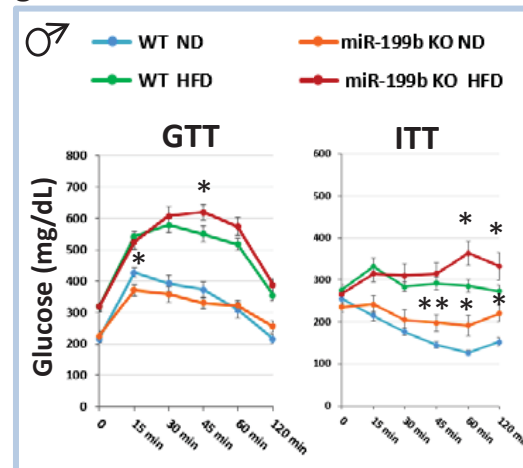
2. Results: Loss of miR-199 reduces body weight in HFD-induced obesity in male mice



3. Results: Loss of miR-199 differentially regulates WAT adipose tissue accumulation in HFD-induced obesity and leads to defects in liver



4. Results: Loss of miR-199 impairs glucose homeostasis



5. Conclusions

- Loss of miR-199b reduces WAT in HFD-induced obesity in male mice,
- Suppression of miR-199b impairs glucose tolerance and insulin sensitivity,
- Loss of miR-199b leads to liver steatosis,
- miR-199b differentially affects fat metabolism in males and females in response to HFD.