Deficiency of FcεR1 increases body weight gain but improves glucose tolerance in diet-induced obese mice

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Figure 2

A. Body weight (g) over Age (week) for WT (n=15) and Fcofr1a−/− (n=15) with HFD. *P<0.001.

B. Food intake (g/day) over Lean mass (g) and Fat mass (g) with WT (n=8) and Fcofr1a−/− (n=6). *P<0.002.

C. Glucose (mg/dL) over Time (minute) for WT (n=15) and Fcofr1a−/− (n=15). *P<0.001.

D. Plasma insulin (ng/mL) over Time (minute) for WT (n=7) and Fcofr1a−/− (n=7). *P<0.02, **P<0.001.

E. Insulin secretion (% islet insulin content) over IgE (μg/mL) for Glucose: 2.8 mM, 11.1 mM, 18.7 mM. *P<0.001.

F. FcRγ1 mRNA (fold change) over IgE (μg/mL).

G. Plasma IgE (ng/mL) over Plasma insulin (ng/mL).

H. IgE (ng/WAT) over IgE (μg/mL).

I. Plasma SAA (serum amyloid A) over Plasma IL6 (pg/mL) and Plasma MCP-1 (pg/mL) for WT (n=15) and Fcofr1a−/− (n=15). *P<0.003, **P<0.022.
Figure 3

A. Mac2 (% in VAT) in WT and FcεR1α−/− mice. Wichita State University.

B. Mac2 (% in SAT) in WT and FcεR1α−/− mice. Wichita State University.

C. CD3 T-cells in VAT. Wichita State University.

D. TUNEL-positive cells (number/mm²) in WT and FcεR1α−/− mice. Wichita State University.

E. AUC VO2 mL/lmin mass (g/hour). Wichita State University.

F. AUC VO2 mL/lmin mass (g/hour). Wichita State University.

G. AUC RER. Wichita State University.

H. AUC heat. Wichita State University.

I. Plasma leptin (ng/mL). Wichita State University.

J. WAT adipocyte size (mm²). Wichita State University.

K. Western blot analysis for UCP1 and GAPDH. Wichita State University.
Figure 6

A) TUNEL (%) as a function of adipogenesis (day) and IgE exposure. *P<0.05, **P<0.001.

B, C) Images showing changes in adipogenic differentiation with IgE exposure.

D) Cell viability (OD495 nm, CCK-8) with increasing IgE concentration.

E) Cytotoxicity (OD495 nm, LDH) with IgE concentration.

F) 2DG6P (μM) uptake with IgE concentration.

G) Western blot analysis showing expression of Glut4, p-AKT, AKT, and β-Actin under control and IgE conditions.

H) Western blot analysis showing expression of FcεR1a, Glut4, p-AKT, AKT, and β-Actin with siRNA treatment.
Figure 7