## **Supplementary Materials**

## Adipose- and muscle-derived Wnts trigger pancreatic β-cell adaptation to systemic insulin resistance

## Kamil Kozinski<sup>1</sup>, Magdalena Jazurek<sup>1</sup>, Pawel Dobrzyn<sup>2</sup>, Justyna Janikiewicz<sup>1</sup>, Katarzyna Kolczynska<sup>1</sup>, Anna Gajda<sup>1</sup> & Agnieszka Dobrzyn<sup>1</sup>

<sup>1</sup>Laboratory of Cell Signaling and Metabolic Disorders, Nencki Institute of Experimental Biology, Polish Academy of Sciences, Warsaw, Poland
<sup>2</sup>Laboratory of Medical Molecular Biochemistry, Nencki Institute of Experimental Biology, Polish Academy of Sciences, Warsaw, Poland

**Correspondence:** 

Agnieszka Dobrzyn

E-mail: adobrzyn@nencki.gov.pl



**Supplementary Figure 1.** Palmitate (16:0) treatment induced insulin resistance in 3T3-L1 (**a**) and C2C12 (**b**) cells. The protein level of AKT and extent of phosphorlation of AKT at Ser473 in BSA- and palmitate-treated cells were analyzed by Western blot.



**Supplementary Figure 2.** Effect of high-fat feeding on insulin mRNA level in pancreas (**a**) and on insulin content in whole pancreas (**b**) and in blood plasma (**c**). Rats were fed high-fad diet for 8 or 16 weeks. mRNA level was measured by real-time PCR. Insulin content was measured using a Rat/Mouse Insulin ELISA Kit (Millipore), according to manufacturer's protocol. The data are expressed as mean  $\pm$  SD, n = 6. \*P < 0.05 vs CHOW group; #P < 0.05 vs 8wk HF group.