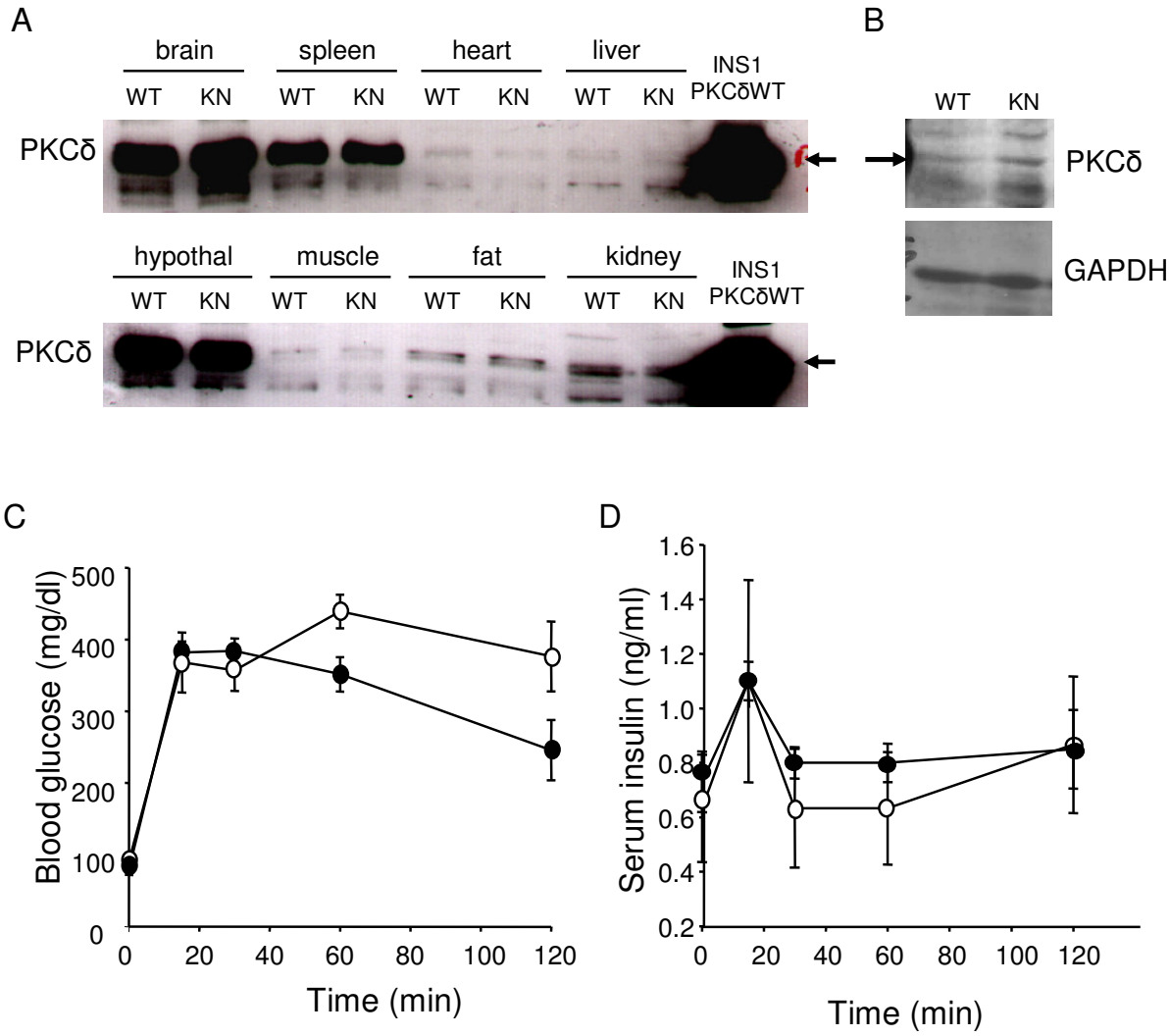
ONLINE APPENDIX

Table S1: Age, diet, body weight, plasma glucose levels and serum insulin concentrations of control and PKCδKN over-expressing mice. Details are described in Research Design and Methods.

mice	**control**	**PKCδKN**	**control**	**PKCδKN**	**control**	**PKCδKN**
Age (months)	1.5	1.5	9	9	3	3
Diet	Chow	Chow	Chow	Chow	HFD	HFD
Body weight (g)	17.0 ± 3.0	16.0 ± 1.0	44.0 ± 4.0	46.1 ± 2.0	42.0 ± 2.0	44.5 ± 2.0
Fasted glucose (mg/dl) (#179)	96.0 ± 25.0	81.0 ± 4.0	175.0 ±19.0	137.0 ± 10.0	157.0 ± 18.0	108.0 ± 4.0*
Fasted glucose (#162)	nd	nd	144.0 ± 4.5	135.0 ± 3.0	91.0 ± 8.6	79.5 ± 7.2
2 h glucose (#179)	101.0 ± 18.0	100.0 ± 1.0	289.0 ± 17.0	218.0 ± 28.0	310.0 ± 82.0	108.0 ± 31.0 *
2 h glucose (#162)	nd	nd	306.0 ± 11.2	267.3 ± 6.2 *	376.3 ± 48.8	245.8 ± 42.2
Fasted insulin (ng/ml) (#179)	0.22 ± 0.14	0.32 ± 0.07	1.30 ± 0.20	1.00 ± 0.10	0.32 ± 0.04	0.55 ± 0.12
2 h insulin (% of basal) (#179)[1]	75.0 ± 28.0	54.0 ± 17.0	77.0 ± 25.0	130.0 ± 13.0	143.0 ± 7.0	193.0 ± 18.0*

[1]glucose load (2g/kg body weight); nd, not determined

* Significantly different to the respective value of control mice



<u>Fig. S1:</u> Expression of PKCδ in tissues of control (WT) and PKCδKN over-expressing mice and blood glucose and plasma insulin during ipGTT after HFD. (A) Western blot analysis of tissue homogenates (50 µg of mouse line #162) reveals a high expression of PKCδ in brain, spleen and hypothalamus (hypothal), but a low expression of PKCδ in heart, kidney, liver muscle and fat in both control (WT) and PKCδKN over-expressing mice (KN). As control, INS-1E cells transfected with PKCδWT (15 µg) are shown on the right. **(B)** Western blot of homogenates of isolated islets from control and PKCδKN mice (#162) discloses a low expression of PKCδKN. **(C** and **D)** Blood glucose and plasma insulin concentrations represented as means ± SEM of control mice (n = 3, white symbols) and of PKCδKN mice (#162, n = 4, black symbols) were measured after intraperitoneally glucose injection (2 g/kg body weight). The transgenic mice (#162) with a low expression of PKCδKN exhibit the tendency to improve glucose homeostasis.

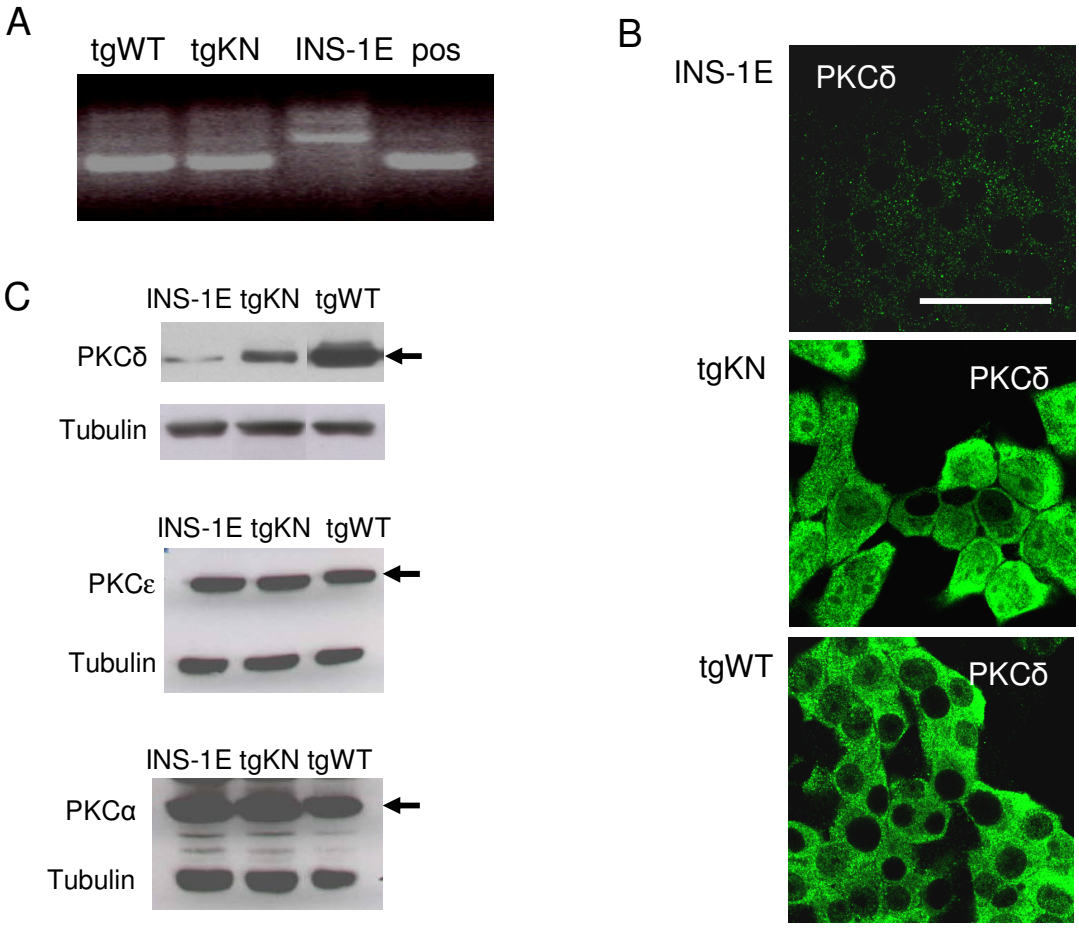


Fig. S2: INS-1E cells overexpressing PKCδWT or PKCδKN. (A) Detection of PKCδ by PCR in INS-1E cells transfected with the respective DNA for PKCδ (tgWT) and the mutated PKCδKN (tgKN) compared to untransfected INS-1E cells and the positive control (pos). **(B)** Immunohistochemical detection of PKCδ in control INS-1E cells and INS-1E cells transfected with WT (tgWT) and KN PKCδ (tgKN). **(C)** Respective Western blots for PKCδ, PKCε and PKCα in control INS-1E cells and INS-1E cells transfected with KN PKCδ (tgKN) and WT PKCδ (tgWT).

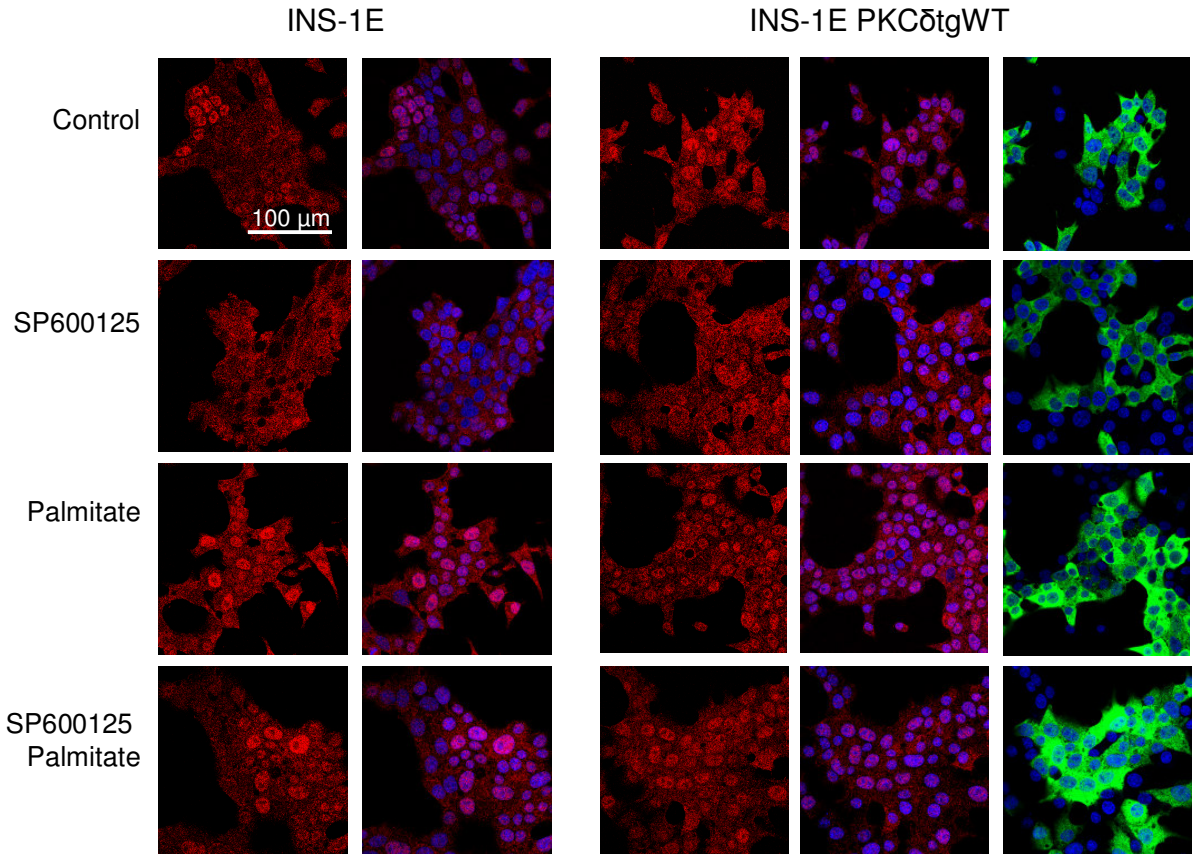


Fig. S3: Nuclear localization of FoxO1 in INS-1E cells depends on PKCδ expression. Representative pictures of FoxO1 staining (red), nuclear staining (blue) and PKCδ staining (green) of INS-1E cells and INS-1E cells transfected with PKCδWT after treatment for 1 d with palmitate (0.6 mmol/l) and with JNK inhibitor SP600125 (10 µmol/l) as indicated. The pink colour indicates increased nuclear localization of FoxO1.