Figure S1

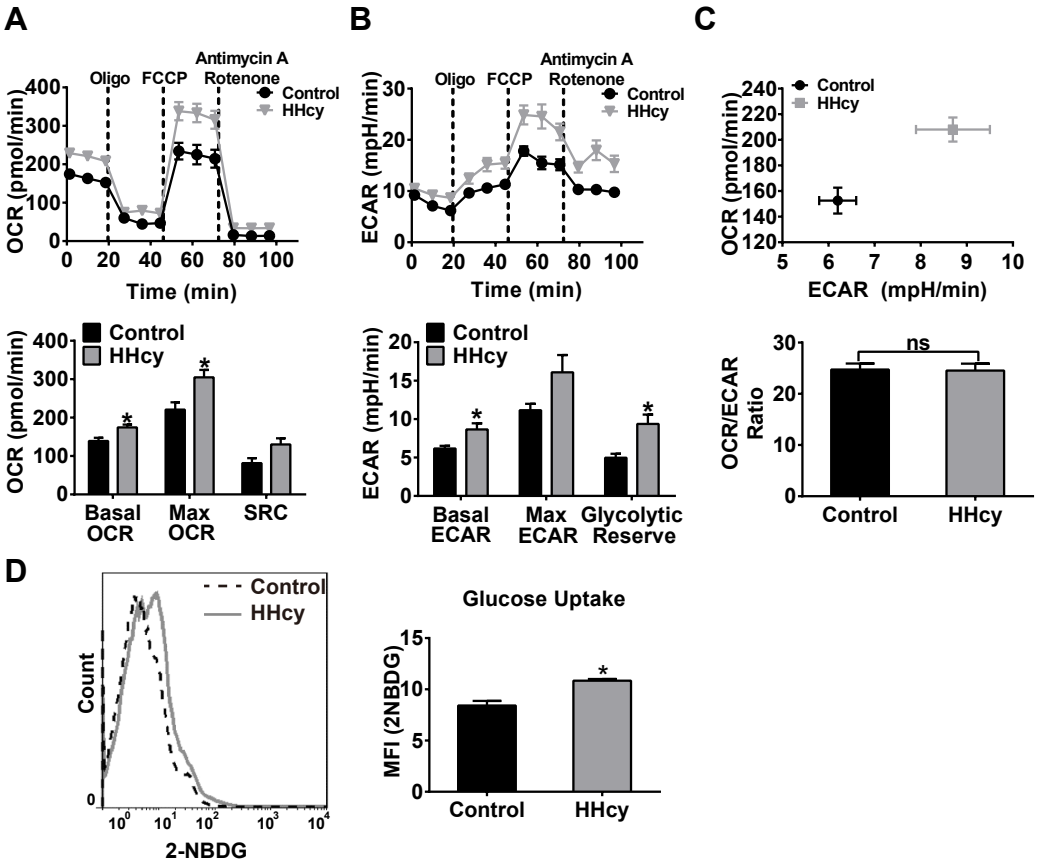

Figure S1. Metabolic changes in B cells from control and HHcy mice in response to LPS stimulation for 24 h. Splenic B cells purified from control and HHcy C57BL/6J mice were treated with 0.1 μg/ml LPS for 24 h *in vitro*. (**A** and **B**) Time courses of OCR and ECAR in B cells are shown. Basal and maximal OCR, SRC (*lower panel* in **A**), ECAR and glycolytic reserve (*lower panel* in **B**) and the OCR/ECAR ratio (**C**) were analyzed via extracellular flux analysis. (**D**) B cells were stained with 2-NBDG to assess glucose uptake via flow cytometry. The data shown are representative of three independent experiments (n = 3 /group). The data are presented as the mean ± SEM. **P* < 0.05 compared with the control. ns, not significant.

Figure S2

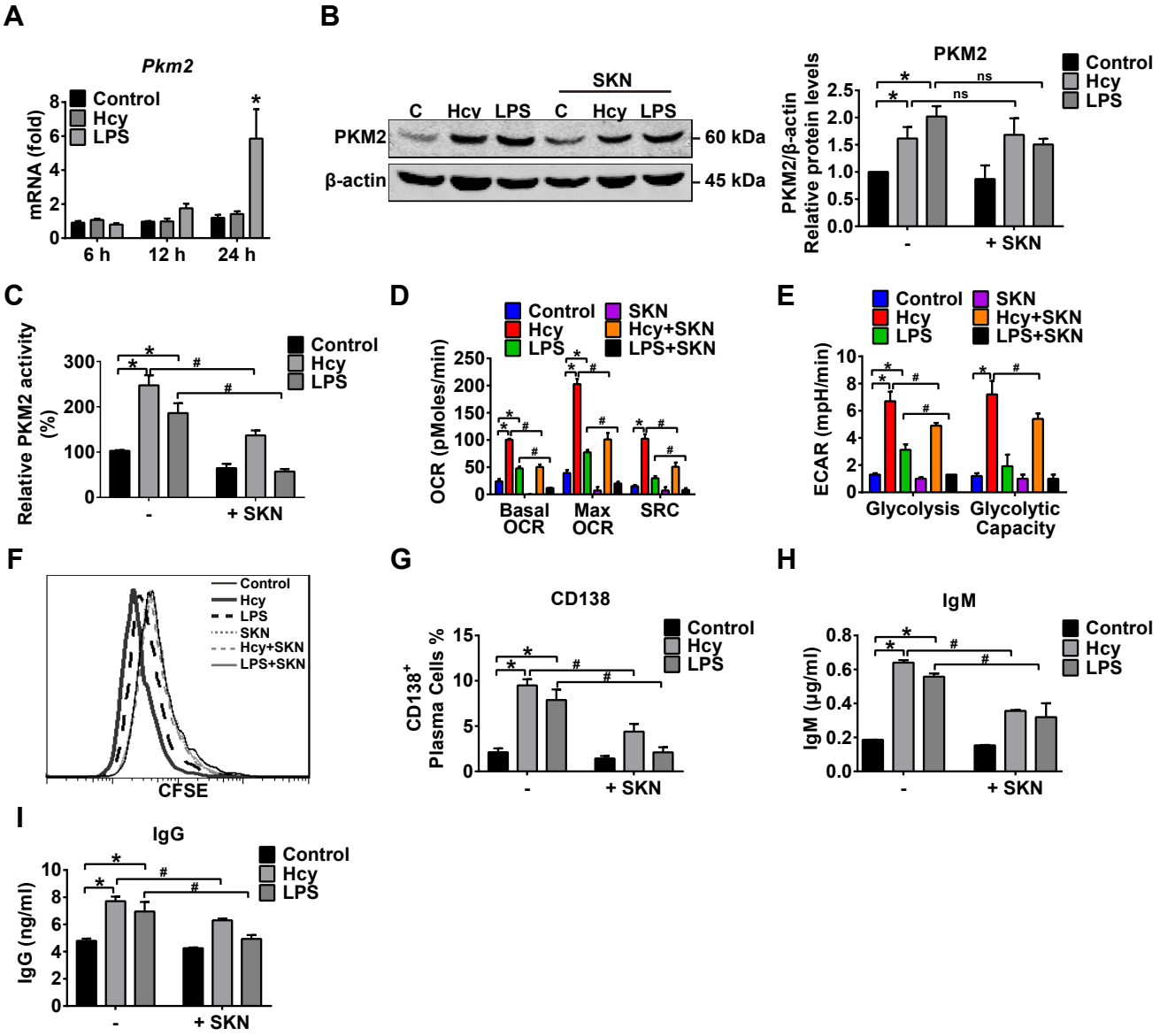

Figure S2. SKN inhibits PKM2-dependent metabolic changes and cell activation in B cells responded to Hcy or LPS. Splenic B cells purified from C57BL/6J mice were pre-incubated with 0.25 μM SKN for 30 min and then cultured with or without 100 μM Hcy or 5 μg/ml LPS for the indicated times. (**A**) Gene expression of *Pkm2* was measured via quantitative PCR in B cells at the indicated times. (**B**) Cell lysates were used to measure and quantify PKM2 protein expression at 24 h via Western blot analysis. (**C**) Measurement of PKM2 enzyme activity in B cells at 24 h. (**D** and **E**) Basal and maximal OCR, SRC, glycolysis and glycolytic capacity at 24 h were analyzed via extracellular flux analysis. Cell proliferation (**F**), $CD138^+$ plasma cells at 48 h (**G**), IgM (**H**)and IgG (**I**) levels in culture supernatants at 72 h are shown. The data shown are representative (*left panel* in **B**, **C**-**I**) and cumulative (**A**, *right panel* in **B**) of at least three independent experiments. The data are presented as the mean ± SEM. *$P < 0.05$ compared with the control. $^{\#}P < 0.05$ compared with Hcy or LPS group. ns, not significant.

Figure S3

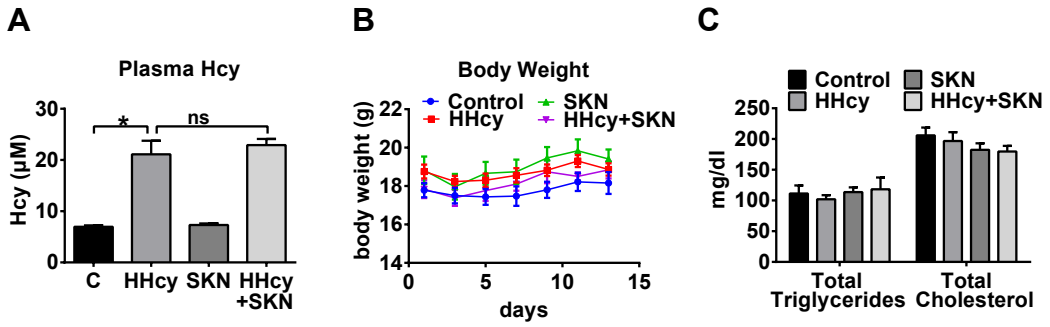

Figure S3. SKN treatment did not affect plasma Hcy, total triglycerides, total cholesterol and body weight. Mice were treated as described in Figure 8. (**A**) Plasma Hcy levels were measured via enzymatic cycling assay. (**B**) The body weight of the mice was recorded during the experiment. (**C**) Plasma total triglycerides and total cholesterol levels are shown. Data shown are representative of at least two independent experiments (n = 4-5 mice in each group). The data are presented as the mean ± SEM. **P* < 0.05 compared with the control. ns, not significant.

Table S1 Plasma Inflammatory Cytokine Levels in ApoE-/- Mice

	Control	HHcy	SKN	HHcy+SKN
IL-12p70 (pg/ml)	6.14 ± 0.27	7.12 ± 0.25	6.26 ± 0.17	6.07 ± 0.07
TNF-α (pg/ml)	4.30 ± 0.08	4.54 ± 0.08	4.23 ± 0.16	4.22 ± 0.14
IFN-γ (pg/ml)	2.45 ± 0.04	2.73 ± 0.09	2.70 ± 0.06	2.50 ± 0.06
MCP-1 (pg/ml)	12.62 ± 0.37	13.66 ± 0.49	12.80 ± 0.60	13.03 ± 0.44
IL-10 (pg/ml)	12.33 ± 0.44	12.55 ± 0.07	13.37 ± 0.20	12.87 ± 0.53
IL-6 (pg/ml)	5.97 ± 0.12	6.15 ± 0.11	6.30 ± 0.20	5.96 ± 0.12

Plasma inflammatory cytokines in ApoE-/- mice were detected by cytometric bead array mouse inflammation kit. Mice were fed with or without Hcy-containing drinking water in the presence or absence of SKN treatment.