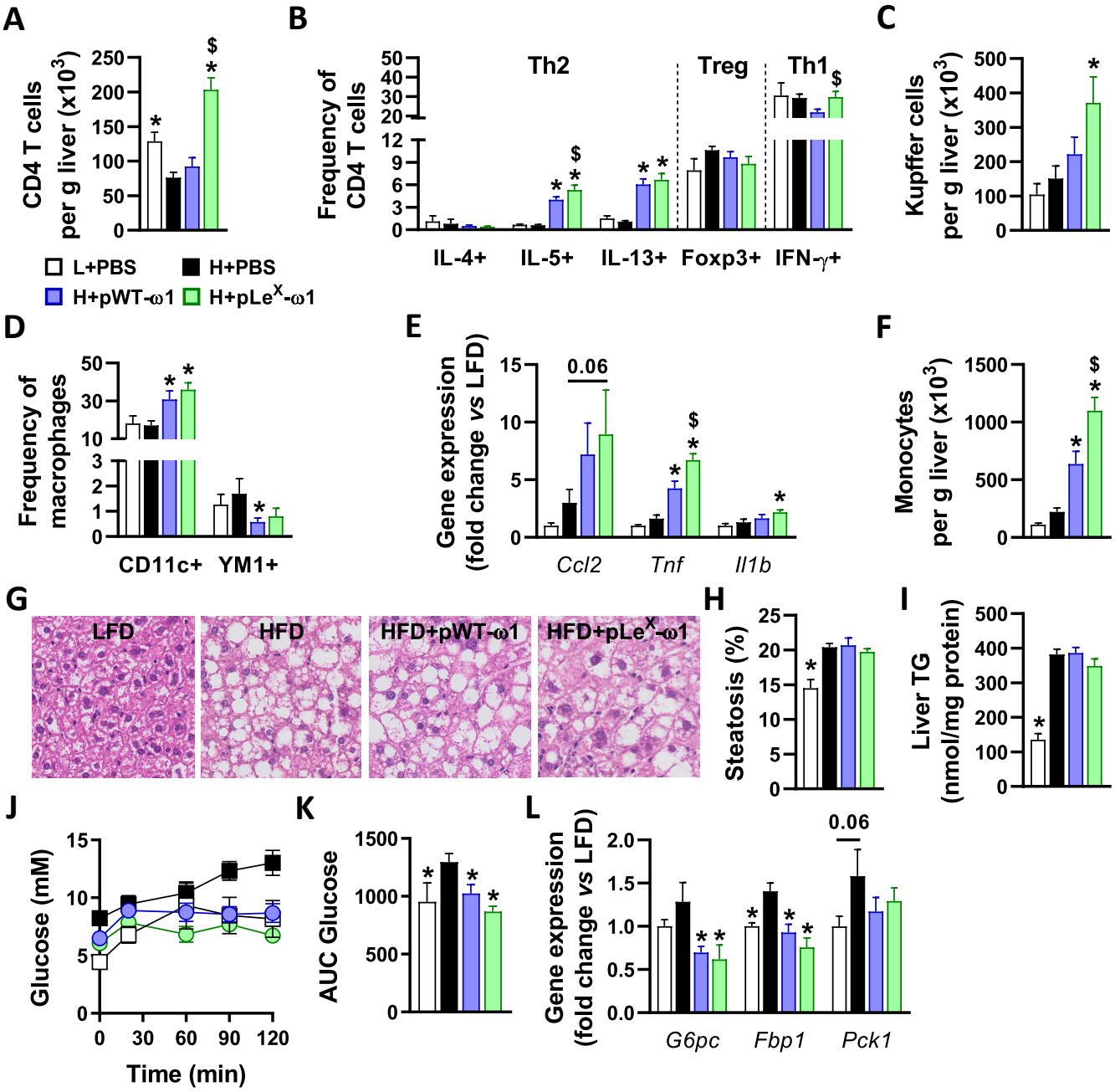

Figure S6. ω1 glycovariants do not affect hepatic steatosis, but reduce gluconeogenesis in obese mice. Mice were fed a LFD (white bars) or a HFD for 12 weeks, and next received biweekly intraperitoneal injections of PBS (black bars) or 50 µg pWT-ω1 (blue bars) or pLe^{X}-ω1 (green bars) during 4 weeks, as described in the legend of Figure 2. At sacrifice, CD45^{+} liver cells were isolated and analyzed by flow cytometry. The numbers of CD4 T cells (*A*), Kupffer cells (KC, *C*) and Ly6C^{hi} monocytes (*F*) per gram tissue, and the frequencies of T helper (*B*) and KC subsets (*D*) were determined. The mRNA expression of pro-inflammatory genes (*E*) was determined. Hepatic steatosis was assessed using hematoxylin/eosin staining in fixed tissues (*G-H*) and hepatic triglycerides content was determined (*I*). Intraperitoneal pyruvate tolerance test was performed during week 4. Blood glucose levels were measured at the indicated time points (*J*) and the AUC of the glucose excursion curve was calculated (*K*). The mRNA expression of the main gluconeogenic genes was determined (*L*). Data shown are a pool of at least two independent experiments, except for J-K. Results are expressed as means ± SEM. * $P<0.05$ *vs* HFD, $ $P<0.05$ *vs* pWT-ω1, # $P<0.05$ *vs* WT (n = 5-18 mice per group in A-L, and 3-6 mice per group in J-K).