Supporting Information

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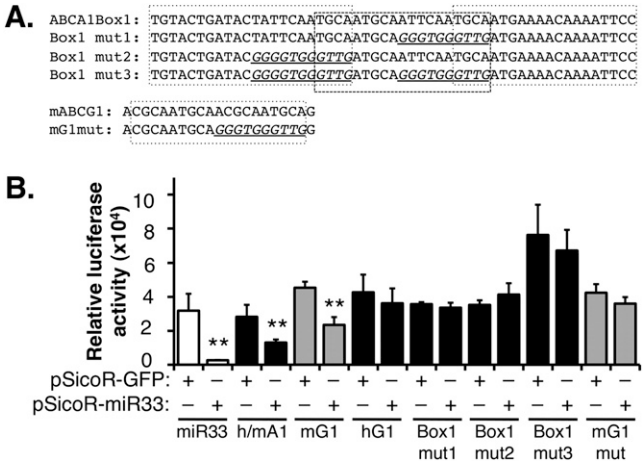

Fig. S1. Mutation of specific sequences abolishes miR-33–mediated silencing. (*A*) Natural and mutated ABCA1 Box 1 and ABCG1 response elements for miR-33. (*B*) HEK293 cells were transfected as described in Fig. 2, and luciferase activity was analyzed 48 h after transfection. Data are mean ± SD of three independent experiments in duplicate.

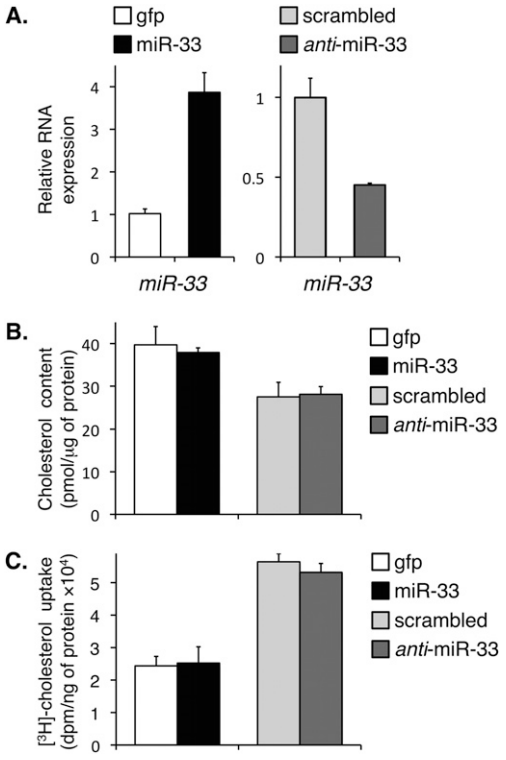

Fig. S2. Manipulation of miR-33 expression levels does not alter cholesterol uptake in HEK293 cells. Cells were transfected with an empty plasmid or a plasmid encoding miR-33, or with scrambled or miR33-specific hairpin inhibitors, as described in Fig. 3 *A* and *B*. (*A* and *B*) The expression of miR-33 (*A*) and total cholesterol and protein content (*B*) in the cells were measured 36 h posttransfection. (*C*) Some wells were then incubated overnight in media supplemented with 1 μCi/mL [^{3}H]-cholesterol. The uptake of the labeled cholesterol (measured in dpm/ng of cell protein) was not altered following overexpression or silencing of miR-33. Data for the different panels are mean ± SD of four (*A*) and eight (*B* and *C*) individual samples.

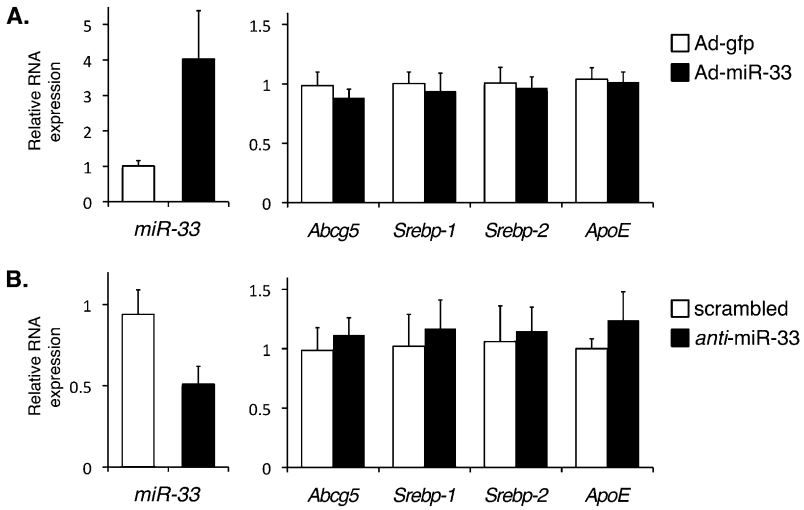

Fig. S3. Manipulation of miR-33 expression levels does not alter the expression of selected lipid-related genes in liver. Mice were infused with adenoviral vectors (*A*) or with antisense oligonucleotides (*B*), as described in Fig. 3 *C–F*. The expression of hepatic miR-33 and selected genes involved in lipid homeostasis was analyzed by real-time PCR. Data are mean ± SD; $n = 5$.