Figure S1. Effect of simvastatin and atorvastatin on CCA cell toxicity. (A) HuCCA-1, (C) KKU-100, (E) KKU-M213 and (G) RMCCA-1 cells were seeded and treated with a variety of concentrations of simvastatin (0-100 μM). (B) HuCCA-1, (D) KKU-100, (F) KKU-M213 and (H) RMCCA-1 cells were treated with various concentrations of atorvastatin (0-100 μM) for 48 h. Cell toxicity was determined using a MTT assay. Data presented the representatives of three independent experiments (mean ± SEM, n=3). Statistical significance was determined by Student's t-test. *P<0.05; **P<0.01; ***P<0.001 vs. 0 μl simvastatin or atorvastatin treatment.

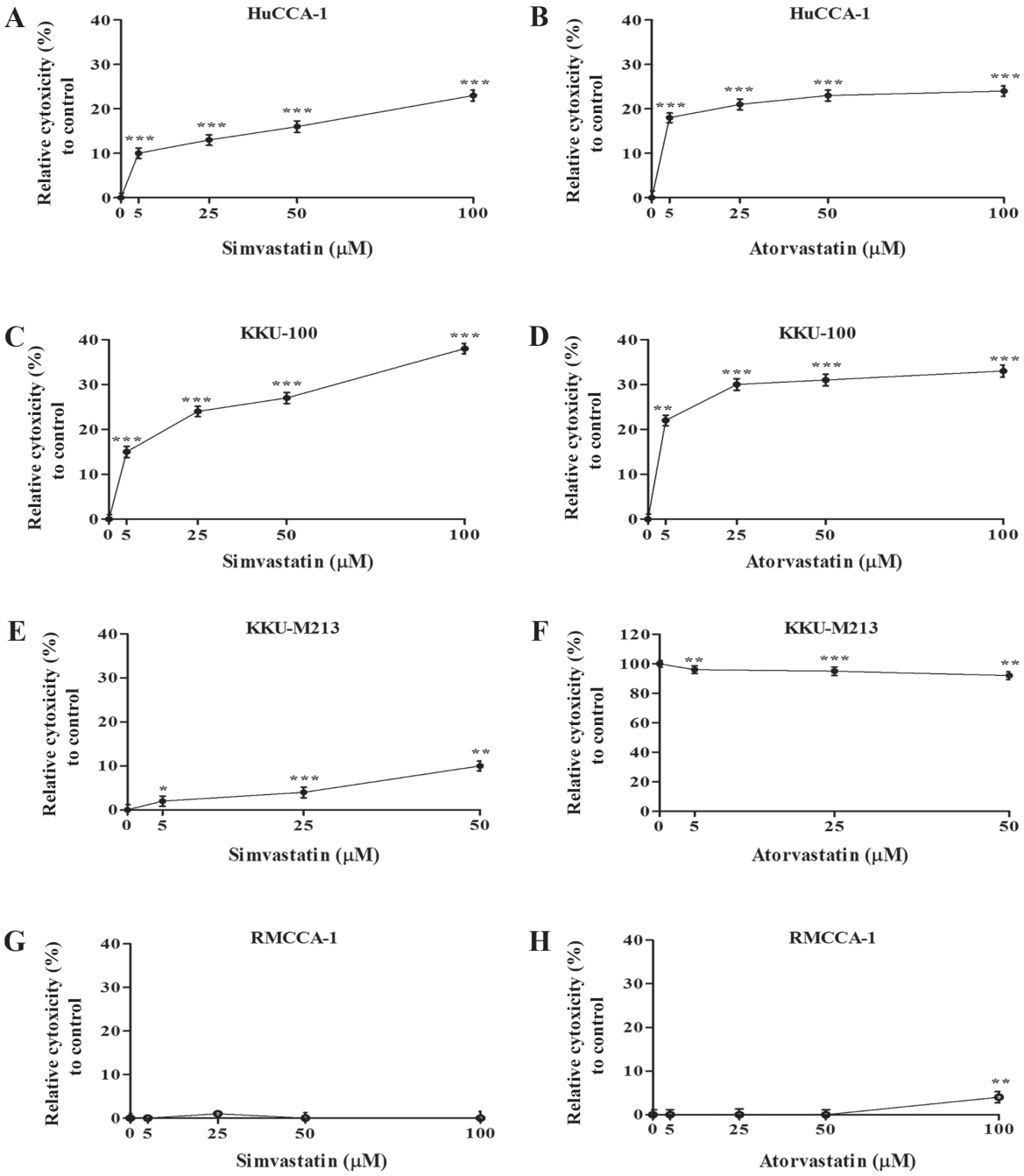

Figure S2. siABCG1 suppressed ABCG1 protein expression, but ABCA1 protein expression remained in the HuCCA-1 cell line. Cells were transfected with siRNA duplexes against ABCG1 at final concentration of 120 pmole for a variety of time points. After transfection, protein expression was evaluated using (A) western blot analysis at various time points and (B) band intensity was evaluated. Data are representative of three independent experiments (mean ± SEM; n=3). Statistical significance was determined using a Student's t-test. $^{*}P<0.05$; $^{**}P<0.01$; $^{***}P<0.001$ vs. siControl. ABCA1 protein expression was also evaluated using (C) western blot analysis at 120 h post transfection of siABCG1 (n=3). Si, small interfering; ABC, ATP-binding cassette.

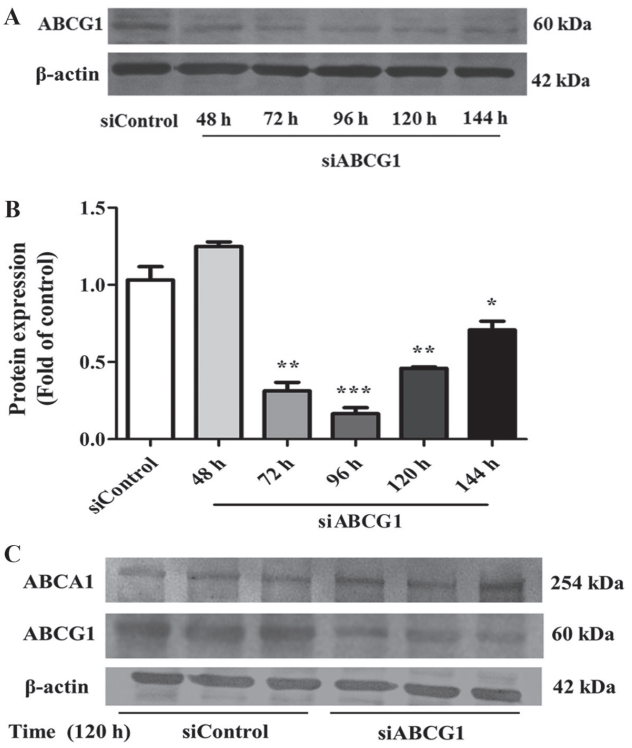

Figure S3. Effect of ABCG1 knockdown on cholesterol efflux in HuCCA-1. siRNA-transfected HuCCA-1 cells were loaded with bodipy cholesterol at 12.5 μM (low) and 25 μM (high) for 1 h. After the addition of HDL (for 6 h), cholesterol efflux was determined. The percentage of bodipy cholesterol efflux to HDL was compared between wild type, siControl and siABCG1. Data are from one experiment (mean ± SEM; n=3). ABC, ATP-binding cassette; Si, small interfering; HDL, HDL, high density lipoprotein.