SUPPLEMENTARY FIGURE LEGENDS

Fig. S1. Effect of sorafenib on the intracellular mitoxantrone accumulation in the MCF7/MX cells. mitoxantrone accumulation in parental MCF7/WT cells or MCF7/MX, which express wild-type ABCG2, following a 30 minute incubation in the absence or presence of soreafenib or Ko143 (2.5 μM). The data are means ± SD from three independent experiments (*P<0.01, compared with 0.1% DMSO vehicle).

Fig. S2. Chemo-sensitization of drug resistance by sorafenib in the MCF7/MX cells. MCF7/WT or MCF7/MX cells, which express ABCG2, were treated with or without IC_{10} of mitoxantrone in the absence or presence of different concentrations of sorafenib or Ko143 (CH: 2.5 μM; CM: 1.25 μM; CL: 0.625 μM) followed by SRB assay. The results are presented as survival rate of MDR cells relative to DMSO vehicle and shown as means ± SD of three independent experiments. Ko143 was used as an ABCG2 inhibitor control

Fig. S3. Effect of sorafenib on ABCG2 expression level in the MCF/MX cells. MCF7/MX cells, which express wild-type ABCG2, were treated with DMSO vehicle, 2.5 μM PZ-38, or sorafenib at 1.25 μM and 2.5 μM concentrations for 3 days followed by western blot analysis for ABCG2 expression. PZ-38 was chosen be a positive control, while 0.1% DMSO was used as negative control.

Fig. S4. Inhibition of ABCG2 function by sorafenib on mitoxantrone efflux. HEK293/ABCG2 cells were incubated with increasing concentrations of sorafenib for 1 h followed by FACS analysis of mitoxantrone level. The data are presented as the mean mitoxantrone accumulation ± S.D. (n=3), normalized to the mitoxantrone accumulation observed in resistant cells incubated with 2.5 μM Ko143 (100% inhibition of ABCG2 function).