Figure S1. Outline of the proposed mechanisms. Garcinol treatment inhibits the phosphorylation of AKT and in turn decreases the proliferation and invasion of HGC-27 cells by downregulating the expression of cylinD1, MMP-2 and MMP-9, thereby exerting its antitumor effects. The phenotypic alterations caused by garcinol can be rescued by SC79, a specific agonist of AKT, at the cellular level. MMP, matrix metalloproteinase.