Supplemental Figure Legends.

Figure S1. MDA-7/IL-24 activates PERK, JNK and p38 MAPK and inactivates ERK1/2 in SKOVIII cells. SKVOIII cells were plated and 24 h after plating infected with Ad.5-*cmv* or Ad.5-*mda*-7 (50 m.o.i.). Forty eight h after infection, cells were isolated, and processed for SDS PAGE and blotting to determine the expression and the phosphorylation of the indicated proteins (n = 2).

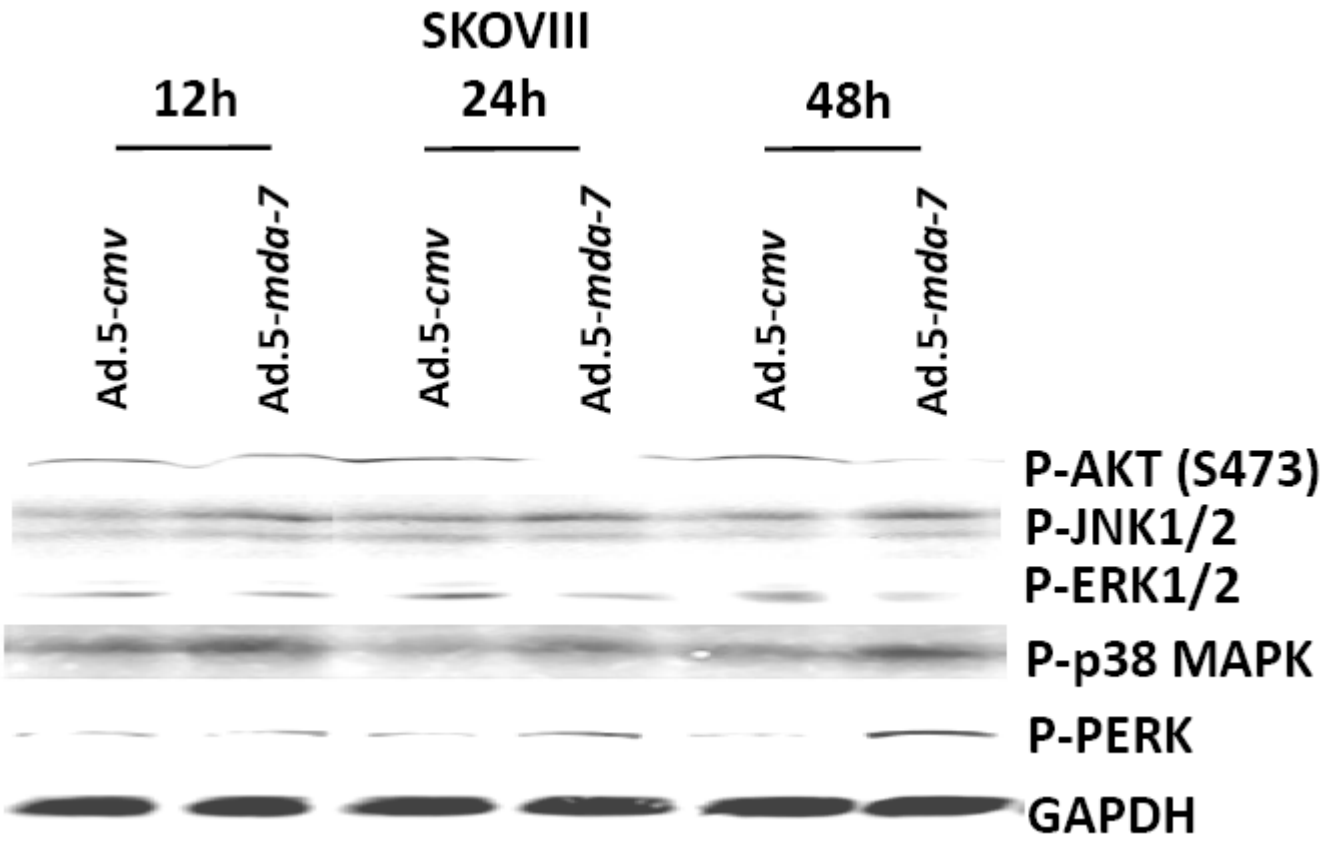

Figure S2. GST-MDA-7 enhances BBR3464 lethality in primary human GBM cells. Primary human glioma cells (GBM6) were plated and 24 h after plating were treated with GST or GST-MDA-7 (20, 40 nM). Cells were treated 30 min later with vehicle or with BBR3464 (100, 200 nM). Cells were incubated for 96 h after which time cell viability was assessed in triplicate by trypan blue assay (± SEM, n = 3).

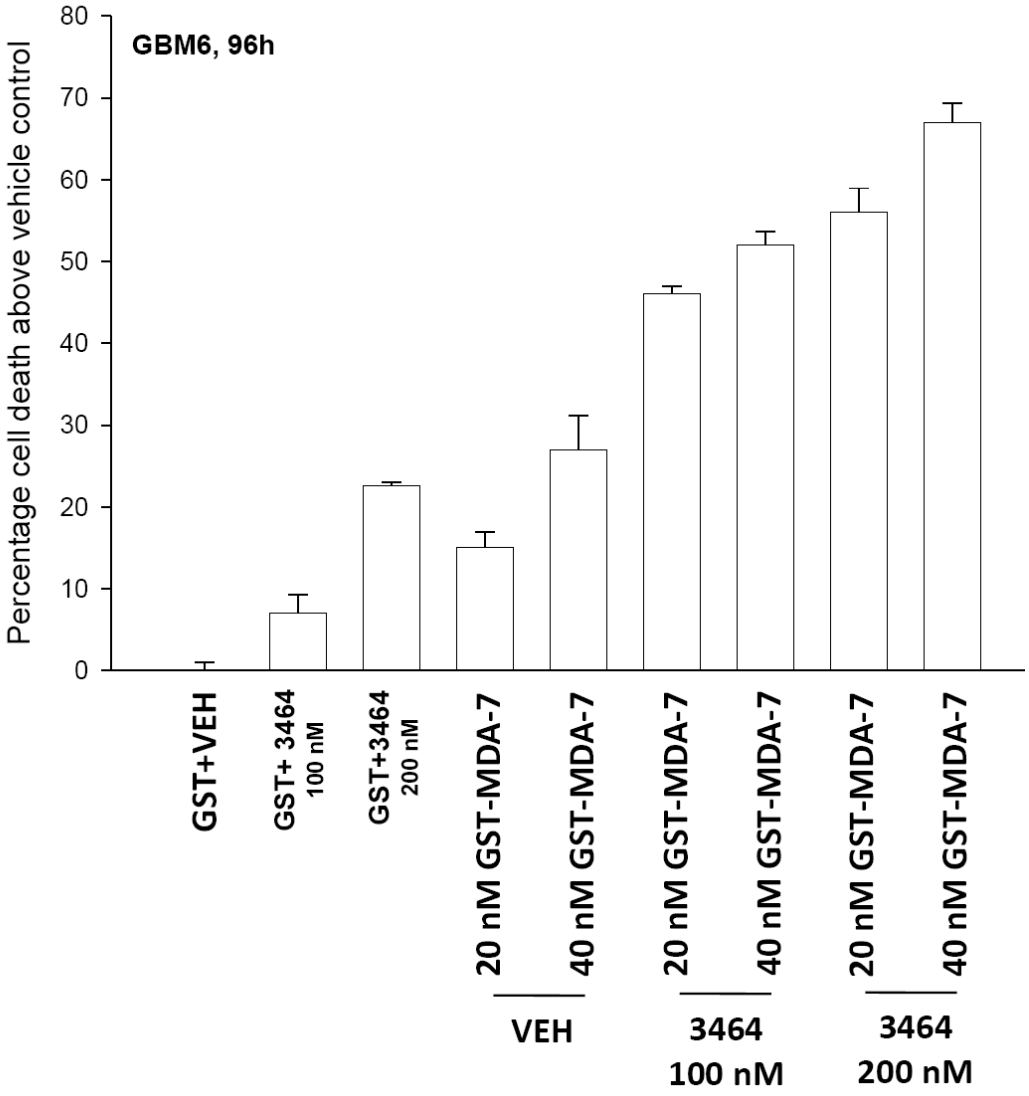

Figure S3. GST-MDA-7 enhances cisplatin lethality in primary human GBM cells. Primary human glioma cells (GBM6) were plated and 24 h after plating were treated with GST or GST-MDA-7 (10, 15, 20 nM). Cells were treated 30 min later with vehicle or with cisplatin (500, 750, 1000 nM). Cells were incubated for 96 h after which time cell viability was assessed in triplicate by trypan blue assay (± SEM, n = 3).

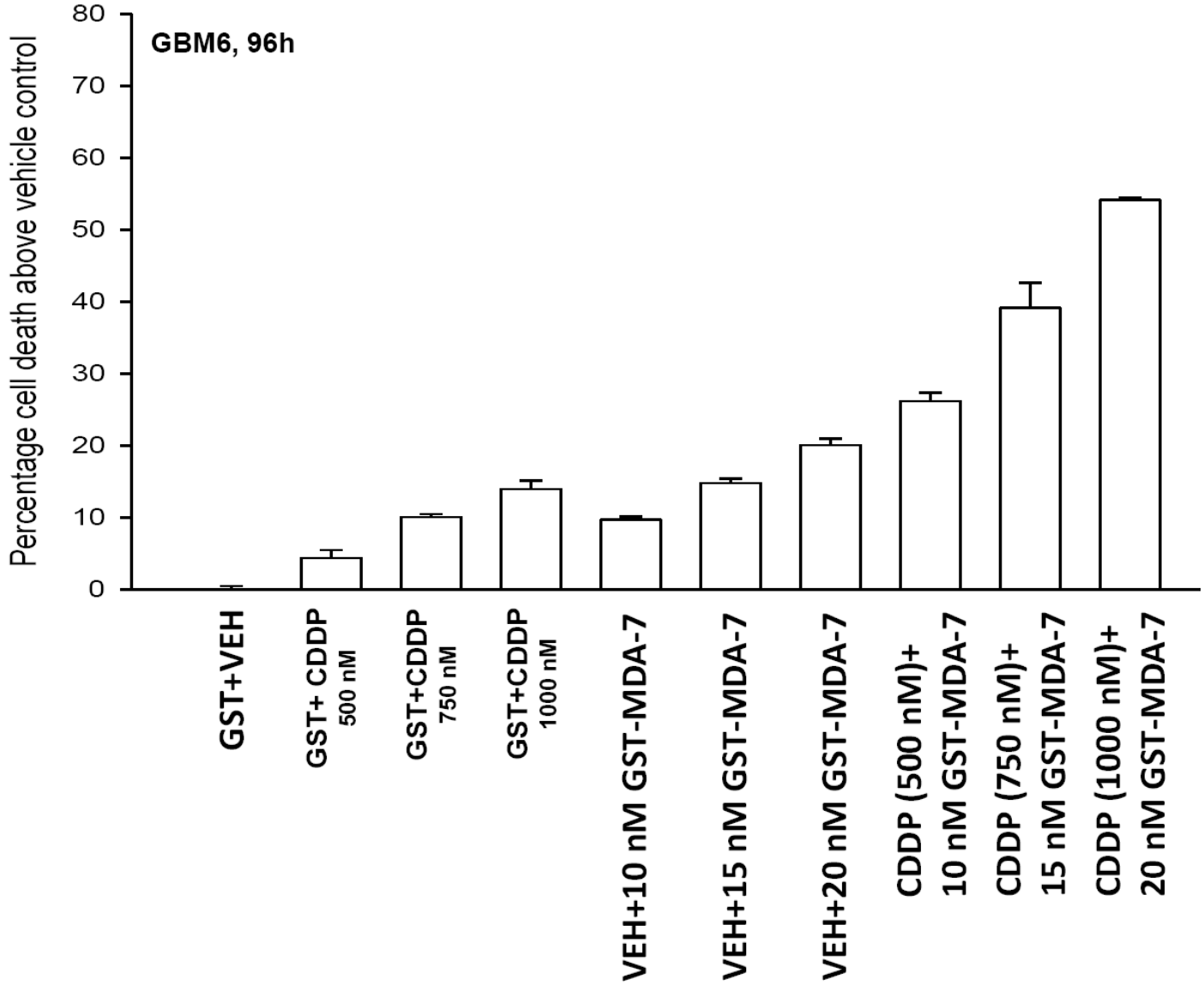

Figure S4. Ad.5-*mda*-7 and cisplatin interact to kill primary human ovarian cancer cells in vitro. Primary human OCCs were obtained from patient ascites. Cells were passaged once and plated for analyses. Cells 24 h after plating infected with Ad.5-*cmv* or Ad.5-*mda*-7 (50 m.o.i.)., and 12 h after infection cells were treated with vehicle (DMSO) or with cisplatin (CDDP, 3 μM). Seventy two h and 96 h after virus infection cell viability was determined by trypan blue exclusion assay on isolated cells (± SEM, n = 3).

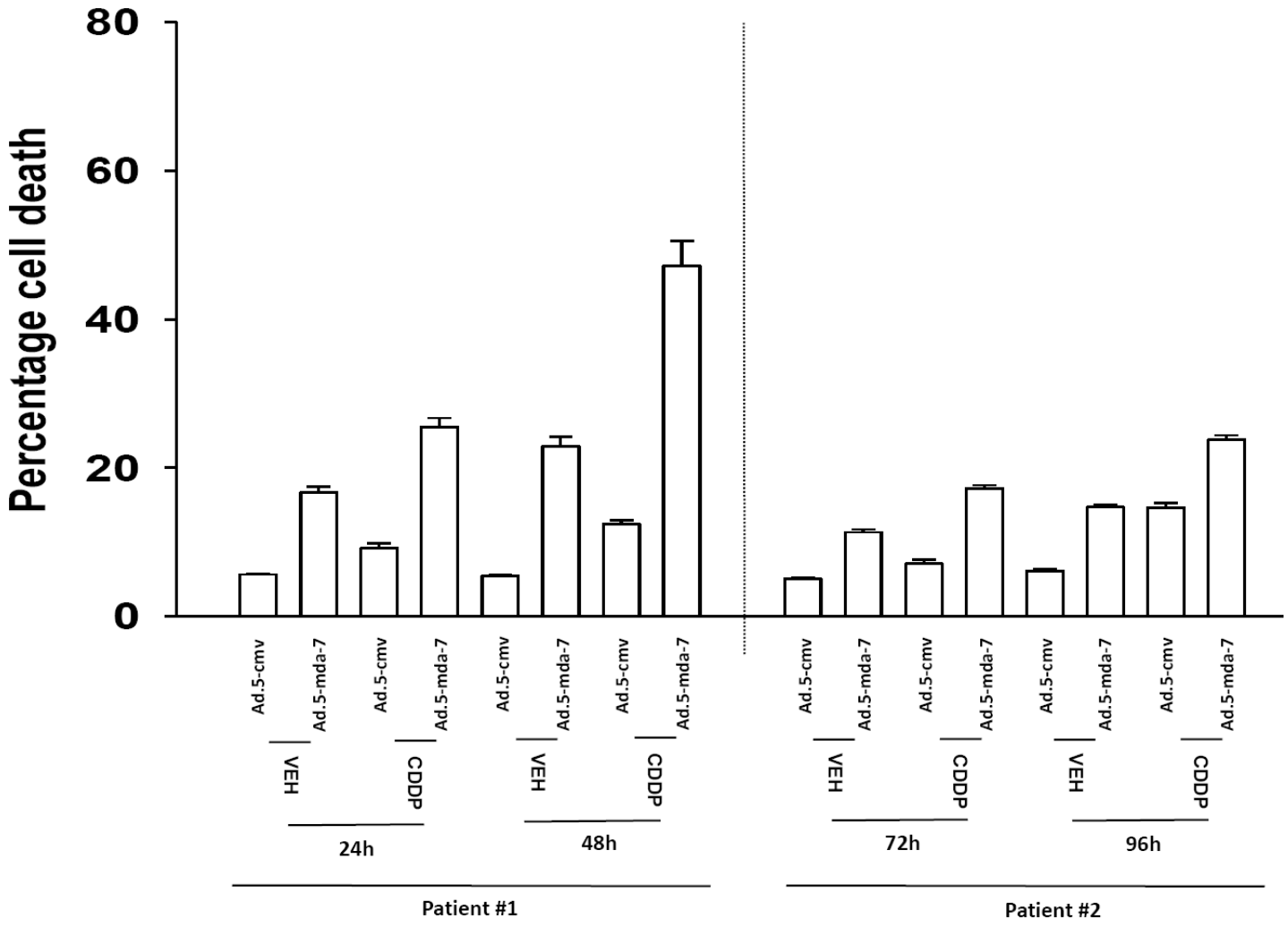

Figure S5. Knock down of ATG5 suppresses Ad.5-*mda*-7–induced cell killing in OCCs. OVCAR cells 24 h after plating infected with Ad.5-*cmv* or Ad.5-*mda*-7 (50 m.o.i.), and 12 h after infection transfected with either a vector control plasmid to express a non-specific scrambled siRNA (siSCR) or with a plasmid to knockdown expression of ATG5 (siATG5). Twenty four h after virus infection cells were treated with vehicle (DMSO) or with cisplatin (CDDP, 3 μM). Seventy two h after virus infection cell viability was determined by trypan blue exclusion assay on isolated cells (± SEM, n = 3).

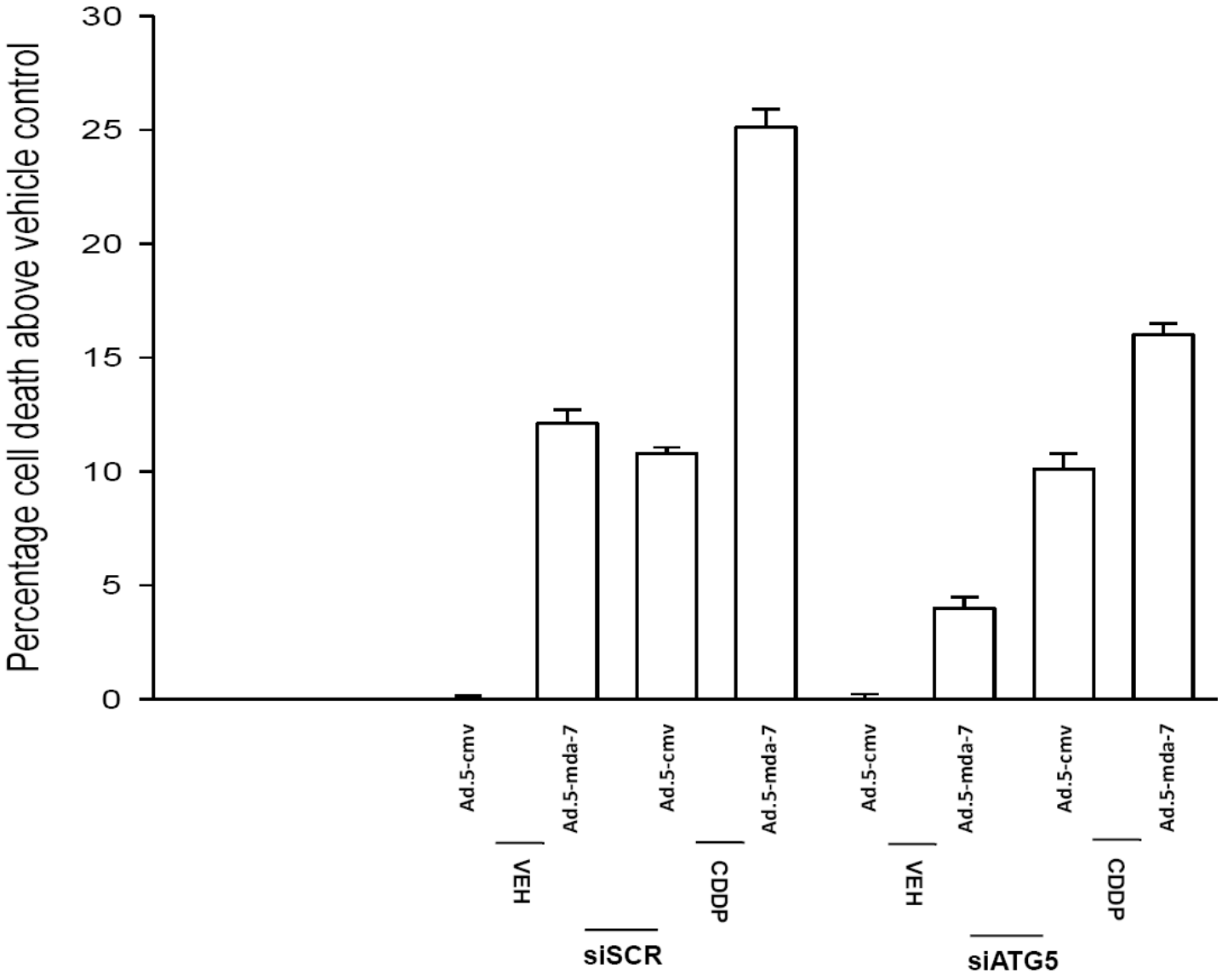

Figure S6. Ad.5-*mda*-7 and [cisplatin + paclitaxel] treatment interact in a greater than additive fashion to kill ovarian cancer cells in colony formation assays. OVCAR cells were plated as single cells in 60 mm dishes (250-1000 cells/dish) in sextuplicate and infected with Ad.5-*cmv* or Ad.5-*mda*-7 (50 m.o.i.), and 24 h after infection as indicated cells were treated with paclitaxel (10 nM) and cisplatin (CDDP, 3 μM), as indicated. Drug containing media was removed 48 h later and cells permitted to form colonies for the following 14 days. Data are normalized to 1.00 for virus infection + vehicle treatment for each virus condition. Ad.5-*mda*-7 reduced survival to 0.83 of control (± SEM, n = 2). * $p < 0.05$ less than corresponding value in Ad.5-*cmv* infected cells.