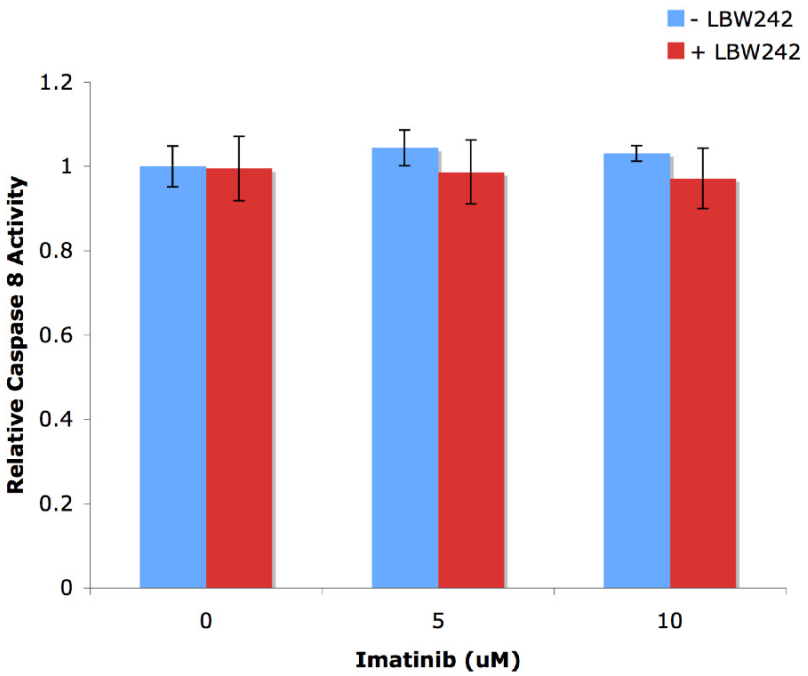
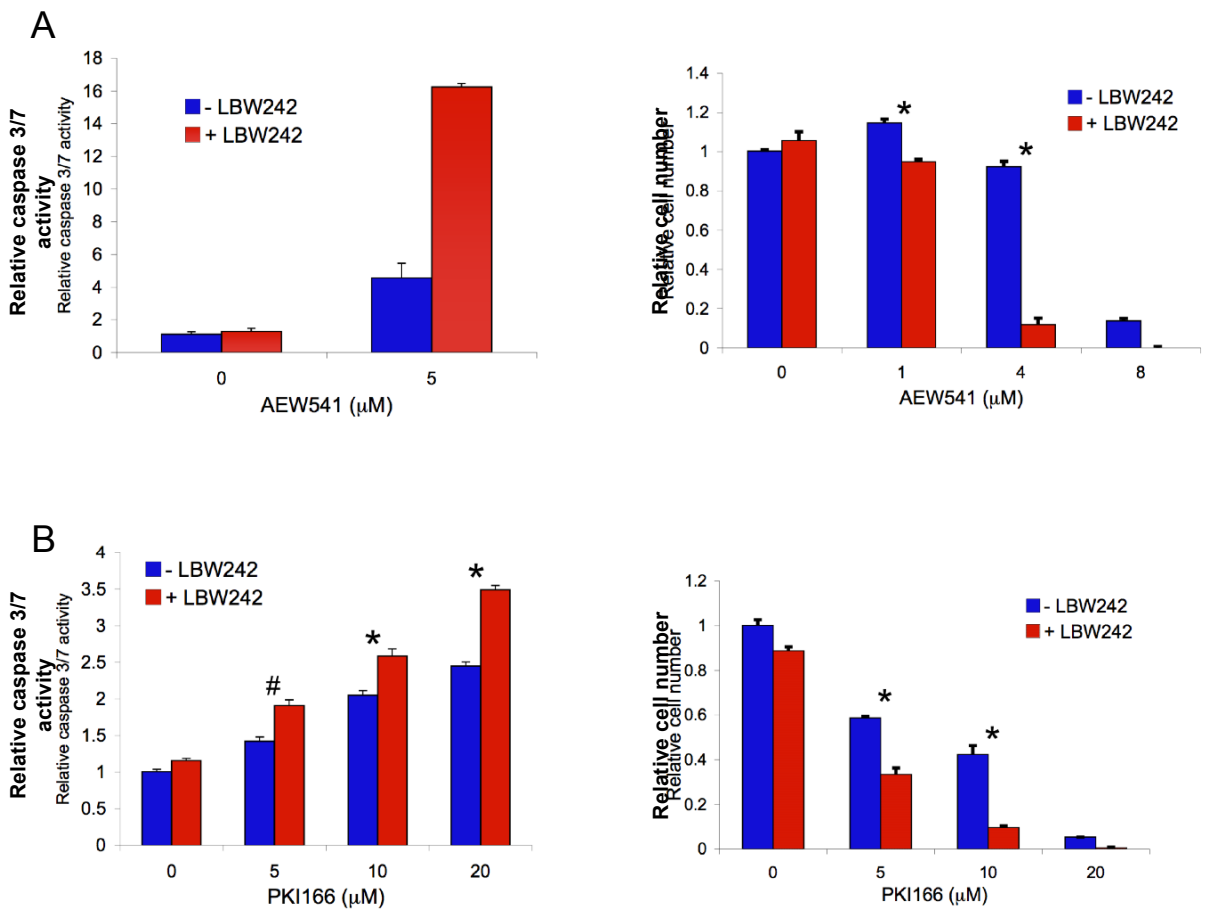
Supplemental Figure 1



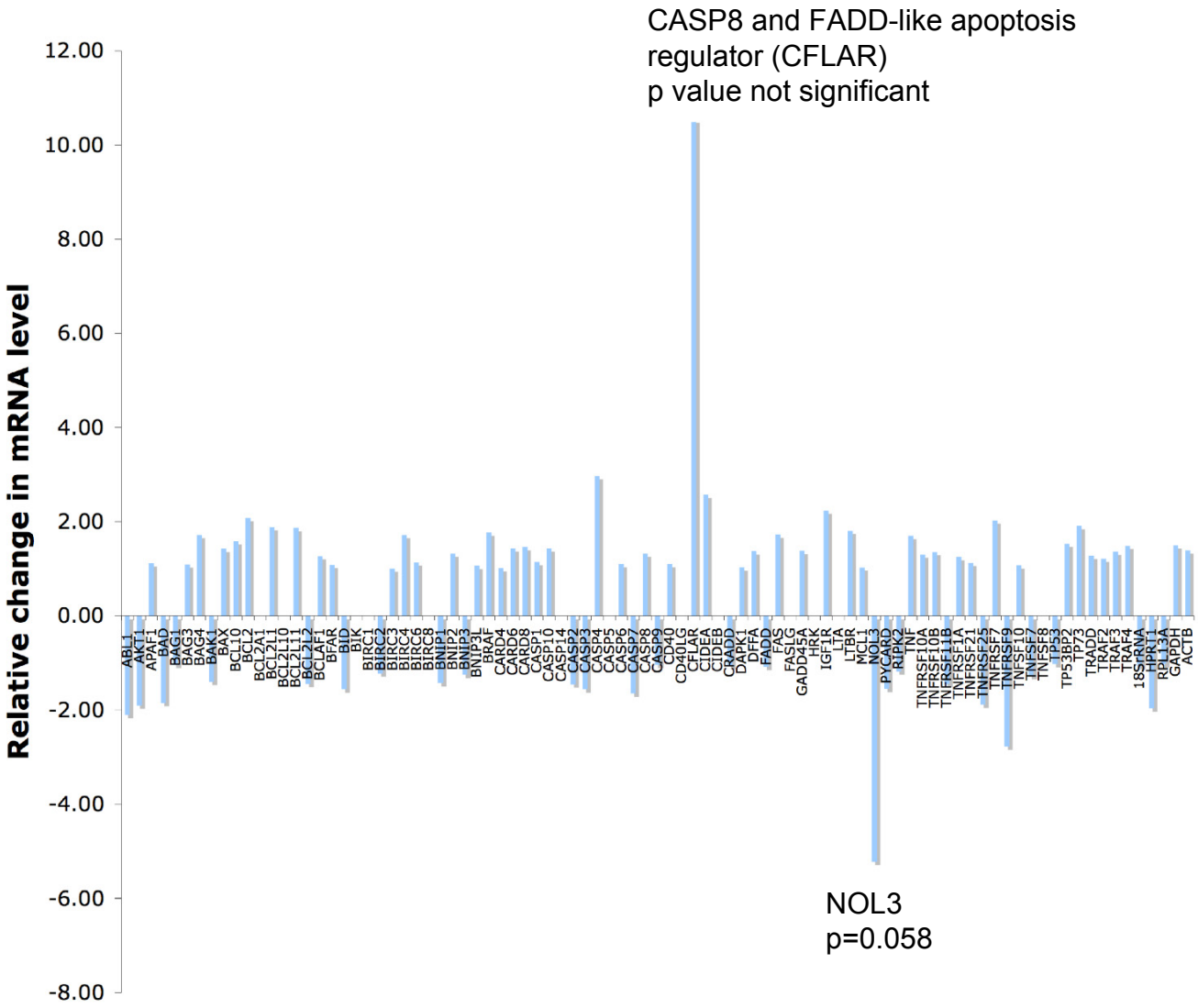
Supplemental Figure 1. PDGFR and IAP inhibition do not activate the extrinsic pathway. U87 cells were seeded in 96 well plates and treated with LBW242 (50 μM) and / or imatinib at the concentrations indicated. After 48 hours incubation relative caspase 8 activity was measured via luminescent assay. No significant difference was seen between any of the treatment arms. Experiments were conducted in triplicate, with data expressed as mean ± SEM.

Supplemental Figure 2



Supplemental Figure 2. Synergistic induction of apoptosis by LBW242 in combination with IGF1R and EGFR inhibition. LN827 cells were seeded in 96 well plates and treated with the IGF-1R inhibitor AEW541 **(A)** or the EGFR inhibitor PKI166 **(B)** at the concentrations indicated. Cells were also treated with either LBW242 (50 μM) or DMSO as a control. After 72 hours incubation relative cell survival was measured by MTS assay and caspase 3/7 activity at 48 hours was measured via fluorescent assay. Experiments were conducted in triplicate, with data expressed as mean ± SEM. *p < 0.001 comparing results +/- LBW242, #p < 0.01 by Student's t-test (two-tailed).

Supplemental Figure 3



Supplemental Figure 3. NOL3 expression is suppressed following administration of imatinib in LN827 cells. Graph depicts results of multiplexed quantitiative RT-PCR analysis of the indicated genes in the apoptosis pathway in LN827 cells. Data represents the average gene expression levels for 3 separate samples treated with or without imatinib, 10 μM for 36 hours. Expression levels of CFLAR were highly erratic, and the apparent increase is not statistically significant. 4 independent follow-up experiments showed no significant change in CFLAR expression following treatment with imatinib (relative change -1.13, 1.05, 1.32 and 1.17)