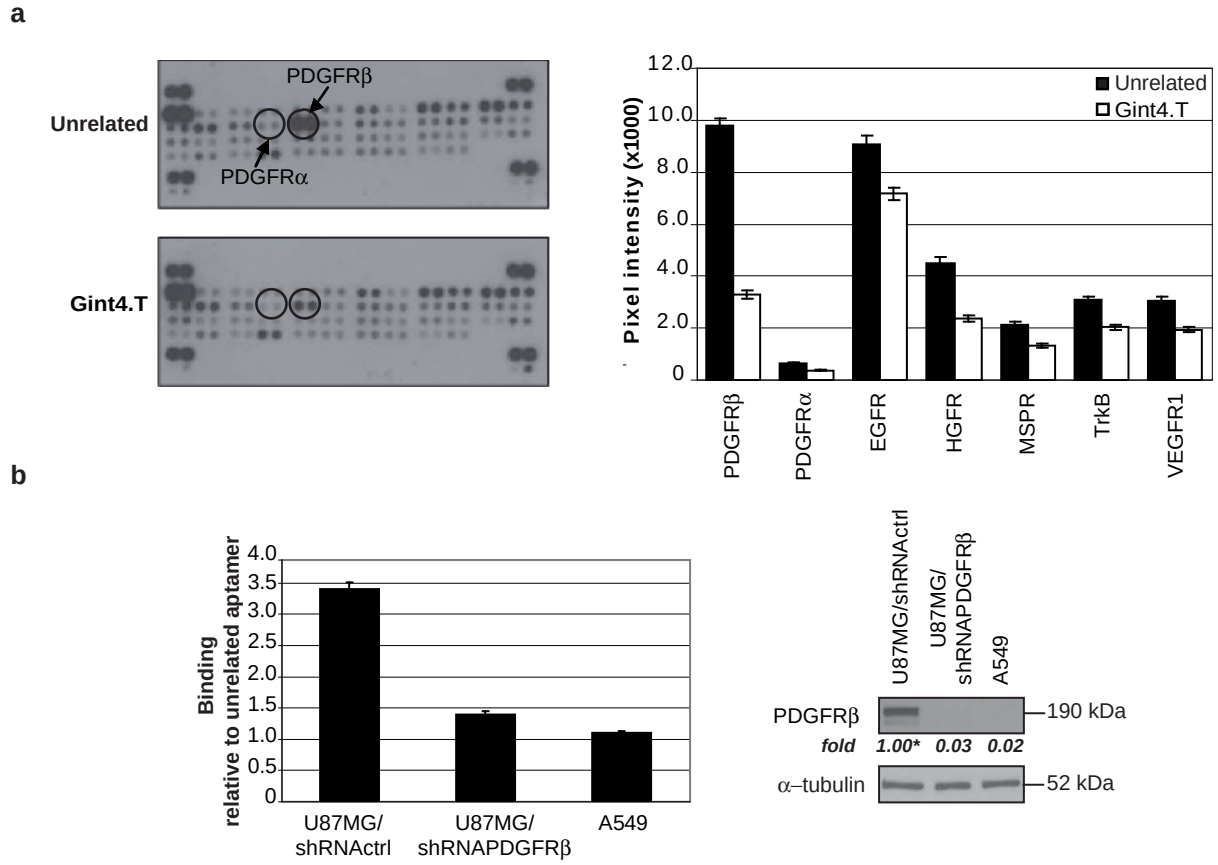

Figure S1. Gint4.T specifically interacts with PDGFRβ. (**a**) U87MG cells were serum starved overnight and then incubated with culture medium supplemented with 20% FBS for 10 minutes in the presence of 200nM Gint4.T or the unrelated aptamer and cell extracts were prepared. 200 µg-lysates were incubated on RTK antibody arrays. Phosphorylation levels were determined by subsequent incubation with anti-phosphotyrosine horseradish peroxidase. Arrows indicate phosphorylated PDGFRβ and PDGFRα. The pixel intensity associated to the phosphorylation status of the receptors whose serum-dependent activation is altered following Gint4.T treatment is reported and error bars depict means ± SD. (n = 4). (**b**) Left, binding of 100 nM radiolabeled Gint4.T to U87MG cells following 72 h-transfection with a specific PDGFRβ short hairpin RNA (U87MG/shRNAPDGFRβ) or a nonrelated shRNA (U87MG/shRNActrl), or to A549 cells. Right, lysates from transfected U87MG or A549 cells were immunoblotted with anti-PDGFRβ antibodies. Values below the blot indicate signal levels relative to shRNActrl-transfected cells, arbitrarily set to 1 (labeled with asterisk). Equal loading was confirmed by immunoblot with anti-α-tubulin antibody. Molecular weights of indicated proteins are reported. The results are expressed relative to the background binding detected with the unrelated aptamer. Error bars depict means ± SD. (n = 3).

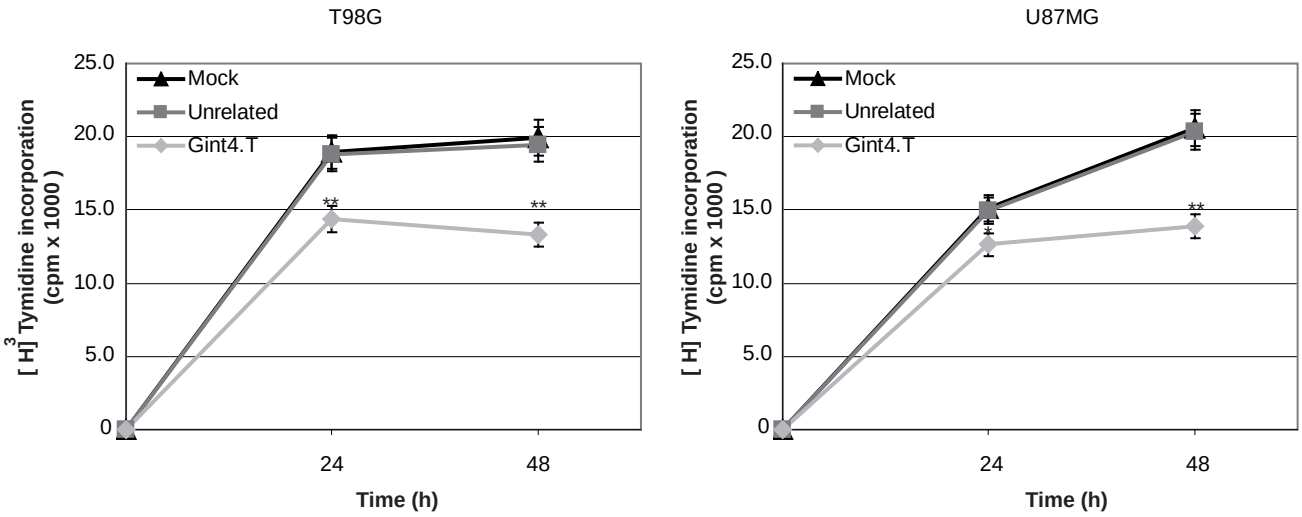

Figure S2. Gint4.T reduces [^{3}H]-thymidine incorporation. T98G and U87MG cells were either mock-treated or treated for 24 and 48 hours with Gint4.T or the unrelated aptamer and proliferation was determined by [^{3}H]-thymidine incorporation. **, $p<0.005$; *, $p<0.05$ relative to unrelated (n=6).

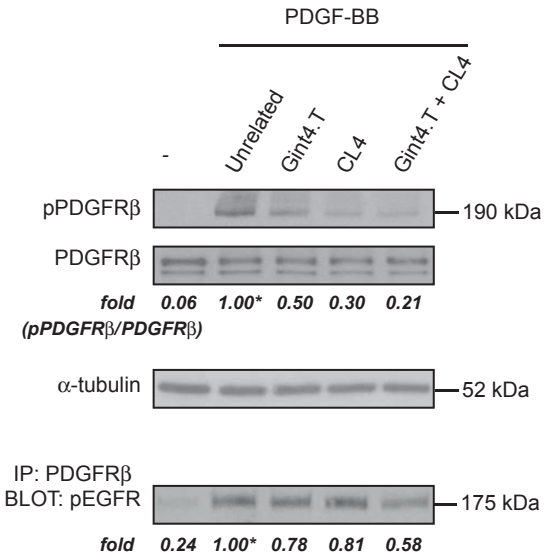

Figure S3. Gint4.T cooperates with CL4 in preventing EGFR transactivation in U87MG cells. Serum-starved U87MG cells were either left untreated or stimulated with PDGF-BB in the presence of Gint4.T, CL4, Gint4.T plus CL4, or the unrelated aptamer. Cell lysates were either immunoblotted with anti-pPDGFRβ and anti-PDGFRβ antibodies or immunoprecipitated with anti-PDGFRβ antibody and immunoblotted with anti-pEGFR, as indicated. Equal loading was confirmed by immunoblot with anti α-tubulin antibody. Values below the blots indicate signal levels relative to PDGF-BB stimulated cells in the presence of the unrelated aptamer, arbitrarily set to 1 (labeled with asterisk). Molecular weights of indicated proteins are reported.

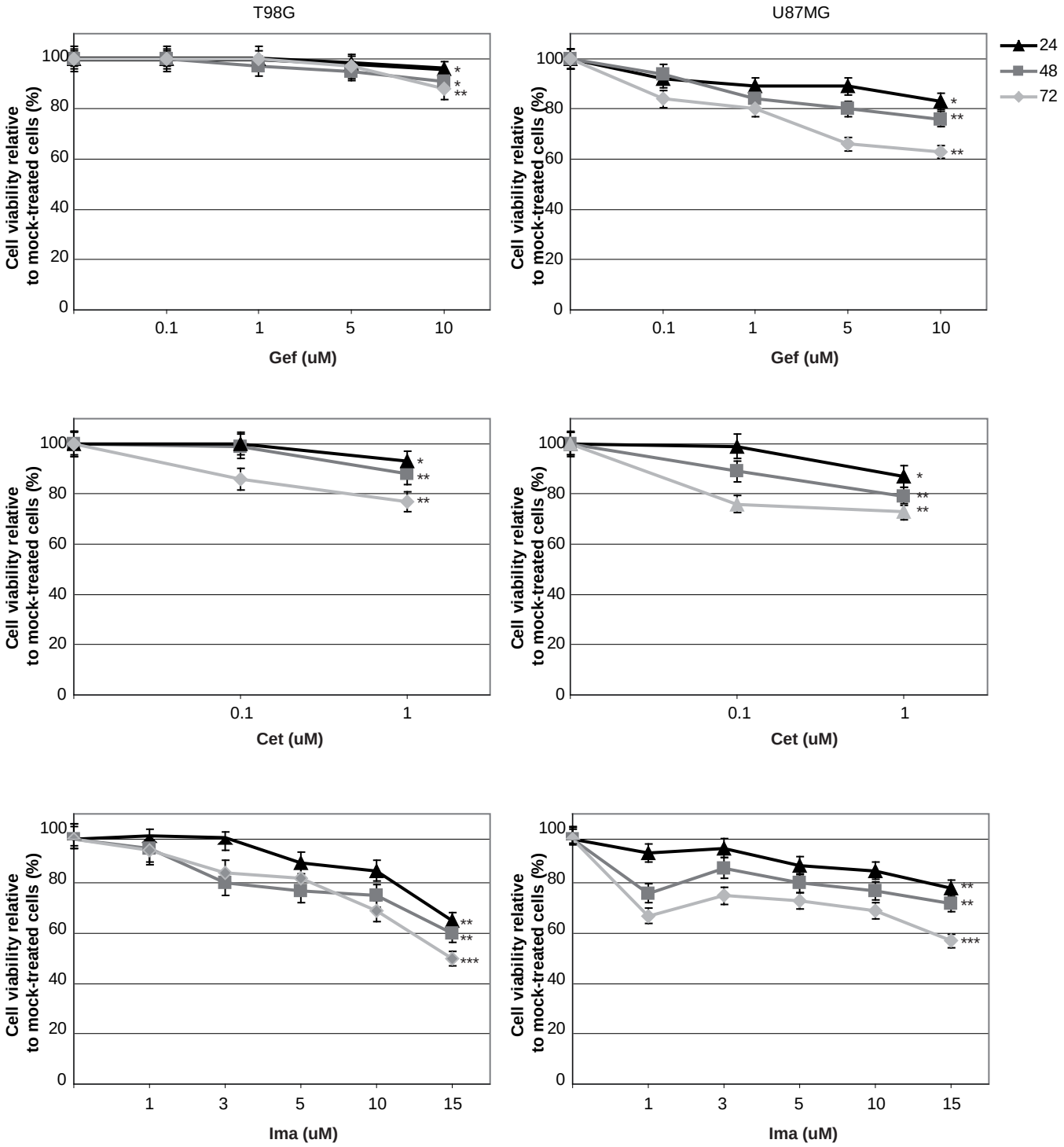

Figure S4. Effect of Gefitinib (Gef), Cetuximab (Cet) and Imatinib (Ima) on cell viability. T98G and U87MG cells were either mock-treated or treated for 72 hours with increasing concentrations of the three inhibitors, as indicated. ***, $p<0.0001$; **, $p<0.005$; *, $p<0.05$ relative to mock-treated (n=6). Error bars depict means ± SD.

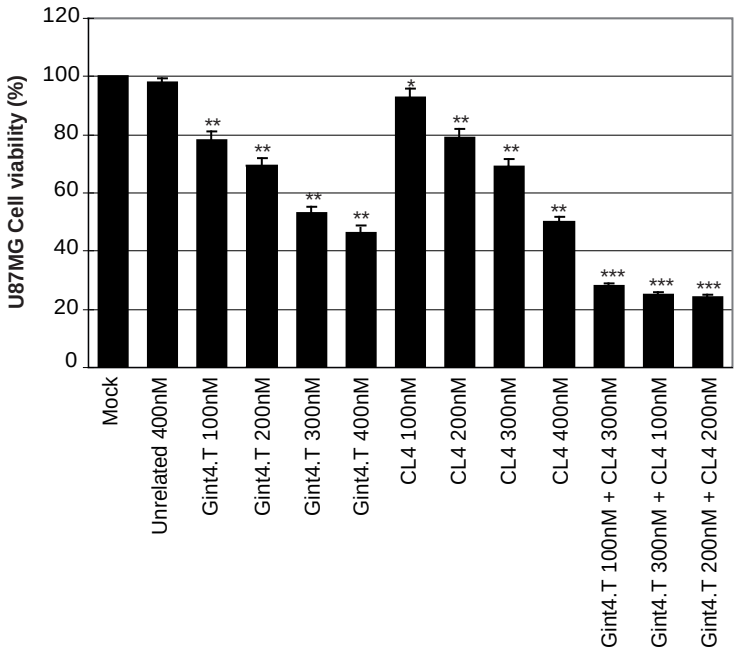

Figure S5. Combined effect of Gint4.T and CL4 on cell viability. U87MG cells were either mock-treated or treated for 72 hours with Gint4.T, CL4, Gint4.T plus CL4 or the unrelated aptamer, as indicated. ***, $p<0.0001$; **, $p<0.005$; *, $p<0.05$ relative to mock-treated (n=6). Error bars depict means ± SD.