

Figure S1 Selective inhibition of Myc/Max-DNA complex. The inhibitory effect and selectivity of ICX-101 at the protein level were confirmed by SPR analysis (A) and electrophoretic mobility shift assay (EMSA)(B). ICX-101 selectively inhibited the binding of Myc/Max dimer to DNA and had little effect on Max/Max dimer or AP-1 having a similar structure.

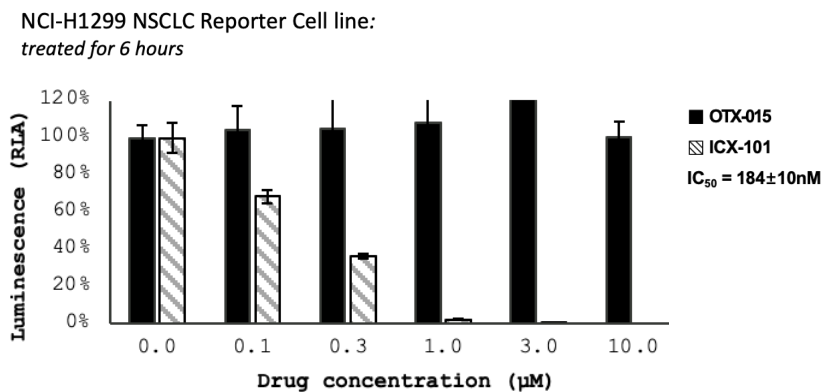


Figure S2 Myc signaling inhibitory effect. ICX-101 strongly inhibited Myc signal in NCI-H1299 reporter cells and had little effect on CMV negative control signal. When the compounds were treated with sequential concentrations for 6 hours, ICX-101 completely blocked the signal of Myc, whereas OTX-015, an indirect inhibitor of Myc through BRD4, had no effect. This is due to the direct inhibitory effect of ICX-101 and shows that it is superior to the indirect inhibitors.

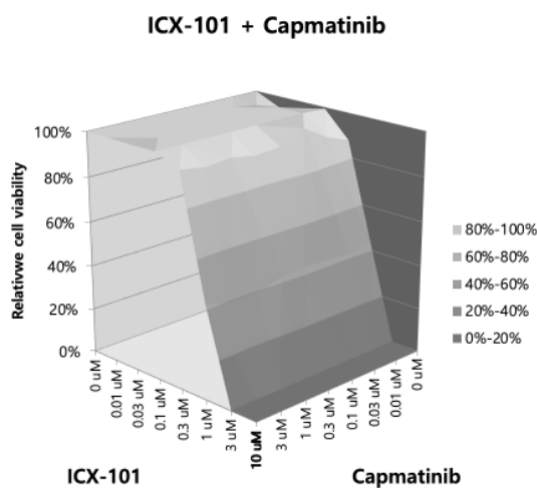


Figure S3 Combination treatment of ICX-101 and capmatinib in patient-derived cells. Patient-derived cancer cells were plated in 96-well plates at passage 3 and treated with each concentration of ICX-101 and capmatinib simultaneously after 24 hours of incubation. Cell viability after 72 hours was measured by ATP measurement method. Capmatinib did not show any antitumor effect up to 10 μ M, and co-treatment with ICX-101 did not show a synergistic effect. Co-treatment with capmatinib did not affect the effect of ICX-101.

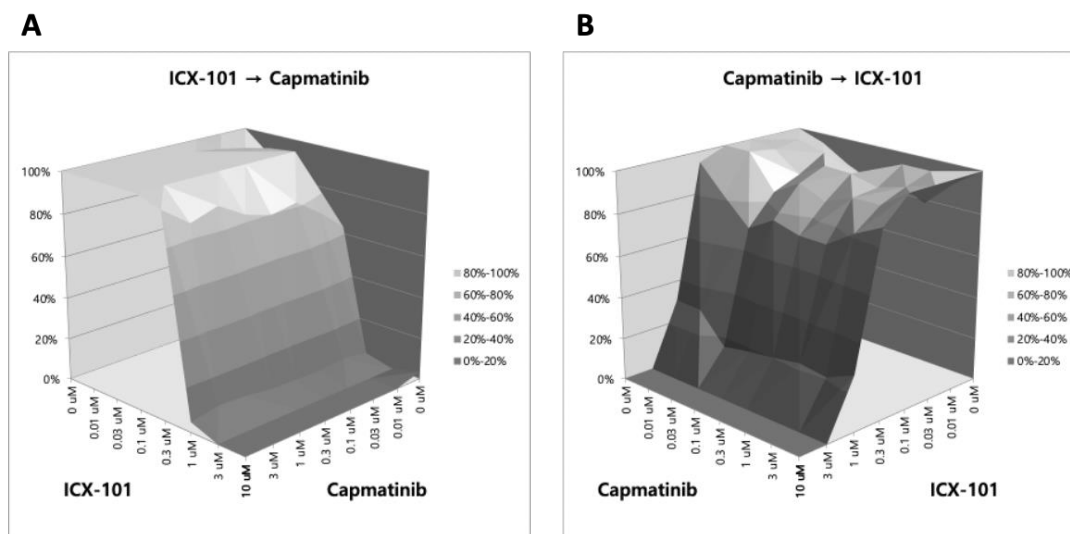


Figure S4 Sequential treatment of ICX-101 and capmatinib in patient-derived cells. Patient-derived cancer cells were seeded in 96-well plates at passage 3, and after 24 hours of incubation, each concentration of ICX-101 or capmatinib was treated. After 48 hours, the other drug was treated in a dose-matrix manner. After further incubation for 72 hours, cell viability was measured by ATP assay. (A) When ICX-101 was treated first, an additive effect, not a synergistic effect, was shown. (B) When capmatinib was treated first, a synergistic effect was shown.