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Supplementary Material: Multiscale Analysis and Validation of Effective Drug Combinations Targeting Driver Kras Mutations in Non-Small Cell Lung Cancer

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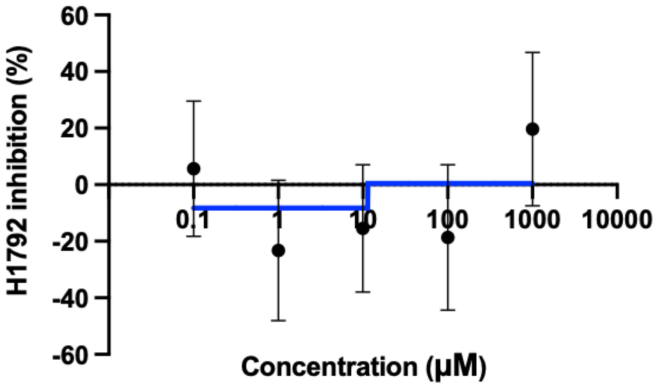

Figure S1. GI50 for BAY-293. The concentration (horizontal axis) of osimertinb is plotted against the H1792 cellular inhibition percentage (vertical axis). The GI50, or concentration required for 50% cellular inhibition, for BAY-293 was unable to be calculated with Graphpad prism 9.0, as there was not a strong enough effect. This indicates that BAY-293 is not effective at decreasing cellular proliferation in H1792 as a single agent.

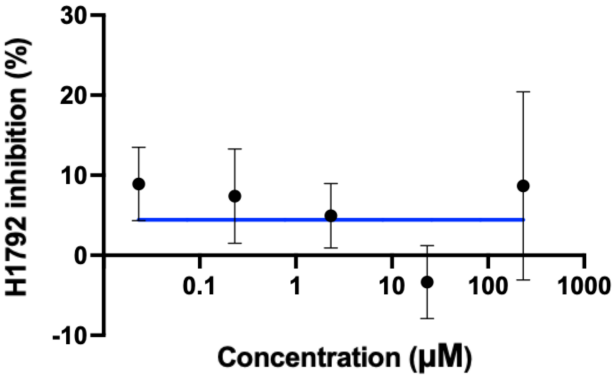

Figure S2. GI50 for ARS-1620. The concentration (horizontal axis) of osimertinb is plotted against the H1792 cellular inhibition percentage (vertical axis). The GI50, or concentration required for 50% cellular inhibition, for ARS-1620 was unable to be calculated with Graphpad prism 9.0, as there was not a strong enough effect. This indicates that ARS-1620 is not effective at decreasing cellular proliferation in H1792 as a single agent.

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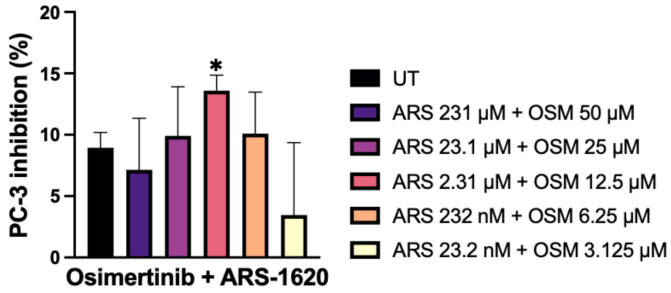

Figure S3. Cellular proliferation for PC-3 with ARS-1620 + osimertinib. The concentration (horizontal axis) of osimertinb and ARS-1620 is plotted against the PC-3 cellular inhibition percentage (vertical axis). The third strongest treatment condition was slightly significant ($p < 0.01$). This indicates that the osimertinib and ARS-1620 combination does not inhibit cellular proliferation in PC-3 relative to the untreated control.

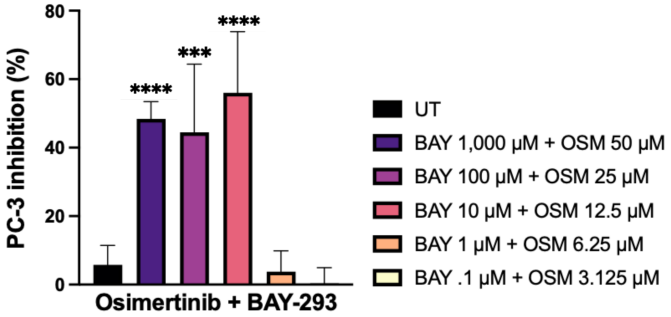

Figure S4. Cellular proliferation for PC-3 with BAY-293 + osimertinib. The concentration (horizontal axis) of osimertinb and BAY-293 is plotted against the PC-3 cellular inhibition percentage (vertical axis). The three strongest treatment conditions were all significantly higher ($p < 0.001$) compared to the untreated control. This indicates that while the osimertinib and BAY-293 combination shows some effect at inhibiting PC-3 cellular proliferation, it is far less than the effect observed with H1792.