Figure Legends

Figure 1. IC_{50} via resazurin assay (A) and Western blot analysis of PI3K and Ras-MEK-ERK pathway activation in UM-SCC-1 (B), 2 (C), 14A (D), 69 (E), 92 (F), and 108 (G) following treatment with HS-173 or BKM120.

Figure 2. Cell viability via resazurin assay and Ras-MEK-ERK pathway activation via Western blot analysis (insets) for UM-SCC-1 (A), 2 (B), 14A (C), 69 (D), 92 (E), and 108 (F) after treatment with trametinib and/or HS-173.

Figure 3. Live and total UM-SCC-69 (A) and UM-SCC-108 (B) cells after 72 hour treatment with increasing concentrations of trametinib and/or HS-173, as measured using a trypan blue exclusion assay.

Figure 4. Cell viability for UM-SCC-14A (A), -69 (B), -92 (C), and -108 (D) after 72 hour treatment with increasing concentrations of gefitinib and/or HS-173, as measured using a resazurin assay.

Figure 5. Western blot analysis of downstream PI3K and RAS-MEK-ERK pathway activation in UM-SCC-14A (A), -69 (B), -92 (C), and -108 (D) following 6 hour treatment with 1 μM gefitinib and/or 1 μM HS-173.

Figure S1. Cell viability for UM-SCC-1, 2, 14A, 69, 92, 108 and fibroblasts after 72 hour treatment with increasing concentrations of PI3K inhibitors BKM120 (A) and HS-173 (B), as measured using a resazurin assay.