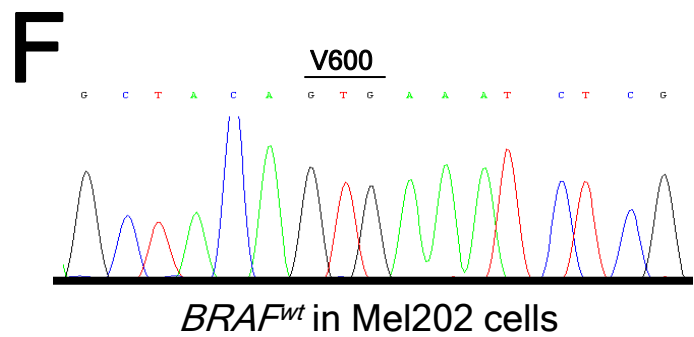
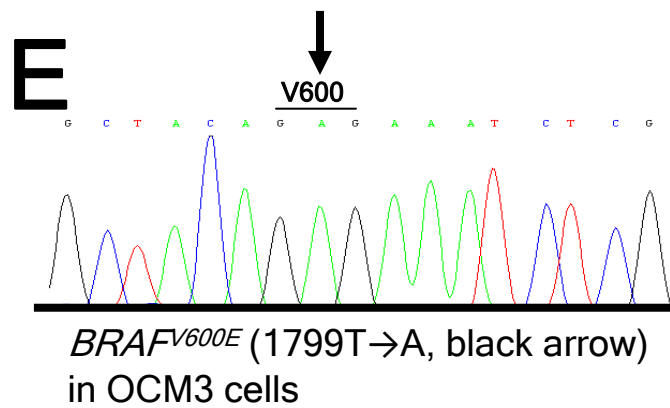
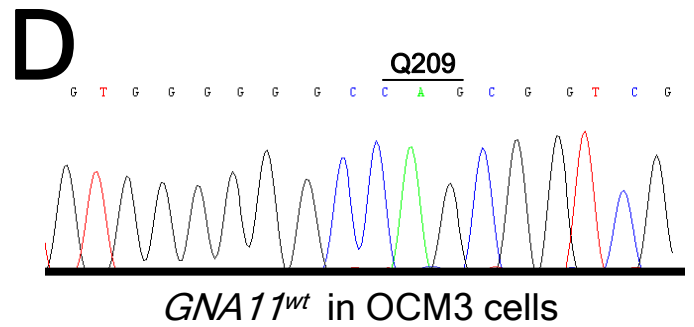
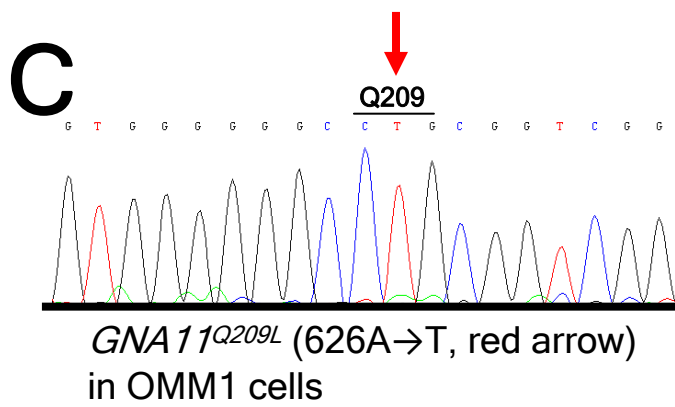
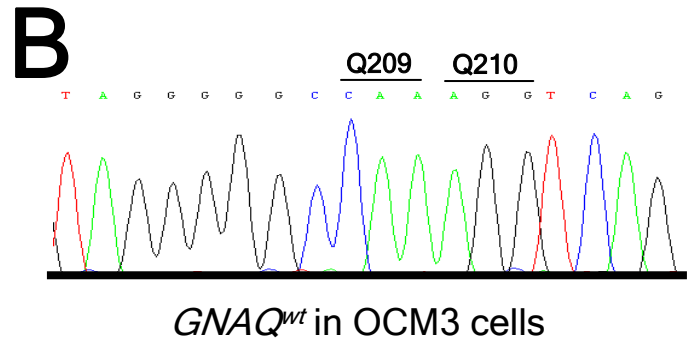
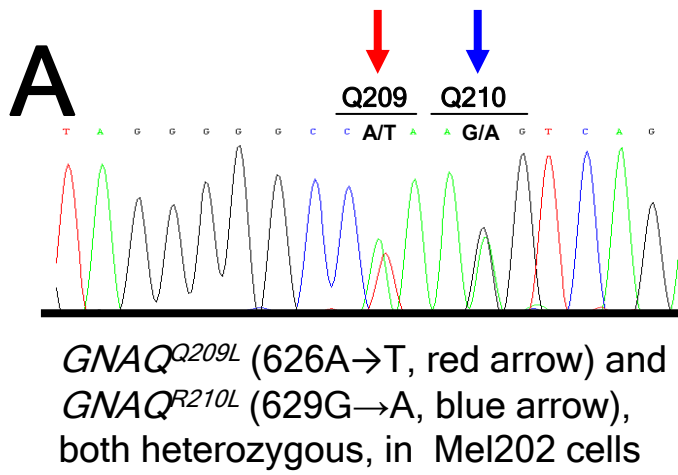
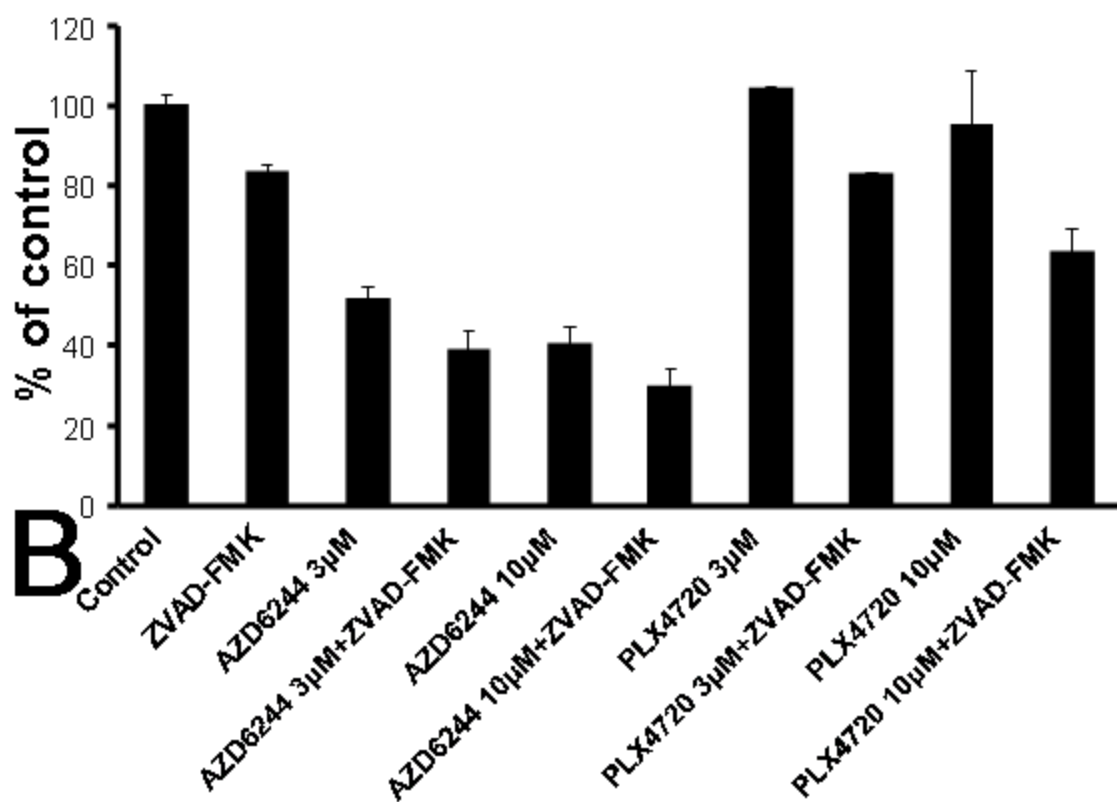
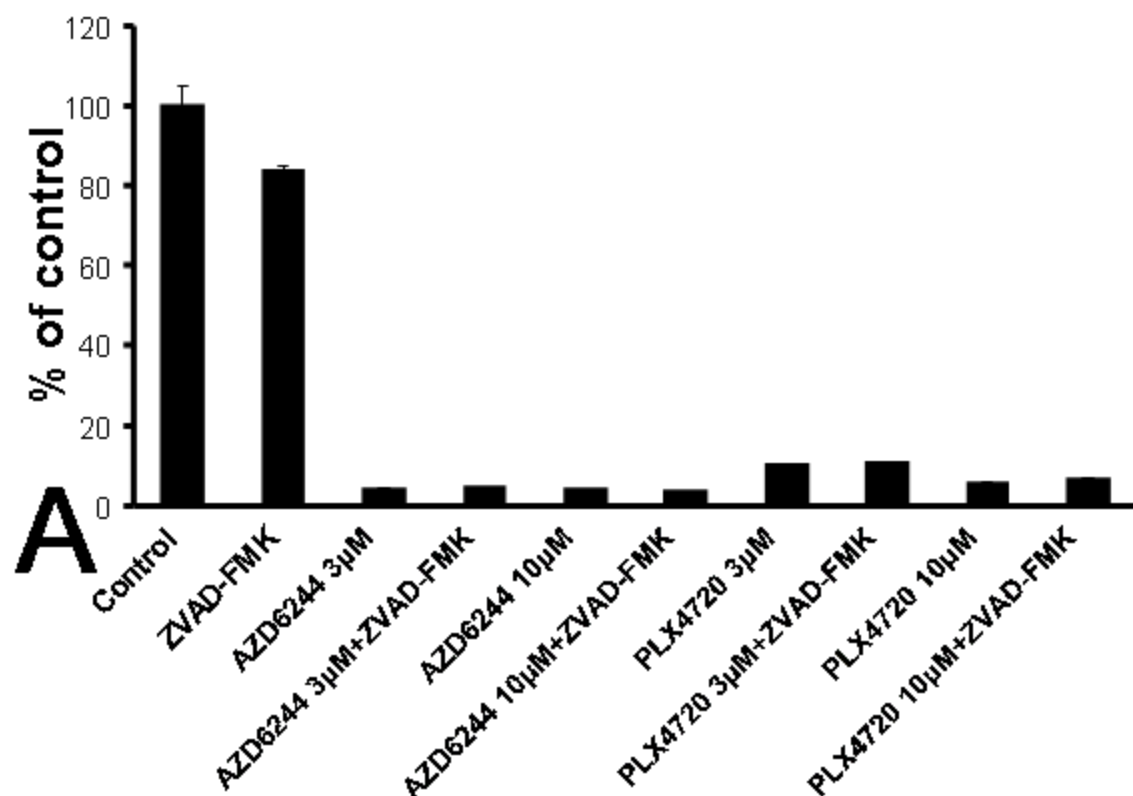


Mitsiades et al. Suppl. Fig. 1





SUPPLEMENTAL FIGURE LEGENDS

Suppl. Fig. 1. **Genotyping of UM cell lines for *GNAQ*, *GNA11* and *BRAF* mutations.**

Sequence electropherograms of PCR products from UM cell lines, depicting the presence of *GNAQ*, *GNA11* and *BRAF* mutations (indicated by arrows). Specifically:

A. In Mel202 cells, a heterozygous c.626A→T (CAA→CTA) transversion was detected in exon 5 of *GNAQ*, leading to a p.Q209L amino acid substitution. We also detected a heterozygous c.629G→A (AGG→AAG) transition, leading to a p.R210L amino acid substitution. Wild-type sequence from OCM3 cells is shown for comparison (B).

C. In OMM1 cells, a c.626A→T (CAG→CTG) transversion was detected in exon 5 of *GNA11*, leading to a p.Q209L amino acid substitution. Wild-type sequence from OCM3 cells is shown for comparison (D).

E. In OCM3 cells, a c.1799T→A (GTG→GAG) transversion was detected in exon 15 of *BRAF*, leading to a p.V600E amino acid substitution. Wild-type sequence from Mel202 cells is shown for comparison (F).

Reference sequences: *GNAQ*, NM_002072.3 / NP_002063.2; *GNA11*, NM_002067.2 / NP_002058.2; *BRAF*, NM_004333.4 / NP_004324.2.

Suppl. Fig. 2. **The impact of AZD6244 and PLX4720 on UM cells is not rescued by the pan-caspase inhibitor ZVAD-FMK.**

The *BRAF*-mutant OCM3 (A) and the *GNAQ*-mutant OMM1.3 (B) cells were pretreated with the pan-caspase inhibitor ZVAD-FMK (20 μM) for 1 hr and then AZD6244 or PLX4720 were added as well at the indicated concentrations for 96 hrs. Cell number was

quantified with the MTT assay and expressed as percentage of control (DMSO) wells (average \pm SD). Overall, the anticancer activity of AZD6244 and PLX4720 was not attenuated by ZVAD-FMK, indicating that apoptosis is not substantially involved in their effects.