Questions week 12

Which residues in the molecule are critical to designing potent inhibitors?

Why measure p-ERK to determine AMG510 activity?

Why did they check a cysteine proteome profile?

What are the main differences between the experiments done *in vivo* using human and mouse cells?

Was RAS inhibitor used as a first line of treatment in lung cancer patients?

What is the effect of carboplatin?

Note: In Figure 5, there is a test of RAS inhibitor in combination with anti-PD1 we will describe the mode of action of anti-PD1 and why it is considered an immune modulatory agent in the next lecture.

Can the combination of RAS inhibitors and immunotherapies be tested in xenografted mouse models using human cells?

Groups for discussion

Figure 1
Figure 2
Figure 3
Figure 4a-b
Figure 4c-e
Figure 5a-c
Figure 5d-g