Questions paper 1_week8

In this study, 2658 genomes from different cancer types were analyzed. To identify cancer mutations, how many samples do they need to examine?

What type of event can lead to hotspot noncoding mutations? Which mechanisms are altered? (fig. 1a)

Why are they defined as candidate drivers? (fig. 1b)

Why did they observe great variability across cancer types? (fig. 1b)

To call non-coding elements drivers, what type of evidence do they include in their analyses? (fig. 2a)

Why consider expression data? (fig.2a)

What type of alterations affect the non-coding regions of p53?

Would the 10 out of 11 patients considered heterozygotes or homozygotes be altered for p53? (fig.2b)

Do all mutations in 3'UTR have the same effect on gene expression? (Fig. 2c-d)

What is a structural variant? (We will discuss this in more detail in week 9 and week 10)

Which parameter/feature is associated with an increase in mutation burden and what type of structural variation? (Fig 3b)

Why microdeletion in 5'UTR of Brd4 reduce the expression of this gene not of NOTCH3? (Fig. 3c-d)

Are oncogenic fusions common to many tumor types? (Figure 4f)

What is the most frequent effect of SRJ?

If we increase the number of WGS in cancer, will we find more driver mutations in noncoding regions?

Why is there variability in the ability to detect mutations in the TERT promoter across tumor types?