Contents for discussion:

* Advantages to the approach
* Innovation of results
* Intracellular signaling network captures relevant tumorigenesis pathways
* Interesting results from z-score ranking
  + Discuss reka paper
* Problems with interactions between soFunDEGs and TFs
  + Even though Transfac predicts an interacton is possible does not mean that there is experimentally validated evidence of it
  + Interactions could exist but there is no literature so nothing we can do
  + By using foc instead of soc we lose out on CL TNBC related genes but we do better than just using DEGs
* Incorporated a very interesting GOF p53 mutation
* Processes for further automation of network construction/consideration of mutational profiles would be helpful
* Incorporating phosphoproteomics
* FVS nodes are known regulators of tumorigenesis
  + Good
  + Downside: using curated databases that may be biased towards known tumorigenic interactions (not identifying novel targets)
* Attractor values clustered the same way as the experimental data
  + Don’t expect values to be exactly same as experiments because RNAseq is just a snapshot of the expression
  + Biological replicates cluster separately 🡪 more data or patient data would be helpful
* The subset of 100,000 perturbations can be expanded in the future
* Why didn’t discrete datasets result in classification overlap?
* Justification for floating point numbers
  + Comparing conditions by computing euclidean distance
* Discuss Perturbation orientation results
  + GSK3B, HDAC3, CTNNB1 don’t agree with literature
  + Follow from experimental conditions
    - Trend with GSK3B
    - Not sure what’s happening with CTNNB1
  + PIAS1 could be new (it’s activation hasn’t been studied)
  + AURKA could be new
  + JUN, RELA, STAT3, TCF3, FOXM1, AKT1 all agree with lit
  + MAPK1 doesn’t seem to matter
* Only studied FVS, but considering source nodes could improve results 🡪 even though adding more control targets, some of them might already be set for the right trajectory
* Synthetic lethality of results
  + None are dependent according to depmap
* Prioritization of reversion targets
  + Number of negative feedback loops
  + Druggability
  + Synthetic lethality
  + Frequency of perturbation