* Carcinosarc as a factor
  + Treating carcinosarc as 1 and all others as 0 as an additional factor
  + Indeed carcinosarc is associated with worse prognosis with log.estimate of 0.338, though not significant due to high sd (low #’s)
  + The significant effect of TIL cluster (for 2 groups) remains!! I tested this on overall survival
* Remove B cells, CD8+Foxp3+ T cells from analysis
  + Experimented with 3 clusters and overall survival – gives a TIL tumor + stroma highest, a TIL tumor + stroma intermediate, and a TIL deplete
  + Basically, insignificant differences between all, with the first cluster having the best outcomes
  + Similarly with 4 clusters, the highest TIL cluster does even better than the intermediate ones, however the highest TIL cluster is very small
    - Results are barely not significant because each group is smaller
* Do analysis where each TIL variable is considered independently(?) in a Coxph model, see which ones are best correlated
  + Could do this perhaps by omitting the clust part of the coxph model on ipython notebook
* CD103/ITGAE’s ligand is E-cadherin – maybe that’s why carcinosarcs have fewer TIL?

Molecular subtypes (p53abn, etc.) => vs angiogenesis markers

When we get the HGSC data overlay and stuff => look at the S-TIL group (which should correspond to C1) => does that correspond to increased angiogenesis

HER2 scores vs VEGFR

Look at clustering HGSC CN signature exposures versus the endometrial ca ones using the Brenton samples + Vancouver samples

* Then do the same with TIL
* This is for Blake’s question

Talk about Wee1 inhibitors in CCNE1 mutated tumours cause we may have significant associations with the CCNE1 data from Dawn’s group

Andrew’s manuscript

* **Given that vascular/endothelial/CAFs correlate with prevalent WGD and immune signalling with rare WGD in HGSC, do we see an anticorrelation between immune and vascular/endothelial/CAF in endometrial serous too?**