**PhyloWGS (Deshwar et al)**

1. The treatment of the TCGA data during testing is quite unclear. The authors do not provide any rationale for removing the SSMs in sites identified for structural variation by BIC-seq. It is also surprising that structural variants (which in this case, I am presuming they mean to say large INDELs) encompass about 4300 SSMs (from a total set of 62). This appears to indicate misalignment in these regions rather than actual novel mutations. What could be a rationale for removing SSMs in sites rich for structural variation events, from the clonality modeling? Won’t the allelic fraction still be informative?
2. The authors used BIC-seq to get the CNV calls. This is a read-depth based method, so an appropriate choice when you are using the CNV inferences to ‘reasses’ VAFs of the somatic mutations and thereby model subclonality. As a negative control, it would have been nice to see them use methods that use the underlying BAF distribtions and het SNPs for CNV prediction – I wonder how using such methods for CNV calling will impact the clonality prediction? At least a comment to this end would have been helpful. I would imagine that the clonality prediction will probably be more effective with tools that use CNVs (ex. THetA), rather than SNVs (ex. pyClone), in that case.

Any comments on controls for parsimony? Loose parsimony seems to be an automatic offshoot of this method.