

**Questions and discussion items Freedman et al. (2011) 43: 513-518**

1. What is the greatest challenge after a genome wide association study?
2. What is the underlying hypothesis with respect to how SNPs exert their effect i.e. confer risk to complex diseases?
3. What are 2 ways to assess the functionality / effect of a SNP?
4. What is different between these two methods.
5. What is a Tag SNP?
6. How could the SNP affect the epigenetically regulated gene expression?
7. What would be criteria for a strong candidate gene?
8. If a strong candidate gene is selected, likely to be affected by the genome wide association signal what would be next steps to proof causality to the disease.