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. Wat is essentially different in this hypothesis compared to studies of early onset osteoarthritis families?
4. What are 2 ways to assess the functionality / effect of a SNP?
5. What is different between these two methods.
6. What is a Tag SNP?
7. How could the SNP affect the epigenetically regulated gene expression?
8. What would be critieria for a strong candidate gene?
9. If you check the functional genomic lecture of Wednesday, do you consider DIO2 a strong candidate gene for Osteoarthritis.
10. 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.