**Generations and Mendel: Study guide and readings**

# Required readings:

# Komatsuda et al. 2007. PNAS 104:1424-1429

# You are responsible for reading the abstract, introduction, and conclusions.

# If you have the opportunity, enjoy reading the full article.

**Study questions:**

1. Why is making a cross, and analyzing the patterns of inheritance in the progeny of the cross, such an important dimension of plant genetics? Why not just study the parents and leave it at that?
2. Who was Gregor Mendel and why did he (or did he not) receive a Nobel prize for his fundamental contributions to genetics?
3. By convention, which parent is shown first in a cross – the male or the female?
4. Considering a single locus in your favorite plant, define homozygosity and heterozygosity.
5. Explain why the percentage of heterozygosity declines by half with each cycle of selfing, assuming the cross was made between two inbred parents. What if the parents were not inbred?
6. What decrease in the percentage heterozygosity do you expect with each generation if you self a completely inbred plant?
7. If you make a cross between two inbred parents and self-pollinated the progeny for 10 generations, would you expect there to be any residual heterozygosity?
8. F1 hybrid seed is the basis of much of the seed industry. Why not F2 seed?
9. What is a backcross, and why might a geneticist or plant breeder choose to make a backcross rather than simply advance the generations through cycles of selfing?
10. What is so special about a testcross?
11. If your goal is to make a population of recombinant inbred lines (RILs) why do you need to self and advance progeny of each F2 plant instead of just advancing the progeny of one F2 plant?
12. How does the doubled haploid generation advance system lead to immediate and complete homozygosity?
13. Compare and contrast making a cross when a plant is a hermaphrodite, monoecious, or dioecious.
14. How does mating biology (e.g. self- or cross- pollinated) affect the likelihood of a prospective parent plant being homozygous or heterozygous?
15. Why are expected ratios different in the F1-derived F2 generation and F1-derived doubled haploid generation?
16. Considering two alleles at a locus, what is dominance?
17. How can more than one gene determine a trait?
18. What is gene flow, in the context of transgenic plants?
19. In the event of gene flow between a transgenic Roundup Ready crop and an organic crop, why would the herbicide resistance trait show Mendelian inheritance?
20. Be able to complete a monohybrid Punnett square for the consequences of selfing a heterozygote.
21. Explain how genetics and/or environment can cause a trait to show qualitative or quantitative inheritance.
22. What is the threshold – in terms of number of genes – for deciding if a trait shows qualitative or quantitative inheritance?
23. What is a QTL and what are two ways to locate QTLs in the genome?
24. Compare and contrast inheritance patterns in the nuclear genome as opposed to the cytoplasmic genomes.
25. Briefly explain the hypothesized origins of the cytoplasm organelles.
26. Why is the following categorical statement not true? “In plants, chloroplasts and mitochondria show maternal inheritance and the only traits these organelles determine are photosynthesis and respiration”.
27. Describe the phenotypes you expect in barley when a plant is *VrsVrs1* and when a plant is *vrs1vrs1*.
28. Which phenotype is due to loss of function: six-row or two-row?
29. What is a homeobox gene?
30. Briefly explain the hypothesized model for the function of alleles at the *vrs1* locus.
31. What types of mutations occurred to give the loss of function at the *vrs1* locus?
32. Explain how, in a diploid plant, there can only be two alleles at a locus but many alternative alleles are possible in a population of the same diploid plants.
33. Why is mutation the principal source of new alleles?
34. How can some mutations not have an obvious effect on phenotype?
35. How can different mutations in the same gene led to the same or different phenotypes?
36. Briefly explain how doubled haploids are produced in barley, using anther culture.
37. How does goodness of fit relate to testing hypotheses regarding the inheritance of a trait?
38. Why is it important to know what generation is being studied (e.g. F2 or F3) when conducting a chi square test?
39. If Mendelian analysis is based on using progeny to understand parents, why are parents usually included in genotyping/phenotyping analyses?
40. Show how you could get a 1:1 phenotypic ratio in F1 progeny and in doubled haploid progeny from an F1, depending on the allelic state of the parents used to make the cross.
41. If you study inheritance using only doubled haploids, why can’t you determine if an allele is dominant or recessive?
42. In a chi square test, it is imperative that you use the correct degrees of freedom. Say you calculate a chi square value of 9. What conclusions will you make if you test goodness of fit at 2 df vs. 6 df?
43. Considering those other genomes: the chloroplasts and the mitochondria - why don’t we use chi square tests to test the expected vs. observed ratios for traits encoded in these genomes?
44. If chloroplasts and mitochondria in monocots usually show maternal inheritance, why are doubled haploids - produced through anther culture - green and able to grow?
45. Be able to calculate chi square tests for mono-hybrid and di-hybrid ratios based on the F2 and doubled haploid generations derived from the cross of two inbred parents.

**The readings**

*Komatsuda et al.*

1. Does the six-row trait guarantee higher yield?
2. Approximately how many years ago was barley domesticated, and where was it domesticated?
3. Was there a one-time, one-place mutation conferring the six-row phenotype?
4. What does the wild type (dominant, Vrs1) allele encode?
5. What do recessive (vrs1) alleles encode?
6. Why are there (apparently) more possible recessive alleles than dominant alleles