

Papers for Thursday January 2011

Bowman, J., Smyth, D. R. and Meyerowitz, E. 1991 (*Development*) Genetic interactions among floral homeotic genes of Arabidopsis

Pelaz S. et.al. 2000 (*Nature*) B and C floral organ identity functions require *SEPALLATA* MADS-box genes

Questions for Thought

As you read the first paper, do not be intimidated by its length, information amount or obscure anatomical details. Focus on the methods used (allelic series, mutant combinations) and the logic applied to arrive at a predictive model for further experiments. Table 6 is a useful summary of all their data and will be the basis of our discussion.

The second paper is a more recent study, using reverse genetics to further explore the molecular underpinnings of floral pattern formation.

As before, **write down questions** you have about the logic or rationale for each experiment, the method employed, and the conclusions drawn. Come up with at least three questions. Turn in your questions as homework at the beginning of class. During class we will discuss your questions along with the QfT below. We will emphasize the questions in bold; the other questions are meant to help you think about each issue.

1) Before reading these papers: unlike animal cells, plant cells cannot move and their identity is largely determined by position. **What biological mechanisms may allow adjacent cells to acquire different cell fates?** Think mechanistically.

Bowman 1991:

2) **What is the major scientific question the authors aim to address?** What was known before this study was published?

3) The authors extend previous work by adding additional alleles. **Why is this a worthwhile approach?** What can you learn from testing different alleles? Think mechanistically and remember the concept of modularity.

4) **What do the single mutant phenotypes imply about the functions of the four genes in flower patterning?** What organs require Agamous function? *Apetala2*? *Pistillata* and *Apetala3*?

5.) What are transheterozygotes? What questions can you address with transheterozygotes? In case you have two lethal alleles resulting in a viable transheterozygote – what is a likely explanation?

6. The authors are using different temperatures for some of their phenotypic analyses. Why? Phenotypes and their severity are changing. What could this imply? Think mechanistically.

5) Remember what you learned about epistasis analysis. **How do they choose the alleles for their double and triple mutant analysis?** What are potential pitfalls? Which mutant combinations hold the most information?

6. What results allows them to conclude that AP2 and AG interact with each other? What kind of interaction seems most likely from the results?

7. Turn to Table 6 and Fig. 9 and think about their how their results allow them to arrive at the ABC model. **What results can the model explain and which aspects are not represented?** What predictions can the model make for expression patterns and protein interactions?

Pelaz 2000:

1) Nine years have passed since the ABC model was first proposed. **Given this new paper's introduction, how has the original model been revised or updated?**

2) The authors conduct a reverse genetic screen. **How does this approach differ from a forward genetic screen?** What do you need to conduct a reverse genetics screen? What are the advantages? What are the disadvantages?

3) The authors create triple mutants without explicitly describing how they proceed. **How do you think this might be accomplished?** What would complicate this approach?

4) **What does the triple mutant phenotype imply?** How does it compare to single and double mutant phenotypes of the previously identified ABC genes? Do these new genes act up-or downstream of the B and C genes?

5.) How come that the B genes *AP3* and *PI* were not redundant in function but the D genes *Sep1-3* are? **What could this observation imply about their molecular mode of action?**

6.) Given the multiple hypothesized AP2 functions and the expression of carpeloid leaves versus true leaves in strong versus weak AP2 mutants, do you think that the expression of ABC(D)E genes would be enough to produce flowers?

7. How can the ABCD model explain different flowers in different evolutionary lineages? Think of how the Arabidopsis flowers (crucifer) differs morphologically from many other flowers.