GENOME553 Winter 2010

Paper for Thursday 28 January 2010
Shippy, T. D., M. Ronshaugen, J. Cande, J.P. He, R. W. Beeman, M. Levine, S. J. Brown, and R. E. Dennell. 2008. Analysis of the *Tribolium* homeotic complex: insights into mechanisms constraining insect Hox clusters. *Dev. Genes. Evol*. **218**: 127–139.

Questions for Thought (QfT)

As you read this paper, refer to FlyBase or BeetleBase for any questions you might have about genes, deficiencies, or nomenclature. **Write down questions** you have about the biology of the system, body patterning, and the results of their screen. **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.

1) What are some of the unusual features of the HOX gene clusters? What might be the advantages to an organism to maintain this HOX-gene organization? Disadvantages? *What would happen if a chromosome break separated some genes of the cluster from the others? Justify your prediction with molecular arguments.* Many investigators have studied HOX-gene function in a variety of vertebrate and invertebrate animals. **Why analyze yet another cluster, this time in beetles?**

2) The authors employed several genetic and molecular methods for identifying genes in the *Tribolium* HOX cluster. Outline their overall strategy and explain the purpose of each approach. **Did they identify all the genes in the region? What genes might they have missed?**

3) Shared regulatory elements could select for maintenance of a clustered set of HOX genes. To test this hypothesis, the authors analyze an allele of the *mxp* gene (*Tribolium castaneum mxp* is thought to be the ortholog of Drosophila *proboscipedia* (pb) and is therefore written: *mxp/Tc-pb*). **What evidence supports the proposed HOX-cluster organization depicted for *mxp*$^{Dch-3}$ in Figure 3a**?

4) The larval and adult phenotypes of *ptl*^{D60} exhibit different degrees of homeotic transformation, with the adult defects being more severe and resembling loss of both *ptl/Tc-Antp* and *Cx/Tc-Scr*. **Why do the authors use RNAi to characterize the *ptl* defects?** Why not simply sequence the mutation? What are the pros and cons of each approach? NOTE: "larval" RNAi disrupts pupal and adult transcripts; "parental" RNAi disrupts embryonic and larval transcripts.

5) A GFP reporter construct present in the KT076 enhancer-trap line is expressed in the same pattern as *Cx/Tc-Scr* yet the authors conclude that the insertion is an allele of *ptl*. **Why?**

6) The authors propose two hypotheses to explain the clustered organization of the HOX genes: shared regulatory elements and phylogenetic inertia. What evidence supports each hypothesis in Tribolium? In Drosophila?

7) Propose two <u>distinct</u> functions for the non-coding RNAs in the HOX clusters. Suggest an experiment that would distinguish between these possible functions.