RESEARCH NOTE

UNUSUAL PHENOTYPE SUGGESTS ROLE FOR HOMEOTIC GENES IN ARACHNID DEVELOPMENT

Studies of segmental mutations in *Drosophila melanogaster* have led to the discovery of several classes of regulatory genes important in determining body pattern (Carroll 1995). These regulatory genes are also known as transcriptional factors because their proteins bind to another gene’s control regions, or promoters, allowing for controlled expression or repression. For example, attachment of maternal effect gene transcripts to specific areas of an ovum in *Drosophila*, initiates the anterior-posterior (AP) body axis (Melton 1991). Diffusion of maternal effect proteins from opposing ovum poles creates a dual concentration gradient that differentially activates or represses additional classes of transcriptional factors. This cascade of regulatory gene expression determines positional information, indicating body polarity and regional specificity.

Regional specificity within body plans is largely determined by a class of transcriptional factors known as homeotic genes. Specific concentrations of maternal effect proteins turn on different homeotic genes controlling the identity of segments along the AP axis of an arthropod’s body. Remarkably, homeotic genes are arranged on chromosomes in linear clusters corresponding to their exact sequence of expression. Those located toward the left end of the complex are expressed in posterior parts of the body, while those to the right are expressed toward the anterior parts (Kenyon 1994).

Bithorax (BX-C) and Antenapedia (ANT-C) are the two known complexes of homeotic genes. ANT-C in *Drosophila* controls the identity of appendages (Carroll 1995). A mutant ANT-C gene causes flies to grow a leg in their antennae socket. BX-C in *Drosophila* controls the morphology of the posterior thorax and abdomen. A mutant BX-C gene results in two rear-thoraces instead of one front and one rear thorax, causing flies to have two pairs of wings instead of one pair of wings and one pair of halteres. In organisms other than *Drosophila*, genetic mechanisms underlying body plan development are less well understood and have only recently been investigated in arachnids (Damen et al. 1998; Telford & Thomas 1998).

Recently, I collected an immature *Misumenops* sp. (Thomisidae) on the Hawaiian Island of Maui which showed a dramatic segmental mutation. It was collected from wet forest in the Nature Conservancy’s Waikamoi preserve on east Maui. This individual closely resembled *Misumenops anguliventris* Simon 1900, one of 17 described species of *Misumenops* endemic to the Hawaiian Islands. Thomisids are one of the few spider families containing genera that are known to be exceptionally diverse in the Hawaiian archipelago (Gillespie 1994).

After closer examination of this individual under a light microscope, I noticed a second set of eight eyes on its abdomen. These “abdominal eyes” displayed the exact pattern of the eight “normal” eyes on the cephalothorax. In addition, the dorsal aspect of the abdomen displayed the same type and pattern of setae also found on the carapace of the cephalothorax. Despite these dramatic morphological aberrations, the posterior aspect of the abdomen resembled a “normal” abdomen. At the time of collection, the spider was a third or fourth instar juvenile. It lived for two months and appeared typical in behavior. After death, the spider was preserved in 70% alcohol and was then prepared and examined using a Hitachi S-800 scanning electron microscope (see Figs. 1, 2). The specimen has been deposited in the Bishop Museum entomological collections.

Kaston (1982) summarized accounts of oc-
ular anomalies in spiders. Of the nine cases he described, only two involved spiders gaining eyes. These specimens, having 14 and 16 eyes, were explained as a result of embryonic duplication of a head region. It is premature to suggest that these phenotypes were a result of a mutation in a major regulatory gene because there are no accompanying figures showing where these eyes were located. If additional eyes were located on a segment other than the cephalic, this confusion in segment identity would strongly suggest abnormal homeotic gene expression.

The “abdominal” eyes and duplicated segmentation pattern shown in the spider I collected may be explained by several different hypotheses. First, a mutation in a homeotic gene of the bithorax complex may account for the abnormal phenotype. However, homeotic mutations are expressed in individual segments and because spider abdomens are composed of several segments, generation of this phenotype would require independent mutations in all segments. It is more likely that this mutant was produced because the wrong set of homeotic genes was turned on, while the correct set was not. This could be brought about by several mechanisms. A mutation in a regulatory gene determining body polarity might turn on an entire group of regionally inappropriate homeotic genes. Alternatively, duplication of anterior structures might arise if maternal nurse cells placed transcripts activating anterior development at the anterior end of the ovum as well as the where the abdomen would normally arise. The mutant phenotype could also be created through a chromosomal aberration produced during gametogenesis. If the linear cluster of homeotic genes is duplicated at the region corresponding to the cephalothorax, this might result in the development of two cephalothoraxes.

Evolution of homeotic genes may explain the immense diversity of body forms seen among arthropods (Kenyon 1994; Carroll 1995). Small mutations in these highly conserved genes result in macro-mutations, providing an evolutionary mechanism for generating novel phenotypes. Homeotic genes have also been identified in cnidarians, nematodes and annelids. Most recently, homeotic genes have been identified in a spider (Damen et al. 1998) and mite (Telford & Thomas 1998). Comparative investigation of homeotic gene expression will undoubtedly play an important role in understanding the evolution of arachnid morphology.

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LITERATURE CITED

Jessica E. Garb: Department of Zoology and Center for Conservation Research and Training, University of Hawaii, Edmundson Hall, Honolulu, Hawaii 96822 USA

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