

The Inheritance of Red Eye Color in *Drosophila*

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Once upon a time, *Drosophila melanogaster* was not bred in laboratories. The poor little fly lived as a vagrant, depending on the careless handouts of Mother Nature and Man, in orchards, breweries, vinegar works, tomato gardens, silos, and garbage pails. But the great housing project began soon after 1900 by a man appropriately named Castle. He carefully coddled the flies in bottles and began studying the effects of inbreeding, crossbreeding, and selection. They were pedigreed like pampered pooches. Still, he did not succeed very well in getting bigger and better flies; in fact they were rather monotonously uniform.

When Prof. Castle gave up *Drosophila* in favor of bigger game (e.g., rabbits), a new savior took up the study. By good luck this man, T. H. Morgan, immediately discovered a very obvious mutation, white eyes. This was a welcome change from the regularity of brick-red eyes seen hitherto. It was propagated and quickly found to be recessive to red, and sex-linked. With all the ballyhoo about it, advanced students joined Morgan in the fly lab, and in the reek of fermenting banana many yeasty ideas developed.

Since the white eye color was recessive to red, as night follows day red must be dominant to white. What could be more simple? But then another eye-color mutation was discovered. This one was a dull wine-color and was christened "purple." Feverish analysis soon revealed that this mutation was also recessive to red, but it was not sex-linked. A slight dilemma now presented itself: red was

dominant to white, but it must also be dominant to purple. Could two things recessive to the same thing be recessive to each other?

This dilemma had to be investigated further, and therefore purple-eyed flies of pure stock were crossed to white-eyed flies also of pure stock. Would the hybrids have purple eyes or white? *Mirabile dictu*, they turned out all to have wild-type brick-red eyes! This was disconcerting: where did the red come from? We had thought we got rid of it in the mutant stocks. A throwback!

The F₂ generation results shed light on the mystery, since the ratio obtained was 9/16 red, 3/16 purple, and 1/4 white. This happened to be a well known ratio in genetics already and had been explained by using two sets of genes. Thus red would be *AABB* or *AABb* or *AaBB* or *AaBb*; purple would be *aaBB* or *aaBb*; and white would be *AAbb* or *Aabb* or *aabb*. This was the modern way of Biology—you couldn't talk about eye color being inherited because only genes are inherited. Weismann's doctrine of the germplasm rather than the soma being transmitted has jurisdiction.

Well, then, which is the red-eye gene? Is it *A*? No. Is it *B*? No. And it isn't *aa* or *bb*. There is only one conclusion—there is no such thing as the red-eye gene. Red eyes are soma, and genes are germplasm, and they cannot be equated. Of course a lot of text books failed to heed this matter, but the *Drosophila* students have had it drummed into them pretty regularly. As the years went by, literally scores of eye-color mutations of all sorts were picked up: ruby, apricot, garnet, vermilion, sepia, plum, prune, brown, etc., etc. Combinations of these yield practically any tint from black to white. To get normal red, no mutant must show itself.

How many genes then are necessary to make red? Nobody knows. Chemical study of the eye pigments reveals pteridine compounds, and it is hypothesized that each mutant involves a block or deviation at a particular step in a long biosynthetic series. "White" is a failure at the very beginning; red is success at all steps.