Class discussions may include the following. 1) About 8% of males and 0.5% of females of European origin have red-green color vision defects (Drummond-Borg et al., 1988). Why is there such a higher frequency of color blindness in males? The reason is that this is a sex-linked trait and hemizygous (XY) males are expected to have a higher frequency than females who must be homozygous (XX) for the defective region. 2) If the red-green sequences are in Hardy/Weinberg equilibrium and the frequency of the defect is 8% in males, what is the expected frequency in females? The answer is 0.08 × 0.08 = 0.6%, close to the reported 0.5%. 3) The assumption that humans are in Hardy/Weinberg equilibrium for the red-green sequences also assumes that females and males that have red-green color blindness are as fit as humans without color blindness, *i.e*., humans with and without the red-green defect have about the same number of offspring. One might ask students if they think this would be true in prehistoric and modern times. What is known is that a higher frequency of red-green color blindness is found in more advanced societies than in some primitive societies (Malhotra, 1978; Narahari, 1993). This may suggest relaxed natural selection for red-green color blindness in modern societies.

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White eye phenotypes and their genetic analysis.

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An interesting case for undergraduate students of general Genetics is to consider that different genes can produce the same or similar phenotypes. We present here an experiment to discover that the same phenotype could be produced by different genes, and then, to carry out the genetic analysis of these genes. For this laboratory study we have used the following *Drosophila melanogaster* strains: *white* (white eyes) and *scarlet – brown* (white eyes).

Initially, students have a couple of strains (named mutant 1 and mutant 2) showing the same phenotype (white eyes) and the first question is, are they mutations from the same gene or from different genes? The classical approach is to carry out reciprocal crosses between them. The crosses and results that would be obtained are:

(P) ♂ mutant 1 × ♀ mutant 2

↓

(F_1) All individuals present normal eyes

(P) ♂ mutant 2 × ♀ mutant 1
↓
(F_1) ♂ white eyes + ♀ normal eyes

Thus, it is possible to deduce that mutant 1 and mutant 2 do not complement, and thus mutant 1 and mutant 2 affect different genes.

For the particular analysis of each mutant strain, reciprocal crosses have to be carried out between individuals from the mutant and normal strains. Thus, for the case of **mutant 1 strain**:

A) (P) ♂ mutant 1 × ♀ normal and **B)** (P) ♂ normal × ♀ mutant 1

In the first cross, all F_1 individuals presented normal eyes. However, in the reciprocal cross all males have white eyes whereas all females are normal. Flies from both F_1 (derived from **A** and **B** parental crosses) have to be independently intercrossed to obtain the respective F_2. In this generation and for the parental **A** cross, the proportions of individuals are approximately: 1/2♀ normal + 1/4 ♂ normal + 1/4 ♂ white eyes. The F_2 obtained from the parental cross B is composed of 1/4♀ normal + 1/4 ♀ white eyes + 1/4 ♂ normal + 1/4 ♂ white eyes. These results are according to a sex-linked inheritance pattern. Checking the *D. melanogaster* essential genetic maps (for instance Gardner *et al*., 1991; Russell, 1992; Griffiths *et al*., 1996; Klug and Cummings, 1997; Pierce, 2009), mutant 1 strain corresponds most likely to the *white* gene.

For analyzing **mutant 2**, the reciprocal crosses carried out are:

C) (P) ♂ mutant 2 × ♀ normal and **D)** (P) ♂ normal × ♀ mutant 2

In both cases (crosses **C** and **D**), the F_1 was constituted by normal individuals. Thus, reciprocal crosses **C** and **D** are equivalents. Males and females of F_1 have to be intercrossed to obtain the F_2. The phenotypes obtained and their proportions are approximately: 9/16 normal + 3/16 bright eyes + 3/16 brown eyes and 1/16 white eyes. Thus, a couple of genes are controlling this trait and new phenotypes (not presented in the parental individuals) arise in the F_2 generation. This is due to a gene interaction between two genes presenting independent transmission. Studying the *D. melanogaster* genetic maps (Gardner *et al*., 1991; Russell, 1992; Griffiths *et al*., 1996; Klug and Cummings, 1997; Pierce, 2009), it is possible to deduce that the genes producing this interaction are likely *scarlet* (located in chromosome III) and *brown* (in chromosome II).

We consider that this *Drosophila* experiment is very useful to students, because it allows working the complementation concept, to study the segregation of a sex-linked gene (*white* gene) and to introduce the fundamentals of gene interaction (*scarlet* and *brown* genes). If a basic *Drosophila* laboratory is available, logistic for the experiment is not difficult. The number of generations (and thus, the weeks needed for the whole experiment) is restricted. Furthermore, the number of weeks can be reduced if, after the first cross (complementation test), some students carry out the genetic analysis of mutant 1 strain, whereas others study mutant 2 strain. Finally, only a basic statistical level is required by the students, because all statistical analyses can be carried out using the χ^2 test.

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