Selfing the F_2 snails gives F_3 progeny, ¾ of which are dextral and ¼ of which are sinistral. The latter are the *d/d* progeny of the F_2 *d/d* female snails; these F_3 snails are sinistral because their phenotype reflects the F_2 genotype of their mother.

The reciprocal cross is of a sinistral (*d/d*) female with a dextral (*D/D*) male (Figure 13.11b). The F_1 snails are all *D/d* in genotype, yet they are sinistral in phenotype because the mother is genotypically *d/d*. Selfing the F_1 produces F_2 snails, all of which are dextral for the same reason as the reciprocal cross just described. The genotypes and phenotypes of the F_2 and F_3 generations are the same as for the reciprocal cross, again for the same reasons.

What is the basis for the coiling? The orientation of the mitotic spindle in the first mitotic division after fertilization controls the direction of coiling. The mother encodes products, deposited in the oocyte, that direct the orientation of the mitotic spindle and therefore the direction of cell cleavage. Thus, a mother of genotype *D/–* deposits a gene product that specify a dextral coiling. A mother of genotype *d/d* either does not produce a gene product, or that product is nonfunctional, and this results in default sinistral coiling.

Maternal effect is also seen for certain genes involved in axis formation during embryo development in *Drosophila melanogaster*. Those genes are discussed in Chapter 19.

Keynote

In the maternal effect, an inherited trait is controlled by the maternal nuclear genotype before the egg is fertilized and is not influenced by the paternal genotype.

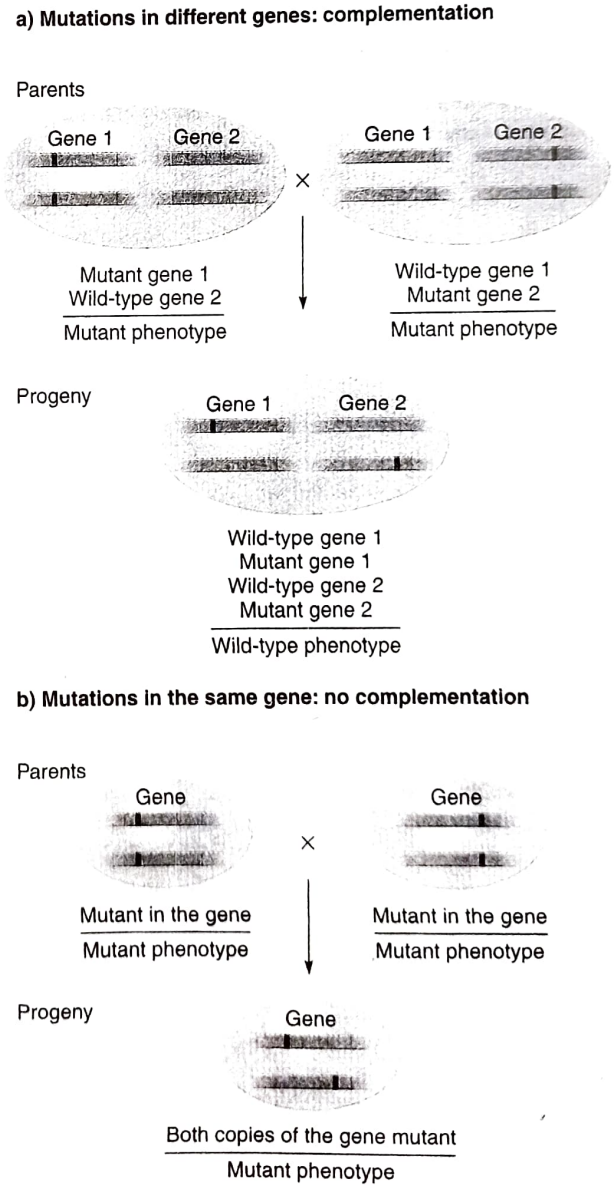
Determining the Number of Genes Involved in a Set of Mutations with the Same Phenotype

Up to this point in the book, each mutation we have discussed has affected a different gene. We will now begin to encounter cases where that is not so. To help us analyze and understand those cases, we need to understand the relationship between the phenotype and the gene in more detail.

We have learned that the general genetic approach to studying a biological process is to isolate mutants which affect that process. Those mutants are identified by their phenotype—the mutant phenotype—which is distinct from the wild-type phenotype. Consider a genetic study in which a large number of mutants are isolated, with each mutant having the same altered phenotype. Our aim is to understand the structures and functions of the genes controlling the biological process involved. Does each mutant define a different gene, or not? We can answer that question with the **complementation test,** also called the **cis-trans test,** which determines whether two independently isolated mutants with the same phenotype have mutations in the same or different genes. The complementation test was developed by Edward Lewis to study genes in *Drosophila*.

In a complementation test, two mutants resulting in the same phenotype are crossed, and the phenotype of the progeny is observed. If the two mutations involved are in different genes, then the progeny will be wild-type/mutant heterozygotes for each of the two genes involved. Because there is a wild-type copy of each gene, the phenotype will be wild type, not mutant (Figure 13.12a). We say that the two mutants complement each other. However, if the two mutations are in the same gene, then the progeny will have a different mutant version of the gene on each of the two homologues, and the phenotype will be mutant

Figure 13.12
Complementation test to determine whether two mutations resulting in the same phenotype are in the same or different genes.

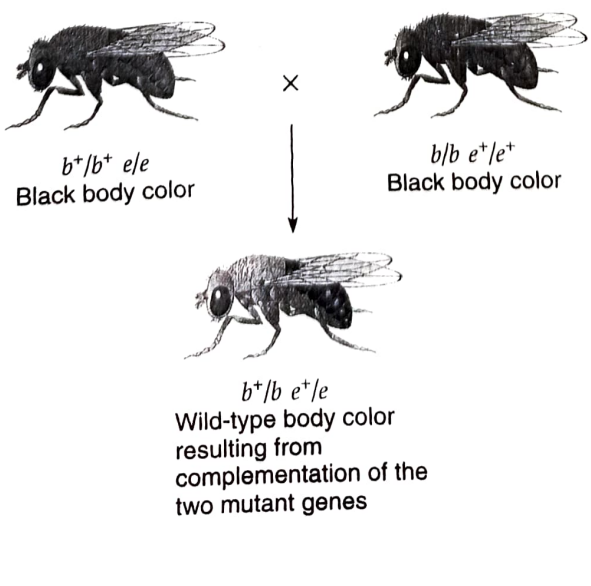


(Figure 13.12b). In this case, we say that the two mutants do *not* complement each other. Of course, the test is done on unknowns, so the interpretation is the other way around. That is, if two mutations complement each other, they must be in different genes, and if two mutations do not complement each other, they must be in the same gene. How many genes are defined by a set of mutations depends on the number of genes involved in the biological process under genetic study.

Let us consider an example from *Drosophila*. Two true-breeding mutant strains have black body color instead of the wild-type grey yellow. When the two strains are crossed, all the F_1 flies have wild-type body color (Figure 13.13). How can these data be interpreted? The simplest explanation is that complementation has occurred between mutations in two genes, each of which is involved in the body color phenotype. That is, homozygosity for a recessive mutant allele of an autosomal gene, *black* (*b*), results in a black body color. Homozygosity for a recessive mutant allele of another autosomal gene, *ebony* (*e*), which is located on another autosome, also results in a black body color when homozygous mutant. Because the two parents are homozygotes, they are genotypically b^+/b^+ *e*/*e* and *b*/*b* e^+/e^+, and each is phenotypically black. The F_1 genotype is b^+/b e^+/e. The F_1 flies have wild-type body color because there is now one wild-type allele of each gene—complementation has occurred. Importantly, *no recombination* is involved in a complementation test. Here, the double heterozygote was produced simply by the fusion of gametes produced by the two true-breeding parents.

If the F_1 flies from the cross between two independently isolated, true-breeding, recessive black-bodied mutant strains are all phenotypically black, that means that the two mutations involved did not complement each other. This result indicates that the two mutations are in the same gene.

Figure 13.13
Complementation between two black-body mutations of *Drosophila melanogaster*.



Keynote

A complementation test determines whether two independently isolated mutants with the same phenotype have mutations in the same or different genes. If a combination of two mutants results in a wild-type phenotype, then the two mutations are in different genes. If combination of two mutants results in a mutant phenotype, then the two mutations are in the same gene.

Gene Interactions and Modified Mendelian Ratios

No gene acts by itself in determining an individual's notype; instead, the phenotype is the result of complex and integrated patterns of molecular rea that are under gene control. All the genetic examp have discussed and will discuss have discrete mo bases, and in a number of cases complex interacti tween genes can be detected by genetic analysis. amine some examples in this section.

Consider two independently assorting gen each with two alleles: *A* and *a*, and *B* and *b*. The of a cross between individuals, each of which is heterozygous (*A*/*a* *B*/*b* × *A*/*a* *B*/*b*), will be nine ge in the following proportions:

1/16 *A*/*A* *B*/*B*
2/16 *A*/*A* *B*/*b*
1/16 *A*/*A* *b*/*b*
2/16 *A*/*a* *B*/*B*
4/16 *A*/*a* *B*/*b*
2/16 *A*/*a* *b*/*b*
1/16 *a*/*a* *B*/*B*
2/16 *a*/*a* *B*/*b*
1/16 *a*/*a* *b*/*b*

If the phenotypes determined by the two are distinct—for example, smooth versus wri or long versus short stems—and there is com nance, then we get the familiar dihybrid phen of 9:3:3:1, where the four phenotypic classes mal and mutant phenotypes controlled by th involved (see Figure 11.12b, p. 309). Deviatio standard 9:3:3:1 ratio of normal and mutant indicates that interaction between alleles genes is involved.

As we showed in Chapter 11, the 9:3:3: ratio can be represented genotypically in a s as *A*/– *B*/–, *A*/– *b*/*b*, *a*/*a* *B*/–, *a*/*a* *b*/*b*, respecti indicates that the phenotype is the same gene is homozygous dominant or heterozy ample, *A*/– means either *A*/*A* or *A*/*a*. This used when incomplete dominance or codo volved, because the *A*/*A* and *A*/*a* genotypes phenotypes.