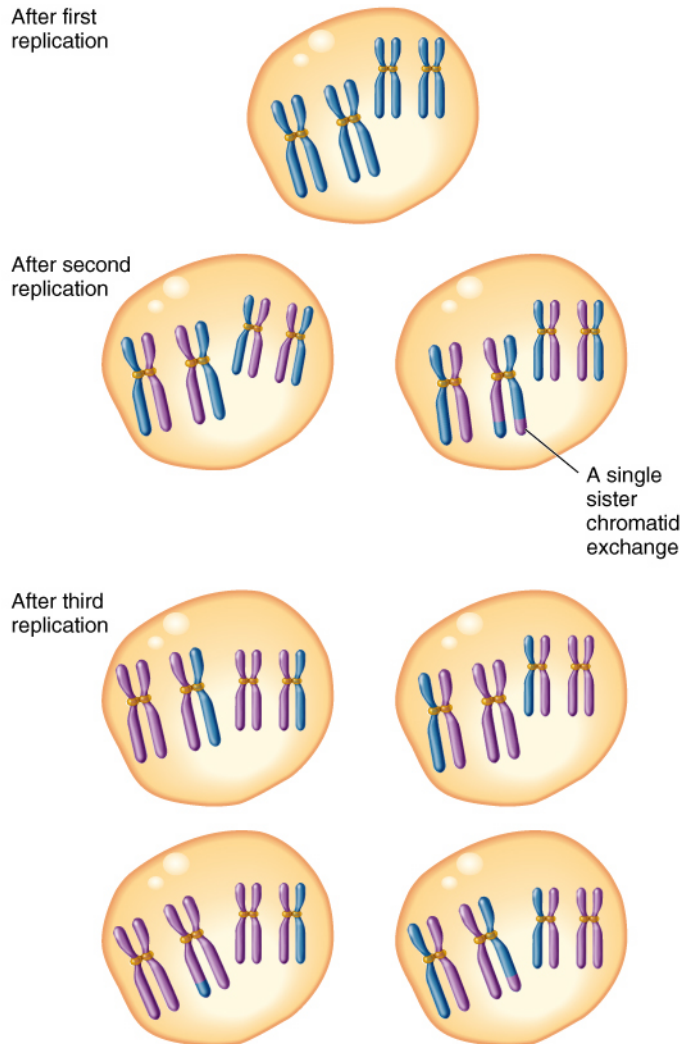


- E1. Due to semiconservative DNA replication, one of the sister chromatids has both of the DNA strands that contain BrdU while the other sister chromatid only has one of its two DNA strands that contain BrdU.
- E2. You would conclude that the substance is a mutagen. Substances that damage DNA tend to increase the level of genetic exchange such as sister chromatid exchange.
- E3. You would add it after the second round of DNA replication but before crossing over occurs during mitosis (i.e., add it during the G_2 phase). In this experiment you are detecting SCEs that occur after the second round of DNA replication. If the mutagen can persist in the cells a long time, you might be able to add it earlier.
- E4. The drawing here shows the progression through three rounds of BrdU exposure. After one round, all of the chromosomes would be dark. After two rounds, all of the chromosomes would be harlequin. After three rounds, the number of light sister chromatids would be twice as much as the number of light sister chromatids found after two rounds of replication.



- E5. BrdU inhibits Giemsa binding. As described in Chapter 8, the binding of Giemsa to chromosomes produces G bands, which are dark. The chromatids that contain both strands of BrdU are light, indicating that BrdU inhibits Giemsa binding.
- E6. When McClintock started with a colorless strain containing *Ds*, she identified 20 cases where *Ds* had moved to a new location to produce red kernels. This identification was possible because the 20 strains had a higher frequency of chromosomal breaks at a specific site, and because of the mutability of particular genes. She also had found a strain where *Ds* had inserted into the red-color-producing gene, so that its transposition out of the gene would produce a red phenotype. Overall, her analysis of the data showed that the sectoring (i.e., mutability) phenotype was consistent with the transposition of *Ds*.

- E7. It is due to the excision of the transposable element. This conclusion was based on the reversion of the phenotype (namely colorless to red), which suggests a restoration of gene function, and the nonmutability of the phenotype.
- E8. Transposon tagging is an experimental method that is aimed at cloning genes. In this approach, a transposon is introduced into a strain, and the experimenter tries to identify individuals in which the gene of interest has lost its function. In many cases, the loss of function has occurred because the transposon has been inserted into the gene. If so, one can make a library and then use a labeled transposon probe to identify clones in which the transposon has been inserted. This provides a way to clone the gene of interest. Later, one would use the transposon-inserted clone to screen a library from an individual with an active version of the gene. In this way, one could then identify a normal copy (nontransposon-inserted) of the gene.
- E9. One could begin with the assumption that the inactivation of a tumor-suppressor gene would cause cancerous cell growth. If so, one could begin with a normal human line and introduce a transposon. The next step would be to identify cells that have become immortal. This may be possible by identifying clumps of cells that have lost contact inhibition. One could then grow these cells and make a genomic DNA library from them. The library would be screened using the transposon as a labeled probe.
- E10. A transposon creates a mutable site because the excision of a transposon causes chromosomal breakage if the two ends are not reconnected properly. After it has moved out of its original site (causing chromosomal breakage), it may be inserted into a new site somewhere else. You could experimentally determine this by examining a strain that has incurred a chromosomal breakage at the first mutable site. You could microscopically examine many cells that had such a broken chromosome and see if a new mutable site had been formed. This new mutable site would be the site into which the transposon had moved. On occasion, there would be chromosomal breakage at this new mutable site, which could be observed microscopically.
- E11. First, you would inject the plasmid carrying the P element and *rosy*⁺ gene into embryos that were homozygous for the recessive *rosy* allele (i.e., *rosy rosy*). Allow these embryos to mature into adult flies, and then mate them to *rosy rosy* flies. Analyze the color of the F₁ offspring. If the eyes are red, at least one copy of the P element has hopped into the chromosome of these flies. Possibly, the P element has hopped into a gene that is involved with wing development. However, at this point, such a fly would have one normal copy of the gene and one copy that has been inactivated by the P element. If the inactivation of the gene were inherited as a dominant phenotype, these flies would have deformed wings. Alternatively (and more likely), however, the inactivation of one copy of most genes would be inherited as a recessive trait. If this were the case, the wings of the red-eyed flies would still appear normal. To identify these recessive genes, take each F₁ offspring with red eyes (and normal wings) and mate them to *rosy rosy* flies. About 1/4 of the F₂ offspring should have red eyes (assuming that one P element has hopped into the genome of the red-eyed flies). Take the F₂ offspring with red eyes (from a single cross) and mate them to each other. In other words, mate F₂ brothers and sisters to each other. From these crosses, 1/4 of the F₃ flies should have *rosy* eyes, 1/2 should have one copy of the *rosy*⁺ P element and red eyes, and 1/4 should be homozygous for the P element and have red eyes. If the P element has inserted into a gene that is required for wing development, 1/4 of the offspring should have deformed wings and red eyes. In this way, you could identify genes that are required for wing development.

Note: This experiment is a lot of work! You would have to identify thousands of flies with red eyes and set up thousands of crosses to identify a few genes that play a role in wing development. Nevertheless, this approach has been successful in the identification of genes that play a role in many aspects of *Drosophila* development.