

- E1. If the physiological adaptation theory had been correct, mutations should have occurred after the cells were plated on the media containing T1 bacteriophages. Since the same numbers of bacteria were streaked on each plate, we would have expected to see roughly the same number of resistant colonies on all of the plates. The number of resistant colonies would not have depended on the timing of the mutation. In contrast, what was actually observed was quite different. If a random mutation occurred early in the growth of a bacterial population (within a single tube), there were a large number of T1-resistant colonies on the plate. Whereas, if a random mutation occurred late, or not at all, there were few or no colonies on the plate.
- E2. When cells from a master plate were replica plated onto two plates containing selective media with the T1 phage, T1-resistant colonies were observed at the same locations on both plates. These results indicate that the mutations occurred randomly while on the master plate (in the absence of T1) rather than occurring as a result of exposure to T1. In other words, mutations are random events, and selective conditions may promote the survival of mutant strains that occur randomly.
- To show that antibiotic resistance is due to random mutation, one could follow the same basic strategy except the secondary plates would contain the antibiotic instead of T1 phage. If the antibiotic resistance arose as a result of random mutation on the master plate, one would expect the antibiotic-resistant colonies to appear at the same locations on two different secondary plates.
- E3. The paracentric inversion does not actually prevent crossing over. However, if crossing happens to occur, this produces an acentric X chromosome and a dicentric X chromosome that will eventually break. Offspring inheriting these abnormalities will probably not survive, so we do not observe them in the crosses. What the *CIB* chromosome actually does is to prevent us from observing living offspring that are the product of a crossover between the X chromosomes in the *CIB* mothers.
- E4. Perhaps the X rays also produce mutations that make the *CIB* daughters infertile. Many different types of mutations could occur in the irradiated males and be passed to the *CIB* daughters. Some of these mutations could prevent the *CIB* daughter from being fertile. These mutations could interfere with oogenesis, etc. Such *CIB* daughters would be unable to have any offspring.
- E5. Müller's experiment is not measuring the mutation rate within a single gene. There are many genes along the X chromosome, which may mutate to produce a lethal phenotype. Müller's experiment is a measure of the rate at which X-linked mutations in many different genes can occur to produce a recessive lethal phenotype.
- E6. You would conclude that chemical A is not a mutagen. The percentage of *CIB* daughters (whose fathers had been exposed to chemical A) that did not produce sons was similar to the control (compare 3 out of 2,108 with 2 out of 1,402). In contrast, chemical B appears to be a mutagen. The percentage of *CIB* daughters (whose fathers had been exposed to chemical B) that did not produce sons was much higher than the control (compare 3 out of 2,108 with 77 out of 4,203).
- E7. Haploid cells are more sensitive to mutation because they have only a single copy of each gene. Therefore, recessive mutations that inhibit cell growth are easily detected. In diploid cells, it would take a mutation in both copies of the same gene to detect its phenotypic effects.
- E8. You would expose the bacteria to the physical agent. You could also expose the bacteria to the rat liver extract, but it is probably not necessary for two reasons. First, a physical mutagen is not something that a person would eat. Therefore, the actions of digestion via the liver are probably irrelevant, if you are concerned that the agent might be a mutagen. Second, the rat liver extract would not be expected to alter the properties of a physical mutagen.
- E9. Absence of mutagen:  $17/10,000,000 = 17 \times 10^{-7} = 1.7 \times 10^{-6}$   
 Presence of mutagen:  $2,017/10,000,000 = 2.0 \times 10^{-4}$   
 The mutagen increases the rate of mutation more than 100-fold.
- E10. The results suggest that the strain is defective in excision repair. If we compare the normal and mutant strains that have been incubated for 2 hours at 37°C, much of the radioactivity in the normal strain has been transferred to the soluble fraction because it has been excised. In the mutant strain, however, less of the radioactivity has been transferred to the soluble fraction, suggesting that it is not as efficient at removing thymine dimers.