AP BIOLOGY · UNIT 7 · TOPIC 7.8

# Continuing Evolution

*Learning objective 7.8.A Explain how evolution is an ongoing process in all living organisms.*

Name: Date: Period:

Use the tool **Still Happening**. Parts 1 and 2 use **The experiment that settled it**, Part 3 uses **What the drug actually does**, and Parts 4 and 5 use **Four places, one process**. Evolution is not something that finished in the past. A flask of bacteria shows the whole process in a week. In 1943 Luria and Delbrück showed that resistant bacteria were present **before** the bacteria were exposed to the agent they resist. Their experiment used a virus that kills bacteria; the tool runs the same test with an antibiotic. This result corrects the most common misconception in this topic.

## Part 1 · The experiment

**60** parallel cultures, each grown from a single cell to about **1,048,576** cells, then plated on antibiotic. The tool models two hypotheses for where the resistant cells come from. Click each hypothesis at the top of the tab to see its cultures, then copy both rows from the table in the panel headed “Why the average could never have answered it.”

| Hypothesis | Mean survivors | Variance | Variance ÷ mean | Cultures with none | Most in one culture |
|---|---|---|---|---|---|
| Mutations arise at random during growth | | | | | |
| The antibiotic induces resistance | | | | | |

**1. The experiment in Part 1 is Luria and Delbrück's fluctuation test.**
**(a) Describe the design of the fluctuation test.**

**(b) Explain what the result of the fluctuation test shows about when resistant bacteria arise.**

**2. In the tool, the two hypotheses give nearly the same mean number of survivors per culture (0.95 and 0.90). Explain why the experiment compared the variance between cultures instead of the mean.**

## Part 2 · Where the jackpots come from

A culture starts as one cell and doubles **20** times, to 1,048,576 cells. For each division in the table, a single mutation for resistance happens at that division. Record the doublings remaining after that division, then calculate the number of resistant cells at the end and the fraction of the culture that is resistant. Assume that all descendants of the mutant cell inherit the resistance.

| Mutation happens at division… | Doublings remaining | Resistant cells at the end | Fraction of the culture |
|---|---|---|---|
| 1 | | | |
| 5 | | | |
| 10 | | | |
| 15 | | | |
| 20 | | | |

**3. Using the Part 2 table, explain why random mutation during growth produces very large differences in the number of resistant cells between cultures that were treated identically.**

**4. Explain why the induction hypothesis cannot produce the large differences between cultures described in question 3, however many cultures are tested.**

## Part 3 · What the drug actually does

Go to **What the drug actually does**. One cell in a million is resistant before any drug is applied. For question 7, open “Why resistance fades when the drug is withdrawn, and why slowly” lower on the tab. Click each condition and record what happens over **200** generations.

| Condition | Frequency after 200 generations | Fold change | Generations to a majority |
|---|---|---|---|
| No antibiotic | | | |
| Low dose | | | |
| Full course | | | |

**5. A student claims that the antibiotic caused the bacteria to become resistant. Explain why the student’s claim is incorrect.**

**6. Another student claims that individual bacteria adapted to the antibiotic. Explain why this claim is also incorrect.**

**7. (a) Using the No antibiotic row of the Part 3 table, describe how the frequency of resistance changes over 200 generations when no antibiotic is present.**

**(b) Explain why the frequency of resistance changes in this way when no antibiotic is present.**

## Part 4 · Four places, one process

Go to **Four places, one process**. The four cases involve different organisms and different chemicals. Click each case (Antibiotic, Pesticide, Herbicide and Chemotherapy) and record its three readouts.

| Case | Selective agent | What survives it | Generation time |
|---|---|---|---|
| Antibiotic resistance in bacteria | | | |
| Pesticide resistance in insects | | | |
| Herbicide resistance in weeds | | | |
| Chemotherapy resistance in tumor cells | | | |

**8. Explain how the evolution of resistance to a chemotherapy drug in a tumor is the same process as the evolution of resistance to an antibiotic in bacteria.**

**9. Evolution is an ongoing process in all living organisms.**
**(a) Identify three different kinds of examples that show evolution is ongoing.**

**(b) For one of your examples, explain how it shows that evolution is ongoing.**

## Part 5 · Evolution you cannot see

Scroll down to the panel headed **Evolution you can measure in one species in one year**. Resistance is the visible case of ongoing evolution; change in the genome is a less visible one. The tool's table shows all seven values. Copy the four given values, then calculate the other three yourself (the rate per base uses the bases in both copies of the genome), showing each calculation in the How you get it column, and compare your results with the tool's. For question 11, open "Pathogens, and why some vaccines are reformulated every year" below the table.

| Quantity | Value | How you get it |
|---|---|---|
| New mutations per person per generation | | Given |
| Size of the human genome | | Given |
| Bases in both copies of the genome | | Given |
| Mutation rate per base per generation | | |
| Human births per year | | Given |
| New mutations in the species each year | | |
| Times each position in the genome is mutated per year | | |

**10. Using the Part 5 table, explain how each human genome can be a nearly exact copy of its parents' genomes while the human species gains billions of new mutations every year.**

**11. The influenza vaccine is reformulated most years. Explain, in terms of natural selection, why the vaccine must be reformulated.**

## What the model simplifies

The fluctuation test is simulated as clean exponential growth with a single mutation rate, no back-mutation, no cell death, and no growth-rate difference between mutant and normal cells. Real cultures have all four, and modern versions estimate mutation rates by fitting a distribution rather than by comparing a variance to a mean. The qualitative result the experiment was designed to settle is robust to all of it.

Resistance is treated as one allele with one fitness effect. Real resistance is often several genes, sometimes carried on plasmids that transfer between bacteria, which spreads it far faster than inheritance alone would, and is why resistance can appear in a species that never evolved it.

The selection panel holds fitness constant for 200 generations. A real infection ends, is cleared by an immune system, or kills its host, and the drug concentration varies through the body and over time.

The middle row of the Part 3 table repays careful reading rather than becoming a slogan. A weak dose is not a stronger selective force: it took 86 generations to bring resistance to a majority against 5 for a full course. What a weak dose does is leave a large population alive while that selection acts. The public-health advice to complete a course rests on more than this model contains, and its evolutionary justification is more debated among researchers than most textbooks suggest.

The genomic figures are round numbers from the literature (roughly 60 new mutations per person per generation, roughly 130,000,000 births a year), used to make an order-of-magnitude point rather than a precise measurement. The 60 new mutations per person are counted across both copies of the genome, so the rate per base divides by both copies (6,200,000,000 bases). That gives about $1 \times 10^{-8}$ per base per generation, the accepted figure for humans.