

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 runs the whole process in a week, and in 1943 Luria and Delbrück found a way to prove that the resistant cells were there **before** the antibiotic was — which is the single most useful thing in this topic, because it settles the misconception almost everyone starts with.

Part 1 • The experiment

60 parallel cultures, each grown from a single cell to about **1,048,576** cells, then plated on antibiotic. Two explanations are on trial. Record what each predicts and what the tool shows.

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

1. Describe the fluctuation test and explain what its result shows.

2. Both hypotheses were set up to give the same average number of survivors. Explain why the experiment measured the variance between cultures instead of the average.

Part 2 · Where the jackpots come from

A culture starts as one cell and doubles **20** times. Work out what fraction of the final culture is resistant if the mutation happens at each of these divisions — assume one mutation, and that all its descendants inherit it.

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

3. Using your table, explain why random mutation during growth produces such enormous differences between identically treated cultures.

4. Explain why induction cannot produce that pattern, however many cultures you run.

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. Record what happens under each condition over **200** generations.

Condition	Frequency at the end	Fold change	Generations to a majority
No antibiotic			
Low dose			
Full course			

5. A student says the antibiotic caused the bacteria to become resistant. Explain what is wrong with that.

6. Explain why it is also wrong to say that an individual bacterium adapted to the antibiotic.

7. Using the first row of your table, explain why resistance declines when an antibiotic is withdrawn, and why it declines slowly.

Part 4 • Four places, one process

Go to **Four places, one process**. Different organisms, different chemicals, different industries. Fill the table and then say what all four have in common.

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

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

9. Explain how evolution is an ongoing process in all living organisms, giving three different kinds of example.

Part 5 · Evolution you cannot see

Open the panel headed **Evolution you can measure in one species in one year**. Resistance is the visible case; this is the quiet one. Do the arithmetic yourself before checking it.

Quantity	Value	How you get it
New mutations per person per generation		Given
Size of the human genome		Given
Mutation rate per base per generation		
Human births per year		Given
New mutations in the species each year		
Times the genome is covered per year		

10. Explain how the human genome can be both a faithful copy of a parent's and changing measurably every year.

11. Explain why the influenza vaccine is reformulated most years, in terms of selection.

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 dose 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.