Activity 1.2.3: Attack of the Superbugs

Step 28: Summary Paragraph

There are a few different mechanisms of antibiotic resistance, all eventually doing the same thing, but taking different routes to get there. To start, selection pressure, essentially "survival of the fittest," occurs when a specific bacteria cell contains a mutation that allows it to survive in a dangerous environment where the same kind of bacteria without the mutation die, leaving only the bacteria cells with the resistant gene (to the antimicrobial/antibiotic) alive. This gene is then passed on to future generations of the bacteria, making it resistant to the antimicrobial for the foreseeable future, often because the antimicrobial cannot reach the target protein. Another method is conjugation, where a donor cell passes on a resistance gene to a recipient gene through a pilus, a spindle-like tail at the end of the cell. This gene lies within the plasmid, an independent section of DNA, outside of the bacterial DNA, which can easily replicate itself and pass the information along. There is also transformation, a method where living cells pick up the "naked" DNA, DNA leftover from dead cells, and incorporate it into its own DNA. And last but not least, there is transduction, a method where infected bacteria inadvertently creates and sustains bacteriophage (or phage) that continue to contaminate more bacteria cells once the original dies and its components are left to float about the body. Overall, all of these methods eventually create stronger, resistant bacteria cells, but through different methods.

Conclusion Questions

1. What was the purpose of culturing bacteria on these four plates?
   Each of the four plates has a different antibiotic, or lack thereof, that has to be separated for accuracy. There was "no antibiotics" which can obviously not have any antibiotics touching it. Having it on a different plate ensures that there is no contamination. This also ensures that the plate with a single antibiotic does not accidentally become contaminated by the other antibiotic, leading to false results.

2. How did your predicted results compare to your observed results?
   My prediction was accurate. I did not know which antibiotic specifically would affect which strain, so that was not included in my guess. I did, however, predict that single antibiotics would only affect one strain, and a mixture would affect both. Overall, my prediction was accurate.

3. Why did the *E. coli* I strain grow on both the LB agar plate and the LB agar plate with streptomycin but not on the LB agar plate with ampicillin?
   E. coli I strain grew on the LB agar plate because there was not an antibiotic present, meaning there was nothing stopping the growth of the bacteria. It also grew on the LB agar plate with streptomycin because it is not affected by that specific antibiotic, so it was not blocked from growing. It was, however, affected by the LB agar plate with ampicillin because it is affected by the antibiotic, so it was blocked from growing.

4. Why did neither strain grow on the plate containing both streptomycin and ampicillin?
   Both strains were blocked by either streptomycin or ampicillin. When both antibiotics were added to the plate, they were both allowed to block a strain. This means that there was an antibiotic "covering" whichever bacteria strain it may have been and preventing it from growing successfully without limiting itself to only one possible antibiotic/strain stunt.

5. Craft an explanation for Angela about what the results indicate about the new strain of bacteria produced when both strains of *E. coli* were mixed together.
   When *E.coli* I and II were mixed together, creating what we can call *E. coli* III, the resistance to their respective antibodies was combined to create an even more resistant bacteria. Theoretically, when both antibodies were added to the mixture of both strains, both strains should have been stunted. This did not happen though because instead of becoming twice as susceptible, the mixture became twice as resistant to the individual antibiotics.

6. What mechanism of gene transfer do you think was responsible for transferring the antibiotic-resistant gene between these two strains of bacteria? Explain your answer.
7. Based on the results of the experiment, as well as what you learned about the mechanism of gene transfer between bacterial cells, was the streptomycin-resistant gene transferred from Strain I to Strain II, or was the ampicillin-resistant gene transferred from Strain II to Strain I? How do you know?
   During conjugation, the most likely way the resistance DNA was transferred between strains given that the strains were in contact with each other and had previously shown no signs of sharing resistance (despite the one-way rule), requires a donor cell's pilus to touch a recipient cell. Through this, the donor cell is able to directly transfer the resistance gene to the recipient. Once this process is completed, the recipient gene is now a donor gene and can do the same to other cells, helping spread the immunity (why you need to get a vaccine… even cells know).
   It would appear that the streptomycin-resistant gene was the one transferred because looking at the images provides of the lab, the patterns of simply streptomycin and streptomycin & ampicillin match much closer than simply ampicillin. This lead me to believe the streptomycin-resistant gene was the one being transferred to yield such a similar result.

Conclusion 6 and 7 combined