

## A new antibiotic kills pathogens without detectable resistance.

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### BACKGROUND/HYPOTHESIS:

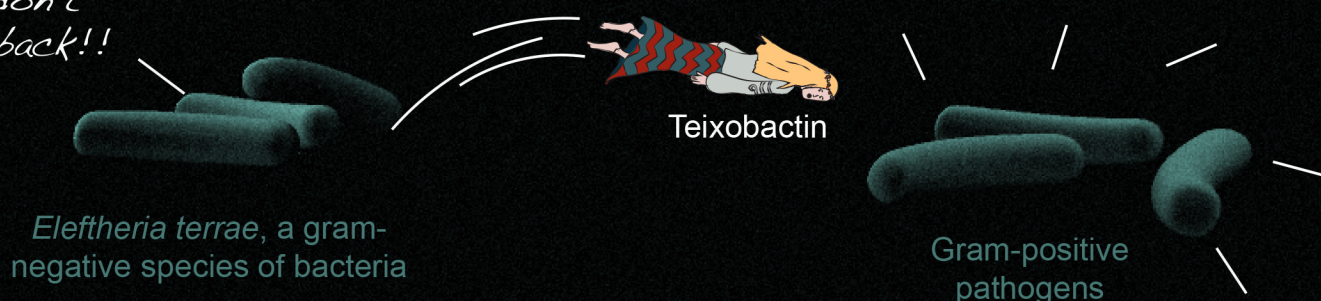
Since the '60's, we have exhausted the search for antibiotics in the 1% of bacterial species that can be cultured in the lab. As for the other 99%, this group of researchers figured, if you can't bring the bacteria to the lab, then bring the lab to them.

### APPROACH:

The iChip is a small device containing several channels, each holding about one bacterium and separated from the environment by semi-permeable membranes. By placing this in native soil, colonies can gain a foothold, and many can then be moved back to the lab for culturing.

**RESULTS:** One such colony produced an antibiotic that kills *Staphylococcus aureus*, *Mycobacterium tuberculosis*, *Clostridium difficile*, *Bacillus anthracis*, and many more gram-positive pathogens, including resistant strains. But they couldn't even be forced to develop resistance to this drug.

*And don't come back!!*



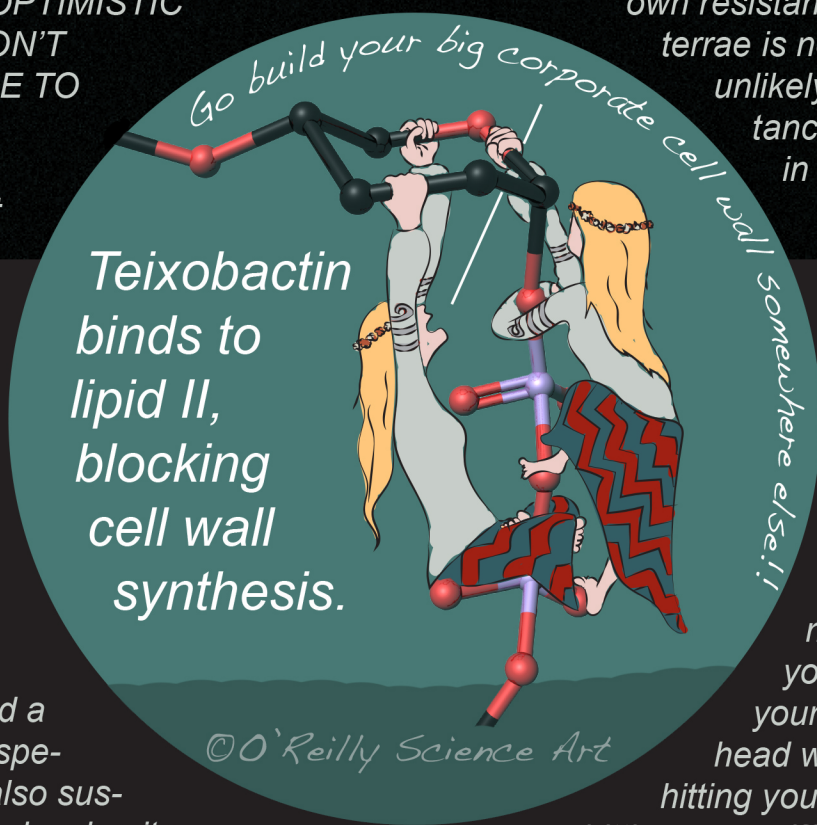
### SO WHY CAN WE BE OPTIMISTIC THAT PATHOGENS WON'T DEVELOP RESISTANCE TO TEIXOBACTIN?

For one thing, the target is a lipid, so pathogens can't just simply mutate a protein or DNA to develop resistance. This is why it took 30 years for resistance to vancomycin to become widespread.

Secondly, vancomycin resistance may have had a head start because the species that produces it is also susceptible to it, and had to develop its

own resistance mechanism. *E. terrae* is not susceptible, so it is unlikely that any such resistance mechanism is already in place.

**Teixobactin binds to lipid II, blocking cell wall synthesis.**



### ADDED BONUS:

Teixobactin ultimately inhibits teichoic acid synthesis, effectively untethering autolysins from the membrane and letting them chew up whatever peptidoglycan there is. It's the microbial equivalent of your big brother grabbing your fist, pounding your head with it, and saying, "Stop hitting yourself. Why are you hitting yourself?" over and over again.