

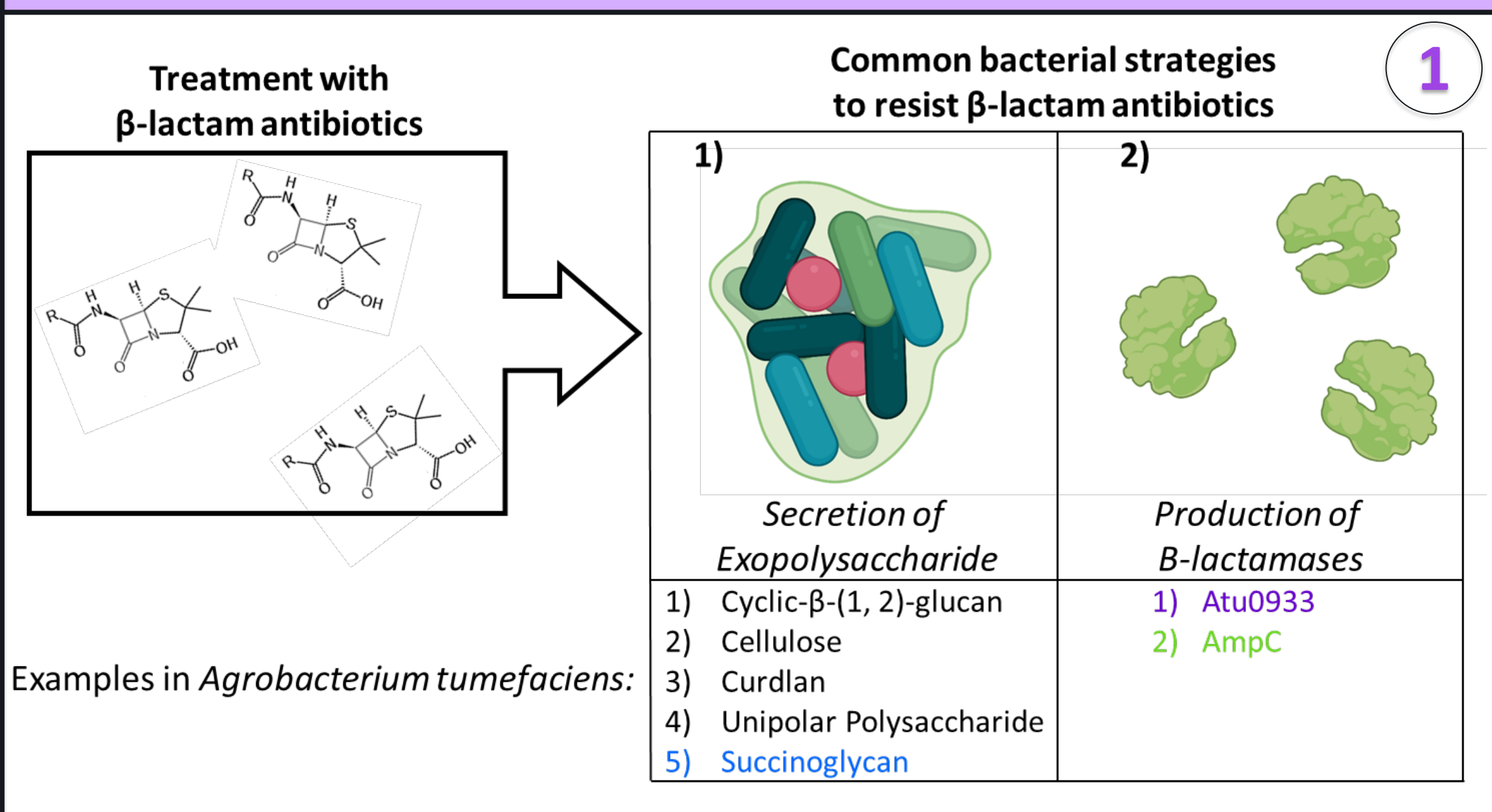


# Succinoglycan and $\beta$ -lactamase Production Confers Resistance to Cell-Wall Targeting Antibiotics in *Agrobacterium tumefaciens*

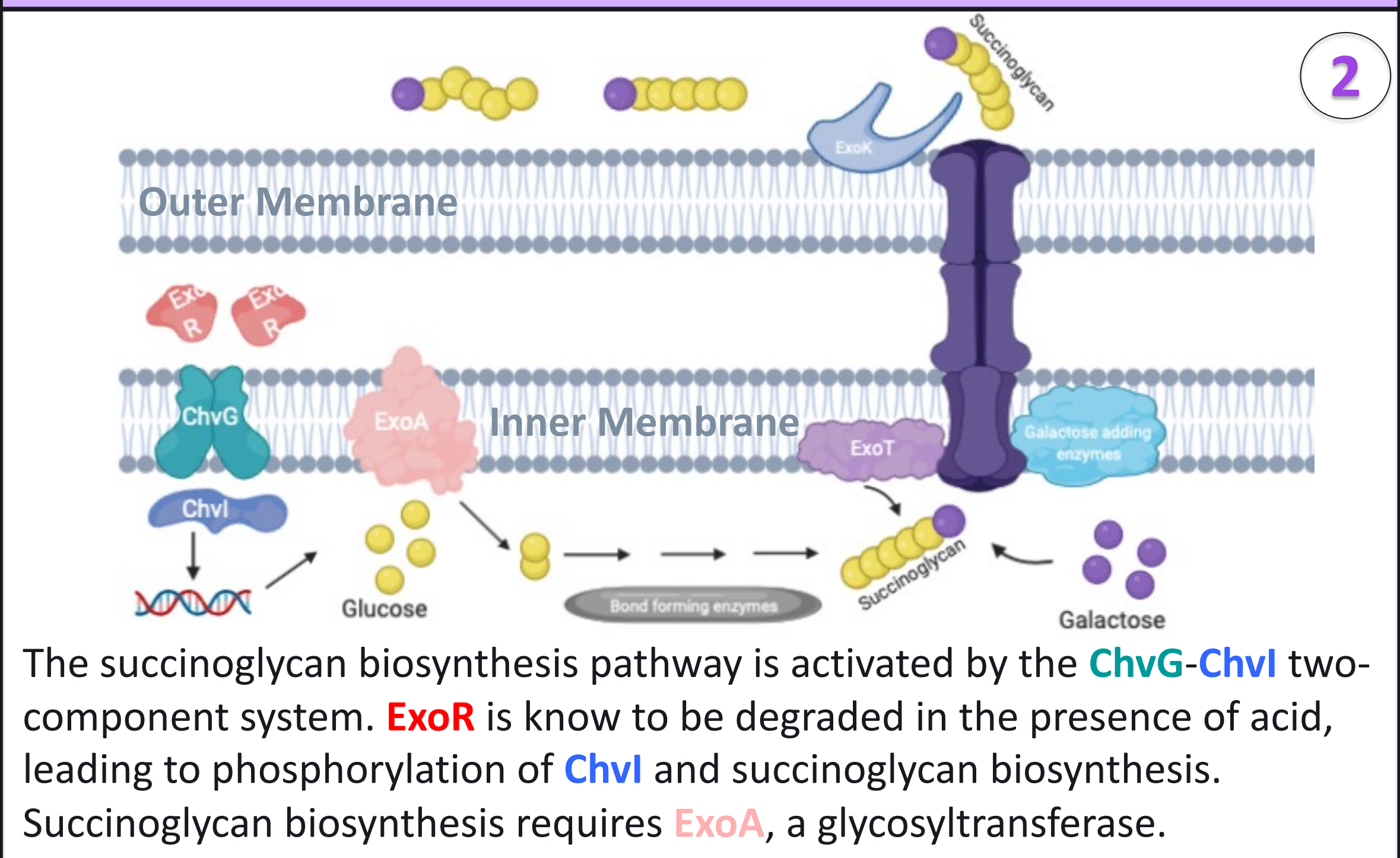


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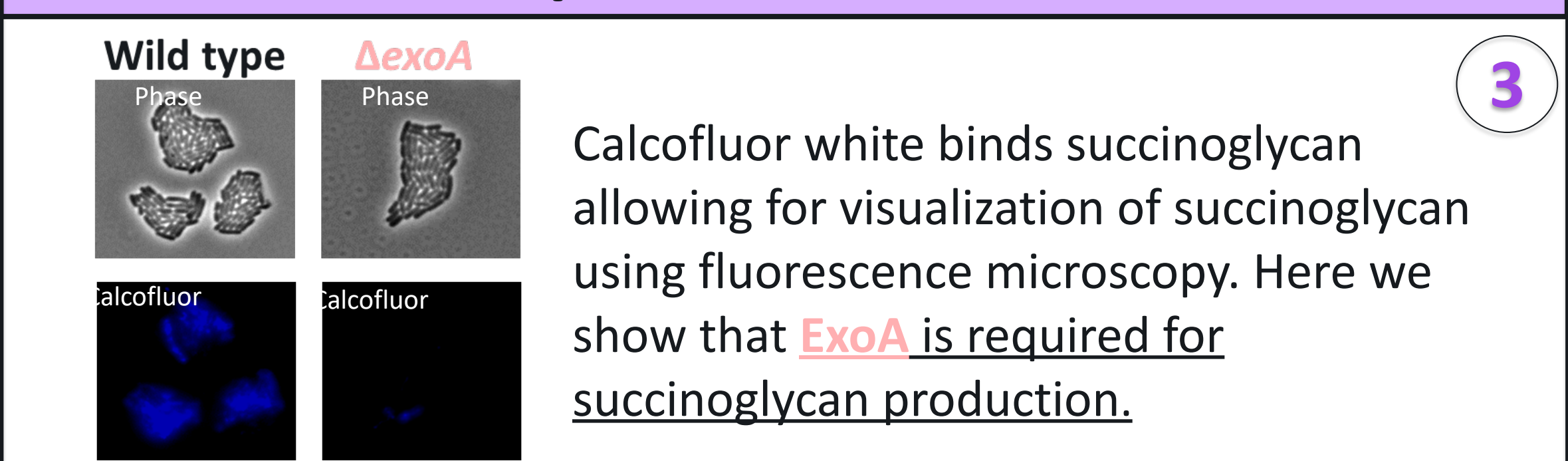
## Common strategies to resist $\beta$ -lactams



## The succinoglycan biosynthesis pathway



## Succinoglycan production and export is dependent on ExoA



## Objective and Hypothesis

### Research Question:

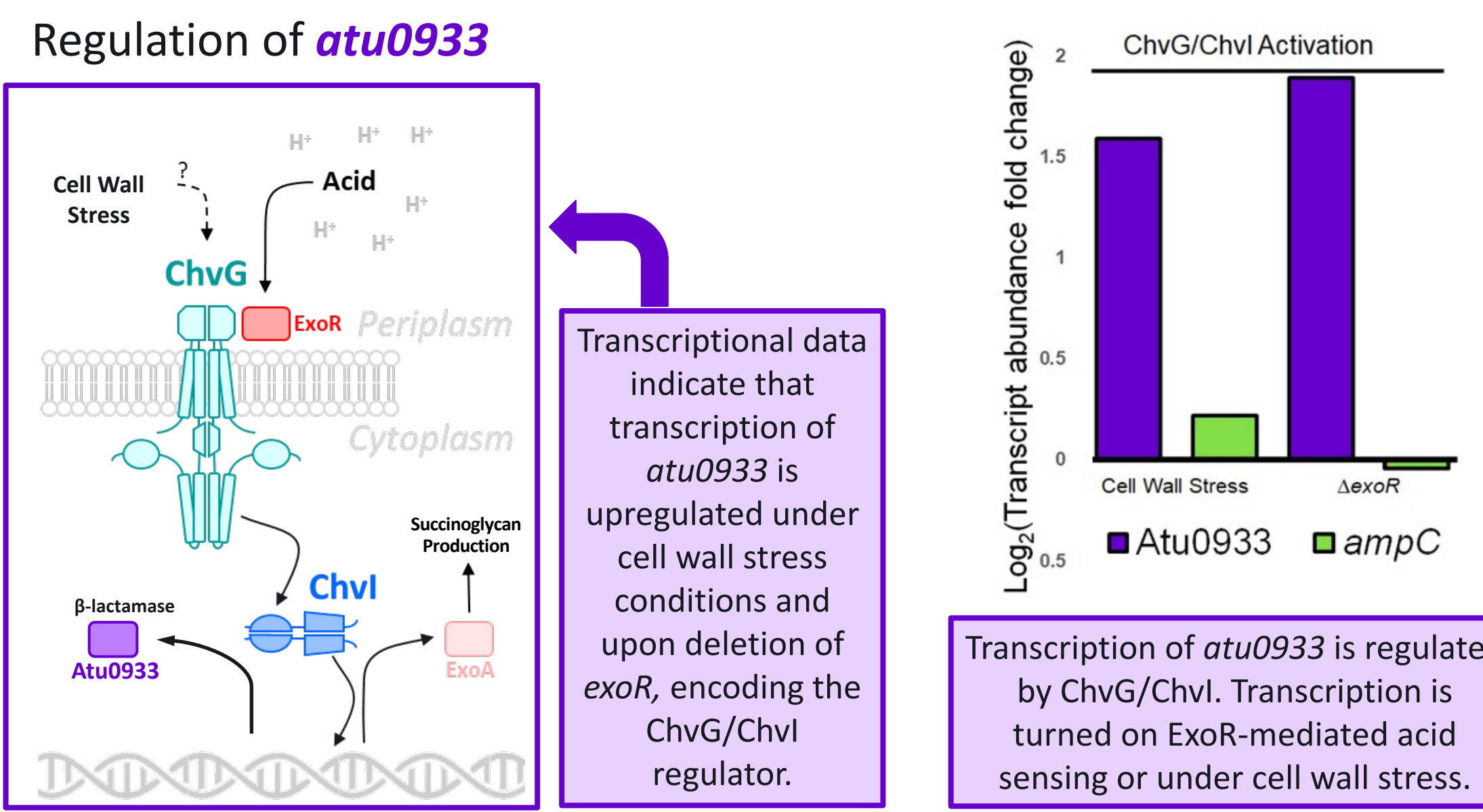
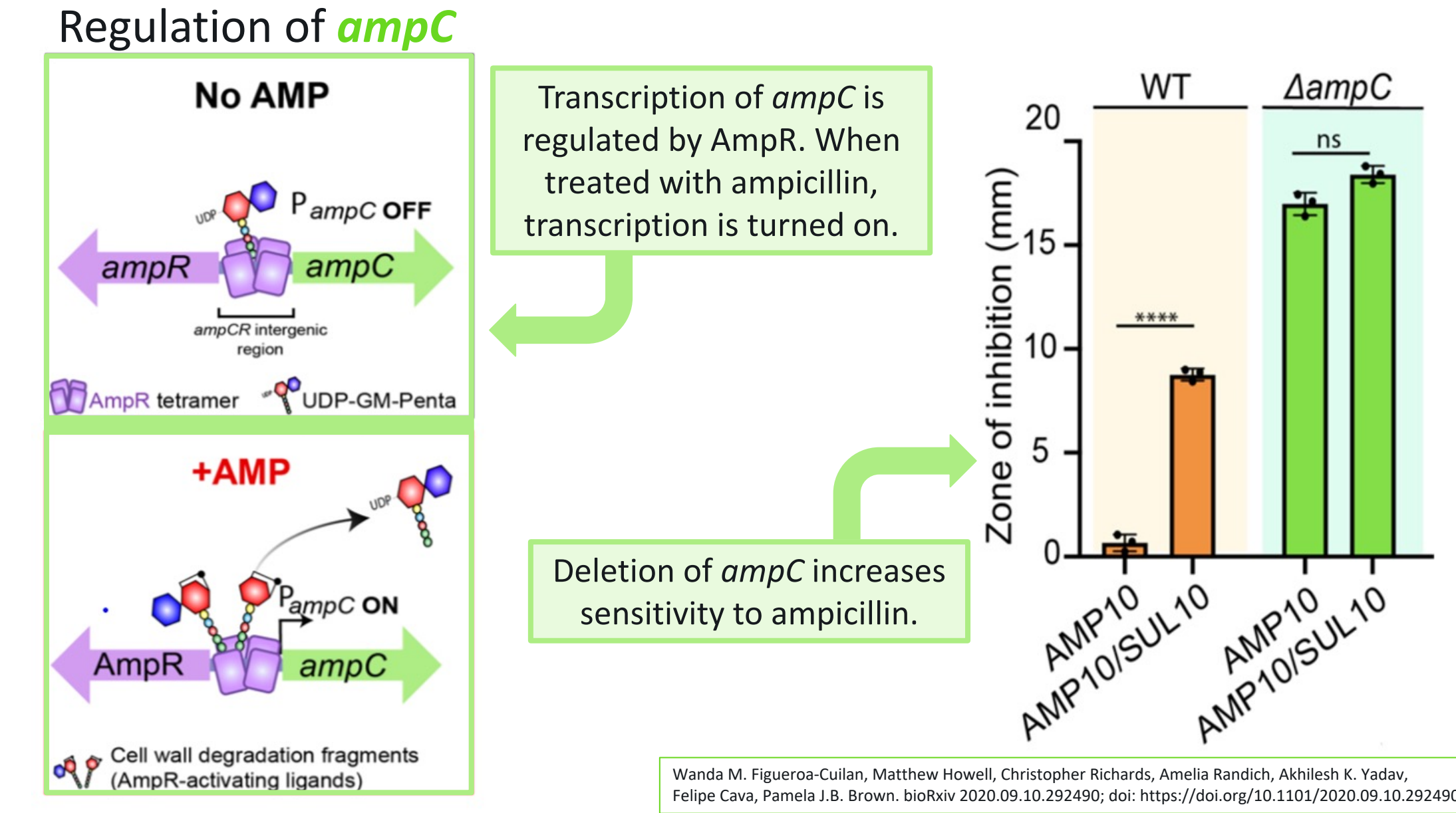
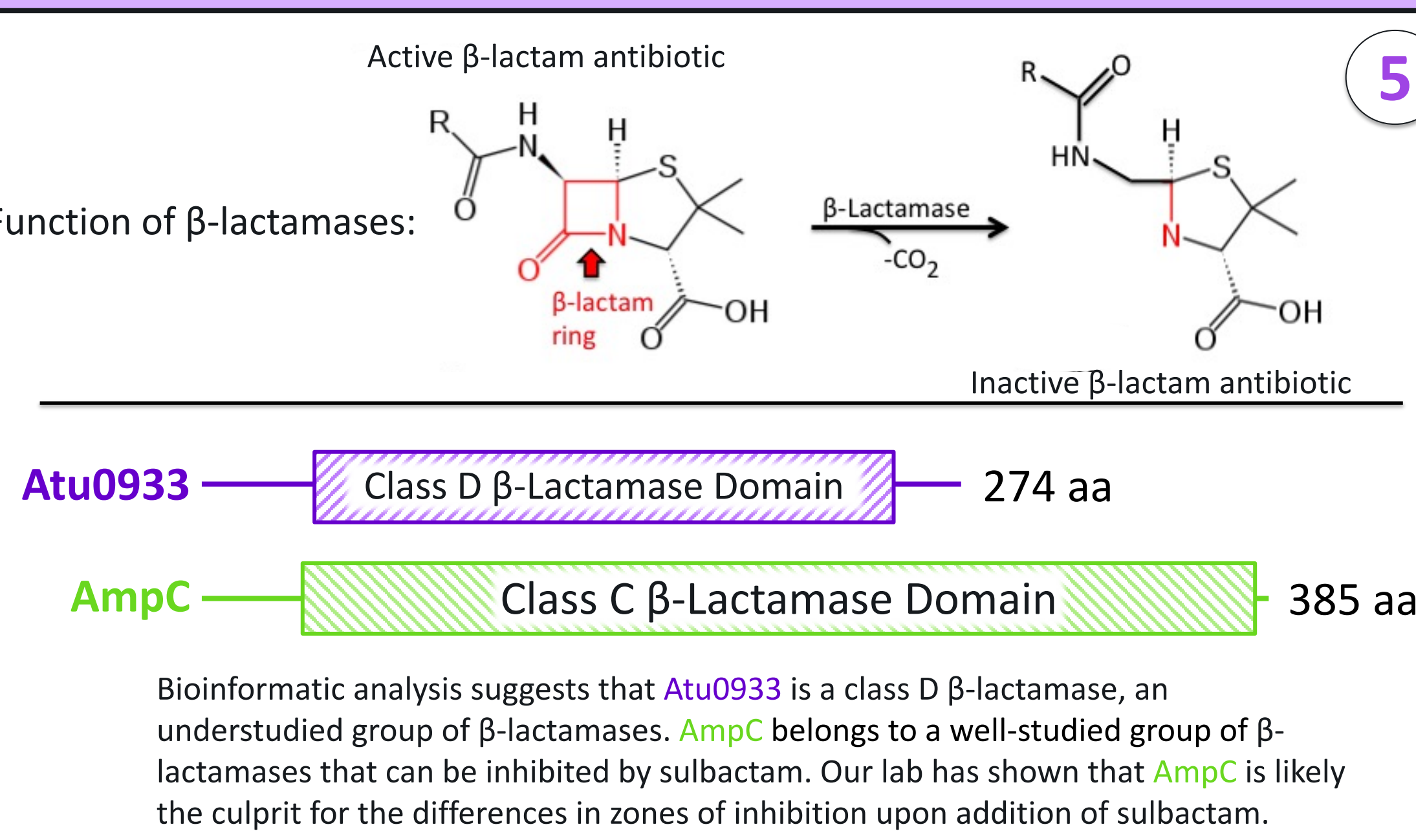
How does *Agrobacterium tumefaciens* tolerate beta-lactam antibiotics?

Using an antibiotic disc diffusion assay we find that ***ΔexoA***, ***ΔchvG***, and ***ΔchvI***, show increased sensitivity to  $\beta$ -lactam antibiotics. We also see increased size in zones of inhibition when adding sulbactam, a  $\beta$ -lactamase inhibitor. From these data we can conclude 2 things:

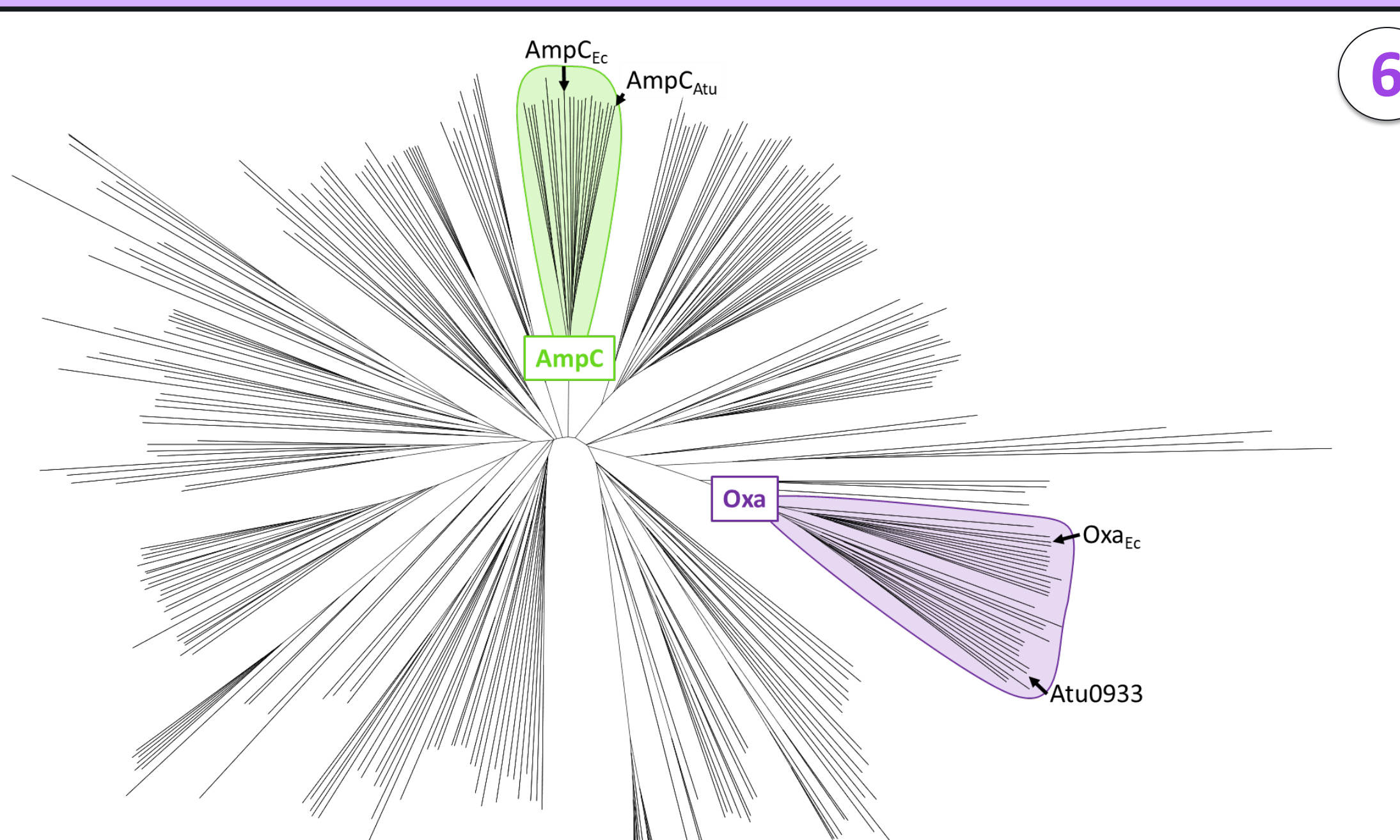
- 1) Succinoglycan contributes to  $\beta$ -lactam resistance.
- 2) There are  $\beta$ -lactamases contributing to  $\beta$ -lactam resistance.

**Hypothesis:** *A. tumefaciens* secretes  $\beta$ -lactamases which contribute to survival during cell wall-targeting antibiotic stress.

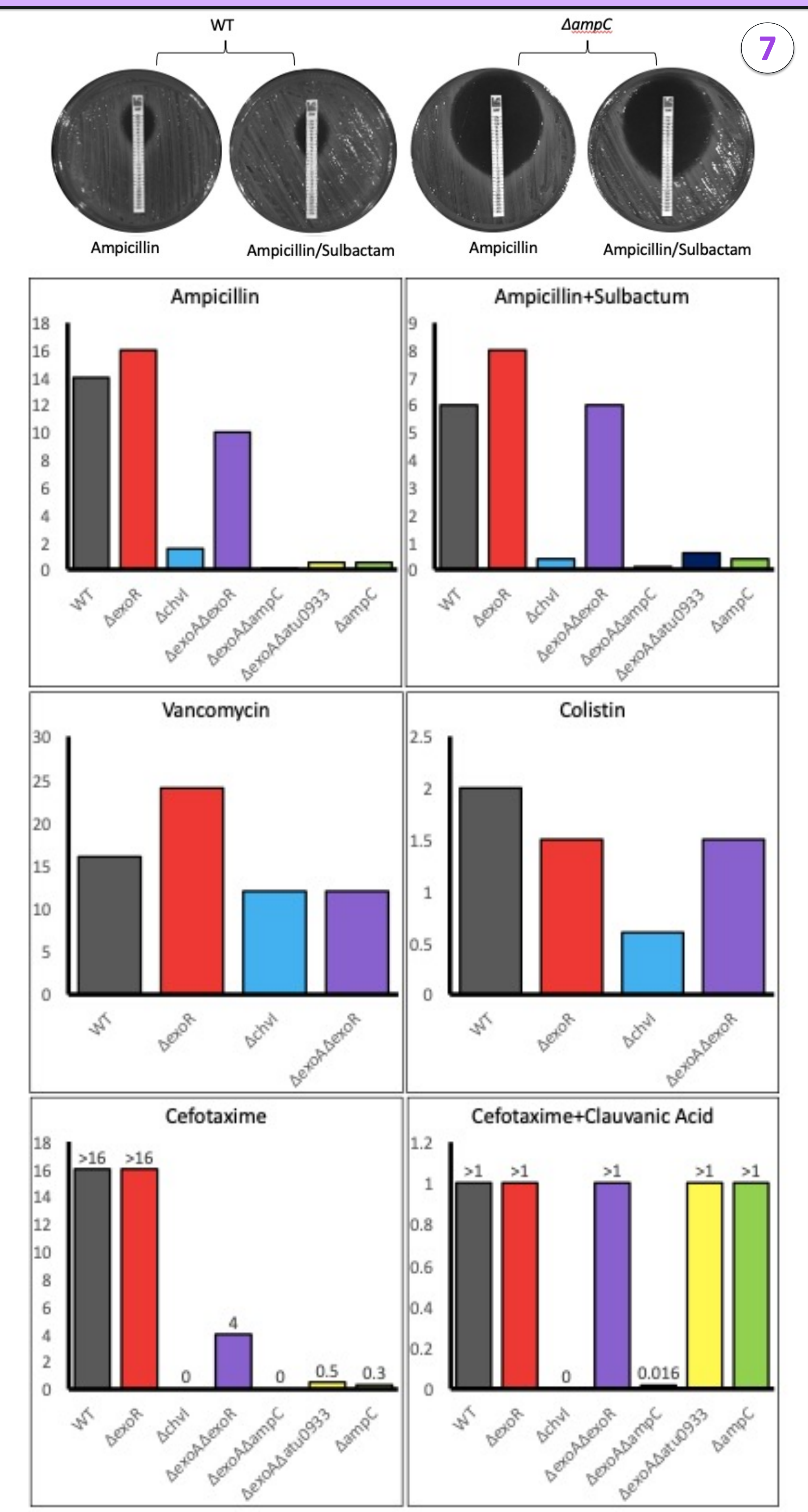
## The genome of *A. tumefaciens* encodes two $\beta$ -lactamases that are differentially regulated



## Phylogenetic analysis indicates Atu0933 may be an Oxa ortholog



## MIC Strip Data



## Summary of Results

- ***ΔexoR*** constitutively produces succinoglycan, providing resistance to each of antibiotic stress
- ***ΔchvI*** has extreme sensitivity to ampicillin and cefotaxime suggesting that the ChvG-ChvI pathway is required for some level of resistance
- The loss of succinoglycan through the deletion of ***ΔexoA***, resulted in increased sensitivity to ampicillin, but not to the level of the chvI deletion, suggesting that chvI regulates additional resistance mechanisms

## Acknowledgements

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