Title: CBP60g as a Salt Stress and Combined Stress Regulator

Plants constantly navigate multiple stressors that challenge both survival and immunity. My research demonstrates that the immune regulator *CBP60g* is not only involved in defense pathways but could also confer salt stress tolerance and increased disease resilience to *Pseudomonas syringae* DC3000 (*Pst* DC3000) under salt stress. Overexpression of *CBP60g* rescues the SA pathway inhibited under salt stress, suggesting a protective role of CBP60g under saline conditions.

Effector translocation assays reveal that *Pst* DC3000 virulence increases under salt stress in Col-0, but not in 35S::CBP60g, which could imply that CBP60g also limits bacterial invasion. Ion content analysis further shows that 35S::CBP60g plants maintain a lower Na^+/K^+ ratio than Col-0, which may contribute to salt tolerance. This indicates a more complex role of CBP60g, not only in response to pathogen infection but also salt stress alone, potentially by modifying the apoplastic environment or limiting ion intake as a whole.

Through transcriptomic, metabolomic, and microbiome analyses, I investigate how CBP60g influences host defense and apoplastic dynamics under salt stress. My findings reveal CBP60g's key role in shaping plant immunity and salt stress adaptation.