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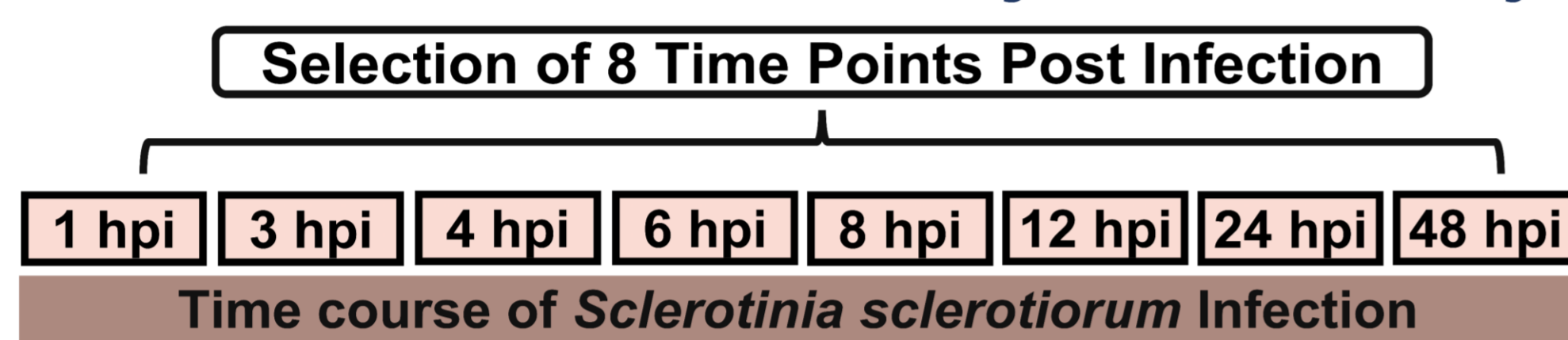
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ABSTRACT

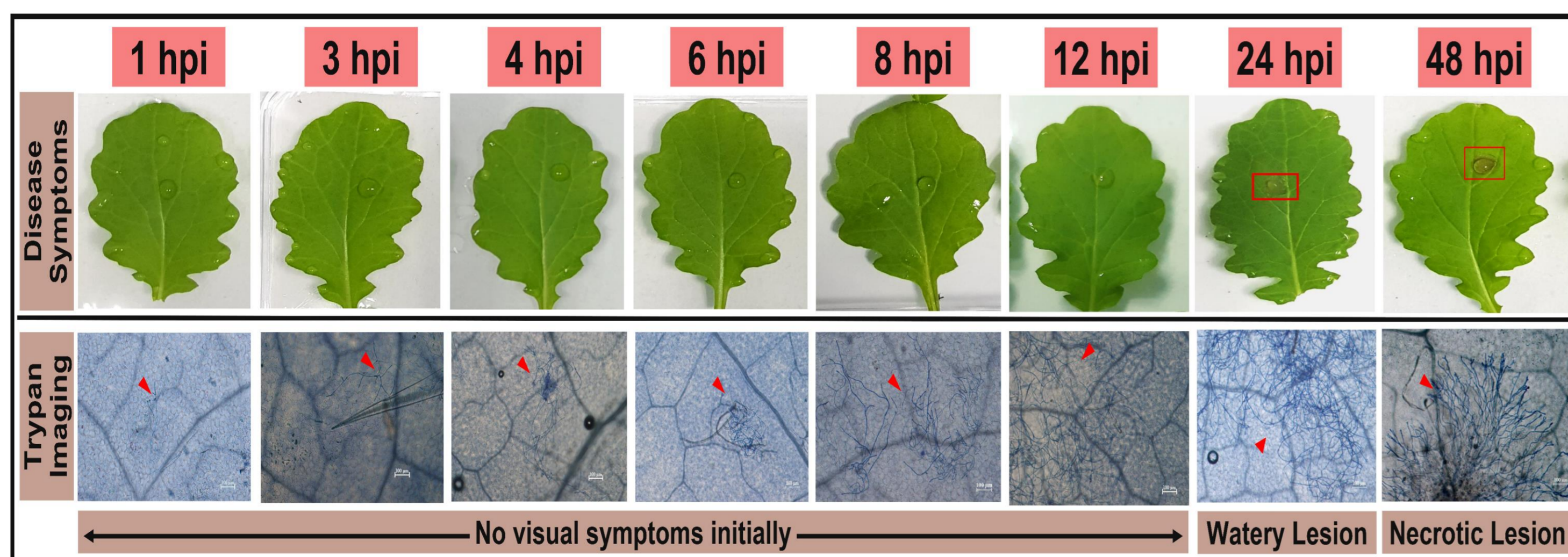
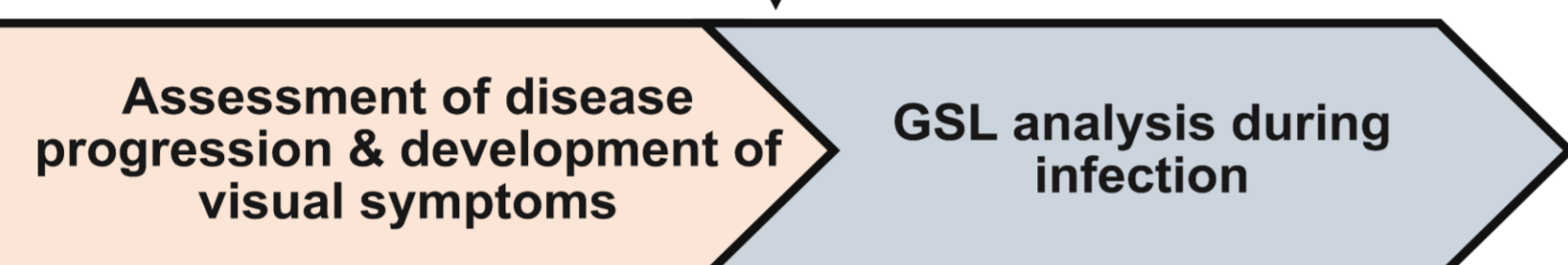
Brassica juncea the major oilseed crop of the Indian subcontinent contributes to >28% of the total oil production, but is under constant threat from environmental challenges, like the white-mold disease caused by the necrotrophic phytopathogen *Sclerotinia sclerotiorum*. White-mold proves a bottleneck to the cultivation of *B. juncea* where it causes >92% losses, leading to qualitative and drastic deterioration of the yield and seeds oil contents. The mustard species also house unique secondary metabolites called glucosinolates (GSLs) which along with their hydrolysis products (GHPs) govern defense against various phytopathogens, including *S. sclerotiorum*. Despite the reported toxicity of GSLs and their products, *Sclerotinia* is able to infect, colonize and cause significant yield losses on glucosinolates producing plants. Therefore in the present work, we establish the dynamics of *S. sclerotiorum*-*B. juncea* pathosystem in terms of the glucosinolate-myrosinase system. We in turn investigate the role of C3 aliphatic glucosinolate derived product, that has proven to be one of the most toxic to curtail the growth of *Sclerotinia*. In addition, we screen 250 natural accessions of *B. juncea* spanning a range of variation in different glucosinolate fractions, particularly C3 GSLs. This in turn will be useful to identify resistance and proposed roles of GSLs to control the menace of white-mold in polyploid mustard species.

1. The disease dynamics of *S. sclerotiorum* infection in Indian oilseed mustard *B. juncea*

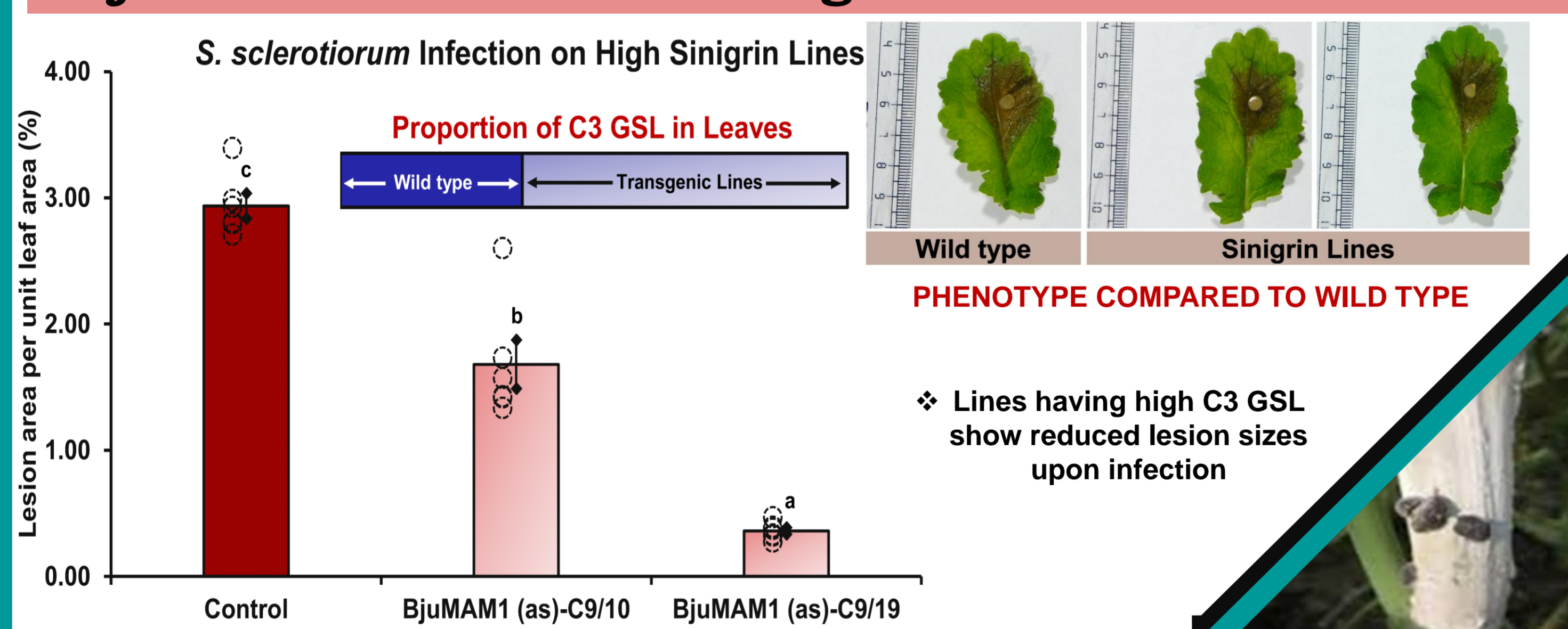
Dynamics of *S. sclerotiorum* - *B. juncea* Pathosystem



Disease progression on *B. juncea* plants



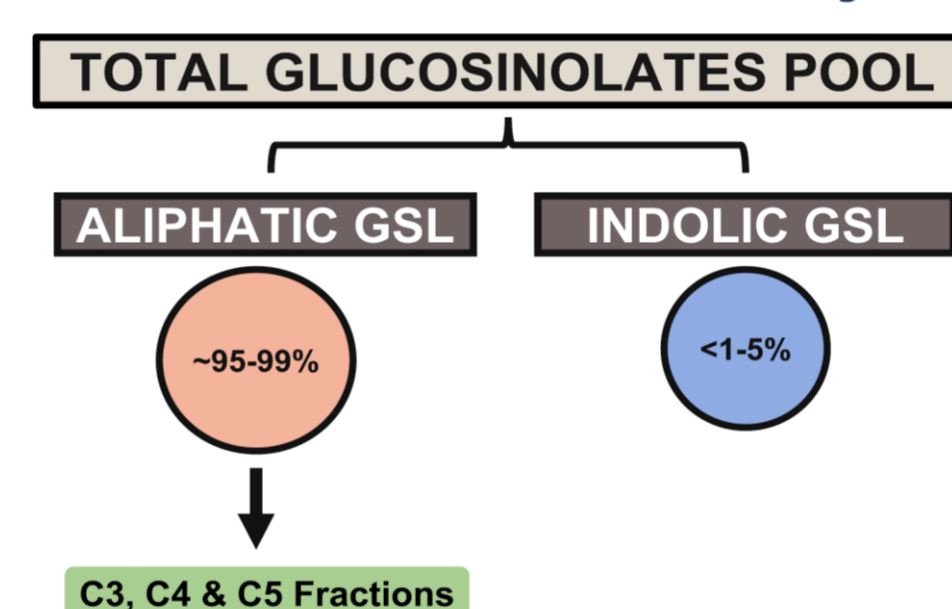
3. High sinigrin content in the genetic background of *B. juncea* shows tolerance against *Sclerotinia*



2. *S. sclerotiorum* infection modulates the levels of in planta glucosinolates in *B. juncea*

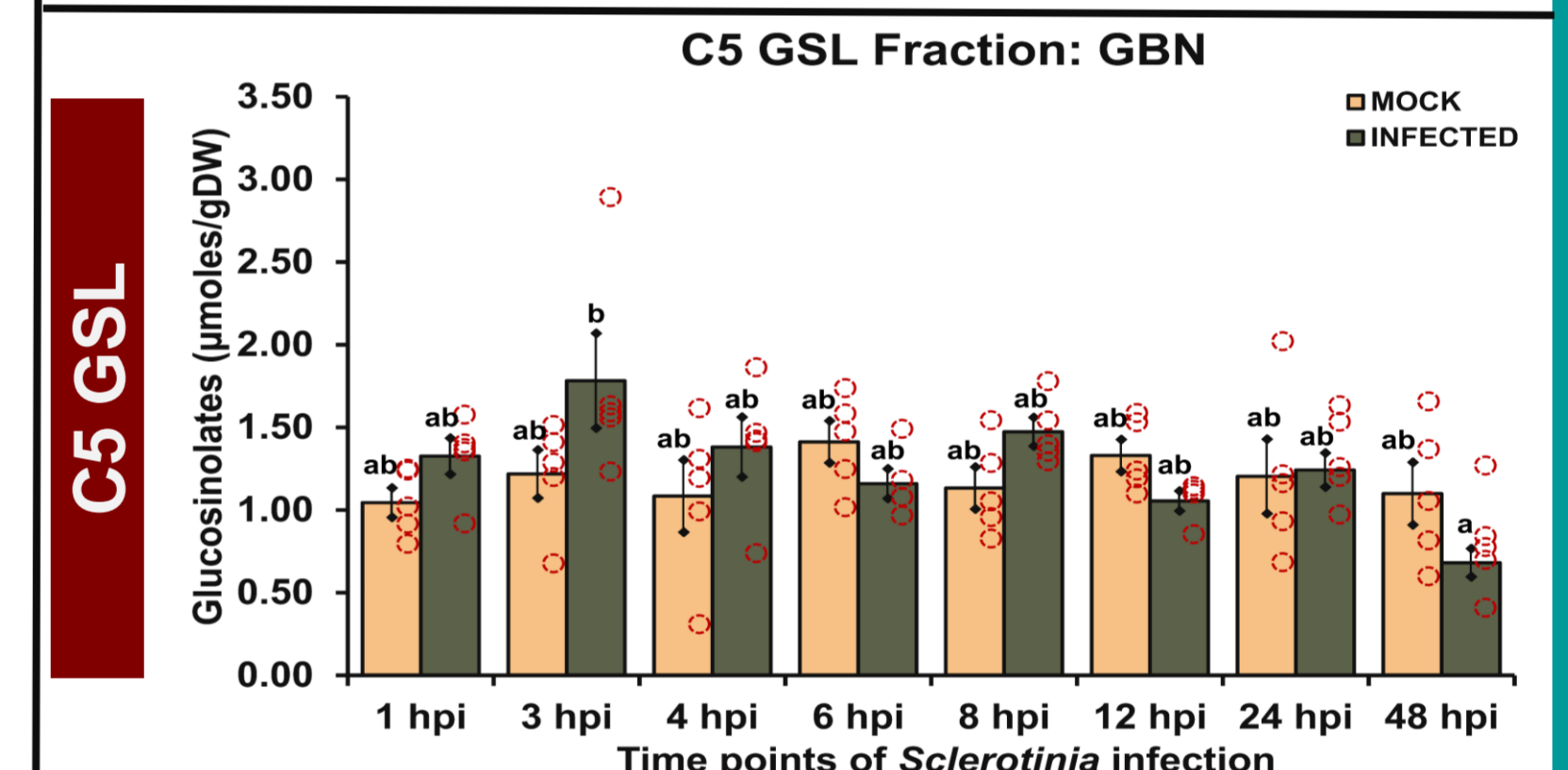
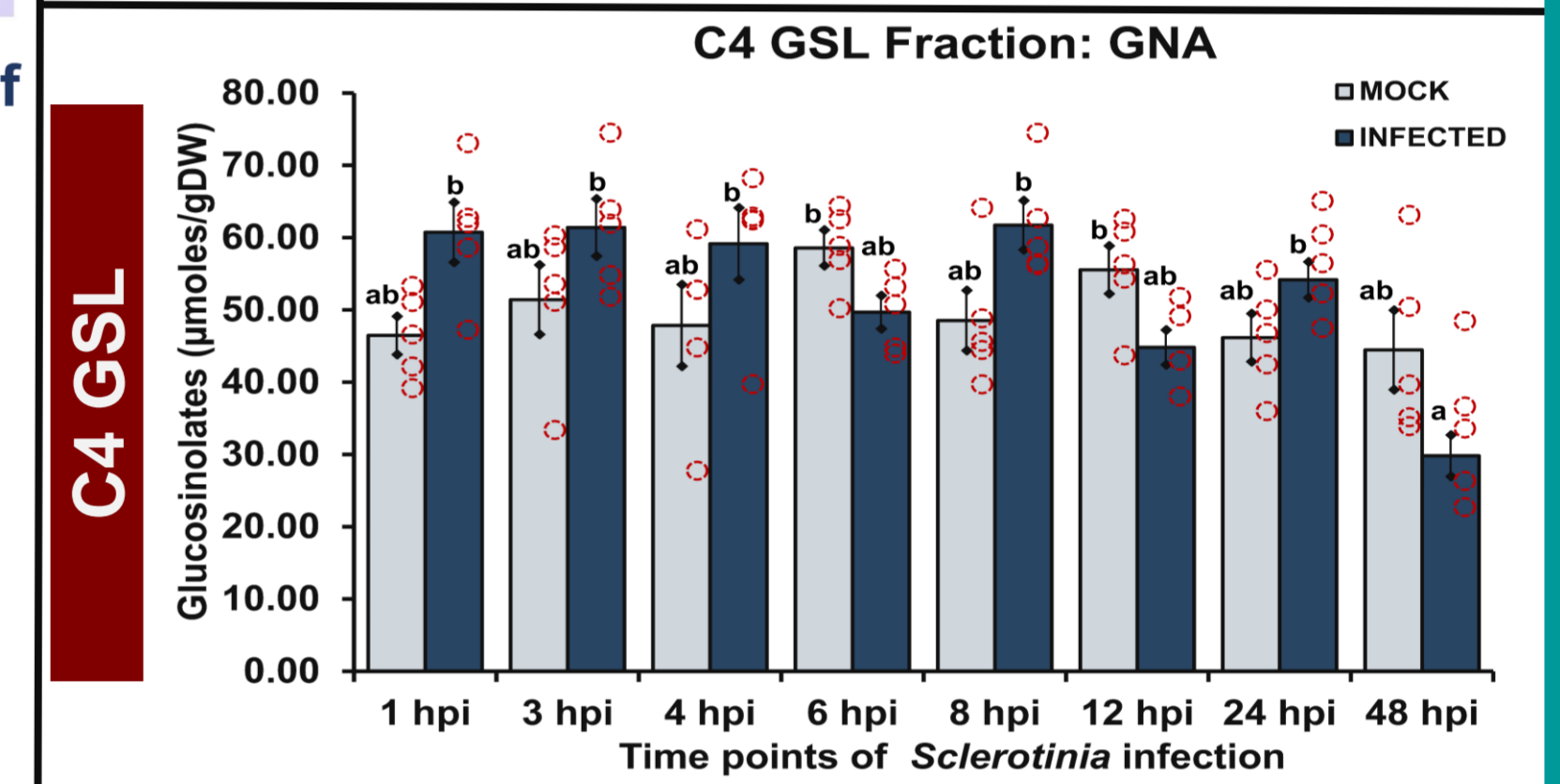
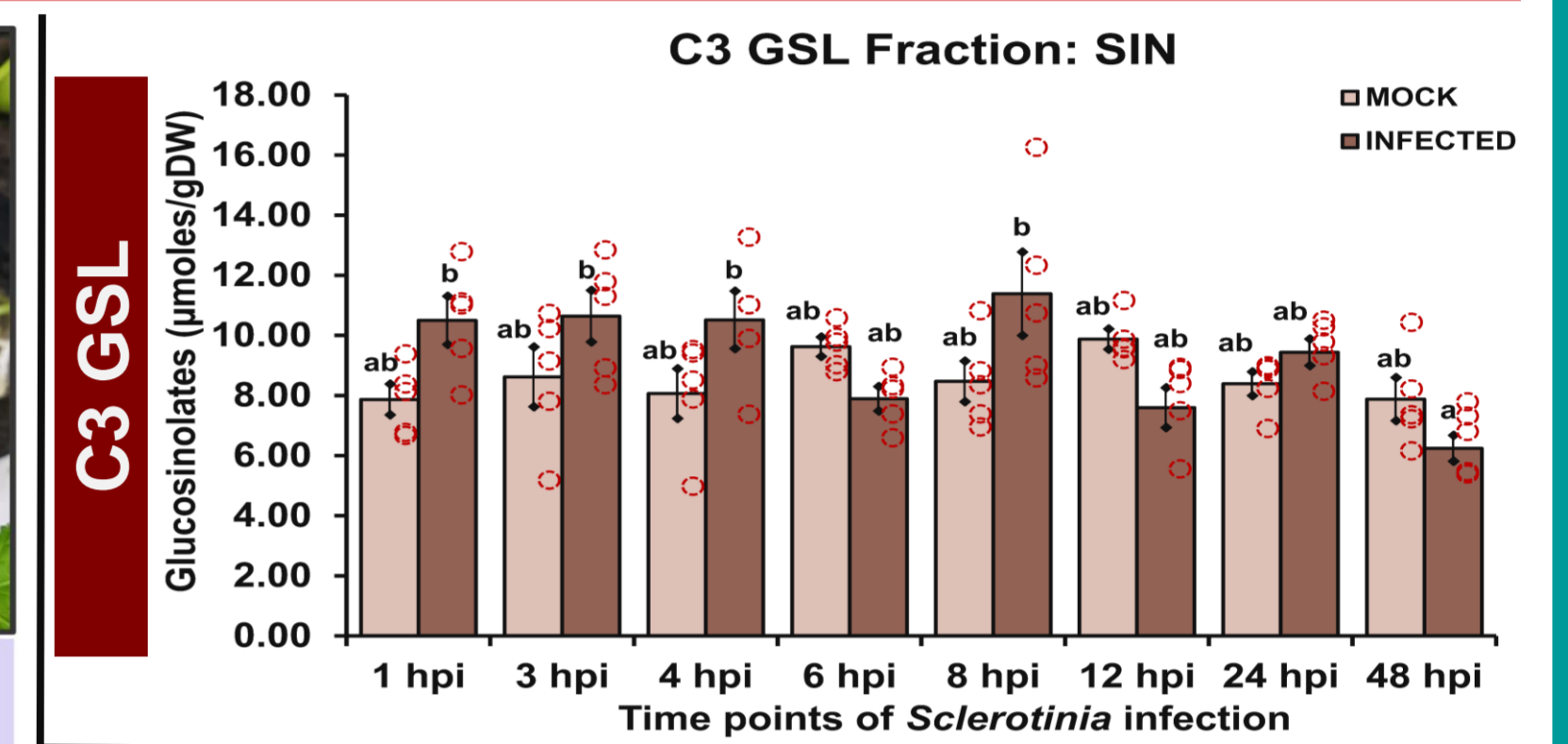


Modulation of GSLs during time course of *S. sclerotiorum* infection in *B. juncea*



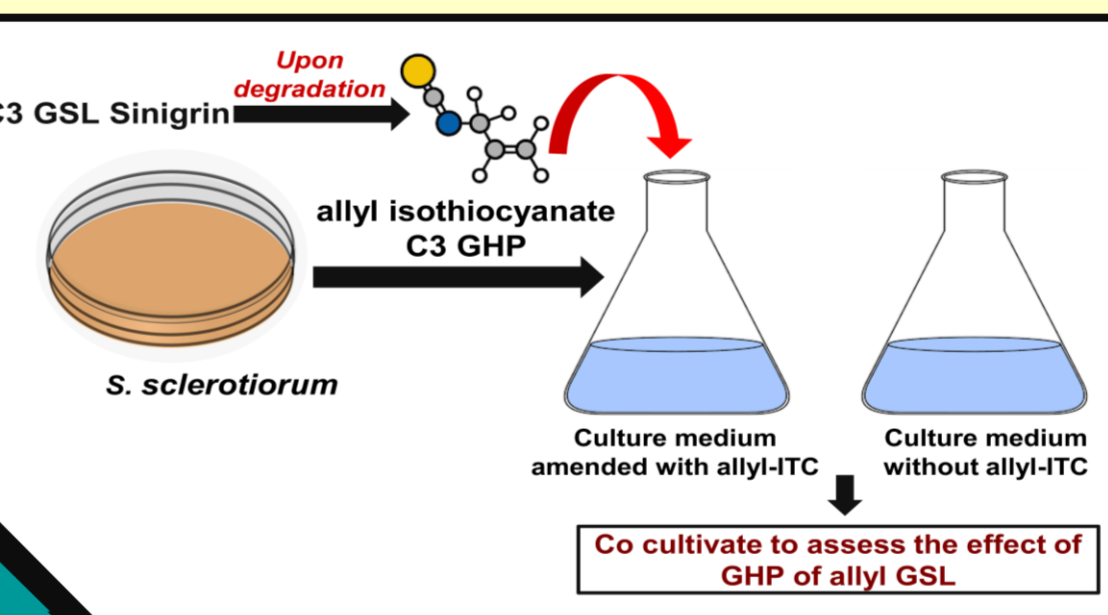
❖ Right panel shows changes in the in planta levels of three major aliphatic GSL fractions (C3, C4 & C5) of *B. juncea* during *S. sclerotiorum* infection.

❖ Our results suggest that *Sclerotinia* infection activates the glucosinolate-myrosinase system by modulating changes in component glucosinolates of *B. juncea* plants throughout the time course of infection.

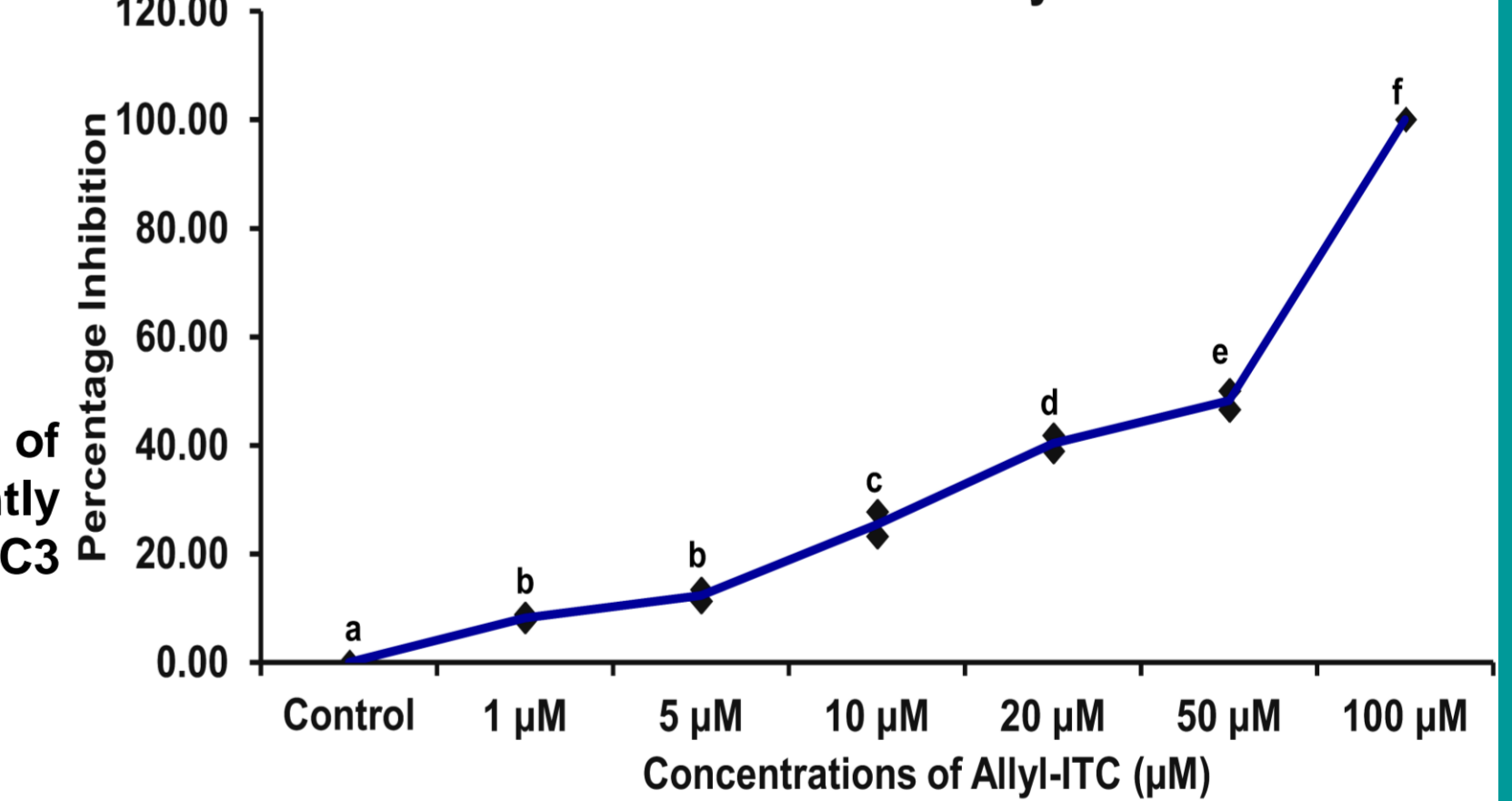


4. *S. sclerotiorum* mycelial growth inhibition in the presence of C3 GSL hydrolysis product

Allyl-ITC's effect on fungal growth was studied as the reduction in the mycelial mass at different concentrations



Percentage Inhibition of Mycelial Mass in the medium amended with Allyl-ITC

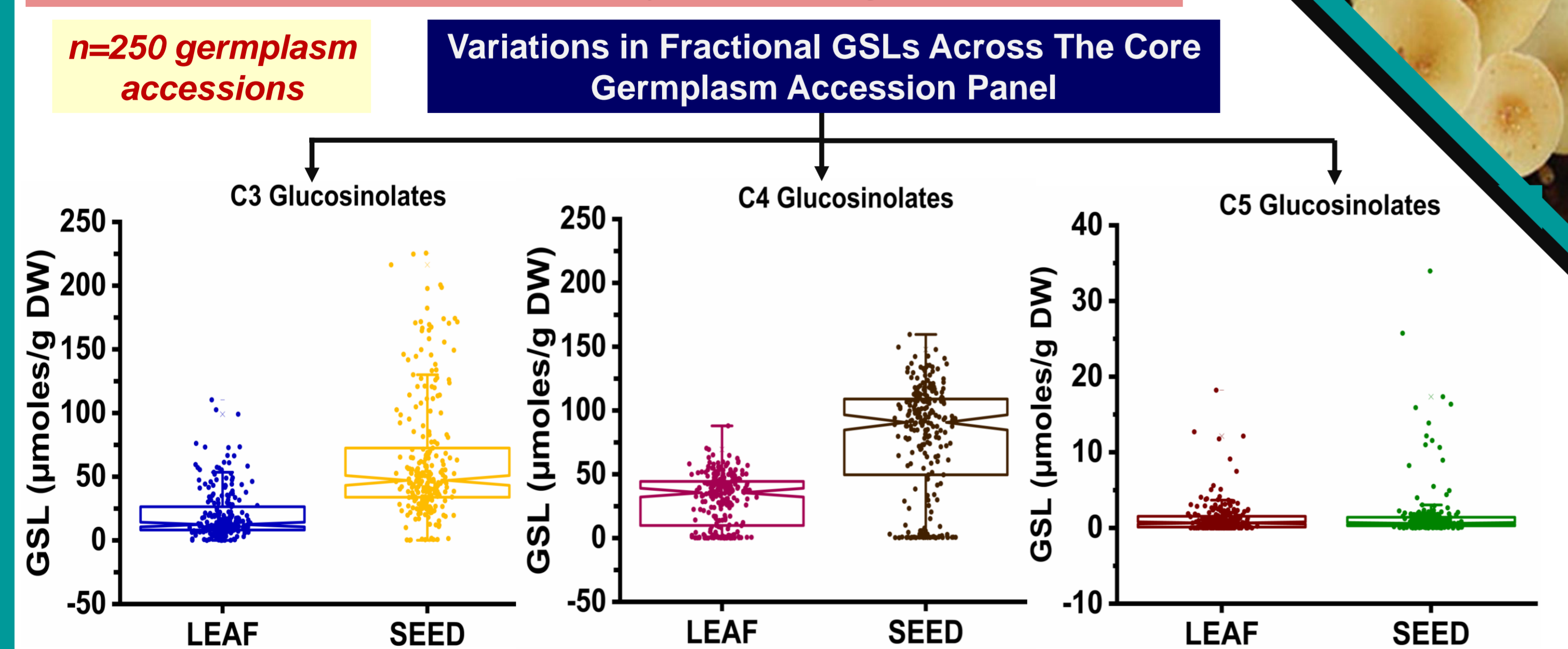


❖ The mycelial growth of *Sclerotinia* was significantly inhibited in presence of C3 GSL's hydrolysis product

THE WHITE-MOLD DISEASE

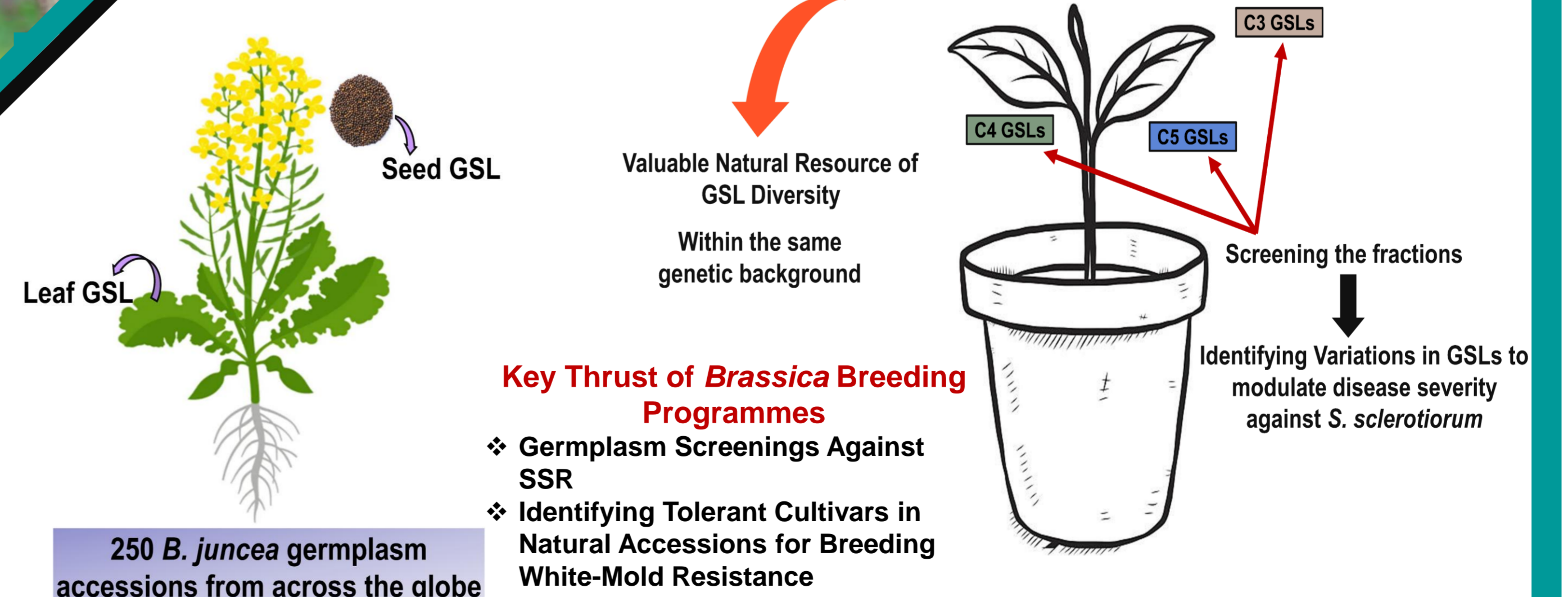
IN OILSEED BRASSICA CROPS

5. GSLs across the *B. juncea* germplasm



Conclusions & Future Perspectives

Germplasm Accessions



References

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Acknowledgements

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