AgriCos e-Newsletter
ISSN: 2582-7049
Volume: 02 Issue: 08 August 2021 Article No: 30

SWEET Genes: A Source of Disease Resistance in Plants

B. K. Namriboi and Bhagyashree Bhatt
Ph.D Scholar, Department of Plant Pathology, College of Agriculture, GBPUAT, Pantnagar, U. S. Nagar, Uttarakhand

SUMMARY

Sugars Will Eventually be Exported Transporters (SWEET) genes encode for diverse physiological functions such as pollen nutrition, nectar secretion, seed filling, phloem loading, and pathogen nutrition. These genes are hijacked by pathogens to co-opt sugar supply of host plant for themselves leading to successful infection of the host plant. Diseases that use TAL effectors are excellent candidates for reducing yield loss by engineering resistant plant lines, because TAL effector binding depends on highly conserved and short cognate EBE sequences present in host S-gene promoters. *SWEET* genes also act as susceptible genes. Therefore, manipulation of SWEET genes using genomic editing technologies such as TALENs and CRISPR-Cas9 can be deploy for creating resistance cultivars without yield penalty.

INTRODUCTION

Plants and pathogens engage in an evolutionary tug-of-war since the beginning of their existence. In order to get the upper hand plant limits pathogen access to nutrients and initiates immune responses while pathogen evolves adaptive strategies to gain access to nutrients and suppress host immunity. Successful pathogens divert nutrient efflux mechanisms of the host to redirect nutrient flow towards them for nutritional gain. Sugars are the main carbon and energy source plants and animals. Phytopathogenic bacteria in the genera *Pseudomonas* and *Xanthomonas* can live in the intercellular space (apoplasm) of plants, where they acquire carbohydrates for energy and carbon. Pathogens induce the expression of different *SWEET* genes, indicating that the sugar efflux function of SWEET transporters is targeted by pathogens for successful invasion.

Sugar transporters

In plants, the sugars synthesized during photosynthesis are transported to sink organs through phloem by various families of sugar transporters. Sugar transporters were categorized into three distinct types:

1. MSTs (monosaccharide transporters),
2. SUTs (sucrose transporters)
3. Sugar Will Eventually be Exported Transporter (SWEET) proteins

MSTs and SUTs function in sugar influx whereas SWEETs primarily function in sucrose efflux. SWEET proteins play role in adaptation to abiotic and biotic stresses as well as host-pathogen interactions.

Sweet Gene

Sugars Will Eventually be Exported Transporters (SWEET) proteins were first identified in plants as the novel family of sugar transporters which mediates the translocation of sugars across cell membranes. *SWEET* genes were first found as nodulins (genes induced during nodulation) and named MtN3 (*Medicago truncatula* nodulin number 3). Later, a homolog of the *MtN3* gene was found in Drosophila. This homolog is expressed in embryonic salivary glands and was named *saliva*. They encode membrane-localized uniporters that transport sugars across cell membranes down a concentration gradient.

Various numbers of *SWEET* genes have been identified in plants; 17 in *Arabidopsis thaliana*, 21 in *Oryza sativa*, 23 in *Sorghum bicolour*, 52 in *Glycine max*, 35 in *Solanum tuberosum*, 29 in *Solanum lycopersicum*, 33 in *Malus domestica*, 17 in *Vitis vinifera*, 59 in *Triticum aestivum* and 22, 31, 55 and 60 in *Gossypium arboreum*, *G. raimondii*, *G.hirsutum* and *G. barbadense*, respectively.

Physiological Role of Sweet Proteins

Various sugar transporters were identified in bacteria, fungi, plants and humans, which perform an important role in development, metabolism, growth, and homeostasis (Chen *et al*., 2010). Family of SWEET sugar transporters in plants perform crucial roles in various developmental processes such as pollen nutrition, nectar

secretion, seed filling, phloem loading function for distance translocation of sucrose, and regulating gibberellin response.

Potential Role of Sweets in Pathogen Nutrition

- *Xanthomonas oryzae pv. oryzae* (Xoo) strain PXO99 A can produce the transcription activator-like (TAL) effector, PthXo1, which binds directly to the *OsSWEET11* promoter. The TAL effectors are delivered to the cytoplasm of plant cells through the type III secretion system and enter the nucleus to induce the expression of specific *SWEET* genes, ensuring the delivery of sucrose to the apoplasts of the colonized cells (Chen *et al.*, 2010).
- *Golovinomyces cichoracearum* infection induces several *AtSWEETs*, most prominently *AtSWEET12* (Chen *et al*., 2010).
- *OsSWEET11/Xa13*, *OsSWEET13/Xa25* and *OsSWEET14* have been identified as targets of Xoo effectors in rice (Hutin *et al.*, 2015).
- The transcript level of *OsSWEET12* was enhanced during the infection of *Xoo* strain transformed with dTALEs. These dTALEs were executed to bind with the putative TATA boxes in the promoter region of *OsSWEET12* which provides susceptibility to *Xoo* strain.
- Pathogen *Xanthomonas axonopodis* pv. *manihotis* TAL effector (TAL20) target *MeSWEET10a* promoter, which is a clade III hexose and sucrose transporter in cassava thereby enhancing the accumulation of sugars (Cohn *et al.*, 2014).
- The TAL effector molecule (Avrb6, a TAL effector determining *Xanthomonas campestris* pv. *malvacearum* pathogenicity) induce the expression of GhSWEET10 confers susceptibility to bacterial blight of cotton.
- The susceptible *Plasmodiophora brassicae* infection triggers the expression of specific *BrSWEET* genes which further enhance cellular glucose and fructose uptake in *Brassica rapa* (Li *et al*., 2018) indicating that *SWEET* genes also act as susceptible genes.

Modification of *Sweet* Gene For Disease Resistance

- Mutations involving the EBE (effector binding element) reduce or eliminate effector binding, prevent *SWEET* gene induction. A single substitution in the second nucleotide of the EBE is sufficient to avoid induction of *OsSWEET11* by PthXo1.
- TALENs-mediated deletions directed at *OsSWEET14* and, CRISPR-Cas9-mediated mutations at *OsSWEET13* have produced plants that are resistant to strains of *Xoo*.
- The recessive resistance gene *b6* in cotton has been associated with alterations of the *GhSWEET10* promoter, which is targeted by the TAL effector Avrb6 of *Xanthomonas citri* subsp. *malvacearum*, the causal agent of cotton blight (Cox *et al.*, 2017).
- The EBEs of AvrXa7 and PthXo3 in the OsSWEET14 promoter were precisely edited by TALENs, which prevents the induction by TALEs. The mutated lines showed strong resistance to both AvrXa7- and PthXo3-dependent *Xoo* strains.
- Knock-out of *OsSWEET11* showed increased resistance to *Rhizoctonia solani*, which causes sheath blight disease (Gao *et al*., 2018).
- The promoter of *Xa13* (*OsSWEET11*) was targeted by CRISPR/Cas9-based disruption, leading to enhanced resistance without affecting rice fertility (Li *et al*., 2019).
- EBEs in the promoters of *OsSWEET11*, *OsSWEET13* and *OsSWEET14* were edited simultaneously by CRISPR/Cas9 technology and rice lines conferring broad-spectrum resistance to *Xoo* were created (Xu *et al.*, 2019).

CONCLUSION

Utilizing modified SWEET *genes* to develop specific disease resistant crop cultivars with the help of genome editing tools can be a game changer in plant disease management without affecting the yield. However, there is need to identify and analyze functions of *SWEET* genes in more crops which may provide more candidate

genes for genetic modification. Moreover, the combination of different types of resistance mechanisms needs to be further elucidated and advances in this field will rely heavily on collaborations between plant pathologists and physiologists.

REFERENCES

Chen, L. Q., Hou, B. H., Lalonde, S.,Takanaga, H., Hartung, M. L., Qu, X. Q., et al. (2010) Sugar transporters for intercellular exchange and nutrition of pathogens. Nature:468: 527–532.doi:10.1038/nature09606

Cox, K.L., Meng, F., Wilkins, K.E., Li, F., Wang, P., Booher, N.J. et al. (2017) TAL effector driven induction of a SWEET gene confers susceptibility to bacterial blight of cotton. Nature Communications: 8 (15588)

Gao, Y., Zhang, C., Han, X., Wang, Z.Y., Ma, L., Yuan, D.P. et al. (2018) Inhibition of OsSWEET11 function in mesophyll cells improves resistance of rice to sheath blight disease. Molecular Plant Pathology: 19: 2149– 2161

Hutin, M., Perez-Quintero, A.L., Lopez, C., and Szurek, B. (2015) MorTAL kombat: the story of defense against TAL effectors through lossof-susceptibility. Frontiers in Plant Science: 6:535

Li, C., Li, W., Zhou, Z., Chen, H., Xie, C., and Lin, Y. (2019) A new rice breeding method: CRISPR/Cas9 system editing of the Xa13 promoter to cultivate transgene-free bacterial blight-resistant rice. Plant Biotechnology Journal: 10.1111/pbi.13217

Li, H., Li, X., Xuan, Y., Jiang, J., Wei, Y., and Piao, Z. (2018) Genome wide identification and expression profiling of SWEET genes family reveals its role during *Plasmodiophora brassicae*- induced formation of clubroot in *Brassica rapa*. Frontiers in Plant Science: 9 (207)

Xu, Z., Xu, X., Gong, Q., Li, Z., Li, Y., Wang, S. et al. (2019) Engineering broad- spectrum bacterial blight resistance by simultaneously disrupting variable TALE- binding elements of multiple susceptibility genes in rice. Molecular Plant: 12: 1434-1446