Plant Biotechnology Journal
Open Access

Plant Biotechnology Journal (2024), pp. 1–15 doi: 10.1111/pbi.14408

Review Article

The role of sugar transporters in the battle for carbon between plants and pathogens

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Received 24 December 2023;
revised 3 May 2024;
accepted 27 May 2024.
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Keywords: sugar transporter, carbon nutrients, plant-pathogen interaction, SWEET, STP, SUT.

Summary

In photosynthetic cells, plants convert carbon dioxide to sugars that can be moved between cellular compartments by transporters before being subsequently metabolized to support plant growth and development. Most pathogens cannot synthesize sugars directly but have evolved mechanisms to obtain plant-derived sugars as C resource for successful infection and colonization. The availability of sugars to pathogens can determine resistance or susceptibility. Here, we summarize current progress on the roles of sugar transporters in plant–pathogen interactions. We highlight how transporters are manipulated antagonistically by both host and pathogens in competing for sugars. We examine the potential application of this target in resistance breeding and discuss opportunities and challenges for the future.

Introduction

As autotrophs, plants generate sugars in leaves by photosynthesis and assimilation. These sugars are transported, metabolized and stored in suitable forms for plant growth and development. In contrast, pathogens as heterotrophs must obtain sugars from host plants to grow and establish a successful infection.

There are two roles for host-derived sugars in plant–pathogen interactions (Bezrutczyk *et al.*, 2018). Host sugars serve as nutrients, feeding the pathogen (Chen *et al.*, 2010) and as signals that can regulate the infection process (Herbers *et al.*, 1996). The two roles of sugars are not mutually exclusive, it is likely that some sugars play a dual role as both signals and nutrients (Liu *et al.*, 2013; Schuler *et al.*, 2015).

Sugars are transported via both intercellular (symplastic) and extracellular (apoplastic) trafficking pathways in plants. Sugars are also exported or imported across the plasma membrane by transporter proteins. Due to the limited carbon resources in host plants, pathogens must compete with plants for nutrients. Therefore, both pathways and transporters are potential targets, manipulated and exploited by the host and pathogen antagonistically in competing for sugars. In this review, we focus on how pathogens manipulate sugar transporters, thus affecting the redistribution of host-derived carbon to support their infection. For sugars as signalling roles in plant–pathogen interaction, we refer readers to previous reviews (Bezrutczyk *et al.*, 2018; Li *et al.*, 2021; Morkunas and Ratajczak, 2014) and will not include this topic here. We also describe some potential applications of this knowledge and explore some open questions and opportunities, for example starvation-mediated resistance breeding as potential new methods with durable resistance.

Transport of sugars in plants during infection

As an obligate parasite, a plant pathogen must obtain carbon from host plants to establish a successful infection at the early stage of invasion. The strategies by which pathogens obtain nutrients from host depend on the types, lifestyle and infection stage of the pathogens (Kanwar and Jha, 2019). The trafficking pathways by which nutrients are transported to infection sites are divided into two routes: apoplastic and symplastic pathways (Figure 1).

Sugar transport from the host to the infection site in plants

Plant cells are connected by plasmodesmata (PD) into a single 'organism' named as symplast. This cytoplasmic and membrane continuity allows for communication and coordination between cells, a prerequisite for multicellularity (Faulkner *et al.*, 2005). The space outside the symplast is known as the apoplast, it includes the cell wall and the aqueous intercellular space (Erickson, 1986). Cell walls are composed of cross-linked polysaccharides with pores, ranging from 5 to 20 nm in size (Cunningham *et al.*, 2018; Wang *et al.*, 2016), allowing solutes to move freely in the apoplast.

When pathogens invade plant tissue, distal nutrients can move to the infect site through both pathways. In the apoplastic pathway, distal site nutrients are released from mesophyll cells into the apoplast and then diffuse to the infection sites (Figure 1). The movement of sugars is driven by concentration gradients.

Please cite this article as: Chen, Y., Miller, A.J., Qiu, B., Huang, Y., Zhang, K., Fan, G. and Liu, X. (2024) The role of sugar transporters in the battle for carbon between plants and pathogens. *Plant Biotechnol. J.*, https://doi.org/10.1111/pbi.14408.

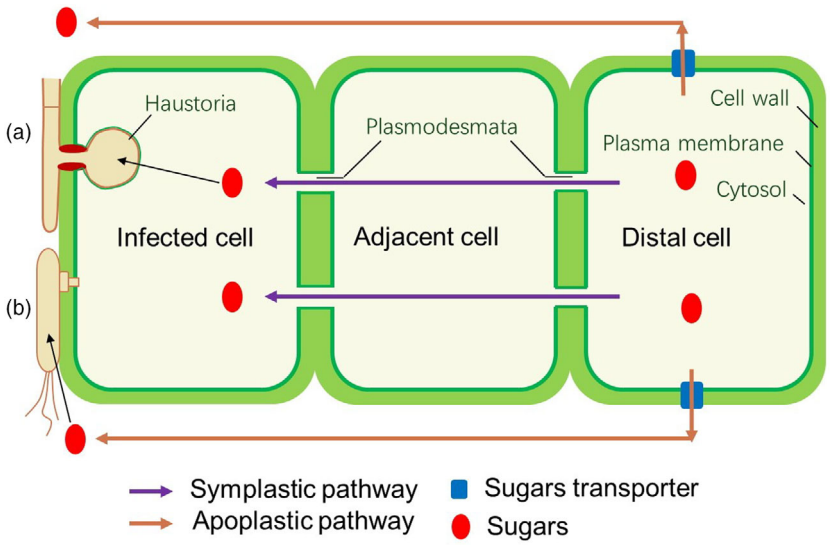

Figure 1 Schematic illustration of two sugars trafficking pathways in plants in plant–pathogen interactions. (a) Haustorial-forming fungal pathogens; (b) Bacterial pathogens.

In the symplastic pathway (Figure 1), nutrients are transported locally, moving cell-to-cell through PD. In this pathway, the sugar concentration is higher in the distal cells than that in the infected cell. Therefore, sugars can diffuse to the infected cell through PD following the concentration gradient, revealing that the PDs are key elements in this pathway (Miras *et al.*, 2022). Reversible callose deposition at PD can determine the diameter of the cytoplasmic sleeve, controlling molecular flux through PD. A detailed review focused on PD and its role in assimilate translocation has been published (Miras *et al.*, 2022).

Sugar transport from the infection site to pathogen in plants

Sugars in the apoplast and symplast cannot exchange freely across the plasma membrane (PM) and this obstacle can provide a selective barrier. Sugars are transported by integral membrane proteins with a range of transmembrane domains or transmembrane helices (Schulz, 2011). These transporter proteins facilitate the movement of sugars across the membrane barrier. Some sugar transporters have been thought to have a dual function as both sugar transporters and sugar sensors in plants (Lalonde *et al.*, 1999), similar to those in yeast (Rolland *et al.*, 2006), but this hypothesis has not been verified experimentally yet in plants. Invertase and hexokinase activities are believed to be a key components of sugar sensing (Moore *et al.*, 2003; Ruan *et al.*, 2010). Plants can regulate the distribution of nutrients by transporter activity in response to internal and external stimuli. This regulation can be by both changes in transcript and post-translational regulatory mechanisms (Devanna *et al.*, 2021). In the context of infection, transporters are key elements that are manipulated and exploited antagonistically by both pathogens and host plants for nutrient distribution.

How pathogens extract nutrients from host plants depends on the pathogen type, lifestyle and infection stage. In general, bacteria absorb nutrients in the apoplast of plant cells (Liu *et al.*, 2022), whereas biotrophs and biotrophic phase of hemitropic fungi/oomycete obtain nutrients mainly from the symplast (Voegele *et al.*, 2001). However, necrotrophic fungi/oomycete or hemitrophic fungi/oomycete in necrotrophic phases take up nutrients mainly from the apoplast (Liu *et al.*, 2022). In bacterial pathogen infections (Figure 1), bacteria take up nutrients and grow in the apoplast directly after invasion, and host nutrients in the cytoplasm can also be exported into the apoplast by transporters (Yamada *et al.*, 2016). In biotrophic fungal pathogen infections (Figure 1), fungi often form structures in host cells called haustoria that acts as sites of nutrient uptake (Voegele *et al.*, 2001). Apoplastic sugars in infected sites can be transferred into the cells across the PM by transporters to supply the fungal haustoria (Chen, 2014). Thus sugars from both pathways can be obtained by pathogenic fungi and bacteria with the assistance of sugar transporters, revealing that transporters play key roles in regulating sugar redistribution to supply for pathogens.

Sugar transporters and their functions

Sugars from the host plant are transported across membranes by a range of sugars transporter proteins. For example (see Figure 2), sucrose can be exported into the apoplast by Sugars Will Eventually Be Exported Transporters (SWEETs) (Chen, 2014; Lin *et al.*, 2014) and be imported back into cytoplasm by Sucrose Transporters (SUTs) (Gottwald *et al.*, 2000). Sucrose outside cell can also be hydrolysed by cell wall invertases (Inv-CW) into hexose: glucose and fructose (Ruan, 2014). These hexoses can be taken up by Sugar Transporter Proteins (STPs) back into cytoplasm (Buttner, 2010). While in the cytoplasm, sucrose can be hydrolysed by neutral invertase (Inv-N) into hexose: glucose and fructose (Ruan, 2014), or by sucrose synthase (SUS) to produce fructose and UDP–glucose (Stein and Granot, 2019). The vacuolar membrane, the tonoplast, is also involved in transporting and distributing sugars. Sucrose in the cytoplasm can be transported by tonoplast membrane-localized tonoplast sugars transporter (TST) into the vacuole as stored sugars (Schulz *et al.*, 2011). Sucrose in vacuole, on one hand, can be efflux by SUT4 into cytoplasm (Schulz *et al.*, 2011); or can be hydrolysed by vacuolar invertase (Inv-v) into monosaccharides (Roitsch and Gonzalez, 2004), then be efflux by early response to dehydration 6 (ERD6) into the cytoplasm (Buttner, 2007). In cytoplasm, monosaccharide can be imported by tonoplast monosaccharide

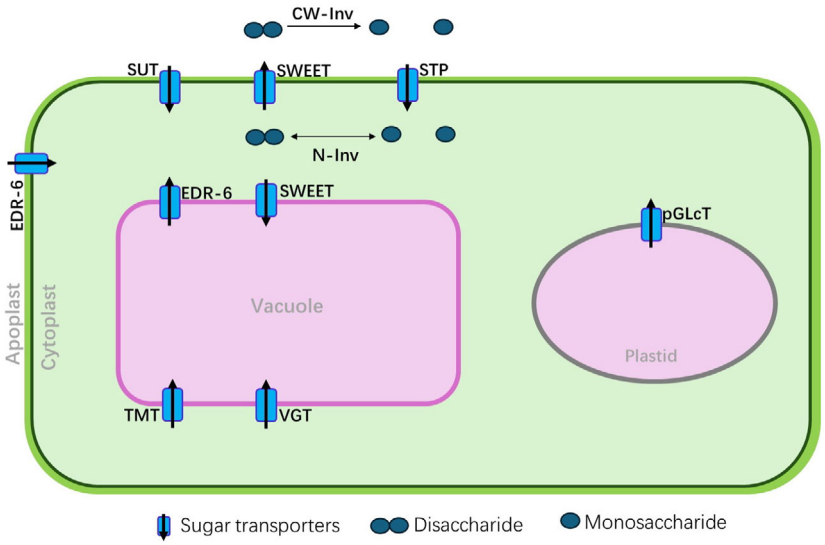

Figure 2 Plant transporters and their functions. Major classes of sugar transporters and their cellular location in the plant are shown in the figure. The direction of the arrow depicts the export or import of sugars from or into the organelle, respectively. ERD6, early response to dehydration 6; CWInv, cell wall invertase; N-Inv, neutral invertase; pGlcT, plastidic glucose transporter; PMT, polyol/monosaccharide transporter; TMT, tonoplast membrane transporter; VGT, vacuolar glucose transporter.

transporter (TMT) or vacuolar glucose transporter (VGT) (Buttner, 2007). It is suggested that plastidic glucose transporter (pGLcT) is involved in plastidic glucose efflux (Weber *et al*., 2000).

Sugars will eventually be exported transporters (SWEETs)

The SWEET family proteins contain a PQ-loop repeat and belong to the transporter–opsin-G protein-coupled (TOG) receptor superfamily (Medrano-Soto *et al*., 2020). The SWEETs catalyse the facilitated efflux and/or influx of sugars (Chen *et al*., 2010). Based on their subcellular localization and substrate specificity, SWEETs have been classified into four clades (Ji *et al*., 2022; Yao *et al*., 2022). Clade I, II and IV transport monosaccharide and clade III preferentially transports sucrose. Clade IV SWEETs are localized in tonoplast, and the members of the other clades are mainly localized in the PM (Yamada and Osakabe, 2018). SWEETs function as facilitated diffusion transporters, meaning that the direction of sugar transport depends on the substrate concentration gradient (Yamada *et al*., 2010).

The SWEET genes are found in almost all cellular organisms, and they are largely conserved across species (Jia *et al*., 2017). Structural analyses indicate that prokaryotes have ancestral SemiSWEETs with only three transmembrane domains (TMDs). Eukaryotic SWEETs have seven TMDs that most likely evolved by internal duplication of the Semi-SWEET 3-TMDs (Xuan *et al*., 2013). Furthermore, some species evolved by multiple internal duplication to generate extraSWEET and superSWEET that possess 15 and 25 TMDs, respectively (Devanna *et al*., 2021) (Figure 3).

This gene family usually has multiple members in higher plants, including 17 in *Arabidopsis thaliana*, 21 in *Oryza sativa*, 23 in *Sorghum bicolor*, 52 in *Glycine max*, 35 in *Solanum tuberosum*, 29 in *Solanum lycopersicum*, 33 in *Malus domestica* and 17 in *Vitis vinifera* (Miao *et al*., 2017).

Phylogenetic analysis of SWEET transporter proteins from bacteria, fungi, oomycote and green plants show that bacterial SWEETs, fungal SWEETs and green plant SWEETs are grouped into three independent clades whereas bacteria SWEETs display a distribution pattern with higher diversity. The bacteria have one branch closely related to oomycete, while another branch evolves independently. Of three independent clades, phylogenetic distance between oomycete and fungi is shorter than green plant (Figure 4).

Plant SWEETs have broad substrates spectrum such as glucose, fructose and sucrose. Substrate of fungal SWEETs are diverse including glucose, fructose and mannose. Bacteria has Semi-SWEETs which can transport glucose and sucrose (Table 1).

Sugar transporter proteins (STPs)

The STP proteins belong to a family of hexose transporters (or Monosaccharide transporters-MSTs), found in both prokaryotes and eukaryotes. The proteins of this family normally have 12 TMDs. Their amino acid sequences are highly conserved among homologous families from algae and protozoa to Mammals (Henderson, 1990). They are H+/sugar symporters and are usually found in the PM of cells (Henderson, 1990; Kong *et al*., 2019). The well-characterized Arabidopsis STPs are all localized in the PM and transport hexoses including galactose, xylose, glucose, fructose and mannose (Rottmann *et al*., 2018). This gene family has also multiple members in higher plant species, including 14 in *Arabidopsis thaliana*, 29 in *Oryza sativa*, 23 in *Sorghum bicolor*, 66 in *Fragaria vesca*, 22 in *Zea mays*, 52 in *Solanum lycopersicum* and 59 in *Vitis vinifera* (Devanna *et al*., 2021).

Phylogenetic analysis of STP transporter proteins from bacteria, fungi, oomycote and green plants show they are grouped into four independent groups (Figure 5).

Plant STPs and fungal STPs have a broad substrate spectrum and glucose is one of main substrates whereas bacterial STP can transport arabinose, xylose and galactose (Table 2).

Sucrose transporters (SUTs)

The SUT (also known as SUC) family of sucrose transporters are disaccharide transporters that are only found in plants and fungi, probably because sucrose is not produced in animals or micro-organisms (Hu *et al*., 2021). In plants, SUTs facilitate the sucrose uptake into companion cells and sieve elements against the concentration gradient (Chen *et al*., 2012). Their transport

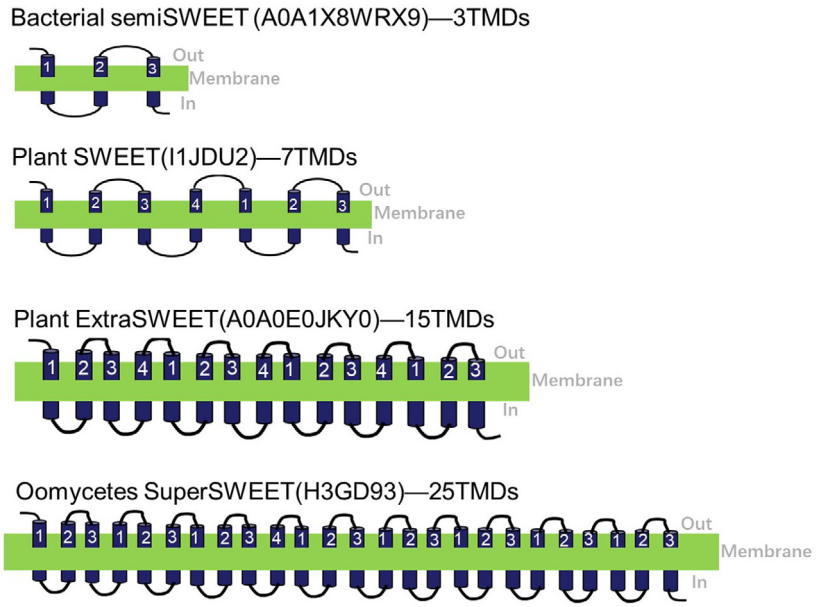

Figure 3 Schematic two-dimensional model of SWEETs transporter from bacteria, plants and oomycetes. Transmembrane helices in proteins are shown as blocks in the figure. Proteins shown as UniProtKB id. TMD, transmembrane domain.

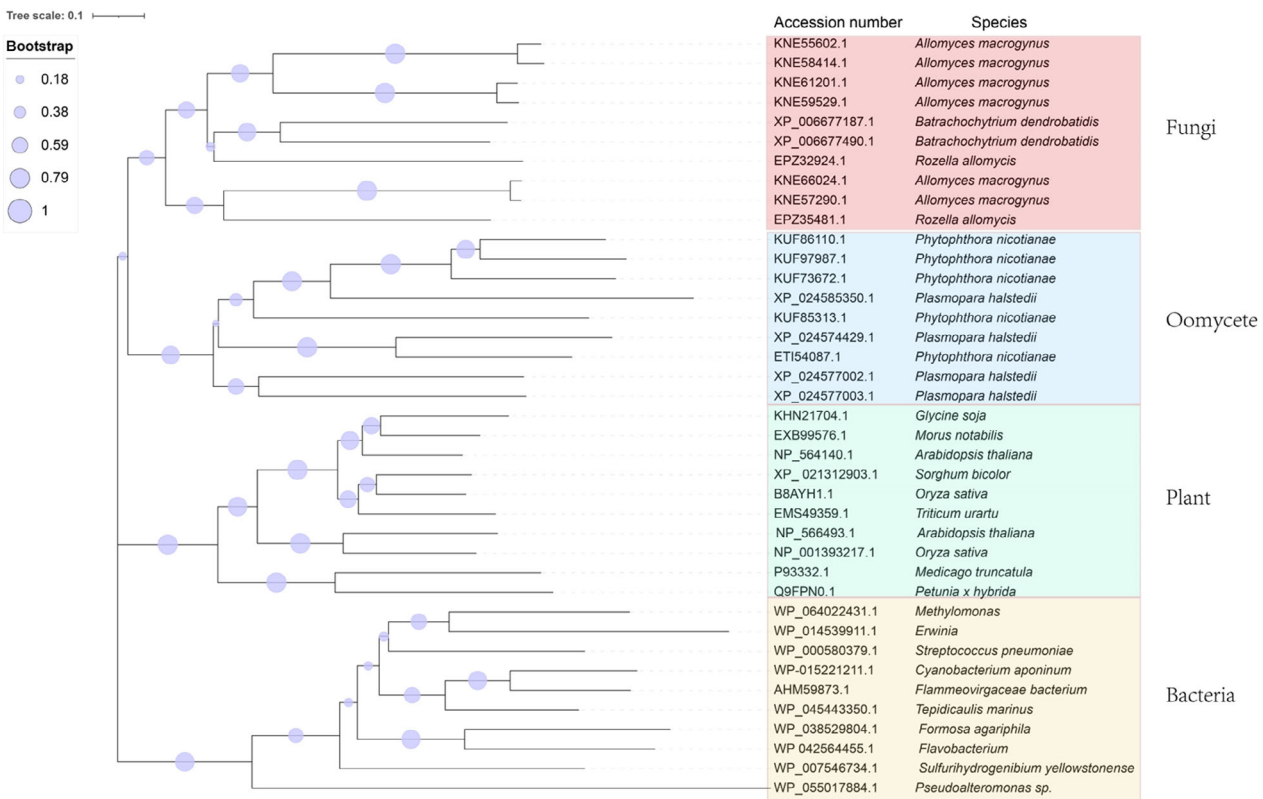

Figure 4 Phylogenetic analysis of SWEET transporter proteins using neighbour-joining (N-J) method with bootstrap values determined by 1000 replicates in MEGA7 (Kumar *et al.*, 2016). The amino acid sequences of SWEET proteins from fungi, bacteria, green plants and oomycota are available at the NCBI database (https://www.ncbi.nlm.nih.gov/protein).

can be bi-directional. SUT1 proteins have been reported to efflux sucrose to the apoplast, whereas sink-specific SUT1 proteins take up sucrose from apoplast into the cells.

SUT has three major classes and their sub-classes include Type-I, Type-II-A, Type-II-B and Type-III (Salvi *et al.*, 2022). Type-I and II SUTs are associated with phloem loading, whereas Type-II-B

Table 1 Example SWEETs from plants, fungi and bacteria and their substrates

	Species	Genes	Substrates	Refs
Plants	*Arabidopsis* *Brassica* *Nicotiana* *Sweet potato*	*SWEET9/10/11/12/13/14/15*	Sucrose	(Chen *et al.*, 2015; Kanno *et al.*, 2016; Li *et al.*, 2017; Lin *et al.*, 2014; Sun *et al.*, 2013)
	Arabidopsis *Vitis vinifera*	*SWEET4/5/8*	Glucose	(Chen *et al.*, 2012; Engel *et al.*, 2005; Sun *et al.*, 2013)
	Arabidopsis *Lotus japonicus*	*SWEET2/3*	2-Dexoyglucose	(Chardon *et al.*, 2013; Sugiyama *et al.*, 2017)
	Arabidospsis	*SWEET17*	Fructose	(Chardon *et al.*, 2013)
Fungi	*Neocallimastigomycota*	*NcSWEET1*	Glucose, fructose and mannose	(Podolsky *et al.*, 2021)
	Batrachochytrium dendrobatidis	*BdSWEET1*	Glucose, fructose	(Hu *et al.*, 2016)
Bacteria	*Bradyrhizobium japonicum*	*BjSemiSWEET1*	Sucrose	(Lee *et al.*, 2015; Xuan *et al.*, 2013)
	Escherichia coli	*EcSemiSWEET*		
	Leptospira biflexa	*Lb SemiSWEET*	Glucose	(Xu *et al.*, 2014)

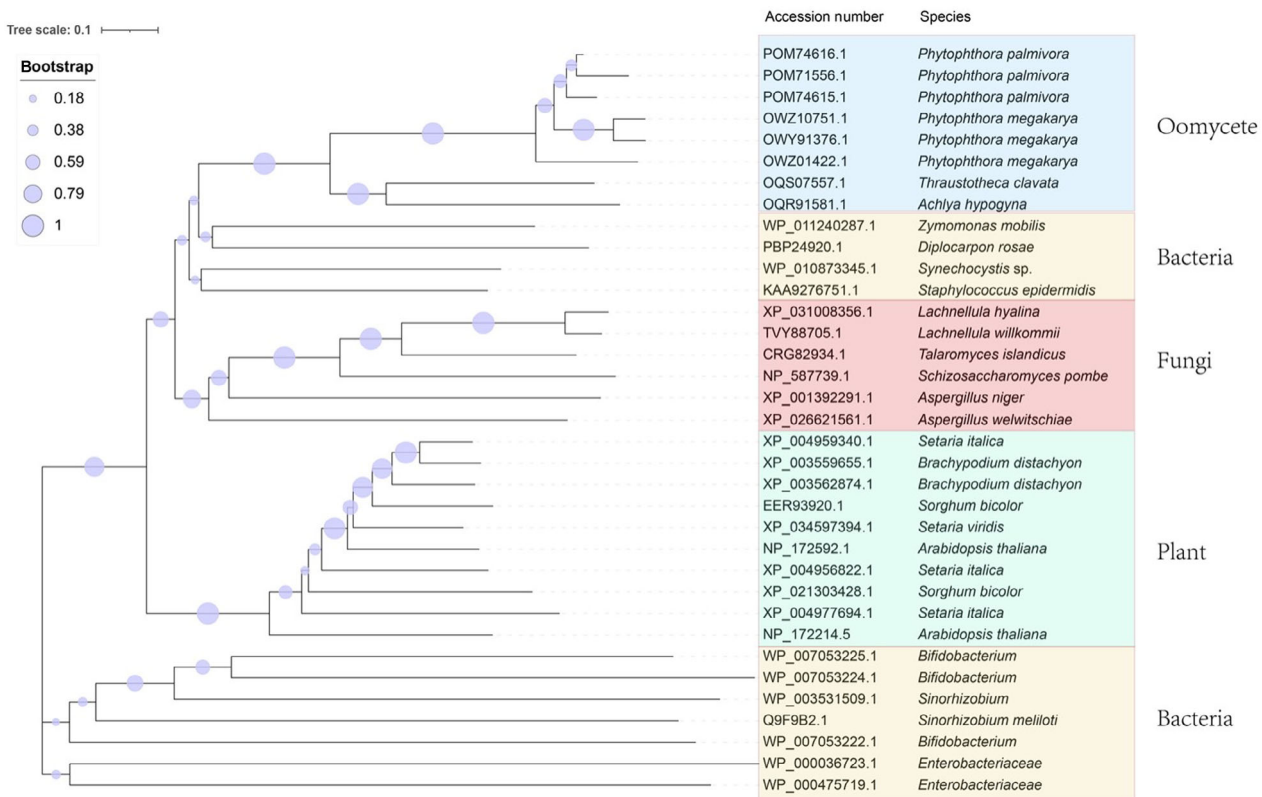

Figure 5 Phylogenetic analysis of STP transporter proteins using neighbour-joining (N-J) method with bootstrap values determined by 1000 replicates in MEGA7. The amino acid sequences of STP proteins from fungi, bacteria, green plants and oomycota are available at the NCBI database (https://www.ncbi.nlm.nih.gov/protein).

functions in phloem unloading and importing the sucrose into sink tissue (Slewinski *et al.*, 2009). All Type-III SUTs are localized in the PM except for a few in the tonoplast. The Type-III tonoplast-located SUTs are reported to be involved in sucrose storage and in modulating cytosolic sucrose concentration (Endler *et al.*, 2006). The PM-localized Type-III SUTs are found to take part in signalling (Endler *et al.*, 2006). Rice has five *SUT* genes. OsSUT2 is tonoplast-localized and the other four are PM-localized (Aoki *et al.*, 2003; Wu *et al.*, 2018). There are nine *SUT* genes in Arabidopsis (Sivitz *et al.*, 2007), 11 in tobacco (Wang *et al.*, 2019) and 7 in maize (Leach *et al.*, 2017).

Phylogenetic analysis of SUT transporter proteins from fungi and green plants show that plant SUTs can generate an independent clade but fungal SUTs show a diverse distribution pattern (Figure 6). Both plant and fungi SUTs have wide range of substrates, but mainly sucrose (Table 3).

Table 2 Example STP transporters of plants bacteria and fungi and their substrates

	Species	Genes	Substrates	Refs
Plants	*Arabidopsis*	*AtSTP2/4/11*	Glucose, galactose, mannose and xylose	(Buttner, 2010; Schneidereit *et al.*, 2005)
	Arabidopsis	*AtVGT1/2*	Glucose	(Aluri and Büttner, 2007)
	Oryza sativa	*OsTMT1*	Glucose	(Cho *et al.*, 2010)
	Oryza sativa	*OsMST1*	Glucose, fructose, mannose and galactose	(Wang *et al.*, 2007)
	Oryza sativa	*OsMST6*	Broad-spectrum monosaccharide	(Wang *et al.*, 2008)
	Arabidopsis	*AtINT1*	Inositol	(Schneider *et al.*, 2008)
	Arabidopsis	*AtSTP14*	Galactose	(Poschet *et al.*, 2010)
	Arabidopsis	*AtSTP7*	L-arabinose and D-xylose	(Rottmann *et al.*, 2018)
Bacteria	*Cyanobacterium Synechocystis*	*GlcP*	Fructose, Glucose	(Zhang *et al.*, 1989)
	E.coli	*Arabinose transporter*	Arabinose	(Maiden *et al.*, 1987)
	E.coli	*Xylose transporter*	Xylose	(Maiden *et al.*, 1987)
	E.coli	*TMG1*	Galactose	(Rotman *et al.*, 1968)
Fungi	*Saccharonmyces cerevisiae*	*SNF3*	Glucose	(Celenza *et al.*, 1988)
	Saccharomyces cerevisiae	*HXT*	Glucose	(Reifenberger *et al.*, 1995)
	Geosiphon pyriformis	*GpMST1*	Glucose, mannose, galactose and fructose	(Schüssler *et al.*, 2006)

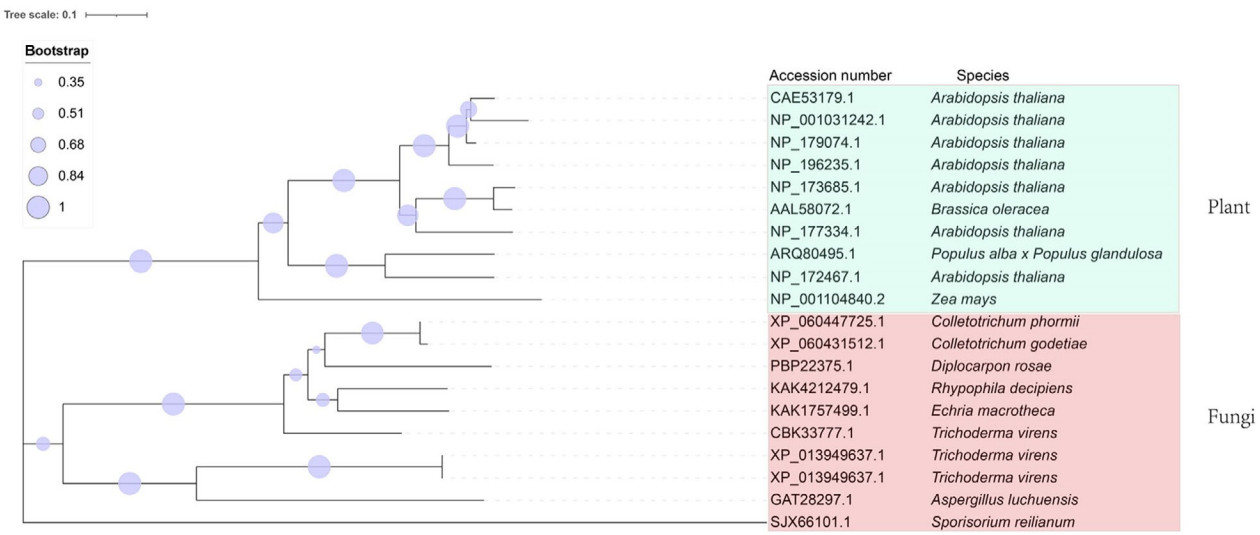

Figure 6 Phylogenetic analysis of SUT transporter proteins using neighbour-joining (N-J) method with bootstrap values determined by 1000 replicates in MEGA7. The amino acid sequences of SWEET proteins from fungi, green plants are available at the NCBI database (https://www.ncbi.nlm.nih.gov/protein).

The roles of host and pathogen sugar transporters in plant–pathogen interactions

Sugars transporters play key roles in plant–pathogen interaction (Table 4). Plants convert carbon dioxide by photosynthesis into sugars that are transported to growing tissues via both apoplastic and symplastic pathways. As 'parasites', pathogens have evolved mechanisms to obtain sugars from nutrients-rich niche of plant tissues. Depending on lifestyle and type, pathogens evolved mechanisms to manipulate sugar transports to guarantee their access to carbohydrate (Figure 7).

The roles of host plant sugar transporters in plant–pathogen interactions

Plant sugar transporters–fungi/oomycete pathogen interactions

Biotrophic fungi and oomycetes can take up sugars from the host's cytoplasm by their specialized invasive organ, the haustoria, thus influx of apoplastic hexoses will benefit a fungal infection by increasing the availability of sugars in the infected cell. Indeed, as importers of sugars, PM-localized STPs transporters play negative roles in defending against biotrophic fungi. For example, the wheat *Lr67sus* gene encodes a homologue protein of STP13, but its natural variation of *Lr67res* has lost glucose uptake activity. Wheat lines expressing *Lr67res* confer a broad-spectrum resistance to biotrophic fungal pathogens such as leaf rust *Puccinia triticina*, stripe rust *Puccinia striiformis* and stem rust *Puccinia graminis* and powdery mildew pathogen *B. graminis* (Moore *et al.*, 2015). Consistent with this idea, knockdown of wheat *TaSTP6* promotes resistance to the rust pathogen *P. striiformis*, whereas expression of *TaSTP6* in Arabidopsis increases plant susceptibility to powdery mildew (Huai *et al.*, 2019).

In contrast, STPs may also play a positive role in resistance to necrotrophic fungal pathogens which extract nutrients from the apoplast. For example, the overexpression of *AtSTP13* enhances Arabidopsis resistance to the necrotrophic fungus *B. cinerea*,

Table 3 Example SUTs from plants and fungi and their substrates

	Species	Gene	Substrates	Refs
Plants	*Oryza stativa*, *Zea mays*	*OsSUT1/2*, *ZmSUC1/2*	Sucrose	(Aoki *et al.*, 2003; Leach *et al.*, 2017)
	Arabidopsis	*AtSUC2/9*	Sucrose and a wide range of glucosides	(Chandran *et al.*, 2003; Sivitz *et al.*, 2006)
	Hordeum vulgare	*HvSUT1*	Sucrose and four glucosides	(Sivitz *et al.*, 2005)
Fungi	*Ustilago maydis*	*Srt1*	Sucrose	(Wahl *et al.*, 2010)
	Schizosaccharomyces pombe	*Sut1p*	Maltose and sucrose	(Reinders and Ward, 2001)
	Saccharomyces cerevisiae	*MAL2T and AGT1*	Sucrose	(Stambuk *et al.*, 2000)
	Trichoderma virens	*TvSut*	Sucrose	(Vargas *et al.*, 2011)
	Colletotrichum graminicola	*MBT1*	Melibiose	(Lingner *et al.*, 2011)

Table 4 Roles of transporters in plant–pathogen interactions

	Pathogen	Plant	Tissue	Transporters	Location	Roles	Ref
Fungi	*Puccinia triticina*	Wheat	Leaf	*TaSTP13*	Plant PM	Susceptible	(Moore *et al.*, 2015)
	Puccinia striiformis	Wheat	Leaf	*TaSTP13*	Plant PM	Susceptible	(Moore *et al.*, 2015)
	Blumeria graminis	Wheat	Leaf	*TaSTP13*	Plant PM	Susceptible	(Moore *et al.*, 2015)
	Puccinia hordei	Barley	Leaf	*TaSTP13*	Plant PM	Susceptible	(Milne *et al.*, 2019)
	Puccinia striiformis	Wheat	Leaf	*TaSTP6*	Plant PM	Susceptible	(Huai *et al.*, 2019)
	Botrytis cinerea	Arabidopsis	Leaf	*AtSTP13*	Plant PM	Resistant	(Lemonnier *et al.*, 2014)
	Pythium irregulare	Arabidopsis	Root	*AtSWEET2*	Plant PM	Resistant	(Chen *et al.*, 2015)
	Fusarium oxysporum	Sweet potato	Root	*IbSWEET10*	Plant PM	Resistant	(Li *et al.*, 2017)
	Colletotrichum higginsianum	Arabidopsis	Leaf	*AtSWEET11/12*	Plant PM	Susceptible	(Gebauer *et al.*, 2017)
	Rhizoctonia solani	Rice	Sheath	*OsSWEET11*	Plant PM	Susceptible	(Gao *et al.*, 2018)
	Ustilago maydis	Corn	Leaf	*UmSRT1*	Pathogen PM	Susceptible	(Wahl *et al.*, 2010)
	Botrytis cinerea	Tomato	Leaf	*FRT1*	Pathogen PM	Susceptible	(Doehlemann *et al.*, 2005)
	Uromyces fabae	Broad bean	Leaf	*HXT1*	Pathogen PM	Susceptible	(Voegele *et al.*, 2001)
	Botrytis cinerea	Arabidopsis	Leaf	*AtSWEET4*	Plant PM	Susceptible	(Chong *et al.*, 2014)
Bacteria	*Pst DC3000*	Arabidopsis	Leaf	*AtSTP13*	Plant PM	Resistant	(Yamada *et al.*, 2016)
	Xoo	Rice	Leaf	*OsSWEET11/13/14*	Plant PM	Susceptible	(Chen *et al.*, 2010; Zhou *et al.*, 2015)
	Xcm	Cotton	Leaf	*GhSWEET10*	Plant PM	Susceptible	(Cox *et al.*, 2017)
Virus	*TYLCV*	Tomato	Leaf	*LeHT1*	Plant PM	Resistant	(Eybishtz *et al.*, 2010)

whereas the mutation of *AtSTP13* results in the opposite effect, implying that STP13 may improve resistance by depriving the fungus of sugar nutrients (Lemonnier *et al.*, 2014).

SWEETs facilitate sugar diffusion across cell membranes. Upon infection, SWEETs generally facilitate the export of sugars out of host cells, which decrease sugar availability to biotrophic fungal pathogens that take up nutrient from cytoplasm through haustorium. However, a cotton glucose transporter GhSWEET42 acts as a susceptibility factor in cotton-*Verticillium dahlia* (a hemi-biotrophic fungal pathogen) interaction (Sun *et al.*, 2021). In this study, total glucose concentration in overexpressed lines has been increased by 3–4 times and decreased in gene-silenced lines, with parallel changes in pathogenic fungal susceptibility(ref.), perhaps indicating that GhSWEET42 affects glucose metabolism and glucose distribution between symplast and apoplast. Necrotrophic fungal pathogens absorb nutrient from dead tissue or apoplast and thus benefit from the activation of host SWEETs that increase apoplastic sugar availability by exporting sugars out of host cells. For example, infection with the necrotrophy *Botrytis cinerea* triggers a strong up-regulation of *VvSWEET* gene expression in *Vitis vinifera*. Knockout mutants in the orthologous *AtSWEET4* are found to be less susceptible to *B. cinerea* (Chen, 2014). Similarly, the necrotrophic fungus *Rhizoctonia solani* induces rice *OsSWEET11*, *OsSWEET2a* and *OsSWEET3a* expression in leaves. The analyses of transgenic plants reveal that *OsSWEETs* mutants are less susceptible whereas overexpression plants are more susceptible to *Rhizoctonia solani* (Gao *et al.*, 2018; Yang *et al.*, 2023).

Root sugar transporters provide an interesting comparison to leaves. For example, Arabidopsis root-expressed vacuolar SWEET2 modulates rhizosphere sugar secretion, possibly by reducing the availability of glucose sequestered in the vacuole, thereby limiting carbon loss to the rhizosphere. Moreover, the reduced availability of sugars in the rhizosphere due to SWEET2 activity sequestering glucose into root vacuoles, adjusts cytoplasmic glucose for sugar efflux and thereby contributes to plant resistance to *Pythium* (Chen *et al.*, 2015). Interestingly, Pythium infection can be induced by a 40-fold up-regulation of SWEET2 but the resulting plants can benefit *Bacillus subtilis* colonization (Yang *et al.*, 2023). This *B. subtilis* colonization can repress the SWEET2 by activating transcription factor AHL29 (Wu *et al.*, 2024), indicating a complicated interaction among pathogens, symbiosis and plants. The balance between sugar supply to pathogenic fungi and symbiotic mycorrhizal fungi in roots is complicated and the factors which switch between these modes may involve specific transporters. The expression of each type of specific transporter may provide a marker for the switch from symbiont to pathogen.

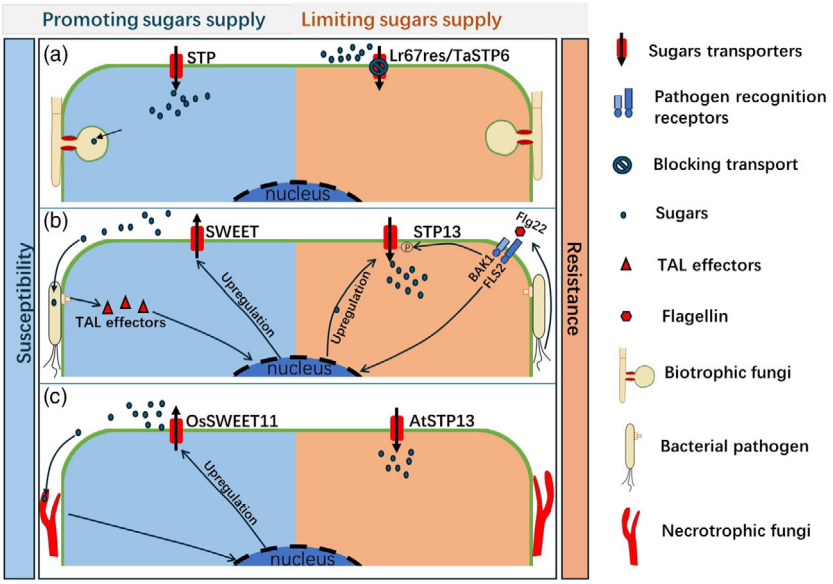

Figure 7 Schematic illustration of roles of sugar transporter in plant–pathogen interactions. (a) Haustorial-forming biotrophic fungal pathogens; STP uptakes hexose, promoting susceptibility and mutant of STP13 lead to resistance to biotrophic fungal pathogens. (b) Bacterial pathogens: Bacteria-derived flg22 activates BAK1 to phosphorylates STP13, enhancing hexose uptake and leading to resistance. Bacteria secret TAL effectors to induce expression of SWEET, leading to susceptibility.(c) Necrotrophic fungal pathogens. necrotrophic fungus Rhizoctonia solani induces expression of OsSWEET11, leading to susceptibility. Overexpression of AtSTP13 enhances resistance to necrotrophic fungal *B. cinera*.

Plant sugar transporters in plant–bacteria pathogen interactions

Different from biotrophic fungal pathogens which take nutrients from the cytoplasm, bacterial pathogens colonize and absorb nutrients directly in the apoplast. Contents and concentrations of apoplastic sugars are regulated tightly by plants through sugar metabolism enzymes and transporters. To become established in plants, bacteria manipulate plant transporters in combination with sugar metabolism to gain access to nutrients in apoplast. Apoplastic availability of sucrose depends mainly on regulations of SWEETs, STPs, SUTs and cell wall invertases (CWIN). SWEETs generally facilitate the export of sucrose or hexose out of cells (Breia *et al.*, 2021; Chen *et al.*, 2015; Pommerrenig *et al.*, 2020), to increase sugar availability in the apoplast to increase bacterial infection. SWEETs are upregulated in host plants upon infection by *Xanthomonas*. These bacteria deliver TAL effectors into leaf cells, directly inducing SWEET sugar transporters to release sucrose into apoplast where the bacteria grow (Boch *et al.*, 2014) and promoting infection (Chen *et al.*, 2010; Liu *et al.*, 2011; Yang *et al.*, 2006; Yu *et al.*, 2011). Apoplastic sucrose can be re-taken up by SUT into the cytoplasm (Chen, 2014), or be cleaved by CWINV into monosaccharides (Ruan, 2014), which can be re-imported by STPs into cytoplast (Buttner, 2010). However, in the context of infection, SUT-mediated uptake of sucrose may not be a good choice for plants due to these reasons (Liu *et al.*, 2022): (1) SUTs have relatively low K_m values (Kühn, 2012), requiring high concentrations of apoplastic sucrose. But high concentrations of apoplastic sucrose will be cleaved quickly by CWINV into monosaccharides and subsequently imported by STPs which have relatively low K_m values (Norholm *et al.*, 2006; Paulsen *et al.*, 2019; Schneidereit *et al.*, 2003); (2) SUT activities are optimal at acidic pH around 5–6 (Rottmann *et al.*, 2018; Sauer, 2007), but bacterial infections induce alkalization of the apoplast which will decrease SUT activity by decreasing the trans-PM proton gradient that drives transport. By contrast, STPs play a positive role in resistance to bacteria by importing monosaccharides into the cytoplasm, leading to low concentrations of apoplastic sugars. For example, bacteria flg22 can be recognized by plant receptor FLS2 and co-receptor BAK1. BAK1 phosphorylates a sugar transporter STP13, which enhances hexose uptake activity into the symplast from apoplast where bacteria grow, leading to resistance to bacterial pathogen (Yamada *et al.*, 2016). Consistent with this idea, the Arabidopsis double mutant *stp1stp13* shows a higher concentration of apoplastic glucose and exhibits an increased susceptibility to bacterial pathogens (Yamada *et al.*, 2016).

Plant sugar transporters in plant–virus interactions

Plant viruses are one of the smallest and most complex pathogens to utilize the symplast of the cell and its molecular and structural machinery to induce infection and spread in the plant host. Plant viruses utilize the nutrients directly from cytoplasm. For example, tomato yellow leaf curl virus (TYLCV) is a devastating disease resulting in significant crop losses each year (Moriones and Navas-Castillo, 2000). The hexose transporter gene *LeHT1* transcript is strictly regulated in the resistant line of the two inbred tomato lines (Resistant line and Susceptible line) (Eybishtz *et al.*, 2010). Silencing the gene *LeHT1* in R line leads to a LeHT1-silenced resistant line (termed Ri line) which has susceptibility to TVLCV infection, but not to the extent observed in S lines lacking *LeHT1* expression. In Ri and S lines, the virus exhibits increased mobility. Interestingly, Ri line also undergoes programmed cell death after infection with the virus, a response that has not been observed in R or S lines. This indicates the possible function of this hexose transporter in defence against TYLCV, since it would not be necessary to sequester sugars from a virus. It has been suggested that silencing *LeHT1* could increase PD permeability and thereby increase TYLCV mobility (Eybishtz *et al.*, 2010). Thus, the LeHT1 protein may be involved in manipulating the symplastic trafficking pathway by regulating PD permeability (Julius *et al.*, 2017).

Table 5 Example sugar transporters and their affinity for sugar substrates

	Species	Transporters	Substrates	Km (mM)	Refs
Pathogen	*Ustilago maydis*	*Srt1*	Sucrose	0.026	(Wahl *et al.*, 2010)
	Geosiphon pyriformis	*GpMST1*	Glucose	1.2	(Schüssler *et al.*, 2006)
	Saccharomyces cerevisiae	*MAL11*, *MAL1*	Maltose	4	(Cheng and Michels, 1991; Stambuk *et al.*, 2000)
		MAL2T	Maltose	70–80	
			Sucrose	120	
	Saccharomyces cerevisiae	*AGT1*	Sucrose	8	(Reinders and Ward, 2001)
			Maltose	20–35	
	Schizosaccharomyces prombe	*Sut1p*	Maltose	6.5	(Reinders and Ward, 2001)
			Sucrose	36.3	
	B.cinerea	*FRT1*	Fructose	0.16	(Doehlemann *et al.*, 2005)
	Ustilago maydis	*HXT1*	Glucose	0.018	(Voegele *et al.*, 2001)
Plant	*Fava bean*	*VfSUT1*	Sucrose	1.4	(Weber *et al.*, 1997)
		VfSTP1	Glucose	0.030	
	Arabidopsis	*AtSUC1*	Sucrose	0.25	(Zhou *et al.*, 1997)
	Plantago major	*PmSUC3*	Sucrose	5.5	(Zhou *et al.*, 1997)
	Hordeum vulgare	*HvSUT1*	Sucrose	7.5	(Zhou *et al.*, 1997)
		HvSUT2		5	

The roles of pathogen sugar transporters and metabolism enzyme in plant–pathogen interactions

In addition to manipulating host transporters to obtain host-derived nutrients, pathogens also utilize their own transporters to compete with the host for uptake of host-derived nutrient. In the interface of plant–pathogens, pathogen transporters contact directly with the same solutes as host transporters do. To obtain host-derived sugars from this shared interface pool, pathogens have developed the three strategies listed below.

Pathogen transporters have a higher affinity to sugar substrates than host transporters

Sugar transporters from both plants and pathogens often have different substrate affinities (Table 5). For example, the corn smut fungus (*Ustilago maydis*) encodes a novel, high-affinity sucrose transporter, UmSRT1, which is more efficient in sucrose uptake than that of the host ZmSUT1 protein (Wahl *et al.*, 2010). Upon deletion of UmSrt1, pathogen virulence is greatly decreased, suggesting this transporter efficiently competes for extracellular sucrose with the adjacent cells of its host at the plant–fungus interface (Wahl *et al.*, 2010). This result shows pathogen sugar transporter substrate affinity (K_m) may be as a target for resistance.

Pathogens can directly take up hexoses from the host plant. In *B. cinerea*, a fructose transporter FRT1 plays a key role in pathogenesis (Doehlemann *et al.*, 2005). FRT1 is highly specific for fructose and contributes to fructose-induced germination of fungus. Rust fungus *U. fabae* expresses a hexose transport protein HXT1 in rust haustoria but is negligible in other fungal structures. HXT1 has assigned a substrate specificity for D-glucose and D-fructose (Voegele *et al.*, 2001), indicating that pathogen can utilize such haustoria hexose transporters to uptake sugars and increase pathogenesis.

Pathogens can disturb sugar partitioning using invertases

It has been speculated that sugar transporters act in combination with sugar metabolism enzymes such as invertases, which can hydrolyse sucrose into monosaccharides. Activation of plant CWINs that hydrolyse sucrose into monosaccharide can trigger plant immune responses (Zhang *et al.*, 2023). To overcome host CWIN-triggered plant immune response, obligate biotroph fungus *Uromyces fabae* uses its own invertase UfINV1 to disturb sugar partitioning and promote infection during host–pathogen interactions (Voegele *et al.*, 2006).

Pathogens can utilize the specific forms of sugars from hosts

In order to avoid directly competing with host for sugars, some pathogens can utilize the specific forms of carbon from the host and exploits it as a carbon source to support primary infection and development in plant tissue. For example, *Phytophthora sojae*, an oomycete causing stem and root rot of soybean, directly acquires trehalose from the host and exploits it as a carbon source to support infection (Zhu *et al.*, 2023).

Manipulation of plant sugar transporters as targets for pathogen resistance

Sugar metabolism and transport are essential biological functions for plants. As susceptible (S) factors, plant sugar-related genes often are hijacked by pathogens to benefit themselves and to promote infection (Cohn *et al.*, 2014). S genes have important physiological functions in host plants and therefore their mutations are typically accompanied by a variety of undesired pleiotropic effects on plant growth, development and crop yields, which greatly limits the application of S genes in plant disease resistance breeding (Deng and Cao, 2022). However, disruption of S genes usually confers durable and broad-spectrum disease resistance in crops and is an attractive breeding strategy for conferring disease resistance (Li *et al.*, 2022; Wang *et al.*, 2014; Yang *et al.*, 2006). Here, we will discuss some applications as examples.

Genetic modification of transporters in a constitutive manner

Upon infection of plants by the bacterial pathogen *Xanthomonas* spp., many *Xanthomonas* strains secrete transcription activator-like(TAL) effectors, which enter the host cell nucleus and activate

host SWEETs at effector binding elements(EBEs) in the promoter, inducing the transporters and promoting susceptibility in host plants (Bezrutczyk *et al*., 2018; White *et al*., 2009). For example, rice SWEET11, SWEET13 and SWEET14 are targeted by *Xanthomonas* TAL effector PthXo1, PthXo2 and PthXo3 at the SWEET promoter EBE region, respectively, to activate expression of these *SWEETs*, leading to susceptibility (Antony *et al*., 2010; Streubel *et al*., 2013; Yang *et al*., 2006). Recognition between TAL effector and EBE of promoter is specific and depend on the TAL domain and the EBE sequence (Boch *et al*., 2009; Moscou and Bogdanove, 2009). Thus, mutation in the promoter EBE region of SWEET genes can abrogate the recognition and increase resistance likely without losing their sugar transport function in host plants (Antony *et al*., 2010; Yu *et al*., 2011). For instance, CRISPR–Cas9-mediated genome editing was used to introduce mutations in three SWEET gene promoters, leading to broad-spectrum resistance to bacterial blight in rice (Oliva *et al*., 2019). Although some mutations in SWEET promoter such as naturally occurring SWEET11 promoter variants *xa13* promoter confer resistance and do not negatively affect yield (Sakthivel *et al*., 2017), it is conceivable that promoter-edited lines or variants could impair yield, if the promoter variations affect normal gene function in uninfected plants. To overcome this, a diagnostic kit was developed that includes a SWEET promoter database, RT–PCR primers for detecting SWEET induction, engineered reporter rice lines to visualize SWEET protein accumulation and knockout rice lines to identify virulence mechanisms in bacterial isolates. With this kit, SWEET knockout lines are generated using CRISPR-Cas9 to investigate their roles in resistance and yield (Eom *et al*., 2019). With this strategy, *sweet13* and *sweet14* knockout lines show resistance but did not show detectable growth or yield defects under greenhouse conditions, nor were obvious differences observed in a single-season field experiment (Eom *et al*., 2019).

Genetic modification of transporter genes in a spatiotemporally dependent manner

Constitutive mutation of transporters, which play important roles in plant growth and development, often have undesired pleiotropic effects on plants. An alternative strategy with less pleiotropic effects is to engineer S genes in a tissue-specific or infection-induced manner. This type of approach can reduce the negative effect on plant growth but induce plant resistance. For example, a tissue-specific promoter will narrow the S gene silencing in particular tissues without affecting others. Rice Xa13 (OsSWEET11) is essential for rice pollen viability (Chen *et al*., 2010), and Xa13 is exploited by bacterial pathogens for virulence by direct binding of a bacterial effector to SWEET promoter (Yang *et al*., 2006). Constitutive suppression of *Xa13* leads to enhanced resistance, but significantly reduced the pollen viability (Chu *et al*., 2006; Yang *et al*., 2006). Instead, tissue-specific promoters *pOsrbcsp* were used to silence *Xa13* in the non-anther tissues but maintain normal expression in pollen, thus generating highly bacterial blight-resistant transgenic plants with normal pollen viability (Li *et al*., 2012). Another interesting example is OsSWEET14, which positively regulates rice resistance to sheath blight (ShB). Non-specific overexpression of SWEET14 significantly reduced yield production, suggesting that SWEET14 plays a role in both yield production and defence. DOF11 is identified as a direct transcriptional regulator of SWEET14, and DOF11 overexpression increased resistance to ShB but reduced yield production. Interestingly, tissue-specific activation of DOF11 by fusion of VP16 a transcriptional activation domain (Li *et al*., 2013) increased both yield production and resistance to ShB (Kim *et al*., 2021). Furthermore, some plant genes are induced only during infection and expressed just in the infected cells such as Downy Mildew Resistance 6 (DMR6) (van Damme *et al*., 2008). Thus, the promoter of DMR6 can be used to drive the expression of a RNAi construct which target certain S genes and knockdown their expression in a particular spatiotemporal manner to increase resistance during pathogen infection and minimize unwanted pleiotropic effects.

Natural variation in transporters

The greatest limitation to the introduction of resistance in plants by manipulating plant S genes is the fitness cost because most S genes have essential functions. However, within plant species, there is considerable natural variation of S genes that have been shaped by differences in selection pressure (Thompson, 2005). These natural genetic variations of S genes are thought to be maintained by trade-offs between the benefits from increased resistance and the fitness cost of the sacrificed essential functions (Zaidi *et al*., 2018). Some of natural variation in sugar transporters has been found to confer resistance without any penalty in plant growth and development. For example, naturally occurring SWEET11 promoter variants xa13 promoter confer resistance and do not negatively affect yield (Sakthivel *et al*., 2017). By mining a rice diversity panel for mutations in the promoter of *OsSWEET13* and *OsSWEET14*, natural variations at the EBE of both genes are identified and displayed resistance to *Xanthomonas oryzae* Xoo (Zaka *et al*., 2018). Wheat *Lr67sus* gene encodes a homologue protein of STP13, but its natural variant *Lr67res* losses glucose uptake activity. Wheat lines expressing *Lr67res* confer a broad-spectrum resistance to biotrophic fungal pathogens (Moore *et al*., 2015). Thus, natural variation may offer a promising opportunity for sugar transport-related resistance, and gene editing (GE) technology may be able to quickly exploit this information in many crops.

Conclusions and future opportunities

Sugar provide energy and building blocks for both plants and pathogens. Upon infection, pathogens need to acquire the host-derived sugars to establish a successful infection because they cannot synthesize sugars themselves. Sugars can move to the plant infection site through two pathways: Apoplastic and symplastic pathway but can only be exchanged through membrane transporters between apoplast and symplast. Plants regulate the distribution of sugars via sugar transporters and metabolic enzymes, whereas pathogens hijack plant transporters or utilize their own transporters and metabolic enzymes to redistribute host-derived sugars to benefit infection. The main strategies currently to generate resistance are focused on engineering plant transporters to produce different types of mutant lines. However, yield penalties and other undesired pleiotropic effects often are inevitable by mutating the plant sugar transporters which have essential roles in host plants. New ideas of how to generate sugar starvation-mediated resistance require more studies on the pathogen transporters in addition to those on the plant side.

In the plant–pathogen interface, there is direct competition for extracellular sugars between plant transporters and pathogen transporters. The transporter with high affinity (low K_m) will compete for sugars more efficiently (Doehlemann *et al*., 2005;

Voegele *et al*., 2001; Wahl *et al*., 2010), leading to resistance whether plant transporters have higher affinity or susceptibility if pathogen transporters is more competitive. Investigation of sugar binding pockets and key residues in high-affinity transporters using high-resolution structure of the transporter proteins (Bavnhøj *et al*., 2021, 2023) is an exciting opportunity to improve the binding affinity of plant sugar transporters by gene editing techniques. Furthermore, artificial intelligence(AI) and revolutionary computational tools such as AlphaFold2 and RoseTTAFold offer highly accurate predictions of three-dimensional protein structures to aid this research (Baek *et al*., 2021; Jumper *et al*., 2021; Smorodina *et al*., 2022; Varadi *et al*., 2022). Together these tools can provide information for designing high-affinity transporters as targets for gene editing in crops for the fight against pathogens.

At the infection site, pathogens need to manipulate plant sugar transporters, in combination with their own sugar transporters, to get access to host-derived sugars. Therefore, blocking these plant and pathogen sugar transporters but only at the infect site by transporter inhibitors could be a potential strategy for sugar starvation locally, leading to resistance. There are some artificial blockers which bind irreversibly to the sugar substrate binding site on both plant and pathogen transporter proteins. These molecules are often non-metabolizable sugar analogues such as 2-deoxy-D-glucose (Pajak *et al*., 2019) and sucralose (Schiffman and Rother, 2013). Engineering of the plant sugar transporters to be insensitive to these blockers can possibly limit supply of sugars for pathogens and lead to resistance when externally applying these inhibitors to infect sites. Further, some natural products can block sugar transporters (e.g. Phloridzin; Lemoine and Delrot, 1987) or invertases (e.g. INH1; Palmer *et al*., 2015). Engineering pathogen-inducible synthetic pathways in plants for these types of natural blockers in infection site may be another way to generate starvation-mediated resistance (Jumper *et al*., 2021).

Plants evolved multiple mechanisms to defend against pathogens. In addition to activating their immune system to eliminate pathogens, plants also actively block pathogen access to sugars to prevent colonization. In response, pathogens developed strategies to modulate the host immunity and also to manipulate plant sugar transporters to meet their needs for carbon during infection. Plant immunity involves many genes working together in a complex network, but sugar transport in plants is relatively simple and the major transporter families are identified. This might suggest that sugar starvation-based resistance strategies could be a good alternative to immune-based resistance strategies. A combination of both strategies may provide novel opportunities to design more durable resistance in agriculture.

Acknowledgements

This work was financially supported by the Jiujiang Natural Science Foundation (Liu xiaokun); Jiangxi Natural Science Foundation (20224BA205006); and Talents program of Jiangxi Province (jxsq2023101036).

Conflict of interest

The authors declare no conflicts of interest.

Data availability statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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