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REVIEW

The role of sugar signaling in plant defense responses against fungal pathogens

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Abstract In most fungal pathogen–plant systems, a high level of sugars in plant tissues enhances plant resistance. Several hypotheses have been proposed to explain the mechanisms of "high-sugar resistance". Sugars constitute the primary substrate providing energy and structural material for defense responses in plants, while they may also act as signal molecules interacting with the hormonal signaling network regulating the plant immune system. Sugars enhance oxidative burst at early stages of infection, increasing lignification of cell walls, stimulate the synthesis of flavonoids and induce certain PR proteins. Some sugars act as priming agents inducing higher plant resistance to pathogens.

Keywords Sugar signaling · Plant immune system · Fungal pathogens · Plant defense responses · Abiotic stress

Abbreviations

TFs	Transcription factors
PAMPs	Pathogen-associated molecular patterns
MAMPs	Microbe-associated molecular patterns
EF-Tu	Bacterial Elongation Factor-Tu
PRRs	Transmembrane pattern recognition receptors
PTI	PAMP-triggered immunity
LRR	Leucine-rich repeat
LysM	Lysin motifs
MAPK	Mitogen-activated protein kinase
ROS	Reactive oxygen species
ETS	Effector-triggered susceptibility
NB-LRR	Nucleotide-binding site leucine-rich repeat protein
ETI	Effector-triggered immunity
PCD	Programmed cell death
PR	The pathogenesis-related proteins
HR	Hypersensitive response
HXK	Hexokinase
CAB	Chlorophyll *a*/*b*-binding protein
RGS1	G-Protein signaling protein 1
T6P	Trehalose-6-phosphate
SnRK1	Sucrose non-fermenting-1 related protein kinase 1
AMPK	50-AMP-activated protein kinase
G6P	Glucose-6-phosphate
Suc	Sucrose
bZIP	The basic region-leucine zipper motif
AtbZIP1	*Arabidopsis* group C/S1 basic leucine zipper (bZIP)
KIN10/11	*Arabidopsis* protein kinases (also known as AKIN10/At3g01090 and AKIN11/At3g29160)
AtSTP	*Arabidopsis* sugar transporter protein
SUT1	Sucrose transporter
VvHT5	Stress-inducible hexose transporter
SWEETs	Class of sugar transporters
HXT1	Hexose transporters
SAR	Systemic acquired resistance
ISR	Induced systemic resistance
SA	Salicylic acid
JA	Jasmonic acid
ET	Ethylene

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ABA Abscisic acid
SPS Sucrose phosphate synthase

Introduction

Sugars, owing to their regulatory function, affect all phases of the life cycle of plants and, interacting within phytohormones, control the processes of growth and development of plants (Wind et al. 2010; Stokes et al. 2013). There are many reports on the importance of sugar levels in plant resistance to diseases caused by fungal pathogens and oomycetes, but their role as signal molecules in defense responses has only been described in recent publications (Doehlemann et al. 2008; Morkunas et al. 2011; Bolouri Moghaddam and Van den Eden 2012). This influx of novel data has been provided by studies on mutants, primarily *Arabidopsis thaliana*, with disturbed sugar signaling pathways, on transgenic plants and thanks to the results supplied by analyses of gene expression (Cho et al. 2012; Schenk et al. 2012). Research on molecular plant responses to abiotic stresses also provides information which is useful in the interpretation of reactions occurring in plants during fungal pathogen attack (Hey et al. 2010). Most environmental changes are stressful, although some may be beneficial. To counteract stressful changes and grow successfully, a majority of plants launch resistance mechanisms to stressful environments by reprogramming metabolism and gene expression, and acquiring a new equilibrium between development and defense (Yu et al. 2010). At the same time, attempts have been made to discuss the role of sugar level in resistance to abiotic stresses (e.g., Rosa et al. 2009). Based on the results of ecological and agronomic studies were stated that there is a strong correlation between soluble sugar concentration and stress tolerance. In this review are present examples of such correlation also occurring in the case of many, although not all, biotic stresses. In biotic stresses caused by pathogenic fungi additional problems are faced in the interpretation of the dependence of resistance on sugar levels. Pathogens interfere with the metabolism of their host and do so not only through uptake of sugars and other metabolites for their own needs but may also disturb plant metabolism to different degrees. Plants and pathogens engage in an evolutionary tug-of-war, in which the plant limits pathogen access to nutrients and initiates immune responses, whereas the pathogen evolves adaptive strategies to gain access to nutrients and suppress host immunity (Boller and He 2009; Chen et al. 2010).

Involvement of sugars in plant immune system

The innate resistance of plants to pathogens and systemic resistance induced by signals originating from the infection site have been known for a long time. However, only the recent development of genomics has made it possible to obtain data facilitating a thorough comparison of relationships in different plant–pathogen systems. Nishimura and Dangl (2010) reported that following the establishment of a complete genome sequence of *Arabidopsis*, an explosion of information regarding both disease resistance and susceptibility to pathogens has been observed. They calculated that the curve illustrating the number of publications concerning plant–pathogen interactions is similar in shape to the exponential curve. Accumulation of these data made it possible to formulate certain generalizations on the immune response of plants to pathogen attack, as, e.g., the zig-zag model developed on the basis of work conducted by many laboratories (Jones and Dangle 2006). In this model the plant immune system is divided into four phases. Although numerous modifications are continually being made to the details of this model, it still provides a good basis to explain molecular events (Ahmad et al. 2010; Zipfel and Robatzek 2010; Rampitsch and Bykova 2012; Chujo et al. 2013). Jones and Dangle (2006) distinguished two classes of molecules which plants are capable of distinguishing as pathogen attack. Conserved microbial molecules are referred to as pathogen-associated molecular patterns (PAMPs) or microbe-associated molecular patterns (MAMPs). PAMPs include a growing list of microbial molecules: lipooligosaccharides of gram-negative bacteria, bacterial flagellin, bacterial Elongation Factor-Tu (EF-Tu), glucans and glycoproteins from oomycetes, chitin from fungus cell walls, etc. (Zhang and Zhou 2010). PAMPs are perceived by host receptor proteins known as transmembrane pattern recognition receptors (PRRs), and their recognition causes PAMP-triggered immunity (PTI). Known plant PRRs are modular proteins harboring an extracellular domain consisting of LRR (leucine-rich repeat) or lysin motifs (LysM). PTI relies on MAP kinase (MAPK) activation, production of reactive oxygen species (ROS), transcriptional reprogramming, hormone biosynthesis and deposition of callose, a high molecular weight β-(1,3)-glucan polymer in the cell wall. This is, according to the model proposed by Jones and Dang, the first 'zig' towards resistance. In this model phase 2 occurs when successful pathogens deliver effectors that interfere with PTI, or otherwise enable pathogen nutrition and dispersal, resulting in effector-triggered susceptibility (ETS). Fungal and oomycete effectors can act either in the extracellular matrix or inside the host cell. These effectors can suppress host defense (the 'zag'). In phase 3, one effector is recognized by an NB-LRR (nucleotide-binding site leucine-rich repeat) protein, activating more specific (gene-for-gene) resistance responses denominated effector-triggered immunity (ETI). ETI is an amplified version of PTI that often passes a threshold for the induction of hypersensitive cell death (HR). In phase 4, natural selection drives pathogens to avoid ETI either by shedding or diversifying the recognized effector gene, or by acquiring additional effectors that suppress ETI.

Although Jones and Dangle (2006) did not mention the role of sugars, in the opinion of some researchers sugar signals may also contribute to immune responses against pathogens. They probably function as priming molecules leading to pathogen-associated molecular patterns (PAMP)-triggered immunity and effector-triggered immunity in plants (Gómez-Ariza et al. 2007; Bolouri Moghaddam and Van den Eden 2012). This novel concept of "sweet priming" predicts specific key roles to saccharides in perceiving, mediating and counteracting both biotic and abiotic stresses (Bolouri Moghaddam and Van den Eden 2012) (Fig. 1).

There are attempts to explain the phenomenon of higher resistance to fungal diseases of plants with higher levels of sugar in their tissues. This phenomenon was initially described as a characteristic of plants prone to low-sugar diseases (Horsfall and Diamond 1957). In recent literature it is termed "high-sugar resistance" and includes the induction of several plant defense mechanisms (Ferri et al. 2011). Basing on the latest published research results, an attempt will be made to clarify the varied involvement of sugars in the immune system of plants.

Sugar sensing and signaling

Defense response results in a substantial reprogramming of plant cells (Bolton 2009; Doehlemann et al. 2008). Many plant responses to the attack of a fungal pathogen are closely connected with the pathways regulating the level of sugar in the plant cell and ensuring energy homeostasis (Hey et al. 2010). A significant role in these responses is played by sugars themselves, acting as signaling molecules. Several such mechanisms have been described (Rolland et al. 2006). Sugars regulate cellular activity at multiple levels, from transcription and translation to protein stability and activity (Rolland et al. 2006). Hexokinase (HXK1) is the best investigated glucose sensor, while this protein also serves an enzymatic function, catalyzing the first step of glycolysis—conversion of glucose to glucose 6-phosphate (Smeekens et al. 2010). Hexokinase isoforms have been found in the cytosol, chloroplast, mitochondria and the nucleus (Hanson and Smeekens 2009; Cho et al. 2009). This diversity of subcellular localizations of hexokinases may reflect their roles in a variety of cellular processes. Mitochondria-associated hexokinases play a role in the control of programmed cell death (PCD). Kim et al. (2006) showed that hexokinase-mediated PCD promotes the expression of many of the pathogenesis-related (PR) genes induced during hypersensitive response (HR) cell death, indicating that some features of HR cell death are conserved in the hexokinase-mediated PCD process. On the basis of the results of analyses of gene expression in the HXK mutant or transgenic *Arabidopsis thaliana* plants it was shown that nuclear hexokinase signaling integrates nutrient and hormone signals to regulate gene expression and plant growth, physiology, and development (Bolouri-Moghaddam et al. 2010). It is nuclear HXK that is responsible for the repression of gene transcription of

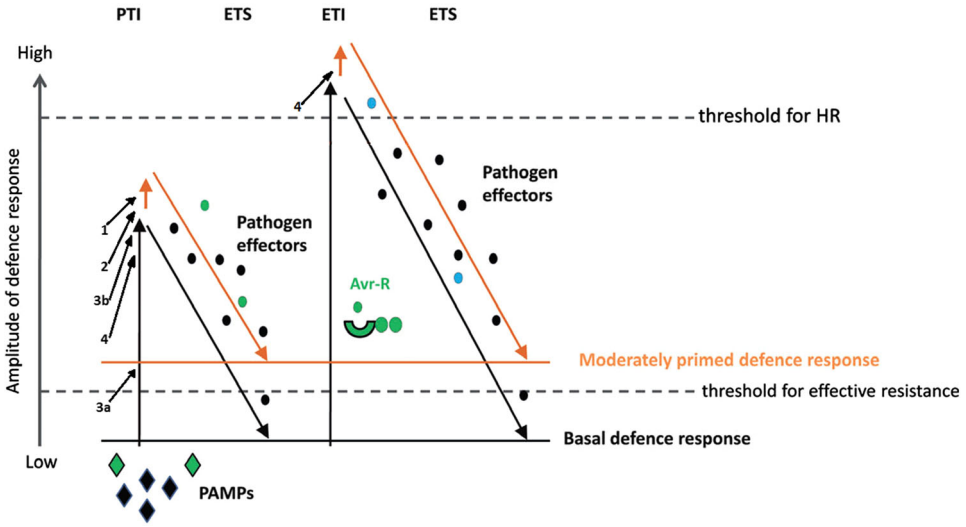

Fig. 1 Sugars influence the plant immune system as priming molecules, probably moderately stimulating it. This figure presents a modification of the zig-zag model (Jones and Dangle 2006) proposed by Ahmad et al. (2010), which occurs during moderate primed defense response (*orange*). *Numbers with arrows* indicate selected examples of immune system stimulation induced by sugars, which are proposed in this paper: *1* Stimulation of intensity of respiration processes and enhancement of oxidative burst, *2* lignification of cell walls, *3* stimulation of the phenylpropanoid pathway: *a* a higher flavonoid level in host cells enhances the basic defense response, *b* a higher flavonoid level affects the pathogen, *4* stimulation of R protein synthesis elevates PTI and ETI. *PTI* PAMP-triggered immunity, *ETS* effector-triggered susceptibility, *ETI* effector-triggered immunity, *HR* hypersensitive cell death, *Avr-R* R protein that recognize a given effector (color figure online)

certain photosynthesis proteins, e.g., chlorophyll *a/b*-binding protein (CAB), which was the subject of one of the first reports on the role of sugars in the regulation of gene expression in plants (Sheen 1990). The molecular mechanisms responsible for glucose-dependent transcriptional repression of the chlorophyll *a/b* CAB2 involve a nuclear HXK1 complex that binds the CAB2 promoter (Cho et al. 2006). Glucose activates the expression of several PR genes. The presence of hexokinase 1 is required for the induction of some of these genes, but it is not connected with the signaling function of this protein, but with its catalytic activity (Xiao et al. 2000). As it was reported by Rampitsch and Bykova (2012), glycerol-3-phosphate acts as a signal for innate immunity in the response to pathogen attack.

Apart from HXK, the G-protein-coupled receptor (regulator of G-protein signaling protein 1—RGS1) is another glucose sensor (Huang et al. 2006; Grigston et al. 2008). Due to its location in the plasma membrane it plays an important role in the transduction of extracellular glucose signaling (Baena-Gonzalez 2010). As it was reported by Perfus-Barbeoch et al. (2004), mutations in the G-protein subunit of rice showed altered responses to elicitors and pathogens, e.g., the rice blast fungus, which, according to those researchers, indicates the involvement of RGS1 in defense responses, i.e., through stimulation of ROS synthesis.

Apart from glucose, sucrose also functions as a signaling molecule (Wind et al. 2010), as it affects the expression of certain genes which enhance the expression of anthocyanin biosynthesis genes. Its involvement in the regulation of translation in certain transcription bZIP factors is discussed below. Trehalose is another disaccharide performing the signaling function in growth and development processes of plants, as well as plant defense responses, while trehalose-6-phosphate (T6P) is considered to be a powerful signaling molecule in plant cells (Paul et al. 2008; Delatte et al. 2011). Trehalose is a well-known non-reducing sugar that has been shown to partially induce resistance against powdery mildew (*Blumeria graminis* f. sp. *tritici*) in wheat by the activation of phenylalanine ammonia-lyase and peroxidase genes (Reignault et al. 2001; Muchembled et al. 2006). Control of sugar and energy metabolism in cells regulated by sucrose non-fermenting-1-related kinase (SnRK1) is a highly important plant defense mechanism against different stresses, both biotic and abiotic (Baena-Gonzalez and Sheen 2008; Hey et al. 2010). It is closely related to the metabolic regulators: sucrose non-fermenting-1 protein kinase (SNF1) of yeast (*Saccharomyces cerevisiae*) and 50-AMP-activated protein kinase (AMPK) of mammals (Polge and Thomas 2007; Robaglia et al. 2012). In *Arabidopsis thaliana* plants, two protein kinases KIN 10 and KIN 11, collectively designated as SnRK1s, are responsible for energy signaling in the cell, formed as a result of stress, both biotic and abiotic (Baena-Gonzalez 2010). SnRKs is thus a link between metabolism and the network of the stress signals in plant cells (Halford and Hey 2009). These stresses also include sugar starvation and metabolism disorders caused by pathogen attack. Jones et al. (2011) showed that in rice infested by *Magnaporthe grisea* sensitive cultivars had a lower level of metabolites involved in energy metabolism than cultivars resistant to that fungus. At a threat of cell energy deficit SnRK1 regulates the expression of over 1,000 genes, restoring cell homeostasis by repressing energy-intensive anabolic pathways and activating catabolism genes (Baena-Gonzalez 2010). Studies conducted on transgenic plants with the expression of the inactive SnRK1 form showed that this kinase is responsible for the activation of genes by stress conditions, as e.g., the expression of two marker genes for the flooding stress response, alcohol dehydrogenase 1 and pyruvate decarboxylase 1 was found only in plants expressing wild-type SnRK1 (Cho et al. 2012). However, this activation may be abolished in wild plants by the addition of exogenous 90 mM sucrose. In the opinion of those authors, it indicates that the application of sucrose causes repression of SnRK1 activity. Inactivation of SnRK1 by sugars, i.e., glucose and sucrose, was previously shown by Baena-Gonzalez et al. (2007), while sugar deficit is a strong inducer of this kinase. SnRK1 is also inhibited by G6P (glucose-6-phosphate) and T6P (trehalose-6-phosphate) (Zhang et al. 2009; Wingler et al. 2012; Nunes et al. 2013b). As it was reported by Nunes et al. (2013a), T6P content is closely related to Suc availability. By inhibiting SnRK1 (and possibly also through SnRK-independent regulation), T6P increases the expression of biosynthetic genes, e.g., for protein, nucleotide, and cell wall synthesis. Changes in gene expression in *Arabidopsis* seedlings with increased or decreased T6P levels are consistent with the inhibition of SnRK1 by T6P in vivo (Wingler et al. 2012). This regulation also corresponds to the role of T6P as a "feast" signal when carbon supply is high. SnRK1 regulates gene expression through the activity of specific transcription factors bZIP (for basic region/Leu zipper motifs). The S-group of bZIP is of particular importance for the regulation of sugar metabolism. They are small proteins, generally involved in sugar and stress signaling. In *Arabidopsis* they are S1, bZIP1, bZIP2, bZIP11, bZIP44 and bZIP53 subgroups whose synthesis is repressed by sucrose at the translation level (Baena-Gonzalez et al. 2007; Hanson et al. 2008; Kang et al. 2010). In the case of certain bZIP members of the S1 subgroup, additional sugar-induced regulations were detected. For example, their transcriptional responses to sugars are variable: while AtbZIP11 is sugar inducible, AtbZIP1, AtbZIP2, and AtbZIP53 are sugar repressible (Price et al. 2004). Translation of bZIP11 mRNA in *A. thaliana* is repressed in

response to sucrose (other sugars tested were found to be less effective—Hummel et al. 2009), whereas in the carbohydrate-consuming sink tissue it is up-regulated at the mRNA level (Rook et al. 1998; Kang et al. 2010). Proteins of S1 bZIP transcription factors bind with proteins belonging to the C-class of bZIPp and only such heterodimers are activated by KIN10/11 (Ehlert et al. 2006; Hanson and Smeekens 2009). Members of the C-class of bZIP proteins which form heterodimers with S1 proteins of bZIP include bZIP9, bZIP10, bZIP 25 and bZIP 53 (Fig. 2 in Hanson and Smeekens 2009). Such heterodimerization facilitates numerous and diverse combinations of members of these two protein groups. It is of great importance, as it facilitates different variants of regulation and modification of plant growth and development, as well as their metabolism in response to several stimuli. The expression of genes in both groups consists in the regulation by numerous stress factors, both biotic and abiotic (Weltmeier et al. 2009). AtbZIP10 was shown to be involved in oxidative stress response, particularly during defense against the fungal biotroph *Hyaloperonospora parasitica* (Kaminaka et al. 2006). It was shown that AtbZIP10 is a positive mediator of basic plant defense responses and hypersensitive response (HR) following pathogen attack.

The formation of a new sink at invasion site caused by fungal pathogen attack

The joint level of soluble carbohydrates in plants attacked by a fungal pathogen, as well as proportions of individual sugars, may be variously modified, both by plant regulatory mechanisms and by pathogen interference. Invasion of pathogenic fungi always causes changes in sugar metabolism of plants, but they may vary depending on the type of the host–pathogen system. There are several causes for quantitative and qualitative changes of sugars at the infection site. The level of sugars is reduced by their consumption for both energy and structural purposes, their uptake by the pathogen, while in autotrophic tissues it happens due to the inhibition of photosynthesis. Sugar losses are compensated for, sometimes in excess, by the influx of sugars thanks to the transformation of the infection site into a sink. Consequently, in different plant–pathogen interactions either a decrease or an increase was observed in the level of sugars in infected tissues (Berger et al. 2007). Some of these interactions will be discussed in greater detail. Induction of cell wall invertase genes and induction of hexose transporter and sucrose transporter genes are considered to be the primary causes for the formation of a sink at the infection site (Sutton et al. 2007; Essmann et al. 2008; Kühn and Grof 2010; Morkunas et al. 2010). Cell wall invertase is an extracellular enzyme which cleaves sucrose. Cell wall invertase is a sink-specific enzyme, normally found in various types of carbohydrate-consuming tissues and its activity is usually low in source leaves (Essmann et al. 2008). However, when leaves are attacked by a pathogen, a rapid increase is observed in the activity of this enzyme (Chou et al. 2000; Fotopoulos et al. 2003; Hayes et al. 2010). Apart from the induction of plant cell wall invertase activity at the infection site, the activity of fungal invertases is observed (Heisterüber et al. 1994; Chou et al. 2000; Voegele et al. 2006), which also degrades sucrose in the apoplast. As it was mentioned above, induction of sugar transporter genes also contributes to the formation of a sink at the infections site. Infection of the fungal biotroph *Erysiphe cichoracearum* on *Arabidopsis* leaves rapidly elicits the defense response and induces a high expression level of a monosaccharide transporter, called sugar transporter protein 4 (AtSTP4) (Fotopoulos et al. 2003). In other fungal pathogen–host systems, the induction of the STP4 transporter, e.g., powdery mildew (*Blumeria graminis*), causes the induction of the AtSTP4 homologue in infested wheat leaves (Sutton et al. 2007). In leaves of maize infested by the fungus *Colletotrichum graminicola*, enhanced expression was observed for the SUT1 sucrose transporter (Vargas et al. 2012). In grapevine leaves infested by obligatory biotrophs *Erysiphe necator* and *Plasmopara viticola* numerous hexose transporters were induced, but the strongest effect was found for *VvHT5*, which was also induced in response to wounding. This, according to the authors, suggests their general role in plant response to stress (Hayes et al. 2010). VvHT5 shows the highest similarity to AtSTP13 and both of them have a comparable high affinity to glucose ($K_m = 89$ μM and $K_m = 74$ μM, respectively) (Norholm et al. 2006; Hayes et al. 2007; Afoufa-Bastien et al. 2010). Furthermore, the expression of these two transporters is described to be induced in response to pathogen attack (Norholm et al. 2006; Hayes et al. 2010). Induction of STP sugar transporters is a characteristic feature of plant response to various stresses, both biotic and abiotic. AKIN10, a central integrator of transcription networks in plant stress and energy signaling, has a significant impact on AtSTP expression levels (AtSTP3: 0.4-fold; AtSTP7: 1.7-fold; AtSTP4: 1.6-fold; AtSTP1: 2.6-fold; AtSTP14: 35-fold), as determined by transient AKIN10 expression in mesophyll protoplasts (Baena-Gonzalez et al. 2007).

Members of the newly described class of sugar transporters, referred to as SWEETs, are also to a varied degree induced during the invasion of pathogenic fungi (Chen et al. 2010). Infection with *Golovinomyces cichoracearum*, a powdery mildew fungus, induces the expression of AtSWEET12, whereas another fungal pathogen, *Botrytis cinerea*, induces AtSWEETs: 4, 15, and 17. This differential regulation suggests that each pathogen has its own

specifically tailored mechanism to hijack host carbohydrates (Slewinski 2011). Fungal pathogens also activate their sugar transporters during invasion of the plant. Hexose (HXT1) transporters were specifically expressed in haustoria, specialized fungal feeding structures that occupy living plant cells by invagination of the plant plasma membrane (Voegele et al. 2001). A comprehensive discussion of the role of plant and fungal sugar transporters in symbiotic and pathogenic interactions was recently presented in a review paper by Doidy et al. (2012).

The formation of a sink at the infection site does not always meet the sugar requirement. At the infection sites sugars are taken up by the attacking fungus, while the attacked plant tissues have high substrate requirements for the initiation of defense responses, e.g., the synthesis of pathogenesis-related (PR) proteins, phenylpropanoids, or papillum formation (Strömberg and Brishammar 1993; Morkunas et al. 2005, 2007; Morkunas and Gmerek 2007; Bolton 2009). It has been shown that the induction of defense is cost intensive (Swarbrick et al. 2006). In infected tissues the intensity of respiration processes is increased (Scharte et al. 2005; Morkunas and Bednarski 2008; Morkunas et al. 2008, 2013; Rampitsch and Bykova 2012). Vargas et al. (2012) found an enhanced expression of respiration-related genes at infection sites on maize leaves inoculated with a hemibiotrophic fungus *Colletotrichum graminicola.* Enhanced sugar metabolism causes changes in the qualitative composition of carbohydrates in infested cells while it may also cause a reduction of their level (Morkunas et al. 2007, 2010; Kawakami and Yoshida 2012). For example, during sunflower cotyledon infection by the necrotrophic fungus *Sclerotinia sclerotiorum*, sucrose level was reduced by 100 %, fructose by 85 %, whereas for glucose it was only 20 % (Jobic et al. 2007). Sugar levels decrease in tomato plants after inoculation with *B. cinerea* (Berger et al. 2004; Bonfig et al. 2006). Sugar deficit may lead to sugar starvation in cells, a phenomenon well characterized in terms of metabolism and at the gene expression level (Morkunas et al. 2003). Sugar starvation may initiate the SnRK1 cascade, which causes a reprogramming of cell metabolism to produce energy (Baena-Gonzalez 2010). However, there are very few studies showing a significant role of SnRK1 in the resistance to biotic stresses (Hao et al. 2003; Gissot et al. 2006). Such a reprogramming of primary carbon metabolism may further enhance the expression of defense-related genes and favor the production of secondary compounds with antimicrobial activity (Bolton 2009). A deficit of sugars and energy at the infection site may pertain also to autotrophic tissues, since fungal infection of leaf tissues typically causes a reduced rate of photosynthesis. A decrease in photosynthesis has been reported in compatible interactions with biotrophic fungi, i.e., *Albugo candida*, *Puccinia coronata* and *Blumeria graminis* (Chou et al. 2000; Scholes and Rolfe 1996; Swarbrick et al. 2006) as well as necrotrophic pathogens such as *Botrytis cinerea* (Berger et al. 2004, 2007). The photosynthetic organs of young leaves of sugar beet are particularly sensitive to the infection by *Aphanomyces cochlioides* (oomycetes) (Chołuj and Moliszewska 2012).

The formation of a competitive sink in leaves infested by pathogenic fungi results in a reduced yielding of diseased crops. In experiments conducted on wheat infested by biotrophic pathogens *Puccinia triticina* it was shown that fungal sporulation had a competitive priority for assimilates over grain filling (Bancal et al. 2012). Activity of the sink formed at the infection site may be enhanced by the chemical interference of the fungal pathogen in the regulation of carbon allocation in the plant. For example, many biotrophic fungi such as *Cladosporium fulvum, Blumeria graminis, Pyrenopeziza brassicae* and *Venturia inaequalis* may produce and secrete cytokinins (Robert-Seilaniantz et al. 2007). Accumulation of cytokinins may stimulate host invertase activity, which in turn contributes to an increase in hexose level, the formation of a nutrient sink and a delay of senescence in leaf infection sites (Walters and McRoberts 2006).

Involvement of sugars in plant defense responses during infection with pathogenic fungi

As it was reported by Biemelt and Sonnewalde (2006), various strategies are used to acquire nutrients by necrotrophs, hemibiotrophs and obligate biotrophs, but the initial phases of pathogenesis do not differ fundamentally between them. These early reactions of the attacked plant include an enhanced production of reactive oxygen species (ROS), primarily superoxide ($O_2^{\cdot-}$) and hydrogen peroxide (H_2O_2). Overproduction of ROS through an oxidative burst is part of plant cell reactions to challenge by a pathogen or elicitor. The association of ROS formation and an increased activity of enzymes participating in their metabolism with the induction of defense responses has been demonstrated in many plant–pathogen interactions (Wojtaszek 1997; Morkunas et al. 2004; Morkunas and Bednarski 2008; Lanubilea et al. 2012; Nikraftar et al. 2013). Enhanced ROS production occurs from the moment of recognition of the attack by the plant and in the case of biotrophic pathogens it is concluded with HR programmed death of the attacked cells and cells surrounding the infection site. In in vitro cultured embryo axes of yellow lupine the infection by hemibiotrophic fungus *Fusarium oxysporum* also caused an increase in respiration and ROS production (Morkunas and Bednarski 2008; Morkunas et al. 2008, 2013). We have shown that these processes are much more intensive when embryos are nourished with an

exogenously supplied sucrose. In infected embryos sugar caused an enhanced generation of superoxide anions, which may be one of the causes for the greater resistance. In infested embryos sugar nutrition also caused an increase in the number of mitochondria with less reduced numbers of cristae (Morkunas and Bednarski 2008). Early ROS induction serves an important role in plant response to the attack of fungal pathogens and so does an early nitric oxide burst. As it was reported by Floryszak-Wieczorek et al. (2007), the elimination of the pathogen is determined by the speed and efficiency of early defense responses initiated by the plant and activates a sequence of events. Apart from enhanced ROS production, an early response to pathogen attack may involve enhanced lignification of cell walls (Rampitsch and Bykova 2012). Strengthening of cell walls is one of the most important plant defense mechanisms against infection by fungal pathogens, as it is then more resistant to the activity of hydrolytic enzymes of the attacking pathogen, it limits its access to water and nutrients and decreases the diffusion of its toxins to plant cells. According to Hammerschmidt (1984), an effective inhibition of an infection caused by necrotrophs is possible only if lignin synthesis is induced shortly after inoculation. In embryo axes of lupine supplemented with sugar the content of lignins as early as 24 h after inoculation with *Fusarium oxysporum* was twofold greater than that in non-supplemented embryos, although even the latter showed an increased lignin level after infection (Morkunas and Gmerek 2007). In addition, it was shown that sugar-supplemented embryos had a greater activity of peroxidases covalently and ionically bound with the cell wall (Morkunas et al. 2007). It has also been reported that sucrose and hexoses can play an important role in resistance to fungal pathogens through stimulation of phenylpropanoid metabolism (Forlani 2010; Morkunas et al. 2011; Gibertia et al. 2012). Phenylpropanoid pathway allows plants to produce various secondary metabolites in defense response to infection (Ferri et al. 2009, 2011). These include flavonoids (isoflavonoids in particular), which can play the role of phytoalexins in plants from the family Fabaceae (Andersen and Markham 2006; Bednarek and Osbourn 2009; Naoumkina et al. 2010). Isoflavonoids can be toxic to fungal pathogens, i.e., reduce the development of fungi by inhibiting the growth of their mycelia, spore germination, while they also limit fungal pathogenicity. Their fungicidal action is related to the damage to the plasmalemma, a rapid blockage of cytoplasmic movement, and disorganization of cell organelles. Moreover, they disturb fungal respiration and nutrient uptake (Weidenbörner et al. 1990; Picman et al. 1995). The high level of isoflavonoid glycosides particularly genistein-7-*O*-glucoside and free isoflavonoid aglycones (i.e., genistein, wighteone, and luteone) constituted an important element of resistance of tissues nourished with sucrose against infections. Accumulation of these metabolites was due to both high phenylalanine ammonia-lyase (PAL) activity and higher supply of substrates for their synthesis in tissues with a high level of carbohydrates (Morkunas et al. 2005, 2007). An increase in β-glucosidase activity which hydrolyses isoflavone glucosides and releases free aglycones was found in infected tissues. It has been concluded that sucrose and hexoses (glucose and fructose) in yellow lupine embryo axes, as carbon skeleton donors, may be redirected to secondary metabolism, and consequently, lead to an increased concentration of isoflavonoids, which are important components of the defense system, considering their antimicrobial properties. Expression of the genes of phenylpropanoid pathway enzymes is increased at early stages of infection (Boddu et al. 2006). Confocal microscopy also revealed a strong accumulation of flavonoid end products at the early phase of infection in inoculated embryo axes with high sucrose levels, which was consistent with the expression of flavonoid biosynthetic genes (Morkunas et al. 2011). Mobilization of defense mechanisms in plant cells, e.g., the synthesis of flavonoids, requires a large amount of energy, often at the expense of basic life functions of the plant (Gould and Lister 2006). These compounds serve their defensive roles only when they are found at a specific place, time, and concentration. Douglas (1996) reported that phenylpropanoid biosynthesis requires an effective flow of carbon to phenylalanine synthesis through shikimate and aromatic amino acid pathways. Phenylalanine is a substrate for the reaction catalyzed by PAL, whose product is cinnamic acid (an important link in isoflavonoid biosynthesis) and lignin. Ehness et al. (1997) noted that independently from each other glucose and the fungal elicitor chitosan induced mRNAs level for PAL from *Chenopodium rubrum*. In *Asparagus* a rapid induction of root epidermal cell death and activation of phenyl ammonia-lyase and peroxidase proteins were associated with a restriction of *Fusarium oxysporum* f.sp. *asparagi* growth (He et al. 2001). Application of PAL inhibitors suppressed basal resistance of sugar beet against *Rhizoctonia solani* (Taheri and Tarighi 2011).

Effect of abiotic stress on carbohydrate content and resistance to diseases

The primary principle in physiological experimentation is to change only this one factor which is the subject of the study, leaving all the others unaltered. For this reason in research on the effect of both biotic and abiotic stresses on plants a vast majority of literature data, particularly those published previously, refer to one, strictly specified stress. However, under natural conditions, plants are rarely exposed to only one adverse effect. Already the results of earlier studies based on enzyme activity showed that many mechanisms of plant response to various stresses are similar and even many metabolic pathways initiated in defense against various stresses are identical. However, only the

results of recent research, particularly concerning the regulation of gene expression, have made it possible to develop certain models explaining the relationships between biotic and abiotic stresses (Goellner and Conrath 2008). They also shed some light on the role of sugars in those responses. Some abiotic stresses at the same time reduce the level of sugar and plant resistance to fungal infections. Vidhyasekaran (1974) tested the influence of photoperiod on carbohydrate content in finger millet leaves and their resistance to the disease caused by *Phytophthora infestans*. In plants kept in continuous light, carbohydrate content was twice as high and the disease index was 20-fold lower than in plants kept in continuous darkness. In plants illuminated for 12 h a day, carbohydrate content was 15 % higher than in plants cultured in the dark, but this was sufficient to lower the disease index 12-fold. Based on these results Vidhyasekaran postulated that the beneficial effect of light on plant resistance to pests and disease is effected thanks to an increase in sugar levels in tissues. However, the latest studies showed that the mechanism of enhancing plant resistance by light is much more complex. It was shown that a significant role in the stimulation of resistance to fungal infection is played by phytochromes interacting with phytohormones (Roberts and Paul 2006; Xie et al. 2011; Cerrudo et al. 2012). Recently several reviews have been published, thoroughly describing the latest discoveries concerning the effect of light on plant resistance to pathogens, including also fungal pathogens (Kazan and Manners 2011; Ballaré et al. 2012; Kangasjärvi et al. 2012; Svyatyna and Riemann 2012).

Excessive nitrogen fertilization results in a decreased carbohydrate level in cultivated plants and it also may be a reason for the limited resistance to some fungal diseases (Yoshida et al. 2008; Huber and Thompson 2007). Rice blast (Kürschner et al. 1992; Long et al. 2000) is the best-known example of such a disease. Among wheat diseases, powdery mildew (Last 1953; Teich et al. 1987), leaf rust (Howard et al. 1994; Teich et al. 1987), stripe rust or yellow rust (Ash and Brown 1991; Danial and Parlevliet 1995) and several other diseases (Howard et al. 1994) have been reported to increase in severity as the rate of nitrogen application is increased. Transcription analyses show that sugar and inorganic nitrogen act as both metabolites and signaling molecules. Price et al. (2004) reported that cluster analysis revealed a significant interaction between glucose and nitrogen in regulating gene expression, because glucose can modulate the effects of nitrogen and vice versa.

However, acclimation processes initiated in plants by abiotic stresses may also have a positive effect on their resistance to biotic stresses. Plants exposed to one stress may become more tolerant to another. This phenomenon, called cross-tolerance, has been known for many years (Płażek and Żur 2003). An example in this respect may be provided by plant acclimation to cold conditions. In plants kept in the cold at temperatures of 0–5 °C several changes are observed, enhancing their resistance to freezing. Plants acclimated to cold conditions show a greater resistance to fungal pathogens (Rapacz et al. 2000; Płażek and Żur 2003). Cellular changes associated with the acquisition of tolerance to chilling and/or freezing include the accumulation of sugar or compatible solutes, changes in membrane composition and synthesis of dehydrin-like proteins (Ruelland and Zachowski 2010). One of the older hypotheses explaining a greater resistance of acclimated plants to pathogens assumes that the cause is connected with the osmotic action of accumulated sugars and osmotically active proteins. According to Tronsmo (1986), a reduced availability of water may partly explain the increased resistance to fungal pathogens in grasses after hardening. However, during the dehardening process a rapid loss of cold resistance is observed in plants while their resistance to pathogens is maintained over a longer period (Rapacz et al. 2000). At present it is known that the process of acclimation to low temperatures is highly complex and includes many changes within cells, both at the molecular and structural levels (see reviews by Chinnusamy et al. 2006; Ruelland and Zachowski 2010). Another example of the positive effect of plant acclimation to abiotic stress on the increase in their resistance to the attack of a fungal pathogen may be connected with adaptation to NaCl (Kuźniak et al. 2010, 2011; Libik-Konieczny et al. 2011, 2012). The positive effect of certain abiotic stresses on plant resistance to biotic stresses may be viewed as their role of defense priming in plants (Goellner and Conrath 2008). "Defence priming is a unique physiological state that can be induced by molecular patterns of microbes or plants, pathogen-derived effectors, beneficial microbes, and treatment with some natural or synthetic compounds and wounding. Primed plants show fast and/or strong activation of defence responses when subsequently challenged by microbes, insects, or abiotic stress" (Conrath 2011). According to the above definition, priming is caused by a wide range of agents, including also the proposed sucrose (Gómez-Ariza et al. 2007; Bolouri-Moghaddam and Van den Ende 2012). Exogenously applied sucrose induced accumulation of the transcript of PR proteins (PR-2 and PR-5) in *Arabidopsis thaliana* (Thibaud et al. 2004). The use of mutants and transgenic plants of *A. thaliana* indicated that salicylic acid (SA) was involved in the sugar-dependent activation of these PR protein-coding genes (Thibaud et al. 2004). Priming is a part of both systemic acquired resistance (SAR) and induced systemic resistance (ISR) (Goellner and Conrath 2008). Whereas SAR is predominantly effective against biotrophic pathogens that are sensitive to SA-dependent defenses, ISR was shown to be effective against pathogens and insects that are sensitive to JA- and ET-dependent defenses (Pieterse et al. 2009).

Phytohormone abscisic acid (ABA) is commonly associated with plant development and abiotic stress, but its role in biotic stress is becoming increasingly evident (see reviews by Asselbergh et al. 2008; Wasilewska et al. 2008; Ton et al. 2009; Łaźniewska et al. 2010; Cao et al. 2011; Robert-Seilaniantz et al. 2011). ABA supports JA-dependent defense against necrotrophic pathogens, while it is an antagonist of SA-dependent defenses and SAR (Pieterse et al. 2009). It is another example of interactions of signaling pathways responsible for defense responses of plants to biotic and abiotic stresses.

Conclusions and future directions

Although a high-sugar level does not always boost the immune system in plants, since we know pathosystems, in which a high-sugar level stimulates the development of the pathogenic fungi (the so-called high-sugar diseases—Horsfall and Diamond 1957), in most plant species, particularly those important in agriculture, sugar enhances resistance. As it was mentioned earlier, sugar transporters are key elements, necessary for the formation of the secondary sink at the site of fungal pathogen invasion. The information that certain sugars may act as priming agents may also be useful in programs to generate stress-resistant cultivars. Moreover, certain sugars may prove an effective substitute to toxic pesticides.

Author contribution Doctor habilitatus Iwona Morkunas—preparation of the following chapters in the review: involvement of sugars in plant immune system, fungal pathogen attack causes the formation of a new sink at invasion site, involvement of sugars in plant defense responses during infection with pathogenic fungi. Preparation of Figure 1. Professor Lech Ratajczak- preparation of the following chapters in the review Abstract, Introduction, Sugar sensing and signaling, Effect of abiotic stress on carbohydrate content and resistance to diseases.

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