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REVIEW

Inducible Lectins and Plant Resistance to Pathogens and Abiotic Stress

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Abstract—Lectin concentration (activity) increases in plant tissues upon infection by pathogens, in response to abiotic stress, as well as during growth and development of tissues. Such a broad range of events accompanied by accumulation of lectins is indicative of their involvement in regulation of integral processes in plant cells. Data concerning the role of lectins in regulation of oxidative stress and stress-induced cytoskeleton rearrangements are presented.

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Lectins comprise a miscellaneous group of proteins capable of reversible and specific binding ("recognition") of carbohydrate residues of different chemical nature. An obligatory condition is the presence of one or several noncatalytic carbohydrate-binding domains along with additional domains of different, in particular, catalytic nature [1].

Differences in lectin structure and carbohydrate specificity suggest differences in their functional load [1]. The known and proposed functions of phytohemagglutinin can be divided into structural (packaging of storage glycoproteins, membrane glycoproteins, and enzyme complexes), transport (transport of mono-, di-, and polysaccharides and of protein subunits), regulatory (blocking oligosaccharide groups, regulation of enzyme-glycoprotein and enzyme-lectin activities), and information (pathogen and elicitor recognition and immobilization).

Although lectins are constitutively present in tissues of healthy organisms, their content can increase many times in response to infection as well as to different stress effects. Since lectins of healthy plants and those induced by infection and other effects are identical in most cases, a question arises concerning the extent of their functional similarity in an intact organism and one under stress.

Abbreviations: ABA) abscisic acid; DMSO) dimethyl sulfoxide; IAA) indoleacetic acid; TMV) tobacco mosaic virus; WGA) wheat germ agglutinin.

Numerous experimental data indicative of a possible protective role of phytohemagglutinins are present in a number of reviews [1-6]. However, most specific mechanisms of the protective effects of lectins still remain unknown.

In recent years, lectin domains have been found within receptor kinases. The extracellular domain of plant lectin-like receptor kinases (LecRK) resembles soluble lectins of legumes and *Arabidopsis* [7-9] or chitinase [10]. Receptor kinases having a lectin domain are also known in animals. In particular, tyrosine kinase evidently plays a role in the integration of *Hydra vulgaris* cells [11].

It is also known that receptor kinases of different classes can be induced by wounding, pathogens, oligogalacturonides, salicylic acid, and oxidative stress [12-15]. Expression of some lectin-like kinase genes also increases in the case of infection, natural ageing, wounding, and treatment by elicitors [10, 16-18]. Expression of LecRK localized in the plasmalemma of *Medicago truncatula* root cells increases upon nitrogen starvation and decreases in response to infection by rhizobia or treatment by Nod-factor [19]. Tissue-specific expression of a number of lectin genes occurs upon plant infection by a symbiotic bacterium [20]. In addition, inoculation of Rhizobia induces changes in the localization of the Nod-factor lectin receptor [21].

Lectins of receptor complexes are present in cells in small amounts. Such proteins might be responsible for processes of specific recognition both inside and outside

the cell [1]. However, along with receptor kinases, classical lectins can also be induced in response to different factors. The main features of the latter are, on one hand, the determinable specificity towards monosaccharides in the case of a small number of isolectin forms, and on the other hand, their relatively high content in plant tissues. As far as specific recognition is concerned, the presence of a large number of copies of relatively "simple" proteins suggests the existence of functions different from those of the above-described lectin-like receptor kinases. The increase in their activity (concentration) in plant tissues is fairly high upon infection by pathogens, under abiotic stresses, as well as during growth and development of these tissues and can be measured by physiological and biochemical methods. Possible protective and regulatory functions of induced lectins of this group are considered in this review.

Increase in the content of lectins under biotic and abiotic stress was observed in cells of living organisms of different taxonomic groups. In particular, the content of galactose/N-acetylgalactosamine-specific lectin of *Entamoeba histolytica* (the pathogen of amebic dysentery), playing an important role in pathogenesis and parasite differentiation, significantly increased under heat stress [22]. The content of lipopolysaccharide-specific immunolectin-2 in plasma of *Manduca sexta* larvae increased 3-4 times after injection of a Gram-negative bacterium or lipopolysaccharide [23].

INDUCTION BY ABIOTIC STRESS

Some experimental data concerning changes in the plant lectin complex under abiotic stress are given in Table 1.

The accumulation of lectins during wounding stress is of interest [24]. It was shown that plasmalemma of the wound periderm not only contains more binding sites for peptidoglycan from the cell wall of phytophthorosis (late blight of potato) pathogen, but it more actively binds the preparation obtained from avirulent strains of this pathogen.

Although most works were carried out with a chitooligosaccharide-specific wheat lectin (wheat germ agglutinin, WGA), lectins of other plant taxons exhibiting different carbohydrate specificity also accumulate in response to various abiotic stresses. The content of N-acetylgalactosamine-specific lectin of *Dolichos biflorus* (leguminous) increased during heat stress [25]. Incubation of developing bean cotyledons at elevated temperature enhanced the synthesis of phytohemagglutinin but inhibited transport of the latter into protein bodies [26]. Mannose-specific lectin of rice was induced under abiotic stress; in this case, two out of four rice isolectins had N-terminal sequence specific for drought-induced proteins [27]. Two isolectins were isolated from rice plants under salt stress [28].

Lectin accumulation under stress is regulated at different levels [6]. The rate of WGA synthesis under salt stress, determined by ^{35}S incorporation into a particular protein, increased transiently (the maximum was noted 3 h after beginning the treatment) [29]. However, the presence in the plant of a pool of specific mRNA and WGA precursors is evidently also able to provide for independent of *de novo* protein synthesis increase in lectin content at early stages of the stress-induced response [6]. Under salt stress, this resulted in retention of WGA induction for 4 and 8.5 h in plants treated by inhibitors of protein (cycloheximide) and mRNA (cordycepin) syntheses, respectively. Translation-independent accumulation of WGA also occurred upon treatment with abscisic acid (ABA) [30].

In many cases, the abiotic stress effect produced transient lectin content (activity) peaks in the plant or in certain organs or organelles. A well-studied reaction of lectins to stress is their increase in response to low temperatures and cold hardening. Low but positive temperature (2°C) induced a transient (5 h) activity maximum of cellular organelle phytohemagglutinins in the meristem tillering node of a frost-resistant wheat variety [31]. Longer transient maxima and minima of lectin content were observed during prolonged exposure accompanied by cold adaptation and increased frost resistance. The maximum of phytohemagglutinin activity in the cell wall fraction from leaves of three-day-old seedlings falls on the third to fifth days. However, the content of lectins in coleoptiles continued to decrease during the whole period of observation (7 days) [32]. The dynamics of phytohemagglutinin activities was different for different cell compartments [33]. Already after one day of incubation in a cold chamber, the lectin content in chloroplasts decreased and then increased in 7 days to a level exceeding the non-hardened control. The peak of lectin concentrations in outer nuclear membranes and their minimum in the microsome fraction were detected on the third day.

Addition of chlorpromazine (antagonist of calmodulin) and verapamil (calcium channel blocker) lowered the basal level of cytoplasmic lectins in non-hardened roots of wheat plants and completely inhibited their induction during cold hardening [34], although the effect on the content of the cell wall lectins was insignificant. This is indicative of a role of the calcium signal system in regulation of lectin content in the soluble fraction.

LECTINS: CHANGES DURING INFECTION AND IN RESPONSE TO RESISTANCE INDUCERS

Protection against pathogens is one of the most important functions attributed to lectins [1, 6]. It is natural to expect that the content of protective compounds, including lectins, should increase upon infection by

Table 1. Changes in lectins under abiotic stresses

Stress	Plant, organ (organelle)	Method of lectin determination	Time, days		Source
			I	II	
Cold hardening	wheat	HA			
	leaves		3-5 (+)		[32]
	coleoptiles		3 (+)	7 (–)	[33]
	nuclei		1 (–)		
	plastids		3 (–)		
	microsomes		5 (+)		
	cell walls		8* (+)		
	organelles			24* (+)	[31]
	leaves		3 (+)		[135]
	roots		1 (+)		
	roots				
	soluble lectins			7 (+)	[136]
	bound lectins			0.25-7 (+)	
Heat	callus	EIA		14-18* (+)	[59]
	Dolichos biflorus, suspension culture	IBE		(+)	[25]
Osmotic stress, heat, salt	wheat	–	4* (+)		[137]
Drought	»	–		(+)	[92]
Drought, osmotic stress	wheat, roots	EIA	5* (+)	3* (+)	[29]
			11* (+)		[138]
			7* (+)		[60]
Salt	rice	–		(+)	[27, 28]

Note: Here and in Tables 2 and 3 the following abbreviations are used. EIA) enzyme immunoassay; HA) hemagglutination; IBE) immunoblots of electrophoregrams; GE) gene expression; EP) electrophoresis; I) extremum time for transient maxima (+) and minima (–) of lectin concentrations; II) increasing interval of significant monotonous change during observations or the time of single measurements.

* Here and in Tables 2 and 3, time in hours.

pathogens. In fact, the increase in lectin contents (usually measured by the level of hemagglutinating activity) in response to inoculation of various nature pathogens (bacteria, fungi, mycoplasms, viruses, nematodes) is one of the well-studied reactions of a plant. Some experimental results are shown in Table 2.

A feature of this reaction in a number of pathosystems is a higher lectin induction upon infection of resistant plants. Thus, avirulent strains of *Pseudomonas pisi* induced in tobacco plants a lectin that agglutinates these bacteria [35]. Accumulation of lectins was observed after inoculation of tobacco mosaic virus (TMV) into hypersensitive varieties of *Nicotiana tabacum* and *Datura metel* [36]. No such increase was observed in plants with systemic necrosis or upon systemic infection of susceptible varieties by cucumber mosaic virus. Tobacco vacuolar protein, interacting with chitin due to the presence of a chitin-binding hevein domain, was induced by TMV in the hypersensitive variety as well as by wounding [37]. In our studies an increase in hemagglutinating activity in the readily soluble protein fraction was registered approximately the next day after tobacco and wild type potato infection by viruses, inducing hypersensitive reaction, and no increase was observed upon inoculation of the pathogen causing systemic infection in a susceptible plant [38, 39].

The advantageous increase in lectin concentration (activity) in the case of inoculation of a pathogen into a resistant plant correlates with data on their accumulation upon treatment by resistance inducers (Table 3). The

Table 2. Changes in lectins upon infection by pathogens

Inducer	Plant, organ	Method of lectin determination	Time, days		Source
			I	II	
P. pisi	tobacco	–		(+)	[35]
TMV (hypersensitivity)	tobacco, thornapple, leaves	HA		1 (+)	[36]
	tobacco, leaves	EP		7 (+)	[37]
	leaves	HA		1 (+)	[39]
Potato virus Y (PVY, hypersensitivity) Potato virus X (PVX, susceptibility)	*S. chacoense*, leaves		22* (+)		[38]
Septoriosis	wheat, shoot	EIA		6-9 (+)	[51]
	developing caryopses		2-3 (+)		[47]
Root rot	*Ttiticum turgidum*			(+)	[52]
Mycoplasm *Acholeplasma laidlawii*	wheat	HA			
	leaves		7* (+)		[139]
	roots		1 (+)		
Nematodes	oat	IBE		4 (+)	[140]

increase in the content of lectin proteins was noted in response to a fungal elicitor [40], double-stranded RNA [38], interferon [39], salicylic acid [41], and hydrogen peroxide [42]. Synthesis of mRNA of the *Arabidopsis* hevein-like protein was induced by viral infection and ethylene and to lower extent by salicylic and 2,6-dichloroisonicotinic acids [43].

In many cases, the accumulation was transient. Studying the dynamics of lectin in *Solanum chacoense* leaves showed the presence of a hemagglutinating activity maximum in the soluble protein fraction approximately a day after treatment by double-stranded RNA or inoculation of potato virus Y (PVY), inducing hypersensitive response of the host plant [38]. However, already on the third day activity of phytohemagglutinins in this fraction was lower than in control. The amount of WGA antigen in the roots of wheat seedlings reached a maximum 8 h after treatment with 24-epi-brassinolide [44] and 2 h after submersion into a hydrogen peroxide solution [42]. Salicylic acid and its analog 2,6-dichloroisonicotinic acid induced enhanced expression of barley jacalin-like lectin, which is constitutively expressed in seeds and coleoptiles 4 h after treatment [45]. Later transient maxima (3-4 days) of lectin content were observed in barley seedlings in response to salicylic acid and infection by *Fusarium* sp. [46], as well as in developing wheat caryopses infected with septoriosis [47].

The shape of lectin accumulation curves upon induction by pathogens or immunomodulators can differ in different protein fractions, like that under abiotic stress. The extremum positions and intensities changed when different inducer concentrations were used. Treatment of *Nicotiana glutinosa* leaves with human interferon resulted in differently shaped dynamic curves, usually the lectin content increased in either 8 or 72 h after treatment [39].

The study of changes in lectin concentrations (phytohemagglutinin activities) in extracts of tobacco leaf discs and potato tubers treated by polysaccharides (chitosan, glucomannan, dextran sulfate), enzymes (cellulase, pectinase), and monosaccharides (glucose, glucosamine) showed that these substances to variable degree induced changes in lectin contents [48]. The time of emergence of the lectin content maximum depended on the polysaccharide species and concentration. The content of membrane lectins in tobacco leaf discs usually decreased significantly during the initial period after treatment (1-2 days) and later increased (2-4 days). On

Table 3. Changes in lectins in response to resistance inducers

Inducer	Plant, organ	Method of lectin determination	Time, days		Source
			I	II	
Fungal elicitor	wheat, roots	EIA		(+)	[40]
Double-stranded RNA	*S. chacoense*, leaves	HA	22* (+)		[38]
Bisol, baytan	wheat				[141]
α-Interferon	tobacco, leaves	HA	1 (+)	3 (+)	[39]
Salicylic acid	wheat	–		(+)	[142]
	barley	HA GE	3–4 (+)	 4* (+)	[46] [45]
	maize, seedlings	HA		(+)	[143]
24-Epi-brassinolide	wheat, roots	EIA	8* (+)	 14* (+)	[44] [60]
24-Epi-brassinolide (seed treatment)	wheat, roots	EIA	3–4 (+)		[144]
Chitosan, glucomannan	tobacco, leaf discs	HA		4 (+)	[48]
Pectinase, cellulase, glucomannan	potato, tubers	HA	2 (+)	4 (+)	[48]
Hydrogen peroxide	wheat, roots	EIA	2* (+)		[42]

the whole, in potato tubers lectin accumulation was prevalent on treatment by inducers. Variant treatments with cell wall degrading enzymes (pectinase and cellulase) were characterized by increase during the whole period of observation (5 days). The enzyme-stimulated accumulation of lectins is probably caused by distortion of the cell wall integrity and formation of endogenous elicitors of polysaccharide or oligosaccharide nature, which resembles processes accompanying the so-called cell wall stress described in yeasts [49].

Fairly complex changes in lectin content (phytohemagglutinin activity) in the case of hypersensitive reaction is observed not only in time but also in the space around virus-induced necrosis [50]. Increase in hemagglutinin activity in the perinecrotic zone compared to that in remote tissues was observed in the fraction of soluble and weakly membrane-bound proteins of the TMV-infected tobacco plant. This difference became less pronounced with time, and at a distance of 15 mm from the center of necrosis it even changed sign. In contrast, in the membrane protein fraction remote from the necrosis center by 5-9 mm, the decrease in phytohemagglutinin activities in at early times (2 days) was replaced by increased activity in 4 days. Compared to uninfected leaves, in tissues remote from necrosis a decrease in activity at early stages and increase during the following period were observed in the protein fraction weakly bound to the membrane. Increased content of lectins was registered in the membrane protein fraction in this zone during both periods. Usually results obtained in different experiments vary much depending on the object, conditions, and other factors. These experiments show that this can be due to the complicated time dynamics of the process as well as to effects of spatial distribution of lectins around the lesion focus.

In all the above-described experiments, accumulation of lectins was closely connected with the induction of resistance in the host plant. However, increase in lectin content was also detected upon infection of susceptible plants. Thus, the infection of wheat by the septoriosis pathogen resulted in increase in lectin content by the ninth day [51]. In this case, the increase in a susceptible variety was more pronounced.

Infection by root rot stimulated increase in lectin contents in wheat leaf bases, and this increase correlated with the degree of infection [52]. It was shown that potato tubers infected with potato virus X (PVX) contain 60% more lectin than healthy ones [53].

A correlation of lectin contents with disease severity can be observed in susceptible plants at later stages of infection. Thus, the dynamics of lectin content is probably characterized by an early transient peak and later pro-

longed quantitative changes. In this case, the first change in most cases correlates with the resistance of the plant, and the later changes are indicative of disease symptoms, i.e. they correlate with susceptibility. One of peaks or both together can be revealed in each experiment. Thus, in the case of wheat infection by septoriosis, in the more resistant variety along with the main peak of lectin concentrations on the ninth day after infection, there was the tendency for accumulation of lectin on the third day, which was absent from the susceptible variety [51]. It follows from Table 1 that at least two types of lectin content dynamics can be also observed under abiotic stress.

LECTINS AND PHYTOHORMONES

The transition of a plant to a state of resistance, as well as many other processes associated with stress, often correlates with immunologically important shifts in the plant hormonal system. This especially concerns ABA, which is even called the stress hormone.

Numerous experimental data are indicative of the important role of ABA in induction of plant protective reactions. The ABA content significantly increases in response to very different stresses of biotic and abiotic nature [54]. It was shown that exogenous ABA stimulates increase in WGA content in the plant [29, 55-58]. Under osmotic stress (0.1-1.0 M mannitol) the ABA peak at 2 h after beginning of treatment preceded the WGA peak at 5 h [29]. Both peaks were transient and at 5 h (ABA) and at 10 h (WGA) the amounts of both compounds returned to control values. The increase in ABA content also preceded that of WGA under heat stress [59] and in response to NaCl [60]. There was no induction of WGA by osmotic stress in the presence of fluridon, which decreases the endogenous ABA content via inhibition of synthesis of carotinoids [29]. Data on ABA and WGA concentration kinetics as well as experiments with the inhibitor of endogenous ABA indicated the existence of the cause-and-effect relationship between the stress-induced ABA accumulation and increased lectin content [29]. However, the WGA content can be influenced by different factors as well, owing to which even for abiotic stresses correlation between the ABA and lectin contents is not always complete. Thus, unlike ABA accumulation, WGA accumulation caused by water stress depended on the stage of development and variety [61, 62]. In some works, no correlation between lectin and ABA contents was observed in infected plants [6, 51].

Detailed investigation of kinetics of WGA induction in wheat plants by some exogenous phytohormones has shown that the effect of indoleacetic acid (IAA) and 6-benzyl-aminopurine (BAP) can be mediated by transient increase in ABA content [58]. Unlike cytokinins and IAA, the increase in lectin content in response to gibberellin and 24-epi-brassinolide was not accompanied by an increase in ABA content, and it is probably an ABA-independent pathway of regulation of this protein [58].

The variability of lectin protein regulation pathways can also be revealed in a somewhat different form. The chitooligomer-specific lectin Nictaba was isolated from tobacco plants [63]. This lectin was absent from untreated tissues, but the protein and its mRNA were accumulated in tobacco leaves after treatment by jasmonic acid methyl ester at optimal concentration. Lectin was localized exclusively in the leaf cell cytoplasm and nuclei, but not in chloroplasts or vacuoles. Salicylic acid, etephon, ABA, gibberellic acid, wounding, and osmotic stress did not induce the lectin. Lectin induction was not accompanied by emergence of PR-proteins or proteinase inhibitor. Purified lectin did not inhibit growth of *Neurospora crassa*, *Fusarium culmorum*, or *Botrytis cinerea*. Thus, Nictaba fundamentally differs from previously described tobacco lectins by the character of induction, intracellular localization, and anti-pathogenic activity [36-39]. Similar nucleotide sequences are found in genomes of a number of members of the Solanaceae family, grasses, and legumes, which suggests that the affiliation of Nictaba is with a new lectin family. Cytoplasmic and nuclear proteins carrying an O-bound residue of N-acetylglucosamine can serve as receptors of this lectin. The function of this lectin is supposed to be the inhibition of growth and metabolic activity without any connection with protective reactions to stress.

The difference in regulatory pathways is probably indicative of different functional activities of lectins. Thus, gibberellin controls daily changes in expression of the *Cucumis sativus* xylem lectin [64]. Treatment with IAA and brassinolides stimulates increase in level of *Cicer arietinum* lectin mRNA. Like WGA, this lectin is excreted, but its amount decreases in response to water stress [65].

There are a number of quite different reactions of lectin proteins to treatment by hormones, especially by jasmonates and ABA. Thus, treatment with methyl jasmonate increased lectin content in callus culture of *Helianthus tuberosus* [66]. Transcripts of the *Brassica napus* mannose-specific lectin were accumulated in plant leaves in response to wounding and treatment with jasmonic acid, ABA, and ethylene precursor. Salicylic acid inhibited this response [67]. Induction of synthesis of the *Griffonia simplicifolia* acetylglucosamine-binding lectin occurred on treatment with methyl jasmonate, while inhibition was stimulated by ethylene and salicylic acid [68]. However, the barley lectin gene *Lem2* was expressed in response to salicylic acid but not to jasmonate, whereas drought and ABA exhibited an inhibitory effect [45]. Inhibition of lectin gene expression together with a number of enzyme genes was detected in etiolated cucumber seed-lobes within 2 h after treatment with cytokinins [69].

Lectins themselves are apparently able to exhibit regulatory effect on metabolism of phytohormones. This was supposed as early as 1982 [70]. Lectins are known to

interact directly with IAA [71]. A number of lectin proteins contain a binding site for hydrophobic compounds that can interact with adenine and related compounds, including many cytokinins [72]. Since lectins occur in certain plant tissues and organs in high concentrations, which are especially specific of seed tissues, even in the case of relatively low affinity to cytokinins the bulk of the phytohormone pool can appear to be bound to lectins, then playing the role of buffer storage [73]. It is interesting that in the presence of some substances the hydrophobic binding site affinity to cytokinins may significantly increase, even exceeding affinity to adenine [73], which evidently gives additional possibilities in the regulation of hormone metabolism by lectins.

LECTINS IN GROWTH PROCESSES

Probably interaction with the hormone system determines the role of lectins in growth and development [74]. Maximal lectin content was found in rapidly growing adventitious roots and leaf base of wheat seedlings [75]. A possible mechanism is the involvement of lectins in inhibition of auxin-dependent catabolism of cell wall polysaccharides and growth by stretching [76, 77]. In the case of compatible pollination in petunia and onion plants, an increase in the content of lectins in the pistil sporophyte tissues was observed along with change in their carbohydrate specificity [78]. Premature germination of immature wheat embryos in *in vitro* culture was accompanied by cessation of WGA synthesis, and reversible inhibition of this germination by ABA restored WGA synthesis even to even more than in intact embryos [55]. Regular changes in lectin localization were observed upon changes in cell differentiation: in root meristem lectin was localized in vacuoles, but in growing roots it was localized exclusively in cell walls [79]. Interruption of the quiescence of *H. tuberosus* in culture *in vitro* was accompanied by redistribution of lectins: their content in parenchymatous tissue decreased, while in buds it increased [80].

A lectin, specific for *D. biflorus* shoots and leaves was also synthesized in callus tissues, and its content correlated with growth rate and viridescence [81]. The seed lectin, usually not found in cultivated tissues of *Psophocarpus tetragonolobus*, appeared upon differentiation [82]. The amount of lectin in morphogenic wheat callus exceeded that in non-morphogenic callus [83]. Increased WGA content was characteristic of morphogenic compared to non-morphogenic callus, morphogenic compared to non-morphogenic regions within a callus, as well as rapidly growing non-morphogenic callus compared to slowly growing callus [84]. Cultivation of non-morphogenic callus in the presence of WGA did not result in enhanced differentiation. The lectin content in embryogenic callus is probably the result of morphogenesis rather than its cause [74].

The above-described tobacco lectin (Nictaba) is evidently involved in regulation of growth but is not associated with the effect of protective phytohormones or with emergence of resistance markers [63]. Obviously, reprogramming of plant metabolism to the resistance state under stress should also be accompanied by changes in the growth and development indexes. In this case, even lectins having rather indirect relationship with their own protective reactions are able to play an important role in regulation of stress response, its coordination with vegetative growth, etc.

ABA-dependent changes in growth under stress probably involve lectins. Preliminary treatment of wheat roots with WGA prevented inhibition of growth indexes (mitotic index and the cell area in the extension zone) by salt stress, while treatment after stress stimulated recovery of growth [85]. In this case, WGA prevented sharp changes in ABA and IAA contents in response to stress in the first case and accelerated IAA accumulation in the second. A certain analogy between the increase in mitotic index during treatment by exogenous WGA [85], obviously exhibiting its activity first of all at the cell wall level, and simultaneous increase of mitotic index and content of cell wall lectin during treatment by oryzalin, causing cytoskeleton depolymerization, should be noted [86].

Thus, there is an obvious relationship between growth and lectin content, but experimentally noted correlations are ambiguous and can have both negative and positive sign. The increase in mitotic index in response to exogenous WGA under stress is indicative of possible antagonism of WGA with ABA, exhibiting mainly inhibition of growth. The role of ABA as an inducer of lectin synthesis under stress was determined quite reliably. The combination of the above-described data suggests that lectin function is not only assurance of the physiological effect of ABA under stress, but rather its inhibition and restriction in time.

All these data show that the lectin accumulation is a rather complicated event; it depends on organ, position relative to necrosis, sampling time, and protein fraction and can correlate both with resistance and symptom severity. Quite similar results can be obtained by analysis of changes in lectin contents under abiotic stress or in response to treatment by phytohormones and resistance inducers. However, there is something in common in all these situations: any stress and any protective reaction is accompanied by increased formation of active forms of oxygen, i.e. by oxidative stress or "oxidative burst". Oxidative stress also often accompanies growth and changes in plant development stages.

LECTINS AND OXIDATIVE STRESS

The role of reactive oxygen species is now intensively studied in connection with resistance to pathogens of

plants and other living organisms. In this case, hydrogen peroxide as a reactive oxygen species in plant tissues can be directly used for synthesis of the pathogen-induced lignin and transverse cross-link of proline-rich structural proteins, and it also exhibits antimicrobial properties. As a secondary messenger, hydrogen peroxide is involved in functioning of the NADPH-oxidase signal system that plays an important role in regulation of plant protective reactions [87] and participates in induction of syntheses of phytoalexins [88] and pathogenesis-related proteins [89]. Usually in response to elicitors, transient increase in hydrogen peroxide synthesis and release into the environment (like that by plant roots) are observed. The amount of this substance increases under oxidative stress, accompanying any unfavorable effect, and often is an immediate cause of the emergence of symptoms.

The ability of plant tissue to recognize elicitor and synthesize hydrogen peroxide appears about 18 h after wounding of the explant epidermis [90]. If preliminary incubation was carried out in the presence of salicylic or 2,6-dichloroisonicotinic acids, known as resistance inducers, then the use of elicitors caused a significantly more intense response. In contrast, treatment with lectin inhibited elicitor-induced (chitooligosaccharide) synthesis of hydrogen peroxide [91]. It was shown in our experiments [42] that treatment with exogenous hydrogen peroxide can stimulate an increase in lectin amount. Our experimental system modeled quite well the transient increase in the concentration of the compound in the zone surrounding wheat germ roots. It is supposed that lectin regulation of hydrogen peroxide synthesis can be involved in plant protection against self-damage in the case of excessive production of reactive oxygen species in an infected plant [91]. The role of the stress-induced WGA in the protection of cells against damaging effects of free radicals was also discussed in [92]. Thus, the increase in lectin content can serve as a mechanism shortening "oxidative burst" duration and thus restriction in time immune reactions, i.e. lectins may serve as suppressors of information transfer via the channel of the superoxide-synthetase signal system. The inhibitory effect of lectins is in some sense directed opposite to the effect of salicylic acid stimulating the emergence of cell competence towards recognition of elicitors and synthesis of hydrogen peroxide [90].

It seems that the increase in lectin contents observed by many authors in response to different biotic and abiotic stresses is mediated by enhanced synthesis of hydrogen peroxide and other reactive oxygen species. This hypothesis can explain both the data on correlation of lectin induction with protective reactions of a resistant plant and experiments showing the existence of a clear relationship between lectin concentrations and the extent of damage to a susceptible plant. In fact, the necessity of protection against oxidative stress can emerge both in a resistant plant (at early stages of induction of protective reaction) and due to metabolic changes in a susceptible plant at later stages of pathogenesis.

LECTINS AND CYTOSKELETON

Another integral event in vital activity of cells (like motility of organelles, cell division, growth, and development that define cell shape and its interaction with the biotic environment) is cytoskeletal rearrangement [93]. Cytoskeleton is a quite mobile structure, and in experiments *in vitro* the level of dynamic instability of plant microtubules is much higher than that in animals [94]. In a number of experimental systems using animal cells, treatment with exogenous lectins influenced the state of cytoskeletal elements or induced cytoskeleton-associated changes in cell shape [95-98].

Much information has accumulated concerning the role of cytoskeleton in pathogenesis in plants and animals as well as upon interaction with symbiotic microorganisms. It is assumed that the main principles of the regulation of cytoskeletal function are conservative in eukaryotic cells [99]. Since successful infection requires regulation of adsorption, penetration into the host cell, phagocytosis, intra- and intercellular translocations, and other functions, the presence in a pathogen of proteins interacting with regulatory elements of the host cytoskeleton is a prerequisite for successful pathogenesis [100].

Microfilaments of cytoskeleton interact with the cytoplasmic domain of plasmalemma transmembrane proteins via a complex of linker proteins [101]. The carbohydrate moiety of the cytoskeleton-bound proteins involved in regulation of its state can be the site for interaction with cytoplasmic and membrane lectins. Thus, the change in shape of phytolectin-treated erythrocytes is attributed to interaction of the latter with the outer carbohydrate moiety of glycophorin, the transmembrane protein of erythrocyte membrane whose cytoplasmic domain interacts with cytoskeleton [97].

Many proteins bound to cytoskeletal elements have been found to be modified by N-acetylglucosamine residues [102-104]. The chitooligosaccharide-specific lectins, in particular WGA, are able to interact with N-acetylglucosamine residues of glycoconjugates and can be used for detection of modified proteins. It is assumed that such modification is reversible and very mobile; it carries out regulatory functions like phosphorylation. It was found in many cytoplasmic and nuclear proteins [105]. For example, cytoskeletal changes in neuron culture caused by colchicine and cytochalasin are followed by change in topography of the WGA-interacting sites, i.e. those containing N-acetylglucosamine [106].

Acetylglucosylation and phosphorylation are reciprocal and influence the resistance of proteins to proteases and the state of the cytoskeleton; disturbance of these processes results in severe diseases. Thus, tau-proteins

associated with neuronal cytoskeleton in the norm contain over four monosaccharide residues per protein molecule, which are attached in the sites responsible for interaction with cytoskeleton [103]. In Alzheimer's syndrome, these sites are hyperphosphorylated, whereas the tau-proteins appear to be incapable of binding microtubules. It is difficult to imagine that regulatory possibilities caused by the presence of a number of lectins specific towards this monosaccharide residue could remain unused.

There are examples of direct interactions of lectins with cytoskeletal proteins. The *E. histolytica* lectin specific to Gal-GalNAc and serving as the virulence factor of the parasite contains a spectrin-binding site in the cytoplasmic domain [107]. Comitin, the mannose-specific lectin of *Dictyostelium discoideum*, contains an actin-binding site and is probably involved in interaction of the Golgi membrane apparatus with cytoskeleton [108].

The association of selectin with actin cytoskeleton plays an important role in the interaction of vascular endothelial cells and leukocytes, which define the direction of the latter to the focus of inflammation [109]. In this case, transmembrane E-selectin not only provides for purely mechanical adhesion of leukocytes, but carries out signal functions [110]. Selectin interacts with cytoskeleton via the cytoplasmic domain [111]. The content and activity of selectins in different type cells are regulated by various mechanisms. E- and P-selectins are absent from non-activated endothelial cells, i.e. they are inducible [112].

Different stresses are followed by changes in the plant cytoskeleton [113-116]. Data showing the effect of phytohormones on the state of cytoskeletal elements are considered in review [117]. The opposite effect of cytoskeleton on hormone balance is also known: disintegration or stabilization of tubulin cytoskeleton in maize root cells stimulated ABA accumulation [118].

We have already discussed above experimental data showing the change in lectin concentration (activity) in plant tissues in response to phytohormones and biotic and abiotic factors. It was shown that a soybean lectin influences the state of protoplast plasmalemma, while the lectin does not exhibit such activity in the presence of colchicine [119]. Thus, there is a possible correlation between changes in cytoskeleton and lectin contents, although the existence of a functional relationship between these events requires additional experimental justification.

Two hypotheses concerning the effect of cytoskeletal rearrangements on cell functioning are of interest for understanding the mechanisms of lectin action both under stress and in the case of changes in the plant cell physiological state, which accompany growth, differentiation, and change in ontogenic phases [113, 120]. According to the first [113], cold hardening causes in wheat roots a phase change in cytoskeletal stability: in the first stage (2-3 days) there appear more stable but less active associations of microtubules, which is accompanied by inhibition of growth. In the second stage (4-7 days) there are changes favoring lower stability and higher activity with partial restoration of growth. It should be noted that the minimum of lectin content in microsome fraction corresponds to the first stage, whereas maximum in the fraction of wheat seedling cell walls corresponds to the second stage [33].

According to the second hypothesis, one of the functions of lectins in establishing legume–rhizobial symbiosis is stabilization of cytoskeletal changes induced by Nod-factor reception [120]. This can explain similar nodulation anomalies in transgenic plants carrying lectin sequence both in the sense and antisense orientations, i.e. upon increase and decrease in endogenous lectin content [121]. The change in cytoskeletal structures with different physiological activities apparently takes place just in the case of changes in lectin contents. These processes are important for change in cell physiological state, which happens in particular under stress conditions. The correlation between any intact plant parameter (like lectin or hormone content) and resistance to unfavorable factors suggests the interrelation of this parameter with dynamic instability of microtubules. Such a correlation was found during investigation of frost resistance [113]. In connection with this, a question arises concerning the relationship between adaptation to stress and cytoskeletal changes as well as concerning ABA and lectin effects on the state of the actin–tubulin system.

It was shown that hardening and treatment with ABA enhance the resistance of microtubules to the depolymerizing effect of oryzalin [122]. However, unlike hardening resulting in enhancement of the interactions of microtubules with microfilaments, ABA stimulated depolymerization of actin cytoskeleton and decrease in the content of tubulin proteins, probably at the expense of unstable fraction. Generation of more stable microtubules probably provides for enhanced frost resistance, which is confirmed by a correlation between microtubule stability in response to oryzalin in ABA-treated cells in the zone of differentiation and frost resistance of the variety [113]. The appearance in hormone-treated cells of more stable but less active microtubules is probably the basis of the ABA-induced growth retardation under stress conditions [113]. Data of the same authors on correlation between dynamic instability of microtubules from intact plants growing at normal temperature and growth parameters, potential frost resistance, and capability of hardening emphasize the functional significance of cytoskeletal rearrangements in stress reactions upon growth rate regulation, change in development phases, and other processes requiring change in the physiological state of cells.

Inhibition of tubulin polymerization and assembly of microtubules in response to oryzalin also lowered the content of soluble cytoplasmic lectins and increased somewhat the content of cell wall lectins [34, 86].

Colchicine exhibited similar activity towards cell wall lectins, and cytoskeleton stabilization by dimethyl sulfoxide (DMSO) decreased the amount of this lectin fraction [122]. In varieties with higher frost resistance, these effects were less pronounced. In the cold-adapted and frost-resistant wheat variety Albidum 114, the content of cell wall lectins did not change after oryzalin treatment, while the amount of cytoplasmic lectins increased [86]. In the presence of ABA, the effect of oryzalin on the cell wall lectins in a slightly frost-resistant wheat variety brought its parameters close to those of the highly resistant variety [122]. The effect of oryzalin on lowering the content of soluble lectins was significantly weakened in hardened plants in the presence of Ca^{2+} in relatively high concentrations; in this case, concentration of cell wall lectins significantly increased both in hardened and control plants. In hardened plants, the effects of oryzalin disappeared after addition of chlorpromazine [34]. The authors suggested the existence of plasmalemma-mediated interactions between the cell wall lectins and cytoskeleton [86]. The high stability of lectin content under the influence of oryzalin was attributed to formation during cold adaptation of a population of more stable microtubules in plant cells. Thus, the change in lectin content, especially of the cell wall lectins, upon low-temperature hardening might be associated with microtubule assembly and disassembly [123]. In this case, the release of these proteins from the cell wall is due to rearrangement of the latter followed by enhanced instability of the cytoskeleton.

Some analogies with the above-described changes in lectin complex in plants can be detected during stimulation of human neutrophils. In this case, even distribution of membrane selectins is gradually replaced by formation of discrete clusters. In non-activated neutrophils the interaction of L-selectins with cytoskeleton lowers their mobility, prevents cluster formation, and as a result the adhesion of neutrophils with blood vessel endothelial cells. In the case of cytoskeletal disintegration during activation, the lateral motility of lectins increases, and the extent of cluster formation is enhanced along with change in localization in membrane domains. The motility of lectins is enhanced upon treatment with latrunculin A as well as in cells expressing L-selectins, which are devoid of the cytoplasmic domain responsible for interaction with actin cytoskeleton [111, 112].

Our data on the induction of lectin upon treatment with hydrogen peroxide [42] suggest the involvement of these proteins in alteration of the state of the cytoskeleton during oxidative stress. The enhancement of the synthesis of reactive oxygen species is a specific cell response upon treating epithelial cells with amebic lectin [124] and mouse neutrophils with the galactose-specific lectin frutalin [125].

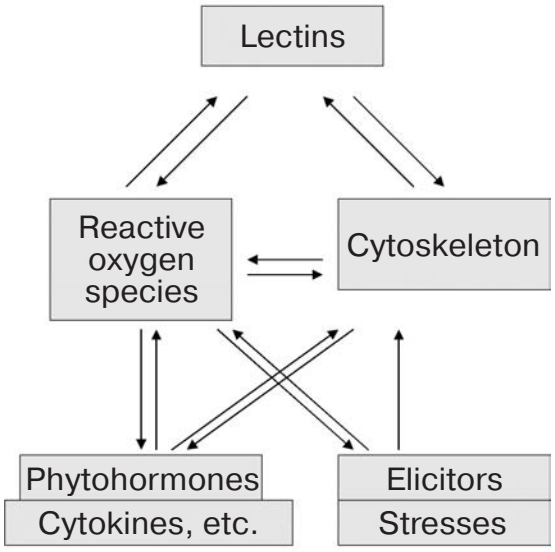
Actin microfilaments comprise the cytoskeletal component most sensitive to oxidative stress [126]. Oxidative stress emerging in vascular endothelium cells, for example, upon treatment by hydrogen peroxide, reduces the cortical actin cords and enlarges stress fibrils [127]. Treatment of a monolayer of endothelial cells from human umbilical vein with 100 μM hydrogen peroxide within 1 min causes filamin translocation from the membrane into the cytosol [128-130]. Since filamin stimulates F-actin branching and binds it to membrane glycoproteins, filamin translocation is followed by rearrangement of actin cytoskeleton and increase in the permeability of the monolayer. Damage to pulmonary artery endothelial cells by 1 mM hydrogen peroxide induces depolymerization of actin cytoskeleton, while microtubules disintegrate at higher temperatures [131]. Actin oxidation establishing intermolecular disulfide bonds can also influence the state of cytoskeleton because it weakens interaction with actin-bound proteins and between actin subunits [132]. The oxidation-reduction state of a cell is probably an important regulatory factor of actin cytoskeleton stability that defines the cell sensitivity to oxidative stress [133].

Experimental results showing that formation of extracellular hydrogen peroxide in plant pathosystem involves actin cytoskeleton is interesting for these reasons [134].

Regular changes in lectin content accompanying cytoskeleton modification are indicative of their important role in all these processes and are evidently the key to the question how lectin can combine functions of constitutive and inducible proteins and participate in different aspects of plant life (stress responses, interaction with pathogens, growth and development, accumulation of stored substances in seeds).

Practically any change in the physiological state of cells under conditions of stress or morphogenesis is revealed in cytoskeleton and is accompanied to different extent by increased level of reactive oxygen species (oxidative stress). In this case, elicitors, phytohormones, and lectins can play the role of direct inducers. The possible interaction of these elements is shown on the Scheme. Examples of the relationships marked by arrows are given above. Some of them are peculiar both to the plant and animal tissues, while others are so far found only in animal and human tissues.

Reception of inducers or any other signals and following activation of signal transduction pathways (including those with involvement of reactive oxygen species) finally result in switching on of the mechanism changing the state of the cytoskeleton. In this case, an old configuration is cytoskeleton dissipated and a new one is created. However, probably any disturbance in cell structure can result in an increased content of reactive oxygen species. Then coupled changes in cytoskeleton and content of reactive oxygen species induce secondary changes revealed in increase in lectin contents, change in hormone balance, or emergence (or modification) of secondary elicitors. The apparent increase in activity of



Possible interactions resulting in changes in the level of lectins under stress and other processes accompanied by changes in the physiological state of cells

lectins in extracts can occur either as a result of *de novo* synthesis or liberation (lowering binding strength) of lectins associated with disintegrating cytoskeleton. Lectin binding to and dissociation from cytoskeletal components along with *de novo* synthesis probably also contribute to stress-induced changes in experimentally measured activities of different lectin fractions. Data in the literature indicate a possible effect of lectins on the destabilization and stabilization cycle of the cytoskeleton, which plays a key role in regulation of plant response to biotic and abiotic stimuli. Increase in lectin content also results in inhibition of oxidative stress and decrease in the amount of reactive oxygen species, which along with stabilization of a new cytoskeletal configuration returns the cell to the unexcited state.

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