We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS BOOK CITATION INDEX INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

3

Plant Organelles-to-Nucleus Retrograde Signaling

Nadezhda Yurina and Margarita Odintsova
A.N. Bach Institute of Biochemistry, Russian Academy of Sciences
Russia

1. Introduction

Plant cells contain two types of energy producing organelles: chloroplasts, which convert solar into chemical energy, and mitochondria, which convert stored energy into ATP. Cell organelles comprise thousands of various proteins; most of them are encoded by nuclear genes; only few genes, which encode mainly the components of the system of gene expression per se and of the respiratory (in mitochondria) or photosynthetic (in chloroplasts) chains, are localized in the genomes of organelles. Thus, the maintenance of cell organelle functional activity during cell growth and development depends predominantly on the nuclear genome encoding most organelle proteins and their own genomes encoding the limited but substantial number of proteins. In this connection, organelle ribosomes, photosystems, complexes of mitochondrial respiratory chain are mosaic in their origin; they are multiprotein complexes comprising subunits encoded by the nucleus and organelles. Therefore, special mechanisms are required for coordination of gene expression in various cell compartments. Since the nuclear genome plays a key role in the biogenesis and functioning of cell organelles, the main recent attention was paid to the analysis of so called anterograde regulation, which controls the flow of information from the nucleus and cytoplasm to organelles. Along with anterograde regulation, retrograde signaling occurs in the cells, when signals emitted by organelles control gene expression in the nucleus (Pesaresi et al. 2007; Yurina & Odintsova, 2007). In this review, we summarize the recent understanding of chloroplast-to-nuclear and mitochondria-to-nuclear retrograde regulation in plant, which involves multiple potential signaling pathways in relation to abiotic stress.

2. Plastid-generated signals and their role in nuclear gene expression

The very first evidence for the existence of plastid-generated signals that control the expression of nuclear genes encoding the chloroplast proteins was obtained about 30 years ago in the studies of *albostrians* and *Saskatoon* mutants of barley (*Hordeum vulgare* L., cv. Haisa) (Emanuel et al., 2004). The subsequent studies of mutant plants with impaired carotenoid biosynthesis (manifested as photobleached plastids) demonstrated that the absence of functionally active chloroplasts considerably diminished the expression of several photosynthetic genes residing in the nucleus (Oelmüller et al., 1986). Similar results were obtained when carotenoid biosynthesis in the seedlings was reduced by norflurazon,

an inhibitor of phytoene desaturase, the enzyme participating in carotenoid biosynthesis (Yurina et al., 2006). In addition to photobleaching, the expression of nuclear genes was inhibited when chloroplast development was blocked by inhibiting the expression of plastid genes with tagetitoxin or nalidixic acid (Gray et al., 1995). Numerous signaling pathways were found to function in the cell and coordinate the expression of nuclear genes depending on chloroplast requirements (Beck, 2005).

Protein synthesis in plastids was found to correlate with the expression of nuclear genes coding for plastid proteins. The contents of several nuclear-encoded proteins transferred to plastids, e.g., the Calvin cycle enzymes, and also of proteins functionally related to plastids, such as nitrate reductase, and to peroxisomes, such as glycolate oxidase, catalase, and hydroxypyruvate reductase, were reportedly lower in the leaves of *albostrians* barley with deficient chloroplast ribosomes than in the wild-type plants. The activity of nuclear genes encoding the chloroplast-localized enzymes in the seedlings of white mustard (*Sinapis alba* L.) declined in the presence of chloramphenicol, an inhibitor of translation in chloroplasts, whereas the activity of phytochrome-induced cytoplasmic enzymes, such as chalcone synthase, was not affected and in some cases even increased (Oelmüller et al., 1986). In pea (*Pisum sativum* L.) plants, chloramphenicol inhibited the red- and blue light-induced expression of the gene encoding early light-induced protein (ELIP) (Adamska, 1995).

Treating plants with specific inhibitors of translation in chloroplasts, such as lincomycin, erythromycin, and streptomycin, also down-regulated the expression of nuclear genes encoding the photosynthetic proteins. The inhibitors of protein synthesis were shown to affect the expression of nuclear genes only at the early stages of seedling development, during the initial two–three days of germination, and the authors presumed that the products of protein synthesis at the early steps of germination may act as plastid signals (Gray, 1995). As a whole, the evidence already amassed presumes that protein synthesis in plastids generates some signal forerunning the expression of several nuclear genes. These nuclear genes encode the plastid components and also the proteins localized in other cell compartments. However, the data are missing as to whether the inhibition of protein synthesis in the plastids would decrease the expression of any nuclear genes. The current experimental evidence lists among the probable sources of chloroplast signals the reactive oxygen species (ROS), the changes in the redox state of the components of photosynthetic electron transport chain (ETC) of the stromal components of plastids, and also the metabolites produced in the course of photosynthesis (Piippo et al., 2006).

In plants, ROS are continuously synthesized as byproducts of numerous metabolic pathways in various cell compartments. ROS include oxygen ions, free radicals, and inorganic and organic peroxides. The contents of ROS are dramatically elevated under stress conditions, such as high illuminance, low temperature, etc. ROS accumulation provokes oxidative stress and damages cell structures. ROS are neutralized by enzymes, such as superoxide dismutase and catalase, and by antioxidant systems. The singlet oxygen $^1O_2$ produced by PS II and the superoxide anion generated by PS I are the major forms of ROS produced by chloroplasts under the stress conditions of high illuminance; the superoxide anion is rapidly dismutated into hydrogen peroxide (Apel & Hirt, 2004). Both these ROS were shown to act as plastid signals. The singlet oxygen $^1O_2$ generated in plastids affects the expression of several nuclear genes. Plastids of the *flu* mutant of *Arabidopsis thaliana* amass protochlorophyllide (protoChlide), which generates the singlet oxygen under illumination. In *Arabidopsis*, the singlet oxygen was shown to enhance the expression of 70 genes and

inhibit the expression of nine genes. Two arguments are put forward to advance the idea that the singlet oxygen serves as a chloroplast signal. First, it is short-lived, about 200 ns, with the transduction distance (the action at the distance) up to 10 nm. Therefore it is thought to perform the role of a plastid-generated signal, which specifically activates the genetically determined program of cell responses to the stress conditions. The EXECUTER 1 protein recently identified in *Arabidopsis* plants is presumed to recognize or transduce this signal. The N-terminal region of this protein resembles the signal sequences of chloroplast proteins encoded by nuclear genes and essential for the import of these proteins (Wagner et al., 2004). Localization of EXECUTER 1 in plastids would establish an important segment of the signaling pathway linking the plastid-generated singlet oxygen to the expression of nuclear genes.

The singlet oxygen is the major constituent of ROS generated by PS II in plants that are deficient in carotenoids due to mutations or following the treatment with herbicides, which inhibit carotenoid synthesis, such as norflurazon. In carotenoid-deficient plants, the plastid structure is considerably damaged, and the expression of several nuclear genes, mostly encoding the photosynthetic proteins, is inhibited. However, the expression profiles of the nuclear genes inhibited by carotenoid deficiency differs from those activated or inhibited by the singlet oxygen generated by illumination in the protoChlide -accumulating *flu* mutants. Therefore the singlet oxygen generated by PS II in the absence of carotenoids most probably activates the signaling pathway different from the pathway activated by the singlet oxygen produced from protoChlide or generated by PS II in the presence of 3-(3,4- dichlorophenyl)-1,1-dimethylurea (DCMU) (Wagner et al., 2004). The idea of alternative signaling pathway was substantiated by the analysis of *gun* mutants with lower sensitivity of gene expression than in the wild-type plants (Beck, 2005).

Hydrogen peroxide is another form of ROS generated by chloroplasts. It accumulates when plants are moved from temperate to strong light, and the superoxide radicals generated by PS I are transformed into hydrogen peroxide. The latter is reduced to water by chloroplast ascorbate peroxidase (APX). While the singlet oxygen, due to its high reaction capacity, mostly remains in the chloroplasts, the excess hydrogen peroxide easily diffuses across the chloroplast envelope into the cytoplasm. Treating plants with H_2O_2 was shown to induce the expression of nuclear genes related to plant responses to stress factors; thus, the expression of *cAPX* , the gene for cytoplasmic APX, is enhanced by excess light. The crucial role of hydrogen peroxide in *cAPX* expression was demonstrated in the experiments with *Arabidopsis* leaves infiltrated with catalase before excess light treatment: while the induction of *APX2* (*Arabidopsis* cAPX) was inhibited, the expression of the superoxide dismutase gene was not affected (Chang et al., 2004).

Recent studies of transgenic tobacco plants expressing the genes for catalase or thylakoid-type APX demonstrated that *APX* expression involved both hydrogen peroxide and the redox state of plastoquinone pool. Under excess light stress, cell levels of H_2O_2 in the transgenic tobacco plants were considerably lower and the induction of *cAPX* was saturated much earlier than in the wild-type plants. It follows that chloroplast-generated H_2O_2 would represent a redox signal to activate *cAPX* expression in the nucleus. The initial induction observed under photooxidative stress conditions was related to the redox state of plastoquinone pool (see below for more details), whereas the subsequent mRNA accumulation was in line with continuous accumulation of H_2O_2 (Yabuta et al., 2004). The correlation between the *APX2* expression and H_2O_2 generation under the excess light stress

was also elucidated by comparing the expression of luciferase gene under the *APX2* promoter to H_2O_2 accumulation demonstrated by cytochemical staining with methylviologen (Chang et al., 2004). This dye was shown to enhance the formation of singlet oxygen and hydrogen peroxide in chloroplasts, and the concomitant *cAPX* expression correlated with the methylviologen-generated H_2O_2 accumulation in the cells.

Both the location and the mechanism of action of plastid-generated H_2O_2 are unknown. Similar to water, hydrogen peroxide is thought to freely diffuse across the biological membranes and in this way directly interact with out-of-plastid systems of signal transduction. Nonetheless it is not clear how cells discern between the plastid-generated H_2O_2 and hydrogen peroxide produced in other cell compartments, e.g., on the plasma membrane when cells are attacked by pathogens. The stress responses to pathogens are known to widely differ from those induced by excess light stress (Beck, 2005).

Thus, we conclude that at least two chloroplast-generated forms of ROS, the singlet oxygen and hydrogen peroxide, participate in the transduction of specific signals from the plastids to the nucleus. Two ROS forms induce different responses at the level of gene expression and probably perform in the different signaling pathways. The mechanism of ROS interaction with the nucleus is also unknown. In this aspect, the experiments with mutants, such as *EXECUTER1* where the singlet oxygen participates in the plastid signal blockade, are very promising (Beck, 2005).

The environmentally induced changes in redox state of the components of photosynthetic ETC act as signals that regulate gene expression in the chloroplasts and partly in the nucleus (Pfannschmidt & Liere, 2005). It means that photosynthesis is a source of information essential for the control over the nuclear gene expression that is not recognized by cytoplasmic photoreceptors, while the chloroplasts themselves serve as sensors for the changes in light quantity and quality and in this way induce the physiological responses of photoacclimation (Beck, 2005).

The participation of the redox state of ETC components in nuclear gene expression was established in several ways. To change the redox state of ETC components, plants grown at low light were transferred at regular intervals into strong light and back. The same goal was attained by the light conditions that primarily excited either PS I or PS II, by changing the growth temperature and the level of carbohydrates and electron acceptors, such as O_2 and CO_2 , and by herbicides DCMU and 2,5-dibromo-3-methyl-6-isopropyl-p-benzoquinone (DBMIB), which specifically blocked electron transport from PS II to the cytochrome b_6/f complex at the site upstream (DCMU) or downstream of plastoquinone pool (DBMIB). These data led to the conclusion that the redox state of plastoquinones was the initial signal that regulated the expression of particular genes (Nott et al., 2006). Recently the role of the redox state of ETC as the source of chloroplast signal(s) transferred into the nuclear compartment has been supported by the analysis of expression profile of numerous nuclear genes in *Arabidopsis*: 2661 out of 3292 genes under analysis encoded chloroplast proteins and only 631, nonchloroplast ones (Richly et al., 2003). In the cases when the plants grown under PS I-inducing light were transferred under PS II-inducing illumination, the changes in mRNA contents were registered for 2133 genes: the changes were positive (enhanced expression) for 1121 genes and negative (diminished expression) for 1012 genes. The in depth study demonstrated that among these genes, 286 are immediately regulated by the redox state of photosynthetic ETC signals: the expression of 86 genes was up-regulated and 200 down-regulated (Fey et al., 2005). Whatever small were the changes in the expression

levels of most genes, this evidence seems to prove that the redox state of the ETC components regulate the expression of many nuclear genes. In green algae, the redox state of the plastoquinone pool was shown to affect *Lhcb* expression. In higher plants, however, expression of *Lhcb* has been correlated with the phosphorylation status of LhcII. The thylakoid protein STN7 has been identified as the protein kinase for LHCII (Inaba, 2010). The control exerted by the redox state of plastoquinone pool on the nuclear gene expression in the higher plants is of lesser importance than in the green algae.

Chloroplast redox signals were shown to affect the nuclear gene expression at both transcriptional and posttranscriptional (*Fed-1*, *PetE*) levels by controlling mRNA stability and binding to polyribosomes (Sherameti et al., 2002). The redox state depends on illumination, and therefore the photosynthetic apparatus can be seen as a photoreceptor recording photon quality and quantity (Beck, 2005).

In addition to the redox state of chloroplast ETC components, the nuclear genes are also controlled by the redox state of the stromal components, such as glutathione and the ferredoxin–thioredoxin system. It follows that in mature leaves of higher plants, the redox state of the components at the donor side of PS I is more important for light-dependent changes in the expression of nuclear genes coding for plastid proteins than the redox state of plastoquinone pool, especially at the early steps of signal transduction. Such conclusion confers with the evidence from the experiments with *Synechocystis* sp. 6803: in the latter case, the expression of few genes was related to the redox state of plastoquinone pool (Piippo et al., 2006). Following short exposure to extreme environmental conditions, the leading role in signal transduction from the chloroplasts to the nucleus would pass on to the stromal redox components, the products of CO_2 fixation, and the ATP/ADP ratio.

Recently many studies were focused on the role of Mg-protoporphyrin IX (MgProtoIX) and its methyl ester (MgProtoIXMe) as the regulators of the nuclear gene expression. The first proof for the participation of chlorophyll precursors in the transduction of plastid signals was obtained in the experiments with *Chlamydomonas reinhardtii* cells. In synchronous cultures of this alga, mRNA of the gene *Lhcb* accumulated already in two hours after the cultures were transferred into the light, mostly due to enhanced transcription. The studies of higher plants support the idea that chlorophyll precursors participated in the transduction of plastid-generated signals. Thujaplicin treatment of etiolated cress seedlings interfered with protochlorophyllide synthesis and resulted in MgProtoIX accumulation and decline in the light-induced synthesis of *Lhcb* mRNA. When treated with 5-aminolevulinic acid (ALA), such seedlings produced only half of light-induced mRNA *Lhcb* as compared to the seedlings treated with water. When treated with amitrole, an inhibitor of carotenoid biosynthesis, the ethiolated barley seedlings accumulated ALA, MgProtoIX, and MgProtoIXMe, with the concomitant decline in light-induced *Lhcb* and *RbcS* expression. In *Arabidopsis*, several nuclear genes encode proteins essential for plastid–to–nucleus signal transduction have been identified (Susek et al., 1993). The *Lhcb* promoter equipped with the proper selection marker was built into the nuclear genome, and seeds were mutagenized with ethyl methanesulfonate. These lines were used to isolate mutants with *Lhcb* expression independent of the chloroplast functional state. While due to chloroplast photobleaching, the expression of reporter genes under the *Lhcb* promoter was low in the wild-type plants grown under continuous illumination in the presence of norflurazon, the mutants exhibited high expression of *Lhcb* on the norflurazon-supplemented medium. Such screening identified five nonallelic loci with diminished capacity for plastid-to-nucleus signaling;

these mutants were called "genomes uncoupled", or *gun*-mutants (Susek et al., 1993). Four *gun*-mutants have been already characterized at the molecular level: several impaired enzymes, such as heme oxygenases, phytochromobiline synthases, and H subunits of Mg-chelatase or MgProtoIX-binding protein, were localized in the plastids and shown to participate in the porphyrin biosynthesis. The mutations at these genes decreased MgProtoIX accumulation. Damaging other enzymes in the biosynthetic pathway leading to MgProtoIX also diminished the plastid control of *Lhcb* expression. The plastid signal transduction is apparently affected by plant developmental stage: while the expression of *Lhcb, HEMA1*, and *Elip* was inhibited in norflurazon-treated seedlings of *Arabidopsis* and barley, similar treatment of adult plants downregulated only *Lhcb* expression (Pogulskaya et al., 2006). A considerable progress has been recently reported in characterizing the sequences of the target genes for plastid signals, such as MgProtoIX and others. The study of light-responsive promoters of the genes controlled by plastid signals and mostly encoding the components of photosynthetic apparatus demonstrated that light and plastid signals affected the nuclear gene expression at one and the same *cis*-elements. In these experiments (Kusnetsov et al., 1996), the plastid signal controlling the nuclear gene expression was activated with norflurazon, which promoted photooxidation of the thylakoid membrane in the light.

Based on the above-mentioned data, it was suggested that in higher plants a plastid-to-nucleus signal is induced by MgProtoIX and/or MgProtoIXMe (Nott et al., 2006; Pogulskaya et al., 2006; Strand et al., 2003; Yurina et al., 2006).

Subsequent studies showed that accumulation of MgProtoIX is not always accompanied by inhibition of *Lhcb* expression (Mochizuki et al., 2008; Mochizuki et al., 2010; Moulin et al., 2008;). Determination of ProtoIX, MgProtoIX and MgProtoIXMe concentrations in *Arabidopsis* seedlings grown in the presence of norflurazon showed that norflurazon inhibits significantly the formation of the intermediates of tetrapyrrole biosynthesis, the inhibition was more pronounced in older seedlings. The expression levels of some other genes, such as *Lhcb1, RbcS, HEMA, BAM3* (encodes beta-amylase) and *CA1* (encodes carbonic anhydrase) were also reduced. It was also shown that the *Arabidopsis* mutants *cs* and *ch42* with impaired Mg-chelatase subunit I (ChII) did not display *gun* phenotype, although the production of MgProtoIX in these mutants was considerably decreased (Mochizuki et al., 2001).

The herbicide 2,2'-dipyridyl supposed to induce accumulation of the MgProtoIXMe inhibited *gun2* and *gun5* phenotypes (Mochizuki et al., 2008). In norflurazon-treated seedlings, the *Lhcb* expresssion was decreased, however no accumulation of porphyrins was observed (Gadjieva et al., 2005).

Contradictory data concerning the signaling role of MgProtoIX in the repression of nuclear plastid protein genes may be associated with problems of accurate quantitative determination of tetrapyrrole biosynthesis intermediates. A detailed study of the role of MgProtoIX (and other chlorophyll biosynthesis intermediates) as signaling molecules during retrograde regulation revealed the absence of correlation between MgProtoIX accumulation and expression of nuclear plastid protein genes. It was shown that in norflurazon-treated plants at different growth conditions, nuclear gene expression was inhibited, however, the accumulation of MgProtoIX or other intermediates of chlorophyll biosynthesis did not occur (Moulin et al., 2008). Conversely, elevation of the MgProtoIX endogenous level by the addition of the tetrapyrrole precursor ALA caused an induction not repression of the nuclear photosynthetic genes. Chemical or genetic modification of the

tetrapyrrole levels and light conditions revealed no correlation between the intermediates of tetrapyrrole biosynthesis (including MgProtoIX) and *Lhcb* expression (Mochizuki et al., 2010). *Gun* mutations had no effect on heme accumulation (Voigt et al., 2010). The determination of tetrapyrrole content in *Arabidopsis* plants under light stress conditions by a conventional procedure revealed no significant changes in MgProtoIX concentration in norflurazon-treated plants as compared to control plants (our data). A certain decrease in MgProtoIX concentration was even recorded (unpublished data). Thus, it remains unclear whether MgProtoIX serves as a signal during retrograde regulation in higher plants.

Based on conflicting experimental data it was suggested that not MgProtoIX molecules but their derivatives, degradation products or ROS, such as singlet oxygen or hydrogen peroxide, may induce retrograde signaling cascades (Mochizuki et al., 2010; Moulin et al., 2008). There is experimental evidence that expression of nuclear plastid protein genes *Lhcb* and *RbcS* is controlled by hydrogen peroxide and singlet oxygen (La Rocca et al., 2001). Based on these data it was hypothesized that a simple signaling cascade mechanism connecting MgProtoIX accumulation with inhibition of nuclear plastid protein gene expression is unlikely (Mochizuki et al., 2008; Moulin et al., 2008).

The discrepant results may partially arise from different conditions of norflurazon treatment of seedlings (Zhang et al., 2011). It cannot be excluded that MgProtoIX-generated signals are short-living and function in particular cellular compartments and thus are difficult to detect.

Transcriptome analysis of the green unicellular algae *C. reinhardtii* grown in the presence of MgProtoIX or heme showed that tetrapyrrole biosynthesis intermediates transiently but considerably changed expression of approx. 1000 genes. They include a limited number of photosynthesis-related genes, the genes for the tricarbonic acid cycle enzymes, the genes for heme-binding proteins, several stress-responsive genes, as well as genes involved in protein folding and degradation. Both tetrapyrroles act as secondary messengers in adaptive response of the whole cell, not only cellular organelles (Voss et al., 2011).

Another important process that may generate a plastid signal is protein import into plastids (Inaba, 2010). It was shown that in a mutant with a deficiency of the main receptor for the imported proteins Toc159, expression of nuclear plastid protein genes is inhibited. This abnormality in protein import into the plastids serves as a plastid signal (Kakizaki & Inaba, 2010). By this mechanism retrograde signals regulate expression of nuclear plastid protein genes in accordance with the requirements of these organelles and provide efficient assembly of multisubunit complexes encoded both by nuclear and chloroplast genes. It should be noted that the signaling molecules triggering retrograde regulation have not been identified so far (Pfannschmidt, 2010).

2.1 Components of the retrograde signaling pathways

The GUN1 protein plays an important role in retrograde signal transduction. It was shown that the *gun1* mutation is not associated with tetrapyrrole biosynthesis. This follows from the differences between the *gun1* and *gun2-gun5* genes (Cottage et al., 2010). Treatment of plastids with lincomycin, an inhibitor of protein synthesis, represses expression of nuclear photosynthetic genes in wild-type *Arabidopsis* seedlings and *gun2-gun5* mutants, but not in *gun1* mutants (Koussevitzky et al., 2007). The *gun1* gene codes for a protein of plastid nucleoids, which contains 10 copies of a pentatricopeptide repeat (PPR) and the so-called SMR-domain (a minor MutS-associated domain) close to the C-terminus of the polypeptide chain (Cottage et al., 2010; Koussevitzky et al., 2007). The SMR-domain binds DNA, while

the PPR-motif participates in RNA processing and is found in many mitochondrial and plastid proteins. Since lincomycin does not inhibit expression of nuclear photosynthesis-related genes in *gun1* mutants, it was suggested that GUN1 may be involved in signal transduction induced by impaired expression of plastid genes (Armbruster et al., 2011). GUN1-dependent signal transduction may also be involved in coordination of nuclear photosynthetic gene expression with the efficiency of protein import into the plastids (Kakizaki & Inaba, 2010). It was demonstrated that GUN1 mediates signals induced by tetrapyrrole biosynthesis intermediates and redox state of the electron transport chain. Since *gun1* mutants display abnormal reaction to high-intensity light, it was hypothesized that GUN1 integrates signals induced by norflurazon, lyncomycin and high-intensity light (Koussevitzky et al., 2007). A comparative study of anthocyan and *Lhcb1* transcript accumulation in *gun1-1* mutants and wild-type *Arabidopsis* seedlings indicated that the deficiency of functional GUN1 impairs early development of seedlings and alters the sensitivity of plants to sucrose and abscisic acid (see below). However, the mode of GUN1 action is presently unknown (Cottage et al., 2010).

In the retrograde signaling pathway downstream of GUN1 operate two nuclear transcription factors: AP2 (Apetala 2)-like transcription factor ABI4 and GLK1. The *Arabidopsis abi4* mutant has a weak *gun* phenotype. The *abi4* gene is highly expressed in seeds in contrast to seedlings. The *abi4* expression is induced by glucose and probably other sugars. ABI4 is a negative regulator of the *Lhcb* expression. In response to plastid signals ABI4 competitively binds to G-box of the *cis*-element and inhibits *Lhcb* expression (Inaba, 2010). The transcription factors of the Apetala 2 (AP2)- type act as repressors of transcription in the presence of abscisic acid, ethylene and jasmonic acid (Koussevitzky et al., 2007). By a still unknown mechanism GUN1 activates ABI4. It was shown that just this mechanism is used by retrograde signals induced by tetrapyrrole biosynthesis intermediates, plastid gene expression and redox state of the electron transport chain, but differs from the mechanisms utilized by signals associated with abnormal protein import into the plastids (Inaba, 2010). In this case, the transcription factor GLK1 operates. In contrast to ABI4, GLK1 is a positive regulator of *Lhcb* expression, which coordinates expression of nuclear photosynthetic genes (Kamikaze & Inaba, 2010). Under stressful conditions, such as norflurazon treatment and impaired protein import, GLK1 expression is considerably decreased that leads to inhibition of photosynthesis-related genes. For GLK1 repression, GUN1 is necessary.

It was shown that many nuclear photosynthetic genes controlled by plastid signals contain the ACGT sequence in their promoter regions that also serves as the major (core) element involved in cell response to abscisic acid. This points to the involvement of components of ABA-signaling cascade (or abscisic acid levels) in retrograde signaling (Jung & Chory, 2010; Koussevitzky et al., 2007). Analysis of the ABA-deficient (*aba*) and ABA-insensitive (*abi*) *Arabidopsis* mutants showed that in regulation of nuclear gene expression and plant adaptation to stressful conditions, the components of the ABA cascade are tightly associated with the Mg-Proto retrograde signaling pathway. The interaction between the plastid and abscisic acid signals is mediated by ABI4, which is a 'master switch' that controls expression of a large number of genes in response to diverse signals. The ABI4 transcription factor is not only a component of the plastid retrograde signaling pathway but is also involved in mitochondrial retrograde signaling, thus it serves as a convergence point of these signaling pathways (Cottage et al., 2010).

It was shown that the H subunit of Mg-chelatase (ChIH) participates in signal transduction from the plastids to the nucleus and in ABA-dependent signaling cascades. The ChIH protein is a receptor of ABA-induced signals. In *Arabidopsis* it is a positive regulator of signaling induced by Mg-Proto and chlorophyll biosynthesis intermediates. Plants with enhanced expression of this protein are supersensitive to abscisic acid, while ChIH-deficient mutants possess an ABA-insensitive phenotype. It was assumed that the plastid proteins EX1 and EX2 (Inaba, 2010) and protein kinases, in particular the thylakoid membrane protein STN7, which is a Lhcb protein kinase (Inaba, 2010; Pfannschmidt, 2010), are also involved in retrograde signaling. However these proteins are located in the plastids. The cytosolic mediators of the plastid signals have not been identified. The nuclear transcription factor Whirly1 (Why1), which was originally described as a telomere-binding protein, is also supposed to be involved in retrograde signaling.

Abundant experimental data indicate that plastid retrograde signaling cascades interact with light signaling. Light activates expression of a large number of photosynthesis-related nuclear genes, such as *Cab* encoding chlorophyll *a/b* – binding proteins, *RbcS* (encoding the small subunit of Rubisco) and *Pc* coding for plastocyanin. Retrograde signals may act synergistically with the light signals or transform them from inducers into repressors of photosynthetic gene expression (Larkin et al., 2008; Osipenkova et al., 2010). It is generally accepted that the plastid retrograde signals are endogenous regulators of the light signaling and that integration of the light and plastid signals helps plants to overcome chloroplast dysfunction during organelle biogenesis under unfavorable light conditions (Larkin et al., 2008). Both types of signals act on the same *cis*-elements in the promoters of the nuclear plastid protein genes (Kusnetsov et al., 1996). The ABI4 participates both in retrograde and light signaling. This protein binds to the promoter region of the gene regulated by a retrograde signal through the conserved CCAC motif, which is a core element necessary for binding. In such a way ABI4 inhibits G-box mediated light-inducible expression of photosynthesis-related genes when the chloroplast development is inhibited (Koussevitzky et al., 2007).

Earlier a strong correlation between the light and sucrose signaling pathways was revealed (Dijkwel et al., 1997). It was shown that the sucrose signaling pathways may interact (at least in some tissues) with other retrograde signaling pathways and thus modulate the response of nuclear genes to retrograde signals. In the study of *sun6* (sucrose-uncoupled6) *Arabidopsis* mutants (allelic *abi4*-mutants), a correlation between redox signals and sucrose-regulated gene expression was established (Oswald et al., 2001). It was also shown that the SUN6 protein is involved in sucrose repression of phytochrome A-dependent signaling pathways (Dijkwel et al., 1997). These data indicate interactions of sucrose, light and plastid signaling pathways.

The data presented indicate that plant cells possess a complex network of signaling pathways that function independently or interact with each other due to common intermediates or final cascade components. Tetrapyrroles play a key role in this network, their metabolism regulates the plant cell functions.

The transfer of numerous genes from cell organelles into the nucleus in the course of cell evolution (Odintsova & Yurina, 2006) led to gene redistribution in cell compartments and the coordinated expression of nuclear and organellar genes. The expression of nuclear genes in plastids and mitochondria must be harmonized with the functional state of these organelles, and such coordination was provided by evolution of the so-called retrograde

(organelle-to-nucleus) control over the expression of nucleus-coded organellar genes. The pathways of such signal transduction have not been sufficiently clarified: we do not know how the plastid or mitochondrial signal crosses the organelle membranes, what signal molecules stay in the cytosol and nucleus, and whether their action upon the regulatory proteins of the nucleus is direct or mediated by the induction of other signals. In most cases the plastid signals are related to the redox state of organelles, e.g., the ETC components and redox-active stromal compounds, such as thioredoxin and glutathione, or with the chlorophyll biosynthesis. ROS, the side products of photosynthesis and mitochondrial respiration, also can participate as signal molecules in the organelle-to-nucleus signal transduction. ROS are formed by both types of organelles, and therefore can provide for the coordinated expression of nuclear genes in the plastids and mitochondria at the transcriptional level. The analysis of the nucleus-encoded chloroplast transcriptome demonstrated that the transcription of the nuclear genes coding for plastid proteins was controlled by several types of plastid signals comprising the complicated signal network within plant cells (Leister, 2005). The attempts to investigate this network using the mutant plants are problematical because alternative control mechanisms may function in the mutants impaired in one and the same photosynthetic complex (Beck, 2005).

3. Mitochondrial retrograde regulation in plants

Most data about mitochondrial retrograde regulation (MRR) in plants were obtained on material with disturbed functioning of mitochondria. Expression of some nuclear genes is activated in response to disturbances in mtETC, tricarbonic acid cycle, and also mtDNA (Rhoads & Subbaiah, 2007). Plant mitochondria dysfunction induced by mutations results frequently in male sterility, embryo lethal phenotype, or chlorosis in plants, which could not complete their life cycle (Newton et al., 2004). Plant mitochondria dysfunction can be induced by biotic and abiotic stresses. A bulk of information was obtained demonstrating a great contribution of mitochondria in general plant response to stress.

Mitochondrial retrograde regulation in plants is usually studied on the systems with disturbed mitochondrial functions induced by mutations, chemical agents, or biotic and abiotic stresses. Dysfunction of mitochondria leads to changes in the nuclear gene expression. Plant response to mitochondrial function disturbance is an expression induction of genes encoding proteins involved in the restoration of mitochondria functioning, such as alternative oxidase (AO) or alternative NADPH-dehydrogenases, and genes encoding antioxidant enzymes, such as glutathione transferases, catalases, ascorbate peroxidases, and superoxide dismutases, normalizing ROS level (Rhoads & Subbaiah, 2007). At present, most studied plant response to mitochondria dysfunction is the changes in expression of the nuclear gene encoding AO (Mackenzie & McIntosh, 1999; Rhoads & Vanlerberghe, 2004). It is believed that the two pathways of signal transduction to the nucleus operate at AO expression: one with the involvement of ROS and another with the involvement of organic acids (Gray et al., 2004).

Cytoplasmic male sterility (CMS), i.e., plant failure to produce viable pollen, is induced by signals from mitochondria and is one of the characteristic examples of MRR in plants. Male sterility is often results from mutation-induced mitochondria dysfunction. Changes in mtDNA or mitochondrial gene expression induce changes in nuclear gene expression, result in the modification of stamen phenotype, and finally in the suppression of pollen formation. CMS is a commonly occurring phenomenon observed in more than 150 plant species. It is

met frequently in hybrid lines obtained due to intra- or inter-specific crosses, i.e., in alloplasmic lines where the nucleus of one species functions in the cytoplasm of another species.

The fact that CMS is determined by the interaction between the nucleus and mitochondria was proved by maternal inheritance of male sterility phenotype and by suppression of male sterility with nuclear genes of fertile restoration (Rf genes). These genes (except a single gene) encode proteins belonging to the family of PPR proteins (proteins containing pentatricopeptide repeats). These proteins in *Arabidopsis* are supposed to be localized in mitochondria or plastids. Some of them participate in the processing of various RNAs in organelles (Carlsson et al., 2008).

It should be noted that not only retrograde signaling pathways but also other mechanisms could result in CMS, i.e., diverse mechanisms could determine male sterility. However, the target genes of retrograde signaling pathways and functions of genes for fertility restoration are evidently common for many CMS systems. Since several Rf genes could suppress the phenotype of male sterility and restore male fertility in various species, we can suppose that changes in transcription, translation, or RNA processing in mitochondria are represent the common mechanism of restoration. The homeotic genes, i.e., genes, which mutations result in changes of floral organs, are most probable targets for the retrograde signaling pathways directed from CMS-inducing mitochondria to the nucleus. However, the signal for CMS origin is unknown (Carlsson et al., 2008; Kubo & Newton, 2008).

Plant mitochondria respond to abiotic and biotic stresses. They can serve stress sensors and initiate responses (or be involved in responses) to particular stress types. Thus, MRR is involved in the plant cell response to oxygen stress. It is known that, under definite conditions (flooding for example), oxygen is a limiting factor for plant growth and survival. Under normal growth and development, plant also could experience hypoxia because oxygen diffusion in tissues with closely packed cells and small intercellular spaces is hindered. It is also known that even plants acclimated to land conditions manifest a great tolerance to oxygen deficit, which indicates the presence in them of highly sensitive system for oxygen sensing and well developed responses to oxygen stress (Bailey-Serres & Chang, 2005). Adaptation to a rapidly changing oxygen level includes rapid changes in gene expression (potentially induced by preceding rapid changes in the level of ROS, redox state, and/or energetic status) permitting a correction of the metabolism (for example, suppression of carbon entry in TCA cycle with parallel increase in the carbon entry into glycolytic and other enzymic pathways) (Rhoads & Subbaiah, 2007). Reactions leading to programmed cell death are also one of the plant responses to anaerobiosis (Subbaiah & Sachs, 2003).

The genes controlled by oxygen could be roughly divided into aerobic genes (transcribed at atmospheric oxygen level), hypoxic genes (induced at lowered oxygen level), anoxic genes (induced in the absence of oxygen), and hyperoxic genes (transcribed under oxidative conditions). Certainly, such a division is rather conditional (Rhoads & Subbaiah, 2007).

Since oxygen is primarily absorbed by cell mitochondria, changes in the level of available oxygen should be primarily sensed by these organelles, resulting in MRR of nuclear genes. The levels of transcripts and proteins (TCA enzymes or mtETC components) were shown to be reduced at oxygen removal and rapidly restored after oxygen level restoration (Branco-

Price et al., 2005). In experiments with maize cell culture, it was shown that, in the absence of oxygen, immediate and reversible increase in the Ca^{2+} level in the cytosol ($[Ca^{2+}]_{cyt}$) occurs. Changes in the Ca^{2+} level are observed around mitochondria and disappeared by the blocker of mitochondrial calcium channel ruthenium red. This dye suppresses induction of genes sensitive to anoxia, alcohol dehydrogenase 1 (*adh1*) and sucrose synthase (sh1) in maize seedlings and cell culture (Subbaiah et al., 1998). Maize seedlings treated with ruthenium red are especially sensitive to anoxic stress. Ca^{2+} addition neutralizes ruthenium red effects, confirming the supposition that ion changes in maize evidently initiated by mitochondria are the signals for responses to anoxia. Calcium ions were shown to be an important component of the signal transduction pathway related to hypoxia in *Arabidopsis*, rice, and barley, which indicates the conserved nature of this pathway in plants (Rhoads & Subbaiah, 2007). The conclusion that mitochondria are a source of $[Ca^{2+}]_{cyt}$-signal initiating nuclear gene activation is supported by dynamics of mitochondrial Ca^{2+} ($[Ca^{2+}]_{mt}$) in response to anoxia. $[Ca^{2+}]_{mt}$ release was shown to occur in maize cells immediately after ceasing oxygen influx.

Heat stress is known to reduce crop yield. One of the ways to decline heat shock effects is the induction of genes encoding heat shock proteins (HSPs), including low-molecular HSPs (sHSPs). Trasgenic plants expressing HSPs are a convenient model for studying mitochondrial responses to heat shock. Thus, transgenic *Arabidopsis* plants were obtained expressing transgene encoding maize mitochondrial sHSP, ZmHSP22 (Rhoads et al., 2007). This protein is weakly constitutively synthesized in etiolated maize seedlings and induced by heat shock. In transgenic line, this transgene under the control of cauliflower mosaic virus 35S promoter was actively expressed constitutively. Using confocal immunofluorescent microscopy and the analysis of isolated mitochondria, it was shown that ZmHSP22 penetrates *Arabidopsis* mitochondria and processed there with the formation of mature protein. In transgenic plants subjected to heat stress, expression of several nuclear genes encoding endogenous mitochondrial sHSP of *Arabidopsis* (AtHSP23.6) and also HSPs localized in chloroplasts (AtHSP25.3 and AtHSP70-6) and cytosol (AtHSP17.4 and AtHSP70-1) was changed. In wild-type plants, AtHSP23.6 is weakly expressed but induced by heat stress. These data allow a supposition that heat-induced MRR could affect HSP expression.

The common component of various abiotic stresses is an oxidative stress induced by ROS generation. Oxidative stress could induce mitochondria dysfunction in plants and result in inactivation of definite hormonal signals (Rhoads & Subbaiah, 2007). Proteomic approach for identification of mitochondrial components sensitive to oxidative damage showed that they are enzymes of the TCA cycle, components of mtETC and oxidative phosphorylation (Sweetlove & Foyer, 2004). In the leaves, glycine decarboxylase, the enzyme of the photorespiratory pathway, turned out to be especially prone to oxidative damage (Taylor et al., 2002).

3.1 Components of signal transduction pathways related to plant mitochondrial retrograde regulation

The common feature of abiotic and biotic stresses in plants is an increased level of ROS in the cells, which leads to changes in expression of nuclear genes (Vranova et al., 2002). However, the precise role of ROS in plant responses to stresses is unknown. A specific ROS property is that they are damaging compounds and simultaneously signal molecules during

plant responses to stresses. However, at different stresses, ROS elevated level induces different changes in gene expression; ROS can interact with other stress-factors, such as calcium, hormones, or changes in the cell redox state (Gadiev et al., 2006). mtROS are the part of the total ROS pool generated in the cells in response to stress. The contribution of mitochondria and other cell compartments into the ROS level is undetermined; changes in nuclear gene expression induced by each of these compartments are unknown as well.

It was shown that inhibition of the cytochrome respiratory pathway results in the increase in the mtROS content (especially hydrogen peroxide), which evidently induces MRR signaling and AO gene expression, which leads to the reduction in mtROS generation. However, this does not mean that mtROS are required for all MRR pathways functioning in the plants. Monofluoroacetate is a mighty inducer of AtAOX1a gene expression (Zarkovic et al., 2005), but it does not induce a strong enhancement in ROS generation in the cell (Rhoads & Subbaiah, 2004). Although the role of mtROS and MRR at inhibition of plant mtETC by antimycin seems to be proven, other signaling components of mitochondrial regulation downstream ROS are not identified.

It was shown that cell redox state and signals emitted by the photosynthetic ETC are involved in the control of nuclear and chloroplast gene expression. Changes in the redox state have also a great significance for plant responses to stresses (Foyer & Noctor, 2005). Mitochondria can change the cell redox state, in particular due to ROS generation and functioning of the glutathione–ascorbate cycle. It is supposed that in plants glutathione and ascorbate are the components of redox signaling, which induce expression of defense genes (Dutilleul et al., 2003; Foyer & Noctor, 2005).

Glutathione is also supposed to be a component of the signal transduction pathway related to cold stress (Kocsy et al., 2001). Until now, only few components of the MRR signaling pathways in plants are identified. Among data available, especially interesting ones are that calcium is involved in MRR related to hypoxia; indeed, Ca^{2+} ions participate in signal transduction in mitochondrial retrograde signaling pathways most frequently met in higher eukaryotes (Butow & Avadhani, 2004). It is supposed that Ca^{2+} is involved as a signal not only in plant MRR under hypoxia but also in other stress types. All these data argue for an important role of Ca^{2+} transport for plant responses to stresses. It is unknown how a signal from the increase in the $[Ca^{2+}]_{cyt}$ determined by mitochondria is amplified, transmitted to the nucleus, and results in changes in nuclear gene expression. It is believed that Ca^{2+}-binding proteins participate in this process, viz., calcium-dependent protein kinases, transcription factors, and regulatory proteins (such as 14-3-3 proteins). It is not excluded that Ca^{2+} signal per se is transferred to the nucleus. Some data are obtained that changes in the Ca^{2+} content in the nucleus affect directly gene expression.

Plant genomes (at least sequenced genomes of *Arabidopsis* and rice) contain more genes encoding transcription factors than sequenced genomes of other organisms; in particular, they comprise unique gene families characteristic of only plants (Chen et al., 2002). It is supposed that these factors could be additional factors tuning plant responses to stresses. Some of them are evidently involved in MRR, although no transcription factors specific for MRR are identified until now. It might be that such factors as WRKY, bZIP, and Dof are involved in plant MRR because they participate in responses to biotic and abiotic stresses, including oxidative stress (Chen et al., 2002).

The elucidation of molecular mechanisms of plant MRR is only at its start. Mitochondria responses to stresses, which are the part of total plant responses, could determine the fate of

plant cells, resulting in the restoration of their vital activity or in their death. Thus, induction of AO and alternative NADPH-dehydrogenases in tobacco cell suspension at suppression of cytochrome respiratory pathway prevent cell death. In the cells lacking AO, ROS accumulation induced by mtETC inhibition evidently shifts metabolism toward programmed cell death (Robson & Vanlerberghe, 2002). A capability of the induction of AO expression could also affect the independent mitochondrial pathways of programmed cell death.

Several retrograde mitochondrial signaling pathways could function in the plant cells (Zarkovic et al., 2005), and they could initiate specific changes in nuclear gene expression in response to specific disturbances in mitochondria functioning.

Retrograde mitochondrial signaling pathways interact with each other. In addition, they interact with retrograde chloroplast signaling pathways (Pesaresi et al., 2006, 2007) and other signal transduction pathways in the plant cell including sugars (Pesaresi et al., 2007), hormones (Kwak et al., 2006), enzymes (Subbaiah et al., 2006), etc.

The interaction between retrograde signaling from mitochondria and plastids is of a great interest because these signals control expression of nuclear genes encoding organelle components in dependence on organelle functional state. Chloroplast and mitochondrial metabolisms are known to be connected. Photosynthesis provides substrates for mitochondrial respiration and, in its turn, depends on some compounds synthesized by mitochondria. In darkness, mitochondria are the major source of ATP for cell processes, including those in chloroplasts. In addition in darkness, ATP supports the proton gradient across the thylakoid membrane, thus protecting chloroplasts against photoinhibition after the start of illumination. In the light, mitochondria provide chloroplasts with carbon-containing compounds, produced in the TCA cycle, for NH_4^+ assimilation, whereas ATP supports diverse biosynthetic reactions, including the restoration of photosystem II functions (Pesaresi et al., 2006).

Data concerning signal molecules and components of inter-organelle signaling pathways are few. The molecular analysis of these pathways supposes their multiple interactions. It is believed that NO, ascorbate, and ROS could fulfill the role of signals in the mitochondria and chloroplast interactions. However, the ways of signal transduction from organelles to the nucleus and the incorporation of these signals into the general system of expression regulation are not known so far (Pesaresi et al., 2006).

4. Conclusion

Retrograde regulation of nuclear gene expression by organelles is best studied in budding yeast *S. cerevisiae* and higher plant chloroplasts. In chloroplasts, retrograde signals are mostly related to the redox state of organelles or to chlorophyll biosynthesis. Intermediates of tetrapyrrole biosynthesis and the products of organellar protein synthesis are evidently major retrograde signals (Pesaresi et al., 2006, 2007). Changes in the nuclear gene expression dependent on plastid retrograde signaling pathways include a multilevel control of transcription and the involvement of the ABI4 transcription factor (Pesaresi et al., 2007). ROS, harmful by-products of photosynthesis and mitochondrial respiration, could also serve signal molecules transmitting a signal from organelles to the nucleus. Since ROS are generated in both types of organelles, they could provide for coordinated expression of nuclear genes of plastid and mitochondria at the level of transcription. Most identified

regulons are shown to contain nuclear genes of chloroplast and mitochondria, and this allows coordinated regulation of activities of these organelles (Rhoads & Subbaiah, 2007). Despite the fact that investigations of plant organelles-to-nucleus retrograde signaling have been the subject of intensive research for several decades, the available data are fragmentary. The molecules, which induce the signal, are still unknown, as well as the mechanisms of signal transduction and the components of the signaling cascade involved. Not much is known on the specificity and cross-talk between different signaling pathways in plants. Usage of classic and novel methodological approaches help identify signal molecules involved in mitochondrial retrograde regulation in plants. Further research exploiting the traditional methods of biochemistry, direct and reverse genetics, proteomics and metabolomics will help to identify the signaling molecules triggering retrograde cascades, to unravel the mechanisms of signal transduction.

5. Acknowledgment

This study was supported by the Molecular and Cell Biology program of the Presidium of RAS.

6. References

Adamska, I. (1995). Regulation of Early Light-Inducible Protein Gene Expression by Blue and Red Light in Etiolated Seedlings Involves Nuclear and Plastid Factors. *Plant Physiol.*, Vol.107, No.4, (April 1999), pp. 1167-1175, ISSN 1532-2548

Apel, K. & Hirt, H. (2004). Reactive Oxygen Species: Metabolism, Oxidative Stress, and Signal Transduction. *Annu. Rev. Plant Biol.*, Vol. 55., pp. 373-399, ISSN 1543-5008

Armbruster, U.; Pesaresi, P.; Pribil, M.; Hertle, A. & Leister, D. (2011). Update on Chloroplast Research: New Tools, New Topics, and New Trends. *Mol. Plant.*, Vol.4, No.1, (January, 2011), pp. 1-16, ISSN 1752-9867

Bailey-Serres, J. & Chang, R. (2005). Sensing and Signaling in Response to Oxygen Deprivation in Plants and Other Organisms. *Ann. Bot.*, Vol.96, No.4, (September, 2005), pp. 507-518, ISSN 0305-7364

Beck, Ch.F. (2005). Signaling Pathways from the Chloroplast to the Nucleus. *Planta*, Vol. 222, No.5, (November 2005), pp. 743-756, ISSN 0032-0935

Branco-Price, C.; Kawaguchi, R.; Ferreira, R.B. & Bailey-Serres, G. (2005). Genome-Wide Analysis of Transcript Abundance and Translation in Arabidopsis Seedlings Subjected to Oxygen Deprivation. *Ann. Bot.*, Vol. 96, No.4, (September, 2005), pp. 647-660, ISSN 0305-7364

Butow, R.A. & Avadhani, N.G. (2004). Mitochondrial Signaling, the Retrograde Response. *Mol. Cell.*, Vol.14, No.1, (April, 2004), pp. 1-15, ISSN 1097-2765

Carlsson, J.; Leino, M.; Sohlberg, J.; Sundstrom, J.F. & Glimelius, K. (2008). Mitochondrial Regulation of Flower Development. *Mitochondrion*, Vol. 8, No.1, (January, 2008), pp. 74-86, ISSN 1567-7249

Chang, C.C.; Ball, L.; Fryer, M.J.; Baker, N.R.; Karpinski, S. & Mullineaux, P.M. (2004). Induction of Ascorbate Peroxidase 2 Expression in Wounded Arabidopsis Leaves Does Not Involve Known Wound-Signalling Pathways but Is Associated with Changes in Photosynthesis. *Plant J.*, Vol. 38, No.3, (May, 2004), pp. 499-511, ISSN 0960-7412

Chen, W.; Provart, N.J.; Glazebrook, J.; Katagiri, F.; Chang, H.S.; Eulgem, T.; Mauch, F.; Luan, S.; Zou, G.; Whitham, S.A.; Budworth, P.R.; Tao, Y.; Xie, Z.; Chen, X.; Lam, S.; Kreps, J.A.; Harper, J.F.; Si-Ammour, A.; Mauch-Mani, B.; Heinlein, M.; Kobayashi, K.; Hohn, T.; Dangl, J.L.; Wang, X. & Zhu, T. (2002). Expression Profile Matrix Arabidopsis Transcription Factor Genes Suggests Their Putative Functions in Response to Environmental Stresses. *Plant Cell*, Vol.14, No.3, (March, 2002), pp. 559-574, ISSN 1040-4651

Cottage, A.; Mott, E.K.; Kempster, J.A. & Gray, J.C. (2010). The Arabidopsis Plastid-Signalling Mutant gun1 (genomes uncoupled1) Shows Altered Sensitivity to Sucrose and Abscisic Acid and Alterations in Early Seedling Development. *J. Exp. Bot.*, Vol.61, No.13, (July, 2010), pp. 3773-3786, ISSN 0022-0957

Dijkwel, P.P.; Huijser, C.; Weisbeek, P.J.; Chua, N.H. & Smeekens, S.C. (1997). Sucrose Control of Phytochrome A Signaling in Arabidopsis. *Plant Cell*, Vol.9, No.4, (April, 1997), pp. 583-595, ISSN 1040-4651

Dutilleul, C.; Garmier, M.; Noctor, G.;., Mathieu,C.; Chetrit, P.; Foyer, C.H. & De Paepe, R. (2003). Leaf Mitochondria Modulate Whole Cell Redox Homeostasis, Set Antioxidant Capacity, and Determine Stress Resistance Through Altered Signaling and Diurnal Regulation. *Plant Cell*, Vol.5, No.5, (May, 2003), pp. 1212-1226, ISSN 1040-4651

Emanuel, C.; Weihe, A.; Graner, A.; Hess, W.R. & Börner, T. (2004). Chloroplast Development Affects Expression of Phage-Type RNA Polymerases in Barley Leaves. *Plant J.*, Vol.38, No.3, (May, 2004), pp. 460-472, ISSN 0960-7412

Fey, V.; Wagner, R.; Bräutigam, K.; Wirtz, M.; Hell, R.; Dietzmann, A.; Leister, D.; Oelmüller, R. & Pfannschmidt, T. (2005). Retrograde Plastid Redox Signals in the Expression of Nuclear Genes for Chloroplast Proteins of Arabidopsis thaliana. *J. Biol. Chem.*, Vol. 80, No.7 , (November, 2004), pp. 5318-5328, ISSN 0021-9258

Foyer, C.N. & Noctor, G. (2005). Redox Homeostasis and Antioxidant Interface between Stress Perception and Physiological Responses. *Plant Cell*, Vol. 17, No.7, (July, 2005), p. 1866-1875, ISSN 1040-4651.

Gadiev, I.; Vanderauwera, S.; Gechev, T.S.; Laloi, C.; Minkov, I.N.; Shulaev, V.; Apel, K.; Inze, D.; Mittler, R. & Van Breusegem, F. (2006). Transcriptomic Footprints Disclose Specificity of Reactive Oxygen Species Signaling in Arabidopsis. *Plant Physiol.*, Vol.141, No.2 , (January, 2006), pp. 436-445, ISSN 1532-2548

Gadjieva, R.; Axelsson, E.; Olsson, U. &, Hansson, M. (2005). Analysis of gun Phenotype in Barley Magnesium Chelatase and Mg-protoporphyrin IX Monomethyl Ester Cyclase Mutants. *Plant Physiol. Biochem.*, Vol.43, No.10-11, (October, 2005), pp. 901–908, ISSN 0981- 9428

Gray, G.R.; Maxwell, D.P.; Villarimo, A.R. & McIntosh, L. (2004). Mitochondria / Nuclear Signaling of Alternative Oxidase Expression Occurs through Distinct Pathways Involving Organic Acids and Reactive Oxygen Species. *Plant Cell Rep.*, Vol.23, No.7, (August, 2004), pp. 497-503, ISSN 0721-7714

Gray, J.C.; Sornarajah, R.; Zabron, A.A.; Duckett,C.M. & Khan, M.S. (1995). Chloroplast Control of Nuclear Gene Expression, In: *Photosynthesis: From Light to Biosphere*, Mathis P. (Ed.), pp. 543-550, Kluwer Academic Publishers, ISBN 978-0-7923-3862-8, Dordrecht, Netherlands

Inaba, T. (2010). Bilateral Communication Between Plastid and the Nucleus: Plastid Protein Import and Plastid-to-nucleus Retrograde Signaling. *Biosci. Biotechnol. Biochem*. Vol.74, No.3 , (March 2010), pp. 471-476, ISSN 0916-8451

Jung, H.-S. & Chory, J. (2010). Signaling between Chloroplasts and the Nucleus: Can a Systems Biology Approach Bring Clarity to a Complex and Highly Regulated Pathway? *Plant Physiol.* Vol.152, No.2, (November, 2009), pp. 453–459, ISSN 1532-2548.

Kakizaki, T. & Inaba, T. (2010). New Insights into the Retrograde Signaling Pathway between the Plastids and the Nucleus. *Plant Signal Behav.*, Vol. 5, No.2, (February, 2010), pp. 196-199, ISSN 1559-2316

Kocsy, G.; Galiba, G. & Brunold, C. (2001). Role of Glutathione in Plants. *Physiol. Plant.*, Vol. 113, No.2, (October, 2001), pp. 158-164, ISSN 0032-0889

Koussevitzky, S.; Nott, A.; Mockler, T.C.; Hong, F.; Sachetto-Martins, G.; Surpin, M.; Lim, J.; Mittler, R. & Chory, J. (2007). Signals from Chloroplasts Converge to Regulate Nuclear Gene Expression. *Science,* Vol.316, No.5825, (May, 2007), pp. 715–719, ISSN 0036-8075

Kubo, T. & Newton, K.J. (2008). Angiosperm Mitochondrial Genomes and Mutations. *Mitochondrion*, Vol. 8, No.1, (Novermber, 2007), pp. 5-14, ISSN 1567-7249

Kusnetsov, V.; Bolle, C.; Lubberstedt, T.; Sopory, S.; Herrmann, R.G. & Oelmüller, R. (1996). Evidence That the Plastid Signal and Light Operate via the Same Cis-Acting Elements in the Promoters of Nuclear Genes for Plastid Proteins. *Mol. Gen. Genet.*, Vol.252, No.6, (October, 1996), pp. 631-639.

Kwak, J.M.; Nguyen, V. & Schroeder, J.I. (2006). The Role of Reactive Oxygen Species in Hormonal Responses. *Plant Physiol.*,Vol.141, No.2, (June, 2006), pp. 323-329, ISSN 1532-2548

La Rocca, N.; Rascio, N.; Oster, U. & Rüdiger, W. (2001). Amitrole Treatment of Etiolated Barley Seedlings Leads to Deregulation of Tetrapyrrole Synthesis and to Reduced Expression of *Lhc* and *RbcS* Genes. *Planta*, Vol.213, No.1, (May, 2001), pp. 101–108, ISSN 0032-0935

Larkin, R.M. & Ruckle, M.E. (2008). Integration of Light and Plastid Signals. *Curr. Opin. Plant Biol.*, Vol.11, No.6, (October, 2008), pp. 593–599. ISSN 1369- 5266

Leister, D. (2005). Genomics-Based Dissection of the Cross-Talk of Chloroplasts with the Nucleus and Mitochondria in Arabidopsis. *Gene*, Vol. 354, No.12, (October, 2005), pp. 110-116, ISSN 0378-1119

Mackenzie, S. & McIntosh, L. (1999). Higher Plant Mitochondria. *Plant Cell*, Vol.11, No.4, (April, 1999), pp. 571-586, ISSN 1040-4651

Mochizuki, N.; Brusslan, J.A.; Larkin, R.; Nagatani, A. & Chory, J. (2001). Arabidopsis Genomes Uncoupled (GUN 5) Mutant Reveals the Involvement of Mg-chelatase H Subunit in Plastid-to-nucleus Signal Transduction. *Proc. Natl. Acad. Sci. USA,* Vol.98, No.4, (February, 2001), pp. 2053–2058, ISSN 1091-6490

Mochizuki, N.; Tanaka, R.; Grimm, B.; Masuda, T.; Moulin, M.; Smith, A.G.; Tanaka, A. & Terry, M.J. (2010) The Cell Biology of Tetrapyrroles: a Life and Death Struggle. *Trends Plant Sci.*, Vol. 15, No.9, (July, 2010), pp. 488–498, ISSN 1360-1385

Mochizuki, N.; Tanaka, R.; Tanaka, A.; Masuda, T. & Nagatani, A. (2008). The Steady-state Level of Mg protoporphyrin IX is not a Determinant of Plastid-to-nucleus Signaling

in Arabidopsis. *Proc. Natl. Acad. Sci. USA*, Vol.105, No.39, (September, 2008), pp. 15184–15189, ISSN 1091-6490

Moulin, M.; McCormac, A.C.; Terry, M.Y. & Smith, A.G. (2008). Tetrapyrrole Profiling in Arabidopsis Seedlings Reveals that Retrograde Plastid Nuclear Signaling is not Due to Mg-protoporphyrin IX Accumulation. *Proc. Natl. Acad. Sci. U S A,* Vol. 105, No.39, (September, 2008), pp. 15178–15183, ISSN 1091-6490

Newton, K.J.; Gabay-Laughnan, S. & DePaepe, R. (2004). Mitochondrial Mutations in Plants, In: *Advances in Photosynthesis and Respiration.* Day, D.A., Millar, A.H., Whelan J. (Eds.), pp. 121-142, Kluwer, ISBN 978-90-481-3882-1, Dordrecht. Netherlands

Nott, A.; Jung, H.-S.; Koussevitzky, S. & Chory, J. (2006). Plastid-to-Nucleus Retrograde Signaling. *Annu. Rev. Plant. Biol.*, Vol. 57, pp. 739-759, ISSN 1040-2519

Odintsova, M.S. & Yurina, N.P. (2006) Chloroplast Genomics of Land Plants and Algae, In: *Biotechnological Applications of Photosynthetic Proteins: Biochips, Biosensors and Biodevices*, Giardi M.T., Piletska E.V., (Eds.), pp. 57-72, Landes Bioscience/Eurekah. Com., ISBN 0-387-33009-7, Georgetown (Texas), USA

Oelmüller, R.; Levitan, I.; Bergfeld, R.; Rajasekhar, V.K. & Mohr, H. (1986). Expression of Nuclear Genes as Affected by Treatments Acting on the Plastids. *Planta*, Vol. 168, No.4, (November, 1986), pp. 482-492, ISSN 0032-0935

Osipenkova, O.V.; Odintsova, M.S. &Yurina, N.P. (2010). The Influence of Light, Hormonal, and Carbohydrate Signal Systems on ELIP genes expression in gun_mutants Arabidopsis thaliana. *Applied Biochemistry and Microbiology*, Vol. 46, No. 3, pp. 331–338. ISSN 0003_6838

Oswald, O.; Martin, T.; Dominy, P.J. & Graham, I.A. (2001). Plastid Redox State and Sugars: Interactive Regulators of Nuclear-encoded Photosynthetic Gene Expression. *Proc. Natl. Acad. Sci. U S A*, Vol. 98, No.4, (January, 2001), pp. 2047-2052, ISSN 1091-6490

Pesaresi, P.; Masiero, S.; Eubel, H.; Braun, H-P.; Bhushan, S.; Glaser, E.; Salamini, F. & Leister, D. (2006). Nuclear Photosynthetic Gene Expression Is Synergistically Modulated by Rates of Protein Synthesis in Chloroplasts and Mitochondria. *The Plant Cell*. Vol.128, No.4, (March, 2006), pp. 970-991, ISSN 1040-4651

Pesaresi, P.; Schneider, A.; Kleine, T. & Leister, D. (2007) Interorganellar Communication. *Curr. Opinion in Plant Biology*, Vol.10, No.6, (August, 2007), pp. 600-606, ISSN 1369-5266

Pfannschmidt, T. & Liere, K. (2005). Redox Regulation and Modification of Proteins Controlling Chloroplast Gene Expression. *Antioxidants Redox Signal*, Vol.7, No.5-6, (May-June, 2005), pp. 607-618, ISSN 1523-0864

Pfannschmidt, T. (2010). Plastidial Retrograde Signalling – a True ''Plastid Factor'' or Just Metabolite Signatures? *Trends Plant Sci*., Vol. 15, No.8, (January, 2010), pp. 427–435, ISSN 1360-1385

Piippo, M.; Allahverdiyeva, Y.; Paakkarinen, V.; Suoranta, U.-M.; Battchikova, N. & Aro, E.-M. (2006). Chloroplast-Mediated Regulation of Nuclear Genes in Arabidopsis thaliana in the Absence of Light Stress. *Physiol. Genomics*. Vol. 25. pp. 142-152, ISSN 1094-8341

Pogulskaya, E.N.; Yurina, N.P. & Karapetyan, N.V. (2006) Tetrapyrrole Involvement in Expression Regulation of a Nuclear Gene of Low-Molecular-Weight Plastid Protein ELIP. *Applied Biochemistry and Microbiology*, Vol. 42, No. 3, pp. 320–325, ISSN 0003-6838

Rhoads, D.M. & Subbaiah, C.C. (2007). Mitochondrial Retrograde Regulation in Plants. *Mitochondrion*. Vol.7, No.3, (May, 2007), pp. 177-194, ISSN 1567-7249

Rhoads, D.M. & Vanlerberghe, G.C. (2004). Mitochondria-Nucleus Interactions, Evidence for Mitochondrial Retrograde Communication in Plant Cells, In: *Advances in Photosynthesis and Respiration.* Day, D.A., Millar, A.H., Whelan J. (Eds.), Vol. 17, pp. 83-106, Kluwer Academic Publishers, ISBN 978-90-481-3882-1, Dordrecht. Netherlands

Rhoads, D.M.; White, S.J.; Zhou, Y.; Muralidharan, M. & Elthon, T.E. (2005). Altered Gene Expression in Plants with Constitutive Expression of a Mitochondrial Small Heat Shock Protein Suggests the Involvement of Retgrograde Regulation in the Heat Stress Response. *Physiol. Plant.*, Vol.123, No.4, (March, 2005), pp. 435-444, ISSN 0031-9317

Richly, E.; Dietzmann, A.; Biehl, A.; Kurth, J.; Laloi, C.; Apel, K.; Salamini, F. & Leister, D. (2003). Covariations in the Nuclear Chloroplast Transcriptome Reveal a Regulatory Master-Switch. *EMBO Rep.*, Vol.4, No.5, (May, 2003), pp. 491-498, ISSN 1469-221X

Robson, A.C. & Vanlerberghe, G.C. (2002). Transgenic Plant Cells Lacking Mitochondrial Alternative Oxidase Have Increased Susceptibility to Mitochondria-Dependent and -Independent Pathways of Programmed Cell Death. *Plant Physiol*, Vol.129, No.4, (August, 2002), pp. 1908-1920, ISSN 1532-2548

Sherameti, I.; Nakamura, M.; Yamamoto, Y.Y.; Pfannschmidt, T.; Obokata J. & Oelmüller R. (2002). Polyribosome Loading of Spinach mRNAs for Photosystem I Subunits Is Controlled by Photosynthetic Electron Transport. A Crucial Cis Element in the Spinach PsaD Gene Is Located in the 5′-Untranslated Region. *Plant J.*, Vol. 32, No.5, (December, 2002), pp. 631-639, ISSN 0960-7412

Strand, A.; Asami, T.; Alonso, J.; Ecker, J.R. & Chory, J. (2003). Chloroplast to Nucleus Communication Triggered by Accumulation of Mg-protoporphyrin IX. *Nature*, Vol. 421, No.6918, (January, 2003), pp. 79–83, ISSN 0028-0836

Subbaiah, C.C. &, Sachs, M.M. (2003). Molecular and Cellular Adaptations of Maize to Flooding Stress. *Ann. Bot.*, Vol. 91, No.2, (January, 2002), pp. 119-127, ISSN 0003-3847

Subbaiah, C.C.; Bush, D.S. & Sachs, M.M. (1998). Mitochondrial Contribution to the Anoxic Ca^{2+} Signal in Maize Suspension-Cultured Cells. *Plant Physiol.*, Vol.118, No.3, (November, 1998), pp. 759-771, ISSN 1532-2548

Subbaiah, C.C.; Palaniappan, A.; Duncan, K.; Rhoads, D.M. & Huber, S.C. (2006). Mitochondrial Localization and Putative Signaling Function of Sucrose Synthase in Maize. *J. Biol. Chem.,* Vol. 81, No.23, pp. 15625-15635, ISSN 0021-9258

Susek, R.E.; Ausubel, F.M. & Chory, J. (1993). Signal Transduction Mutants of Arabidopsis Uncouple Nuclear CAB and RBCS Gene Expression from Chloroplast Development. *Cell,* Vol.74, No.5, (September, 1993), pp. 787-799, ISSN 0092-8674

Sweetlove, L.J. & Foyer, C.H. (2004). Roles for Reactive Oxygen Species and Antioxidants in Plant Mitochondria Plant Mitochondria: From Genome to Function, In: *Advances in Photosynthesis and Respiration.* Day, D.A., Millar, A.H., Whelan J. (Eds.), Vol. 17, pp. 307-320, Kluwer, ISBN 978-90-481-3882-1, Dordrecht. Netherlands

Taylor, N.L.; Day, D.A. & Millar, A.H. (2002). Environmental Stress Causes Oxidative Damage to Plant Mitochondria Leading to Inhibition of Glycine Decarboxylase. *J. Biol. Chem.*, Vol.277, No.45, (November, 2002), pp. 42663-42668, ISSN 0021-9258

Voigt, C.; Oster, U.; Börnke, F.; Jahns, P.; Dietz, K.J.; Leister, D. & Kleine, T. (2010). In-depth Analysis of the Distinctive Effects of Norflurazon Implies that Tetrapyrrole Biosynthesis, Organellar Gene Expression and ABA Cooperate in the GUN-type of Plastid Signalling. *Physiol. Plant*. Vol.138, No.4, (December, 2009), pp. 503-519, ISSN 0031-9317

Voss, B.; Meinecke, L.; Kurz, T.; Al-Babili, S.; Beck, C.F. & Hess, W.R. (2011). Hemin and Mg-protoporphyrin IX Induce Global Changes in Gene Expression in *Chlamydomonas reinhardtii*. *Plant Physiol*., Vol.155, No.2, (December, 2010), pp. 892-905, ISSN 1532-2548

Vranova, E.; Inze, D. & Van Breusegem, F. (2002). Signal Transduction during Oxidative Stress. *J. Exp. Bot*., Vol.53, No.372, (May, 2002), pp. 1227-1236, ISSN 0022-0957

Wagner, D.; Przybyla, D.; Camp, R.; Kim, C.; Landgraf, F.; Lee, K.P.; Würsch, M.; Laloi, C.; Nater, M.; Hideg, E. & Apel, K. (2004). The Genetic Basis of Singlet Oxygen-Induced Stress Responses of Arabidopsis thaliana. *Science,* Vol.306, No.5699, (November, 2004), pp. 1183-1185, ISSN 0036-8075

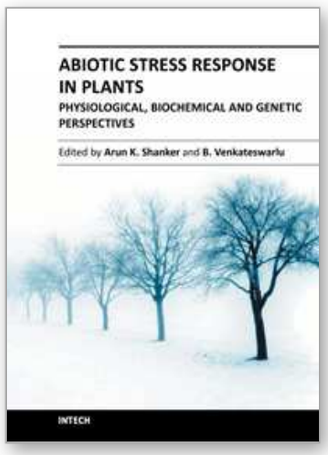
Yabuta, Y.; Maruta, T.; Yoshimura, K.; Ishikawa, T. & Shigeoka, S. (2004). Two Distinct Redox Signaling Pathways for Cytosolic APX Induction under Photooxidative Stress . *Plant Cell Physiol*., Vol. 45, No.11, (Novemer, 2004), pp. 1586-1594, ISSN 0032-0781

Yurina, N.P. & Odintsova, M.S., (2007) Plant Signaling Systems. Plastid_Generated Signals and Their Role in Nuclear Gene Expression. *Russ. J. Plant Physiol*., Vol.54, No.4, pp. 427–438, ISSN 1021-4437

Yurina, N.P.; Pogulskaya, E.N. & Karapetyan, N.V.. (2006) Effect of Photodestruction of Plastids from Norflurazon_Treated Barley Seedlings on Expression of Nuclear Genes Encoding Chloroplast Stress Proteins. *Biochemistry (Moscow*), Vol.71, No.4, pp. 430-436, ISSN 0006_2979

Zarkovic, J.; Anderson, S.L. & Rhoads, D.M. (2005). A Reporter Gene System Used to Study Developmental Expression of Alternative Oxidase and Isolate Mitochondrial Retrograde Regulation Mutants in Arabidopsis. Plant Mol. Biol., Vol.57, No.6, pp. 871-888, ISSN 1573-5028

Zhang, Z.W.; Yuan, S.; Feng, H.; Xu, F.; Cheng, J.; Shang, J.; Zhang, D.W. & Lin, H.H. (2011) Transient Accumulation of Mg-protoporphyrin IX Regulates Expression of PhANGs - New Evidence for the Signaling Role of Tetrapyrroles in Mature *Arabidopsis* plants. J. Plant Physiol., Vol.168, No.7, (January, 2011), pp. 714-721, ISSN 1532-2548



Abiotic Stress Response in Plants - Physiological, Biochemical and Genetic Perspectives

Edited by Prof. Arun Shanker

ISBN 978-953-307-672-0
Hard cover, 346 pages
Publisher InTech
Published online 29, August, 2011
Published in print edition August, 2011

Plants, unlike animals, are sessile. This demands that adverse changes in their environment are quickly recognized, distinguished and responded to with suitable reactions. Drought, heat, cold and salinity are among the major abiotic stresses that adversely affect plant growth and productivity. In general, abiotic stress often causes a series of morphological, physiological, biochemical and molecular changes that unfavorably affect plant growth, development and productivity. Drought, salinity, extreme temperatures (cold and heat) and oxidative stress are often interrelated; these conditions singularly or in combination induce cellular damage. To cope with abiotic stresses, of paramount significance is to understand plant responses to abiotic stresses that disturb the homeostatic equilibrium at cellular and molecular level in order to identify a common mechanism for multiple stress tolerance. This multi authored edited compilation attempts to put forth an all-inclusive biochemical and molecular picture in a systems approach wherein mechanism and adaptation aspects of abiotic stress are dealt with. The chief objective of the book hence is to deliver state of the art information for comprehending the effects of abiotic stress in plants at the cellular level.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Nadezhda Yurina and Margarita Odintsova (2011). Plant Organelles-to-Nucleus Retrograde Signaling, Abiotic Stress Response in Plants - Physiological, Biochemical and Genetic Perspectives, Prof. Arun Shanker (Ed.), ISBN: 978-953-307-672-0, InTech, Available from: http://www.intechopen.com/books/abiotic-stress-response-in-plants-physiological-biochemical-and-genetic-perspectives/plant-organelles-to-nucleus-retrograde-signaling1

INTECH
open science | open minds

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

IntechOpen