# Photosynthesis, a global sensor of environmental stress in green plants: stress signalling and adaptation†

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**Photosynthesis serves as a global stress sensor in plants, algae and cyanobacteria. In this overview, we focus on higher plants only. Although several structural and functional components of the photosynthetic apparatus are responsive to stress, photosystem II (PS II) and ribulose-1,5-bisphosphate carboxylase oxygenase (Rubisco) act as the major stress sensors. Stress sensing is primarily reflected in cellular energy imbalance, which, in this review, is discussed in terms of perturbation in photostasis, imbalance in redox homeostasis and changes in cellular sugar level. Signals generated by these changes bring about photochemical, metabolic and molecular reprogramming for stress adaptation through different signal transduction pathways. Recent redox and sugar signalling models, which explain stress response of green plants, are discussed here. This review concludes with a brief description of some of the challenging and unresolved areas of stress study, which we hope would be addressed in the near future.**

**Keywords:** Photostasis, photosynthesis, redox homeostasis, stress sensor, sugar signalling.

ALL environmental stresses, irrespective of their targets and nature of perception in plants, algae and cyanobacteria bring perturbation in the cellular energy homeostasis. The stress-adaptive mechanisms developed by these photosynthetic organisms are primarily based on re-establishment of cellular energy balance[1,2]. In this background, photosynthesis, the energy-producing process, plays a central role in modulating energy signalling and balance which have significant implications for energy homeostasis of the whole organism. In this review, we have focused on higher plants, i.e. the green plants. Although molecular details of stress response of plants are yet to be worked out, new and novel ideas are emerging in recent years on the perception of stress signal and its transduction into appropriate metabolic response in photosynthetic tissues. For the basics of photosynthesis, see Rabinowitch and Govindjee[3], and Blankenship[4].

This overview discusses the thesis that photosynthesis serves as a global sensor of environmental stress that induces cellular energy imbalance as reflected in the distinct alteration in redox chemistry associated with thylakoid membranes and alteration in cellular sugar status. The signals generated by these changes develop several signal transduction pathways for metabolic and molecular reprogramming for final stress response that ends with either cellular death or survival. For detailed information on abiotic stress in plants, the readers are referred to edited books in the field[5–7].

## Chloroplasts: the sensor of stress

Photosynthesis is a process by which plants, algae and photosynthetic bacteria convert solar energy into chemical energy of organic molecules[3,4]. The process in plants and algae occurs in chloroplasts involving a series of photochemical and biochemical reactions. The light energy absorbed by pigments initiates the reactions leading to primary photochemical reactions associated with the two photosystems: photosystem I (PS I)[8] and photosystem II (PS II)[9], that are interconnected with several redox components. This is followed by reactions that terminate in the production of stable organic compounds[10]. The photosynthetic process is conveniently divided into the 'light reactions' that drive electron transport from water to $NADP^+$ in the thylakoid membranes and the 'dark reactions' involving $CO_2$ fixation that occur in the stroma of chloroplasts. It is important to note, however, that the

†This is the fifth article on the theme 'Photosynthesis and the Global Issues', being guest-edited by Govindjee, George C. Papageorgiou and Baishnab C. Tripathy. The first article by Lars Olof Björn and Govindjee discussed the evolution of photosynthesis and the chloroplasts and was published in 2009 (**96**, 1466–1474). The second article, by Maria Ghirardi and Prasanna Mohanty, discussed hydrogen production by algae, and was published in 2010 (**98**, 499–507). The third article by Gernot Renger published in 2010, discussed the light reactions of photosynthesis (**98**, 1305–1319). The fourth article by Attipalli R. Reddy, *et al.* was also published in 2010 (**99**, 46–57). Here, the authors discussed the impact of global elevated $CO_2$ concentration on photosynthesis and plant productivity.

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only true light reactions are when light energy is converted to chemical energy at the reaction centres of PS I and II. These two sets of reactions not only occur in different locations within the chloroplast, but the time-spans of different reactions associated with them are also different. Nevertheless, the process is highly integrated and well regulated, and is sensitive to environmental changes[11].

The modifications of the chloroplast in response to various environmental stresses have been widely studied in different laboratories and, thus the literature in the area is vast. The stress is sensed at the levels of pigment composition, structural organization, primary photochemistry and the $CO_2$ fixation[12,13]. Excellent reviews are available on the photosynthetic response of plants to various environmental stresses[6,13–22]. During the last several years, our laboratories in Orissa have been involved in monitoring photosynthetic changes primarily at the level of photochemical reactions associated with thylakoid membranes of chloroplasts in the leaves of higher plants experiencing dark stress[23,24], water stress[25,26], high light stress[27–29], UV stress[30–32] and flooding stress[33]. These stresses alter the rates of primary photochemical reactions associated with the process and show significant loss in the activity of the enzymes of the Calvin–Benson cycle[34,35]. Reports showing quantitative loss in photosynthetic pigments, differential changes in chlorophyll *a* and chlorophyll *b*, remarkable changes in carotenoid composition in general, and alteration in the composition of xanthophyll cycle pigments in particular, in response to these stress factors are available in the literature[12]. Among all the components of the photosynthetic apparatus that are known as the targets of stress, PS II and ribulose-1,5-bisphosphate carboxylase oxygenase (Rubisco) are considered as the major stress sensors[12,13]. In natural environment, most of the stresses are sensed by photosynthetic organisms through these components. PS I is known to be relatively less sensitive, than PS II, to most stresses, but it is highly sensitive to stresses such as freezing[12].

## Structural features of PS II that make this photosystem sensitive to stress

PS II is a protein complex containing several polypeptides as subunits and chemical moieties responsible for electrochemical reactions (Figure 1). Both PS I and PS II are photosensitive devices which absorb photons, transfer them as excitons, trap them and convert them to electrochemical energy resulting in photoredox reactions[10]. They act in the timescale from femtoseconds to milliseconds. The photoredox reactions result in the evolution of $O_2$ by the oxidation of water at the $Mn_4Ca$ metal complex on the electron donor side of PS II and the reduction of plastoquinone (PQ) to plastoquinol ($PQH_2$)[10]. A recent crystallographic study of PS II at 1.9 Å resolution[36] would add to our current understanding of the structure and the function of this photosystem.

The photosensitivity of PS II gives it the ability to act as an environmental sensor. Light, which governs the photosynthetic process, is the major component of the environment. Excess light causes oxidative damage to the photosensor, mainly PS II. Increase in the intensity of light may cause an increase in the oxidized species on the donor side, or the reduced species on the acceptor side of PS II, throwing off the redox balance. The system tries to nullify the adverse effects of the environment as far as possible. Most of the abiotic stresses ultimately result in oxidative stress. Thus PS II senses the stress and transmits signals for appropriate response[13].

The most sensitive part of PS II is the metal centre at the oxygen-evolving complex. Its high sensitivity is associated with the Mn cluster, which forms stable metal centres with high oxidation states of Mn (III or IV). A still higher oxidation state (V) may be formed during the oxidation of water. Stress may lead to the production of a high population of high oxidation states, with a tendency to oxidize the neighbouring chemical species, like ligand residues of the protein and, in turn, Mn getting reduced to Mn (II)[37]. Once Mn is reduced to the oxidation state II, stability of the metal complex decreases and the metal centre breaks down, Mn ions may then leave the centres. On the acceptor side of PS II, the PQ pool may have a

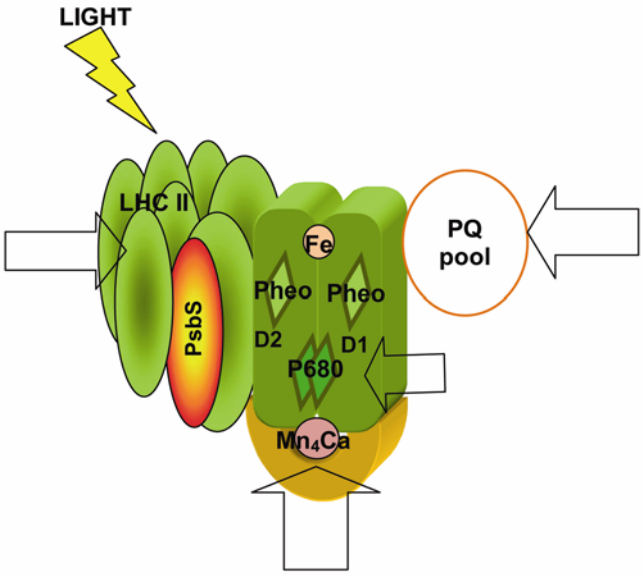


**Figure 1.** An artistic representation of some important environmental sensing subunits of photosystem II (PS II). The sensitivity is depicted by the thickness of the arrows pointing to the respective subunits. LHC II, Light harvesting complex of PS II; PsbS, Protein associated with non-photochemical quenching; P680, A special pair of chlorophyll *a* which acts as an exciton trap and primary electron donor of PS II; Pheo, Pheophytin *a* acting as a primary electron acceptor of PS II; $Mn_4Ca$, Oxygen evolving metal complex of PS II; Fe, Non-heme iron complex of PS II; D1, D2, Core protein subunits of PS II and PQ, Plastoquinone.

higher population of reduced $PQH_2$. Accumulation of reduced PQ may lead to the formation of toxic oxygen species, which may generate a cascade of signals modulating stress acclimation of the system[38].

The light-harvesting chlorophyll *a* proteins of PS II (LHC II), that absorb most of the light energy for PS II, respond to stress by reorganizing their association and decelerating the rate of the redox processes at PS II. A protein, PsbS, which is involved in the photoprotection of many plants, is believed to sense high light stress and regulate the organization of LHC II (ref. 29). Nature might have tailored the weak protein–protein interaction to facilitate re-organization or migration of LHC II. When the plant is under stress, the weak interaction also results in the dissociation of LHC II from the core proteins, i.e. D1, D2, Cytb559, 43 kDa (CP43) and 47 kDa (CP47) polypeptides of the PS II complex. This dissociation may prevent further photodamage of the PS II core polypeptides under stress. The mechanism of PS II sensing of stress and adaptation to stress at the molecular level remains to be fully understood.

## General features of stress-sensing mechanisms in plants

Plants have developed mechanisms for stress adaptation, defence and repair. These mechanisms involve stress perception, transduction of stress signal, and what we may call the 'final response'. Even in the most stressful environment, plants can efficiently sense the stress, correctly recognize the resulting signal(s) and use them as cues to bring forth specific changes at various levels in structure, physical and physiological behaviour, biochemical pathways, and expression of stress-specific genes as a response[7]. Photosynthetic tissues experiencing stress exhibit dynamic stress adaptation behaviour and tend to maintain optimal photosynthetic efficiency as much as possible[11–13,22,39].

The events associated with chloroplast-specific stress sensing mechanisms and responses of plants experiencing various environmental stresses in the presence of light are depicted in Figure 2 (modified after Biswal *et al.*[22]). Figure 2 shows the major sensors of different environmental stresses which the plants experience as monitored under laboratory conditions or in different combinations in the field. The stress-induced changes in the sensors result, in energy imbalance, changes in the redox status of electron transport components of thylakoids and alteration of cellular sugar level. These changes are suggested to initiate signalling cascades that lead to physical and molecular readjustments for stress adaptation. Short-term adaptation includes non-photochemical quenching (NPQ) of excess unused quanta, and modulation of toxic oxidative environment through several antioxidant systems. Expression of stress-responsive genes regulated by redox and sugar signalling is a long-term adaptive strategy of plants (Figure 2).

## Alteration in photostasis, signalling and adaptation

To provide a clear picture of stress perception and response, all the structural and functional features of chloroplasts are grouped into three basic components: light energy harvesting system (energy 'source'), utilization of energy by carbon, nitrogen and sulphur assimilation processes (energy sink), and channelling of energy from source to sink through a series of redox reactions associated with the electron transport chain of thylakoids. Stress sensing mechanisms of chloroplasts have primarily developed through source, sink and redox components of electron transport chains as the sensors of stress. These components not only coordinately carry on the photosynthetic process, but also make the process the global sensor of stress. The stress is decoded basically by energy imbalance between the source and the sink[40,41]. The changes induced by environmental stress in the activity of the source (determined by the redox state of the chloroplast) and the capacity of the sink (determined primarily by carbon and nitrogen metabolism) generate a signalling system for the expression of specific genes leading to modification in cellular metabolism for photosynthetic adaptation[40]. Photosynthesis maximizes the use of absorbed light energy in carbon and nitrogen assimilatory processes at sink, and also dissipates excess unused quanta at the source.

Under normal conditions, there exists a balance between the source and the sink, which is referred to as photostasis[40] (Figure 3). Several thought-provoking articles have been published that provide an understanding of the complexities of photostasis in plants[40–42]. The energy balance state has been expressed mathematically by Falkowski and Chen[43] as follows:

$$\sigma_{\mathrm{PS\ II}} E_{\mathrm{k}} = \tau^{-1}, \tag{1}$$

where $\sigma_{\mathrm{PS\ II}}$ is the effective absorption cross-section of PS II, $E_{\mathrm{k}}$ the irradiance at which the maximum photosynthetic quantum yield balances the photosynthetic capacity, and $\tau^{-1}$ the rate at which the photosynthetic electrons are utilized by the sink.

If the stress is severe, a significant change in photostasis may result in the death of the plant. However, plants can survive by restoring photostasis through modulation either at the source, the sink or both. Although eq. (1) explains certain physiological conditions in chloroplasts, it is quite complex since the phenomenon is linked to several metabolic pathways. Both the source efficiency and the sink capacity could be modulated by several factors. For example, the metabolic energy sink could be

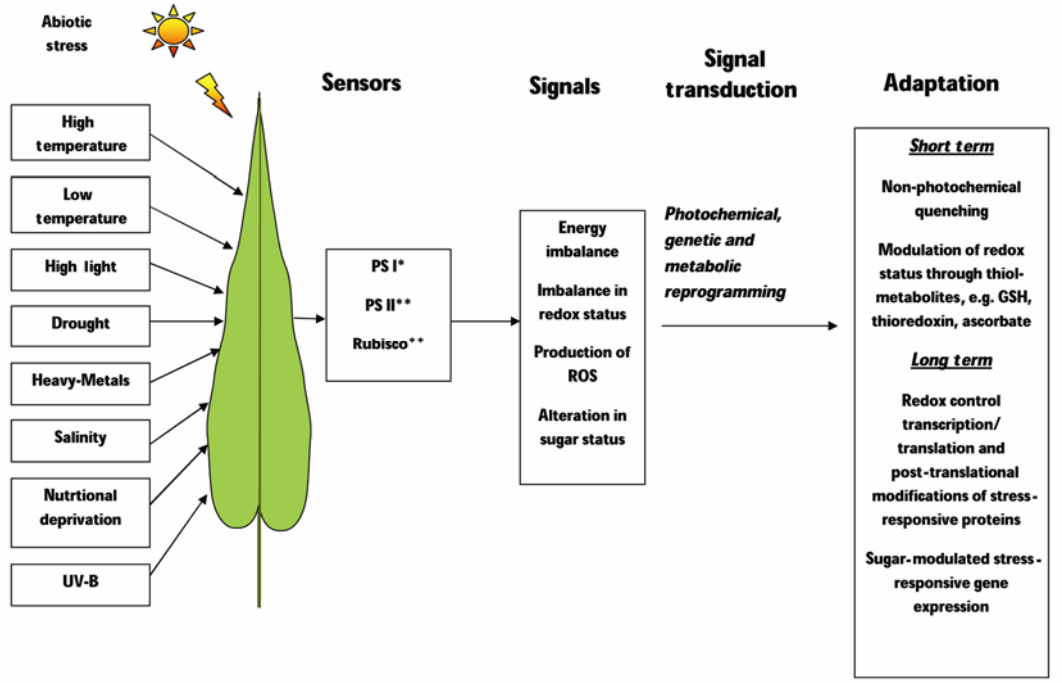


**Figure 2.** A scheme depicting a green leaf as the major target of environmental stresses, both PS I and PS II, and Rubisco being the primary stress sensors in the many chloroplasts in the leaf. The stress-induced changes in the sensors generate the stress signals like energy imbalance, redox changes associated with electron transport system, production of reactive oxygen species (ROS) and changes in the sugar level. These changes through several signal transduction pathways result in photochemical, metabolic and molecular reprogramming for stress adaptation. The leaf exhibits short-term adaptational mechanisms like state transition with a change in PS II absorption cross-section, alteration in PS II : PS I stoichiometry and dissipation of excess energy as heat through non-photochemical quenching of chlorophyll excited state. Extensive gene expressions, both nuclear and plastidic, regulated by stress-induced alteration in redox status of electron transport components and in the content of cellular sugars are associated with long-term stress adaptation. Stress sensing component of chloroplast: PS II and Rubisco are more sensitive than PS I (modified after Biswal *et al.*[22]). GSH, Reduced glutathione.

linked to other cellular pathways, including respiration. A change in any point(s) of the pathways may generate photostasis signal. Photosynthetic organelles, however, have a dynamic acclimation capacity to integrate the stress-induced changes, restore photostasis and develop stress adaptive mechanisms[44]. Although the concept of photostasis is based on the response of photosynthetic organisms to low temperature[40], the results of imbalance in energy budget of photosynthesis induced by other abiotic stresses like high irradiance[45], nitrogen deficiency[46], and salt stress[47] are consistent with the above view.

Spatial and temporal complexity of photosynthesis makes photostasis prone to stress. The sequence of photosynthesis is known to cover a wide time-span and begins with photophysical and photochemical events, i.e. light absorption, excitation energy transfer and charge separation in the timescale of femtosecond ($10^{-15}$ s) to nanosecond ($10^{-9}$ s). This is followed by electron transport in the microsecond ($10^{-6}$ s) to millisecond ($10^{-3}$ s) range, and finally by enzyme-mediated reactions in the millisecond to second range (see Martin Kamen's classical figure depicted in Govindjee and Gest[48]). Relatively slow reactions are rate-limiting and thus, incompatible with the fast reactions. Further, the fast primary photochemical reactions are relatively stress-resistant compared to temperature-dependent slow enzyme-mediated reactions associated with the electron transport system and carbon dioxide fixation in the Calvin–Benson cycle. This results in the development of excitation pressure at the source. Since plants are photoautotrophs, light at any intensity in combination with other environmental stresses can bring a change in photostasis in terms of accumulation of excess unutilized quanta because of weakened sink demand induced by stress. In addition, high light always accumulates excess energy at the 'source'. NPQ of excess quanta at the source is one of the major processes for restoration of the balance and maintenance of photostasis.

NPQ takes into account the combined effects of different types of processes at PS II such as: (i) energy-dependent quenching (qE) of the excited state of chlorophyll; (ii) state transition related decrease ($q_T$), which is due to physical movement of mobile LHC II from PS II to PS I, and (iii) photoinhibitory quenching ($q_I$) of the excited state of chlorophyll. These three types of quenching (the second one not being a true quenching as it involves decrease in the concentration of fluorescent chlorophylls in PS II) are distinguished by their induction and relaxation kinetics, which are relatively slow in $q_T$

and $q_I$ compared to the faster qE type of quenching[49]. The qE type of quenching resulting from the energization of the thylakoid membrane, with the build-up of ΔpH, plays a dominant role in photoprotection under high light conditions. In fact, the qE type of antenna quenching, through the operation of xanthophyll cycle, has become the focus of recent research dealing with NPQ. It is basically a process that dissipates excess quanta through a kind of feedback de-excitation[50]. The interaction between PsbS and zeaxanthin, a xanthophyll, is associated with qE[29]. The direct involvement of PsbS in qE has been demonstrated in *Arabidopsis thaliana* mutant npq4-1 that lacks *PsbS* gene and loses its capacity for qE[50]. The mutant, however, exhibits normal light harvesting and photochemistry in its thylakoid membrane. Although the precise roles of PsbS and zeaxanthin in qE type of quenching remain unclear, a model is available, where a low pH-induced formation of PsbS–zeaxanthin complex is suggested to be responsible for this quenching[29]. In addition to the antenna quenching through qE, PS II reaction centre quenching is also considered as one of the important components of NPQ. It occurs slowly in the timescale of hours to days and significantly complements the quenching by qE and state transition[41,42,51]. For further information on photoprotection and photoinhibition, see relevant chapters in Demmig-Adams *et al.*[52]. Other factors for the restoration of photostasis include the following: (i) Changes in the antenna cross-section of PS II[53,54]. (ii) Alteration of the PS II : PS I stoichiometry[55]. (iii) Changes in the levels or activity of the Calvin–Benson cycle enzymes, enzymes associated with sucrose and starch biosynthesis[56], and enzymes associated with nitrogen and sulphur assimilation. (iv) A suitable combination of any of the above processes.

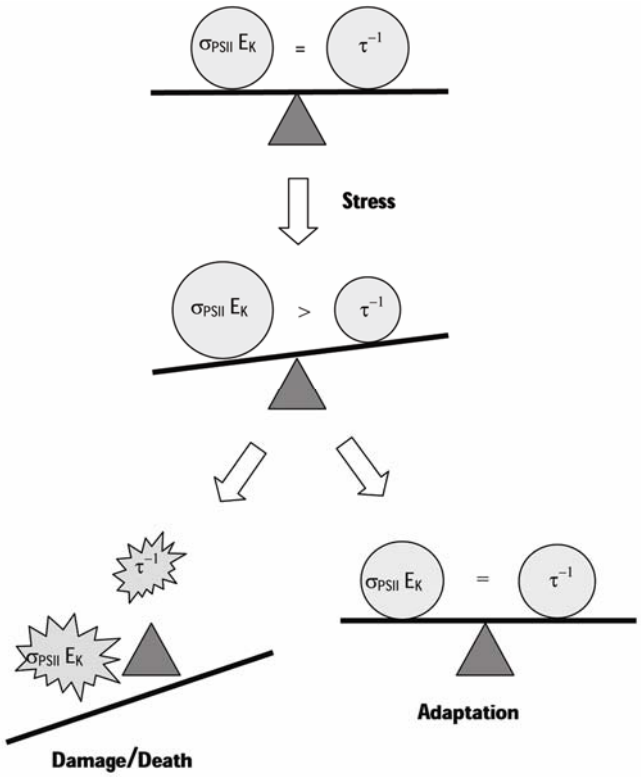


**Figure 3.** A diagrammatic representation of photostasis, the balance between the rate of energy supplied ($\sigma_{PS\ II}E_k$: source) and the rate of energy utilized ($\tau^{-1}$: sink), where $\sigma_{PS\ II}$ is the effective absorption cross-section of PS II, $E_k$ the irradiance at which the photosynthetic quantum yield is maximum; and $\tau^{-1}$ the rate at which the photosynthetically produced electrons are consumed by the terminal electron acceptor under light saturated conditions. Under normal conditions, the balance is represented by two circles of the same size. Stress-induced imbalance between these two factors is shown by two circles of different sizes, such that $(\sigma_{PS\ II}E_k) > \tau^{-1}$, which down-regulates the energy source, enhances the rate of energy utilization or adopts a suitable combination of both these processes to restore photostasis. The circles of the source and sink here are shown to be of the same size indicating restoration of photostasis that results in stress adaptation. Failure of adaptation (distorted source and sink) via either of the two above processes leads to cell death (modified after Ensminger *et al.*[40]).

The plants acclimatize to stress conditions by adopting an array of mechanisms instead of developing a stress-specific response to establish photostasis. In fact, the plant responses to energy starvation and/or energy excess condition encompass several signalling and stress responses.

## Redox signalling and stress adaptation

Redox chemistry of thylakoids couples the energy source and the sink. The redox active components of the electron transport chain maintain a redox homeostasis under normal conditions. But stress perturbs this homeostasis, which signals for several cellular readjustments for its restoration.

### *Redox state of plastoquinone modulates the overall redox balance of the system*

The redox state of the PQ pool plays a major role in maintaining the redox homeostasis. The stress-induced change in cellular energy is distinctly sensed by this redox active quinone, which generates appropriate signalling for homeostasis. For example, if either of the photosystems is selectively damaged by stress, the PQ pool of the intersystem electron transport chain is either reduced or oxidized. The reduced state of the PQ pool in cooperation with cytochrome (Cyt) $b_6f$ activates a LHC II-specific redox-sensitive kinase that phosphorylates a mobile LHC II. The phosphorylated LHC II moves towards PS I and gets attached to it[57]. On the other hand, when the PQ pool is oxidized LHC-specific kinases are deactivated and a phosphatase is activated, which de-phosphorylates mobile LHC II (ref. 57) resulting in relocation of LHC II to PS II. This state transition, a short-term response employed by the chloroplast, helps in redistributing the excitation energy between the two photosystems evenly to balance the redox state of the intersystem electron

transport chain (for a historical perspective on state changes, see Papageorgiou and Govindjee[58]). Although the kinases like Stt7 in the green alga, *Chlamydomonas reinhardtii*[59] and STN7 in *A. thaliana*[60] have been identified, the exact mode of activation/deactivation of the kinase by the reduced/oxidized PQ has not yet been understood. On the other hand, photosystem stoichiometry adjustment, a long-term response, involves favoured expression of PS I genes by reduced PQ and expression of PS II genes by oxidized PQ. The redox state of PQ pool determines not only the rate-limiting photosystem, but affects photosynthesis gene expression, both in the chloroplast and in the nucleus to bring forth changes in the stoichiometry of the two photosystems[12].

### *Reactive oxygen species and redox homeostasis*

Cellular redox homeostasis is significantly affected by stress-induced production of reactive oxygen species (ROS). ROS, however, generate signals for the synthesis of defence enzymes and other antioxidant systems against stress. Several models have been proposed to understand stress-related ROS metabolism[61]. Foyer and Noctor[62] have proposed a model suggesting interaction of ROS and antioxidants as an interface between metabolic and stress signals. They have proposed a possible ROS regulation mechanism of gene expression for metabolic readjustment under stressful environment. Although some basic information on ROS-induced expression of appropriate genes and their downstream regulation is available, the mechanism of ROS signalling and their role in cellular redox homeostasis is not yet clearly understood[63].

Data are available on ROS metabolism of chloroplasts and their contribution to cellular redox homeostasis in plants experiencing stress. Source–sink imbalance induced by stress usually disengages the electron transfer from normal pathways. When the electrons from PS I are transferred to molecular oxygen ($O_2$), ROS, such as $O_2^{\cdot-}$, $H_2O_2$ and $HO^{\bullet}$, are formed[64,65]. Further, the stress-mediated breakdown of the donor side of PS II enhances the life of $P_{680}^{+}$, a radical (the oxidized form of PS II reaction centre) with high oxidizing potential (1.17 V). The radical thus finds an opportunity to oxidize other pigments and the protein in its vicinity. Similarly, stress-induced inhibition at the electron acceptor side of PS II leads to the formation of the triplet state of $P_{680}$, $^3P_{680}$ by charge recombination of $Q_A^{-}/Q_B^{-}$ with $P_{680}^{+}$. $^3P_{680}$ can transfer its energy to $O_2$, which is easily available in the vicinity of PS II, forming toxic singlet oxygen ($^1O_2$) (Figure 4).

Chloroplast system may, however, employ multiple antioxidant enzymes or chemical species as an adaptation strategy[66,67]. Redox species like $QH_2$, thioredoxin, glutathione and $H_2O_2$ are the possible redox regulators that produce defence systems, including antioxidative enzymes through gene expression[22,68]. These antioxidants act as redox buffers between the oxidants and the reductants in chloroplasts. The possible involvement of ROS signalling in modulating redox homeostasis is depicted in Figure 4, which shows the link of excitation pressure developed at PS I and PS II to redox signalling molecules that finally regulate gene expression required for stress adaptation.

## Stress-induced changes in photosynthetic production of sugar: sensing, signalling and molecular reprogramming

Sugar, in addition to its role as a major source of energy, acts as a signalling molecule in cellular and whole-plant metabolic network. Sugar production through photosynthesis and changes in its level at different environmental

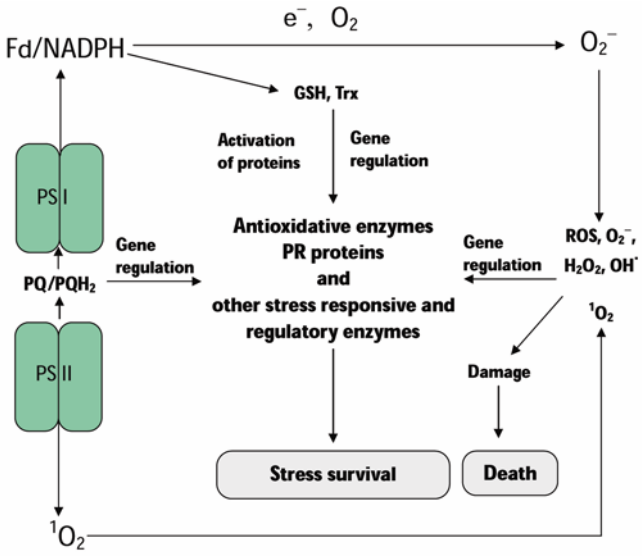


**Figure 4.** A model showing redox signalling network and specific signal transduction pathways leading to either adaptation or cell death. Development of antioxidant signalling and the downstream regulation of gene expression for the restoration of redox homeostasis lead to adaptation. The PQ pool that carries electrons from PS II to PS I, under over-reduction activates phosphorylation of a mobile LHC II and gene expression for adaptation. Imbalance distribution of light energy between PS I and PS II and/or impairment of $CO_2$ fixation build(s) excitation pressure and initiate(s) the production of superoxide radical ($O_2^{\cdot-}$) from the electron acceptor side of PS I or generate(s) singlet $O_2$ ($O_2^1$) due to a back reaction and the transfer of 'energy' from $P_{680}$ to $O_2$. The $O_2^{\cdot-}$ radicals generate hydrogen peroxide ($H_2O_2$) and hydroxyl radicals ($HO^{\bullet}$). The ROS (reactive oxygen species) generated by both the photosystems under stress regulate the gene expression for scavenging systems, including enzymes like superoxide dismutase (SOD), ascorbate peroxidase (APX), and other stress-responsive and regulatory enzymes. $PQH_2$ and ROS are shown as a class of signalling molecules that establish a link between the stress-induced excitation pressure and the gene expression for adaptation. Glutathione (GSH) and thioredoxin (Trx) are well known redox active molecules, which activate stress-related proteins and/or regulate stress-responsive gene expression. In photosynthetic tissues of higher plants, the presence of a large pool of these small molecular-weight antioxidants buffers the reductants and the oxidants for the maintenance of redox homeostasis (modified from Foyer and Noctor[62]).

conditions determine the growth, development and stress response[69]. Stress-induced changes in cellular status of sugar integrate carbon metabolism of photosynthesis, respiration and nitrogen in a well-regulated and coordinated manner[41].

Environmental stress can modulate the regulatory processes and results in metabolic changes at the cellular and the whole-plant level, specifically during source–sink transition. Sugar is known to modulate gene expression and enzyme activities, both in sugar-producing (source) and sugar-consuming (sink) tissues, thereby providing scope for the adaptation of carbon metabolism to the changing environmental conditions and to the availability of other nutrients[70,71]. In general, low sugar status enhances photosynthesis and reserve mobilization, whereas excess sugar stimulates growth and carbohydrate storage. The sensing of sugars, being the end-product of photosynthesis, guarantees an appropriate response of metabolism to specific situations.

Although stress-induced sugar sensing and signal transduction pathways are gradually being understood, analysis of the mechanisms of sugar-induced response is quite complex because of the dual functions of sugar, both as a nutrient as well as a signalling molecule. Sugar-linked signal transduction pathway is interconnected with other cellular signalling pathways. Extensive literature is available on the crosstalk between sugar signalling and the signalling pathways associated with ethylene, abscisic acid, cytokinins, auxin, G-protein and calcium[69,72,73]. In addition, interaction of sugar metabolism with nitrogen metabolism makes sugar signalling more complex[21]. The expression of several sugar-regulated genes is significantly modulated by the level of cellular nitrogen. Under stressful environment, the decline in photosynthetic efficiency may result in sugar-limited conditions in plants. Such a condition generates a signalling cascade that finally makes the energy available from other sources primarily by upregulating some specific catabolic processes[23,24] and thus protecting cells against nutrient stress. In fact, several dark inducible genes responsible for enhancing catabolic pathways have been identified[74]. These genes are expressed in dark-induced sugar starvation conditions. The role of one of the genes, *din2*, which codes for the cell wall-bound $\beta$-glucosidase is known in detail[23]. Research in this area has been extended in our laboratory[24]: we have demonstrated that darkness and water stress induce increase in the activity of cell wall-bound hydrolases, $\beta$-glucosidase[24] and $\beta$-glucanase (B. Biswal, unpublished), in the background of loss in photosynthesis. These enzymes cause the breakdown of cell-wall polysaccharides to sugars, and thus provide an alternative source of energy for the survival of plants under sugar-limiting conditions. The expression of the genes for these processes is suggested to be mediated by SnRK1 (sucrose non-fermenting 1-related kinase 1), the master regulator of transcription[21]. SnRK1 integrates all diverse sugar signalling and triggers catabolic processes necessary to sustain respiration and metabolic activity of energy-depriving cells.

The molecular mechanism of SnRK1 response to stress has been studied in *A. thaliana*[75]. In response to sugar deficit, SnRK1 in *Arabidopsis* is observed to repress many genes associated with cellular biosynthetic processes, including the synthesis of lipids, proteins, sucrose, starch, amino acids and nucleotides[69]. Downregulation of biosynthetic pathways is a strategy of the cell to conserve energy under stress conditions. On the other hand, stress causes induction of genes responsible for the degradation of starch, sucrose and polysaccharides associated with cell walls, lipids, proteins and amino acids. The requirement of catabolic products as the alternative sources of energy for stress survival, during photosynthesis-limiting condition induced by stress, is supported by the study of mutants with overexpression of KIN10, a member of SnRK1 family[75]. Compared to wild-type plants, the mutants exhibit higher capacity for stress survival and significant delay in the process of senescence when both the wild type and the mutants are grown in nutrient-deprived conditions. These data support the concept that SnRK1 triggers a convergent type of transcriptome that links genetic reprogramming to appropriate readjustment in cellular metabolism coping with the stress (Figure 5).

The mode of sugar signalling is different when stress causes an enhancement in the sugar level. Increase in sugar level can be induced by elevated concentration of atmospheric $CO_2$, in nitrogen-deficient environment and/or by changes in phloem loading/unloading for transport of sugar under different stress conditions. The increase in sugar level is sensed by hexokinase (HXK), a well-known conserved sugar sensor[21,72]. It is known that overproduction of sugar can suppress photosynthetic genes by HXK, leading to a loss in photosynthetic production of sugar. On the other hand, loss in sugars can upregulate photosynthetic genes to restore the loss. The stress, however, may not permit the recovery causing continuation of photosynthetic loss. Thus, the loss in photosynthesis recovery is likely to activate the catabolic pathways for alternative sources of energy through the SnRK1 pathway. However, the precise meeting point of both the sugar sensors, HXK and SnRK1, remains unresolved.

## Looking at the future: open questions

The following are the unresolved areas that must be addressed in the future.

(1) Photosynthesis in plants is undoubtedly a major sensor of stress. The process is likely to integrate all stresses into a cellular response with a stress-adaptive programme. But it is a challenge to understand how the process integrates the diverse stress signals, which act in time and space with different intensities. Photostasis redox homeostasis and sugar balance in plants are closely

interconnected and interdependent. They have been usually studied separately in different contexts with limited objectives. Stress-induced perturbation in any area can bring changes in the other areas. However, spatial and temporal integration of the photosystems overloaded with absorbed light, redox signalling generated from electron transport system in chloroplasts and changes in cellular sugar status in stressful environment largely remain unclear.

(2) The interaction of ROS and antioxidants constitutes an interface of the signalling generated from environmental stress and metabolic response, which significantly modulates redox homeostasis and stress adaptation in green plants. Chemistry of the interface is poorly understood, although several models have been proposed in this area of study[62]. The signal transduction from ROS produced by chloroplasts under stress conditions and gene expression in the nucleus for production of antioxidants through downstream regulation remains hypothetical. Although the interplay between the two is being slowly understood[62], it needs further research.

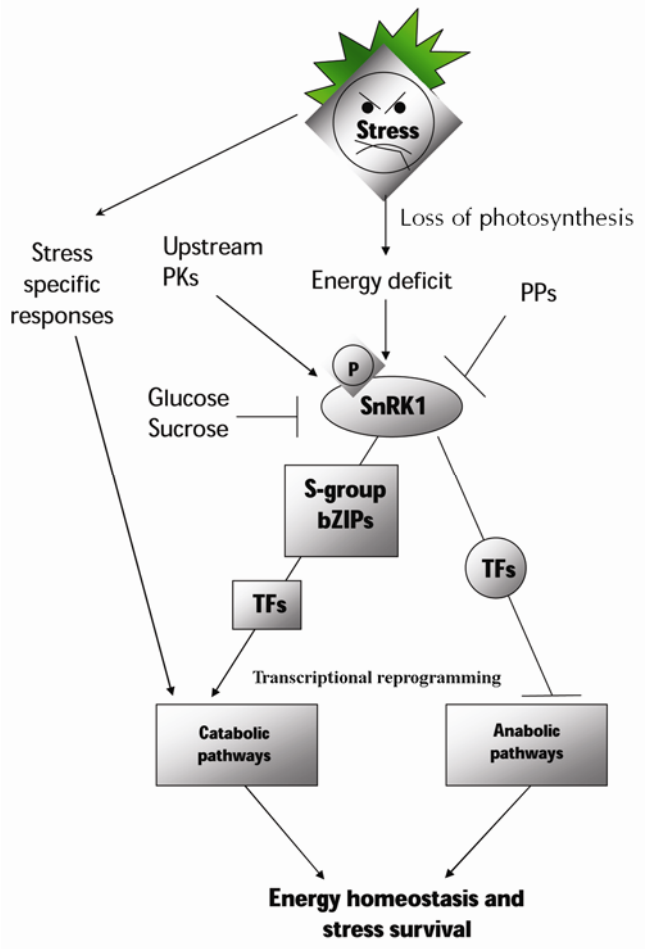


**Figure 5.** A model showing the sugar deficiency sensor SnRK1 as the key sensor of all stresses and its activation is suggested to lead to transcriptional reprogramming culminating in energy homeostasis and stress survival. Almost all stresses result in the loss of photosynthesis that ultimately creates an energy deficiency condition which is sensed by SnRK1. The activation of the sensor is also modulated by upstream protein kinases (PKs) and protein phosphatases (PPs), whereas SnRK1-targeted genes are repressed by glucose and sucrose. Activated SnRK1 initiates many energy-generating catabolic processes, including degradation of amino acids, lipids, cell wall, starch and sucrose through s-group basic leucine zipper (bZIP) transcription factors (TFs) and checks many energy-utilizing processes, including amino acid biosynthesis, cell-wall synthesis, and glycolysis through transcriptional reprogramming by repressing protein synthesis and many TFs. Activation and suppression are indicated by → and –|, respectively (modified after Baena-Gonzalez and Sheen[21]).

(3) Involvement of sugar signalling in stress response of plants is complex and, therefore, a challenging area of research for the future. The diverse stresses ultimately result in loss of photosynthesis, consequently a loss in cellular sugar. We have gained some insight into the mechanism of integration of diverse stresses through the convergent regulator, SnRK1 (ref. 75). Action of SnRK1 ultimately results in transcription reprogramming to meet the sugar-deficit environment induced by stress. But the process of reprogramming, i.e. how SnRK1 coordinately regulates induction of catabolism and repression of anabolic processes to provide energy for stress survival remains a black box. Also unresolved is the precise relationship between the action of SnRK1 and HXK, another important well-known sugar sensor.

(4) Since the genetic basis of many stress-induced photosynthetic responses, signalling and downstream regulation of gene expression, both nuclear and plastidic, is known, the genetic manipulation of stress response is possible. Initially, plant molecular biologists concentrated on specific individual genes associated with stress response. However, microarray transcript analysis now provides opportunities for the simultaneous examination of several genes induced by stress. Most of these genes have now been identified and characterized. In some cases, the expressed genes are metabolic and in others they are regulatory. The modification of the identified and targeted genes for the development of stress tolerance of crop plants constitutes a major area of agriculture biotechnology. Of course, this type of research has already begun since stress-sensitive and tolerant mutants have already been generated by gene manipulation[17,76].

(5) To study plant responses to environmental stress, individual stresses are normally monitored in the laboratories that obviously do not simulate the situation in the field, where plants experience multistress environment. In the field studies, it is, however, difficult to separate and examine individual stress effects without the interference of other stresses. Since plants are autotrophs, their response to either individual stress in the laboratory or multistress environment in the field involves light that makes stress monitoring more complex. We believe that the stress monitoring must be streamlined and this kind of experimental limitation must be addressed.

(6) The mechanism of energy sensing by cellular sensors is complex and has not yet been clearly understood. Further, the stress signal transduction pathways are not

straightforward. The crosstalk between signal transduction pathways associated with changes in energy balance, redox reaction and sugar status in chloroplasts and the stress-induced metabolic pathways of other cellular organelles, including mitochondria makes stress study complex and a challenge to stress biologists.


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