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Forum Review

Glutathione-Associated Regulation of Plant Growth and Stress Responses

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ABSTRACT

A reduced form of glutathione (GSH) is considered to protect the cell from oxidative damage, based on its redox buffering action and abundance in the cell. However, in plants, the high redox potential molecule ascorbate exists at comparable or higher concentrations and is used for scavenging hydrogen peroxide as an electron donor. Recently, examples that cannot be explained simply by the antioxidant activity of GSH have been increasing in number. This article summarizes the recent findings on the glutathione-associated events in plants, in particular, growth and development including cell differentiation, cell death and senescence, pathogen resistance, and enzymatic regulation. *Antioxid. Redox Signal.* 7, 973–981.

INTRODUCTION

A REDUCED FORM OF GLUTATHIONE (GSH) exists in plants at millimolar concentrations (25, 61) although plants have the high redox potential molecule ascorbate (AsA) at high concentrations. The reactive oxygen species (ROS) generated following biotic and abiotic stresses are scavenged by the AsA-GSH cycle in plants, in which AsA is used for scavenging hydrogen peroxide (H_2O_2) as an electron donor (2, 4, 5–7, 15, 25, 61). The univalent oxidation product monodehydroascorbate (MDA) is re-reduced to AsA by the flavin-containing enzyme MDA reductase using NADPH (80, 81) or by reduced ferredoxin (57). Part of MDA is spontaneously disproportionated to dehydroascorbate (DHA) and AsA. DHA is regenerated to AsA by DHA reductase using GSH as an electron donor, and the resulting oxidized glutathione (GSSG) is reduced to GSH by glutathione reductase (GR) using NADPH (25). In the cycle, GSH is used in the backup system for AsA regeneration. Thus, the physiological significance of GSH as an antioxidant in plants becomes obscure and we need to reconsider the role of GSH in cellular regulation other than scavenging ROS.

Recently, GSH has been found to play multiple roles: tracheary element differentiation, a programmed cell death process to transform the cells into a water-conducting pipe (36), the G1-S transition in the cell cycle (88), flowering (69–72, 95), anthocyanin accumulation (93), enzymatic regulation (37), translational and transcriptional regulations (9, 41, 53, 97), and detoxification of xenobiotics (49) and heavy metals (16). Furthermore, examples that cannot be explained simply by the antioxidant activity and are inconsistent with the general idea brought by the antioxidant activity have been increasing in number. For example, the role of GSH is to terminate life and shorten the plant life cycle (64, 95). This is obviously inconsistent with the general belief that oxidative stresses brought by ROS shorten life.

Recent findings on the GSH-associated events in plants and the specificity of GSH and/or GSSG to the events are described, along with possible mechanisms that determine the specificity of glutathione.

GLUTATHIONE BIOSYNTHESIS AND PHOTOSYNTHESIS

GSH, the tripeptide γ-GluCysGly that exists abundantly in plant chloroplasts, is synthesized in two reactions that are catalyzed by γ-glutamylcysteine synthetase (γ-ECS) and glutathione synthetase. The γ-ECS-catalyzed reaction is the rate-limiting step of GSH biosynthesis in plants (61, 62, 72). Photosynthetic production of ATP restricts the rate of GSH biosynthesis (72), consequently regulating such glutathione-

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associated responses as flowering (72), cell death (83), and pathogen resistance responses (83). Although subcellular compartmentation of GSH biosynthesis has been intensively discussed (62), there is a single copy of the gene in the *Arabidopsis* genome regarding γ-ECS and glutathione synthetase, and almost all of the γ-ECS transcripts possess extra sequences for plastid targeting (72). The only known γ-ECS transcript with no targeting signal peptide sequences is in the monocot maize bundle sheath cell with specialized metabolism (30). The key reaction of cysteine biosynthesis that is catalyzed by serine acetyltransferase (SAT) takes place in chloroplasts, cytosol, and mitochondria in *Arabidopsis* (63), among which cytosolic and mitochondrial SATs are under the feedback inhibition by cysteine. Chloroplastic SAT is free from such feedback regulation (63). Cysteine accumulates when GSH biosynthesis is limited by the photosynthetic production of ATP, indicating that the major site of GSH biosynthesis is the chloroplast (70, 72). This idea is not inconsistent with the high levels of ATP and cysteine in the *Arabidopsis cad2-1* mutant defective in γ-ECS.

GLUTATHIONE AS A REGULATOR OF PLANT GROWTH

In our experience, plant growth is strongly dependent on light. Phototropism and photoperiodism of plants are well known examples of control by specific light wavelengths (12, 27, 33, 45), and, in addition, plant growth is strongly dependent on light intensity (72). The first two involve photoreceptors such as phytochromes and cryptochromes, but the third has been explained tentatively by a photosynthetic production of sugars or by photostress-associated response. Recently, the photosynthesis-dependent GSH biosynthesis accounts for the light-intensity-dependent growth (72). Mutant *ch1*, defective in photosynthetic light harvesting, shows not only reduced levels of GSH, but also delayed growth and flowering. This growth and flowering phenotype is restored by the supplementation of GSH or overexpression of the GSH-biosynthesis-limiting γ-ECS gene under the control of the cauliflower mosaic virus 35S promoter (72). Altogether, plant growth is regulated by GSH synthesized during photosynthesis. The finding that GSH promotes the somatic embryogenesis of white spruce (86) may implicate that plant growth is universally regulated by GSH. In this respect, the *fca* mutation that causes late flowering and alters plant GSH levels is a cue to understanding the universal mechanism (69).

TERMINATION OF PLANT LIFE CYCLE BY GLUTATHIONE

In this point, one question may be raised: is the plant's life shortened by GSH? Flowering, or the transition from the vegetative growth phase to the reproductive, is one important step in the plant's life cycle. The involvement of GSH in determining flowering (69–72) implies that, in an unexpected manner, GSH participates in determining when the plant's life will end.

Like flowering, plant senescence of *Eustoma grandiflorum*, a rosette plant, is delayed by buthionine sulfoximine (BSO), a potent inhibitor of GSH biosynthesis, and accelerated by GSH (M. Yanagida and K. Ogawa, unpublished observations). This plant shows bolting (which is an event indicative of the transition from the vegetative leaf rosette stage to the reproductive) and flowering only when a certain period of the oxidative stress chilling is given (95), which is consistent with the general idea that an oxidative stress shortens the plant's life. However, GSH can make the plant bolt without chilling, and the bolting-inducing effect of chilling stress is completely blocked in the presence of BSO (95). Generally, plants given oxidative stresses show increased GSH levels (61, 56, 85), which is consistently observed in chilled *Eustoma* plants (95). Taken together, GSH acts not to prolong, but to terminate the plant's life cycle. Such an insight into glutathione is not inconsistent with the fact that transgenic tobacco plants with elevated glutathione levels are paradoxically susceptible to the oxidative damage under photooxidative conditions (18). In this regard, it may be noted that adenosine 5′-phosphosulfate reductase activity, involved in cysteine biosynthesis, is feedback regulated by GSH at the protein and mRNA levels (34). Such a feedback regulation may function as a limiter of GSH biosynthesis to keep cellular GSH at appropriate levels, consequently regulating life span.

GLUTATHIONE IN PATHOGEN-RELATED RESPONSE

ROS are generated in the plant–pathogen interaction (21), following which plant GSH and GSSG change (22, 51–53, 87). Early studies revealed that glutathione causes massive and selective induction of plant defense genes (91). Recently, it has been reported that a key activator of pathogen-related responses, NPR1 [nonexpressor of pathogenesis-related protein-1 (PR-1)], is regulated by the redox status of its cysteine residues (59). As a reduced form of NPR1 is active, it was postulated that the redox change in glutathione might contribute to the activation of NPR1. However, an NPR1-regulated response, the accumulation of PR-1 protein, is not suppressed by the inhibition of such a redox change in glutathione, but is strongly associated with its absolute amount in *Arabidopsis* (83). The resistance signal salicylic acid also induces the accumulation of PR-1 protein (39), which occurs according to the plant glutathione level (83). Thus, changes in the amount of total glutathione following the plant–pathogen interaction may contribute to the regulation of pathogen resistance in plants. This insight is supported by the evidence shown by Mullineaux's group that stress defense gene expression is closely linked to plant glutathione biosynthesis (10).

GLUTATHIONE IN PROGRAMMED CELL DEATH

Cell death may occur in response to pathogen invasion, in which pathogen-related responses as described above are observed. This type of cell death is programmed by resistance

signal(s) such as salicylic acid and ROS. Many mutants exhibiting spontaneous cell death similar to pathogen-related cell death have been isolated (19), among which the *lsd1* mutation mediates cell death (20, 38) that is shown to require salicylic acid accumulation and NPR1 function (8). The cell death that is mediated by the *lsd1* mutation can be suppressed in the presence of BSO, and this suppression is abolished by the application of GSH (83). In this case, cell death is not inhibited by the suppression of the redox change in glutathione, but of the absolute level of glutathione (83), although the NPR1 function to induce cell death is regulated by the reductant dithiothreitol (DTT) or GSH (59).

Unlike the cell death occurring following the plant–pathogen interaction, tracheary element differentiation in the cultured cells of *Zinnia elegans*, another type of programmed cell death in plant development, is associated with the transient accumulation of GSSG (36). The effect of GSSG application on tracheary element differentiation is dependent on culture time: before cellular GSSG achieves a peak level, the application of GSSG has a promotional effect on the differentiation; after the peak, the effect is reversed. On the contrary, the application of GSH has no effect on the differentiation before the peak, but it has a suppressive effect after the peak. These facts suggest that the redox change in glutathione is probably involved in the regulation of cell differentiation.

REDOX REGULATION OF PROTEIN FUNCTION

The redox status of the cysteine residues in proteins modulates the protein function (40, 82). Thioredoxin (Trx) and glutaredoxin (Grx) reduce the specific cysteine residues in numerous proteins, participating in the redox control of protein functions (54). Such a redox regulation of the specific protein cysteine residues by Trx and Grx has recently been focused on (11, 44, 46, 58, 94), but *in vitro* DTT also reduces the protein cysteine residues to modulate the protein function. DTT has a much lower redox potential (high reducing power) than GSH. However, DTT has little or no activity to regulate the above events that are regulated by GSH (Table 1), suggesting that the above events are regulated not by a simple thiol exchange reaction. One may suppose that the specificity of Grx determines that of glutathione, but, if so, DTT should be replaceable with GSH, partially or completely. Thus, there seems to be another factor determining the specificity of glutathione in plant cells.

Table 1. Specificity of Glutathione in Glutathione-Associated Physiological Events in Plants

Glutathione-associated physiological events	*Specificity*						*References*
	GSH	*GSSG*	*GSH/GSSG*	*BSO*	*DTT or MetOH*	*Trx*	
Growth and development							
Bolting/flowering	Time, genotype, and concentration			Time and genotype			69
	+			−			72
	+	Stress	No	−	No		95
Somatic embryogenesis	+						86
Cell division	+			−			79
	+	No	No	−	No		88
Root hair length	+			−	+		76
Tracheary element differentiation	Time	Time	Probably yes	Time			36 See text
Stress-related							
Hypersensitive response cell death	+	+	No	−			83
PR protein expression	+	+	No	−			83
NPR1 function	+		Yes		+		59
Cold stress tolerance	+						43
Photostress tolerance	−						18
Salt stress tolerance	+			−			56
Heavy metal tolerance	+						16, 76
Glutathionylation enzymes							
Cytosolic triose phosphate isomerase	BSA	BSA	BSA		No	No	37
Plastidic FBA	pH	pH	pH		−	−	73

The symbols "+" and "−" indicate promotional and suppressive roles, respectively. Terms such as Time, BSA (bovine serum albumin), pH, and Stress indicate that the effect of the indicated redox reagents is dependent on the indicated conditions. Yes or No means whether it has any effect or not. MetOH, mercaptoethanol; DTT, dithiothreitol.

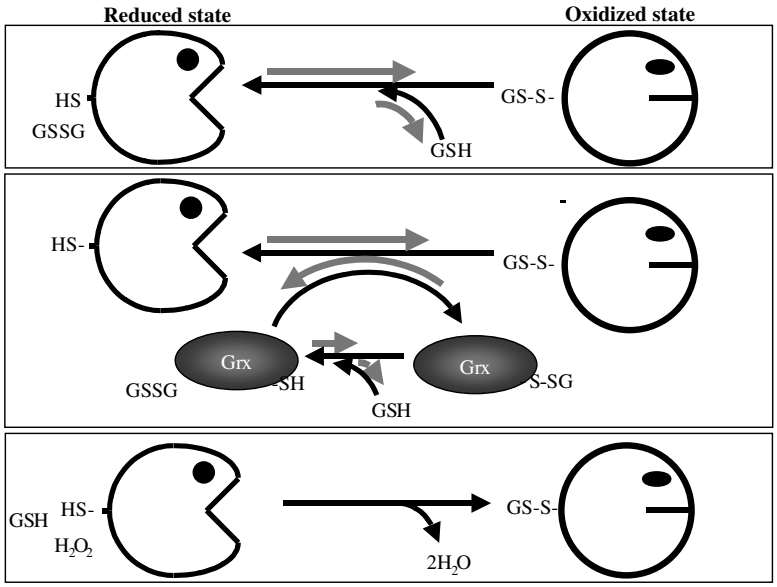

FIG. 1. Protein glutathionylation. The glutathionylated/deglutathionylated protein ratio is equilibrated accordingly to the [GSH]/[GSSG] ratio (**upper**), and this equilibration reaction can be mediated via the monothiol mechanism of Grx (**middle**). Protein glutathionylation is promoted by such an oxidant as H_2O_2 (**lower**), and this reaction is further promoted by peroxidase activity. Arrows with the same tone in a panel show one series of reaction flows (a glutathionylation or deglutathionylation process). In addition to the above three, *S*-nitrosoglutathione (GSNO) has been shown to lead to protein glutathionylation (14).

PROTEIN GLUTATHIONYLATION/ DEGLUTATHIONYLATION

Protein glutathionylation (Fig. 1), which is the covalent binding of the GS-moiety to the protein through the intermolecular disulfide bridge, modulates protein function (17), which may explain the glutathione-specific events. As protein glutathionylation is promoted by such an oxidant as H_2O_2 (Fig. 1, upper and lower panels), it may be a switch turning the function of some proteins from a normal mode to an oxidative-stress mode. Considering these together with the facts that ROS is generated for a developmental regulator (65) and metabolic substrate (68) and that ROS-scavenging enzymes are microcompartmentalized at the site of ROS generation (5, 6, 66, 67), even in sound plants, such a glutathionylation-dependent switch may be regulated by oxidative stimuli at a microlocal site of the cell.

Plastidic fructose 1,6-bisphosphate aldolase (FBA), a glutathionylated protein from the suspension-cultured cells of *Arabidopsis* (37), may be a cue to understanding what makes the events specific to glutathione. At pH 8, this enzyme is activated by both GSH and GSSG with conformational changes, but inhibited by DTT and Trx (50, 73). This indicates that the regulation of protein function by glutathione is substantially different from a simple thiol/disulfide exchange reaction. Considering that a number of physiological events are specific to glutathione, such a glutathione-specific redox regulation of protein functions must be common in plant physiology, and the clarification of proteins to be regulated specifically by glutathione should be essential for further comprehension.

Grx (but not Trx) has been shown to mediate the glutathionylation/deglutathionylation of proteins (Fig. 1, middle panel) (24), and the redox potential of Grx is regulated according to the redox potential ($[GSH]^2/[GSSG]$) (92), which may explain why the total glutathione level increases with little change in the GSH/GSSG ratio in such an event as PR-1 protein accumulation in resistance response to pathogens (83).

GLUTATHIONE AS AN ELECTRON DONOR FOR STRESS-ASSOCIATED ENZYMES

Glutathione is used as an electron donor for many enzymes, such as glutathione-dependent formaldehyde dehydrogenase (FDH), glutathione peroxidase (GPX), glutathione *S*-transferase (GST), and Grx.

Plant GPX and GST family genes always respond to various oxidative stress, such as chilling, excess photon, and drought (3, 89). Overexpression of GPX or GST results in elevated tolerance against oxidative stress (77, 89, 98), indicating that GPX and GST play a protective role in oxidative stress. GST also mediates the conjugation of GSH to a metabolite such as flavonoids, and the resulting molecule may serves as a signaling molecule. Such a speculation may be the case for phytohormone signaling, because the phytohormone auxin has been shown to bind GST (13) and GST genes are under the control of phytohormones (84). It is also possible that signal molecules may be converted from metabolites via the GSH-dependent peroxidase reaction of GPX. The peroxidase activity of GPX and GST may promote protein glutathionylation as shown in Fig. 1 (lower panel) to modulate the protein function. In fact, GST-dependent glutathionylation of 1-Cys peroxiredoxin has also been reported to activate the protein (48). These observations may explain why plant GST family genes always respond to oxidative stress. Thus, the diversity of plant GST may be a cue to understanding a mechanism that determines which protein(s) should be glutathionylated. Furthermore, as FDH has *S*-nitrosoglutathione reductase activity (78), we may have to take into account nitric oxide signaling.

RELATIONSHIP BETWEEN GLUTATHIONE BIOSYNTHESIS AND GLUTATHIONE-SPECIFIC REGULATION IN PLANT CHLOROPLASTS

The glutathione-dependent regulation of the plastidic FBA is considered to be closely linked to glutathione biosynthesis in chloroplasts. FBA is activated by pH transition from 7 to 8 (73). In chloroplasts, following illumination, the Calvin cycle fixing the atmospheric CO_2 to sugars is activated and the stromal pH condition changes from 7 to 8 (1, 35, 90). The photosynthetic production of ATP relies on the illumination-dependent formation of proton gradient across the thylakoid membranes (42), restricting glutathione biosynthesis (72). The ATP production at the beginning of photosynthesis is accelerated by cyclic electron flows (47), which are essential for photosynthesis (60). These facts with the above suggest that, following illumination, the FBA in the Calvin cycle is activated by the photosynthesis-dependently synthesized GSH and stromal alkalization.

Under photooxidative conditions, an electron acceptor in photosystem I, $NADP^+$, is apt to run short due to the restricted consumption of NADPH in the Calvin cycle (5, 6, 26, 74, 75). In this respect, the activation of the FBA by GSSG is physiologically important to maintain the Calvin cycle to produce $NADP^+$. Thereby, the photooxidative stress may be reduced in chloroplasts.

NUMEROUS PROTEINS ARE UNDER THE CONTROL OF GLUTATHIONE, FORMING METABOLIC AND SIGNALING NETWORKS

Comprehensive proteomic analysis on glutathionylated proteins has been performed in animal cells (28), revealing that numerous proteins are to be glutathionylated in cells, although such an attempt has not been done intensively in plants. The target proteins of Trx and Trx itself are potential targets of glutathione. As it has been shown that glutathionylation of human Trx inhibits the Trx-dependent activation of the insulin reductase (14) and some of the plant Trx targets overlap glutathionylated proteins in human T lymphocytes, plant Trx and its targets are regarded as the target of glutathione. In addition to the above, the reductant DTT modulates the functions of many proteins, although it is not specific to glutathione. Thus, cellular redox changes in glutathione following oxidative stresses and/or changes in light conditions may regulate a broad range of metabolic and signaling pathways, forming the glutathione-dependent metabolic and signaling networks through the redox status of the cysteine residue(s) in proteins (Fig. 2). Besides, in unstressed plants, microlocal oxidative stimuli may be used for the glutathione-dependent regulation of protein functions.

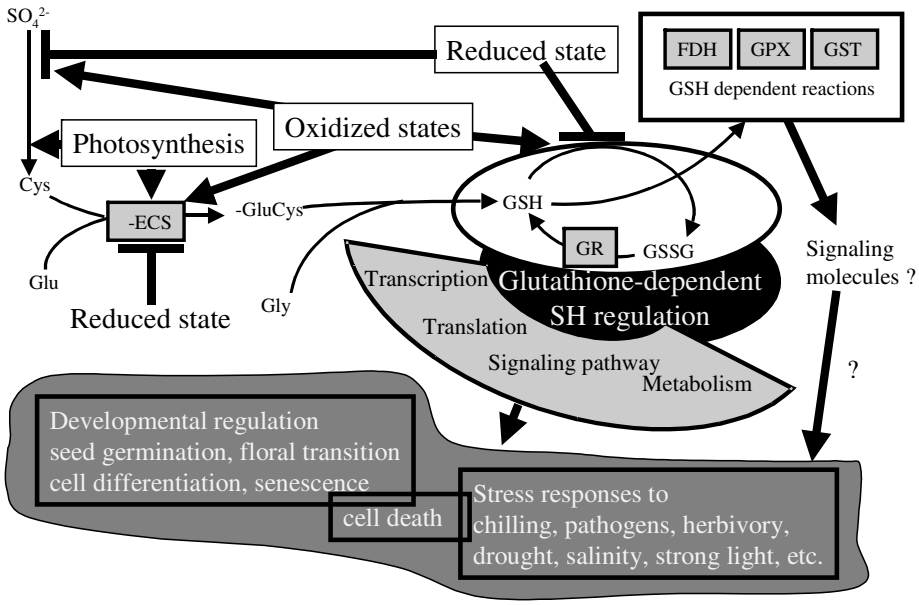

FIG. 2. Schematic metabolic and signaling networks mediated by glutathione in plants. The rate of glutathione biosynthesis is regulated via the γ-ECS reaction through oxidative stimuli or photosynthesis, and the synthesized glutathione participates in many physiological events, such as flowering, cell differentiation, and stress responses. There are numerous key factor proteins with redox-sensitive cysteine residues in each event, and their functions can be controlled simultaneously by glutathione, forming complicated metabolic and signaling networks. GSH-dependent reactions mediated by such enzymes as FDH, GPX, and GST may produce signaling molecules. Phytochelatin [$(\gamma\text{-GluCys})_n$Gly] is synthesized from GSH, chelating heavy metals.

CONCLUDING REMARKS

Glutathionylated proteins in the suspension-cultured cells of *Arabidopsis* have been studied using a biotinylated GSH ester (37). However, their functions in plants have not been fully elucidated. Continuous attempts to identify glutathionylated proteins in plants and the functional analysis of the proteins identified will lead to advances in plant physiology. Considering that plants are strongly dependent on photosynthesis that consists of chain redox reactions and that they deal with a large amount of redox molecules, the regulation of protein functions by the most major thiol, glutathione, should be intensively explored. As there are many reactions that require glutathione as an electron donor, it may also be important to consider the product of the reactions as a signaling molecule.

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ABBREVIATIONS

AsA, ascorbic acid; BSO, buthionine sulfoximine; DHA, dehydroascorbate; DTT, dithiothreitol; γ-ECS, γ-glutamylcysteine synthetase; FBA, fructose 1,6-bisphosphate aldolase; FDH, formaldehyde dehydrogenase; GPX, glutathione peroxidase; GR, glutathione reductase; Grx, glutaredoxin; GSH, reduced glutathione; GSSG, oxidized glutathione; GST, glutathione *S*-transferase; H_2O_2, hydrogen peroxide; MDA, monodehydroascorbate; NPR1, nonexpressor of pathogenesis-related protein-1; PR-1, pathogenesis-related protein-1; ROS, reactive oxygen species; SAT, serine acetyltransferase; Trx, thioredoxin.

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