Free Radical Biology and Medicine 65 (2013) 724–730

Contents lists available at ScienceDirect

Free Radical Biology and Medicine

journal homepage: www.elsevier.com/locate/freeradbiomed

Review Article

Glutathione and plant response to the biotic environment

Pierre Frendo [a,b,c,*], Fabien Baldacci-Cresp [a,b,c], Sofiane M. Benyamina [a,b,c], Alain Puppo [a,b,c]

[a] Université de Nice–Sophia Antipolis, Institut Sophia Agrobiotech, F-06903 Sophia Antipolis Cedex, France
[b] INRA UMR 1355, Institut Sophia Agrobiotech, F-06903 Sophia Antipolis Cedex, France
[c] CNRS UMR 7254, Institut Sophia Agrobiotech, F-06903 Sophia Antipolis Cedex, France

ARTICLE INFO

Article history:
Received 31 January 2013
Received in revised form
22 July 2013
Accepted 23 July 2013
Available online 1 August 2013

Keywords:
Plant
Microbe
Pathogenesis
Symbiosis
Glutathione
Redox

ABSTRACT

Glutathione (GSH) is a major antioxidant molecule in plants. It is involved in regulating plant development and responses to the abiotic and biotic environment. In recent years, numerous reports have clarified the molecular processes involving GSH in plant–microbe interactions. In this review, we summarize recent studies, highlighting the roles of GSH in interactions between plants and microbes, whether pathogenic or beneficial to plants.

© 2013 Elsevier Inc. All rights reserved.

Contents

Introduction

Glutathione (GSH) is a tripeptide (γ-glutamylcysteinylglycine) present in a broad range of organisms, from bacteria to humans. It is synthesized in a two-step process. In the first step, γ-glutamylcysteine synthetase or γ-glutamylcysteine ligase (γ-GCL, GSH1) catalyzes the formation of γ-glutamylcysteine (γGC) from glutamate and cysteine, in an ATP-dependent reaction. Surprisingly, although GSH is present in many organisms, including bacteria, plants, and animals, the primary sequence of GSH1 is not conserved in these different groups of organisms [1]. In the second step, glutathione synthetase (GS or GSH2) catalyzes the formation of GSH from γGC and glycine, in another ATP-dependent reaction. The primary sequence of GS differs between eukaryotes and prokaryotes [1]. In plants, the GSH synthesis pathway takes place in the plastid and cytosol (Fig. 1). γ-GCL is encoded by a nuclear gene (*GSH1*) and is targeted to plastids [2,3]. GS is also encoded by a nuclear gene (*GSH2*) and is found in both the plastids and the cytosol [3,4]. In plants, GSH accumulates to millimolar concentrations within cells. Multiple GSH homologs have been detected in plants. One of the most frequently observed is homoglutathione (hGSH), which replaces or is present in addition to GSH in the large and diverse plant family *Leguminosae* [5,6]. Its synthesis requires a specific homoglutathione synthetase, encoded by a gene derived from the *GS* gene by gene duplication [7].

The biological functions of GSH relate principally to reversible redox reactions of the cysteine sulfur group, resulting in the coexistence of a reduced state (GSH) and an oxidized state (GSSG), in which two GSH molecules are linked via a disulfide bound. The cellular GSH pool is mostly reduced under optimal conditions. The redox status of GSH is kept high by glutathione reductase (GR), which uses NADPH to supply reducing power. GSH can also react

* Corresponding author at: Institut Sophia Agrobiotech, 400 Route des Chappes, F-06903 Sophia Antipolis Cedex, France.
E-mail address: frendo@unice.fr (P. Frendo).

0891-5849/$ - see front matter © 2013 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.freeradbiomed.2013.07.035

with protein cysteine residues to form mixed disulfides via a glutathionylation process. Protein glutathionylation has been extensively investigated in animals [8–11], but much less is known about this process in plants [12,13]. Glutaredoxins (GRXs), which couple GSH redox potential to changes in protein thiol–disulfide status, are involved in the deglutathionylation process and in the regeneration of multiple enzymes, such as peroxiredoxins and methionine sulfoxide reductases [14,15]. GSH may also react with numerous endogenous and xenobiotic electrophilic compounds, via glutathione *S*-transferases [16,17]. Finally, GSH also protects plants against heavy metals, through the formation of phytochelatins (PCs), which are GSH polymers. PCs are synthesized by phytochelatin synthase, which uses GSH as a substrate [18].

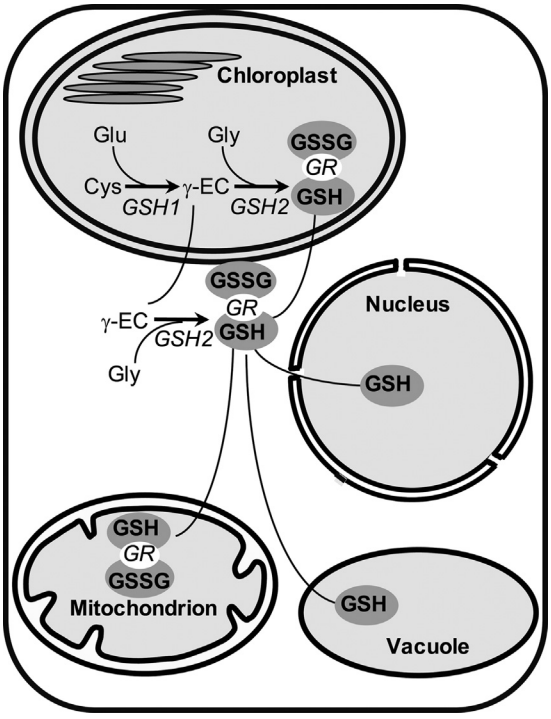

Fig. 1. Synthesis and transport of glutathione in a plant cell. Glutathione (GSH) is synthesized in a two-step biosynthetic pathway involving γ-glutamylcysteine ligase (GSH1) and glutathione synthetase (GSH2). The redox state of GSH is regulated by glutathione reductase (GR).

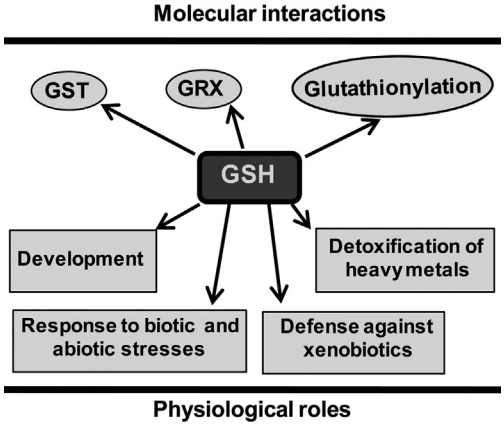

Fig. 2. General roles of glutathione in plants. Glutathione (GSH) is used as a substrate by glutaredoxins (GRX) and glutathione *S*-transferases (GST); it is also involved in protein glutathionylation. In plants GSH is involved in development, abiotic and abiotic stress responses, and protection against heavy metals and xenobiotics.

GSH plays a crucial role in plant development (Fig. 2). Analyses of the phenotypes of *Arabidopsis thaliana* GSH-deficient mutants have shown that GSH is involved in embryo and meristem development [19,20]. Abiotic and biotic stresses play a crucial role in the regulation of development and the adaptation of plants to their environment [21,22]. In this context, GSH has been shown to be involved in light signaling, in studies of the *Arabidopsis rax1* mutant, which has only half the normal level of GSH in its leaves and displays constitutive expression of the photo-oxidative stress-inducible ascorbate peroxidase 2 [23]. However, the role of GSH is not restricted to the regulation of the plant growth and adaptation to the abiotic environment. This molecule is also involved in the response of the plant to its biotic environment. In this review, we analyze the links between glutathione metabolism and the adaptation of the plant to its biotic environment.

Glutathione and plant–pathogen interactions

Studies in the late 1980s showed that the treatment of cultured plant cells with exogenous GSH induced the accumulation of plant defense-related transcripts for proteins such as phenylpropanoid biosynthetic enzymes, phenylalanine ammonia-lyase, and chalcone synthase, which is involved in lignin and phytoalexin production [24,25]. Treatment with pathogen-derived elicitors was then shown to induce GSH accumulation in cell cultures [26] or in plants, during defense induction [27,28]. The plant defense response to pathogens also modifies the redox state of GSH [29]. Moreover, GSH levels increase after treatment with the defense-related plant hormone salicylic acid (SA), and the redox state of this molecule shifts toward a more reduced state [30–32].

The first genetic evidence of a role for GSH in defense reactions was provided by the isolation of *Arabidopsis* phytoalexin-deficient (*pad*) mutants [33]. The *pad2* mutant line displays impaired production of the phytoalexin camalexin and enhanced susceptibility to the pathogenic bacterium *Pseudomonas syringae*. This mutant line has also been shown to be susceptible to the pathogenic oomycetes *Phytophthora porri* and *Botrytis cinerea* [34,35]. Levels of pathogenesis-related protein 1 and of SA are very low in the *pad2* mutant line [34]. The identification of *PAD2* as *GSH1* demonstrated the existence of a clear link between GSH metabolism and plant defense mechanisms [28]. This association between GSH content and plant defense has also been demonstrated with other GSH1-deficient mutants, *cad2-1* and *rax1-1*, which are less resistant than the wild type to avirulent strains of *P. syringae* [23].

Thiol–disulfide redox status is clearly involved in the regulation of a major regulatory protein, NPR1 (nonexpressor of PR gene 1) [36]. NPR1 must be converted from its oligomeric form to a monomer for translocation from the cytosol to the nucleus, and this requires the reduction of the disulfide bonds of the oligomeric form [31]. It has also been shown that the disulfide bonds can be reduced in vitro in vitro with a GSH:GSSG buffer at physiological concentration [31]. Furthermore, NPR1 can also be reduced by thioredoxins (Trxs) [37]. However, NPR1 oligomerization may also be in vitro regulated in vitro by nitrosylation, with *S*-nitrosoglutathione (GSNO) [37]. Moreover, the SA-induced monomerization of NPR1 and its nuclear translocation are inhibited in the *atgsnor1-3* mutant, which lacks the GSNO reductase and has high levels of S-nitrosylation activity [37,38]. The resistance of *atgsnor1-3* mutants to pathogens is severely compromised [38], and *atgsnor1-3* mutants also display alterations to SA metabolism, with lower levels of SA

Download English Version:

https://daneshyari.com/en/article/8270827

Download Persian Version:

https://daneshyari.com/article/8270827

Daneshyari.com