Cyclophilin 20–3 is positioned as a regulatory hub between light-dependent redox and 12-oxo-phytodienoic acid signaling

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ABSTRACT
The jasmonate family of phytohormones plays central roles in plant development and stress acclimation. However, the regulatory modes of their signaling circuitry remain largely unknown. Here we describe that cyclophilin 20–3 (CYP20–3), a binding protein of (+)-12-oxo-phytodienoic acid (OPDA), crisscrosses stress responses with light-dependent redox reactions, which fine-tunes the activity of key enzymes in the plastid photosynthetic carbon assimilation and sulfur assimilation pathways. Under stressed states, OPDA – accumulated in the chloroplasts – binds and promotes CYP20–3 to transfer electron (e−) from thioredoxins (i.e., type-f2 and -x) to 2-Cys peroxiredoxin B (2-CysPrxB) or serine acetyltransferase 1 (SAT1). Reduction (activation) of 2-CysPrxB then optimizes peroxide detoxification and carbon metabolisms in the photosynthesis, whereas the activation of SAT1 stimulates sulfur assimilation which in turn coordinates redox-resolved nucleus gene expressions in defense responses against biotic and abiotic stresses. Thus, we conclude that CYP20–3 is positioned as a unique metabolic hub in the interface between photosynthesis (light) and OPDA signaling, where controls resource (e−) allocations between plant growth and defense responses.

INTRODUCTION
The phytohormones, jasmonic acid (JA) and its precursors and derivatives, collectively known as jasmonates are pivotal signal molecules widely distributed throughout the plant kingdom, and convey defense (adaptive) responses toward various forms of abiotic and biotic stresses, including wounding, UV damage, insect attack and microbial pathogen infection. They also play critical roles in plant reproduction, and developmental processes such as fruit ripening, root growth, tuberization, senescence, and tendril coiling. These important but diverse activities of jasmonates must be reflected by their versatility as molecular modulators. However our current knowledge regarding their modes of action is incomplete.1,2,17

Lately, molecular underpinnings have been revealed for (+)-12-oxo-phytodienoic acid (OPDA, a primary precursor of jasmonate family) signaling in the regulation of stress-responsive cellular redox homeostasis.3 Once it is produced, OPDA binds cyclophilin 20–3 (CYP20–3), a bifunctional enzyme in the chloroplasts that remodels target proteins (peptidylprolyl isomerase activity) and/or transfers electrons to peroxide substrates (reductase activity).3–8 The binding stimulates CYP20–3 to form a complex with serine acetyltransferase 1 (SAT1), which triggers the formation of a hetero-oligomeric Cysteine (Cys) synthase complex (CSC) with O-acetylserine(thiol)lyase B.3,5,10 CSC formation then leads to the production of Cys (sulfur assimilation) and subsequently thiol metabolites, which increases cellular reduction potentials. The enhanced reduction capacity in turn coordinates the expression of a subset of OPDA-responsive genes that actuate and calibrate pathogen defense and stress adaptation processes.1 The results substantiated a role of OPDA as an autonomous metabolic messenger, connecting stress cues to the production of additional small molecules and the regulation of cellular redox homeostasis in transmitting signal from the chloroplasts to nucleus gene expressions (referred to as ‘retrograde signaling’).3,11,12

Thioredoxins (Trxs) are small (~12 to 14 kDa) disulfide oxidoreductases to which deliver electron, and restructure target proteins. In Arabidopsis, the chloroplasts contain ~10 Trx isoforms, categorized into five types (f, m, x, y and z), that relay the light-dependent activation of enzymes in the Calvin cycle (carbon fixation) such as fructose 1,6-bisphosphatase (FBPase), glyceraldehyde-3-phosphate dehydrogenase, sedoheptulose 1,7-bisphosphatase and phosphoribulokinase, which balance energy conversion and consumption in photosynthesis.19–21

Recent studies, however, have started to unveil that the plastid Trxs can also target other, Calvin cycle-unrelated, proteins and corroborate their activities in broad cellular processes during plant growth and survival such as photorespiration and stress acclimation.20,22 For instance, affinity survey of chloroplast lysates via Trx-immobilized resins revealed the several molecular modulators in plastid photosynthesis and stress responses (reduction of 2-CysPrxB).4,5,11,12

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binding candidates of Trxs including CYP20–323, a key regulator of i) stress-responsive OPDA signaling and ii) photosynthetic pathway as an electron donor of 2-Cys peroxiredoxins (2-CysPrxs). Arabidopsis genome encodes two plastid 2-CysPrx (denoted A and B) isoforms, both are highly abundant thiol-based peroxidases that function in protecting and modulating photosynthesis. Once reduced (activated) via NADPH-dependent thioredoxin reductase C (NTRC) and other electron donors such as Trxs and CYP20–3, they metabolize the detoxification of a toxic byproduct in photosynthesis (i.e., H₂O₂) and the activation of Calvin cycle enzymes such as FBPase. The interaction of Trxs with CYP20–3, thereby, positioned CYP20–3 as a redox sensor of the light-dependent redox reaction chain (also called electron transport chain, ETC; Fig. 1A), transferring electron (e⁻) from Trxs toward 2-CysPrxs and/or SAT1. Reduction (activation) of 2-CysPrxs then continues peroxide detoxification and activates photosynthetic carbon metabolisms, whereas the activation of SAT1 stimulates sulfur assimilation which coordinates redox-resolved nucleus gene expressions in defense responses against biotic and abiotic stresses. In line with this scenario, the stress-induced OPDA binds and, perhaps, modulates the functional and conformational states of CYP20–33 to which adjusts its subsequent binding and electron transfer between 2-CysPrxs and/or SAT1.

To further understand the regulatory modes of jasmonate family signaling circuitry, this study used in vitro protein-protein interaction assays, and delineated the functional dynamics of Trx isoforms in conjunction with CYP20–3-dependent OPDA signaling. A series of pull-down assays demonstrated that OPDA binds CYP20–3, and stimulates its interactions with and electron transfers from Trxs (i.e., type-x and -f2), as well as toward 2-CysPrxB and SAT1 (see Fig. 1), highlighting the unique roles of CYP20–3 in controlling the interplay between plant light and hormone signaling to fine-tune energy inputs into outputs that shape plant growth and defense responses (‘fitness’) toward various environmental stress cues.

Results and discussion

**OPDA stimulates Trx-mediated reduction (activation) of CYP20–3**

To delineate the roles and modus operandi of CYP20–3 in the interface between the light-dependent ETC and OPDA signaling, we profiled the assembly dynamics of CYP20–3 with its binding ligand (OPDA) and reductant proteins, Trxs (i.e., Trx-x6) and SAT13,4 (Fig. 1B). In this pull-down assay, CYP20–3 was capable of co-purifying SAT1 and concurrently Trx-x in the presence of OPDA, and these trimetric interactions were enhanced in an OPDA concentration-dependent manner. These observations indicated that OPDA signaling fosters the sequential interactions between (1) Trx-x and CYP20–3, and (2) CYP20–3 and SAT1, especially since little – if any – direct interaction was shown between Trx-x and SAT1 (Fig. S1A). Indeed, a following pull-down assay showed that the increased concentrations of Trx-x elevate the interaction of CYP20–3 with SAT1 (Fig. 1C), further corroborating that Trxs are electron donors that relay OPDA signaling to potentiate the activity of CYP20–3, which subsequently 1) recruits SAT1, 2) facilitates CSC formation, 3) produces thiol metabolites, and 4) ultimately affects cellular redox potentials resulting in differential nucleus gene expressions during plant stress defense responses.3

**OPDA signaling shepherds the light-dependent ETC to strengthen the activity of CYP20–3**

Plastid Trxs share little sequence homology (Fig. S2), but often demonstrate functional and biochemical redundancy to each other.19,20 In agreement, all plastid Trxs displayed binding affinity to CYP20–3 (Figs. 2A and S3A). Moreover, a subset of those interactions such as CYP20–3 with Trx-f2, -m1 or -m4 was promoted by OPDA (Fig. 2A). In fact, type-f2, -m1 and -m4 showed stronger binding capacity to CYP20–3 than Trx-x (Fig. 2B, 4 left lanes). However, Trx-m1 and -m4 were, with or without OPDA, not co-purified with CYP20–3 when SAT1 was supplemented (Figs. 2B, 5 right lanes, and S3B), indicating that CYP20–3 binds preferentially to SAT1 over Trxs, except Trx-f2 (Fig. 2B, red arrow). Trx-f2 was concurrently pull-downed along with SAT1 by CYP20–3, and the Trx-f2-CYP20–3 and CYP20–3-SAT1 interactions were markedly enhanced in parallel with the increased concentrations of OPDA (Fig. 2C). Note that control experiments again showed that Trx-f2 has little if any binding affinity toward SAT1 (Fig. S1B), further substantiating that OPDA signaling regulates a flow route of the light-dependent ETC, stimulating the Trx-mediated (i.e., type-f2 and -x) reduction (activation) of CYP20–3, which in turn promotes the formation of CYP20–3-SAT1 complex and subsequent metabolic cascades such as sulfur assimilation and cellular redox regulations in the coordination of plant defense, OPDA-responsive, gene expressions.3

As alluded, 5 types of plastid Trxs (f, m, y, x and z) were reported for Arabidopsis, of which type-f and -m consisting of 2 and 4 isoforms are the most abundant (Fig. 3A).19,21 and their expressions, besides type-f1 and -m4, were rapidly (< 60 min) upregulated to 2-fold upon perceiving wounding (Fig. 3B and Table S1). Since wounding induces OPDA
accumulations with a peak at ~60 to 180 min-post-wounding (mpw), the stress-responsive rearrangement of Trx transcripts occurs in prior or parallel to the OPDA biosynthesis, supporting i) the intrinsic roles of Trxs in mediating ETC during stress resolution processes, and ii) the presence of two different modes of Trx-mediated CYP20–3 activations, at the transcriptional and posttranslational (i.e., OPDA signaling) levels. Together, these results propose that CYP20–3 controls an allocation of light-derived energy (e\(^{-}\)) between the photosynthetic carbon fixation and OPDA signaling, which perhaps shapes plant growth/development and defense tradeoffs (fitness).

Figure 2. OPDA promotes the sequential interactions between Trx-f2 and CYP20–3, and CYP20–3 and SAT1. In vitro pull-down assays for (A) Trxs (type-f2, -m1 and -m4) and CYP20–3 in the presence of various concentrations of OPDA, (B) comparing the binding affinities of Trxs (type-f2, -m1 and -m4) to CYP20–3 without (4 left lanes) or with (5 right lanes) SAT1, and (C) determining the physical association of Trx-f2, CYP20–3 and SAT1 at various OPDA concentrations. The lower panels (Input) show Coomassie blue-stained gels, indicating the amount of bait protein used in each pull-down assay. Parallel immunoblots for proteins that co-purified with GST:SAT1 were probed with monoclonal anti-His antibody (upper panel, IB HIS). For (B), the same concentration (2.5 nM) of Trx was used, and red arrow indicates Trx-f2.

Figure 3. Wounding triggers the rapid rearrangement of Trx transcript levels. (A) Basal levels of plastid Trx mRNAs in WT Arabidopsis (Col-0) plants, measured by the quantitative (q)RT-PCR. (B) Time-resolved qRT-PCR analyses of plastid Trx genes (type-x and -z (i), -f (ii), -m (iii), and -y (iv) isoforms) in wounded WT Col-0 plants. Total RNAs were prepared from leaves at 0, 30, 60, 90, 120, 150 and 180 min-post-wounding (mpw) as described in Materials and Methods. Values were normalized to the expression of UBC gene (means ± SD; n = 3), and different letters indicate significant differences (P < 0.05, the Student’s t-test). Note that the results of statistical analysis on the wound responsive expressions of Trx-m isoforms [3B(iii)] are summarized in Table S1.
OPDA promotes CYP20–3 and 2-CysPrxB interaction

Our data propose that OPDA signaling stimulates the buildup of reduction potential of CYP20–3, which activates SAT1-dependent sulfur assimilation and regulates defense (OPDA-responsive) gene expressions. On the other hand, recent studies reported that CYP20–3 is also able to target 2-CysPrxs (Fig. 4A, B), ubiquitous peroxidase that function in peroxide detoxification and controls carbon metabolisms in photosynthesis.23 Therefore, we investigated the potential effects of OPDA on the interactions of CYP20–3 with 2-CysPrxA or -B.5,6,24 Unexpectedly, OPDA promoted only CYP20–3–2-CysPrxB, but not CYP20–3–2-CysPrxA, interaction (Fig. 4A, B, upper panels). In line with this scenario, OPDA enhanced the CYP20–3–2-CysPrxB, as well as CYP20–3–SAT1 interactions simultaneously, but promoted only the CYP20–3–SAT1 interaction when they were co-incubated with 2-CysPrxA (Fig. 4A, B, lower panels). Unlike Trxs, the expression of both 2-CysPrxA and -B (Fig. 4C), as well as SAT13, was constitutive regardless of wounding, suggesting that OPDA signals the post-translational modification of, perhaps structural and/or functional states of, CYP20–3, which subsequently enhances its binding capacities to the downstream target proteins (i.e., 2-CysPrxB and SAT1) in two distinctive cellular processes; plant photosynthesis (growth) and OPDA signaling (defense) pathways.

Conclusion

Light is the principle energy source for plant growth and survival. In the chloroplasts, photo-receptors capture, and transform light into chemical energy for the photosynthetic carbon fixations and biomass productions, as well as create a chain of redox reactions (or ETC) which coordinates growth and defense responses (‘plant fitness’), programing optimal phenotypes under different ecological conditions.28,29 Here, environmental stressors appear as a major determinant as plants must continue growing and, at the same time, need to compete with their neighbors as well as defend themselves from the various forms of abiotic and biotic stresses. Defense (adaptive) responses toward the environmental stressors are, however, often constrained by resource availability. Diversion of resources to defense could limit growth processes, whereas their allocation to growth could reduce investment in defense responses. Therefore, plants must strike a precise balance in their responses to a myriad of environmental stress cues.17,29

Recently, emerging evidence has illuminated a unique activity of plant hormone signaling in converting light inputs into outputs that shape plant fitness. For instance in Arabidopsis, the perception of competing for light with neighboring plant species by photoreceptors activates shade-avoidance growing, and concurrently ensures defense responses against pathogens and insects (reviewed in ref. 29). A main mechanism
underlying the heightened defense state is to activate jasmonate signaling by low red:far-red (R:FR) radiation ratios. The low R:FR ratios appear to inactivate photoreceptors, leading to the upregulation of a subset of JA-responsive genes (e.g., Vegetative Storage Protein 1 and 2), and the increased induction of OPDA accumulations. In agreement, blocked OPDA signaling in cyp20–3 KO plants displayed hypersensitive reactions to oxidative stresses originated by several environmental cues including high-light intensity, substantiating a critical role of jasmonates, perhaps OPDA, in a key facet of light signaling.

In line with this scenario, the present study has located CYP20–3 at a regulatory hub where crossovers light and OPDA signaling (Fig. 4D). Light signaling converys ETC via Trx and NTRC-redox reaction systems (Fig. 4D, resting states), wherein CYP20–3 relays OPDA signaling in controlling ETC routes and efficiency (Fig. 4D, stressed states). Once it is induced under stressed states, OPDA binds CYP20–3, and stimulates its interactions and electron transfers from Trxs to SAT1 (e→Trxs→CYP20–3→SAT1) and 2-CysPrxB (e→Trxs→CYP20–3→2-CysPrxB). Reduction (activation) of 2-CysPrxB then continues peroxide detoxification in photosynthetic carbon metabolisms, while the activation of SAT1 promotes CSC formation and builds up reduction capacity that in turn triggers the retrograde regulation of nucleus defense gene expression. This model shed new light on i) a unique interface between light and hormone (OPDA) signaling, which ii) fine-tunes resource (e−) allocations between growth and defense responses iii) in making instant and appropriate adaptive decisions while being challenged constantly by environmental stressors, and maintaining necessary growth and development, ultimately balancing optimal growing phenotype.

Materials and methods

In vitro Protein-protein interaction assay

Recombinant protein preparation, and in vitro pull-down assay were performed as described previously. Briefly, coding sequences for full-length proteins were cloned into pGEX-4T-1 (GE Healthcare) and pET28a (Novogen) to obtain N-terminal GST-fused CYP20–3 and SAT1, and N-terminal 6× HIS/T7- tagged SAT1, Trxs and 2-CysPrxs, respectively. The recombinant proteins were prepared from Escherichia coli BL21 (DE3, New England Biolabs), and purified using a nickel- (Ni-NTA, Qiagen), or a glutathione- (GSH, GBiosciences) affinity column chromatography. The GST-fused CYP20–3 or SAT1 was then immobilized on GSH-affinity beads (Sigma) with Trxs, 2-CysPrxs and/or SAT1 in the presence of various concentration of OPDA for 1 h at room temperature, washed with 50 mM sodium phosphate buffer (pH 7.5, containing 150 mM NaCl), and elution with GSH. Next, all pull-downed proteins were resolved by SDS/PAGE, and probed with a monoclonal anti-His antibody (Invitrogen).

Quantitative RT-PCR

Total leaf RNA was prepared using TRizol reagent (Invitrogen) and RNase-free DNase (RQ1; Promega) according to the manufacturer’s instructions. RNA qualities were assessed by agarose gel electrophoresis and NanoDrop (A260/A280 > 1.8 and A260/A320 > 2.0). RT reactions were performed by using an oligo(dT) reverse primer and a reverse transcriptase (SuperScriptII; Invitrogen). The cDNA were assessed by quantitative PCR with 2 sets of housekeeping genes, POLYUBIQUITIN (UBC) and GAPDH. Quantitative PCR was performed with the SensiMix SYBR and Fluorescein kit (Bioline) in the CFX96 Touch™ (Bio-Rad) PCR system cycled 40 times by using genespecific primer sets (Table S2). The annealing temperatures for the primer pairs were 53 °C. To determine the relative abundance of target transcripts, the average threshold cycle (i.e., Ct) was normalized to that of UBC as 2−ΔΔCt, where −ΔΔCt = (Ctgene−CtUBC).

Statistical analysis

All statistical analyses followed by the Student’s t-test were performed using JMP® 13 software (SAS institute, USA).

Disclosure of potential conflicts of interest

The authors declare that they have no conflict of interest.

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