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Generation of Hydrogen Peroxide by Plant Peroxidases Mediated Thiol Oxidation

By

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S u m m a r y

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Thiol oxidation by plant peroxidases is an oxygen consuming process independent of hydrogen peroxide addition. The *in vitro* system consists of two interdependent reactions, i.e. hydrogen peroxide mediated oxidation of thiols via the enzymatic intermediates compound I and II yielding thiyl radicals, and further the non-enzymatic reaction of thiyl radicals to the corresponding disulphides or reaction with molecular oxygen yielding superoxide radicals and hydrogen peroxide. The thiyl radical generation per time (regulated by enzyme, thiol, and phenol concentration) determines the amount of hydrogen peroxide generated and as a consequence the amount of phenol which is oxidized by this plant enzyme. In the presence of phenols and with increasing pH oxygen consumption, thiol oxidation, and as a consequence the amount of hydrogen peroxide generated, increases. Considering phenol oxidation the system apparently becomes biphasic with a slow phenol oxidation phase A followed by a rapid phenol oxidation phase B. During phase A oxygen consumption occurs and thiol is oxidized completely. We present reaction schemes and discuss the biological relevance with regard to a possible biosynthetic route of hydrogen peroxide in plants.

I n t r o d u c t i o n

The oxidation of the thiol group of cysteine is involved in several functions of proteins and low molecular weight non-protein thiols, e.g. glutathione. The one-electron oxidation of the thiol group of cysteine results in the formation of a thiyl radical, which can undergo dimerization to the corresponding disulfide or react with oxygen, superoxide radicals or hydrogen peroxide according to the well known reactions presented in table 1 [(reactions (4) - (6) and (14) - (18)].

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One of the mechanisms by which thiyl radicals of cysteine or related compounds are formed *in vivo* is by reaction with haem peroxidases. Haem peroxidases (EC 1.11.1.7) are known to be poorly specific for their substrates. They can catalyse the oxidation of various organic and inorganic compounds such as phenols, arylamines, halides, and thiols, and with most combinations of peroxidases and substrates such oxidations have been found to proceed univalently (NAKAMURA & al. 1984, 1986). The formation of thiyl radicals has been confirmed for thiol oxidation promoted by horseradish peroxidase (HARMANN & al. 1984, 1986), lactoperoxidase (MOTTLEY & al. 1987), prostaglandine H synthase (ELING & al. 1985), and myeloperoxidase (SVENSSON & al. 1993) by electron spin resonance (ESR) spectroscopy using 5,5'-dimethyl-1-pyrroline N-oxide (DMPO) as spin trap.

The conventional peroxidase cycle involves reaction with hydrogen peroxide producing the enzyme intermediate compound I (which is two oxidizing equivalents above the resting enzyme ferriperoxidase) and two one-electron reductions back to the ferric-enzyme via the second enzyme intermediate compound II. Hydrogen peroxide is reduced to water and phenols are oxidized to phenoxy radicals which dimerize. No oxygen consumption is observed [reactions (1), (8), (9) in table 1, showing bimolecular reaction rates of the reaction of both peroxidase compounds with e.g. cinnamyl alcohols, the monomers for the lignification process].

By way of contrast in the presence of cysteine oxygen is consumed and the reaction becomes independent of hydrogen peroxide. The oxygen consumption by solutions containing cysteine and HRP has been shown to be not affected by further addition of hydrogen peroxide but decreased by DMPO. These results have been interpreted with thiyl radicals being formed and then trapped by DMPO before further reactions of the cysteinyl radical can lead to oxygen consumption.

In the meanwhile several thiol compounds have been studied. Some of the reactions were strongly dependent on peroxides, but in some cases addition of exogenous hydrogen peroxide was unnecessary (SVENSSON 1988a,b,c, SVENSSON & LINDVALL 1988, OSSWALD & al. 1989, PICHORNER & al. 1992, SVENSSON & al. 1993, OBINGER & al. 1996, BURNER & OBINGER 1997).

The principal features of peroxidase reactions with oxygen consumption have been found to be auto-oxidation of the substrate, yielding hydrogen peroxide. This peroxide is used by enzyme to oxidize the substrate to an intermediate, which can reduce molecular oxygen, whereby hydrogen peroxide and/or organic peroxides are regenerated (YAMAZAKI & YOKOTA 1973, HARMAN & al. 1984, WEFERS & al. 1985). Therefore it is necessary to differentiate between the enzymatic reactions (initiation reaction and reaction of the peroxidase intermediates I and II with thiols [(reaction (1) - (3))] and the non-enzymatic free radical reactions [e.g. reactions (4) - (7), (10) - (12), (14) - (18)].

This paper is a general view of the *in vitro* systems horseradish peroxidase/cysteine and horseradish peroxidase/cysteine/homovanillic acid and discuss the biological relevance in regard to hydrogen peroxide generation and regeneration necessary for peroxidase mediated oxidation of endogenous phenols.

The enzymatic reactions

Under aerobic conditions thiol oxidation by horseradish peroxidase is independent of added hydrogen peroxide. It is a well known fact (MISRA 1974) that thiols are oxidized by molecular oxygen in the presence of (nanomolar) free iron and/or copper ions producing the disulfide and traces of hydrogen peroxide required to initiate the peroxidase cycle via reaction (1). Such minute autooxidation has the consequence that compound I formation is observed upon addition of thiol solutions to peroxidases and that at nanomolar concentrations the enzymes exist in their compound II oxidation state (OBINGER & al. 1996).

Compared with phenols and arylamines [reactions (8) and (9)] aliphatic thiols are very bad substrates for HRP compound I and above all for compound II (OBINGER & al. 1996, BURNER & OBINGER 1997). With the exception of aromatic thiols and cysteine methyl ester the bimolecular rate constants for the reaction of horseradish peroxidase compound I and II with thiols are very low [reactions (2) and (3)]. In contrast to good substrates, which disappear by recombination or disproportionation, poor substrates are excellent candidates for free-radical chain processes. Bimolecular rate constants as shown in table 1 [reactions (2) and (3)] mean that the rates of thiol radical production are low with the consequence of oxygen consumption and hydrogen peroxide production via the well known reactions (5) - (7).

As long as phenols are absent both thiol oxidation and oxygen consumption are slowly. Compound II accumulates on addition of thiols to horseradish peroxidase reflecting the extremely low bimolecular rate constants of the reaction of HRP compound II with thiols. Hydrogen peroxide regeneration by the free radical processes discussed below is not the rate limiting step in this system. Addition of hydrogen peroxide does not affect oxygen consumption, because hydrogen peroxide already exists in the system owing to thiol autooxidation and regeneration [Fig. 1, reactions (5) - (7)].

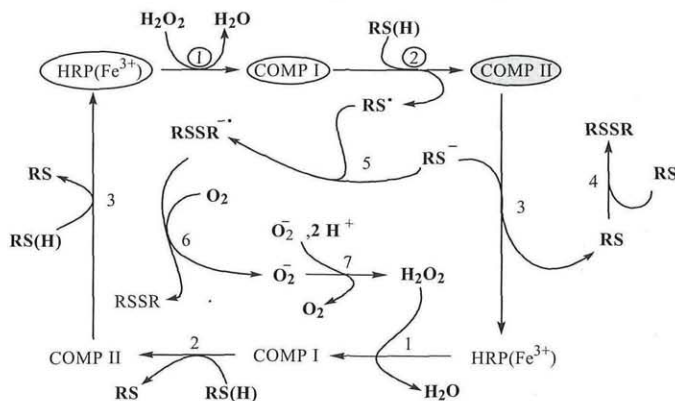


Fig. 1. The in vitro system horseradish peroxidase/cysteine accumulates compound II and hydrogen peroxide.

In the range 6 - 9 pH shows no influence on the rate constants of both compound I and II with cysteine and related compounds.

Non-enzymatic free radical reactions

In terms of oxygen consumption and disulphide production methyl and ethyl esters of cysteine are more active than cysteamine, whereas cysteine itself is poorly active and glutathione and N-acetylcysteine are nearly inactive. These facts (OBINGER & al. 1996, BURNER & OBINGER 1997) reflect the enzymatic reaction rates shown in table 1 [reactions (2) - (3)]. Release of superoxide anions is hardly detectable in the plant peroxidase catalyzed reaction (OBINGER & al. 1996, BURNER & OBINGER 1997). Again, only upon esterification of cysteine superoxide radical formation is detectable.

In contrast to the reaction rates measured by sequential-stopped flow analysis (OBINGER & al. 1996, BURNER & OBINGER 1997) overall thiol oxidation strongly depends on pH. Disulphide formation and oxygen consumption both increase with pH in the pH-range 5-9. This dependence of activity on pH coincides with the deprotonation of thiols.

The fate of thiyl radicals will reflect the kinetics of reactions that produce and remove them. The main conjugative reactions of thiyl radicals in cellular systems are conjugation with thiolate or molecular oxygen or dimerization to the corresponding disulfide (WARDMAN & VON SONNTAG 1995). Their reactions are controlled by pH, oxygen concentration, and thiol concentration. The steady-state concentration of thiyl radicals is in the nanomolar region and hence much lower than thiol (μM for cysteine) or oxygen ($30\text{-}40\mu\text{M}$ in oxygenated tissues) concentration. Therefore reactions of thiyl radicals with thiolate or oxygen predominate above combination forming disulphides via reaction (4).

It is a well known fact that thiyl radicals react more rapidly with thiolate anions than with the protonated forms [see reaction rates of reaction (5)] explaining the pH-dependence of the overall thiol oxidation. The formed disulfide radical anion is relatively stable and a powerful reductant. It reduces molecular oxygen [reaction (6)] producing superoxide radicals which disproportionate to molecular oxygen and hydrogen peroxide [reaction (7)].

Thiyl radicals can also react with molecular oxygen according reaction (14), but reactions of thiylperoxyl radicals are likely to be slower than those of the thiolate anions under physiological conditions (WARDMAN & VON SONNTAG 1995). Thiylperoxyl radicals may either rearrange (forming a sulfonyl radical) or transfer an oxygen atom to thiol (forming a sulfinyl radical). But both slower reaction rates and the fact that cystine is nearly the only oxidation product of cysteine oxidation by plant peroxidases indicate that thiyl radicals react via the superoxide releasing pathway [reactions (5) - (7)].

Table 1. Elementary reactions participating in the HRP-catalyzed oxidation of thiols in the absence or presence of phenols.

Reaction	Rate Constant	pH	Ref.
(1) $\text{HRP}(\text{Fe}^{3+}) + \text{H}_2\text{O}_2 \rightarrow \text{Compound I} + \text{H}_2\text{O}$	$1.8 \times 10^7 \text{ M}^{-1} \text{ s}^{-1}$	7.0	DOLMAN & al. 1975
(2) $\text{Compound I} + \text{RSH} \rightarrow \text{Compound II} + \text{RS}^{\cdot}$			
RSH : cysteine	$240 \text{ M}^{-1} \text{ s}^{-1}$	7.0	OBINGER & al. 1996, BURNER & OBINGER 1997
Cysteamine	$320 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Penicillamine	$24 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
N-Acetylcysteine	$40 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Homocysteine	$130 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Cysteine methyl ester	$5800 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Cysteine ethyl ester	$660 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Glutathione	$41 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
(3) $\text{Compound II} + \text{RSH} \rightarrow \text{HRP}(\text{Fe}^{3+}) + \text{RS}^{\cdot}$			
RSH : cysteine	$< 50 \text{ M}^{-1} \text{ s}^{-1}$	7.0	OBINGER & al. 1996, BURNER & OBINGER 1997
Cysteamine	$< 100 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Penicillamine	$\sim 0 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
N-Acetylcysteine	$\sim 0 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Homocysteine	$< 50 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Cysteine methyl ester	$\sim 300 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Cysteine ethyl ester	$< 100 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Glutathione	$\sim 0 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
(4) $\text{RS}^{\cdot} + \text{RS}^{\cdot} \rightarrow \text{RSSR}$	$1-3 \times 10^9 \text{ M}^{-1} \text{ s}^{-1}$	7.0	WARDMAN & SONNTAG 1995
(5) $\text{RS}^{\cdot} + \text{RSH} \leftrightarrow [\text{RSS}(\text{H})\text{R}]^{\cdot}$	$\sim 10^3 - 10^4 \text{ M}^{-1} \text{ s}^{-1}$		WARDMAN & SONNTAG 1995
$\text{RS}^{\cdot} + \text{RS}^{\cdot} \leftrightarrow [\text{RSSR}]^{\cdot}$	$\sim 5 \times 10^8 - 5 \times 10^9 \text{ M}^{-1} \text{ s}^{-1}$		
(6) $[\text{RSSR}]^{\cdot} + \text{O}_2 \rightarrow \text{RSSR} + \text{O}_2^{\cdot -}$			WARDMAN & SONNTAG 1995
glutathione	$5 \times 10^9 \text{ M}^{-1} \text{ s}^{-1}$		
α -lipoic acid	$9 \times 10^8 \text{ M}^{-1} \text{ s}^{-1}$		
(7) $\text{O}_2^{\cdot -} + \text{O}_2^{\cdot -} + 2 \text{H}^+ \rightarrow \text{H}_2\text{O}_2 + \text{O}_2$	$1.8 \times 10^7 \text{ M}^{-1} \text{ s}^{-1}$	5.1	RABANI & NIELSEN 1969
	$\sim 10^6 \text{ M}^{-1} \text{ s}^{-1}$	7.0	BIELSKI & al. 1985
(8) $\text{Compound I} + \text{AH}_2 \rightarrow \text{Compound II} + \text{AH}^{\cdot}$			
(AH ₂): Homovanillic acid	$8 \times 10^4 \text{ M}^{-1} \text{ s}^{-1}$	7.0	OBINGER & al. 1996, BURNER & OBINGER 1997
Coniferyl alcohol	$8.0 \times 10^7 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Sinapyl alcohol	$6.2 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	7.0	FURTMÜLLER & al. 1996
			FURTMÜLLER & al. 1996
(9) $\text{Compound II} + \text{AH}_2 \rightarrow \text{HRP}(\text{Fe}^{3+}) + \text{AH}^{\cdot}$			
(AH ₂): Homovanillic acid	$9 \times 10^3 \text{ M}^{-1} \text{ s}^{-1}$	7.0	OBINGER & al. 1996, BURNER & OBINGER 1997
Coniferyl alcohol	$1.0 \times 10^7 \text{ M}^{-1} \text{ s}^{-1}$	7.0	
Sinapyl alcohol	$4.1 \times 10^4 \text{ M}^{-1} \text{ s}^{-1}$	7.0	FURTMÜLLER & al. 1996
			FURTMÜLLER & al. 1996
(10) $\text{AH}^{\cdot} + \text{AH}^{\cdot} \rightarrow \text{HA-AH}$			
AH [·] : tyrosyl radical	$4.5 \times 10^8 \text{ M}^{-1} \text{ s}^{-1}$		SIMIC & HUNTER 1984
(11) $\text{AH}^{\cdot} + \text{RSH} \rightarrow \text{AH}_2 + \text{RS}^{\cdot}$			WARDMAN & SONNTAG 1995
(12) $\text{RSH} \rightarrow \text{RS}^{\cdot}$			
			MISRA 1974
(13) $\text{Compound II} \rightarrow \text{HRP}$	$3 \times 10^{-3} \text{ s}^{-1}$	7.4	KRYLOV & DUNFORD 1996
(14) $\text{RS}^{\cdot} + \text{O}_2 \rightarrow \text{RSOO}^{\cdot}$			WARDMAN & SONNTAG 1995
RSH: glutathione	$3.2 \times 10^3 \text{ M}^{-1} \text{ s}^{-1}$		
(15) $\text{O}_2^{\cdot -} + \text{AH}^{\cdot} + \text{H}^+ \rightarrow \text{AH}_2 + \text{O}_2$			
AH [·] : tyrosyl radical	$1.5 \times 10^9 \text{ M}^{-1} \text{ s}^{-1}$		SIMIC & HUNTER 1984
(16) $\text{O}_2^{\cdot -} + \text{RSH} + \text{H}^+ \rightarrow \text{GSO}^{\cdot} + \text{H}_2\text{O}$			WINTERBOURN & METODIEVA 1995
RSH: glutathione	$10^3 \text{ M}^{-1} \text{ s}^{-1}$		
(17) $\text{GSO}^{\cdot} + \text{GS}^{\cdot} + \text{H}^+ \rightarrow \text{GS}^{\cdot} + \text{GSOH}$			
(18) $\text{GSOH} + \text{GSH} \rightarrow \text{GSSG} + \text{H}_2\text{O}$			

(224)

Oxygen uptake and thiol oxidation are stimulated by addition of phenols. In the presence of phenols (Fig. 3) under turnover conditions ferriperoxidase accumulates meaning that hydrogen peroxide regeneration is the rate limiting step. With nanomolar enzyme concentrations the system is biphasic as phenol oxidation starts at a very low rate (phase A in figure 2, I). During this phase thiol oxidation and concomitant oxygen consumption occurs (PICHORNER & al. 1992, OBINGER & al. 1996, BURNER & OBINGER 1997). Increasing peroxidase concentration shortens phase A and at high enzyme concentrations the system becomes monophasic (Fig. 2, II). Besides the length of phase A the total amount of phenol oxidized during phase B strongly depends on enzyme and thiol concentration (OBINGER & al. 1996, BURNER & OBINGER 1997).

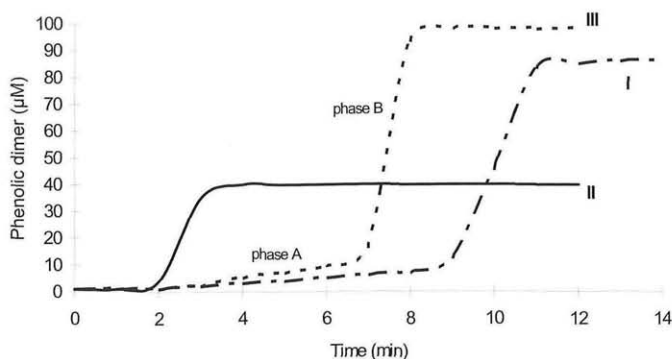


Fig. 2. (I) Schematic presentation of the biphasic behavior of peroxidase mediated phenol and thiol oxidation of the in vitro system horseradish peroxidase (7 nM)/cysteine (25 μM)/homovanillic acid (500 μM). During phase A all thiol is oxidized (accompanied by oxygen consumption), whereas during phase B phenol is oxidized following the classical peroxidase pathway [reactions (1), (8) and (9)]. At high (0.1 μM) enzyme concentrations (II) the system becomes monophasic. Necessary hydrogen peroxide derives from reactions happening during phase A. Superoxide dismutase (Cu/Zn 150 units) shortens phase A and increases the amount of H-A-A-H [III, enzyme as in I].

Phenolic compounds are promoters of the peroxidase-catalyzed thiol oxidation by reducing the rate-limiting HRP intermediate, HRP-II and/or by oxidation of thiols by free radicals of phenols which helps to explain the biphasic behaviour of the system peroxidase/thiol/phenol. Addition of phenols accelerates oxygen consumption, but has no effect on the total amount of oxygen consumed, which only depends on thiol concentration. As long as thiols are present phenol radicals are repaired by thiols by hydrogen (or electron/proton) donation ("thiol-pumping") as shown in equation (11). Increasing enzyme turnover has the consequence that phenol radical dimerization becomes more and more important (shortening phase A) as well as thiol radical combination via reaction (4) (decrease in hydrogen peroxide formation and hence phenol dimerization). The maximum

hydrogen peroxide yield strongly depends on the relative concentrations of enzyme, thiol and phenol (OBINGER & al. 1996, BURNER & OBINGER 1997).

Addition of superoxide dismutase (SOD) shortens the lag phase and increases the total amount of phenolic dimer (HA-AH) produced, whereas in the presence of catalase phenol oxidation is suppressed completely. The presence of SOD prevents possible reactions of superoxide anions with phenoxy radicals and/or thiols as indicated by reactions (15) and (16). The physiological relevance of this in vitro system has to be discussed. Firstly it represents a reaction system which is likely to occur in vivo because it has been investigated with representative thiol, phenol, and enzyme concentrations (OBINGER & al. 1996, BURNER & OBINGER 1997) and secondly it makes the peroxidase system independent of a hydrogen peroxide generating enzymatic system.

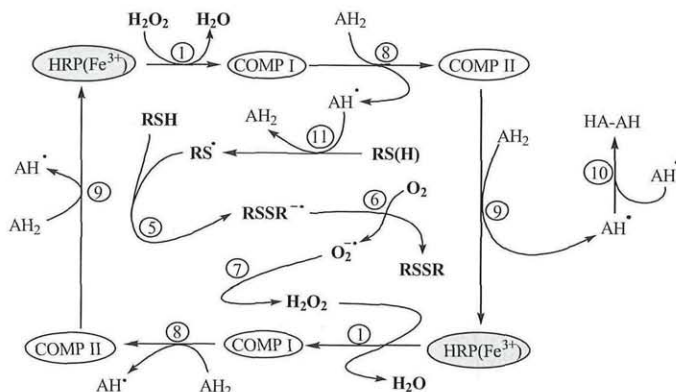


Fig. 3. The in vitro system horseradish peroxidase/cysteine/phenol accumulates ferriperoxidase.

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