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Reactive Oxygen Species and Apoplastic Switch

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K e y w o r d s : Oxygen stress, reactive oxygen species, growth and defence, antioxidants, regulation.

S u m m a r y

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One main common problem during most diseases both of plants and animals is „oxygen stress". The biochemistry of oxygen - activation and - detoxification analyzed in the past has led to the identification of many similar or more or less identical features both in plants and animals.

Plants developed a special strategy for defence combining avoidance and tolerance reactions with a sophisticated set of chemicals synthesized either constitutively or when needed. Coevolution of animals took advantage of the synthesizing capacity of plants sparing the synthesis of certain „expensive" groups of chemicals such as phenolics. These phenolics are involved in defence against pathogens and as "preformed" molecules potentially also in the decision between growth or defence by governing the peroxidase-oxidase activities in the apoplast working as "apoplastic switch".

Oxygen Stress in Plants

The term stress originally stems from physics and has been extended and introduced in medicine and botany. In general mechanics stress is clearly defined as the point of bending of an elastic system next to just symptomless reversibility and irreversible deformation or break.

In medicine and botany, stress is supposed to indicate all situations beyond normal, defined by the observer, sometimes a scientist.

All organs of higher plants (with some exceptions) perform aerobic metabolism and are subject to activated oxygen species, and thus oxygen "stress". This

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(10)

may occur under various different conditions, underlying a cascade of events (Fig.1).

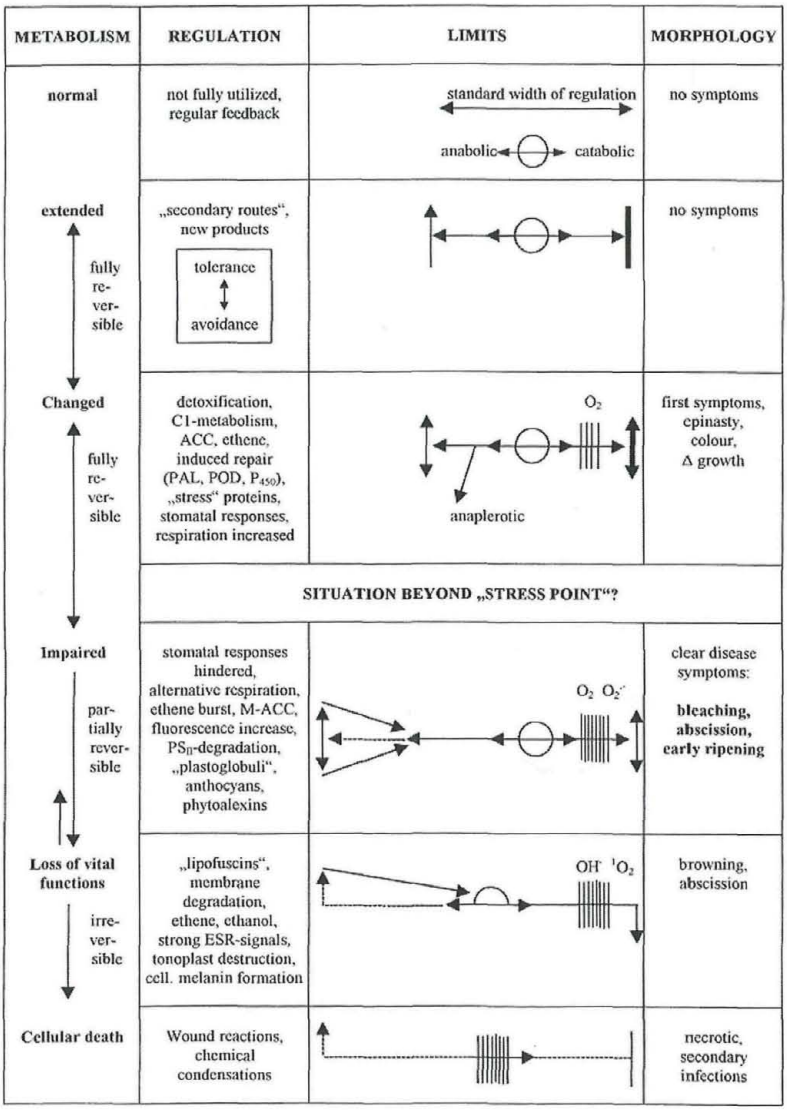

Fig. 1. The stress cascade in plants (SCHEMPP & al. 2005a).

Defence reactions against pathogens both in plants and animals produce reactive oxygen species (ROS) in several cellular compartments by different mechanisms. Thus for protection against damage antioxidative strategies have been developed.

In this review basic reactions operating during oxidative stress situations (ELSTNER 1982, 1987, 1991, ELSTNER & al. 1996) will be dealt with.

Air pollution and radiation

Certain trace gases, especially ozone and SO_2 as air pollutants (SCHEMPP & al. 2005b) and the increase of UVB as an effect of the decrease of the stratospheric ozone layer together with radioactive deposition (Tchernobyl remainder) must be seen as potentially toxic for plants.

UV - damage in plants is negligible due to the plant´s protective systems on the basis of a complex set of UV - absorbant phenolics (JANSEN & al. 2001). SO_2 in most regions of the industrial world (western hemisphere) due to binding with CaO (forming gipsum) after the burning process in the power plants is ameliorated. In former times, when SO_2 concentrations in industrial regions or in their downstream exhaust could reach up to $1000\mu g/m^3$ it has caused serious pathological problems in both animals and plants (HIPPELI & ELSTNER 1996a,b). Ozone may reach concentrations in the atmosphere which urges the plants to react and seems to be of higher relevance to plants as compared to animals and man (ELSTNER & al. 1996). On the other hand, Ozone has been shown to act as a abiotic elicitor inducing oxidative defence and has been used as a tool for analyzing stress responses in terms of predisposals to pathogen attack (SANDERMANN & al. 1998, LANGEBARTELS & al. 2000).

Metabolic events in green plants forming ROS

Stressors such as cold, drought and many others cause internal oxygen activation via feed-back of metabolic blocks to the photosystems. External stress always has a change of internal metabolism as consequence, mediated by a complex interaction of hormones. Adaptation to drought, for example, is counteracted by stomatal closure. This event is initiated by the hormone abscisic acid mediated by H_2O_2 produced via a NADPH oxidase (ZHANG & al. 2001).

Green plants have adapted to extremely different environmental conditions. Since normally plants, in contrast to animals, cannot escape, they have either to adapt or to die. Most of these visible or measurable symptoms have been shown to be connected with oxygen activation (ELSTNER 1982, 1987, ELSTNER & OßWALD 1994, HIPPELI & ELSTNER 1996a,b) where principally a transition from heterolytic (two electron transitions) to increased homolytic (one electron transitions) reactions is observed. Homolytic reactions create free radicals. We thus address these situations as „oxidative stress".

Avoidance and defence strategies

Every episode during a hypothetic stress cascade is characterizable by the balance between pro- and antioxidant capacities (SCHEMPP & al. 2005a). In the

beginning the plant tries to minimize electron pressure arising from photosystem II (PSII) by

- reducing the „optical diameter" i.e the light harvesting systems (LHCII) of PSII by transfer and integration of the chlorophyll-protein complex into photosystem I (ALBERTSSON 2001) via phosphorylation where molecular recognition dominates the organization of the thylakoid structure and function (ALLEN & FORSBERG 2001), and by non-photochemical quenching (MÜLLER & al. 2001).
- induction of photorespiration, connecting several cellular compartments by „carbon-idling" and enhancement of C-1 metabolism (ELSTNER 1987, COSSINS 1987, HANSON & al. 2000),
- H_2O_2 cycling via the „Beck-Halliwell-Asada-cycle" (ASADA 1999),
- the xanthophyll cycle (MÜLLER & al. 2001, DEMMING-ADAMS & ADAMS 1996, POLLE & al. 2001) protecting PSII,
- photoinhibition by inacitvating the electron - „outlet" of PSII via radical-induced degradation of the D-protein (MELIS 1998), and ATP - dependent resynthesis (ALLAKHVERDIEV & al. 2005),
- via the novel and very recently decribed plastid terminal oxidase (quinone-oxygen oxidoreductase) resembling the cyanide-resistant alternative oxidase linked to carotene desaturation wich is responsible for chlororespiration (CAROL & KUNTZ 2001).

Both photoinhibition (e) and chlororespiration (f) are tuned by the redox-state of the plastoquinone-cycle which seems to play the dominant, pivotal role in the feedback regulation of the high fidelity - functioning of the photosytems and thus the security of the whole thylakoid system.

By induction of key enzymes of the shikimat- and mevalonate pathways, a wealth of antibiotic and/or antioxidative molecules belonging to several structural classes are de-novo synthesized. Most of these molecules are supposed to be involved in the over-all strategy of plant´s defence and are called phytoalexins; their inductions may be brought about by either abiotic (ozone, wounding) or biotic (pathogens) effectors, or both (KUC 1995, HAMMERSCHMIDT 1999).

In the following we shall concentrate on basic redox-mechanisms during oxidative stress in plants.

Pathways of oxygen activation in plants (c.f.SCHEMPP & al. 2005a)

A radical is a compound containing an unpaired electron. There are stable and unstable radicals. Most free radicals are highly reactive creating new radicals thus initiating chain reactions. Oxygen is a very stable biradical in the triplet ground state ($^3O_2$) and has to be activated in order to react with atoms or molecules in the "normal" singlet ground state thus circumventing spinforbidden reactions. The most important reactions of oxygen activation are briefly adressed in the following.

Light reactions

Oxygen can be activated by photodynamic reactions:

$$P + \text{light} \rightarrow P^* \qquad (1)$$

$P^* + {}^3O_2 \rightarrow {}^1O_2 + P$ (exciton transfer forming singlet oxygen)
P represents a pigment in its ground (singlet) state and P^* it is activated triplet form

Reaction (1) represents a photodynamic reaction classified as photodynamic reaction type II where singlet oxygen ($^1O_2$) is formed.

In contrast to atmospheric oxygen, $^1O_2$ is not subject to the spin rule and reacts rapidly with most organic molecules (RH), especially at double bonds, producing hydroperoxides:

$$RH + {}^1O_2 \rightarrow ROOH \qquad (2)$$

ROOH in turn can be reduced by one electron donors (E^- representing reduced transition metal ions like Fe^{2+} or Cu^+, semiquinones, heme- and nonheme proteins, isoalloxazines or pteridines) yielding alkoxyl (RO·)-radicals:

$$E^- + ROOH \rightarrow E + RO\cdot + OH^- \qquad (3)$$

These RO· radicals may initiate chain reactions thus reacting further cooxidizing other molecules, for example by initiating cooxidative bleaching of pigments:

$$RO\cdot + RH \rightarrow R\cdot + ROH \qquad (4)$$

Photodynamic reactions undergoing charge separation within the excited pigment are called photodynamic reaction type I (where may $_+P$ represent a photooxidized, i.e. bleached pigment:

$P + \text{light} \rightarrow P^*$ (pigmant activation) (5)

$P^* \rightarrow {}_+P^-$ (charge separation) (5a)

${}_+P^- + O_2 \rightarrow {}_+P + O_2{}^{\cdot-}$ (superoxide formation) (5b)

Phototoxins

Production of ROS is observed after illumination of cercosporin, a perylenequinone toxin produced by several phytopathogenic Cercospora species. Cercosporin (Cerc) mainly seems to induce the formation of singlet oxygen and superoxide (YOUNGMAN & al. 1983, DAUB & HANGARTER 1983, YOUNGMAN & ELSTNER 1984) in photodynamic reactions both of type I and type II (see reactions (1) and (5)):

$Cerc + \text{light} \rightarrow Cerc^*$ (triplet state)
$Cerc^* + {}^3O_2 \rightarrow Cerc + {}^1O_2$ (type II reaction) (5c)
or:

(14)

$Cerc^* \rightarrow {}_+Cerc^-$

${}_+Cerc^- + O_2 \rightarrow {}_+Cerc + O_2^{\cdot -}$ (type I reaction)

Similar light dependent reactions are well known and observed with other plant - derived products such as hypericins from St. John´s wort or several furanocoumarins from different Apiaceae.

Under illumination cercosporin induces lipid peroxidation in plant cells (CAVALLINI & al. 1979, DAUB & al. 1983) followed by changes in membrane structure. Singlet oxygen quenchers like DABCO (diazabicyclooctane) delayed killing of cells by cercosporin (DAUB & al. 1983). The formation of ROS by bacterial and fungal phytotoxins has recently been reviewed by HEISER & al. 1998, 2005.

Reductive oxygen activation

In the presence of appropriate reductants (see above; high affinity for oxygen; negative redox potential: Eo of the redox pair $O_2/O_2^{\cdot -} = -330$ mV; the reducing site of PSI has a redox potential of approximately -600mV or even lower), superoxide may be formed from atmospheric oxygen:

$$E^- + O_2 \rightarrow E + O_2^{\cdot -} \quad \text{(superoxide formation)} \qquad (6)$$

Superoxide dismutates at neutral pH in aqueous media with a rate constant $k = 2 \times 10^5\ L \cdot M^{-1}\ sec^{-1}$, yielding hydrogen peroxide:

$$O_2^{\cdot -} + O_2^{\cdot -} + 2\,H^+ \rightarrow H_2O_2 + O_2 \qquad (7)$$

Similar to reaction (3), hydrogen peroxide may be reduced by the certain electron donors yielding the highly reactive hydroxyl radical (OH, redox potential close to +2V):

$$H_2O_2 + E^- \rightarrow E + OH^- + \cdot OH \text{ or} \qquad (8)$$

$$H_2O_2 + Fe^{2+} \rightarrow Fe^{3+} + OH^- + \cdot OH \qquad (8a)$$

Reaction (8a) is known as "Fenton"-reaction.

Fe^{3+} in can be re-reduced to by superoxide ion:

$$Fe^{3+} + O_2^{\cdot -} \rightarrow Fe^{2+} + O_2 \qquad (9)$$

The sum of reactions (8a) and (9):

$$H_2O_2 + O_2^{\cdot -} \rightarrow \cdot OH + OH^- + O_2 \qquad (10)$$

is known as "Haber-Weiss" reaction as recently reviewed by HIPPELI & ELSTNER 1999.

Under in vivo condition, the production and function of the „free OH radical" is under doubt (SARAN & al. 2000) and „Crypto OH" (YOUNGMAN & ELSTNER 1981) or metal-peroxide/peroxide -electron donor complexes have been proposed instead (WINTERBOURN 1999).

Lipid peroxidation and induction of chlorosis and necroses by ROS

Oxidative processes such as lipid peroxidation occur if ROS are produced at a cellular level and the detoxification system is overloaded or exhausted. The peroxidation of unsaturated fatty acids is induced by hydroperoxy radicals (HO_2) or by OH-radicals or electron donor-H_2O_2-complexes i.e. "crypto-OH" (YOUNGMAN & ELSTNER 1981, WINTERBOURN 1999) primarily yielding lipid radicals via abstraction of a hydrogen atom. Singlet oxygen directly produces lipid hydroperoxides in analogy to reaction 2.

Alkoxyl radicals (LO·) from this process can attack pigments such as chlorophyll (CHL) or carotenes which are oxidized (bleached) by "cooxidation" (HEISER & al. 1998).

$$LO\cdot + CHL_{red} \rightarrow LOH + CHL_{ox} \qquad (11)$$

Phytopathological aspects

Several authors and editors (c.f. PELL & STEFFEN 1991, SCHEMPP & al. 2005 a+b) reported on developmental processes and ecological and pathological factors connecting stress and oxygen activation. Wounding or other mechanical impacts influence transport across membranes or in the apoplast by increasing metabolic feedback till to the photosystems thus inducing oxidative processes. Fatty acid peroxidation through decompartmentalization, ionizing or UV- radiation, drought, flooding, osmotic impacts (high salt concentrations, desiccation), deficiency in macro- or micronutrients, dramatic temperature changes such as heating, chilling or freezing as well as poisoning by air pollution (SCHEMPP & al. 2005b) soil pollution (HORST & al. 2005, HU & SCHMIDHALTER 2005), herbicide treatment (FEDTKE & DUKE 2005) cause changes in pro- and antioxidative potentials and are responded by hormone synthesis or release which in turn may increase resistance, stability, avoidance, tolerance or repair.

Again, the most important notion is that all these impacts underly feedback to the chloroplast: independent on the site of transport- or metabolic "block", be it in the roots (salt, drought, mineral deficiency), in the transport system (xylem or phloem blocks by infections), in phloem loading (photooxidants, infections), limitations in CO_2 fixation (stomatal closure, lack of Calvin cycle activities due to enzyme inhibition) or in photosynthetic electron transport itself (Fig. 2).

As a consequence photosynthetic oxygen activation and/or photodynamic processes are provoked, leading to a chain of reactions as outlined by ELSTNER & OßWALD 1994, HIPPELI & ELSTNER 1996b and SCHEMPP & al. 2005a.

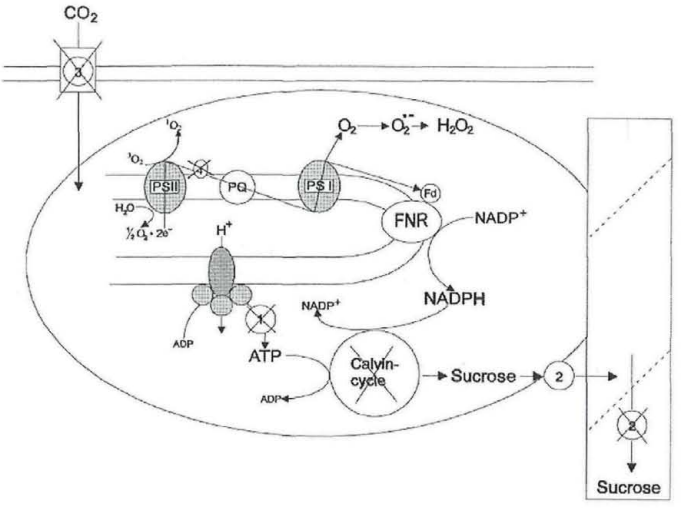

Fig. 2. Stress feedback to the chloroplast.

Toxins

An important field of research on oxygen activation during host-pathogen interactions (BARNA & KIRALY 2005, HEISER & al. 2005) concerns the investigations on reaction mechanisms of toxins produced by the pathogenic fungi and bacteria. Several such toxins have been shown to act as redox cyclers or photodynamically, i.e. through the light dependent formation of activated oxygen. In this context the quinoid derivatives cercosporin, dothistromin and dihydrofusarubin have to be mentioned (see above, HEISER & al. 1998). In other cases bacterial or fungal toxins introduced into the the plants react in analogy to certain herbicides such as the "quat"dyes: As recently documented, naphthazarin toxins such as dihydrofusarubin produced by certain strains of Fusarium solani induce redox cycling and superoxide production with similar kinetics as methylviologen after reduction by photosystem I (ALBRECHT & al. 1998) or by certain NAD(P)H oxidoreductases (such as diaphorases). These redox reactions are responsible for the observed bleaching and necrotization of the treated plants. All oxygen activating phytotoxins have been shown to include both photodynamic and reductive mechanisms.

Protection from oxygen stress

Since activated oxygen is toxic it has to be continuously under strict control of integral detoxification processes, detoxificating enzymes and organic antioxidants. One principle way to deal with oxygen toxicity is „avoidance", i.e. circumventing one or two electron donating processes towards oxygen (see above). This can be achieved by „tight" coupling of electron transport chains operating at the electronegative region of oxygen activation or by stoichiometric coupling of

oxygen activating processes with utilization of activated oxygen. Another possibility is the inhibition or inactivation of oxygen activating processes or enzymes. This has been shown for xanthine oxidase, lipoxygenases, prostaglandine cyclase, NAD(P)H oxidases and other enzymes by a wealth of compounds used in medicine. The so-called NSAIDs (non-steroidal antiinflammatory drugs) and several flavonoids are good examples for this principle.

On the other hand induction of defense systems against oxidative stress by ROS sensors such as oxyR or soxRS, responsive to hydrogen peroxide and to superoxide and NO, respectively, seem to be ancient prerequisits for survival, already present in bacteria such E.coli. New ROS - "defense genes" have recently described for higher plants such as rice (TSUKAMOTO & al. 2005). In tomato, the cryptochrome 2, a flavin-containing blue light photoreceptor, has been shown to induce the synthesis of antioxidants such ascarotenoid (lycopene) and flavonoids (chlorogenic acid, rutin) (GILIBERTO & al. 2005).

Integral detoxification processes

Integral detoxification processes connect elementary reactions of intermediary metabolism with detoxification of reactive oxygen species. This may be achieved

i.) by activation of enzymes or induction of isoenzymes such as peroxidases, DT-diaphorase
or members of the P_{450}-group;

ii.) through coupling of peroxide-utilization with NADPH-oxidation via ascorbate peroxidase, ascorbate-glutathione-glutathione reductase

iii.) by the „peroxidized membrane repair team", including phosoplipase(s) and glutathione peroxidase thus concertantly opening membraneous positions of peroxidized fatty acids for renewing activities.

Thus, detoxification in a wider sense also concerns the replacement of damaged molecules such as DNA, proteins and membrane lipids by a complex „crew" of integrated repair enzymes and replacement processes. A continuous involvement of these repair processes, however, would render them inactive since they also continuously function as targets of these reactive oxidants. Therefore, another batch of first aid molecules such as phenolics is biologically more than logic:

The only „help" for the final repair teams are small molecules with „Kamikazee-type" virtues, representing antioxidants with or without chance to be metabolically repaired themselves.

Shortcomings of detoxifying enzymes

As already mentioned, detoxification by enzymic processes is only possible, if the reactivity of the oxygen species under question is reasonably low under physiological conditions so that the enzymic reaction allows at least one k-order of magnitude between the reaction under enzyme catalysis and the non-catalyzed spontaneous reaction between the oxygen species and any reaction partner in its „molecular" neighbourhood. Therefore, the reactions of $OH^{\cdot}$, $O_2^{\cdot}$, $RO^{\cdot}$, $ROO^{\cdot}$ and

HOO˙are not under enzymic control; their reaction constants with potential reaction partners in their typical „environments" is too fast (generally k>>108) for enzyme catalysis. Thus, the reactions of biomolecules with these oxygen species have to be „amended" after damage. In order not to „flood" these repair process the above mentioned antioxidative molecules serve as scavengers and quenchers of acitivated states.

Enzyme-catalyzed detoxifications mainly concern superoxide, peroxides and epoxides (produced by cytochrome-P_{450}-activities) as more or less „stable" reduced oxygen species.

The Apoplastic Compartment: The Cell Walls as Battle Field

Plant cells, in contrast to animal cells contain more or less rigid cell walls which, together with the intercellular space, comprise the apoplastic compartment. Oxygen activation in this extracellular space includes both products derived from the NADPH oxidase of the plasma membranes and the products of cell wall intrinsic enzymes such as peroxidase(s) (ELSTNER 1991, ELSTNER & OßWALD 1994). These peroxidases produce hydrogen peroxide at the expense of NAD(P)H in a manganese catalyzed reaction allowing an extremely complex crosslinkage of C6-C3 phenylpropanoids forming lignine (HATFIELD & VERMERRIS 2001) as an essential element of woody plants and generally in pathogen defence.

Phenolic compounds play an important role in this context acting in addtion to antioxidants, as inducers of enzymes, as transition metal chelators thus avoiding Haber-Weiss-Fenton-chemistry and cofactors of regulation of enzymic activities. In the following chapter we wish to present some experiments documenting a potential role of the phenolics in vitro for an "Apoplastic Switch Hypothesis" possibly governing the transition from growth to defense.

Plant peroxidases - is their oxidase activity modulated by distinct phenolics thus acting as apoplastic switch?

It is long known that plant peroxidases, i.e. HRP, are excellent catalysts of the H_2O_2-independent oxidation of indole acetic acid (IAA) in vitro. IAA-oxidase activity is thought to occur mainly in the apoplastic space of higher plants, where several peroxidase isozymes as well as diverse phenolics and ascorbic acid are present. Figure 3 shows a cartoon of the oxidative and peroxidatve cycles of apoplastic peroxidases working either as indole acetic acid oxidase or as peroxidase.

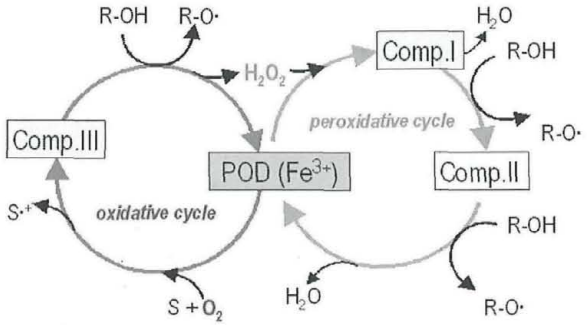

Fig. 3. Oxidative and peroxidative cycle of peroxidase. (R-OH=phenol; RO.=phenoxy radical; S=oxidase-substrate; S.+=oxidized substrate).

The plant hormone IAA stimulates growth i.e. cell elongation. During the polar transport from the IAA producing meristemal cells IAA occurs in the apoplast wher several peroxidase isoenzymes are present. It is long known that these PODs are excellent oxidases of IAA in the absence of H_2O_2. This reaction may, at least partially, be responsible for the control of apoplastic IAA concentrations. As shown earlier (VOLPERT & al. 1995), different phenolics influence these activities of peroxidases: monophenols stimulate oxidase acitivity while diphenols and methoxylated diphenols are inhibitory (Fig. 4).

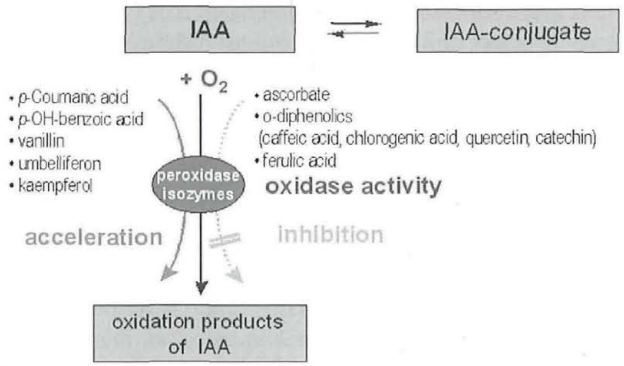

Fig. 4. Control of apoplastic IAA-level via degradation or conjugation.

This regulatory influence occurs within an extremely narrow cocentration range: 10 µM p-coumaric acids stimulate POD-catalyzed IAA oxidation; the addition of increasing amounts of either caffeic or ferulic acid (FA) exhibit and abrupt

jump from p-coumaric acid dependent stimulation into inhibition as shown in Fig. 5.

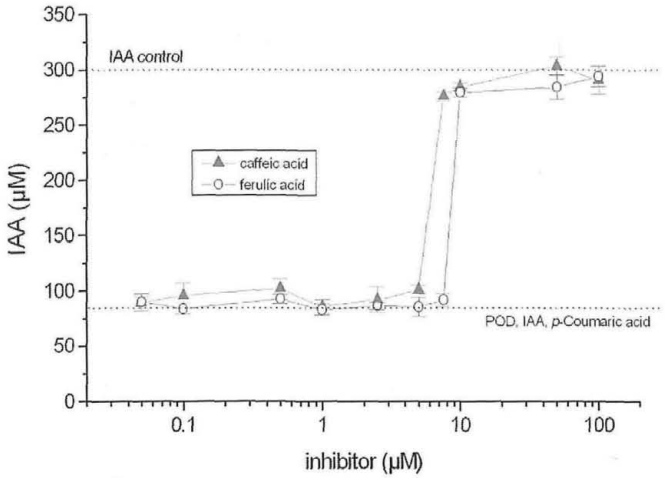

Fig. 5. Inhibition of HRP-IAA-oxidase activity by caffeic and ferulic acid (conditions: phosphate 50 mM pH 6.0, IAA 300 µM, p-Coumaric acid 10 µM, HRP 0.3 u/ml; incubation: 30 min at 25 °C; detection of IAA via UV-abs. at 275 nm after HPLC separation).

There is also strong evidence for dimerization of ferulic acid as a rapid response in the initial stages of plant defence against pathogens. Driven by the oxidative burst cell wall peroxidases catalyze crosslinking of FA estrified to arabinoglycans yielding effective hindrance of pathogens getting close to the plasmalemma. However, crosslinking of FA will be only efficient after depletion of ascorbate since it reduces FA radicals back to FA thereby indirectly performing an ascorbate-dependent detoxification of hydrogen peroxide. These sensible changes within micromolar concentrations of stimulatory versus inhibitory phenolics may modulate the transition from growth to defence.

Yet another reaction may be involved in this extremely complex issue: the ethene precursor, also present in the apoplastic space is counteracting the IAA depending cell growth. Cell wall peroxidases may also contribute to ethylene formation from ACC, also regulated by phenolics as shown in Fig. 6.

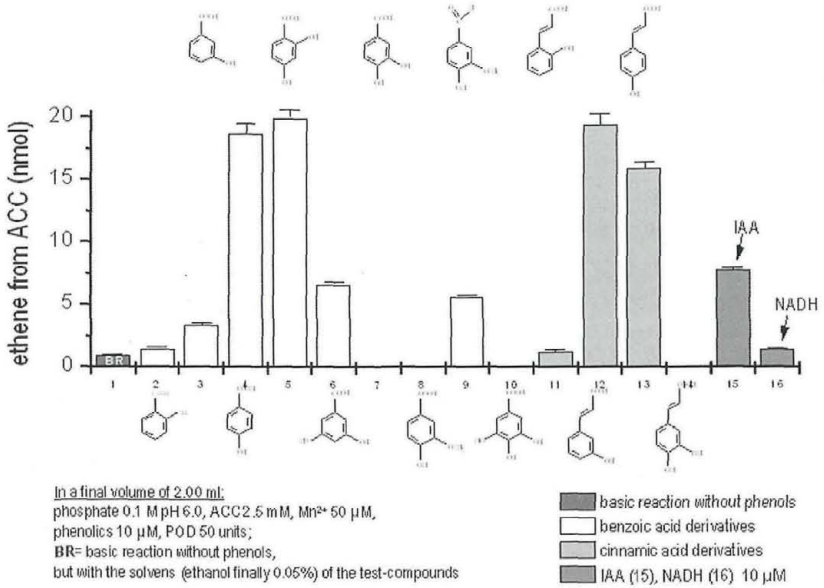

Fig. 6. Influence of diverse hydroxylated or methoxylated phenolics on the formation of ethane from ACC catalyzed by Mn^{2+} and HRP.

In summary, we have investigated the influence of different phenolics present in the apoplast on peroxidase mediated IAA oxidation. Ascorbic acid (AA), ferulic acid (FA) and caffeic acid abruptly switch off peroxidase mediated IAA-oxidation in a very narrow concentration range, even in the presence of stimulatory phenolics, i.e. p-coumaric acid or vanillin. In general IAA oxidase activity is assumed to be important in the control of cellular IAA steady state concentrations influencing growth and developmental processes. In addition to lignification, stiffening of cell walls by peroxidase mediated cross linking of phenolics (i.e. tyrosine in extensin and FA esterified to arabinoglycans) is responsible for cessation of cell growth in parallel to IAA degradation. Furthermore, cell wall reinforcement as a rapid defense response could hinder invading pathogens to come into contact with the plasmalemma membrane. In this context the oxidative burst initiated cell wall cross linking and thus stabilization towards pathogen attack consumes FA after depletion of AA. These sensible changes within micromolar concentrations of stimulatory versus inhibitory phenolics may initiate IAA oxidation and may also contribute to ethylene formation from 1-aminocyclopropane-1-carboxylic acid (ACC) as an Mn^{2+}-dependent (HORST & al. 2005) property of cell wall peroxidases. Oxidase activity of peroxidase towards NAD(P)H and ACC is similarly modulated by the investigated phenols. Thus we hypothesize the existence of an

"apoplastic switch" between plants decision: "growth or denfense" (MATYSSEK & al. 2002).

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