Physiological Role of Reactive Oxygen Species in Chill-Sensitive Plants

By

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S u m m a r y

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Maize (*Zea mays* L.), a very important C4 crop, is a chill-sensitive plant. Its chill resistance is related to various elements of the antioxidative system at the cellular level. The specific anatomy and physiology of the C4 photosynthetic type makes its antioxidative strategy more complex compared to C3 plants. The chill resistance of maize depends on several superoxide dismutase (SOD) isozymes which are unequally distributed among the bundle sheath and mesophyll cells, and also on catalase (CAT) and ascorbate peroxidase (APX) activity. CAT is present as three isoforms in both types of photosynthetic cells, whilst APX is restricted to the bundle sheath. CATs show differentiated susceptibility to thermal inhibition. The role of xanthophyll cycle components, α-tocopherol and polyamines in literature dealing with the chill resistance of maize is also reviewed. Difficulties can be encountered with the construction of chill-resistant maize transformants because signalling processes in the mesophyll and bundle sheath are not properly recognised.

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

In the literature dealing with plant physiology, the term 'chilling' means the exposure of plants to temperatures of 0-15°C. Many crops, e.g. maize, tomato, cucumber, and bean, are thermophilic, and their chill sensitivity is the reason for their late sowing date compared to other cultivated species. The physiological effect of chilling is different from that of frost because there is no ice nucleation in

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plant cells; however, it may be severe enough to lead to yield loss. Chilling stress is often accompanied by oxidative stress - an overproduction of reactive oxygen species (ROS) (WISE & NAYLOR 1987, PASTORI & al. 2000b).

A large number of studies dealing with the chill sensitivity of plants (VIGH & al. 1981, MUSTARDY & al. 1982, STAMP 1987) and chill-dependent oxidative stress (PRASAD & al. 1994a,b, 1995, PRASAD 1997, BURGENER & al. 1998, PASTORI & al. 2000b, DE GARA & al. 2000, BĄCZEK-KWINTA & KOŚCIELNIAK 2003) have been performed on maize. In addition, some attempts to improve chill resistance via manipulating the antioxidative pathways have been performed on this species (VAN BREUSEGEM & al. 1999a,b, PASTORI & al. 2000a). However, maize is just one of ca. 7500 species of C4 plants (SAGE & al. 1999), strong economic pressure is pushing their cultivation towards cool climate zones. The specific anatomy and physiology of C4 plants gives an intriguing insight into chill-associated oxidative stress and defence strategies (HE & EDWARDS 1996, DOULIS & al. 1997, PASTORI & al. 2000a,b).

C4 plants have two types of photosynthetic cells: mesophyll and bundle sheath cells (Kranz anatomy). Atmospheric CO_2 is bound in mesophyll cells by the primary acceptor, phosphoenolpyruvate (PEP). This reaction is catalysed by PEP-carboxylase. Organic acid (in maize - malate) containing 4 carbon atoms is formed and, as a storage of CO_2, transported to the thick-wall cells of the bundle sheath. The released CO_2 is fixed again by the secondary acceptor, ribulosebisphosphate (RuBP), and 3-phosphoglycerate (PGA) is formed. Its reduction to the primary photosynthetic sugar, 3-phosphoglyceride aldehyde, requires back transportation to mesophyll cells because of the lack of NADPH in the bundle sheath, equipped with photosystem I (PS I) only (HATCH & OSMOND 1976, LEEGOOD & EDWARDS 1996). In C4 plants, the efficiency of the dark phase of photosynthesis is improved through reduced photorespiration.

Like the components of the photosynthetic machinery, those of the antioxidant system are distributed - between mesophyll and bundle sheath cells - and require an efficient transport system (PASTORI & al. 2000b, KOPRIVA & al. 2001). This means that any disturbances in the flow of metabolites between these types of tissue may enhance ROS production (KINGSTON-SMITH & FOYER 2000a, PASTORI & al. 2000b). However, these compounds may act signally diminishing susceptibility to chill (PRASAD & al. 1994a,b), and this is dependent on the capacity of the antioxidative system.

The range of chilling injuries depends on the plant organ, the duration of stress, and whether the stress affects the plant at night or during illumination. The range of chilling temperatures, different in the various experiments (5-7 *vs.* 14-15°C), makes it difficult to compare obtained results.

Generation and Toxicity of ROS during Chilling

According to LYONS 1973, the primary reason for chilling injury is the membrane lipid phase transition from liquid-crystalline state to a more viscous

structure, which leads to enhanced permeability of mitochondrial and chloroplast envelopes (LYONS & al. 1964, DE SANTIS & al. 1999, MURATA & YAMAYA 1984). The disturbance in membrane integrity results in the enhancement of reactions generating free radical compounds (WISE & NAYLOR 1987) and lipid peroxidation, especially in chill-sensitive genotypes (DE SANTIS & al. 1999). Other sources of chill-dependent oxidative stress are mainly considered to be chloroplasts and mitochondrial electron transport chains. Photochemical energy in chloroplasts is often excessive due to the suppression of the enzyme-dependent dark phase of photosynthesis. In C4 plants, the photosynthetic performance of both the mesophyll and bundle sheath is constrained during chilling (STAMP 1987, LONG 1983, LEEGOOD & EDWARDS 1996). In this case, superoxide anion O_2^- is formed, mainly within PS I in a Mehler reaction (MEHLER 1951). O_2^- may react with numerous biomolecules, causing them to be disrupted. H_2O_2 is formed via disproportionation (dismutation) of O_2^-, which is both spontaneous and catalysed by superoxide dismutase (SOD) (SCANDALIOS 1993). Hydrogen peroxide may be decomposed by catalases (CATs) to H_2O and O_2, or to H_2O by peroxidases (PRASAD & al. 1994a,b). However, in the presence of transition metals (and in the presence of FeS complexes present in biochemical systems), H_2O_2 reacts with O_2^-, generating a powerful oxidiser, hydroxyl radical ·OH, which may destroy many cellular constituents, leading to various metabolic dysfunctions (ELSTNER 1982, JAKOB & HEBER 1996, SONOIKE 1996, TERASHIMA & al. 1998). PS II is the source of singlet oxygen $^1O_2$, formed within the photosynthetic antenna when the excitation energy of chlorophyll (^{3}Chl) is transferred to triplet oxygen in a ground state $^3O_2$ (ASADA 1994a,b). Singlet oxygen may be also formed in the PS II core (for a review, see NIYOGI 1999). $^1O_2$ induces damage to D1 protein, and this results in a limitation in the light phase of photosynthesis, called photoinhibition (SOMERSALO & KRAUSE 1989). D1 degradation is reversible, but under strong stress, e.g. during the interaction of chill and excessive light, the process of resynthesis is restricted, resulting in irreversible limitation of photosynthetic efficiency (GONG & NILSON 1989, FEIERABEND & al. 1992). In mitochondria, O_2^- is formed as a by-product of the respiratory chain (for a review, see MØLLER 2001).

It has been emphasised that oxidative stress always accompanies the primary effects of chilling stress, and the resistance of some genotypes of chill-sensitive plants is linked to the ability of tissues to activate various elements of the antioxidative system (JAHNKE & al. 1991, MASSACCI & al. 1995, HODGES & al. 1997a, SKRUDLIK & al. 2000, BĄCZEK-KWINTA & KOŚCIELNIAK 2003).

Distribution and Chill Response of the Antioxidative System in Maize Leaves

Antioxidative enzymes are characterised by differentiated sensitivity to chilling temperature. Usually, SOD is primarily taken into consideration as a scavenger of O_2^- generated rapidly in photosynthetic and non-photosynthetic tissues. The SOD (EC 1.15.1.1.) family in plants consists of the FeSOD, MnSOD and

CuZnSOD classes. The presence of plant SOD isozymes and their genes was first demonstrated in experiments performed on maize (BAUM & SCANDALIOS 1979, 1982). SOD protein is relatively resistant both to low and high temperatures (JAHNKE & al. 1991, BURKE & OLIVIER 1992, MISZALSKI & al. 1998). There are 10 genetically and biochemically different forms of SOD in maize (SCANDALIOS 1993, VAN BREUSEGEM & al. 1999b, PASTORI & al. 2000b). The abundance of SODs may complicate the analysis of experimental data due to the different responses of particular enzymes to environmental factors. For example, studies by HODGES & al. 1997a,b performed on several maize genotypes (inbreds and hybrids) did not show any relationship between total leaf SOD activity and resistance to chilling stress. Intriguingly, the mesophyll of maize is equipped only with FeSOD, present in chloroplasts, and the other isoforms are located in the bundle sheath (DOULIS & al. 1997, PASTORI & al. 2000b). PASTORI & al. 2000b demonstrated that in the total SOD pool, all Cu/Zn isoforms and FeSOD activities were enhanced by chill (15°C), whereas MnSOD was inactivated. However, in this study, enzyme activity was related to chlorophyll content, therefore cannot be directly compared with the literature data referring to the protein content. Additionally, 14-15°C in many experiments was considered not as chilling temperature per se, but rather as acclimatory/hardening treatment prior to stress (4-5°C, ANDERSON & al. 1995, LEIPNER & al. 1997).

The next links in the antioxidative system chain are CATs and peroxidases. CAT (EC 1.11.1.6) in maize leaves exists as three isoforms: in peroxysomes and glyoxysomes (CAT-1), cytosol (CAT-2) and mitochondria (CAT-3) (PRASAD 1994a,b, SCANDALIOS & al. 1997). CAT enzymes are equally distributed between the mesophyll and bundle sheath (DOULIS & al. 1997). However, as a haemoprotein containing ferroporphyrin, CAT is prone to photoinactivation (FEIERABEND & al. 1992, and references therein, LEE & LEE 2000). Recovery processes associated with protein turnover are hindered by chill, due to the stimulative effect of low temperature on proteases and inhibitory on post-translative modification (FEIERABEND & al. 1992, WISE 1995, STREB & FEIERABEND 1995). The mitochondrial CAT-3 isoform seems to be the most susceptible to thermal inhibition (AUH & SCANDALIOS 1997), and it is noteworthy that the respiratory chain is the source of O_2^{-}, and consequently, of H_2O_2. Rapid and long-term accumulation of H_2O_2 enhances low-temperature sensitivity (PRASAD & al. 1994a,b). The efficiency of the prevention of CAT proteolysis and activation of the enzyme together with its synthesis de novo is higher in chill-resistant than in chill-sensitive plants (SARUYAMA & TANIDA 1995, AUH & SCANDALIOS 1997). A function similar to that of mitochondrial catalase related to H_2O_2 scavenging during severe chilling may be performed by cytochrome c peroxidase (EC 1.1.1.5) (PRASAD & al. 1995). Another peroxidase, ascorbate peroxidase (APX, EC 1.11.1.11), is the key enzyme in removing H_2O_2 from chloroplasts (ASADA 1992). DE GARA & al. 2000 reported that the seedlings of maize inbred with elevated APX activity possessed high vigour. HULL & al. 1997 showed that APX in the leaves of chill-tolerant Zea diploperennis had ca. 3-fold higher kinetic power (V_{max}/K_m) than APX in Zea mays leaves. It is noteworthy that in maize chloroplasts, APX probably exists as two isoforms: stro-

mal and membrane-bound (CHEN & ASADA 1989, DOULIS & al. 1997). However, their presence is restricted to the bundle sheath (DOULIS & al. 1997); ascorbate (AsA), an electron donor of APX, may be regenerated in the mesophyll in the ascorbate-glutathione cycle, and then must be transported to the bundle sheath. The efficiency of transport processes may be reduced in chilling conditions (PASTORI & al. 2000b) leading to inactivation of the enzyme (CHEN & ASADA 1989). The consequence of this is an overproduction of H_2O_2 in the chloroplasts of the bundle sheath (PASTORI & al. 2000b) causing oxidative damage to its proteins (KINGSTON-SMITH & FOYER 2000a).

Among non-enzymatic antioxidants, tripeptide glutathione (GSH) should be considered first. GSH plays a number of roles in plant physiology (for a review, see KOCSY & al. 2001, TAUSZ & al. 2004). Its antioxidative role involves scavenging oxygen free radicals and singlet oxygen (HALLIWELL & GUTTERIDGE 2000), and it plays a part in the antioxidative systems detoxifying H_2O_2. GSH is also a cofactor of glutathione peroxidase (GPX, EC 1.11.1.9), the enzyme that decomposes H_2O_2 (ESHDAT & al. 1997), and in the ascorbate-glutathione cycle GSH serves as an electron donor for the regeneration of ascorbate from its oxidised form, dehydroascorbate (NOCTOR & FOYER 1998). As a consequence, the oxidised form of glutathione, glutathione disulphide (GSSG), is generated. GSH is then reconstituted by NADPH-dependent glutathione reductase (GR, EC 1.6.4.2). The enzymes involved in glutathione recycling are considered the key ones in the chill resistance of maize (VAN BREUSEGEM & al. 1998, KOCSY & al. 2000, and references therein). Another protective role of GSH in plant metabolism that has been proposed is the protection of sulhydryl (thiol -SH) groups of proteins from oxidation (FOYER & HALLIWELL 1976, KRANNER & GRILL 1996). In maize leaves, both GSH synthesis and regeneration are dependent on transport processes between the mesophyll and bundle sheath. Cysteine, one of the three amino acids forming this tripeptide, is synthesised in the bundle sheath, whereas GSH resynthesis and its regeneration from GSSG by glutathione reductase occurs in mesophyll cells (BURGENER & al. 1998, KOPRIVA & al. 2001). The absence of GR in the cells of the bundle sheath is related to post-transcriptional regulation. This is considered to be one of the factors involved in maize chill sensitivity, triggered in a transport-dependent mechanism similar to that described for APX (Table 1, PASTORI & al. 2000b).

If enzymatic antioxidants fail, a mechanism for dissipating excessive light energy is required. Carotenoid pigments seem to be good candidates for mode of action study in chill-resistant genotypes of chill-sensitive plants. The xanthophyll cycle increases during long-term chilling in maize (C4) and in various species of tomato (C3) of differentiated sensitivity (HALDIMANN 1996, VENEMA & al. 1999), so this response seems unrelated to the type of photosynthesis. This mechanism for photochemical energy dissipation is located mainly in the inner chloroplast membrane, forming thylacoids. However, the chloroplasts of bundle sheath are capable for cyclic electron transport (KUBICKI & al. 1996). As this process creates electrochemical gradient sufficient for ATP synthesis, thus, NPQ-related dissipation of excess light energy should also be considered there. A similar question may be raised regarding the level of alpha-tocopherol, a membrane-bound protectant of

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lipid membranes. Interestingly, its level may be correlated with chilling tolerance, as was shown in model maize genotypes (LEIPNER & al. 1999). Also, polyamines may be considered chill-protectants (SZALAI & al. 1997) due to their membrane-protective and antioxidative properties (DROLET & al. 1986, ROBERTS & al. 1986). Another point which has not been established is the location of polyamines in the mesophyll and bundle sheath.

Table 1. Characteristics of two types of photosynthetically active cells of maize and distribution of antioxidants. The sources: DOULIS & al. 1997, BURGENER & al. 1998, KOPRIVA & al. 2001, PASTORI & al. 2000b.

Characteristics	Mesophyll	Bundle sheath
$NADPH+H^+$	regenerated from NADP+	taken from the mesophyll
Photosystem	PS II and PS I	PS I
chloroplasts	granal	non-granal
PEP-carboxylase	+	-
RuBisCo and RuBP	traces	+
Photosynthates synthesised	sucrose	starch
SOD	1 chloroplastic isoenzyme (Fe-SOD)	9 isoenzymes
APX	-	+
AsA	regenerated from monodehydroascorbate	taken from the mesophyll
DHAR	+	-
MDAR	+	+
Cysteine	taken from the bundle sheath	synthesised
GSH	regenerated from GSSG	taken from the mesophyll
GR	+	-, but transcripts present
CAT	+	+
polyamines	?	?
xanthophyll cycle	?	?

Can ROS Protect Plants from Chill?

ROS formation with chilling stress is potentially harmful; however, it is also considered as (1) a dissipative mechanism of excitation energy in chloroplasts and (2) a signalling mechanism in various organelles and tissues. The mode of action of ROS depends on their concentration in relation to the pool of scavengers.

The dissipative mechanism is linked with PS I, where O_2 is reduced, giving a superoxide radical. O_2^- may be scavenged in a cycle named the water-water cycle, or the Mehler-ascorbate peroxidase reaction (ASADA 1999, NIYOGI 1999). Another possibility is pseudocyclic electron transport. In maize, the participation of enzymes involved in the water-water cycle (elevated SOD and APX activity) was demonstrated by MASSACCI & al. 1995, but in mild chilling treatment (16/14°C, day/night), not in severe chill conditions. The Mehler reaction itself, due to proton consumption, may partly enhance ΔpH between the lumen and stroma, resulting in increased ATP formation. However, the role of this process in chill-sensitive C4

plants has been questioned (LAISK & EDWARDS 1998, and references therein); a pH gradient is also necessary for the non-photochemical quenching (NPQ) of excessive absorbed light energy (SCHREIBER & NEUBAUER 1990). NPQ is important in maize leaves as it protects PS II from photoinhibition and may also reduce the risk of oxidative damage caused by $^1O_2$ (HARBINSON & FOYER 1991).

The signalling role of hydrogen peroxide in chilling stress per se (in dark treatment) was shown by PRASAD & al. 1994a,b. The pre-treatment of roots with 0.1 mM H_2O_2 at 14°C had an inductive effect on mitochondrial catalase transcript levels in mesocotyl and caused increased CAT and guaiacol peroxidase activity during subsequent chilling at 4°C. Non-acclimated seedlings accumulated an excess of H_2O_2 in mitochondria, which was too high to be scavenged, resulting in oxidative damage to proteins (PRASAD 1997).

H_2O_2 is the universal signalling molecule involved in a wide range of plant responses to various stress factors (FOYER & al. 1997, NEILL & al. 2002). As a non-charged molecule it is able to cross biological membranes. Its relatively long life span (half-life ca. 1 ms) allows it to diffuse to various distances from the sites of origin (VRANOVÁ & al. 2002). DESIKAN & al. 2001 showed a large number of genes up-regulated by H_2O_2, and among them antioxidants. These experiments were performed on *Arabidopsis* culture cells, and it would be worth clarifying this mechanism in maize. Interestingly, *aox-1*, one of the genes encoding mitochondrial protein, alternative oxidase, is also activated in this way (WAGNER & KRAAB 1995). Such activation may reduce $O_2^{.-}$ production in maize mitochondria in the cyanide-resistant respiratory pathway (VAN DE VENTER 1985, STEWART & al. 1990).

H_2O_2 also affects the functioning of stomata. Stomatal closure, preventing the plants from experiencing excessive water loss via transpiration, is an important physiological response to stress. The mechanism of stomatal movement in maize during chilling has been studied for many years (MCWILLIAM & al. 1982, JANOWIAK & DÖRFFLING 1996). It has been established that stomata closure is often disturbed in chill-sensitive plants, and this accelerates leaf desiccation (MCWILLIAM & al. 1982). The fact that H_2O_2 promotes stomatal opening in both ABA-dependent and -independent mechanisms (WAGNER & KRAAB 1995, ALLAN & FLUHR 1997, LEE & al. 1999, PEI & al. 2000, ZHANG & al. 2001) may be significant in such studies.

Problems with the Construction of Chill-Resistant Transformants

The antioxidative system consists of numerous elements, so the first obvious conclusion is that one of the ROS scavengers, depending on its pool, may affect the functioning of others, in some cases paradoxically enhancing oxidative stress (BRÜGGEMANN & al. 1999, CREISSEN & al. 1999). The other problem is that the ROS pool, and its ratio to nitric oxide, may be active in terms of signalling (NEILL & al. 2002, VRANOVÁ & al. 2002). Taken together, the disruption of the

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redox balance in the cell, or in the particular cellular compartment, may give unpredictable results. Genes encoding various antioxidative proteins are subject to redox control (PFANNSCHMIDT & al. 2001, PFANNSCHMIDT 2003). As described earlier, NADPH and GSH pools vary as regards mesophyll and bundle sheath cells, and this may serve as such a signal (PASTORI & al. 2000a, WINGATE & al. 1988, KARPINSKI & al. 1997, KOCSY & al. 2001). It was demonstrated that not only native, but also targeted, genes are subject to such regulation. Transformation of maize with MnSOD enhanced total foliar SOD activity (VAN BREUSEGEM & al. 1999b, KINGSTON-SMITH & FOYER 2000b); however, this was still restricted to bundle sheath cells (KINGSTON-SMITH & FOYER 2000b). On the other hand, GR activity in transformants occurred in the mesophyll only (PASTORI & al. 2000a).

The issue of how to overcome chill sensitivity via manipulation of antioxidants has not yet been resolved. The C4 photosynthetic type causes additional problems, and transformation of C3 chill-sensitive plants such as tomato (BRÜGGEMANN & al. 1999) and cotton (KORNYEYEV & al. 2003, LOGAN & al. 2003) to better ROS tolerance did not improve thermotolerance. Interestingly, expression of animal antiapoptotic genes delayed the development of chilling injury in tomato seedlings, and increased the level of anthocyanin (XU & al. 2004). Is this the appropriate strategy for new maize transgenes? Conventional breeding programmes focusing on high yield and pest resistance dominate and are effective. Irrespective of this, basic studies on maize chill sensitivity, including gene manipulation, are still of interest.

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