

## Lipid peroxidation and defense related enzymes in maize infected by *Rhizoctonia solani*

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Host plants are endowed with several potential defense mechanisms, while pathogens produce an array of weapons to evade or suppress these defense mechanisms. Montalbini (8) found that when the pathogens invade host tissues, different oxygen radicals are produced which induce synthesis of various defense chemicals. Besides this, a pathogen may suppress the generation of active oxygen species. Pathogen may also activate generation of scavengers of oxygen species. Superoxide dismutases (SOD) scavenge the superoxide anion (9). Catalase and peroxidases remove H<sub>2</sub>O<sub>2</sub> very efficiently (11). Low levels of O<sub>2</sub> and OH radicals are normally maintained by the action of SOD and catalase (4). Catalase activity increases in susceptible interactions to scavenge hydrogen peroxide (8). The present study was undertaken to assess defense related enzymes and lipid peroxidation in maize and *R.solani* interaction.

The level of lipid peroxidation in diseased and healthy leaf sheath tissue was measured in terms of malondialdehyde (MDA), a product of lipid peroxidation, by the thiobarbituric acid (TBA) reaction. The concentration of MDA was calculated using its extinction coefficient of 155 nm<sup>-1</sup> cm<sup>-1</sup> (6).

Catalase was assayed by measuring the initial rate of disappearance of hydrogen peroxide by the method of Chance and Maehky (1). The decrease in hydrogen peroxide conc. was followed as a decline in A<sub>240</sub> using UV-visible spectrophotometer. The activity was expressed in units, where one unit of catalase represents a decrease in absorbance of one minute. The activity of

superoxidedismutase (SOD) was assayed by measuring its ability to inhibit the photochemical reduction of nitroblue tetrazolium (NBT) using the method of Dhindsa *et al.*, (3). The absorbency by the reaction mixture at 560 nm was read. A irradiated reaction mixture, which did not develop colour, served as control. The reaction mixture-lacking enzyme developed the maximum colour and this decreased with increasing volume of enzyme extract. One unit of SOD activity was equal to an increase in difference in optical density by one in one hour.

Malondialdehyde (MDA), a decomposition product of lipid peroxidation, especially of polyunsaturated fatty acids (PUFA) present in host cell membranes, is often found to increase during pathogenesis. In the present investigation, a varietal difference in accumulation of MDA in host cells, in response to pathogen attack, has been observed. The genotype CHH 62, which has been found to be resistant to pathogenesis, showed non-significant accumulation of MDA whereas the other two genotypes (JK 2595 and FH 3088), that are relatively susceptible to banded leaf and sheath blight pathogen, accumulated higher amounts of MDA (Table 1). Gonner and Schlosser (5) also reported that when oat leaves were inoculated with a virulent pathogen, *Drechslera avenae*, the MDA content in leaves significantly increased. Moreover, this increase in MDA coincided with complete bleaching of chlorophyll as reported earlier also by Kauss (7) and Rosen and Halpern (10). They further suggested that the oxygen species have a role in symptoms development

**Table 1.** Malondialdehyde levels and activities of superoxidedismutase and catalase enzymes in maize genotypes after inoculation with *R.solani*

Varieties		MDA ( $\mu$ mole/ 100 mg f. wt)	SOD (SOD/h/ 100mg f. wt)	CAT CAT/min/ 100 mg f. wt)
CHH-62	I	0.376	48.300	0.006
	UI	0.343	15.70	0.021
JK-2595	I	0.499	21.80	0.030
	UI	0.290	17.90	0.026
FH-3088	I	0.695	36.60	0.050
	UI	0.501	14.10	0.026
SEm $\pm$		0.050	01.74	0.005
CD (0.05)		0.140	03.80	0.009

I= Inoculated, UI= Un inoculated

rather than in any defense mechanism. Therefore, higher MDA content in host cells are indicative of their susceptibility to pathogenesis. In the present investigation, superoxidedismutase (SOD) activity increased significantly in the maize cultivars infected by banded leaf and sheath blight pathogen (Table. 1). Though genotypic differences in SOD activity are non-significant, it may be subject to expression of different forms (isozymes) of SOD during infection (11). Gonner and Schlosser (11) also detected a substantial increase in SOD activity after 40 hrs of inoculation with the highly virulent pathogen *Drechslera avenae* in oat leaves. On the contrary, no such increase in SOD activity was detected in oat leaves, inoculated with weakly virulent *D. nobleae*. In the present study, the noted increase in SOD activity, as time of infection progresses, may be due to response given by the host cells to scavenge excessive formation of superoxide free radicals as induced by the pathogen (9).

Catalase activity has been shown to increase during infection as a mechanism to scavenge fungitoxic  $H_2O_2$  (8). In the present investigation, the genotype CHH 62 showed a significant inhibition of catalase activity due to resistance with to disease. Also, at morphological level, this genotype manifests mild disease symptoms against banded leaf and sheath blight pathogen. Conrath *et al.* (2) reported that plant defense-promoting compounds are generated during pathogenesis, in resistant plant cells, that inhibit catalase activity, as a hypersensitive defense response.

## REFERENCES

1. **Chance, B. and Machly, A.C.** (1995). *Methods Enzymol.* **2**: 264-775.
2. **Conrath, U., Chen, Z., Ricigliano, J.R. and Klessig, D.** (1995). *Proc. Natl. Acad. Sci, USA*, **92**: 7143-7147.
3. **Dhindsa, R.S., Plumb Dhindsa, P.L. and Thorpe, T.A.** (1981). *J. Exptl. Bot.* **32**: 93-101.
4. **Galliard, T. and Chan, H.W.S.** (1980). In : *The Biochemistry of Plants*, Vol. 4 (Eds Stumpf, P.K. and Conn, E.E.). pp. 131-161, Academic Press. New York.
5. **Gonner, V.M. and Schlosser, E.** (1993). *Physiol. Mol. Plant Pathol.* **42**: 221-234.
6. **Heath, R.L. and Packer L.** (1968). *Arch Biochem. Biophys.* **125**: 189-198.
7. **Kauss, H.** (1990). In: *The Plant Plasma Membrane* (Eds., Larsson, E. and Moller, I.M.), pp. 320-351. Springer-Verlag, Berlin,
8. **Montalbini, P.** (1991). *Physiol. Mol. Plant Pathol.* **39**: 173-188.
9. **Moreau, R.A. and Osman, S.F.** (1989). *Physiol. Plant Pathol.* **35**: 1-10.
10. **Rosen, G.M. and Halpern, H.J.** (1990). *Methods Enzymol.* **186**: 611-621
11. **Scandalios, J.G.** (1993). *Plant Physiol.* **101**: 7-12.

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