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# LIPID PEROXIDATION, AUDPC, YIELD, AND TEST WEIGHT AND THEIR CORRELATION DURING *BIPOLARIS SOROKINIANA* INFECTION IN WHEAT

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**ABSTRACT**
The 32 resistant and 2 susceptible (check) (table 1) wheat genotypes were evaluated for the estimation of Lipid peroxidation (MDA), AUDPC, yields and test weight. The resistant genotype have the least MDA (0.106 nmoles/ g fresh weight) content than the susceptible genotype having highest MDA (0.415 nmoles/g fresh weight) content and also found positively correlated with the AUDPC (r= 0.772) and negatively correlated with the test weight (r= -0.438) and the yield (r= -0.317). MDA was increase in the both resistant and susceptible genotype over the time period but due to lack of some antioxidant enzymes it shows the more damage to plant and estimated in terms of MDA content and found more at the 48 hours (0.208 nmoles/g fresh weights) time interval after the inoculation. Present study concluded the susceptible genotype shows the more damage after the inoculation of the *Bipolaris sorokiniana* then the resistant genotypes of wheat due to lake of antioxidant enzymes which reduces the harmful effects of ROS.

## INTRODUCTION

Wheat (*Triticum spp.)* is a cereal crop. The ûrst cultivation of wheat occurred about 10000 years ago, as a part of the 'Neolithic Revolution'. The earliest cultivated forms were diploid (genome AA) (Einkorn) and tetraploid (genome AABB) (Emmer) wheat and their genetic relationships indicate that they originated from the south-eastern part of Turkey (Dubcovsky and Dvorak, 2007). *Triticum aestivum* (Maxican dwarf wheat) presently grown in everywhere in country and also called common bread wheat. It is grown throughout the world, in a wide variety of climates between 30º and 60º N and 40º S latitudes (Nutteson, 1955). It has good nutrition profile with 12.1 percent protein, 1.8 % lipids, 1.8 % ash, 2.0 % reducing sugars, 6.7 % pentosans, 59.2 % starch, 70 % total carbohydrates and provides 314K cal/100 g of food (USDA, 2012). *Bipolaris sorokiniana* (Sacc.) Shoem. *(Helmnthosporium sativum,* telomorph *Cochliobolus sativum*), the causal agent of spot blotch disease of wheat is a major biotic stress in the warm humid tropic. The fungus *C. sativus* is the telomorph (sexual stage) which is causal agent of a wide variety of cereal diseases. *Bipolaris sorokiniana* usually induce visible necrotic symptoms on the leaf, leaf sheath and stem (Chand and Joshi, 2004). It produces oblong, necrotic, dark lesions (up to 20 mm long) which are scattered throughout the leaves. These lesions increased in size and finally coalesce to form a large spot that covers and kills large portions of the leaf, with severely infected leaves senescencing prematurely (Steffenson, 1997). *Bipolaris sorokiniana* a dematious fungi belong to the Dueteromycota group. In nature, it reproduces asexually by means of conidia. Conidia are attached to the geniculate conidiophores. Conidia have thick walled with 8-10 transverse septa. *Bipolaris sorokiniana* germinate completely in four hours, and then forms appressoria at the juncture of epidermal cell wall after eight hours and hyphae from initially infected cells enter adjacent cells in 24 hours, which results in the granularisation of the host cytoplasm (Bisen and Channy, 1983). The germ tube then swells to produce appresorium from which infection hyphae are developed. The infection hyphae enter the host tissue either through stomata or by rupturing the epidermis. (Acharya *et al*. 2011). A common consequence of most biotic stresses is an increase production of reactive oxygen species (ROS) (Polle and Rennenberg, 1993). ROS such as superoxide radical ($O_2^-$), hydrogen peroxide ($H_2O_2$) and hydroxyl radical ($OH^-$) are toxic byproducts of processes such as photosynthetic or respiratory electron transport. The malondialdehyde (MDA) or thiobarbituric acid-reactive-substances (TBARS) assay has been used extensively since the 1950s to estimate peroxidation of lipids in membrane and biological systems (Heath and Packer, 1968). Malondialdehyde is formed through auto oxidation and enzymatic degradation of polyunsaturated fatty acids in cells. However, in addition to cellular carbohydrates, phenyl propanoid type pigments (e.g. flavonoids like anthocyanins) present in plant tissue (Harborne, 1984) resulting in overestimation of the MDA content. This procedure has not been carried out in plant studies in the past where lipid peroxidation has been assessed by the TBARS method (Landry *et al*., 1995;

Predieri *et al*., 1995), potentially resulting in initiation of reported MDA levels. MDA concentration was a widely used method to analyse lipid peroxidation in biological material (Taulavuori *et al*., 2001). Bansal and Srivastava(2012) reported lipid peroxidation leading to oxidative damage during water logging. This paper aims to find out the biochemical damage of plant in terms of MDA and to screen the resistant line on the basis of biochemical and the morphological damage like the AUDPC which ultimate decreases the yields of the plants.

## MATERIALS AND METHODS

The experiments were conducted during the Rabi season at the Agricultural Research Farm, Banaras Hindu University, Varanasi. The experimental field was well drained with uniform topography and assured source of water supply.

**Preparation of spore suspension of the inoculums**

Spore suspension was prepared on the day of inoculation in the field. Inoculums produced on sorghum grains were mixed in sterile water in a bucket. Then it was stirred so that all spores on sorghum grains should dislodge in water. It was then filtered with plastic net. The concentration of spore suspension was maintained to $10^4$ spores $ml^{-1}$ with the help of spore count/100$\mu$l. Then 0.05% Tween-80 was added to increase the stickiness of the spore suspension.

**Inoculation to the field crop**

Inoculation of pathogen (*Bipolaris sorokiniana*) was done at 50% flowering stage (GS 65). Inoculation was done in the evening hours at 5:00-6:00 pm in order to provide sufficient moisture for longer period for spore germination and pathogenesis. Inoculums were spread with help of knapsack sprayer.

**Experimental Materials**

The experiment consists of total 34-lines (Table 1) including two checks and it was sown in 3-replications in randomized block design (RBD). These lines were obtained from CRP project (CGIAR) where purity of each line was maintained by SSD method. The samples for Lipid peroxidation analysis were collected in aluminium foil after that samples were dipped in liquid nitrogen (-196$^0$C) and transported the container to laboratory and preserve at -80$^0$C.

**Assessment of Lipid peroxidation**

For the analysis of lipid peroxidation (MDA) in leaf samples of wheat collected at 0 hrs, 24 hrs, and 48 hrs after the inoculation of the pathogen. The level of lipid peroxidation was estimated as the MDA content, determined according to the method of Heath and Packer, 1968.

**Table 1: Pedigree of genotypes used for SOD and lipid peroxidation**

| Genotypes | Pedigree |
|---|---|
| 1 | ATTILA/3*BCN//BAV92/3/TILHI |
| 2 | TILHI/PALMERIN F2004 |
| 3 | CROC_1/AE.SQUARROSA (205)//KAUZ/3/ENEIDA |
| 4 | TILHI/SOKOLL |
| 5 | PFAU/MILAN//TROST/3/PBW65/2*SERI.1B |
| 6 | CNDO/R143//ENTE/MEXI_2/3/AEGILOPS SQUARROSA (TAUS)/ 4/ WEAVER/ 5/ PASTOR |
| 7 | YAV_3/SCO//JO69/CRA/3/YAV79/4/AE.SQUARROSA (498)/5/2*OPATA |
| 8 | CHIRYA.3 |
| 9 | JUPARE C 2001 |
| 10 | ALTAR 84/AE.SQUARROSA (219)//OPATA/3/WBLL1/FRET2//PASTOR |
| 11 | W462//VEE/KOEL/3/PEG//MRL/BUC |
| 12 | VORB/4/D67.2/PARANA 66.270//AE.SQUARROSA (320)/3/CUNNINGHAM |
| 13 | HD 2967 |
| 14 | BECARD |
| 15 | ATTILA/3*BCN//BAV92/3/TILHI/4/SHA7/VEE#5//ARIV92 |
| 16 | CNDO/R143//ENTE/MEXI_2/3/AEGILOPS SQUARROSA (TAUS)/4/WEAVER/5/2*KAUZ |
| 17 | ASTREB/OAX93.10.1//SOKOLL |
| 18 | ALTAR 84/AEGILOPS SQUARROSA (TAUS)//OPATA |
| 19 | CMH79A.955/4/AGA/3/4*SN64/CNO67//INIA66/5/NAC/6/RIALTO |
| 20 | SW89-5124*2/FASAN |
| 21 | BCN/RIALTO |
| 22 | CROC_1/AE.SQUARROSA (205)//KAUZ/3/SASIA/4/TROST |
| 23 | MILAN/KAUZ/3/URES/JUN//KAUZ/4/CROC_1/AE.SQUARROSA(224)//OPATA |
| 24 | NL 750 |
| 25 | PBW343*2/KUKUNA//PBW343*2/TUKURU/3/PBW343 |
| 26 | NL748/NL837 |
| 27 | TILHI |
| 28 | UP2338*2/4/SNI/TRAP#1/3/KAUZ*2/TRAP//KAUZ/5/MILAN/KAUZ//CHIL/CHUM18/6/UP2338*2/4/SNI/TRAP#1/3/KAUZ*2/TRAP//KAUZ |
| 29 | ASTREB/OAX93.10.1//SOKOLL |
| 30 | GAN/AE.SQUARROSA (897)//OPATA/3/BERKUT |
| 31 | SURUTU-CIAT |
| 32 | WESTONIA/4/KRICHAUFF/FINSI/3/URES/PRL//BAV92 |
| 33 | SONALIKA |
| 34 | CIANO T-79 |

**Disease severity**

Disease severity of spot blotch for each genotype was evaluated on five randomly tagged plants in the field at three different growth stages (GS) viz., GS 65 (half anthesis to half complete), GS 69 (anthesis complete) and GS 77 (late milking) (Zadoks *et al*., 1974) following double digit scale (DD, 00-99) (Saari and Prescott, 1975).

% Disease severity = (D1/9) × (D2/9) ×100

**Area Under Disease Progress Curve (AUDPC)**

The Disease Severity was recorded at minimum three time points at 7 to 10 days intervals, using the double-digit scale (00–99) of Saari and Prescott (1975). The AUDPC are calculated using the per cent disease severity estimations corresponding to the disease ratings, as outlined by Roelfs *et al*., (1992).

$$AUDPC = \sum_{i=1}^{n-1} [(\chi_i + \chi_i + 1)/2](t_i + 1 - t_i)$$

Where,

xi = disease level at time ti, (ti + 1)-ti = time (days) between two disease scores, and n = number of dates at which spot blotch will be recorded.

**Test Weight of grains**

The test weight was estimated from 20 tillers harvested separately from the experimental plots and grains were threshed and packed in envelopes and taken to laboratory. Counted 1000 seeds, from each genotype including resistance

**Table 2: Correlation among the SOD, MDA, Test weight, Yield and AUDPC**

| | MDA | Test weight | Yields |
|---|---|---|---|
| MDA | 1 | | |
| Test weight | -0.4381(0.0096) | 1 | |
| Yields | -0.3175(0.0672) | 0.6905(<0.0001) | 1 |
| AUDPC | 0.7723(<.0001) | -0.2547(0.1460) | -0.1272(0.4734) |

Data given in parenthesis is probability

**Table 3: Mean of AUDPC, SOD, MDA, Test weight and Yields in the year 2013**

| Genotypes | AUDPC | MDA nmoles/g fresh weight | Test weight (g) | Yield of 20 tillers (g) |
|---|---|---|---|---|
| $1^R$ | 442.90 | 0.1069 | 34.00 | 40.67 |
| $2^R$ | 572.53 | 0.1246 | 36.00 | 35.00 |
| $3^R$ | 464.51 | 0.1272 | 35.00 | 40.00 |
| $4^R$ | 353.40 | 0.1489 | 34.67 | 47.00 |
| $5^R$ | 367.28 | 0.1431 | 34.67 | 38.33 |
| $6^R$ | 496.91 | 0.1191 | 35.00 | 41.33 |
| $7^R$ | 254.63 | 0.1413 | 34.00 | 36.33 |
| $8^R$ | 334.88 | 0.1321 | 35.00 | 42.33 |
| $9^R$ | 442.90 | 0.1587 | 33.67 | 27.33 |
| $10^R$ | 388.89 | 0.1434 | 28.67 | 27.67 |
| $11^R$ | 378.09 | 0.1199 | 33.33 | 34.67 |
| $12^R$ | 421.30 | 0.1207 | 33.00 | 32.67 |
| $13^R$ | 410.49 | 0.1152 | 30.67 | 42.67 |
| $14^R$ | 507.72 | 0.1499 | 35.33 | 42.33 |
| $15^R$ | 550.93 | 0.1747 | 35.67 | 40.67 |
| $16^R$ | 291.67 | 0.1377 | 36.33 | 36.00 |
| $17^R$ | 486.11 | 0.1360 | 30.67 | 30.33 |
| $18^R$ | 464.51 | 0.1147 | 28.00 | 34.00 |
| $19^R$ | 486.11 | 0.1539 | 32.33 | 37.00 |
| $20^R$ | 307.10 | 0.1554 | 30.67 | 32.67 |
| $21^R$ | 529.32 | 0.1472 | 33.67 | 38.67 |
| $22^R$ | 367.28 | 0.1543 | 31.33 | 38.67 |
| $23^R$ | 453.70 | 0.1405 | 27.33 | 25.33 |
| $24^R$ | 648.15 | 0.1432 | 31.00 | 36.00 |
| $25^R$ | 388.89 | 0.1298 | 32.33 | 34.00 |
| $26^R$ | 432.10 | 0.1558 | 30.67 | 30.67 |
| $27^R$ | 410.49 | 0.1543 | 32.67 | 36.33 |
| $28^R$ | 518.52 | 0.1459 | 30.33 | 31.33 |
| $29^R$ | 421.30 | 0.1579 | 29.00 | 34.33 |
| $30^R$ | 226.85 | 0.1461 | 32.33 | 31.33 |
| $31^R$ | 496.91 | 0.1605 | 32.67 | 35.00 |
| $32^R$ | 547.33 | 0.1628 | 37.33 | 41.67 |
| $33^S$ | 1254.63 | 0.4159 | 29.67 | 33.33 |
| $34^S$ | 712.96 | 0.3758 | 24.00 | 24.33 |

Superscript R = resistant lines and S = susceptible lines

and susceptible lines were weighed to find the test weight.

**Grain yield**

Grain yield from each genotype 20 tillers selected random from each plot. Threshing of each genotype was done and yield of 20 tillers of each genotypes were computed.

## RESULTS

In this experiment, 34 wheat genotypes (Table 1) were evaluated for Lipid peroxidation, AUDPC, Test weight, and yield. Out of these, first 32 lines were resistant and last 2 lines were susceptible.

Table 2, shows the correlation among 5 variables viz.: MDA, Test weight, Yield and AUDPC of 32 resistant and 2 susceptible genotypes. MDA shows the negative and significant correlation with test weight (r = -0.4281) and yield (r = -0.3175). A positive and significant correlation (r = 0.7723) with AUDPC.

Table 3, shows the mean of AUDPC, MDA, Test weight (g), and Yield of 20 tillers (g) and in resistance and susceptible wheat genotypes. The mean AUDPC ranged 226.85 to 1254.63, MDA 0.106 to 0.415, test weight 24-37.33 and yield 24 to 47g among the genotypes.

Analysis of variance (Table 4) reveals significant (< 0.0001) of genotype for the MDA. Time has also significant effect on MDA activity while the interaction of time and genotype was also significant.

Table 5, shows the mean value of MDA activity at three time intervals *i.e.* 0 hrs, 24 hrs, and 48 hrs after the inoculation of

**Table 4: ANOVA of MDA content at different time interval in different genotype**

| Source | DF | Mean Square | F Value | Pr > F |
|---|---|---|---|---|
| Time | 2 | 0.2408 | 447.04 | < .0001 |
| Genotype | 33 | 0.0355 | 66.05 | < .0001 |
| Replication | 2 | 0.0137 | 25.45 | < .0001 |
| Time*Genotype | 66 | 0.0017 | 3.31 | < .0001 |

**Table 5: MDA content in wheat samples at different time intervals after pathogen inoculation**

| Genotypes | nmoles/g fresh weight | | | |
|---|---|---|---|---|
| | 0 hrs | 24 hrs | 48 hrs | Genotypes(mean) |
| $1^R$ | 0.0952 | 0.1078 | 0.1179 | 0.1069 |
| $2^R$ | 0.1036 | 0.1175 | 0.1527 | 0.1246 |
| $3^R$ | 0.0854 | 0.1383 | 0.1582 | 0.1272 |
| $4^R$ | 0.0964 | 0.1396 | 0.2110 | 0.1498 |
| $5^R$ | 0.1125 | 0.1419 | 0.1752 | 0.1431 |
| $6^R$ | 0.1082 | 0.1071 | 0.1422 | 0.1191 |
| $7^R$ | 0.1071 | 0.1387 | 0.1782 | 0.1413 |
| $8^R$ | 0.0969 | 0.1337 | 0.1658 | 0.1321 |
| $9^R$ | 0.1205 | 0.1453 | 0.2103 | 0.1587 |
| $10^R$ | 0.0918 | 0.1632 | 0.1752 | 0.1433 |
| $11^R$ | 0.0961 | 0.1071 | 0.1567 | 0.1199 |
| $12^R$ | 0.0869 | 0.0998 | 0.1756 | 0.1207 |
| $13^R$ | 0.0823 | 0.1084 | 0.1811 | 0.1239 |
| $14^R$ | 0.1002 | 0.1130 | 0.2366 | 0.1499 |
| $15^R$ | 0.1176 | 0.1962 | 0.2104 | 0.1747 |
| $16^R$ | 0.1056 | 0.1246 | 0.1832 | 0.1377 |
| $17^R$ | 0.0742 | 0.1246 | 0.2093 | 0.1377 |
| $18^R$ | 0.0816 | 0.0954 | 0.1673 | 0.1360 |
| $19^R$ | 0.1157 | 0.1462 | 0.1998 | 0.1147 |
| $20^R$ | 0.1067 | 0.1432 | 0.2166 | 0.1539 |
| $21^R$ | 0.0998 | 0.1378 | 0.2043 | 0.1554 |
| $22^R$ | 0.1108 | 0.1383 | 0.2142 | 0.1472 |
| $23^R$ | 0.0887 | 0.1409 | 0.1920 | 0.1544 |
| $24^R$ | 0.0809 | 0.1551 | 0.1938 | 0.1432 |
| $25^R$ | 0.0920 | 0.1332 | 0.1642 | 0.1298 |
| $26^R$ | 0.1106 | 0.1538 | 0.2031 | 0.1558 |
| $27^R$ | 0.1129 | 0.1469 | 0.2032 | 0.1543 |
| $28^R$ | 0.0938 | 0.1510 | 0.1931 | 0.1459 |
| $29^R$ | 0.1175 | 0.1537 | 0.2025 | 0.1579 |
| $30^R$ | 0.0871 | 0.1574 | 0.1940 | 0.1461 |
| $31^R$ | 0.1216 | 0.1561 | 0.2038 | 0.1605 |
| $32^R$ | 0.0991 | 0.1650 | 0.2244 | 0.1628 |
| $33^S$ | 0.3174 | 0.3833 | 0.5471 | 0.4159 |
| $34^S$ | 0.2740 | 0.3432 | 0.5105 | 0.3758 |
| LSD(0.05) Genotype (0.0064), Time (0.0216) | | | | |
| Mean(time) | $0.1114^C$ | $0.1502^B$ | $0.2080^A$ | |

pathogen on different wheat genotype (resistant and susceptible). MDA varied significantly ($<0.0001$) over the time. The lowest value (0.106 nmoles/g fresh weight) of MDA was observed in resistant and highest in susceptible genotypes (0.415 nmoles/g fresh weights). The MDA also increased with the progress of time after inoculation. Highest activity (0.547 nmoles/g fresh weight) was recorded at 48 hrs among the genotypes. Rate of MDA activity was more in susceptible lines was more as compared to the resistant lines.

## DISCUSSION

As plants are confined to the place where they grow, they have to develop a broad range of defense responses to cope with pathogenic infections, and environmental stresses. The oxidative burst a rapid transient product of huge amounts of ROS). This is one of the earliest observable aspects of a plants defense strategy. ROS include a variety of short and long-lived molecules such as superoxide ($O_2^-$), hydroxyl radicals ($OH^-$), and hydrogen peroxide ($H_2O_2$) (Apel and Hirt, 2004). Accumulation of ROS is toxic to plant tissue and may results in DNA, RNA, Lipid and membrane damage and loss of other cellular activities. Under normal conditions, ROS are produced as byproducts of a variety of metabolic pathways (Respiration and photosynthesis etc.) and are detoxified by different antioxidant enzymes present in plants as an effective cellular scavenging mechanism (Apel and Hirt, 2004) and produced during stress (Sharma *et al*., 2016). However, when challenged by stresses, Lipid peroxidation is quantifying in terms of the MDA contents and it is the byproducts of lipid peroxidation (enzymatic and non-enzymatic) which causes the membrane disintegration and DNA damage to the plants (Silva and Coutinho, 2010). MDA content is found higher in the susceptible genotypes than the resistant genotypes because the production of ROS that equally increases in both resistant and susceptible genotypes. Resistant genotypes due to higher antioxidant enzymes over comes the ROS damages (Jatav and Chand, 2016). In this research we found that in wheat genotypes, *B. sorokiniana* induces stresses in the plants by disturbing of physiological activities like respiration and photosynthesis in the plants which causes the production of reactive oxygen species (ROS). In 32 resistant cultivars, low MDA, AUDPC and higher yields were observed as compare to 2 susceptible checks. This finding further supports that the resistant cultivar was more successful in detoxifying ROS which leads to less lipid peroxidation (MDA) than the susceptible (Neto *et al*., 2005). Information presented from this study indicates that susceptible plants were unable to maintain optimum levels of ROS under continuing *B. sorokiniana* infection pressure even by using antioxidants produced by the host. The loss of ROS scavenging ability along with increased ROS levels suggests that it is the factor contributes to the susceptible response. Thus production of ROS increases in both resistant and susceptible genotypes but in case of resistant genotypes the antioxidants are optimize the ROS.

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