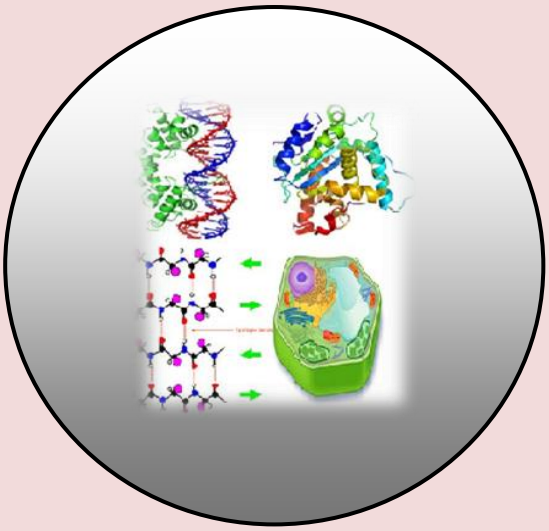
An Important Antioxidant Superoxide Dismutase of *Vigna radiata*: during seed Germination and Seedling Development in both Stressed and Unstressed Conditions

By
Kiran Vati and Samir Sharma

ISSN 2319-3077 Online/Electronic
ISSN 0970-4973 Print

UGC Approved Journal No. 62923
MCI Validated Journal
Index Copernicus International Value
IC Value of Journal 82.43 Poland, Europe (2016)
Journal Impact Factor: 4.275
Global Impact factor of Journal: 0.876
Scientific Journals Impact Factor: 3.285
InfoBase Impact Factor: 3.66

J. Biol. Chem. Research
Volume 36 (1) Part D, 2019 Pages No. 171-182



Journal of Biological and Chemical Research

An International Peer Reviewed / Referred Journal of Life Sciences and Chemistry

Indexed, Abstracted and Cited in various International and National Scientific Databases

Published by Society for Advancement of Sciences®

J. Biol. Chem. Research. Vol. 36, No. 1: Part D, 171-182, 2019
(An International Peer Reviewed / Refereed Journal of Life Sciences and Chemistry)
Ms 36/01/8888/2019

ISSN 2319-3077 (Online/Electronic)
ISSN 0970-4973 (Print)

Kiran Vati
http:// www.sasjournals.com
http:// www.jbcr.co.in
jbiolchemres@gmail.com

REVIEW ARTICLE

Received: 18/04/2019 Revised: 28/05/2019 Accepted: 29/05/2019

An Important Antioxidant Superoxide Dismutase of *Vigna radiata*: during seed Germination and Seedling Development in both Stressed and Unstressed Conditions

Kiran Vati and Samir Sharma
Department of Biochemistry, University of Lucknow, U.P., India

ABSTRACT

In imbibed seeds due to reserve mobilization different metabolic activities occurs lead to generation of reactive oxygen species (ROS). Moderate concentration of ROS perform signaling events like endosperm weakening, radical growth, oxidation of polysaccharides, protein carbonylation, seed reserve mobilization and germination. During germination ROS level more than in dry seeds. After germination increased respiration and other vital activities lead to burst in ROS, cause oxidative damage of protein, lipid and nucleic acid, ultimately lead to cell death. So to protect plant from oxidative damage antioxidants like Superoxide dismutase (SOD), Catalase (CAT), Ascorbate peroxidase (APX), Guaiacol peroxidase (GPX), etc. occur in plants, but their level change according to change in level of ROS. Superoxide dismutase (SOD) antioxidant occurs in all stages in plants and scavenges superoxide ($0_2^{\cdot-}$), primary ROS so it provides first line of defense against oxidative damage. ROS level increases more in stress conditions. SOD play important role in both abiotic and biotic stress. Abiotic stress include drought stress, salt stress, chilling stress, metal stress, UV-B radiation stress, 0_3 stress, $S0_2$ stress, herbicide stress and biotic stress include burst of ROS production by attacking pathogen. In all these stresses ROS level increased so to balance their level antioxidant enzyme activity increased in stress conditions. So ROS is both beneficial and harmful for plant it depend only on their level so ROS can't be eliminated completely only their level can be balanced by antioxidants.

This review mainly focuses about the effect of SOD in dry seeds and also in every stage of plant and play important role during seed germination. This review tell about how SOD play role in stress condition to balance ROS level and how it affected by different type of metal salt and different types of inhibitors.

Keywords: SOD, ROS, Seed germination and Antioxidants.

INTRODUCTION

Oxygen presents in all aerobic organism and is essential for life. Generally ground state oxygen is very less reactive and contain 2 unpaired electrons with parallel spin but by absorbing sufficient energy spin of one of its unpaired electron become reverse and generate singlet oxygen $^1O_2$ and by transferring electrons to oxygen different type of reactive oxygen species (ROS) like $O_2^{\cdot-}$,H_2O_2 and $OH^{\cdot}$ generate (Klaus and Heribert, 2004) (Sharma et al, 2012).

In comparison of ground state O_2 ROS are highly reactive. In plants ROS are continuously generated in different metabolic pathways especially in electron transport chain in mitochondria and chloroplast and in peroxisome by inevitable leakage of electrons to oxygen (Sharma et al., 2012). In chloroplast $O_2^{\cdot-}$ formation enhance when NADPH/NADP+ ratio is increased (Scandalios, 1993) and second when ferredoxin (Fd) (electron acceptor) level is low then electron from PS1 passed to 0_2 and generate $O_2^{\cdot-}$ (Asada et al., 1994; Fridovich., 1995 and 1986; Rubinstein and Luster, 1993). In peroxisome $O_2^{\cdot-}$ form in matrix by xanthine oxidase and in peroxisomal membrane by NADPH oxidase (Lopez Huertas et al.,1996) (Del Rio et al., 1998).

In normal conditions in plant and animal ROS level remain in equilibrium conditions but under stress conditions their level become increase this is called oxidative stress which cause great damage to cell by causing oxidative destruction of biomolecules like protein, lipid and DNA which lead to death of cell but when ROS level is low then they mediate different signaling events in plants (Sharma et al., 2012) like stomata closure (Neill et al., 2002) (Yan et al., 2007) (Kwak et al., 2003) programmed cell death (Bethke and Jones., 2001), gravitropism (Jung Hee Joo et al., 2001) tolerance to biotic and abiotic stress so we never eliminate ROS completely (Alscher et al., 2002) (Zelko et al., 2002).

ROS are scavenged by many enzymatic and non enzymatic antioxidants in which SODs are first line of defense against oxidative stress this enzyme dismutate 2 $O_2^{\cdot-}$ radical in to O_2 (dioxygen) and H_2O_2 (Mc Cord and Fridovich, 1969).

In animals low level of ROS help in signaling of many pathways but when their concentration increase then it is responsible for causing many diseases like Cancer, Hypertension, Diabetes, Atherosclerosis, Inflammation and Premature aging (Zelko et al., 2002). Because of higher mid point reduction potential of high-spin Mn^{+3}/Mn^{+2} it has lower Fenton reactivity so it is safer in oxidative stress (Miller, 2012).

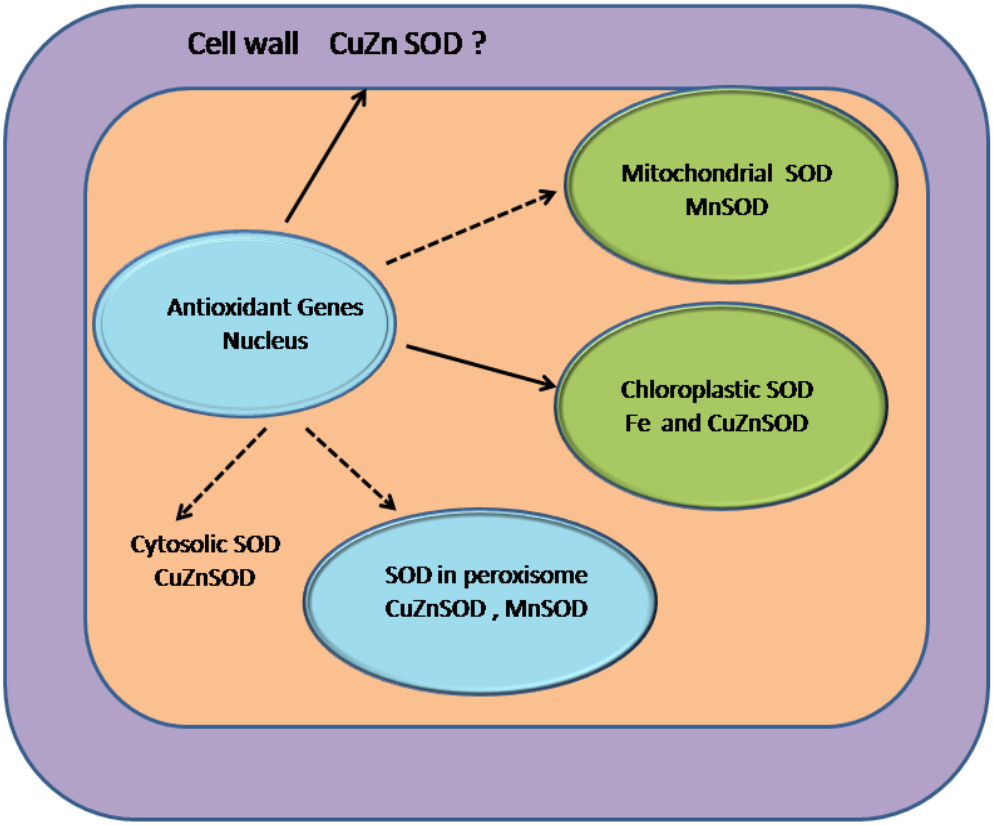


Figure 2. Location of SODs throughout plant cells (Alscher G.R. et al., 2002).

Superoxide dismutase

Because of change in chemical nature of environment nature of SOD change with time (Miller, 2012). In earlier atmosphere O_2 was very less so iron occur in reduced form (Fe^{+2}) and act as active metal cofactor in FeSOD but as O_2 increase in environment Fe^{+2} oxidized to Fe^{+3} so become less bioavailable and at that time Mn^{+3} was abundant so switch to Mn SOD occur (Bannister et al., 1991). Iron by reacting with $O_2^{\cdot-}$ form $OH^{\cdot}$ Radical in both Fenton and Haber Weiss reaction, which cause great oxidative damage to cell this reaction not shown by Mn SOD so is safer than Fe SOD (Gutteridge and Bannister, 1986; Bannister et al., 1991). When environment completely replenish with O_2 then fe^{+2} is very less bioavailable then insoluble Cu^{+1} oxidised to soluble Cu^{+2} form so then CuZn SOD become abundant in which Cu^{+2} is active metal cofactor (Erturk, et al. 1999) (Alscher, et al 2002). Fe SOD is structurally similar to Mn SOD but both are metal specific and totally different from CuZn SOD (Erturk et al, 1999). On basis of metal cofactor SOD classified in to 4 types Fe SOD, Mn SOD, CuZn SOD and Ni SOD (Abreu et al., 2010) (Miller, 2012). Fe SOD, Mn SOD and CuZn SOD identified by reaction with NaCN and H_2O_2 in which Fe SOD inhibited by H_2O_2 CuZn SOD inhibited by both NaCN and H_2O_2 and Mn SOD is resistant toward both (Reddyd and Venkaiah, 1984). Superoxide of different class located in different cellular compartment of plant cells like Fe SOD located in chloroplast, Mn SOD located in mitochondria and peroxisome, CuZn SOD located in cytosol, chloroplast, peroxisome and cell wall (Alscher, et al., 2002).

Iron superoxide dismutase

Fe SOD in some plant species contain a tripeptide sequence SRL or ARL close to the carboxyl terminus of enzyme, direct protein to peroxisome but in some species this sequence is not present so it clear that this is not obligatory sequence for this enzyme (Van Camp et al., 1994). Fe SOD in nature are of 2 types occurring one is homodimer and other is tetramer (Yost and Fridovich 1973) (Puget and Michelson, 1974). Tetrameric Fe SOD found mostly in higher plants and it contains 4 equal subunits of 80-90 kDa with 2-4 gram iron atom in their active center (Alscher G.R. et al., 2002).

Mechanism of FeSOD

$Fe^{3+}SOD + O_2^{\cdot-} \rightarrow Fe^{2+}SOD + O_2$ (1)

$Fe^{2+}SOD + O_2^{\cdot-}(+2H^+) \rightarrow Fe^{3+}SOD + H_2O_2$ (2)

$Fe^{2+}SOD + O_2^{\cdot-} \leftrightarrow (Fe^{2+}SOD\text{-}O_2^{\cdot-}) + (2H^+) \rightarrow Fe^{3+}SOD + H_2O_2$ (3)

$Fe^{3+}SOD + H_2O_2 \rightarrow Fe^{2+}SOD + HO_2 + H+$ (4)

$Fe^{2+}SOD + H_2O_2\,(+2H+) \rightarrow Fe^{3+}SOD....OH + OH-$ (5) (Abreu A. I. et al. 2010).

Fe^{+2} in active site of FeSOD coordinated by 3 histidine and 1 aspartic acid residue (Perry et al., 2010).

Manganese superoxide dismutase

Mn SOD is a Mn containing superoxide dismutase occur both in dimeric and tetrameric form (Abreu A. I. et al. 2010). Mn SOD becomes non-functional by removing Mn^{+2} from their active site (Fridovich 1986). In eukaryote nuclear encoded Mn SOD contain mitochondrial targeting sequences at their N-terminal end by which it imported to mitochondria and after reaching mitochondria this sequence cleaved (Perry P.J.J.et al .,2010). In bacteria cytosolic Mn SOD occur (Abreu A. I. et al. 2010).

Mechanism of MnSOD

$Mn^{3+}SOD\,(OH^-) + O_2^{\cdot-}(+H^+) \rightarrow Mn^{2+}SOD(H_2O) + O_2$ (1)

$Mn^{2+}SOD\,(H_2O) + O_2^{\cdot-}(+H^+) \rightarrow Mn^{3+}SOD(OH^-) + H_2O_2$ (2)

$Mn_{2+}SOD\,(H_2O) + O_2^{\cdot-} \rightarrow Mn^{3+}SOD(H_2O)\text{-}O_2^{\cdot-}$ (3)

$Mn^{3+}SOD\,(H_2O)\text{-}O_2^{\cdot-}(+H^+) \rightarrow Mn^{3+}SOD(OH^-) + H_2O_2$ (4)

Above these reactions show mechanism of Mn SOD in which $Mn^{3+}SOD\text{-}O_2^{\cdot-}$ formed in parallel with $Mn^{3+}SOD$, often called the inhibited complex (Abreu et al., 2010). Mutation of some amino acids in the active center of Mn SOD decreased the catalytic activity (Abreu et al., 2010). In the active site of MnSOD positively charged amino acid present by which $O_2^{\cdot-}$ radical attracted and enter in the active site and become reduced by metal present in active site (Asada 1994, Bowler et al. 1994) (Erturk, et al, 1999).

Copper zinc superoxide dismutase

CuZn SOD is a dimeric enzyme each monomer of enzyme have 1 Cu and 1Zn both are connected to one another by a imidazole group of histidine beside this Cu connected by 3 other histidine residues and 1 water molecule and Zinc connected by 2 histidine and 1 aspartic acid residues (Abreu et al. 2010).

Electrostatic guidance to O_{2}^{-} provided by positive charged arginine present at entrance of active site of CuZn SOD (Fisher et al., 1994)). Oxidative state of Cu in this active site change from +2/+3. In CuZn SOD Zn is not redox active metal and it play important role in stability of CuZnSOD (Abreu et al. 2010).

Mechanism of CuZn SOD

$Cu^{2+}Zn^{2+}SOD + O_{2}^{-} \rightarrow Cu^{+}Zn^{2+}SOD + O_2$ (1)

$Cu^{+}Zn^{2+}SOD + O_{2}^{-}(+2H^{+}) \rightarrow Cu^{2+}Zn^{2+}SOD + H_2O_2$ (2)

$Cu^{2+}Zn^{2+}SOD + H_2O_2 \rightarrow Cu^{+}Zn^{2+}SOD + HO_2 + H^{+}$ (3)

$Cu^{+}Zn^{2+}SOD + HO_2^{-} \rightarrow Cu^{2+}Zn^{2+}SOD\text{-}OH + OH^{-}$ (4)

In overall mechanism dismutase and peroxidase cycle are combined in which O_2, H_2O_2, OH, OH^- all generated. In this enzyme predominant site of OH Radical attack is His61 which bridging both Cu & Zn in active site (Abreu A. I. et al. 2010). In Human Familial amylotrophic lateral sclerosis (FALS) genes on chromosome21 which is responsible for CuZn SOD expression becomes damaged (Rosen et al. 1993). Site specific mutation of some amino acids in active site of enzyme lead to increase in their catalytic rate (Getzoff et al. 1992).

In CuZn SOD Cu is critical for their catalytic activity and zinc play role in their functional stabilization if we replace Cu from any other metal then its catalytic activity gone but if we replace zinc with any other metal then their catalytic activity not affected but if we completely remove zinc then only little effect occur on activity because zinc provide structural stability to the active site (Bordo et al. 1994, Cudd and Fridovich 1982).

Nickel superoxide dismutase

NiSOD first discovered in Streptomyces (Gotto et. al., 2000) and also reported in actinobacteria and other bacteria. Ni SOD fusion gene occurs in eukaryotic green algae Osterococcustauri. Ni SOD not reported in higher plants and animals (Abreu, et al. 2010).

ROS in seed germination

ROS play significant role in seed germination and release of dormancy. In dry seeds ROS are produced by lipid peroxidation but in hydrated status many metabolic pathways are activated which leads to generation of superoxide like lipid catabolism in glyoxisome, purine catabolism in peroxisome, electron transport chain (ETC) in mitochondria and chloroplast. (Vertucci et al. 1995) (Sun and Leopold 1995) (Bailly et al 2004). Mitochondrial ETC directly give electrons to O_2 by which O_2 become reduced to form 0_{2}^{-} but in case of chloroplast O_2 which is generated in photosynthesis can take electrons which are passing from PSI and PSII, and become reduced to O_{2}^{-} (Rhoads et al 2006) , (Rodriguez Serrano et al. 2009) (EI- Maarouf Bouteau and Bailly 2008). O_{2}^{-}cannot cross the membrane so when O_{2}^{-} by SOD convert in to H_2O_2 which can further react with Fe^{+2} or Cu^{+2}, OH Radical generated which can cross membrane and move outside (Puntarulo et al. 1998). In dry seeds sight of ROS action is close to their site of origin but in hydrated seeds or imbibed seeds their site of action is farther because water allow to move ROS to their farther site of action (Bailly et al. 2008) when ROS attack their target like protein, lipid or nucleic acid then it lead to generation of more ROS then it lead to alteration of which cause alteration in gene expression (Gill and Tuteja 2010). ROS provide protection against pathogen, heat stress, chilling stress, high light exposure which leads to photo bleaching in leaves of plants (Levine et al., 1996), (Prasad 1996). During seed germination ROS play very important role in endosperm weakening, mobilization of food reserve in seed, protection from pathogen and in programmed cell death (EI Maarouf –Bouteau, Bailly, 2008). During germination amount of ROS in seeds controlled by giberrellin (GA) and abcissic acid (ABA) homones. GA increase ROS concentration and ABA decreased ROS concentration hence GA promote germination. O_{2}^{-} by reacting with metals like Fe^{+2} or Cu^{+2} in Fenton's reaction convert to OH Radical which leads to seed reserve mobilization, endosperm weakening, radical growth, oxidation of polysaccharide, carbonylation of protein which lead protein more prone to degradation it also lead to their mobilization during germination (Job et al., 2005), oxidation of lipid these all lead seed reserve mobilization, cell wall loosening (Bailly et al 2004).

ROS cause protein modification through carbonylation, lipid by peroxidation and nucleic acid by mutation (Bailly et al. 2008) (Bailly et al. 1996, Munne-Bosch et al. 2011, Pukackaand Rata Jczak 2007).

ABA inhibit all these processes hence it called germination inhibiting enzyme (Gomes and Garcia, 2013) while in stressful conditions excess ROS generated which prevent radicle emergence. Normally during germination level of ROS increase because SOD activity decrease this increased level of ROS is necessary for seed germination and dormancy control in seeds because these ROS cause seed reserve mobilization , endosperm weakening which is necessary for seed germination. In seed dormancy control ROS play important role by interacting with plant hormone which are involve in seed germination like auxin, gibberellins, ABA, ethylene, brassinosteroids etc. In non dormant embryo $O_2^{\cdot-}$ accumulated in radicle of embryo which enhance seed germination but in dormant seeds $O_2^{\cdot-}$ accumulated irregularly level of NaDPH oxidase also increase during seed germination which act as source of $O_2^{\cdot-}$. Seed dormancy alleviation in plants by ROS associated with mRNA oxidation, this oxidation does not occur until seeds are dormant (Bazil et al. 2011). In plants when treated with H_2O_2 then it up regulate or increase the expression of GA induced gene and inhibit the expression of those genes which are involved in GA catabolism and enhanced expression of genes which are involved in GA synthesis, but not affect ABA sensitivity (Bailly et al 2004.

ROS detection

Reactive oxygen species which has unpaired electron detected by electron paramagnetic resonance EPR like $O_2^{\cdot-}$ radical and H_2O_2 has no unpaired electron so not detected by EPR. ROS should be stable and accumulated in measureable level so for make them stable either do extreme cooling or use spin trap. In spin trap $O_2^{\cdot-}$ react with an adduct which is nonreactive so by this way we make $O_2^{\cdot-}$ stable (Stowe and Camara, 2009). Intra matrix fluorescent indicator 2'7'-dichlorofluorescin (DCF) or fluorescent amplex red/ horseradish peroxidase used for detecting (H_2O_2) ROS in mitochondria (Stowe and Camara, 2009).

Role of SOD in oxidative stress

When in plants ROS generated at high level which exceeds the defensive system of plants then this increase in ROS cause great damage to plants by causing oxidative damage of protein ,lipid, carbohydrate. This condition of plant cell is caused oxidative stress (Sharma et al. 2012) lead to death of plant. Oxidative stress is of 2 types' abiotic stress and biotic stress. Abiotic stress include damage caused by ROS in drought stress, salt stress, chilling stress, metal stress, UV B radiation stress, O3 stress, SO2 stress, herbicide stress etc (Burdon et al. 1996, Fadzilla et al. 1997, Hernandez et al. 1995, Kangasjarvi et al. 1994, Well burn and Well burn, 1996) and biotic stress caused by increase production of ROS by attacking pathogen to plants(Hammond Kosack and Jones 1996, Snijder et al.1996).In drought stress photosynthetic electron transport chain become over reduced due to lacking of electron acceptor so excess electron leak to reaction lead to generation of more ROS (Sharma et al, 2012). High light intensity in drought stress disturbs balance between light capture and light utilization so photo respiratory pathway is enhanced (Noctors et al. 2002) which lead to 70%of total H2O2 production. Activity of different antioxidant enzymes like SOD, POD, APX, GR etc increased in this stress (Sharma et al, 2012).

Salt stress

In salt stress because of high salt concentration electron transport pathway in mitochondria, chloroplast disturbed and photorespiration induced lead to high level of ROS generation like $O_2^{\cdot}$, $\cdot OH$, H_2O_2 and $^1O_2$ (G. Tanou et al. 2009) (J.A. Hernandez et al. 2000) (Sharma et al, 2012). Carbon fixation inhibited by high salt stress induced by stomata closure because of reduction in the availability of CO_2 to leaves so lead to over reduction of photosynthetic electron transport system and also induce photorespiration causing excess ROS production (Hernandez et al. 2000).

Chilling stress

chilling stress in plant lead to imbalance between light absorption and light use cause over reduction of mitochondrial electron transport chain which lead to enhanced production of ROS (B.A. Logan et al. 2006) (Hu et al. 2008), cause lipid peroxidation and protein carbonylation excessively (Prasad. 1997) so activities of different antioxidant enzymes like SOD, APX, MDHAR, DHAR and GR increased (Fryer et al. 1998).

Metal stress

Some heavy metals like cu, cd, Zn, Ni are essential micronutrient for growth and development of plant but when these metals present in high concentration in plants then create oxidative stress in plants and plant development and their growth affected (Li al al. 2005).

Cu stress negatively affecting light reaction components (Vinit Dunand et al. 2002) and CO_2 fixation which badly affect photosynthesis and lead to enhanced production of ROS such as $O_2^{\cdot-}$, OH, H_2O_2. Redox active metals like Fe, Cu, Cr produce high level of ROS by redox cycle while redox inactive metals like Pb, Cd, Hg and other lead to reduction in major antioxidants (Shah et al, 2001) (Maheshwari and Dubey 2009) (Srivastava and Dubey, 2011, Sharma and Dubey, 2007, Verma and Dubey 2003). Increased activities of different antioxidant enzyme in metal stress provide protection to plant against oxidative damage (Shah et al. 2001).

In plants when concentration of copper is below 5µg-g^{-1} DW then it lead to limiting plant productivity but as its concentration reaching above 30 µg-g^{-1} DW then it is toxic to plants (Marschner 1995). In plants like *Arabidopsis thaliana, Brassica juncea, lycopersicum lycopersicum, Oryza sativa* during copper limiting condition CuZn SOD is down regulated and FeSOD is upregulated these plants can maintain $O_2^{\cdot-}$ scavenging capacity and save copper which then transported to plastocyanin which is necessary for photosynthesis (Christopher M. Cohu and Marinus Pilon, 2007) but this is not occure with all plant varieties. In Arabidopsis when copper is sufficient then both cytosolic and chloroplastic CuZn SOD expressed and active but Fe SOD is not detected but when Cu is limiting then both cytosolic and chloroplastic CuZn SOD down regulated and Fe SOD is upregulated (Christopher M. Cohu and Marinus Pilon, 2007).

Plant which sprayed with Manganese (Mn) lead to increased dry weight of plant and inhibition of lipid peroxidation through decreasing malondialdehyde, prevent chlorophyll from decomposition and maintained the integrity of cell membrane. Senescence induced by drought in perennial rye grass can be delayed by manganese. When MnSOD expression increased in plants then it enhance tolerance towards environmental stresses and manganese (Mn) is very important in increasing the yield. Mn increase resistant towards senescence in plants induced by drought and other oxidative stresses (Yu-Tong Wang et al. 2010).

In plant uptake of phosphate occur more efficiently than arsenate (Meharg and Macnair, 1994). In mild arsenic concentration SOD activity increase but as arsenic concentration increase to high level then it cause decrease in SOD activity. The plant supplied with high phosphorus decrease oxidative damage caused by arsenic because it limit the conversion of arsenate to arsenite so decrease formation of ROS but this protective effect was counteracted by high arsenic concentration (Gunes et al., 2009)

Deficiency or excess of zinc generate oxidative stress by generating enhanced level of ROS which disturb redox homeostasis in mulberry plants and cause oxidative damage (Tewari, et. al. 2008). Loss of membrane integrity by lipid peroxidation is act as marker of damage by ROS is primary effect of zinc deficiency (Cakmak and Marschner, 1988). Deficiency of Zn increase $O_2^{\cdot-}$ by increasing activity of NADPH-dependent oxidase activity and by decreasing NADP to NADPH as consequence of decreased uptake (Cakmak, 2000) and Photosynthetic fixation of CO_2 (Sharma et al., 1994, 1995). In plant Zn level is marked by carbonic anhydrase (CA) activity because CA activity is well correlated to total Zn tissue concentration.

Different types of ROS and NO in plants under Cd stress detected by confocal laser scanning microscopy (CLSM) by using Cd specific fluorescent probe like $O_2^{\cdot-}$ detected by Dihydroethidium (DHE) and it produce red fluorescence in CLSM, H_2O_2/peroxides by 2′,7′-Dichlorofluorescein diacetate (DCF-DA) produce green color and NO by 4,5- diaminofluoresce indiacetate (DAF-2DA) fluoresce with green color by seeing with CLSM (Fricker and Meyer, 2001; Sandaloi et al., 2008).

In some plants like wheat, pea, bean Cd dependent reduction of SOD activity occur while in some plants like Alyssum plant, sunflower, coffee, radish root, its opposite effect occur because of difference in metal concentration and treatment and type of plant tissue studied(Milano et al., 2003, Sandalio et al. 2001) (Schickler and Caspi 1999).

UV-B Radiation

In plants UV-B radiation limited the CO_2 assimilation which leads to generation of excessive ROS (Allen et al., 1997). UV-B radiations in plants activate NADPH oxidases which lead to generation of active oxygen species which cause great damage to cell (Rao et al, 1996).

Herbicides like Bipyridinium and Bipyridylium (methyl viologen) lead to enhanced production of ROS by causing electron transfer to O_2 (Bolwell, 1996) so antioxidant concentration like SOD increase to provide protection against ROS (Alscher, et al., 2002).

Water logging stress

In Mung bean plants (*Vigna radiata*) water logging leads to enhanced production of ROS by inducing membrane linked NADPH oxidase which lead to increased production of SOD and this increase in SOD mainly because of CuZn SOD then Mn SOD and finally by some increase in Fe SOD these all provide protection against oxidative injury (Sairam et al. 2011).

Biotic stress

When pathogen attack the plant then NADPH oxidase of plasma membrane activate lead to production of O_2^{-}, which after dismutation by SOD convert in O_2 and H_2O_2. H_2O_2 strengthen the cell wall where pathogen attack so prevent the pathogen spread because tissues around that side where pathogen attack become dead (Hammond kosack and Jones, 1996). When pathogen attack the antioxidant enzyme express in different plant according to their need like in some plant SOD, CAT, APX and POD activities increase while in some other plants CAT and APX activities decrease (Sharma et al. 2012). SO antioxidants in plants on pathogen attack regulate generation of ROS (Sharma et al. 2012).

Effect of salt on SOD

When plant seeds treated with NaCl salt then it lead to decrease chlorophyll content, decrease biomass production and decrease root and shoot elongation and cause oxidative damage but when we apply some phytoharmone like gibberellins (GA3) or polyspermine (PAn) reverse the adverse effect of NaCl so these hormone act as antagonist of NaCl. In plants after imposition of NaCl salt stress SOD and CAT level increased. SOD is more potent scavenger of O_2^{-} radical which is generated after salt stress so SOD provide a first line of defense against cellular injury due to abiotic stress (Ghosh, S. et al., 2014).

SOD alone are not responsible for protecting plants oxidative damage because SOD convert O_2^{-} to H_2O_2 which if not removed by CAT or APX or GPX could react with remaining O_2^{-} to form highly dangerous $\cdot OH$ radical which cause great oxidative damage to protein, lipid and DNA (Sharma et al. 2012).

Regulation of SOD activity: In human when CuZn SOD react with H_2O_2 then metal bound $\cdot OH$ radical generate bound to metal then it lead to oxidation of histidine residues which lead to coordination of Cu so Cu dissociate from active site so it lead to enzyme inactivation(Randi H Gottfredsen et al. 2013).

$Enz\text{-}Cu^{2+} + H_2O_2 \leftrightarrow Enz\text{-}Cu^{+} + O_2^{-} + 2H^{+}$ (1)

$Enz\text{-}Cu^{+} + H_2O_2 \leftrightarrow Enz\text{-}Cu_2^{+}\cdot OH + OH^{-}$ (2)

$Enz\text{-}Cu^{2+}\cdot OH + ImH \rightarrow Enz\text{-}Cu^{2+} + Im\cdot + H_2O$ (3)

Enz =enzyme ImH= Imidazole moity of histidine residue in active site of enzyme (M.B. YIM et al 1990). Mn SOD not catalyzes metal bound. OH radical formation from H_2O_2 so it is not inactivated by H_2O_2 (Archibald and Fridovich, 1982).

In CuZn SOD cyanide and azide after entering in to active site bind with metal ion and provide protection from H_2O_2(Mota de freitas et al,1984)(Hodgson et al,1975). Fe SOD also lead to generation metal bound .OH radical which lead to degradation of Fe SOD through Haber weiss reaction.

$$H_20_2 + 0_2^{-} \xrightarrow{Fe^{2+},\ Fe^{3+}} OH^{-} + O_2 + OH\cdot$$

(Halliwell 1987, Cadenas 1989; Fridovich 1989) (Ed. W.T. Sang, Chris Bowler et al 1991). In Human cyclin B1/cdk1 lead to phosphorylation of mitochondrial antioxidant Mn SOD at ser-106 which lead to enhanced enzymatic activity and stability of this enzyme (Dement Candas, Ming Fan et al. 2012). In Human PI3k/Akt upregulate CuZn SOD expression through activation of Nf-kB (Ana I. ROJO, Marta Salinas et al. 2004).

Inhibition of sod

In mung bean cytoplasmic CuZn SOD is highly sensitive to inhibition by cyanide and azide, inhibitory effect of cyanide occur on cytoplasmic CuZn SOD immediately in very minute amount than in chloroplastic CuZnSOD. Chloroplastic CuZn SOD is more readily inhibited by diethyldithiocarbamate (DDC) and o-phenanthroline then cytoplasmic CuZn SOD but both isoenzyme show same denaturation pattern with heat, guanidinium chloride and urea.

DDC has specificity towards copper according to the accessibility of copper in these 2 enzymes they show different susceptibility toward inhibitors or metal chelators (Reddy and Venkaiah, 1984). Denaturation by Gdmcl (guanidinium chloride) and urea is reversible for the mung bean isoenzymes but chloroplastic CuZn SOD is more susceptible towards these denaturation then cytoplasmic CuZn SOD but this denaturation is not reversible for Fe SOD and Mn SOD this show that CuZn SOD are more stable then Fe SOD or Mn SOD (Puget and Michelson., 1974; Lumsden et al., 1976). Nitration of Mn SOD by peroxynitrite completely inhibited enzymatic activity. In human Mn SOD, tyr34 at active site is highly susceptible to peroxynitrite mediated nitration. Nitration and inactivation of Mn SOD increase mitochondrial superoxide level which lead to generate more peroxynitrite which amplifying nitration and oxidation of other mitochondrial proteins (Lee Ann Mac Millan Crow et al., 1997). Peroxynitrite attack nucleophilic amino acids including Lysine, cysteine, methionine, tyrosine and tryptophan. In Arabidopsis nitration of tyr63 caused almost complete inactivation of Mn SOD by $ONOO^-$.

CONCLUSION

ROS generate in plant at each stage but their level in remain balance. When ROS generate in low or moderate level then play important role in different signaling event but when generate in high level then cause great oxidative damage to plant. Antioxidants balance the level of ROS in plant and SOD scavenge $O_2^{\cdot-}$ so form first line of defense against oxidative damage and play important role in seed germination and plant development in both stressed and unstressed conditions.

ACKNOWLEDGEMENTS

The authors thank the Department of Biochemistry, University of Lucknow for providing the central research facilities for carrying out this work.

REFERENCES

Miller, A.F. (2012). Superoxide dismutase: Ancient enzymes and new insights. FEBS, 586: 585-595.

Asada, K. (1994). Production and action of active oxygen species in photosynthetic tissues. In: Foyer and Mullineaux, Causes of Oxidative stress and amelioration of defense systems in plants pp. 78-99, Bocca Raton, FL CRC Press.

Apel, K. and Hirt, H. (2004). Reactive oxygen species: metabolism, oxidative stress, and signal transduction. *Annu Rev Plant Biol,* 55: 373-399.

Zelko, I.N., Mariani, T.J. and Folz, R.J. (2002). Superoxide dismutase multigene family: A comparison of the CuZnSOD (SOD1), MnSOD (SOD2), and EC-SOD (SOD3) gene structure, evolution and expression. *Free Radical Biology and Medicine*, 33: 337-349.

Mc Cord, J.M. and Fridovich, I. (1969). Superoxide dismutase: An enzymatic function for erythrocuprein (Hemocuprein). *J. Biol. Chem.* 22: 6049-6055.

Sharma, P., Jha, A. B., Dubey, R. S. and Pessarakli, M. (2012). Reactive oxygen species, oxidative damage, and antioxidative defense mechanism in plants under stressful conditions. *Journal of Botany*, Article ID 217037, 26 pages, doi:10.1155/2012/217037.

Fridovich, I. (1995). Superoxide radical and Superoxide dismutases. *Annu. Rev. Biochem*. 64:97-112.

Fridovich, I. (1986). Superoxide dismutases. in: Meister A. (eds) Advances In: Enzymology, pp 61-97, New York, John Wiley and Sons.

Rubinstein, B. and Luster, D.G. (1993). Plasma membrane redox activity: Components and role in plant processes. *Annu. Rev. Plant Physiol. Plant Mol. Biol*. 44:131-155.

Del Rio, L.A., Pastori, G.P., Palma, J.M., Scandalio, L.M., Corpas, F.J., Jimenez, A., Lopez-Huertas, E., and Hemandez, A.J. (1998). The activated oxygen role of peroxisomes in senescence. *Plant. Physiol*. 116:1195-2000.

Neill, S. Desikan, R. and Hancock, J. (2002). Hydrogen peroxide signalling. *Current Opinion in Plant Biology*, 5: 388–395.

Yan, J., N. Tsuichihara, N., Etoh, T., and Iwai, S., (2007). "Reactive oxygen species and nitric oxide are involved in ABA inhibition of stomatal opening," *Plant Cell Environment*, 30:1320-1325.

Kwak, J. M., Mori, I, C., Pei, Z, M., et al. (2003). "NADPH oxidase Atrboh D and Atrboh F genes function in ROS-dependent ABA signaling in Arabidopsis," EMBO Journal, 22, pp. 2623-2633.

Bethke, P.C. and Jones, R.L. (2001). "Cell death of barley aleurone protoplasts is mediated by reactive oxygen species," *Plant Journal*, 25, pp.19-29.

Joo, J.H., Bae, Y. S. and Lee, J. S. (2001). "Role of auxin-induced reactive oxygen species in root gravitropism," *Plant Physiology*, 26, pp. 1055-1060.

Ruth Grene Alscher, R.G., Neval Erturk, N. and Heath, L.S. (2002). Role of Superoxide dismutases (SODs) in controlling oxidative stress in plants: *Journal of Experimental Botany*, 53, pp. 1331-1341.

Bannister, W.H., Bannister J.V., Barra, D. and Bossa, F. (1991). Evolutionary aspects of Superoxide dismutase: The Copper/Zinc enzymes, Free Radical Research Communication, 12-13 Pt 1: 349-61.

Gutteridige, John. M.C. and Joe V. Bannister, Joe V. (1986). Copper Zinc and Manganese Superoxide dismutase inhibit deoxyribose degradation by the superoxide driven Fenton reaction at 2 different stages. *Biochem. J.* 234, 225-228.

Isabel, A. Abreu, I.A. Diane, E. and Cabellib, D.E. (2010). Superoxidedismutases: A review of the metal-associated mechanistic variations, *Biochimica et Biophysica Acta* 180,263-274.

Reddy, C.D. and Venkaiah, B. (1984). Purification and characterization of CuZn superoxidedismutases from Mung bean (*Vigna radiata*) seedlings, *J. Biosci*, 6 PP.115-123.

Bowler, C., Van Camp. W., Van Montague, M. and Inze, D. (1994). Superoxide dismutase in plants. *Crit. Rev. Plant Sci*, 13,199-218.

Yost, JrFj and Fridovich, I. (1973). An Iron containing Superoxide dismutases From Escherichia coli. *Journal of Biological Chemistry*, 248, 4905-4908.

Puget, K. and Michelson, A. M., (1974). Iron containing Superoxide dismutases from luminous bacteria. *Biochimie*, 56, 1255-1267.

Perry, J. J.P., Shin, D.S., Getzoffa, E.D. and Tainer, J.A. (2010). The structural biochemistry of the Superoxide dismutases. *Biochimicaet Biophysica Acta*. 1804, 245-262.

Bowler, C., Van Camp. W., Van Montague. M. and Inze, D. (1994). Superoxide dismutase in plants. *Crit. Rev. Plant Sci*.13, 199-218.

Getzoff, E.D., Cabelli, D.E., Fischer, C.L., Parge, H.E., Viezzoli M.S., Banci, L. and Hallewell, R.A. (1992). Faster Superoxide dismutase mutants designed by enhancing electrostatic guidance. *Nature,* 358, 347-351.

Bordo, D., Djinovic, K. and Bolognesi, M. (1994). Conserved patterns in the CuZn superoxide dismutase family. *J. Mol. Biol.* 238, 366-386.

Cudd, A. and Fridovich, I. (1982). Electrostatic Interactions in the Reaction mechanism of Bovine Erythrocyte Superoxide dismutase. *J. Biol. Chem.* 257, 11443-11447.

Goto, J.J., H. Zhu, R.J. Sanchez, A. Nerissian, E.B. Gralla, J.S. Valentine and D.E. Cabelli (2000). Loss of in Vitro metal ion binding specificity in mutant copper-zinc superoxide dismutases associated with familial amyotrophic lateral sclerosis, *J. Biol-Chem.* 275, 1007-1014.

Bailly, C., H.E.M. Bouteau, and F. Corbineau (2008). From intracellular signaling networks to cell death: the dual role of reactive oxygen species in seed physiology. *C. R. Biologies*. 331, 806-814.

Vertucci, C.W. and Farrant, J.M. (1995). Acquisition and loss of desiccation tolerance. In: Kigel J; Galili G, eds. Seed Development and Germination. New York: Marcel Dekker, 237‑71.

Bailly, C., A. Benamar, and F. Corbineau (1996). Changes in malondialdehyde content and in superoxide dismutase, catalase and glutathione reductase activities in sunflower seeds as related to deterioration during accelerated aging. *Physiol. Plant.* 97, 104-110.

Bailly, C., J. Leymarie, A. Lehner, S. Rousseau, D. Côme, and F. Corbineau (2004). Catalase activity and expression in developing sunflower seeds as related to drying. *J. Exp. Bot*. 55: 475-483.

Bouteau, H., EI., Maarouf and Bailly C. (2008). Oxidative Signalling in seed germination and dormancy. *Plant Signalling and Behavior* 3, 175-182.

Puntarulo, S., Sanchez R., A. and Boveris A. (1998). Hydrogen peroxide metabolism in soybean embryonic axes at the onset of germination. *Plant Physiol*, 86,626‑30.

Sun, W.Q. and Leopold, A.C. (1995). The Maillard reaction and oxidative stress during aging of soybean seeds. *Physiol Plant*, 94, 94‑104.

Bailly, C. (2004). Active oxygen species and antioxidants in seed biology. *Seed Sci Res*, 14, 93‑107.

Job, C., Rajjou, L., Lovigny, Y., Belghazi, M. and Job, D. (2005). Patterns of protein oxidation in Arabidopsis seeds and during germination. *Plant Physiol*, 138,790‑802.

Gill, S.S. and and Tuteja N. (2010). Reactive oxygen species and antioxidant machinery in abiotic stress tolerance in crop plants. *Plant Physiology and Biochemistry*. 48, pp 909-930.

Stowe, D., F. and Camara A. K. (2009). Mitochondria reactive oxygen species production in excitable cells: Modulators of mitochondrial and cell function. *Antioxid Redox Signal,* 11, 1373–1414.

Sharma, P., Jha, A., B., Dubey, R., S. and Pessarakali, M. (2012). Reactive oxygen species, oxidative damage and antioxidative defense mechanism in plants under stressful conditions, *Journal of Botany*, 2012, 1-26.

Burdon, R.H., O. Kane., Fadzillah, N., Gill, V., Boyd, P.A. and Finch, R.R. (1996). Oxidative stress and responses in Arabidopsis thaliana and oryza subjected to chilling and salinity stress. Biochem. *Soc. Trans*. 24. 469-472.

Fadzilla, N.M., Finch, R.F. and Burdon, R.H. (1997). Salinity, oxidative stress and antioxidant responses in shoot cultures of rice. *J. Exp. Bot.* 48: 325-331.

Hernandez, J.A., Corpaz, F. J. Gomez, M., Delrio, L.A. and Sevilla, F. (1993). Salt induced oxidative stress mediated by activated oxygen species in per leaf mitochondria. *Physiol. Plant.* 89: 103-110.

Kangasjarvi, I., Talvinen, J., Utriainen, M. and Karjalainen, R. (1994). Plant defense systems induced by ozone. *Plant Cell Environ*. 17:783-794.

Wellburn, A.R. and Wellburn, F.A.M. (1996). Gaseous pollutants and plants defense mechanisms, *Biochem. Soc Trans.* 24:461-464.

Hammond-Kossack, K.E. and Jones, J.D.G. (1996). Resistance gene-dependent plant defense responses. *Plant Cell* 8:1773-1791.

Snijder, A.J., Wastie, R.L., Glide Well, S.M. and Goodman, B.A. (1996). Free redicals and other paramagnetic ions in interactions between fungal pathogens and potato tubers. *Biochem. Soc. Trans.* 24: 442-446.

Noctors, G., Veljovic Jovanovic, S., V., S., Driscoll, S., S., Novits Kaya, L. and C.H. Foyer, C.H. (2002). "Drought and oxidative load in the leaves of C3 plants: a predominant role for photorespiration "*Annals of Botany*, 89, pp. 841-850.

Tanou, G., Molassiotis, A. and Diamantidis, G. (2009). "Induction of reactive oxygen species and necrotic death-like destruction in strawberry leaves by salinity," *Environmental and Experimental Botany*, Vol. 65, no.2-3, pp. 270-281.

Hernandez, J., A., A. Jimenez., A., Mullineaux., P., and F. Sevilla. F., (2000). "Tolerance of Pea (*Pisum sativum* L.) to long-term salt stress is associated with induction of antioxidant defenses," *Plant, Cell and Environment*, Vol. 23, No. 8, pp. 853-862.

Logan, B., A., Kornyeyev, D., Hardison, J. and Holaday, A.S. (2006). "The role of antioxidant enzymes in photoprotection," *Photosynthesis Research*, 88, pp. 119-132.

Hu, W.H., Song, X.S., Shi, K., Xia, X.J., Zhou, Y. H., and J. Q. Yu, J.Q. (2008). "Changes in electron transport, Superoxide dismutase and ascorbate peroxidase isoenzymes in chloroplasts and mitochondria of cucumber leaves as influenced by chilling," "*Photosynthetica*, 46, pp.581-588.

Prasad, T. K. (1997). "Role of Catalase in inducing chilling tolerance in pre-emergent maize seedlings," *Plant Physiology*, 114, PP. 1369-1376.

Freyer, M.J., J.R., Andrews, K. Oxborough, D.A. Blowers, and N.R. Baker (1998). "Relatioships between (02 assimilation, Photosynthetic electron transport, and active 02 metabolism in leaves of maize in the field during periods of low temperature," *Plant Physiology*, Vol.116, no.2.pp.571-580, 1998.

Dunand, F. V., Epron, D., Sosse B. A., and Badot, P.M. (2002). "Effects of Copper on growth and on photosynthesis of mature and expanding leaves in cucumber plants," *Plant Science*, 163, pp. 53-58.

Shah, K., Kumar, R.G., Verma, S. and Dubey, R.S. (2001). "Effects of cadmium on lipid peroxidation, Superoxide anion generation and activities of antioxidant enzymes in growth rice seedlings," *Plant Science*, 161, pp. 1135-1144.

Maheshwari, R. and Dubey, R.S. (2009). "Nickle-induced oxidative stress and lowers the role of antioxidant defence in rice seedlings," *Plant Growth Regulation*, 59 pp. 37-49.

Srivastava, S. and Dubey, R.S., (2011). "Manganese-excess induces oxidative stress, lowers the pool of antioxidants and elevates activities of key antioxidative enzymes in rice seedlings," *Plant Growth Regulation*, pp. 1-16.

Sharma, P., and Dubey, R.S. (2007). "Involvement of oxidative stress and role of antioxidative defense system in growing rice seedling exposed to toxic concentration of aluminium," *Plant Cell Reports*, 26, pp 2027-2038.

Verma, S. and Dubey, R.S. (2003). "Lead toxicity induces lipid peroxidation and alters the activities of antioxidant enzymes in growing rice plants, "*Plant Science*", 164, pp 645-655.

Cakmak, I. (2000). Possible roles of Zinc in protecting plant cells from damage by reactive oxygen species. *New Phytology,* 146,185-205.

Cakmak, I. and H. Marschner (1988). Increase in membrane permeability and exudation in roots of Zinc deficient plants. *Journal of Plant Physiology,* 132, 356-361.

Marschner, H. (1995). Mineral nutrition of higher plants. 2nd Edition. Academic Press, *San Diego*. 889 pp.

Cohu, C.M. and Pilon, M. (2007). Regulation of Superoxide dismutase expression by copper availability. *Physiol. Plant.* 129, 747-755.

Meharg, A.A., Macnair, M.R. (1994). Relationship between plant phosphorus status and the kinetics of arsenate influx in clones of *Deschampsia cespitosa* (L.) Beauv that differ in their tolerance to arsenate. *Plant and Soil* 162: 99–106.

Yu tong Wang, Kun Wang and Xin-quing Shao (2010). Manganese Delays the Senescence induced drought in Perennial rye grass (*Lolium Perenne* L).

Tewari, R.K., Kumar, P. and Sharma, P.N. (2008). Morphology and Physiology of Zinc-stressed mulberry plants. *J. Plant Nutr. Soil Sci*, 171, 286-294.

Sharma, P.N., Tripathi, A. and Bisht, S.S. (1994). Effect of Fe on transpiration& photosynthesis in wheat (*Triticum aestivum* L. cv. up 115) grown in sand culture. *Indian J Exp. Biol.* 32,736-759.

Sharma, P.N., Tripathi, A. and Bisht, S.S. (1995). Zinc requirement for stomatal opening in cauliflower. *Plant Physiol.* 107, 751-756.

Milone, M.T., Sgherri, C., Clijsters, H. and Navarri, Izzo F. (2003). Antioxidant responses of wheat treated with realistic concentration of cadmium. *Environ Exp Bot*, 50: 265-276.

Sandalio, L.M., Dalurzo, H.C., Gome, Z. M., Romero, Puertas M.C. and del, Rio L.A. (2001). Cadmium induced Changes in the growth and Oxidative metabolism of pea plants. *J. Exp Bot* 52: 2115-2126.

Schickler, H. and Caspi, H. (1999). Response of antioxidative enzymes to nickel and Cadmium stress in hyperaccumulator plants of the genus Alyssum. *Physiol*: 105:39-44.

Rao, M. V., G. Paliyath, and D.P. Ormrod (1996). "Ultraviolet-B- and ozone- induced biochemical changes in antioxidant enzymes of Arabidopsis thaliana," *Plant Physiology*, Vol. 110, No. 1, pp. 125-136.

Bolwell, G.P. (1996). The origin of the oxidative burst in plants. *Biochemical Society Transactions* 24: 438–442.

Bolwell, G.P. (1996). The origin of the oxidative burst in plants. *Biochemical Society Transactions* 24: 438–442.

Bolwell, G.P. (1996). The origin of the oxidative burst in plants. *Biochemical Society Transactions*, 24. 438-442.

Sairam, R.K., Kumutha, D., Lekshmy, S. and Viswanathan, C. (2011). Expression of antioxidant defense genes in mungbean (*Vigna radiata* L.) roots under water logging is associated with hypoxia tolerance, *Acta Physiol. Plant*, 33, 735–744.

Hammond-Kossack, K.E. and Jones, J.D.G. (1996). Resistance gene-dependent plant defense responses. *Plant Cell* 8:1773-1791.

Ghosh, S., Mitra, S. and Paul, A. (2014). Physiological studies of sodium chloride on mung bean (Vigna radiate L. wilczek) and its possible recovery with spermine and *Gibberellic* acid, Hindwai Publishing corporation, *The Scientific World Journal*, Article ID858016.

Randi H. Gottfredsen, Ulrike G. Larsen, Jan. J. Enghild and Steen V. Petersen (2013). Hydrogen peroxide induce modifications of human extracellular superoxide dismutase that results in enzyme inhibition; *Redox Biology,* 1, 24-31.

Yim, M.B., P.B. Chock, and E.R. Stadtman (1990). Copper, Zinc superoxide dismutasecatalyzes hydroxyl radical production from hydrozen peroxide: *Proc. Natl. Acad. Sci*: USA, Vol. 87, PP5006-5010, July 1990, *Biochemistry*.

Mota de Freitas, D. and Valentine, J.S. (1984). *Bilochemistry,* 23, 2079-2082.

Hodgson, E.K. and Fridovich, I. (1975). *Biochemistry,* 14, 5294-5299.

Halliwell, B. (1987). Oxidative damage, Lipid Peroxidation and antioxidant Protection in Chloroplast. *Chem. Phys. Lipids,* 44, 327-340.

Cadenas, E. (1989). Biochemistry of Oxygen toxicity. *Annu-Rev. Biochem*. 58, 79-110.

Fridovich, I. (1989). Superoxide dismutases. An adaptation to a Paramagnetic gas. *J.Biol.Chem*. 264, 7761-7764.

Sang, E.W.T., Bowler, C., Herouart, D., Wim Van Camp, W., Villarroel, R., Genetello, C., Van Montagu, M. and Inze, D. (1991). Differential Regulation of Superoxide dismutases in Plants Exposed to Environmental Stress: *The Plant Cell*, Vol. 3, 783-792, August 1991© 1991 American Society of Plant Physiologists.

Rojo, I. A., Salinas, M., Martin, D., Perona, R. and Cuadrado, A. (2004). Regulatio of Cu/Zn Superoxide Dismutase Expression Via the Phosphatidylinositol 3 kinase/Akt Pathway and Nuclear Factor-KB. *The Journal of Neurosciences*, 24 (33), 7324-7334.

Puget, K. and Michelson, A.M. (1974). Iron containing Superoxide dismutases from luminous bacteria. *Biochimie*, 56, 1255-1267.

Mac Millan Crow L.A., Crow, J. P. and Thompson, J.A. (1998). Peroxynitrite-mediated inactivation of manganese superoxide dismutase involves nitration and oxidation of critical tyrosine residues. *Biochemistry,* 37:1613-1622.

Fisher, C.L., Cabelli, D.E., Tainer, J.A., Hallewell, R.A. and Getzoff, E.D. (1994). The role of arginine143 in the electrostatics and mechanism of Cu,Zn superoxide dismutase: computational and experimental evaluation by mutational analysis, Proteins 19,24–34.

Rosen, D.R., Siddique, T., Patterson, D., Figlewicz,, D.A., Sapp, P., Hentati, A., Donaldson, D., Goto, J., O'Regan, J.P., Deng, H.X. et al., (1993). Mutations in Cu/Zn superoxide dismutase gene are associated with familial amyotrophic lateral sclerosis, Nature 362, 59–62.

Allen, D.J., Mckee, I. F., Farage P. K. and Baker, N.R. (1997). Analysis of limitations to CO_2 assimilation on exposure of leaves of two *Brassica napus* cultivars to UV-B," *Plant, Cell and Environment*, 20, 633–640.

Gunes, A., Pilbeam, D. J. and Inal, A. (2009). Effect of arsenic-phosphorus interaction on arsenic induced oxidative stress in chickpea plants, Plant Soil 314:211-220.

Archibald, F. S. and Fridovich, I. (1982). *Arch. Biochem. Biophys*. 214, 452-463.

Corresponding author: Dr. Kiran Vati, Department of Biochemistry, University of Lucknow, U.P., India
Email id: kiran27aug@gmail.com