

# Redox State as a Central Regulator of Plant-Cell Stress Responses

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Francisco J. Corpas  
Editors

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 Springer

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# Preface

It is known that reactive oxygen species (ROS) are the by-products of aerobic breakdown and are inescapably formed by a number of metabolic pathways and electron transport chains. ROS are partially condensed form of molecular oxygen and normally result from the transfer of electrons to  $O_2$  to form, in a succession of univalent reductions, superoxide radical ( $O_2^{\cdot-}$ ), hydrogen peroxide ( $H_2O_2$ ), and hydroxyl radical ( $\cdot OH$ ), respectively, or through an electron-independent energy transfer till an excited form of oxygen (singlet oxygen) (Gupta et al. 2016; Halliwell and Gutteridge 2015). Redox signal transduction is a complete feature of aerobic life enriched through evolution to balance evidence from metabolism and the environment. Like all other aerobic creatures, plants maintain most cytosolic thiols in the reduced ( $-SH$ ) state because of the low thioldisulfide redox potential imposed by millimolar amount of the thiol buffer including glutathione.

Plants have developed cellular tactics where the endogenous content of antioxidant enzymes deliver them with amplified defense against harmful effects of oxidative stress encouraged by heavy metal and other stress sources (Palma et al. 2013). Stress-induced upsurges in ROS level can cause different degree of oxidation of cell components and a gross change in the redox status. Plant cells generally cope very well with high rates of generation of superoxide,  $H_2O_2$ , and even singlet oxygen. When the increment of ROS in plant cells quickly augments and the scavenging systems of ROS do not operate appropriately, a condition of oxidative stress and oxidative injury happens (Gupta et al. 2015). In plants, chloroplast is the most important among the organelles in respect of ROS generation as  $O_2$  is constantly provided through the water autolysis and freely available inside the organelle (Gupta et al. 2015). In plant cells, compartmentalization of ROS production in the different organelles includes chloroplasts, mitochondria, or peroxisomes, and they also have a complex battery of antioxidant enzymes usually close to the site of ROS production (Corpas et al. 2015). Plant cells also contain a series of ROS-scavenging non-enzymatic antioxidants such as ascorbic acid, glutathione (GSH), and carotenoids, as well as a set of enzymes such as superoxide dismutase (SOD), catalase, glutathione peroxidase (GPX), peroxiredoxin (Prx), and the

ascorbate–glutathione cycle (Corpas et al. 2015). The total pool of redox-active complexes which are found in a cell in reduced and oxidized forms generates cellular redox buffers where NAD(P)H/NAD(P)<sup>+</sup>, ascorbate/dehydroascorbate (AsA/DHA), glutathione/glutathione disulfide (GSH/GSSG), and reduced thioredoxin/oxidized thioredoxin ( $\text{Trx}_{\text{red}}/\text{Trx}_{\text{ox}}$ ) are the main pairs. AsA and GSH are major constituents of the soluble redox shielding system, and they contribute pointedly to the redox environment of a cell. AsA cooperates tightly with GSH ( $\gamma$ -Glu-Cys-Gly) in the Foyer–Halliwell–Asada cycle (ascorbate–glutathione cycle), involving three codependent redox couples: AsA/DHA, GSH/GSSG, and NAD(P)H/NAD(P)<sup>+</sup>. It undertakes subsequent reduction/oxidation reactions catalyzed by ascorbate peroxidase (APX), monodehydroascorbate reductase (MDAR), dehydroascorbate reductase (DHAR), and glutathione reductase (GR) that is universally responsible for H<sub>2</sub>O<sub>2</sub> sifting and keeping AsA and GSH in the reduced state at the outflow of NADPH, this cycle being situated in all cellular partitions in which ROS detoxification is required.

One of the major consequences of stresses in plant cells is the enhanced generation of ROS which usually damage the cellular components such as membranes, nucleic acids, proteins, chloroplast pigments, and alteration in enzymatic and non-enzymatic antioxidants. The molecular mechanisms of signal transduction corridors in higher plant cells are vital for processes such as hormone and light sensitivity, growth, development, stress resistance, and nutrient uptake from soil and water (Gupta et al. 2013).

It is really great achievement for the plant biotechnologists who are working for years to know how redox state handled by plants. This edited volume will provide the recent advancements and overview to the plant scientists who are actively involved in redox signaling states and also a key player for cellular tolerance in plant cells under different stresses (biotic and abiotic). Other key features of this book are cellular redox homeostasis as central modulator, redox homeostasis and reactive oxygen species, redox balance in chloroplasts and in mitochondria, and oxidative stress and its role in peroxisome homeostasis. Some chapters are also focusing on glutathione-related enzyme system and metabolism under metal(ed) stress. Abiotic stress-induced redox changes and programmed cell death are also addressed in the edition. In summary, the information compiled in this volume will bring depth knowledge and current achievements in the field of redox state chemistry in plant cell.

Dr. Dharmendra K. Gupta, Prof. José M. Palma, and Dr. Francisco J. Corpas individually thank all authors for contributing their valuable time, knowledge, and enthusiasm to bring this book into in the current shape.

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# Contents

<b>1 Cellular Redox Homeostasis as Central Modulator in Plant Stress Response</b> . . . . .	1
C. Paciolla, A. Paradiso and M.C. de Pinto	
<b>2 Plant Cell Redox Homeostasis and Reactive Oxygen Species</b> . . . . .	25
A. Trchounian, M. Petrosyan and N. Sahakyan	
<b>3 Redox Balance in Chloroplasts as a Modulator of Environmental Stress Responses: The Role of Ascorbate Peroxidase and Nudix Hydrolase in <i>Arabidopsis</i></b> . . . . .	51
T. Ishikawa, T. Maruta, T. Ogawa, K. Yoshimura and S. Shigeoka	
<b>4 Physiological Processes Contributing to the Synthesis of Ascorbic Acid in Plants</b> . . . . .	71
C.G. Bartoli, M.E. Senn and G.E. Gergoff Grozeff	
<b>5 Redox State in Plant Mitochondria and its Role in Stress Tolerance</b> . . . . .	93
N.V. Bykova and A.U. Igamberdiev	
<b>6 Oxidative Stress and its Role in Peroxisome Homeostasis in Plants</b> . . . . .	117
T. Su, Q. Shao, P. Wang and C. Ma	
<b>7 Glutathione-Related Enzyme System: Glutathione Reductase (GR), Glutathione Transferases (GSTs) and Glutathione Peroxidases (GPXs)</b> . . . . .	137
J. Csiszár, E. Horváth, K. Bela and Á. Gallé	
<b>8 Glutathione Metabolism in Plants Under Metal and Metalloid Stress and its Impact on the Cellular Redox Homeostasis</b> . . . . .	159
Luis E. Hernández, A. González, A. Navazas, Á. Barón-Sola, F. Martínez, A. Cuyper and C. Ortega-Villasante	

<b>9</b>	<b>Glutathione and Related Enzymes in Response to Abiotic Stress</b> . . . . .	183
	I. Štolfa, D. Špoljarić Maronić, T. Žuna Pfeiffer and Z. Lončarić	
<b>10</b>	<b>The Function of Cellular Redox Homeostasis and Reactive Oxygen Species (ROS) in Plants Tolerance to Abiotic Stresses</b> . . . .	213
	Qinghua Shi and Biao Gong	
<b>11</b>	<b>Abiotic Stress-Induced Redox Changes and Programmed Cell Death in Plants—A Path to Survival or Death?</b> . . . . .	233
	S.R. Kumar, G. Mohanapriya and R. Sathishkumar	
<b>12</b>	<b>The Role of ROS and Redox Signaling During the Initial Cellular Response to Abiotic Stress</b> . . . . .	253
	Jos H.M. Schippers and R. Schmidt	
<b>13</b>	<b>The Cadmium-Binding Thioredoxin <i>O</i> Acts as an Upstream Regulator of the Redox Plant Homeostasis</b> . . . . .	275
	Moêz Smiri, Sami Boussami, Takwa Missaoui and Amor Hafiane	
<b>14</b>	<b>Arsenic Tolerance in Plants: Cellular Maneuvering Through Sulfur Metabolites</b> . . . . .	297
	D. Talukdar	
<b>15</b>	<b>Regulation of Stomatal Responses to Abiotic and Biotic Stresses by Redox State</b> . . . . .	331
	Y. Murata, S. Munemasa and I.C. Mori	
<b>16</b>	<b>The Antioxidant Power of Arginine/Nitric Oxide Attenuates Damage Induced by Methyl Viologen Herbicides in Plant Cells</b> . . . . .	349
	N. Correa-Aragunde, P. Negri, F. Del Castello, N. Foresi, J.C. Polacco and L. Lamattina	
<b>17</b>	<b>Protein S-Nitrosylation and S-Glutathionylation as Regulators of Redox Homeostasis During Abiotic Stress Response</b> . . . . .	365
	J.C. Begara-Morales, B. Sánchez-Calvo, M. Chaki, R. Valderrama, C. Mata-Pérez, F.J. Corpas and J.B. Barroso	



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