

Signaling and Communication in Plants

P. Vidhyasekaran



# PAMP Signals in Plant Innate Immunity

Signal Perception and Transduction

 Springer

# Signaling and Communication in Plants

## Series Editor

František Baluška

Department of Plant Cell Biology, IZMB, University of Bonn, Kirschallee 1, D-53115  
Bonn, Germany

For further volumes:

<http://www.springer.com/series/8094>



P. Vidhyasekaran

# PAMP Signals in Plant Innate Immunity

Signal Perception and Transduction

 Springer

P. Vidhyasekaran  
Plant Pathology  
Tamil Nadu Agricultural University  
Coimbatore, Tamil Nadu, India

ISSN 1867-9048  
ISBN 978-94-007-7425-4  
DOI 10.1007/978-94-007-7426-1  
Springer Dordrecht Heidelberg New York London

ISSN 1867-9056 (electronic)  
ISBN 978-94-007-7426-1 (eBook)

Library of Congress Control Number: 2013952933

© Springer Science+Business Media Dordrecht 2014

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed. Exempted from this legal reservation are brief excerpts in connection with reviews or scholarly analysis or material supplied specifically for the purpose of being entered and executed on a computer system, for exclusive use by the purchaser of the work. Duplication of this publication or parts thereof is permitted only under the provisions of the Copyright Law of the Publisher's location, in its current version, and permission for use must always be obtained from Springer. Permissions for use may be obtained through RightsLink at the Copyright Clearance Center. Violations are liable to prosecution under the respective Copyright Law.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

While the advice and information in this book are believed to be true and accurate at the date of publication, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Springer is part of Springer Science+Business Media ([www.springer.com](http://www.springer.com))

# Contents

<b>1</b>	<b>Introduction</b> .....	1
1.1	Classical PAMPs .....	2
1.2	Plant Pattern Recognition Receptors (PRRs).....	3
1.3	Second Messengers in PAMP Signal Transduction .....	4
1.4	Plant Hormone Signals in Plant Immune Signaling System.....	6
1.5	War Between Host Plants and Pathogens and the Winner Is ----- ? .....	7
	References.....	8
<b>2</b>	<b>PAMP Signaling in Plant Innate Immunity</b> .....	17
2.1	Classical PAMPs as Alarm Signals .....	18
2.2	Effector-Like PAMPs .....	19
2.3	PAMPs Found Within Effectors .....	21
2.4	Toxins Acting as PAMPs.....	22
2.5	PAMP-Induced HAMPs (DAMPs/MIMPs/PAMP Amplifiers/Endogenous Elicitors).....	23
2.6	Bacterial PAMPs .....	24
2.6.1	PAMPs from Various Bacterial Structures .....	24
2.6.2	PAMPs Detected in Flagella .....	25
2.6.3	Lipopolysaccharide Components Acting as PAMPs.....	27
2.6.4	Muropeptides and Sugar Backbone Structure PAMPs in Peptidoglycans .....	29
2.6.5	elf18 PAMP Epitope in Elongation Factor Tu (EF-Tu).....	31
2.6.6	Cold-Shock Protein (CSP22) as PAMP .....	31
2.6.7	Harpins with PAMP and Protein Secretion Structure .....	31
2.6.8	Ax21 Sulfated Protein as a PAMP .....	33
2.6.9	Rhamnolipids as PAMPs.....	34
2.6.10	Superoxide Dismutase (SOD) as a PAMP .....	35
2.6.11	Bacterial DNA as PAMP .....	35
2.6.12	NEP1-Like Proteins as Bacterial PAMPs.....	35

2.7	Fungal PAMPs.....	35
2.7.1	Chitooligosaccharides as PAMPs.....	35
2.7.2	$\beta$ -Glucan PAMPs Isolated from Fungal Cell Wall .....	37
2.7.3	Fungal Cell Wall Ergosterol PAMP.....	37
2.7.4	EIX Protein as PAMP.....	37
2.7.5	Cerebrosides as PAMPs .....	37
2.7.6	NEP1-Like Proteins as PAMPs .....	38
2.8	Oomycete PAMPs .....	38
2.8.1	PEP-13 as an Oomycete PAMP .....	38
2.8.2	Elicitins as Oomycete PAMPs.....	39
2.8.3	Oomycete Cell Wall Glucans as PAMPs.....	40
2.8.4	Cell Wall Glycoprotein CBEL with CBD Motifs as PAMPs.....	40
2.8.5	NEP1-Like Proteins as Oomycete PAMPs.....	41
2.9	Viral Elicitors .....	41
2.9.1	Several Viral Components Show Elicitor Function.....	41
2.9.2	Viral Double-Stranded RNAs May Be PAMPs.....	42
2.10	Host-Associated Molecular Patterns as Endogenous Elicitors .....	42
2.10.1	Oligogalacturonides as HAMPs.....	42
2.10.2	Cellodextrins as HAMPs.....	44
2.10.3	<i>Arabidopsis</i> AtPep Peptides .....	44
2.10.4	Soybean GmSubPep Peptide.....	45
2.10.5	Maize <i>ZmPep1</i> Peptide.....	45
2.11	Plant Pattern Recognition Receptors.....	46
2.11.1	Structure of PRRs.....	46
2.11.2	FLS2, the PRR for the PAMP Flagellin ( <i>flg22</i> ) .....	47
2.11.3	EFR, the PRR for the PAMP EF-Tu.....	48
2.11.4	XA21, the PRR for the PAMP Ax21 .....	49
2.11.5	CERK1, the PRR for the PAMP Chitin.....	49
2.11.6	CEBiP, the Second PRR for the PAMP Chitin.....	51
2.11.7	NbLRK1, the PRR for the PAMP INF1 Elicitin .....	51
2.11.8	NgRLK1, the PRR for the PAMP Elicitin Capsicein.....	52
2.11.9	LeEIX1 and LeEIX2, the PRRs for the PAMP EIX Proteins .....	53
2.11.10	Glucan—Binding Proteins .....	54
2.11.11	Mannose-Binding Lectin Receptors.....	54
2.11.12	RLK Receptor for the PAMP Lipopolysaccharides .....	55
2.11.13	Peptidoglycan-Binding Proteins.....	55
2.11.14	Pep Receptors for the HAMPs Pep Proteins .....	55
2.11.15	WAK1 as a Receptor for the HAMP Oligogalacturonides .....	56
2.12	Transmembrane Proteins Interacting with PRRs in PAMP-PRR Signaling Complex .....	57
2.12.1	Signaling Adapters/Amplifiers in PAMP-PRR Signaling Complex.....	57

2.12.2	BAK1 Is Required for Proper Functionality of PRRs .....	58
2.12.3	BAK1 Acts Downstream of PRR Perception by PAMP .....	58
2.12.4	BAK1 Functions as an Adapter or Signaling Partner for Regulation of PRRs.....	59
2.12.5	Rapid Heteromerization and Phosphorylation of PRRs and Their Associated Kinase BAK1 .....	59
2.12.6	BIK1 .....	60
2.12.7	PBL Proteins .....	61
2.12.8	ERECTA Protein .....	62
2.12.9	AtPHOS32, AtPHOS34, and AtPHOS43 Proteins.....	63
2.12.10	BIR1 .....	63
2.13	PAMP Triggers Increased Transcription of PRR Gene and Accumulation of PRR Protein .....	64
2.13.1	PAMPs Activate Expression of Genes Encoding PRRs .....	64
2.13.2	PRRs Are Activated by Widely Varying PAMPs.....	64
2.13.3	PRRs May Act Additively in Perception of PAMP .....	65
2.13.4	PRRs Bind with PAMPs for Their Activation.....	65
2.13.5	Ethylene Regulates Transcription of PRRs on PAMP Perception .....	65
2.14	PAMPs Induce Phosphorylation of PRRs .....	66
2.14.1	PAMP-Induced Autophosphorylation of PRRs.....	66
2.14.2	PRR Autophosphorylation Is Essential for PRR to Bind to Its Negative Regulators .....	67
2.15	Negative Regulation of PRR Signaling.....	68
2.16	Translocation of PRRs from Plasma Membrane to Endocytic Compartments .....	69
2.16.1	Endocytosis of PRRs.....	69
2.16.2	Clathrin-Mediated Endocytosis.....	70
2.16.3	Ubiquitin-Proteasome System May Be Involved in PRR Endocytosis.....	71
2.16.4	Phosphorylation of PRR May Be Involved in PRR Endocytosis.....	71
2.16.5	Involvement of EHD in Endocytosis.....	71
2.16.6	What Is the Role for Endocytosis in PAMP-PRR Signaling? .....	73
2.17	ER-QC (for <u>E</u> ndoplasmic <u>R</u> eticulum <u>Q</u> uality <u>C</u> ontrol) Pathways in Biogenesis of PRRs .....	73
2.17.1	ERQC Mechanisms Monitor Protein Folding in ER .....	73
2.17.2	Calnexin (CNX)/Calreticulin (CRT)/UGGT System .....	74
2.17.3	BiP/ERdj/SDF2 System .....	74



2.17.4	Function of ERD2 in ER-QC .....	75
2.17.5	ER Quality Control Components Required for Biogenesis of the Pattern Recognition Receptor EFR .....	76
2.17.6	ER Quality Control Components Required for Biogenesis of the PRR FLS2.....	77
2.17.7	Role of BiP3 in ERQC of the Rice PRR XA21 .....	78
2.18	<i>N</i> -Glycosylation of PRRs.....	79
2.18.1	Glycosylation of PRRs Is Required for Binding PAMP Ligands.....	79
2.18.2	<i>N</i> -Glycosylation Is Required for Transport of PRRs from Endoplasmic Reticulum to Plasma Membrane.....	79
2.19	Significance of PRRs in Innate Immunity.....	80
2.20	PAMPs-Induced Early Signaling Events Downstream of PRRs .....	80
2.20.1	PAMPs Trigger Complex Networks of Signaling Pathways.....	80
2.20.2	Ca <sup>2+</sup> Signaling System.....	82
2.20.3	H <sup>+</sup> Fluxes and Extracellular Alkalinization.....	84
2.20.4	G-Proteins .....	85
2.20.5	ROS Signaling System .....	85
2.20.6	Nitric Oxide Signaling System.....	86
2.20.7	Mitogen-Activated Protein Kinase (MAPK) Cascades.....	87
2.20.8	Salicylate Signaling System.....	89
2.20.9	Jasmonate Signaling System.....	91
2.20.10	Ethylene Signaling System .....	92
2.20.11	Abscisic Acid Signaling System .....	93
2.20.12	PAMP-Induced Expression of Transcription Factors .....	95
2.20.13	Hierarchy of PAMP-Induced Signaling Systems .....	96
2.21	Different PAMPs and HAMPs May Induce Similar Early Signaling Systems .....	96
2.22	Magnitude and Timing of Expression of Early Signaling Systems May Vary Depending on Specific PAMPs.....	97
2.23	PAMPs May Differ in Eliciting Various Defense Responses.....	98
2.24	Synergism and Antagonism in Induction of Plant Immune Responses by PAMPs/HAMPs .....	100
2.24.1	Multiple PAMPs May Be Required to Activate the Complex Defense Signaling Systems .....	100
2.24.2	Different PAMPs May Act Synergistically .....	101
2.24.3	Some PAMPs May Show Antagonistic Effect in Activating Defense Responses .....	101

2.25 Amount of PAMP/HAMP Determines the Intensity of Expression of Defense Signaling Genes..... 102

2.26 Amount of PAMP Available in the Infection Court May Determine the Level of Induction of Immune Responses..... 103

2.27 PAMPs May Trigger Different Signaling Systems ..... 103

    2.27.1 Some PAMPs May Activate Only Specific Signaling Systems..... 103

    2.27.2 Some PAMPs May Activate Multiple Hormone Signaling Systems..... 105

2.28 PAMPs May Function Differently in Different Plants..... 105

2.29 Specificity of PAMPs in Triggering Immune Responses in Plants..... 105

2.30 Role of PAMPs and Effectors in Activation of Plant Innate Immune Responses..... 106

2.31 Effectors May Suppress PAMP-Triggered Immunity ..... 107

    2.31.1 Inhibition of PAMP-Triggered Immunity ..... 107

    2.31.2 Effectors May Degrade PRRs ..... 107

    2.31.3 Effectors May Bind the Receptor Kinase PRRs to Block PAMP-Triggered Immunity ..... 108

    2.31.4 Effectors May Prevent Interaction of Co-receptor BAK1 with PAMPs ..... 108

    2.31.5 Effectors May Target the Receptor-Like Cytoplasmic Kinases BIK1 and PBL1..... 109

    2.31.6 Effectors May Inhibit Autophosphorylation of PRRs ..... 110

2.32 PAMP-Induced Small RNA-Mediated RNA Silencing..... 110

    2.32.1 RNA Silencing Is an Immune System in Plants..... 110

    2.32.2 Flg22 Triggers Accumulation of miRNAs, Which Cleave and Down-Regulate Auxin Signaling Genes ..... 112

    2.32.3 Flg22 Suppresses Accumulation of Some miRNAs, Which Have a Negative Role in PAMP-Triggered Innate Immunity ..... 113

    2.32.4 Importance of miRNA-Directed RNA Silencing Pathway in PAMP-Triggered Immunity (PTI)..... 114

    2.32.5 Small RNAs May Also Be Involved in Effector-Triggered Immunity (ETI)..... 114

References..... 115

**3 G-Proteins as Molecular Switches in Signal Transduction..... 163**

    3.1 G-Proteins Switch on Plant Innate Immunity Signaling Systems ..... 164

    3.2 Heterotrimeric G-Protein Signaling..... 166

3.2.1	Subunits of Heterotrimeric G-Proteins.....	166
3.2.2	G-Protein-Coupled Receptor.....	167
3.2.3	G $\gamma$ Protein Triggers Plasma Membrane Targeting of G $\beta\gamma$ to Trigger Immune Responses.....	167
3.2.4	Activation of G-Protein Heterotrimer in Elementary G-Protein Signaling.....	168
3.3	Small G-Proteins Signaling.....	168
3.4	Heterotrimeric G-Protein G $\alpha$ May Act Upstream of Small G-Protein in Immune Signaling.....	169
3.5	Different G-Protein Subunits in Heterotrimeric G-Proteins Play Distinct Roles in Plant Innate Immunity.....	170
3.6	Small G-Proteins Activate Plant Innate Immunity.....	171
3.7	Small G-Proteins May Be Involved in Susceptible Interactions.....	172
3.8	RAR1-SGT1-HSP90-HSP70 Molecular Chaperone Complex: A Core Modulator of Small G-Protein-Triggered Plant Innate Immunity.....	173
3.9	PAMP Signal May Convert the G-Proteins from Their Inactive State to Their Active State to Trigger Immune Responses.....	175
3.10	PAMP-Activated G-Proteins Switch on Calcium Ion-Mediated Immune Signaling System.....	176
3.10.1	G-Proteins Activate InsP3-Gated Channels.....	176
3.10.2	G-Proteins Stimulate H <sup>+</sup> -ATPase and Regulate Ca <sup>2+</sup> Channel.....	177
3.10.3	G-Proteins Activate Ca <sup>2+</sup> Signaling System Through Modulation of Phosphorylation/ Dephosphorylation System.....	177
3.11	G-Proteins May Trigger Efflux of Vacuolar Protons into Cytoplasm to Activate pH-Dependent Signaling Pathway.....	178
3.12	G-Proteins Switch on ROS Signaling System.....	178
3.12.1	G-Proteins Trigger Generation of ROS to Induce Immune Responses.....	178
3.12.2	G-Proteins Switch on Ca <sup>2+</sup> Influx – RBOH-Mediated ROS Signaling Pathway.....	180
3.12.3	Interplay Between ROP, RBOH, CDPK, Ca <sup>2+</sup> <sub>[cyt]</sub> , and ROS in G-Protein-Mediated ROS Signaling.....	180
3.12.4	Phosphatidic Acid Activates G-Protein-Mediated Pathway of ROS Generation.....	180
3.12.5	Small G-Proteins May Trigger Accumulation of ROS by Suppressing the Action of ROS Scavengers.....	182
3.12.6	G-Proteins Act as Redox Regulators in ROS Signaling.....	183

3.13 G-Proteins Activate Nitric Oxide Signaling System ..... 183

3.14 Close Relationship Between G-Proteins and MAPKs  
in Signal Transduction..... 184

3.15 G-Proteins Induce Biosynthesis of Polyamines  
Which Act as Second Messengers Triggering  
Early Signaling Events ..... 185

3.16 G-Proteins Modulate Salicylic Acid Signaling Pathway..... 187

3.17 G-Proteins Trigger Ethylene Signaling Pathway ..... 188

3.18 G-Proteins Switch on Jasmonate Signaling System..... 189

3.19 G-Proteins Switch on Abscisic Acid Signaling System ..... 190

    3.19.1 G-Proteins May Be Involved in Abscisic Acid  
    Biosynthesis ..... 190

    3.19.2 G-Proteins May Act as Abscisic Acid Receptors ..... 191

    3.19.3 G-Proteins May Regulate Inward K<sup>+</sup> Channels  
    and Slow Anion Channels Activated by ABA ..... 191

    3.19.4 ABA Increases Expression of Genes Encoding  
    Heterotrimeric G-Proteins..... 192

    3.19.5 G-Protein May Play a Role in ABA Signaling  
    Between ABA Reception and ROS Production ..... 192

    3.19.6 G-Proteins May Be Involved in Modulation  
    of ABA-Induced Stomatal Closure Immune Response.... 192

3.20 G-Proteins May Participate in Gibberellic Acid Signaling ..... 193

3.21 G-Proteins Participate in Brassinosteroid Signaling ..... 193

3.22 Interplay Between G-Proteins and Auxin  
Signaling Systems ..... 194

3.23 G-Proteins Activate Defense-Related Enzymes ..... 194

References ..... 195

**4 Calcium Ion Signaling System: Calcium Signatures  
and Sensors ..... 207**

4.1 Calcium Signature in Plant Immune Signal  
Transduction System ..... 208

4.2 Upstream Events Leading to Activation  
of Ca<sup>2+</sup> – Permeable Channels..... 209

    4.2.1 PAMP-Triggered Ca<sup>2+</sup> Influx and Elevations  
    in Cytosolic Free Calcium..... 209

    4.2.2 PAMP – Activated G-Proteins May Initiate  
    Ca<sup>2+</sup> Influx ..... 210

    4.2.3 Reactive Oxygen Species (ROS) Regulate  
    Ca<sup>2+</sup> Influx ..... 211

4.3 Ca<sup>2+</sup> Influx Channels in Plant Cell Plasma Membrane ..... 212

    4.3.1 Voltage-Gated Ca<sup>2+</sup>-Permeable Channels..... 212

    4.3.2 Cyclic Nucleotide-Gated Ion Channels (CNGCs) ..... 214

    4.3.3 Glutamate Receptor-Like Ion Channels ..... 216

    4.3.4 Annexins as Calcium Transporters..... 217

4.4	Ca <sup>2+</sup> Release Channels Involved in Releasing Stored Ca <sup>2+</sup> in Vacuole and Endoplasmic Reticulum into Cytosol .....	218
4.4.1	Inositol 1,4,5-Trisphosphate(InsP3)-Activated Ca <sup>2+</sup> Channel .....	218
4.4.2	Cyclic Adenosine 5'-Diphospho-Ribose (cADPR) Gated Channels .....	219
4.4.3	Slowly Acting Vacuolar (SV) Channel.....	219
4.4.4	NAADP-Activated Ca <sup>2+</sup> Efflux Channel.....	219
4.5	Ca <sup>2+</sup> Efflux from Cytosol to Vacuole and Endoplasmic Reticulum (ER) .....	220
4.5.1	H <sup>+</sup> /Ca <sup>2+</sup> Antiport System .....	220
4.5.2	Calcium Ion Pumps .....	220
4.6	Plasma Membrane H <sup>+</sup> -ATPases in Ca <sup>2+</sup> Signaling .....	221
4.7	Anion Channels in Ca <sup>2+</sup> Influx and Increase in [Ca <sup>2+</sup> ] <sub>cyt</sub> .....	223
4.8	K <sup>+</sup> Channels in Ca <sup>2+</sup> Influx .....	224
4.9	K <sup>+</sup> /H <sup>+</sup> Exchange Response in Ca <sup>2+</sup> Signaling System.....	225
4.10	PAMPs and DAMPs May Trigger Calcium Ion Influx/Efflux Through Different Ca <sup>2+</sup> Channels.....	225
4.11	Induction of Increases in Concentration, Oscillations and Waves in Cytoplasmic Calcium Ion ([Ca <sup>2+</sup> ] <sub>cyt</sub> ) .....	226
4.12	Ca <sup>2+</sup> Sensors in Ca <sup>2+</sup> Signal Transduction .....	227
4.13	Calmodulins as Ca <sup>2+</sup> Sensors.....	228
4.14	Calmodulin-Binding Proteins.....	230
4.14.1	Function of Calmodulin-Binding Proteins in Different Signaling Systems .....	230
4.14.2	CaM Binds to CNG Channel Protein.....	231
4.14.3	Ca <sup>2+</sup> -ATPases as CaM-Binding Proteins .....	231
4.14.4	Protein Kinases as CaM-Binding Proteins.....	231
4.14.5	Receptor-Like Kinases as CaM-Binding Proteins .....	232
4.14.6	NAD Kinase as CaM-Binding Protein .....	232
4.14.7	Catalases as CaM-Binding Proteins .....	233
4.14.8	NO Synthase as CaM-Binding Protein .....	234
4.14.9	MAPK Phosphatase as a CaM-Binding Protein.....	234
4.14.10	Calmodulin Binding Transcription Activators (CAMTAs).....	234
4.14.11	Calmodulin-Binding OsCBT, NtER1, and AtSR1 Transcription Factors.....	235
4.14.12	Calmodulin-Binding WRKY Transcription Factors .....	235
4.14.13	Calmodulin-Binding MYB Transcription Factors .....	236
4.14.14	Calmodulin-Binding TGA Transcription Factors .....	237
4.14.15	Calmodulin-Binding Homeodomain Transcription Factors.....	239
4.14.16	CaM-Binding Protein Involved in Glucosinolate Metabolism.....	239
4.14.17	CaM Binds with MLO Protein to Regulate Defense Response .....	239

4.14.18 CaM-Binding Protein Involved in HR-Associated Cell Death..... 240

4.14.19 CBP60 Family of Calmodulin-Binding Proteins ..... 240

4.15 Calmodulin-Like Proteins as Ca<sup>2+</sup> Sensors ..... 241

4.16 Calcineurin B-Like Proteins as Ca<sup>2+</sup> Sensors..... 241

4.17 NADPH Oxidase as Calcium-Binding Protein ..... 242

4.18 Ca<sup>2+</sup>-Binding Proteins Without EF-Hands ..... 243

4.19 Calcium-Dependent Protein Kinases as Ca<sup>2+</sup> Sensors..... 244

    4.19.1 Structure of CDPKs..... 244

    4.19.2 PAMP/Elicitor Triggers Activation of CDPK ..... 244

    4.19.3 Stimulation of CDPK Activity by 14-3-3 Proteins..... 246

    4.19.4 Enhancement of CDPK Activity by Phospholipids..... 246

    4.19.5 CDPKs Target Proteins Involved in Immune Signaling System..... 246

4.20 Nuclear Free Calcium Ion ([Ca<sup>2+</sup>]<sub>nuc</sub>) in Ca<sup>2+</sup> Signaling..... 248

4.21 Downstream Events in Ca<sup>2+</sup> Signaling System ..... 249

    4.21.1 ROS Generation ..... 249

    4.21.2 NO Generation ..... 250

    4.21.3 MAPK Signaling System..... 251

    4.21.4 Salicylate Signaling System..... 253

    4.21.5 Jasmonate Signaling System..... 254

    4.21.6 Ethylene Signaling System ..... 256

    4.21.7 Abscisic Acid Signaling System ..... 257

    4.21.8 Phytoalexin Biosynthesis ..... 258

4.22 Importance of Calcium Signaling System in Activation of Plant Innate Immunity ..... 258

References..... 260

**5 Reactive Oxygen Species and Cognate Redox Signaling System in Plant Innate Immunity..... 283**

5.1 Reactive Oxygen Intermediates Involved in Oxidative Burst ..... 284

5.2 Upstream Events in ROS Signaling System..... 284

    5.2.1 Enzymes Involved in ROS Generation..... 284

    5.2.2 Early PAMP-Induced Events Leading to Activation of NADPH Oxidase to Generate ROS ..... 286

    5.2.3 Cell Wall Peroxidases Are Involved in ROS Production in Some Plant Systems ..... 287

5.3 ROS-Scavenging Systems May Be Involved in Fine-Tuning Accumulation of ROS ..... 288

5.4 Site of Production of ROS..... 289

5.5 Biphasic ROS Production..... 289

5.6 ROS Plays a Central Role in Triggering Immune Responses ..... 289

5.7 Interplay Between ROS and Ca<sup>2+</sup> Signaling System..... 290

5.8 Interplay Between ROS and NO Signaling Systems ..... 291

5.9 Interplay Between ROS and MAPK Signaling Systems..... 291

5.10	Interplay Between ROS and Salicylic Acid Signaling Systems.....	291
5.11	Interplay Between ROS and Ethylene Signaling Systems .....	292
5.12	Interplay Between ROS and Jasmonate Signaling Systems.....	293
5.13	Interplay Between ROS and Abscisic Acid (ABA) Signaling Systems .....	293
5.14	ROS Activates Phosphorylation/Dephosphorylation Systems .....	294
5.15	Function of ROS in Ubiquitin-Proteasome System .....	295
5.16	ROS May Regulate Expression of Transcription Factors .....	295
5.17	Redox Signaling System .....	295
5.18	ROS Signaling System May Activate Transcription of Defense Genes .....	298
5.19	Pathogens May Cause Disease by Interfering with ROS Signaling System in Host Plants .....	298
	References.....	299
<b>6</b>	<b>Nitric Oxide Signaling System in Plant Innate Immunity .....</b>	<b>307</b>
6.1	Nitric Oxide as a Component of the Repertoire of Signals Involved in Plant Immune Signaling System .....	308
6.2	PAMP-Induced Biosynthesis of NO in Plants.....	308
6.3	Upstream Events in NO Production.....	310
6.3.1	Ca <sup>2+</sup> Influx into Cytosol May Be an Early Upstream Event in NO Production .....	310
6.3.2	Role of Calmodulin in NO Production.....	311
6.3.3	ROS and ABA Act Upstream of NO Production .....	311
6.4	Nitric Oxide-Target Proteins .....	313
6.5	Interplay Between NO and Ca <sup>2+</sup> Signaling Systems .....	314
6.6	Interplay Between NO and ROS Signaling Systems .....	316
6.7	Role of NO in SA, JA, and Ethylene Signaling Systems .....	317
6.8	Role of NO in Protein S-Nitrosylation .....	318
6.9	Role of NO in Protein Nitration .....	321
6.10	Role of NO in Salicylic Acid-Regulated Systemic Acquired Resistance.....	322
	References.....	322
<b>7</b>	<b>Mitogen-Activated Protein Kinase Cascades in Plant Innate Immunity .....</b>	<b>331</b>
7.1	MAPK Signaling Three-Kinase Modules .....	332
7.2	MAP Kinases Involved in Plant Immune Responses.....	334
7.2.1	<i>Arabidopsis thaliana</i> MPK3 and MPK6 Positively Regulate Plant Immune Responses .....	334
7.2.2	<i>Arabidopsis thaliana</i> MPK4 Negatively Regulates Plant Immune Responses .....	335
7.2.3	<i>Arabidopsis thaliana</i> MPK11 in Plant Immune Responses.....	336
7.2.4	<i>Arabidopsis</i> MPK9 and MPK12 Positively Regulate ROS-Mediated ABA Signaling .....	336

7.2.5	Soybean GmMPK4 Negatively Regulates SA and ROS Signaling Systems.....	337
7.2.6	Rice OsMPK6 Positively Regulates Local Resistance and Negatively Regulates Systemic Acquired Resistance.....	337
7.2.7	Oilseed Rape BnMPK4 Positively Regulates JA-Mediated Defense Responses .....	337
7.2.8	Cotton GhMPK2 Are Involved in Ethylene Biosynthesis - Mediated Plant Immune Responses .....	338
7.2.9	Cotton GhMPK7 Triggers SA Signaling System.....	338
7.2.10	GhMPK16 Activates ROS-Mediated Signaling System ..	338
7.2.11	SIPK and WIPK Activates Plant Immune Responses by Modulating SA and JA Signaling Systems .....	339
7.3	MAPK Kinases (MAPKKs) in Plant Immune Responses .....	339
7.3.1	MKK1 in Plant Immune Responses.....	339
7.3.2	Arabidopsis MKK2 Differentially Induces Defense Responses Against Different Pathogens.....	340
7.3.3	Arabidopsis MKK3 Positively Regulates Immune Responses.....	340
7.3.4	Arabidopsis MKK7 Positively Regulates SA-Mediated SAR .....	341
7.3.5	Cotton GhMKK5 Triggers ROS-Mediated Signaling Systems .....	341
7.4	MAPKK Kinase EDR1 Modulates SA-JA-ET Signaling .....	342
7.5	MAPK Pathways Involved in Defense Signal Transduction May Be Interconnected .....	344
7.6	14-3-3 Protein Enhances Signaling Ability of MAPKKK in Activating Plant Innate Immune Responses.....	345
7.7	Role of MAPKs in Priming Plants for Augmented Defense Gene Activation.....	346
7.8	PAMP Signals Activate MAP Kinases .....	347
7.9	Signals and Signaling Systems Activating MAPK Cascades.....	348
7.10	MAPKs May Function Downstream of G-Proteins, Ca <sup>2+</sup> , ROS, SA, ABA, and NO Signaling Pathways.....	349
7.11	Some MAPKs May Act Upstream of SA, JA, and ET Signaling Pathways.....	349
7.12	Some MAP Kinases Act Downstream of Phosphoinositide (PI) Signal Transduction Pathway.....	350
7.13	MAP Kinase Cascades May Act Either Upstream or Downstream of ROS Signaling System .....	350
7.14	MAP Kinases Positively or Negatively Regulate SA Signaling System.....	353
7.15	MAP Kinase Cascades Activate JA Signaling System.....	354
7.16	Some MAP Kinase Cascades Are Involved in Biosynthesis of Ethylene and Ethylene-Mediated Signaling Systems .....	355



7.17	Involvement of MAP Kinase in Crosstalk Between SA and JA/ET Signaling Systems .....	357
7.18	MAPK Phosphatases as Negative Regulators of MAP Kinases .....	358
7.19	MAP Kinase Cascades Modulate Phosphorylation of Transcription Factors to Trigger Transcription of Defense Genes .....	359
7.20	MAPKs Regulate Defense Gene Expression by Releasing Transcription Factors in the Nucleus.....	360
7.21	Role of MAPK Signaling Cascade in Triggering Phytoalexin Biosynthesis .....	361
7.22	Role of MAPK Signaling Cascade in Stomatal Immune Response .....	361
7.23	Effectors Inhibit PAMP-Triggered MAPK Signaling to Suppress Plant Immune Responses.....	363
	References.....	365
<b>8</b>	<b>Phospholipids Signaling System in Plant Innate Immunity.....</b>	<b>375</b>
8.1	Biosynthesis of Phospholipids-Derived Second Messengers.....	375
8.2	Phospholipids in Ca <sup>2+</sup> Signaling System.....	377
8.3	Phosphatidic Acid in G Proteins-Mediated Signaling System.....	378
8.4	Phosphatidic Acid in ROS Signaling System.....	378
8.5	Phospholipids in JA Signaling System.....	380
8.6	Phospholipid Signaling System in ABA Signaling Network.....	380
8.7	Phosphatidic Acid in Phosphorylation/Dephosphorylation System.....	381
	References.....	381
<b>9</b>	<b>Protein Phosphorylation and Dephosphorylation in Plant Immune Signaling Systems .....</b>	<b>385</b>
9.1	Protein Phosphorylation Plays Key Roles in Plant Immune Signal Transduction .....	386
9.2	Protein Phosphorylation Is an Early PAMP/Elicitor-Triggered Event.....	386
9.3	Protein Phosphorylation Is Carried Out by Different Protein Kinases.....	387
9.4	PAMPs/Elicitors Activate Receptor-Like Kinases .....	388
9.5	PAMP/Elicitor Induces Phosphorylation of Calcium-Dependent Protein Kinases .....	390
9.6	PAMP/Elicitor Triggers Phosphorylation of MAP Kinases .....	390
9.7	Role of 14-3-3 Proteins in Protein Phosphorylation .....	391
9.8	PAMP/Elicitor Triggers Phosphorylation of PEN Proteins.....	392
9.9	Protein Phosphorylation Involved in Early Defense Signaling Events Triggered by PAMPs/Elicitors .....	392

9.10	Phosphorylation of Proteins Involved in H <sup>+</sup> Fluxes Induced by PAMP/Elicitor .....	394
9.11	Phosphorylation of Proteins Involved in ROS Signaling System .....	394
9.12	Phosphorylation of Proteins Involved in Ethylene-Signaling System .....	396
9.13	Phosphorylation of Proteins Involved in Salicylic Acid Signaling System .....	397
9.14	Protein Phosphorylation in ABA Signaling System.....	397
9.15	Phosphorylation of Transcription Factors .....	399
9.16	Phosphorylation Events Induced by MAP Kinases in Various Signaling Systems .....	399
9.17	Dephosphorylation Induced by Phosphatases May Negatively Regulate Innate Immune Responses .....	400
	References.....	400
<b>10</b>	<b>Ubiquitin-Proteasome System-Mediated Protein Degradation in Defense Signaling</b> .....	<b>409</b>
10.1	Ubiquitin-Proteasome System in Plants.....	409
10.2	Ubiquitin-Proteasome in Jasmonate Signaling System.....	412
10.2.1	JAZ Proteins Act as Repressors of JA Signaling Pathway .....	412
10.2.2	JA Signaling Pathway Is Activated by the Removal of the JAZ Repressor Proteins by Ubiquitination .....	412
10.2.3	Ubiquitin-Proteasome-Mediated Proteolysis in JA Signaling System .....	414
10.3	Ubiquitin-Proteasome in Ethylene Signaling System .....	415
10.4	Ubiquitin-Proteasome in SA Signaling System .....	417
10.4.1	Ubiquitin-Proteasome May Be Involved in Regulation of SA Levels in the SA Signaling System.....	417
10.4.2	Role of an E3 Ubiquitin Ligase, OsRHC1, in SA-Dependent NPR1 Signaling.....	418
10.4.3	Role of SON1 (F-Box Protein in E3 Ubiquitin-Ligase Complex) in SA-Mediated Immune Responses .....	418
10.5	Ubiquitin-Proteasome in R-Gene Mediated Early Signaling System .....	419
10.6	Small Ubiquitin-Like Modifier (SUMO) in Plant Immunity.....	420
10.6.1	Role of SUMOylation in SA Biosynthesis.....	420
10.6.2	Role of SUMOylation in SA-Mediated Systemic Acquired Resistance .....	421
10.7	Pathogens May Subvert Ubiquitin-Proteasome System to Cause Disease.....	422
	References.....	423
	<b>Index</b> .....	<b>431</b>

# Chapter 1

## Introduction

**Abstract** Innate immunity is the first line of defense against invading microorganisms in plants. Pathogen-associated molecular patterns (PAMPs) are the classical activators of immune responses. These are alarm signal molecules are perceived as ‘nonself’ by plant pattern recognition receptors (PRRs) to switch on the plant immune responses. PAMPs are not only detected in pathogens, but also detected in nonpathogens and even in saprophytes. The PAMPs are often called as microbe-associated molecular patterns (MAMPs). MAMPs are molecular signatures typical of whole classes of microbes and their recognition by PRRs activates the plant innate immunity. Most of the PRRs are receptor-like kinases (RLKs) and RLKs are proteins with a “receptor” and a “signaling domain” in one molecule. The extracellular domains of RLKs bind directly to legands to perceive extracellular signals, whereas the cytoplasmic kinase domains transduce these signals into the cell. PRRs interact with additional transmembrane proteins which act as “signaling amplifiers”. PAMPs induce autophosphorylation of the kinase domain of PRRs and the autophosphorylated PRRs are translocated to endosomes. The biogenesis of transmembrane PRRs occurs through endoplasmic reticulum (ER) with the aid of ER-resident chaperones. The PRR in ER is transported from ER to plasma membrane and *N*-glycosylation of PRRs is required for the transport of PRRs. Second messengers deliver the information generated by the PAMP/PRR signaling complex to the proteins which decode/interpret signals to initiate defense gene expression. Calcium ion is a ubiquitous intracellular second messenger involved in various defense signaling pathways.  $Ca^{2+}$  is a master regulator of gene expression in plants. Calcium signatures are recognized by calcium sensors to transduce calcium-mediated signals into downstream events. Guanosine triphosphate (GTP)-binding proteins (G-proteins) act as molecular switches in signal transduction system. Mitogen-activated protein kinase (MAPK) cascades transduce extracellular stimuli into intracellular responses in plants. Reactive oxygen species is a second messenger in transmitting the PAMP signal. Nitric oxide (NO) is a diffusible second messenger acting in cellular signal transduction through stimulus-coupled S-nitrosylation of cysteine residues. The plant hormones salicylic acid, jasmonate,

ethylene, abscisic acid, auxin, cytokinin, gibberellins, and brassinosteroids play important role in immune response signaling. Plant hormones activate different signaling pathways inducing distinctly different defense genes. These signaling pathways can crosstalk with each other and this crosstalk helps the plant to “decide” which defensive strategy to follow, depending on the type of attacker it is encountering. Potential pathogens produce several effectors to nullify the defense responses induced by the innate immune system. Pathogens may also hijack some signaling systems to cause disease. The war between the plant and pathogen appears to be in fine-tuning the signaling systems to cause disease or to enhance host defense response. Recent advances in our understanding of the molecular basis of plant innate immunity have opened new era in developing potential tools in management of crop diseases.

**Keywords** Pathogen-associated molecular patterns (PAMPs) • Microbe-associated molecular patterns (MAMPs) • Plant pattern recognition receptors (PRRs) • Endocytosis of PRR proteins • PAMP-triggered immunity (PTI) • PAMP-PRR signaling complex

## 1.1 Classical PAMPs

Innate immunity is the first line of defense against invading microorganisms in vertebrates and the only line of defense in invertebrates and plants (Silipo et al. 2010; Zamioudis and Peterse 2012). Several elicitors of microbial origin have been identified as primary danger/alarm signal molecules to switch on the plant immune systems culminating in activation of defense genes (Aziz et al. 2003; D’Ovidio et al. 2004; Cavalcanti et al. 2006; Vidhyasekaran 2007; Thomma et al. 2011). The classical general elicitors reported in plant pathogens resemble the pathogen-associated molecular patterns (PAMPs), the classical activators of innate immune responses in mammals (Nürnberger and Brunner 2002; Nürnberger et al. 2004; Nürnberger and Lipka 2005). These historically termed general elicitors have been renamed as PAMPs (Jones and Dangl 2006; Bent and Mackey 2007). PAMPs are often vital for microbial survival and are therefore not subject to mutational variation (Gust et al. 2007; Zhang and Zhou 2010). PAMPs are defined as evolutionarily conserved building blocks of microbial surfaces that directly bind to plant pattern recognition receptors (PRRs) and induce defense responses (Nürnberger and Brunner 2002; Qutob et al. 2006; Nicaise et al. 2009; Tsuda and Katagiri 2010; Thomma et al. 2011). The molecular signatures in PAMPs are not present in the host and these are perceived as ‘non-self’ by plant pattern recognition receptors (Mackey and McFall 2006).

PAMPs that trigger innate immune responses in various vertebrates and non-vertebrate organisms include eubacterial flagellin, elongation factors, lipopolysaccharides (LPS) from gram-negative bacteria, viral and bacterial nucleic acids, fungal cell wall-derived chitins, glucans, mannans, or proteins and peptidoglycans from gram-positive bacteria (Zipfel and Felix 2005; Jones and Dangl 2006). Similar

PAMPs have been detected in a wide range of plant pathogens (Shinya et al. 2007; Boller and Felix 2009; Silipo et al. 2010; Tsuda and Katagiri 2010; Nürnberger and Kufner 2011). One of the common features of PAMPs is their presence in a broad range of microbial species (Brunner et al. 2002). The general structure of lipopolysaccharides (LPS) is shared by all gram-negative bacteria (Medzhitov 2001) and the protein PAMP flagellin is highly conserved among bacterial taxa (Felix et al. 1999). Chitin is the widespread, conserved, and intrinsic structure detected in fungi (Thomma et al. 2011). CBEL (for Cellulose-Binding Elicitor Lectin) is a glycoprotein PAMP and it occurs widely in the oomycete *Phytophthora* species (Khatib et al. 2004). The PAMP double-stranded RNA is a structural signature of several groups of viruses (Medzhitov 2001; Ding 2010).

PAMPs are exclusively recognized as the molecules involved in triggering innate immunity. PAMPs are actually defined as the molecules, which bind to plant PRRs and induce defense responses (Nicaise et al. 2009; Tsuda and Katagiri 2010). However, most of the PAMPs also have virulence functions besides eliciting defense responses (Thomma et al. 2011). The well characterized PAMP flagellin also has a role in virulence. Glycosylation of the flagellin molecule has been shown to be required for virulence in *Pseudomonas syringae* pv. *tabaci* (Taguchi et al. 2010). *P. syringae* pv. *tabaci* flagellin mutants affected in their elicitor activity also showed reduced virulence in plants due to reduced motility (Naito et al. 2008; Taguchi et al. 2010). The bacterial lipopolysaccharide (LPS) generally acts as PAMP inducing defenses (Tellström et al. 2007; Aslam et al. 2008; Silipo et al. 2008; Thomma et al. 2011). However, changes in composition of LPS affect bacterial virulence, suggesting a role for LPS in virulence of pathogens (Newman et al. 2007). When the PAMP chitin synthesis was disrupted in the fungal pathogen *Botrytis cinerea*, virulence of the pathogen was drastically reduced (Soulie et al. 2006). Mutation of peptidoglycan (PGN) genes reduces the virulence of *Ralstonia solanacearum* and of *Erwinia amylovora* (Cloud-Hansen et al. 2006), suggesting the role of the PAMP peptidoglycan in virulence of pathogens.

PAMPs are detected not only in pathogens, but also in several nonpathogens, and saprophytes. Since the PAMPs are detected in all microbes, the PAMPs are better called as microbe-associated molecular patterns (MAMPs) (Viterbo et al. 2007; Zhang et al. 2007; Denoux et al. 2008; Aslam et al. 2009; Jeworutzki et al. 2010; Thomma et al. 2011; de Freitas and Stadnik 2012). MAMPs are molecular signatures typical of whole classes of microbes, and their recognition plays a key role in innate immunity (Boller and Felix 2009).

## 1.2 Plant Pattern Recognition Receptors (PRRs)

PAMPs are perceived as alarm/danger signals by cognate plant pattern recognition receptors (PRRs) and the PAMP-PRR complex activates the plant immune system (Takakura et al. 2004; Jones and Dangl 2006; Altenbach and Robatzek 2007; He et al. 2007; Wan et al. 2008; Iriti and Faoro 2009). Several receptors for the PAMPs

have been recognized in plasma membrane of plant cells (Nicaise et al. 2009; Petutschnig et al. 2010; Shinya et al. 2010; Schulze et al. 2010; Segonzac and Zipfel 2011). The PRRs identified to date are modular proteins harbouring an extracellular domain consisting of leucine-rich repeat (LRR) or lysine motifs (LysM) (Saijo 2010; Segonzac and Zipfel 2011). Most of the PRRs are receptor-like kinases (RLKs) and the sensors for extracellular molecules consisting of an extracellular ligand-binding domain, a single transmembrane domain, and a cytosolic protein kinase domain are called RLKs (Seifert and Blaukopf 2010). RLKs are proteins with a “receptor” and a “signaling domain” in one molecule. The extracellular domains of RLKs bind directly to ligands to perceive extracellular signals (PAMPs), whereas the cytoplasmic kinase domains transduce these signals into the cell (Bi et al. 2010).

PRRs interact with additional transmembrane proteins which act as signaling amplifiers to achieve their functionality (Zipfel 2009). PAMPs bind with PRRs and induce conformational alteration in PRRs leading to their activation (Ali et al. 2007). PAMPs trigger increased transcription of PRR genes and accumulation of PRR proteins (Qutob et al. 2006; Lohmann et al. 2010). Most of the PRRs are receptor kinases and the PAMPs induce autophosphorylation of the kinase domain of PRRs (Kanzaki et al. 2008; Xiang et al. 2008).

The plasma membrane resident autophosphorylated PRRs are translocated to endosomes and it helps to extend the signaling surface ensuring a robust and efficient cellular signaling system (Geldner and Robatzek 2008). PAMPs induce ubiquitin-proteasome- or clathrin-mediated endocytosis of PRR proteins (Robatzek et al. 2006; Aker and de Vries 2008). PAMP-induced PRR-induced endocytosis has been shown to be dependent on phosphorylation of the PRR (Robatzek et al. 2006). PAMP-induced internalization of PRRs from the plasma membrane is closely correlated with their immune function (Bar et al. 2009; Saijo 2010). The biogenesis of trans-membrane PRRs may occur through the endoplasmic reticulum (ER) with the aid of ER-resident chaperones (Dodds and Rathjen 2010; Popescu 2012). After biosynthesis of PRR in ER, it is transported from the ER to the plasma membrane. *N*-glycosylation of PRRs is required for transport of PRRs from ER to plasma membrane (Häweker et al. 2010). Sustained activation of plasma membrane-resident PRR signaling is important for mounting robust PAMP-triggered immunity (Saijo 2010).

### 1.3 Second Messengers in PAMP Signal Transduction

The plant immune system uses several second messengers to encode information generated by the PAMP/PRR signaling complex and deliver the information downstream of PRRs to proteins which decode/interpret signals and initiate defense gene expression (van Verk et al. 2008; Mersmann et al. 2010; Boudsocq et al. 2010; Hwang and Hwang 2011). It is still not known how the PAMP signals are transmitted downstream of PRR. In plant cells, the calcium ion is a ubiquitous intracellular second messenger involved in numerous signaling pathways (Luan 2009; McAinsh

and Pittman 2009; Abdul Kadar and Lindsberg 2010; DeFalco et al. 2010; Hamada et al. 2012; Stael et al. 2012).

Guanosine triphosphate (GTP)-binding proteins (G-proteins) are the regulatory GTPases, which act as molecular switches in signal transduction system (Yalowsky et al. 2010; Zhang et al. 2011, 2012). Two classes of signaling G-proteins, heterotrimeric G-proteins and small monomeric G-proteins (Ras/Ras-like small GTPases), have been reported. In the Ras superfamily of small GTPases, only the Ras and Rho families have been shown to transmit extracellular signals (Gu et al. 2004). Ras superfamily is named the Ras superfamily because the founding members are encoded by human Ras genes initially discovered as cellular homologs of the viral *ras* oncogene. Plants do not possess a true Ras GTPase such as those that are pivotal signaling in animals. Instead, they have a unique subfamily of Rho-family GTPases, called ROPs (Rho-related GTPase of plants). ROP is the sole subfamily of Rho GTPase in plants. ROPs are also referred to as RAC (for Ras [rat sarcoma oncogene product] related C3 botulinum toxin substrate) proteins (Gu et al. 2004; Kiirika et al. 2012). RAC/ROP small GTPases share a common ancestor with Rho, cdc42 and Rac and they are the only Rho-like GTPases in plants (Gu et al. 2004).

Ca<sup>2+</sup> is a master regulator of gene expression in plants (Galon et al. 2010). Calcium ion acts as a signal carrier (Allen et al. 2000). Calcium signaling is modulated by specific calcium signatures. Ca<sup>2+</sup> signatures are generated in the cytosol, and in noncytosolic locations including the nucleus and chloroplast through the coordinated action of Ca<sup>2+</sup> influx and efflux pathways (McAinsh and Pittman 2009). Specific calcium signatures are recognized by different calcium sensors to transduce calcium-mediated signals into downstream events (Reddy et al. 2011; Wang et al. 2012; Hashimoto et al. 2012).

Mitogen-activated protein kinase (MAPK) cascades are major pathways downstream of sensors/receptors that transduce extracellular stimuli into intracellular responses in plants (Hettenhausen et al. 2012; Zhang et al. 2012). A typical MAPK signaling module consists of three interconnected protein kinases: a MAP kinase kinase kinase (MAPKKK or MEKK [for MAPK/Extracellular signal-regulated kinase Kinase Kinase]), a MAP kinase kinase (MAPKK or MKK), and a MAP kinase (MAPK or MPK) (Mészáros et al. 2006). MAP kinase cascade involves sequence of phosphorylation events (Hirt 2000). MAPKs function at the bottom of the three-kinase cascade and are activated by MAPKKs through phosphorylation on the Thr and Tyr residues in their activation motif between the kinase subdomain VII and VIII. The activity of MAPKKs is, in turn, regulated by MAPKKKs via phosphorylation of two Ser/Thr residues in the activation loop of MAPKKs. MAPKKKs receive signals from upstream receptors/sensors (Ichimura et al. 2002; Li et al. 2012).

The oxidative burst involving rapid and transient production of reactive oxygen species (ROS) is a very rapid response, occurring within seconds (Bolwell et al. 1995) or within a few minutes (Arnott and Murphy 1991) of PAMP treatment, suggesting that the oxidative burst may not require *de novo* protein synthesis but involves the activation of pre-existing enzymes. NADPH oxidase (Bae et al. 2006), peroxidases (Halliwell 1978; Lehtonen et al. 2012), and xanthine oxidase (Allan and Fluhr

1997; Ori et al. 1997) have been shown to be involved in triggering ROS production. ROS is a messenger in transmitting the PAMP signal. Nitric oxide (NO) has been identified as a gaseous second messenger (Besson-Bard et al. 2008; Bellin et al. 2013). NO is a diffusible molecular messenger that plays an important role in the plant immune response signal transduction system (Grennan 2007). PAMPs trigger NO burst within minutes in plant cells (Foissner et al. 2000; Lamotte et al. 2004; Tischner et al. 2010). NO acts substantially in cellular signal transduction through stimulus-coupled S-nitrosylation of cysteine residues (Benhar et al. 2008). It serves as a key redox-active signal for the activation of various defense responses (Klessig et al. 2000).

## 1.4 Plant Hormone Signals in Plant Immune Signaling System

The plant hormones salicylic acid (Mukherjee et al. 2010; Dempsey et al. 2011; Liu et al. 2011a, b), jasmonate (Wang et al. 2009; Sheard et al. 2010; Bertoni 2012), ethylene (Boutrot et al. 2010; Laluk et al. 2011; Nie et al. 2011; Nambeesan et al. 2012), abscisic acid (Yazawa et al. 2012), auxin (Fu and Wang 2011), cytokinin (Choi et al. 2011), gibberellins (Qin et al. 2013), and brassinosteroids (De Vleeschauwer et al. 2012) play important role in defense signaling against various pathogens. It has been demonstrated that specific plant hormone signaling pathways should be activated to confer resistance against specific pathogens. JA and SA signaling systems may differentially contribute for resistance against specific pathogens. JA-mediated pathway effectively confers resistance against necrotrophic fungal pathogens (Berrocal-Lobo and Molina 2004; McGrath et al. 2005; Zheng et al. 2006), while SA-mediated pathway confers resistance against biotrophic fungal pathogens and also against virus and bacterial diseases in some plants (Thomma et al. 1998, 2001; Thaler and Bostock 2004; Nie 2006; De Vos et al. 2006; Spoel et al. 2007; Zheng et al. 2006, 2007). Two forms of induced resistance, systemic acquired resistance (SAR) and induced systemic resistance (ISR), have been recognized based on the induction of specific plant hormone signaling systems (Li et al. 2008). SAR refers to a distinct signaling pathway mediated by SA (Oostendorp et al. 2001), while ISR refers to the signaling pathway mediated by JA and ET. SA signaling system activates not only local resistance, but also systemic acquired resistance (SAR) observed in distal (systemic) tissues. SAR is a SA-dependent heightened defense to a broad spectrum of pathogens that is activated throughout a plant following local infection (Liu et al. 2011a). SAR is associated with priming of defense (Kohler et al. 2002; Jung et al. 2009; Luna et al. 2011) and the priming results in a faster and stronger induction of defense mechanisms after pathogen attack (Conrath 2011). The priming can be inherited epigenetically from disease-exposed plants (Pastor et al. 2012) and descendants of primed plants exhibit next-generation systemic acquired resistance (Slaughter et al. 2012; Luna et al. 2011). The transgenerational SAR has been



recently reported (Luna et al. 2011). Thus, SA signal is transduced not only within the plant tissues, but also transferred even to the next generations.

Plant hormones activate different signaling pathways inducing distinctly different defense genes (Spoel et al. 2007; Zhang et al. 2007; Mitsuhara et al. 2008). These signaling pathways are not simple linear and isolated cascades, but can crosstalk with each other. Both antagonism and synergism between the signaling systems have been reported. Cross-talk between defense signaling pathways is thought to provide the plant with a powerful regulatory potential, which helps the plant to “decide” which defensive strategy to follow, depending on the type of attacker it is encountering (De Vos et al. 2005). It may also allow pathogens to manipulate plants to their own benefit by shutting down induced defense through influences on the signaling network.

## **1.5 War Between Host Plants and Pathogens and the Winner Is ----- ?**

Plant innate immune systems have high potential to fight against viral, bacterial, oomycete, and fungal pathogens and protect the crop plants against wide range of diseases (Knecht et al. 2010; Lacombe et al. 2010; Hwang and Hwang 2011; Alkan et al. 2012). However, potential pathogens produce several effectors to nullify the defense responses induced by the innate immune system (Wu et al. 2011; Akimoto-Tomiyama et al. 2012; Cheng et al. 2012). To avoid or suppress or delay the expression of the defense gene-activating signaling systems, the pathogens secrete several effectors into the host cell (Göhre et al. 2008; Kim et al. 2010; Wu et al. 2011; Cheng et al. 2012). Pathogens may also hijack some signaling systems to cause disease (de Torres-Zabala et al. 2007; Thatcher et al. 2009; El Rahman et al. 2012). It has also been demonstrated that the virulent pathogen may suppress the particular defense signaling system which induce the expression of specific defense genes conferring resistance against the particular pathogen (van Verk et al. 2008; Koornneef and Pieterse 2008; Makandar et al. 2010). Activation of some signaling systems may induce susceptibility, rather than resistance (Atsumi et al. 2009; Yazawa et al. 2012). To overcome antiviral RNA silencing immunity, plant viruses express silencing-suppressor proteins which can counteract the host silencing-based antiviral process (Qu and Morris 2005; Ding and Voinnet 2007; Lewsey et al. 2010).

The war between the plant and pathogen appears to be in fine-tuning the signaling systems to cause disease or enhance host defense. Fast and strong activation of the plant immune responses aids the host plants to win the war against the pathogens (Großkinsky et al. 2011). Overexpression or suppression of some specific signaling systems in the plant immune system has been shown to help the plants to win in the arms race between plants and pathogens (Cheung et al. 2007; Zhang et al. 2008; Hwang and Hwang 2010, 2011; Wu et al. 2010).

Engineering durable nonspecific resistance to phytopathogens is one of the ultimate goals of plant breeding. However, most of the attempts to reach this goal fail as a

result of rapid changes in pathogen populations and the sheer diversity of pathogen infection mechanisms. Recently several bioengineering and molecular manipulation technologies have been developed to activate the ‘sleeping’ plant innate immune system, which has potential to detect and suppress the development of a wide range of plant pathogens in economically important crop plants (Lacombe et al. 2010). Enhancing disease resistance through altered regulation of plant immunity signaling systems would be durable and publicly acceptable (Yamamizo et al. 2006; Shao et al. 2008; Gust et al. 2010; Lacombe et al. 2010). Strategies for activation and improvement of plant immunity aim at enhancing host capacities for recognition of potential pathogens, at boosting the executive arsenal of plant immunity, and interfering with virulence strategies employed by microbial pathogens (Gust et al. 2010). Major advances in our understanding of the molecular basis of plant immunity and of microbial infection strategies have opened new ways for engineering durable resistance in crop plants (Gust et al. 2010; Huffaker et al. 2011). This book describes the most fascinating PAMP-PRR signaling complex and signal transduction systems. It discusses the highly complex networks of signaling pathways involved in transmission of the signals to induce distinctly different defense-related genes to mount offence against different pathogens.

## References

- Abdul Kadar M, Lindsberg S (2010) Cytosolic calcium and pH signaling in plants under salinity stress. *Plant Signal Behav* 5:233–238
- Aker J, de Vries SC (2008) Plasma membrane receptor complexes. *Plant Physiol* 147:1560–1564
- Akimoto-Tomiya C, Furutani A, Tsuge S, Washington EJ, Nishizawa Y, Minami E, Ochiai H (2012) XopR, a type III effector secreted by *Xanthomonas oryzae* pv. *oryzae*, suppresses microbe-associated molecular pattern-triggered immunity in *Arabidopsis thaliana*. *Mol Plant Microbe Interact* 25:505–514
- Ali GS, Prasad KVSK, Day I, Reddy ASN (2007) Ligand-dependent reduction in the membrane mobility of FLAGELLIN SENSITIVE2, an Arabidopsis receptor-like kinase. *Plant Cell Physiol* 48:1601–1611
- Alkan N, Fluhr R, Prusky D (2012) Ammonium secretion during *Colletotrichum coccodes* infection modulates salicylic and jasmonic acid pathways of ripe and unripe tomato fruit. *Mol Plant Microbe Interact* 25:85–96
- Allan AG, Fluhr R (1997) Two distinct sources of elicited reactive oxygen species in tobacco epidermal cells. *Plant Cell* 9:1559–1572
- Allen GJ, Chu SP, Schumacher K, Shimazaki CT, Vafeados D, Kemper A, Hawke SD, Tallman G, Tsien RY, Harper JF, Chory J, Schroeder JI (2000) Alteration of stimulus-specific guard cell calcium oscillations and stomatal closing in *Arabidopsis det3* mutant. *Science* 289:2338–2342
- Altenbach DD, Robatzek S (2007) Pattern recognition receptors: from the cell surface to intracellular dynamics. *Mol Plant Microbe Interact* 20:1031–1039
- Arnott T, Murphy TM (1991) A comparison of the effects of a fungal elicitor and ultraviolet radiation on ion transport and hydrogen peroxide synthesis by rose cells. *Environ Exp Bot* 31:209–216
- Aslam SN, Newman MA, Erbs G, Morissey KL, Chinchilla D, Boller T, Jensen TT, De Castro C, Tearn T, Molinaro A, Jackson RW, Knight MR, Cooper RM (2008) Bacterial polysaccharides suppress induced innate immunity by calcium chelation. *Curr Biol* 18:1078–1083

- Aslam SN, Erbs G, Morrissey KL, Newman M-A, Chinchilla D, Boller T, Molinaro A, Jackson RW, Cooper RM (2009) Microbe-associated molecular pattern (MAMP) signatures, synergy, size, and charge: influences on perception or mobility and host defense responses. *Mol Plant Pathol* 10:375–387
- Atsumi G, Kagaya U, Kitazawa H, Nakahara KS, Uyeda I (2009) Activation of the salicylic acid signaling pathway enhances *Clover yellow vein virus* virulence in susceptible pea cultivars. *Mol Plant Microbe Interact* 22:166–175
- Aziz A, Poinssot B, Daire X, Adrian M, Bezier A, Lambert B, Joubert JM, Pugin A (2003) Laminarin elicits defense responses in grapevine and induces protection against *Botrytis cinerea* and *Plasmopara viticola*. *Mol Plant Microbe Interact* 16:1118–1128
- Bae H, Kim MS, Sicher RC, Bae H-J, Bailey BA (2006) Necrosis- and ethylene-inducing peptide from *Fusarium oxysporum* induces a complex cascade of transcripts associated with signal transduction and cell death in *Arabidopsis*. *Plant Physiol* 141:1056–1067
- Bar M, Sharfman M, Schuster S, Avni A (2009) The coiled-coil domain of EHD2 mediates inhibition of LeEix2 endocytosis and signaling. *PLoS One* 4:e7963
- Bellin D, Asai S, Delledonne M, Yoshioka H (2013) Nitric oxide as a mediator for defense responses. *Mol Plant Microbe Interact* 26:271–277
- Benhar M, Forrester MT, Hess DT, Stamler JS (2008) Regulated protein denitrosylation by cytosolic and mitochondrial thioredoxins. *Science* 320:1050–1054
- Bent AF, Mackey D (2007) Elicitors, effectors, R genes: the new paradigm and a lifetime supply of questions. *Annu Rev Phytopathol* 45:399–436
- Berocal-Lobo M, Molina A (2004) Ethylene response factor 1 mediates *Arabidopsis* resistance to the soilborne fungus *Fusarium oxysporum*. *Mol Plant Microbe Interact* 17:763–770
- Bertoni G (2012) Oxylipins and plant palatability. *Plant Cell* 24:1305
- Besson-Bard A, Courtois C, Gauthier A, Dahan J, Dobrowolska G, Jeandroz S, Pugin A, Wendehenne D (2008) Nitric oxide in plants: production and cross-talk with Ca<sup>2+</sup> signaling. *Mol Plant* 1:218–228
- Bi D, Cheng YT, Li X, Zhang Y (2010) Activation of plant immune responses by a gain-of-function mutation in an atypical receptor-like kinase. *Plant Physiol* 153:1771–1779
- Boller T, Felix G (2009) A renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors. *Annu Rev Plant Biol* 60:379–406
- Bolwell GP, Butt VS, Davies DR, Zimmerlin A (1995) The origin of the oxidative burst in plants. *Free Radic Res* 23:517–532
- Boudsocq M, Willmann MR, McCormack M, Lee H, Shan L, He P, Bush J, Cheng SH, Sheen J (2010) Differential innate immune signalling via Ca<sup>2+</sup> sensor protein kinases. *Nature* 464:418–422
- Boutrot F, Segonzac C, Chang KN, Qiao H, Ecker JR, Zipfel C (2010) Direct transcriptional control of the *Arabidopsis* immune receptor FLS2 by the ethylene-dependent transcription factors EIN3 and EIL1. *Proc Natl Acad Sci USA* 107:14502–14507
- Brunner F, Rosahl S, Lee J, Rudd JJ, Geller C, Kauppinen S, Rasmussen G, Scheel D, Nürnberger T (2002) Pep-13, a plant defense-inducing pathogen-associated pattern from *Phytophthora* transglutaminases. *EMBO J* 21:6681–6688
- Cavalcanti FR, Resende MLV, Lima J, Silveira JAG, Oliveira TA (2006) Activities of antioxidant enzymes and photosynthetic responses in tomato pre-treated by plant activators and inoculated by *Xanthomonas vesicatoria*. *Physiol Mol Plant Pathol* 68:198–208
- Cheng B, Yu X, Ma Z, Dong S, Dou D, Wang Y, Zheng X (2012) *Phytophthora sojae* effector Avh331 suppresses the plant defence response by disturbing the MAPK signalling pathway. *Physiol Mol Plant Pathol* 77:1–9
- Cheung M-Y, Zeng N-Y, Tong S-W, Li FW-Y, Zhao K-J, Zhang Q, Sun SS-M, Lam H-M (2007) Expression of a RING-HC protein from rice improves resistance to *Pseudomonas syringae* pv. *tomato* DC3000 in transgenic *Arabidopsis thaliana*. *J Exp Bot* 58:4147–4159
- Choi J, Choi D, Lee R, Ryu CM, Hwang I (2011) Cytokinins and plant immunity: old foes or new friends. *Trends Plant Sci* 16:388–394
- Cloud-Hansen KA, Brook Petersen S, Stabb EV, Goldman WE, McFall-Ngai MJ, Handelsman J (2006) Breaching the great wall: peptidoglycan and microbial interactions. *Nat Rev Microbiol* 4:710–716

- Conrath U (2011) Molecular aspects of defence priming. *Trends Plant Sci* 16:524–531
- D’Ovidio R, Mattei B, Roberti S, Bellincampi D (2004) Polygalacturonases, polygalacturonase-inhibiting proteins and pectic oligomers in plant-pathogen interactions. *Biochim Biophys Acta Proteomics* 1696:237–244
- de Freitas MB, Stadnik MJ (2012) Race-specific and ulvan-induced defense responses in bean (*Phaseolus vulgaris*) against *Colletotrichum lindemuthianum*. *Physiol Mol Plant Pathol* 78:8–13
- de Torres-Zabala M, Truman W, Bennett MH, Lafforgue G, Mansfield JW, Rodriguez Egea P, Bogre L, Grant M (2007) *Pseudomonas syringae* pv. *tomato* hijacks the *Arabidopsis* abscisic acid signalling pathway to cause disease. *EMBO J* 26:1434–1443
- De Vleeschauwer D, Van Buyten E, Satoh K, Balidion J, Mauleon R, Choi I-R, Vera-Cruz C, Kikuchi S, Höfte M (2012) Brassinosteroids antagonize gibberellin- and salicylate-mediated root immunity in rice. *Plant Physiol* 158:1833–1846
- De Vos M, Van Oosten VR, Van Poecke RMP, Van Pelt JA, Pozo MJ, Mueller MJ, Buchala AJ, Métraux J-P, Van Loon LC, Dicke M, Pieterse CMJ (2005) Signal signature and transcriptome changes of *Arabidopsis* during pathogen and insect attack. *Mol Plant Microbe Interact* 18:923–937
- De Vos M, Van Zaanen W, Koornneef A, Korzelius JP, Dicke M, Van Loon LC, Pieterse CMJ (2006) Herbivore-induced resistance against microbial pathogens in *Arabidopsis*. *Plant Physiol* 142:352–363
- DeFalco TA, Bender KW, Snedden WA (2010) Breaking the code: Ca<sup>2+</sup> sensors in plant signalling. *Biochem J* 425:27–40
- Dempsey DA, Vlot AC, Wildermuth MC, Klessig DF (2011) Salicylic acid biosynthesis and metabolism. *Arabidopsis Book* 9:e0156. doi:10.1199/tab.0156
- Denoux C, Galletti R, Mammarella N, Gopalan S, Werck D, De Lorenzo G, Ferrari S, Ausubel FM, Dewdney J (2008) Activation of defense response pathways by OGs and Flg22 elicitors in *Arabidopsis* seedlings. *Mol Plant* 1:423–445
- Ding S-W (2010) RNA-based antiviral immunity. *Nat Rev Immunol* 10:632–644
- Ding S-W, Voinnet O (2007) Antiviral immunity directed by small RNAs. *Cell* 130:413–426
- Dodds PN, Rathjen JP (2010) Plant immunity: towards an integrated view of plant-pathogen interactions. *Nat Rev Genet* 11:539–548
- El Rahman TA, El Oirdi M, Gonzalez-Lamothe R, Bouarab K (2012) Necrotrophic pathogens use salicylic acid signaling pathway to promote disease development in tomato. *Mol Plant Microbe Interact* 25:1584–1593
- Felix G, Duran JD, Volko S, Boller T (1999) Plants have a sensitive perception system for the most conserved domain of bacterial flagellin. *Plant J* 18:265–276
- Foissner L, Wendehenne D, Langebartels C, Durner J (2000) In vivo imaging of an elicitor-induced nitric oxide burst in tobacco. *Plant J* 23:817–824
- Fu J, Wang S (2011) Insights into auxin signaling in plant-pathogen interactions. *Front Plant Sci* 2:74. doi:10.3389/fpls.2011.0074, Article 2:pp7
- Galon Y, Aloni R, Nachmias D, Snir O, Feldmesser E, Scrase-Field S, Boyce JM, Bouché N, Knight MR, Fromm H (2010) Calmodulin-binding transcription activator 1 mediates auxin signaling and responds to stresses in *Arabidopsis*. *Planta* 232:165–178
- Geldner N, Robatzek S (2008) Plant receptors go endosomal: a moving view on signal transduction. *Plant Physiol* 147:1565–1574
- Göhre V, Spallek T, Häweker H, Mersmann S, Mentzel T, Boller T, de Torres M, Mansfield JW, Robatzek S (2008) Plant pattern-recognition receptor FLS2 is directed for degradation by the bacterial ubiquitin ligase AvrPtoB. *Curr Biol* 18:1824–1832
- Grennan AK (2007) Protein S-nitrosylation: protein targets and roles in signal transduction. *Plant Physiol* 144:1237–1239
- Größkinsky DK, Naseem M, Abdelmohsen UR, Plickert N, Engelke T, Griebel T, Zeier J, Novák O, Strnad M, Pfeifhofer H, Graaff EVD, Simon U, Roitsch T (2011) Cytokinins mediate resistance against *Pseudomonas syringae* in tobacco through increased antimicrobial phytoalexin synthesis independent of salicylic acid signaling. *Plant Physiol* 157:815–830
- Gu Y, Wang Z, Yang Z (2004) ROP/RAC GTPase: an old new master regulator for plant signaling. *Curr Opin Plant Biol* 7:527–536

- Gust AA, Biswas R, Lenz HD, Rauhut T, Ranf S, Kemmerling B, Götz F, Glawischnig E, Lee J, Felix G, Nürnberger T (2007) Bacteria-derived peptidoglycans constitute pathogen-associated molecular patterns triggering innate immunity in *Arabidopsis*. *J Biol Chem* 282:32338–32348
- Gust AA, Brunner F, Nürnberger T (2010) Biotechnological concepts for improving plant innate immunity. *Curr Opin Biotechnol* 21:204–210
- Halliwell B (1978) Superoxide-dependent formation of hydroxyl radicals in the presence of iron chelates. Is it a mechanism for hydroxyl radical production in biochemical systems? *FEBS Lett* 92:321–326
- Hamada H, Kuruu T, Okuma E, Nokajima H, Kiyoduka M, Koyano T, Sugiyama Y, Okada K, Koga J, Saji H, Miyao A, Hirochika H, Yamane H, Murata Y, Kuchitsu K (2012) Regulation of a proteinaceous elicitor-induced  $Ca^{2+}$  influx and production of phytoalexins by a putative voltage-gated cation channel, OsTPC1, in cultured rice cells. *J Biol Chem* 287:9931–9939
- Hashimoto K, Eckert C, Anschütz U, Scholz M, Held K, Waadt R, Reyer A, Hippler M, Becker D, Kudla J (2012) Phosphorylation of calcineurin B-like (CBL) calcium sensor proteins by their CBL-interacting protein kinases (CIPKs) is required for full activity of CBL-CIPK complexes toward their target proteins. *J Biol Chem* 287:7956–7968
- Häweker H, Rips S, Koiwa H, Salomon S, Saijo Y, Chinchilla D, Robatzek S, von Schaewen A (2010) Pattern recognition receptors require *N*-glycosylation to mediate plant immunity. *J Biol Chem* 285:4629–4636
- He P, Shan L, Sheen J (2007) Elicitation and suppression of microbe-associated molecular pattern-triggered immunity in plant-microbe interactions. *Cell Microbiol* 9:1385–1396
- Hettenhausen C, Baldwin IT, Wu J (2012) Silencing *MPK4* in *Nicotiana attenuata* enhances photosynthesis and seed production but compromises abscisic acid-induced stomatal closure and guard cell-mediated resistance to *Pseudomonas syringae* pv. *tomato* DC3000. *Plant Physiol* 158:759–776
- Hirt H (2000) Connecting oxidative stress, auxin, and cell cycle regulation through a plant mitogen-activated protein kinase pathway. *Proc Natl Acad Sci USA* 97:2405–2407
- Huffaker A, Dafoe NJ, Schmelz EA (2011) ZmPep1, an ortholog of *Arabidopsis* elicitor peptide 1, regulates maize innate immunity and enhances disease resistance. *Plant Physiol* 155:1325–1338
- Hwang IS, Hwang BK (2010) The pepper 9-lipoxygenase gene *CaLOX1* functions in defense and cell death responses to microbial pathogens. *Plant Physiol* 152:948–967
- Hwang IS, Hwang BK (2011) The pepper mannose-binding lectin gene *CaMBL1* is required to regulate cell death and defense responses to microbial pathogens. *Plant Physiol* 155:447–463
- Ichimura K, Shinozaki K, Tena G, Sheen J, Henry Y (2002) Mitogen-activated protein kinase cascades in plants: a new nomenclature. *Trends Plant Sci* 7:301–308
- Iriti M, Faoro F (2009) Chitosan as a MAMP, searching for a PRR. *Plant Signal Behav* 4:66–68
- Jeworutzki E, Roelfsema MR, Anschütz U, Krol E, Elzenga JT, Felix G, Boller T, Hedrich R, Becker D (2010) Early signaling through the *Arabidopsis* pattern recognition receptors FLS2 and EFR involves  $Ca^{2+}$ -associated opening of plasma membrane anion channels. *Plant J* 62:367–378
- Jones JDG, Dangl JL (2006) The plant immune system. *Nature* 444:323–329
- Jung HW, Tschaplinski TJ, Wang L, Glazebrook J, Greenberg JT (2009) Priming in systemic plant immunity. *Science* 324:89–91
- Kanzaki H, Saitoh H, Takahashi Y, Berberich T, Ito A, Kamoun S, Terauchi R (2008) NbLRK1, a lectin-like receptor kinase protein of *Nicotiana benthamiana*, interacts with *Phytophthora infestans* INF1 elicitor and mediates INF1-induced cell death. *Planta* 228:977–987
- Khatib M, Lafitte C, Esquerré-Tugayé M-T, Bottin A, Rickauer M (2004) The CBEL elicitor of *Phytophthora parasitica* var. *nicotianae* activates defence in *Arabidopsis thaliana* via three different signalling pathways. *New Phytol* 162:501–510
- Kiirika LM, Bergmann HF, Schikowsky C, Wimmer D, Korte J, Schmitz U, Niehaus K, Colditz F (2012) Silencing of the Rac1 GTPase *MtROP9* in *Medicago truncatula* stimulates early mycorrhizal and oomycete root colonizations but negatively affects rhizobial infection. *Plant Physiol* 159:501–516

- Kim Y-T, Oh J, Kim K-H, Uhm J-Y, Lee B-M (2010) Isolation and characterization of *NgRLK1*, a receptor-like kinase of *Nicotiana glutinosa* that interacts with the elicitor of *Phytophthora capsici*. *Mol Biol Rep* 37:717–727
- Klessig DF, Durner J, Noad R, Navarre DA, Wendehenne D, Kumar D, Zhou JM, Shah J, Zhang S, Kachroo P, Trifa Y, Pontier D, Lam E, Silva H (2000) Nitric oxide and salicylic acid signalling in plant defense. *Proc Natl Acad Sci USA* 97:8849–8855
- Knecht K, Seyffarth M, Desel C, Thurau T, Sherameti I, Lou B, Oelmüller R, Cai D (2010) Expression of *BvGLP-1* encoding a germin-like protein from sugar beet in *Arabidopsis thaliana* leads to resistance against phytopathogenic fungi. *Mol Plant Microbe Interact* 23:446–457
- Kohler A, Schwinding S, Conrath U (2002) Benzothiadiazole-induced priming for potentiated responses to pathogen infection, wounding, and infiltration of water into leaves requires the *NPR1/NIM1* gene in *Arabidopsis*. *Plant Physiol* 128:1046–1056
- Koornneef A, Pieterse CMJ (2008) Cross talk in defense signaling. *Plant Physiol* 146:839–844
- Lacombe S, Rougon-Cardoso A, Sherwood E, Peeters N, Dahlbeck D, van Esse HP, Smoker M, Rallapalli G, Thomma BPHJ, Stakawicz B, Jones JDG, Zipfel C (2010) Interfamily transfer of a plant pattern-recognition receptor confers broad-spectrum bacterial resistance. *Nat Biotechnol* 28:365–369
- Laluk K, Luo H, Chai M, Dhawan R, Lai Z, Mengiste T (2011) Biochemical and genetic requirements for function of the immune response regulator BOTRYTIS-INDUCED KINASE1 in plant growth, ethylene signaling, and PAMP-triggered immunity in *Arabidopsis*. *Plant Cell* 23:2831–2849
- Lamotte O, Gould K, Lecourieux D, Sequeira-Legrand A, Lebrun-Garcia A, Durner J, Pugin A, Wendehenne D (2004) Analysis of nitric oxide signaling functions in tobacco cells challenged by the elicitor cryptogein. *Plant Physiol* 135:516–529
- Lehtonen NT, Akita M, Frank W, Reski R, Valkonen JPT (2012) Involvement of a class III peroxidase and the mitochondrial protein TSPO in oxidative burst upon treatment of moss plants with a fungal elicitor. *Mol Plant Microbe Interact* 25:363–371
- Lewsey MG, Murphy AM, MacLean D, Dalchau N, Westwood JH, Macaulay K, Bennett M, Moulin M, Hanke DE, Powell G, Smith AG, Carr JP (2010) Disruption of two signaling pathways by a viral RNA silencing suppressor. *Mol Plant Microbe Interact* 23:835–845
- Li YM, Zhang ZK, Jia YT, Shen YM, He HM, Fang RX, Chen XY, Hao XJ (2008) 3-acetyl-3-hydroxy-oxindole: a new inducer of systemic acquired resistance in plants. *Plant Biotechnol J* 6:301–308
- Li G, Meng X, Wang R, Mao G, Han L, Liu Y, Zhang S (2012) Dual-level regulation of ACC synthase activity by MPK3/MPK6 cascade and its downstream WRKY transcription factor during ethylene induction in *Arabidopsis*. *PLoS Genet* 8(6):e1002767. doi:10.1371/journal.pgen.1002767
- Liu P-P, von Dahl CC, Klessig DF (2011a) The extent to which methyl salicylate is required for signaling systemic acquired resistance is dependent on exposure to light after infection. *Plant Physiol* 157:2216–2226
- Liu P-P, von Dahl CC, Park S-W, Klessig DF (2011b) Interconnection between methyl salicylate and lipid-based long-distance signaling during the development of systemic acquired resistance in *Arabidopsis* and tobacco. *Plant Physiol* 155:1762–1768
- Lohmann GV, Shimoda Y, Nielsen W, Jørgensen FG, Grossmann C, Sandal N, Sørensen K, Thirup S, Madsen LH, Tabata S, Sato S, Stougaard J, Radutoiu S (2010) Evolution and regulation of the *Lotus japonica* LysM receptor gene family. *Mol Plant Microbe Interact* 23:510–521
- Luan S (2009) The CBL-CIPK network in plant calcium signaling. *Trends Plant Sci* 14:37–42
- Luna E, Bruce TJA, Roberts MR, Flors V, Ton J (2011) Next generation systemic acquired resistance. *Plant Physiol* 158:844–853
- Mackey D, McFall AJ (2006) MAMPs and MIMPs: proposed classification for inducers of innate immunity. *Mol Microbiol* 61:1365–1371
- Makandar R, Nalam V, Chaturvedi R, Jeannotte R, Sparks AA, Shah J (2010) Involvement of salicylate and jasmonate signaling pathways in *Arabidopsis* interaction with *Fusarium graminearum*. *Mol Plant Microbe Interact* 23:861–870