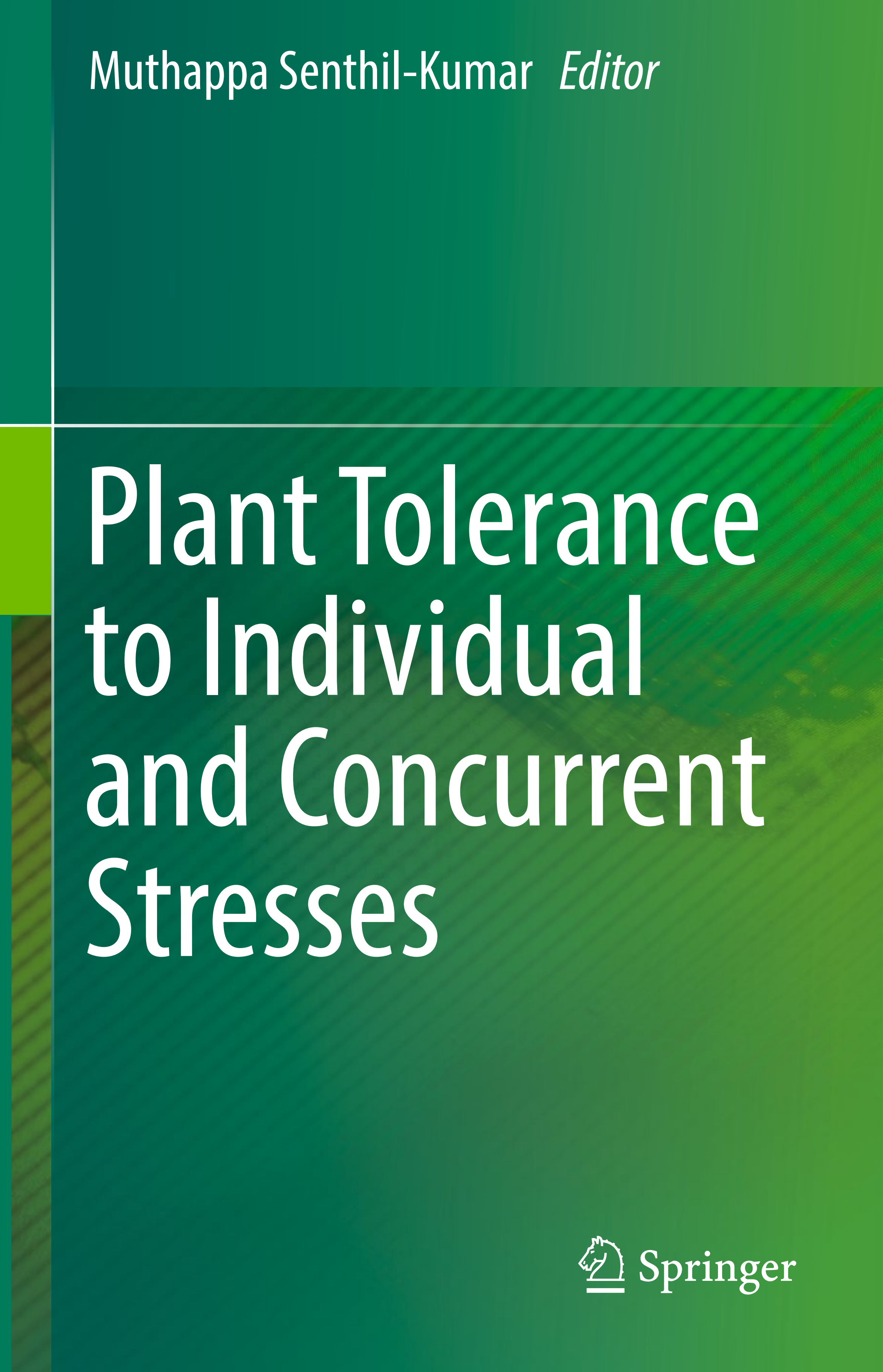
Muthappa Senthil-Kumar *Editor*

Plant Tolerance to Individual and Concurrent Stresses



Springer

Plant Tolerance to Individual and Concurrent Stresses

Muthappa Senthil-Kumar
Editor

Plant Tolerance to Individual and Concurrent Stresses

Springer

Editor
Muthappa Senthil-Kumar
National Institute of Plant Genome Research
New Delhi, India

ISBN 978-81-322-3704-4 ISBN 978-81-322-3706-8 (eBook)
DOI 10.1007/978-81-322-3706-8

Library of Congress Control Number: 2017930398

© Springer (India) Pvt. Ltd. 2017
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.
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.
The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer (India) Pvt. Ltd.
The registered company address is: 7th Floor, Vijaya Building, 17 Barakhamba Road, New Delhi 110 001, India

Preface

In nature, plants are exposed to one or more biotic and abiotic stresses either individually or in combination, which ultimately results in yield loss. During the life cycle, the same plant can face individually occurring one or more stresses. A large number of studies were undertaken to dissect the mechanisms imparting plant tolerance to multiple individual stresses. However, the concurrent stress tolerance has not been adequately studied owing to several complexities involved, including appropriate combined stress imposition method. In the recent past, several research groups around the world have started exploring the concurrent stress tolerance mechanisms under both biotic and abiotic stress combinations. This book compiles the information generated by these research groups along with their research progress and prospects, which would serve as a compendium of knowledge for researchers working on plant stress biology.

This book covers three major aspects under the proposed title. First, it introduces the existence of unique and shared responses in plants exposed to combined stress. Emphasis is given for understanding shared responses in comparison with multiple individual stresses. Second, the influence of abiotic stress on plant-pathogen interaction is elaborately covered. Third, comprehensive information about screening methods to identify genetic variation and the use of various tools to extrapolate information from individual stress studies to understand concurrent stress tolerance is elaborated. The chapterwise coverage of above said information is as follows.

Chapters 1 and 2 cover the overview of physiological and molecular mechanism involved in imparting both individual and combined stress tolerance. Importance is also given to the soil management and agronomic practices that will facilitate cultural management of crops under combined stress. Chapters 3 and 4 enumerates the impact of biotic stresses, namely, weed and pathogen on sequential and simultaneously occurring abiotic stresses including drought and temperature stress. Chapter 5 explains the approaches and avenues available for utilizing the understandings covered in the previous four chapters in terms of genomics-assisted breeding. Chapters 6 and 7 comprehend all previously described stress responses and set tone for specific stress tolerance mechanisms described in subsequent chapters. Chapter 8 focuses on the plant interaction with light and temperature, both as stimuli and stress. This chapter specifically covers the signaling responses and emphasizes the growth changes during combined stress. Hormonal cross talks under combined stress and the coordinated regulation of stress tolerance mechanisms are discussed in Chap. 9. Impact of several individual stresses on plants and strategies for crop improvement are covered in Chap. 10. The Chap. 11 covers the plant-water relations during various pathogen infections. It also enumerates the complexity of these responses in the presence of drought stress. Overall, these 11 chapters delivers scintillating information that not only provide comprehension of up-to-date research outcome in understanding stress interaction and combined stress tolerance, but also enumerate future direction of research. Overall they acts as suitable study material for both students and researchers working this area. This book also delivers prospects for driving future research for developing strategies for crop improvement under multiple stresses.

Eminent researchers from this newly emerging field have contributed to this book as outlined above. This book will be not only served as a one-stop reference point for researchers working in plant responses to both biotic and abiotic stresses but also will be an authority of recent information in this area. It is noteworthy to emphasize the fact that despite the plants grown under field condition exposed to combination of multiple stresses, a comprehensive collection of recent information in this area is lacking. This book will sufficiently address this deficit and act as a reference material for the research community.

I acknowledge all the reviewers who made scientific and technical comments on each chapter included in this book for their valuable time and input.

New Delhi, India Muthappa Senthil-Kumar

Contents

Contributors

Seth Bernard Abugho Department of Soil and Crop Sciences, Texas A&M University, College Station, TX, USA

Muthukumar Bagavathiannan Department of Soil and Crop Sciences, Texas A&M University, College Station, TX, USA

Juhi Bhattacharya National Institute of Plant Genome Research, New Delhi, India

Annvi Dhaka National Institute of Plant Genome Research, New Delhi, India

Urooj Fatima National Institute of Plant Genome Research, New Delhi, India

Raju Ghosh International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, Hyderabad, Telangana, India

Prabhu Govindasamy Department of Soil and Crop Sciences, Texas A&M University, College Station, TX, USA

Aarti Gupta National Institute of Plant Genome Research, New Delhi, India

Sumandeep Juneja Centre for Plant Sciences, School of Basic and Applied Sciences, Central University of Punjab, Bathinda, Punjab, India

Sanjeev Kumar Centre for Plant Sciences, School of Basic and Applied Sciences, Central University of Punjab, Bathinda, Punjab, India

Anuradha Kumari Centre for Biochemistry and Microbial Sciences, Central University of Punjab, Bathinda, Punjab, India

Rui Liu Department of Soil and Crop Sciences, Texas A&M University, College Station, TX, USA

Mehanathan Muthamilarasan National Institute of Plant Genome Research, New Delhi, India

Gunasekhar Nachimuthu New South Wales Department of Primary Industries, Australian Cotton Research Institute, Narrabri, NSW, Australia

Karaba N. Nataraja Plant Molecular Biology Laboratory, Department of Crop Physiology, University of Agricultural Sciences, Bengaluru, India

Prachi Pandey National Institute of Plant Genome Research, New Delhi, India

M. S. Parvathi Plant Molecular Biology Laboratory, Department of Crop Physiology, University of Agricultural Sciences, Bengaluru, India

Manoj Prasad National Institute of Plant Genome Research, New Delhi, India

Wusirika Ramakrishna Centre for Biochemistry and Microbial Sciences, Central University of Punjab, Bathinda, Punjab, India

Aashish Ranjan National Institute of Plant Genome Research, New Delhi, India

Pranav Pankaj Sahu National Institute of Plant Genome Research, New Delhi, India

Muthappa Senthil-Kumar National Institute of Plant Genome Research, New Delhi, India

Mamta Sharma International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, Hyderabad, Telangana, India

Priyanka Singh Centre for Plant Sciences, School of Basic and Applied Sciences, Central University of Punjab, Bathinda, Punjab, India

Roshan Kumar Singh National Institute of Plant Genome Research, New Delhi, India

Upendra Kumar Singh National Institute of Plant Genome Research, New Delhi, India

Vijay Singh Department of Soil and Crop Sciences, Texas A&M University, College Station, TX, USA

Ashley A. Webb New South Wales Department of Primary Industries, Tamworth Agricultural Institute, Calala, NSW, Australia

Renu Yadav Centre for Plant Sciences, School of Basic and Applied Sciences, Central University of Punjab, Bathinda, Punjab, India

About the Editor

Muthappa Senthil-Kumar is a scientist at the National Institute of Plant Genome Research, New Delhi, India. He received his B.Sc. in agriculture from Tamil Nadu Agricultural University, Coimbatore and M. Sc. and Ph.D. in crop physiology from the University of Agricultural Sciences, Bangalore, India. He was post-doctoral fellow at The Samuel Roberts Noble Foundation, Ardmore, Oklahoma, USA. He has published over 40 research articles and several review articles on understanding plant interaction with drought stress and pathogens. Currently, his research team is working to understand the interaction of drought and pathogen stress and their combined impact on plants.

Concurrent Stresses Are Perceived as New State of Stress by the Plants: Overview of Impact of Abiotic and Biotic Stress Combinations

1

Aarti Gupta and Muthappa Senthil-Kumar

Abstract

Crop plants under natural conditions often encounter abiotic and biotic stresses either individually or in combination, single or multiple times in their life cycle. During their concurrence, different stressors interact with each other over the plant interface leading to altered plant responses. Initial stressor can modulate plant physiology and thereby influences plant response towards another stressor. Consequent to the stress interaction, plants encountering concurrent stress show different responses in comparison to the plants exposed to the individual stresses. Additionally, plant defence responses are somewhat skewed towards one stressor during concurrent occurrence of stresses. Such different responses are the cognate 'net effect' of combined stress felt by the plant. The net effect exhibited by plants under combined stress is unique to each stress combination. Thus, in lieu of the combined stress responses, which are different from the individual stress responses, the combined stress has been proposed as a new state of stress. Plant responses towards this new state are not just dictated by either of the individual stresses alone but by more complex interaction. In this chapter, we present an overview of the combined stresses with emphasis on drought and bacterial stressors and discuss the stress interaction effect and net effect.

Keywords

Concurrent stress • Stress interaction • New stress • Unique responses

---

A. Gupta • M. Senthil-Kumar (✉)
National Institute of Plant Genome Research, 10531, JNU Campus, Aruna Asaf Ali Marg,
New Delhi 110 067, India
e-mail: skmuthappa@nipgr.ac.in

M. Senthil-Kumar (eds.), *Plant Tolerance to Individual and Concurrent Stresses*,
DOI 10.1007/978-81-322-3706-8_1

1.1 Introduction

Under field conditions, the environmental constraints do not always occur independently but most often occur in conjunction with pathogens, and this is detrimental to survival of crop plants. Extreme weather patterns have led to the periodic incidences of drought and pathogen infections (Desprez-Loustau et al. 2007; Yáñez-López et al. 2012; Elad and Pertot 2014). Conventionally, disease triangle represents drought altered plant-pathogen interaction by influencing either the host defence or the pathogen virulence (Achuo et al. 2006; Amtmann et al. 2008; Goel et al. 2008; Hanso and Drenkhan 2009; Atkinson and Urwin 2012). Severe drought in 2003 stimulated *Diplodia pinea* (causal agent of Sphaeropsis blight) epidemic on conifers in Central Europe, and *Diplodia pinea* emerged as a new pathogen infecting *Pinus nigra* in Estonia (Hanso and Drenkhan 2009). The concurrence of drought and pathogen stress and their interaction over plant interface leads to altered plant physiology and resistance responses (Choi et al. 2013; Dossa et al. 2016; Gupta et al. 2016b; Sinha et al. 2016). Plant responses to concurrent drought stress and pathogen infection vary depending on the severity and duration of each stress, nature of infecting pathogens and plant genotype (Achuo et al. 2006; Xu et al. 2008; Ramegowda et al. 2013; Dossa et al. 2016; Gupta et al. 2016a; Sinha et al. 2016). Previously, few studies involving concurrent stresses on plants showed that stress interactions provoke a set of unique plant responses wherein some of the acclimation strategies are attuned to the constraints involved and are not seen under either of the individual stress (Xu et al. 2008; Atkinson et al. 2013; Prasch and Sonnewald 2013a; Gupta et al. 2016b). Moreover, combined stress also evokes responses which are 'common' to each of the individual stresses (Prasch and Sonnewald 2013a; Gupta et al. 2016b). Some of these responses are 'similar' between combined and individual stress, whereas certain common responses are evoked to a different level under combined stress when compared to individual stress and are termed as 'tailored responses'. Considering the existence of unique, tailored and similar responses under concurrent drought and pathogen stress compared to individual stresses (Suzuki et al. 2014; Gupta et al. 2016b), it is perceivable that combined stressed plants experience a new state of stress. This underlines the fact that the net impact of a concurrent stress and cognate plant response cannot be studied exclusively from single-stress experiments (Atkinson and Urwin 2012; Suzuki et al. 2014). In this chapter, we attempt to describe the delineation between stress interactions and net impact on plants.

1.2 Stress Interaction

1.2.1 Direct Impact of Drought on Pathogen

Drought stress can influence pathogen survival and spread in environment and thus impacts the disease incidence (Hanso and Drenkhan 2009). In case of rhizosphere-dwelling pathogens, the outcome of interaction between drought stress and bacterial pathogen varies depending upon the nature of the pathogen and whether the pathogen thrives in wet or dry soils. For example, drought favours *Streptomyces scabies* (causal agent of common scab in potato) multiplication in the rhizosphere and thereby increases the opportunity for subsequent infection in plants (Goto 1985). Most often, foliar pathogens experience low water availability in phyllosphere as the most important deterrent. Several studies showed that epiphytic microbial populations increase in wet months (the presence of water) but decline during dry periods (Hirano and Upper 1983, 1990). The leaf surface water does not only support pathogen multiplication but can also be conducive for sustaining plethora of microbial pathogens on phyllosphere and thereby increasing the subsequent chances of plant infection. Ercolani (1991) reported increased diversity of microbial pathogens on olive leaf surface during cooler wet months which however declined during the warmest and driest months of the season. Furthermore, Beattie (2011) reported that abundance of surface water favours bacterial invasion into the leaf tissue. Under water stress, many genes involved in pathogenicity and virulence (of bacteria), including genes in the hypersensitive response and pathogenicity alternative sigma factor (HrpL) regulon, were suppressed in *Pseudomonas syringae* pv. *tomato* DC3000 (causal agent of bacterial speck) (Freeman 2009). Thus, by modulating the pathogen multiplication and survival in the environment, drought (outside the plant) has been shown to regulate incidence of plant infection by these pathogens.

1.2.2 Stress Interactions at the Plant Interface

Drought and pathogen stressors can interact with each other at plant interface wherein drought directly impacts the *in planta* pathogen multiplication and spread. Bacterial movement inside the host is regulated by its flagella, which in turn is favoured by water availability in the leaf apoplast. In an instance, spread of *P. syringae* pv. *syringae* (causes brown spot of beans) in bean seedlings has been shown to be promoted by water (Leben et al. 1970). Instances for drought-induced *in planta* multiplication and spread for other pathogens are also available. Lowered water potential in pea leaves leads to reduced sporulation of *Erysiphe pisi* (causal agent of powdery mildew) (Ayres 1977). Drought stress also restricted *in planta* movement of *Tomato spotted wilt virus* (causal agent of tomato spotted wilt) and attenuated disease symptoms in tomato plants (Córdoba et al. 1991). These examples present a scenario of drought-induced tolerance towards pathogen in plants under combined stress by modulating systemic spread of pathogen.

Drought stress can interfere with plant immunity making the plant susceptible or resistant towards pathogen attack (Mohr and Cahill 2003; Koga et al. 2004; Hatmi et al. 2014). Drought stress imparted susceptibility to *Arabidopsis thaliana* cv. Ler against an avirulent bacterial pathogen *P. syringae* pv. *tomato 1065* (Pst1065) (Mohr and Cahill 2003) and to grapevines against *Xylella fastidiosa* (causal agent of bacterial leaf scorch and Pierce’s disease) (Choi et al. 2013). Drought stress increased the severity and progression of leaf scorch disease caused by *X. fastidiosa* in *Parthenocissus quinquefolia* vine (McElrone et al. 2001). The acclimation of *Nicotiana benthamiana* to moderate drought stress reduced the growth of *P. syringae* pv. tabaci (causes wildfire disease in tobacco) (Ramegowda et al. 2013). However, in the same study, severe drought stress had been shown to increase the susceptibility of the plants to *P. syringae*. Here, drought stress increased the ABA accumulation and hence interfered with plant defence responses (Ramegowda et al. 2013).

Water availability facilitated bacterial pathogenesis by suppressing the plant vasculature defences during effector-triggered immunity (ETI) in *A. thaliana* (Cook and Stall 1977; Freeman and Beattie 2009) and PAMP-triggered immunity in *N. benthamiana* (Oh and Collmer 2005). Freeman and Beattie (2009) showed that plants promote ETI and cause localized desiccation at the site of pathogen infection consequently restricting pathogen multiplication. Drought stress tolerance in grapevine involved activation of polyamine oxidation contributing to improved immune response and low susceptibility to *Botrytis cinerea* (causes grey mould disease) (Hatmi et al. 2014).

Drought stress also instigates physiological changes in plants which may be favourable to the pathogen. Drought-stressed sorghum plants were more susceptible to *Macrophomina phaseolina* (causal agent of charcoal rot) infection (Edmunds 1964). Reportedly, the sorghum root volatiles diffuse more rapidly through dry soil and favour *M. phaseolina* infection under drought conditions (Kerr 1964). In another instance, increased *M. phaseolina* infection in drought-stressed common bean has been reported (Mayek-Perez et al. 2002). Ijaz et al. (2013) suggested that drought stress led to accumulation of carbohydrates and amino acids (viz. asparagine and proline) which served as nutrient for the *M. phaseolina* instigating *in planta* pathogen growth and multiplication. Similarly, the drought-induced proline accumulation and ROS metabolism invoked susceptibility towards *Diplodia pinea* in Austrian pine (Sherwood et al. 2015).

In spite of the drought-imposed obstacles for *in planta* pathogen multiplication and survival, pathogen interacts with plant and tends to establish itself *in planta* during combined stress. In an attempt to overcome the obstacle posed by low water availability, bacteria actively modify the leaf surface habitat during drought stress. For example, bacteria can increase the wettability of leaves by secreting surfactants (Bunster et al. 1989; Hutchison and Johnstone 1993). The water films created by these biosurfactants hydrate epiphytic bacterial cells and facilitate movement of bacteria to more favourable sites (Lindow and Brandl 2003). Bacteria also modify their local environment by producing extracellular polymeric substances (EPS) which helps them hold on to the leaf surface and prevent desiccation by encapsulating cells in a hygroscopic matrix (Wilson et al. 1965; Takahashi and Doke 1984). Synthesis of alginate, a component of EPS, is stimulated by desiccation stress in *P. syringae* (Singh et al. 1992; Keith and Bender 1999) and contributes to epiphytic fitness of this organism during drought stress (Yu et al. 1999). High cell densities induce the expression of particular genes (Pierson et al. 1998; Bassler 1999) and contribute to epiphytic fitness (Monier and Lindow 2003) via quorum sensing cell to cell signals. *Xanthomonas campestris* (causal agent of wilt) was able to reverse stomatal closure induced by ABA via secretion of virulence factors (Gudesblat et al. 2009). Taken together, all these evidences suggest that drought influence pathogen multiplication and survival both outside and inside its host. Although at the same time, pathogen has also adopted combat mechanisms and establishes itself in the plant under combined stress conditions.

Further, studies also show that pathogen influences host plant physiology and water relations to predispose it to drought stress. The vascular wilt pathogens cause desiccation state in host plant which leads to reduced photosynthesis and reduced flow of photo assimilates to the roots and eventually causes reduced root growth. As a result, the host plant is more susceptible to the drought stress. *X. fastidiosa*, a xylem-limited bacterial pathogen, induces drought stress in alfalfa (Daugherty et al. 2010). These pathogens colonize and block xylem vessels and reduce their hydraulic conductivity, thereby aggravating the drought stress conditions in plants (Yadeta and Thomma 2013). Tomato plants infected with *Verticillium dahliae* (causal agent of Verticillium wilt) showed decreased leaf water potential (Ayres 1978).

A. thaliana plants infected with *V. longisporum* were tolerant to drought stress. *V. longisporum* induces the expression of vascular-related NAC domain (*VND7*) gene in these plants and triggered de novo xylem formation which leads to enhanced water storage capacity under drought stress conditions (Reusche et al. 2012). *P. syringae* infection in host plant could interfere with plant-water relation by causing water-soaking and the resultant desiccation of the infection site (Beattie 2011), and such case leads to more drought stress experienced by plants.

Both drought stress and foliar bacterial pathogen infection influence ABA levels and stomatal closure in plants. ABA treatment leads to susceptibility of *A. thaliana* towards avirulent bacterium *P. syringae* pv. *tomato* 1065 (Pst1065) infection where the susceptibility increased in a concentration-dependent manner (Mohr and Cahill 2003). Similarly, application of HopAM1 a type III effector of *P. syringae* increases the multiplication and virulence of *P. syringae* under drought stress (Goel et al. 2008). HopAM1 also enhanced ABA-mediated stomatal closure under drought stress (Goel et al. 2008). Pathogen effectors released inside the plant cell cause increased ABA accumulation and stomatal closure and decreased leaf transpiration rate, which altogether improved drought tolerance in combined stressed plants. For instance, application of purified HrpN-a protein produced by *Erwinia amylovora* (causal agent of fire blight) alleviated drought symptoms in *A. thaliana* (Dong et al. 2005). The increased ABA levels in response to the HrpN treatment enhanced the expression of several ABA-signalling regulatory genes as well as the drought-inducible gene *rd29B* (response to dehydration B), the gene product of which mediates ABA-induced responses (Dong et al. 2005). Root colonization with rhizobacteria, *P. chlororaphis* O6 also induced stomatal closure, reduced water loss by transpiration and increased drought tolerance in *A. thaliana* plants (Cho et al. 2008).

In conclusion, the two stressors when co-occurring influence the plant resistance as a result of stress interaction. Thus, in order to study plant-pathogen interaction, the actual scenarios must be accounted, and such understanding cannot be extrapolated from single-stress studies.

1.3 Net Effect of Combined Stresses

From the earlier discussions, it appears that combined stress is perceived as two interacting stressors by the plants where one could see the reminiscence of two individual stresses. However, looking at the existence of a set of unique responses and net impact, it can be settled that combined stress in plants is perceived as a new state of stress. The simultaneous occurrence of more than one stress influences plants as result of stress interaction and direct net impact of occurring stresses together (Daugherty et al. 2010; Atkinson et al. 2013; Ramegowda et al. 2013; Rasmussen et al. 2013; Bostock et al. 2014; Kissoudis et al. 2014; Prasch and Sonnewald 2013a, 2015; Gupta et al. 2016b). The net impact depends on the specific combination of stresses where the concurrence of two stressors can guard or further disrupt plant processes, and both the stresses, when occurring concurrently, most often act in unison to hamper plant growth and development (positive drought-pathogen interaction) (Fig. 1.1). As a result, the combined stresses can cause severe reduction in crop yield when compared with the losses incurred by individual stresses (Siddiqui 1980; Bhatti and Kraft 1992; McElrone et al. 2001; Janda et al. 2008; Prasch and Sonnewald 2013a; Fig. 1.1). Edmunds (1964) observed that concurrent drought stress and *Macrophomina phaseoli* infection caused more damage compared to individual stressed sorghum plants.

The set of net impact resulting from stress interactions in turn depends on common physiological effect or common traits influenced by the two constituent stressors (of concurrent stress) impacting on plant, and the outcome is more devastating than either of the individual stress. Individual drought stress and *X. fastidiosa* infection both lead to low water potential in leaf and influence reduction in stomatal conductance and xylem dysfunction. As a result of such synergism, *X. fastidiosa*, in combination with drought stress, increases the severity and progression of leaf scorch in *Parthenocissus quinquefolia* causing severe reduction in total biomass as compared to individual stresses (Fig. 1.1; McElrone et al. 2001, 2003). Drought stress invokes stomata closure in the plants (Wilkinson and Davies 2002), while on the other hand, *P. syringae* infection signals stomata opening (Melotto et al. 2008). When *Vicia faba* and *A. thaliana* were subjected to a combination *P. syringae* and water deficit, stomatal closure was more pronounced (Ou et al. 2014). In such case of antagonistic stress interaction, responses to abiotic stresses were found to override the responses to biotic stresses (Ou et al. 2014). Recent studies also suggest that the net impact could be the reminiscent of the stress interaction or due to direct impact of combined stress. In the following section, we attempt to delineate and assess the net impact of combined stresses.

1.4 Assessment of Net Impact of Combined Stress

As stated earlier, combined stressed plants experience net impact as a results of one of the following.

(a) Interaction of each stressor with plant
(b) Interaction between two stressor inside the plant
(c) Interaction of one stressor with plant influencing other stressor

In order to tag a combined stressed plant under natural field conditions, dissection of the component contributing towards net impact is important which so far is not understood. Further, for crop protection and improvement, systematic identification of contributory factors (through interaction) to combined drought and pathogen stress is needed. Foremost prerequisite for such studies is the identification of a common agronomically important parameter targeted by both the stressors. For example, yield reduction is seen in case of individual drought and charcoal rot infection in sorghum (Edmunds 1964). The alteration in the identified parameter can be used to answer how the two stressors are interacting with each other and with plant. During such studies, net impact of combined stress can be instanced in one of the following equations:

(i) $CS=D>P$
(ii) $CS=P>D$
(iii) $CS=D+P$ (additive/positive interaction)
(iv) $CS=D\text{-}P$
(v) $CS=P\text{-}D$
(vi) $CS\neq D$ or P or $D+P$ or $D\text{-}p$ or $P\text{-}D$

Here, D, P and CS denote net impact imparted by individual drought and pathogen stresses and their combination, respectively.

In case (i) drought could be said as the ‘dominant’ stressor, influencing the net impact of the combined stressed plants. The dominant stressor, here, refers to the stress which can modulate the plant processes and decides the plant interaction with subsequent stressor, and also the net impact of the combined stress plants is largely similar to the net impact of dominant stressed plants. In this case, drought can reduce pathogen growth, or it can interfere with plant resistance and impact yield loss (parameter considered here as net impact). For example, drought stress instigated activation of polyamine oxidation and improved immune response which lead to subsequent resistance in grapevine to *Botrytis cinerea* (Hatmi et al. 2014). Likewise, in case (ii) pathogen can be considered as dominant stressor where it can reduce drought effect while interfering with plant-water relations and curb yield loss, e.g. *Erwinia amylovora* alleviated drought symptoms in *A. thaliana* (Dong et al. 2005). In case (iii) the net impact (total loss in yield) is equivalent to the additive losses incurred by individual drought and pathogen stresses and results

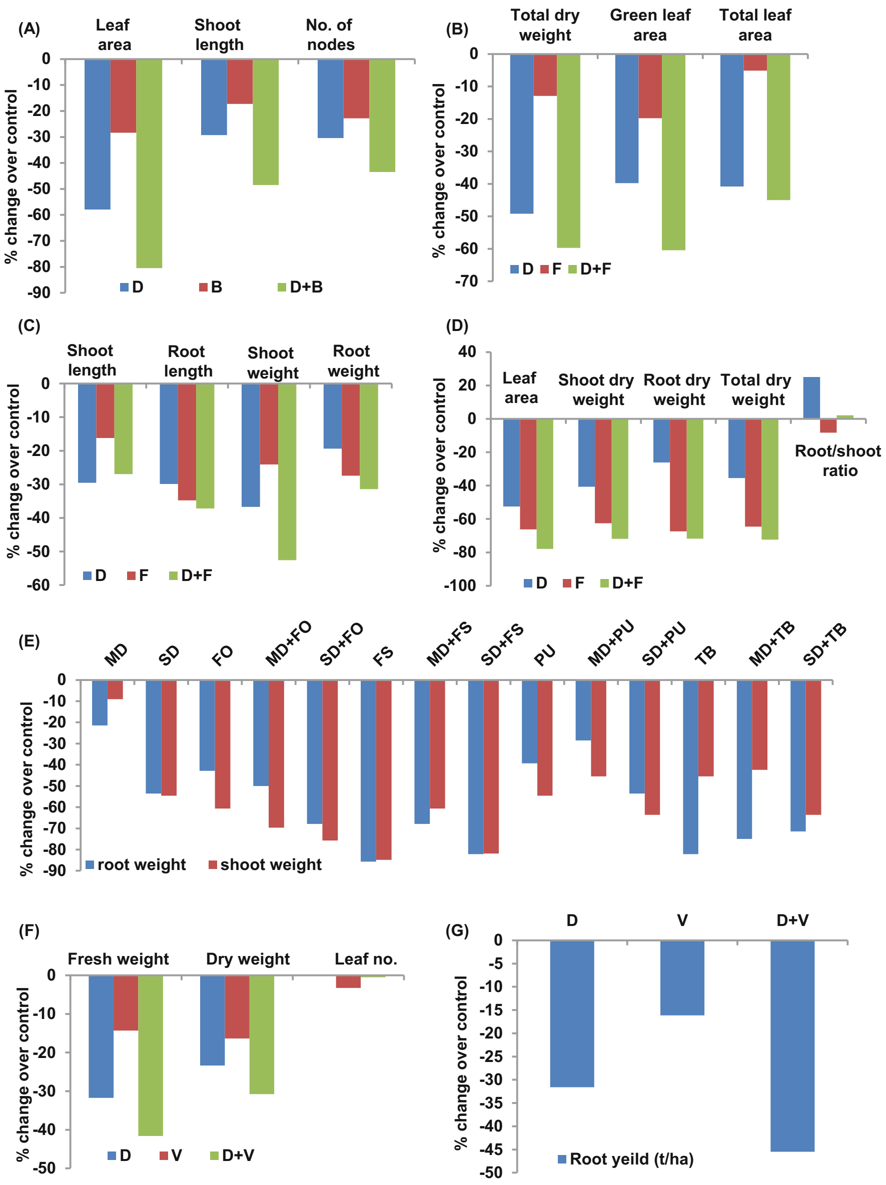


Fig. 1.1 Effects of concurrent drought and pathogen stress on plants. Graphs showing effect of concurrent stresses on yield contributing traits. Drought and bacterial stress (*Xylella fastidiosa*) effect on *Parthenocissus quinquefolia* (McElrone et al. 2001) (**a**), drought and fungal stressor (*Puccinia helianthi*, causes rust) effect on *Helianthus annuus* (Siddiqui 1980) (**b**), drought and fungal (*Drechslera tritici-repentis*) stressor effect on *Triticum aestivum* (Janda et al. 2008) (**c**), drought and fungal (*Macrophomina phaseolina*) stressor effect on *Phaseolus vulgaris* (Mayek-Perez et al. 2002) (**d**), drought and fungal (*Fusarium oxysporum*, FO; *Fusarium solani*,