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Stomate-based defense and environmental cues

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Panchal S, Roy D, Chitrakar R, Price L, Breitbach ZS, Armstrong DW, Melotto M. 2016. Coronatine facilitates *Pseudomonas syringae* infection of Arabidopsis leaves at night. Front. Plant Sci. 7:880.

Abstract

Environmental conditions play crucial roles in modulating immunity and disease in plants. For instance, many bacterial disease outbreaks occur after periods of high humidity and rain. A critical step in bacterial infection is entry into the plant interior through wounds or natural openings, such as stomata. Bacterium-triggered stomatal closure is an integral part of the plant immune response to reduce pathogen invasion. Recently, we found that high humidity compromises stomatal defense, which is accompanied by regulation of the salicylic acid and jasmonic acid pathways in guard cells. Periods of darkness, when most stomata are closed, are effective in decreasing pathogen penetration into leaves. However, coronatine produced by *Pseudomonas syringae* pv. *tomato (Pst)* DC3000 cells can open dark-closed stomata facilitating infection. Thus, a well-known disease-promoting environmental condition (high humidity) acts in part by suppressing stomatal defense, whereas an anti-stomatal defense factor such as coronatine, may provide epidemiological advantages to ensure bacterial infection when environmental conditions (darkness and insufficient humidity) favor stomatal defense.

Plant disease is a successful culmination of three important factors viz. high pathogen virulence, ineffective plant immunity, and favorable environmental conditions. This central dogma of plant pathology is a 50-year-old concept of the disease triangle[1] (Stevens, 1960) and is relevant in all aspects of plant-pathogen interactions[2] (Scholthof 2007). Environmental abiotic factors such as relative humidity (RH) and light conditions have a drastic effect on prevalence of disease in different geographical regions. Plants need to adapt to simultaneous exposure to variable biotic and abiotic stresses, sometimes with opposing effects, for maintenance of healthy whole plant

physiology. For instance, high disease incidence can be explained by the occurrence of climatic conditions that favor pathogen growth and weaken the plant immune system[3] (Panchal et al., 2016a). It is well known that the outbreak of late blight of potato caused by *Phytophthora infestans* that lead to the unfortunate Irish potato famine of 1845 was initiated and spread rapidly mainly because of the unusually wet and cool climatic conditions chronicled for that year[2] (Scholthof 2007). Still, current knowledge on the molecular basis of environment-mediated regulation of plant responses to pathogens is still in its infancy. Moreover, we have gathered evidence that different cell types (*e.g.,* guard cell and mesophyll cell) may have variable molecular responses to the same environmental condition[3] (Panchal et al. 2016) adding additional levels of complexity in plant immune responses.

Plant immune system consists of a complex network of signals tuned to respond to specific types of biotic stresses. One of the first outputs of pattern-triggered immunity (PTI) consists of stomatal defense[4] (Melottto et al. 2006). The microscopic stomatal pores in the leaves are important not only for transpiration and exchange of gases, but also as entry points for some pathogenic microbes, which otherwise could not transit from the phylloplane to the leaf apoplast. However, recognition of microbe-associated molecular patterns (MAMPs) by plant pattern-recognition receptors (PRRs) is a signal to close stomata that serve as guarding gates against microbe invasion[5] (Arnaud and Hwang 2015). A rapid (<2h) bacterium-triggered stomatal closure is also observed when the plant perceives non-pathogens such as *Escherichia coli, Salmonella enterica,* and *Bacillus subtilis*[4,6,7,8] (Melotto et al. 2006; Kroupitski et al. 2009; Roy et al 2013; Kumar et al. 2012).

Molecular mechanisms underlying stomatal defense have been studied mostly in the Arabidopsis-*Pst* pathosystem. This well-studied system has been very useful to decipher both

68 stomatal defense and counter-defense mainly due to the initial PTI response and subsequent

69 induction of coronatine production in the bacterium that overrides PTI[9,10] (Melotto et al. 2017;

70 Xin et al. 2013). This temporal response in the Arabidopsis guard cell is mediated by

71 phytohormones[5] (Arnaud and Hwang 2015). For instance, abscisic acid (ABA), salicylic acid

72 (SA), and jasmonic acid (JA) play important roles in guard cell signaling during Arabidopsis/*P.*

73 *syringae* interaction.

74 Endogenous ABA and SA are important for stomatal closure in response to bacteria or purified

75 MAMPs[4,11,12,13,14,15,16,17] (Melotto et al 2006, Zhang et al 2008, Zeng and He, 2010; Zeng et al

76 2011; Montillet et al 2013, Du et al 2014; Lim et al 2014, Derger et al 2015). By contrast, strong

77 evidence suggests that, similar to its structural and functional mimic coronatine, jasmonoyl-L-

78 isoleucine (JA-Ile) mediates stomatal opening[3,18] (Panchal et al. 2016; Okada et al. 2009).

79 Intriguingly, control of stomatal movement by air RH also seems to operate through hormone

80 signaling. As an example, low RH induced-stomatal closure is associated with ABA

81 biosynthesis[19] (Bauer et al. 2013), whereas activation of stomatal opening by high RH is

82 associated with ABA catabolism[20] (Okamoto et al. 2009). However, we have found that

83 exogenous treatment of ABA does not close stomata to the full extent under high RH as

84 compared to plants at moderate RH[3] (Panchal et al. 2016). This finding indicates that while ABA

85 has a prominent role in RH-mediated stomatal movement, it does not seem to be the only target

86 of high RH in guard cells.

87 Previously, SA-dependent phenotypes have also been shown to be suppressed under high RH[21]

88 (Yoshioka et al. 2001), including the suppression of SA-dependent activation of *PR* genes in

89 Arabidopsis leaves at 24 h after shifting plants to high RH[22] (Zhou et al. 2004). As SA signaling

90 is required for stomatal closure[4,13] (Melotto et al., 2006; Zeng et al., 2011), we performed guard

91 cell-specific analysis and determined that high RH also repressed the expression of *PR1* gene in

92 this cell type[3] (**Fig. 1**; Panchal et al. 2016). On the other hand, JA-responsive genes are

93 upregulated in guard cells within 1h of plant exposure to high RH[3] (Panchal et al; 2016).

94 However, this regulation is independent of the JA-Ile receptor, COI1. COI1-independent and JA-

95 dependent signaling pathway has been previously proposed and induction of some JAZ genes in

96 *coi1* plants has been reported when Arabidopsis leaves are infected with *Sclerotinia*

97 *sclerotiorum*[23] (Stotz et al. 2011). In addition, *P. syringae* pv. *maculicola* ES4326 infection in

98 *coi1-1* plants also leads to induction of JA-regulated genes, indicating that JA response can be

99 activated downstream or independent of COI1[24] (Chen et al. 2001). Moreover, an effector from

100 *Pst* DC3000, HopX1 triggers degradation of JAZ proteins in a COI1-independent manner and

101 promotes stomatal opening[25] (Gimenez-Ibanez et al. 2014). Consistent with this, we observed

102 that the JA biosynthesis genes, *LOX3* and *OPR3* are repressed within 1 h of exposure to high

103 RH[3] (Panchal et al. 2016). This finding suggests that JA-Ile replenishment may not be required

104 as the signaling occurs independent of COI1 in guard cells. Specific branches of the SA and JA

105 signaling pathways regulated by RH are yet to be determined.

106 In several circumstances, JA and SA act antagonistically and some key regulators in this

107 crosstalk have been identified. SA inhibits JA signaling through the regulatory protein,

108 NONEXPRESSOR OF PR GENES 1 (NPR1)[26] (Spoel et al. 2003). By contrast, JA and

109 coronatine inhibit SA biosynthesis genes (isochorismate synthase, *ICS1*) and activate SA

110 degradation genes (benzoic acid/SA carboxyl methyltransferase 1, *BSMT1*) through three NAC

111 transcription factors, ANAC019, ANAC055, and ANAC072[27] (Zheng et al. 2012). However, we

112 observed that both activation of JA and suppression of SA occur simultaneously in guard cells of

113 plants exposed to high RH[3] (Panchal et al. 2016) and hence these pathways are likely to be

114regulated independently by RH. Guard cell response to RH is much quicker (<1h) than that of

115whole leaves (>8h) suggesting the existence of an independent regulation of guard cell signaling

116by RH. However, it is possible that JA/SA antagonism exist in guard cell under high RH at a step

117downstream of the signaling components tested so far, which still needs further investigation.

118Based on current evidence, we propose that the shift of balance between SA and JA signaling

119leads to repression of bacterium-triggered stomatal closure and consequently bacteria that are

120otherwise unable to overcome PTI can still penetrate leaf tissue under high RH (**Fig. 1**).

121High humidity also promotes rapid proliferation of bacteria in the epiphytic phase[28] (Hirano and

122Upper 2000). However, in general, phyllosphere is a water-limiting environment[29] (Beattie 2011)

123that imposes a challenge for epiphytic survival of pathogens in this niche. To counter this

124challenge, bacteria produce extracellular polymeric substances (EPS) to maintain hydration and

125form aggregates on the leaf surface[30,31] (Monier and Lindow 2003; Yu et al 1999). High humidity

126positively affects such aggregate formation of *P. syringae* pv. *syringae* B728a on bean leaf

127surface and aids in rapid proliferation of the bacteria and subsequent entry into the endophytic

128phase[30] (Monier and Lindow 2003). To maintain epiphytic fitness, virulent bacteria can

129physically alter the wettability of the leaf surface by producing biosurfactants[32,33] (Bunster et al.

1301989; Schreiber et al. 2005). Furthermore, bacterial-dependency on high RH to establish

131apoplastic infection while suppressing host immunity has also been demonstrated recently[34] (Xin

132et al. 2016). These observations emphasize that RH participates in multiple steps of molecular

133plant-pathogen interaction and influences its outcome.

134In contrast to high RH that aids plant susceptibility and counteracts stomatal defense, several

135other abiotic factors may favor a robust stomatal defense. In particular, absence of light may lead

136to stomatal closure; indeed, most stomata of C3 and C4 plants are closed at night. This suggests

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137that bacterial penetration of leaves through stomata would be minimal at night. Interestingly, the

138clock proteins CIRCADIAN CLOCK ASSOCIATED 1 (CCA1) and LATE ELONGATED

139HYPOCOTYL (LHY) not only control the circadian stomatal movement, but they are also

140required for flagellin-mediated immune response[35] (Zhang et al. 2013). Disruption of the clock

141activity through CCA1 and LHY resulted in stomata that are less responsive to dark and *P.*

142*syringae* pv. *maculicola*, thus rendering Arabidopsis plants more susceptible to infection at night.

143Furthermore, surface-inoculated plants, but not syringe-infiltrated plants, are more resistant to

144bacterium infection at dusk than at dawn[35] (Zhang et al. 2013). These findings mechanistically

145link stomatal defense and the circadian clock.

146Interestingly, the levels of the two most well-known hormones associated with biotic stress, JA

147and SA, naturally oscillate throughout a 24 h cycle. While the JA level peaks in the daytime, the

148SA level is highest during the night in whole leaves[36,37] (Goodspeed et al. 2012; Grundy et al.

1492015). These oscillations are under the control of the clock and several clock-associated

150proteins[37] (Grundy et al. 2015). If the JA/SA hormone balance determines the opening and

151closing of stomata (**Fig. 1**), then one would assume that inducing JA signaling at night could

152promote stomatal opening. Previously, others and we have determined that coronatine, a

153molecular mimic of JA-Ile, overcomes bacterium-triggered stomatal closure by upregulating JA

154signaling and repressing SA signaling[4,38] (Melotto et al. 2006; Zhang et al. 2015). Consistently,

155*Pst* DC3000 senses the leaf surface, produces coronatine, and opens dark-closed stomata[39]

156(Panchal et al. 2016). It remains to be determined whether coronatine disrupts the natural guard

157cell circadian movement by actively suppressing CCA and LHY1 mediated signaling.

158Nonetheless, it is evident that a stomatal defense-favoring environmental condition such as

159 darkness can be overcome by a virulent pathogen that shifts the hormone balance in guard cell

160 towards JA[3,39] (**Fig. 2**).

161

162 **Acknowledgments**

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