IJASRM
International Journal of Advanced Scientific Research and Management, Volume 3 Issue 12, Dec 2018
www.ijasrm.com
ISSN 2455-6378

Priming Induces Stressful Memories in Plants

Mayukh Das[1], Paulami Koley[1], Monimala Mondal[2] and Surekha Kundu[1*]

[1]Research Scholar, Molecular and Applied Mycology and Plant Pathology Laboratory, Department of Botany, University of Calcutta, 35 Ballygunge Circular Road, Kolkata 700019, India

[2] Research Scholars, Molecular and Applied Mycology and Plant Pathology Laboratory, Department of Botany, University of Calcutta, 35 Ballygunge Circular Road,Kolkata 700019, India

[1*]Assistant Professor, Molecular and Applied Mycology and Plant Pathology Laboratory, Department of Botany, University of Calcutta, 35 Ballygunge Circular Road, Kolkata 700019, India

Abstract

Many pathogens such as fungi, bacteria, virus etc continuously attack the plants. The response by the plants results in the activation of signaling pathways that induce defence reactions. There is growing evidence that priming mechanism induces the plant immune memory. The process of priming involves prior exposure to a biotic or abiotic stresses which makes the plant more resistant to future exposure. Here, we discuss the current knowledge of defence priming. We also discussed the priming of plants with pathogen extract results in the increased disease resistance against similar pathogen which triggers plant immune memory. We discussed the possible mechanisms responsible for the defence priming. Sustained alterations in the levels of proteins or transcription factors could provide an explanation for how plant metabolism is altered by exposure to biotic and abiotic stresses. Alternatively epigenetic changes could play a crucial role by enabling long term changes in gene expression. This potential method can be applied in agricultural fields to improve crop yields and disease resistance capacity of plants.

Keywords: *Priming, Plant immune memory, Biotic and abiotic stresses, Epigenetic change*

1. Introduction

Survival of an organism in its environment is dependent on its inborn immunity or innate immunity (Soto et al., 2006, Staal et al., 2015). This physiological attribute of multicellular organisms not only helps them to fight against pathogens but also to distinguish between "self" and "non self" (Staal et al., 2015). Plants do not have specialized immune cells like T-cells and B-cells or complicated immune systems like somatic adaptive immunity. Yet plants can efficiently discriminate between harmful pathogens and beneficial symbiotic organisms as well as its own cells during defense response (Holub, 2006, Ramirez-Prado et al., 2018). However, there are few reports that sometimes due to lack of distinction between commensalistic organisms and pathogens, the later gain a favor in colonizing the host (Holub, 2006; Soto et al., 2006). So, plants' innate immunity is a result of its cell to cell communication and receiving and perpetuating the signal systematically coming from the attacking site of the pathogen (Staal et al., 2015).

Plant innate immune responses against the pathogens have two types of surveillance systems, one of which acts against the broad range of pathogens that is by strengthening the physical barrier of the host like cell wall, cuticle, waxes etc (Conrath et al., 2006). However this one is so generalised that the plants have evolved another more specific one involving pattern recognition receptors (PRRs) and intracellular nucleotide binding-leucine rich repeat (NLR) proteins, which are encoded by R (resistance) genes (Narusaka et al., 2015).

Plant hormones play an indispensable role in this whole defense context. Several hormones act either synergistically or antagonistically to deter the pathogens. Salicylic acid (SA) act against particularly biotrophic and hemibiotrophic pathogens, whereas Jasmonic acid (JA) and Ethylene (ET) act together against necrotrophic pathogens

(Glazebrook, 2005; Robert-Seilaniantzet et al., 2011). Salicylic acid that plays a role in inducing systemic acquired resistance (SAR), is active broadly against numerous bacteria, fungi, parasites and other microbes by inducing programmed cell death at the infection site and adjacent regions, constituting hypersensitive response (HR) (Dorantes-Acosta A. E. et al., 2012). Therefore perception of MAMP, DAMP, pathogen effectors or abiotic stress leads to normal defence responses in plants (Bruce et al., 2007).

An active field of research in plant defense is defense-priming and plant immunity memory (Song et al., 2018). The development of increased resistance in plants against pathogens using the extract of similar pathogen has recently gained major interest (He et al., 2002). Priming is a process by which an acute environmental stress modifies the plant response to a future challenge and priming induces plant immunity memory (Ramirez-Prado et al., 2018). Memory is a phenomenon by which information about environmental stress is stored in the plant and is maintained for future referral and faster defense response to subsequent stresses (Martinez-Medina et al., 2016). Hence previous exposure to a pathogen results in faster and stronger defence response in the host during the second attack by the pathogen (Bruce et al., 2007). Though priming is known to boost defense response in plants to subsequent infection but the mechanism is still poorly understood.

2. Plants can remember a prior infection:

A few reports are available regarding the priming of host plants with pathogen extracts in order to get better defense response against subsequent infection by the dame pathogen. Nowadays it is widely accepted that plants have the capacity for what can be described as “memory” (Crisp et al., 2016). The effects of biotherapic of Aternaria solani in the severity of early blight disease on tomato plants in the greenhouse have been reported (Carneiro et al., 2010). Prior application of non-pathogenic isolates evoke resistance of Asparagus officinalis on a subsequent challenge with pathogenic strain of the same pathogen (He et al., 2002). Despite this capacity for priming or epigenetic memory, there are many examples where this memory is not achieved and the explanations for this is not well understood (Crisp et al., 2016).

3. Priming induces resistance and plant immune memory:

Priming or a prior exposure of host plants with plant hormones or defence signalling chemicals have been shown to enhance defense response against future attacks by pathogens. SA-induced priming for augmented phenylalanine ammonia-lyase (PAL) gene expression in parsley cell suspensions, β-aminobutyric acid (BABA) induced priming in Arabidopsis for earlier and stronger PR-1 gene expression upon infection by Pseudomonas syringae pv. tomato DC3000 have been reported (Conrath et al., 2006).

4. Complex hormone signaling is involved in plant memory:

When a plant is attacked by a pathogen at one part, the signal is transmitted to distal parts. This SAR response is a SA mediated complicated process where several mobile compounds act as long time signalling molecule and keeping the plant protected from any secondary infection (Staal et al., 2015). Elevated SA level due to local infection raised the reducing power of cell and convert di-sulfide bonded NPR1 oligomer to active monomer (Dong, 2004). Nonexpresser of pathogenesis related gene (NPR1) directly induce SA marker PR genes. Reports show that there are several transcription factors namely TGA2, TGA5 and TGA6 which actually play the intermediate role. This TGA factors act redundantly and bind to the active NPR1 monomer to switch on the PR gene expression cascade (Zhang et al., 2003).

JA, specially jasmonate-isoleucine (JA-Ile) derivatives providing another route of defence against necrotrophic organisms. Upon infection JA accumulate in cells and terminate the transcriptional repression by JAZ (Jasmonate ZIM domain) proteins by COI1 mediating proteolytic degradation of JAZ and thus activating expression of JA responsive genes (Sheard et al., 2010). Not all hormones play a positive role in plant defense and building memory. Abscisic acid (ABA), which has a central role in drought, cold and salt stresses, does not always positively affect biotic stress tolerance. It has been reported that ABA negatively regulate the resistance against Botrytis cinerea infection in tomato by repressing JA/ET response (Staal et al., 2015). On the other hand, ABA is a key regulator in the resistance of Arabidopsis against Pythium irregulare infection. Involvement of ABA responsive elements acts on the promoter region of P. irregulare genes and impart defense against it by affecting the JA biosynthesis (Adie et al., 2007).

5. Possible mechanisms responsible for priming:

Priming is a process by which an acute environmental stress prepared plants to combat against a second challenge. This phenomenon was first described in the study of biotic stress, where the previous exposure to a pathogen resulted in faster and stronger defence response in host during the second attack by a pathogen (Ramirez-Prado et al., 2018). The molecular mechanisms behind priming still not well understood but Bruce et al., 2007 proposed two mechanisms, one involving activation of different proteins, transcription factors and the other involving epigenetic changes. Priming likely involve accumulation of different inactive signalling proteins which are activated after exposure to biotic and abiotic stresses, perhaps by a protein kinase being stimulated by changes in calcium levels. Heat-shock proteins might also get activated by the same route (Bruce et al., 2007). The Arabidopsis Ibs1 mutant is affected in a cyclin-dependent kinase like protein and this mutant could not acquire BABA-induced priming for salicylate-induced defences (Ton et al., 2005). The protein IBS1 functions as a BABA-incuced regulator of the salicylate-induced defence pathway.

Thus primarily, defence priming involves accumulation of different transcription factors that enhance defence gene transcription upon exposure to stress (Conrath et al., 2006). There are forty stress inducible transcription factor genes have been reported in Arabidopsis (Bruce et al., 2007), that might support this molecular mechanism.

Secondly, priming involve some epigenetic changes like modification of DNA by methylation, histone modification or chromatin remodelling without alteration of nucleotide sequence (Ramirez-Prado et al., 2018). This potential mechanism would result in longer term stress imprints to be left in the plant.

6. Future application of plant defense priming and plant defense memory:

Priming imparts tolerance to diseases by bolstering the plant defense machinery rather than directly acting on the pathogens. Therefore though priming we can reduce or altogether avoid the use of harmful chemicals and pesticides. The availability of information on chemical structures of priming agents and recent progress in the mechanistic understanding of plant immune responses have opened up the scope of identifying new or more effecive chemical inducers through rational design (Zhou and Wang, 2018).

Over the last fifteen years, advances in combinatorial chemistry and development of high-throughput screening systems have enabled us to look for synthetic plant immune inducers and led to the discovery new second-generation synthetic elicitors (Bektas and Eulgem, 2015). Through manual inspection one can process only limited numbers of immune elicitors for finding more effective bioactive substructures which are potent immune inducers (He et al., 2017; Luzuriaga-Loaiza et al., 2018). Therefore computational analogues and programmes are being put to use to analyze large amounts of data at ateh same time (Zhou and Wang, 2018).

7. Conclusions

Primed plants can modify their physiology and metabolism upon exposure to further stress more robustly and quickly. Successful plant disease management involve combination of both antimicrobial compounds and priming-inducing agents, thus allowing reduced chemical input into the environment for effective and sustainable plant protection. The knowledge generated in this field of research can be applied in Indian agricultural fields for developing disease tolerance and may be supplied economically to the farmers to increase the crop yields. Most importantly, researches in this fields have contributes significantly to our understanding of plant defense mechanisms specifically with respect to prior infection by a pathogen.

Acknowledgments

The authors are thankful to the Department of Botany, University of Calcutta for providing necessary research facilities. The authors are also grateful to CSIR, Government of India, for providing research funding. The authors declare that there is no conflict of interest.

References

[1] Adie B.A, Perez-Perez J, Perez-Perez M.M, Godoy M, Sánchez-Serrano J.J, Schmelz E.A, Solano R, ABA is anessential signal for plant resistance to pathogens affecting JA biosynthesis and the activation of defenses in Arabidopsis. Plant Cell, 19: 1665-1681, (2007).

[2] Bektas Y, Eulgem T. Synthetic plant defense elicitors. Front. Plant Sci., 5:804, (2015).

[3] Bruce T.J.A, Matthes M.C, Napier J.A, Pickr J.A, Stressful "memories" of plants: Evidence and possible mechanisms. Plant Science, 173: 603-608, (2007).

[4] Chisholm S.T, Coaker G, Day B, Staskawicz B.J, Host-microbe interactions: shaping the evolution of the plant immune response. Cell, 124: 803-814, (2006).

[5] Conrath U, Beckers G. J. M, Flors V, Garcia-Augustin P, Jakab G, Mauch F, Newman M. A, Pieterse C. M. J, Poinssot B, Pozo M. J, Pugin A, Schaffrath U, Ton J, Wendehenne D, Zimmerli L, Mauch-Mani B, Priming: Getting ready for battle. Molecular Plant-Microbe Interaction, 19: 1062–1071, (2006).

[6] Crisp P.A, Ganguly D, Eichten S.R, Borevitz J.O, Pogson B.J. Reconsidering plant memory: Intersections between stress recovery, RNA turn over and epigenetics. Sci. Adv. 2: e1501340, (2016).

[7] Dong X, NPR1, all things considered. Current Opinion in Plant Biology, 7: 547-552, (2004).

[8] Dorantes-Acosta A. E, Sánchez-Hernández C.V, Arteaga-Vázquez M. A, Biotic stress in plants: life lessons from your parents and grandparents. Frontiers in Genetics, 3: 256, (2012).

[9] Glazebrook, J, Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. Annual Review of Phytopathololgy, 43: 205-227, (2005).

[10] He C.Y, Hsiang T, Wolyn D.J, Induction of systemic disease resistance and pathogen defence responses in Asparagus officinalis inoculated with nonpathogenic strains of Fusarium oxysporum. Plant Pathology, 51: 225-230, (2002).

[11] He X. R, Chen X, Lin S.B., Mo X.C, Zhou P. Y, Zhang Z.H. Diversity-oriented synthesis of natural-product-like libraries containing a 3-methylbenzofuran moiety for the discovery of new chemical elicitors. Chemistry open, 6: 102–111 (2017).

[12] Holub E.B, Evolution of parasitic symbiosis between plantsand filamentous microorganisms. Current Opinion in Plant Biology, 9: 397-405, (2006).

[13] Luzuriaga-Loaiza W.P, Schellenberger R, De Gaetano Y, Akong F.O, Villaume S, Crouzet J. Synthetic Rhamnolipid Bolaforms trigger an innate immune response in Arabidopsis thaliana. Sci. Rep, 8:8534, (2018), doi: 10.1038/s41598-018-26838-y.

[14] Narusaka M, Minami T, Iwabuchi C, Hamasaki T, Takasaki S, Kawamura K, Narusaka Y, Yeast Cell Wall Extract Induces Disease Resistance against Bacterial and Fungal Pathogens in Arabidopsis thaliana and Brassica Crop. Plos One, 10(1): e0115864, (2015).

[15] Ramirez-Prado J.S, Abulfaraz A.A, Rayapuram N, Benhamed M, Hirt H, Plant Immunity: From signalling to epigenetic control of defence. Trends in Plant Science, 23(9): 833-844, (2018).

[16] Robert-Seilaniantz A, Grant M, Jones J.D, Hormone crosstalk in plant disease and defense: more than just jasmonate-salicylate antagonism. Annual Review of Phytopathololgy, 49: 317–343, (2011).

[17] Sheard L.B, Tan X, Mao H, Withers J, Ben Nissan G, Hinds T.R,etal. Jasmonate perception by inositol-phosphate potentiated COI1-JAZ co-receptor. Nature, 468: 400-405, (2010).

[18] Song G.C, Ryu C.M. Evidence for volatile memory in plants: Boosting defence priming through the recurrent application of plant volatiles. Mol. Cells, 41(8): 724-732, (2018).

[19] Soto M.J, Sanjuan J, Olivares J, Rhizobia and plantpathogenicbacteria: common infection weapons. Microbiology, 152: 3167–3174, (2006).

[20] Staal J, Dixelius C, Plant Innate Immunity. In: Encyclopedia of Life Sciences (ELS). John Wiley & Sons, Ltd., Chichester, (2009).

[21] Ton J, Jakab G, Toquin V, Flors V, Iavicoli, A, Maeder M.N, Metraux J.P, Mauch-Mani B, Dissecting the b-aminobutyric acid-induced priming phenomenon in Arabidopsis, Plant Cell, 17: 987-999, (2005).

[22] Zhang Y, Tessaro M. J, Lassner M, Li X, Knockout Analysis of Arabidopsis Transcription Factors TGA2, TGA5, and TGA6 Reveals Their Redundant and Essential Roles in Systemic Acquired Resistance. The Plant Cell, 15: 2647-2653 (2003).

[23] Zhou M, Wang W. Recent Advances in Synthetic Chemical Inducers of Plant Immunity. https://www.frontiersin.org/articles/10.3389/fpls.2018.01613/full.