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Review

Views on biotic stress response links to transcription factors in plants

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Abstract:
It is known that biotic and abiotic stress hinder plant growth and development. The expression of transcription factors in response to pathogen-related (PR) proteins is observed. The role of various signalling pathways and stress-responsive genes in the defence mechanism against biotic stress is documented. Therefore, it is of interest to document data related to the molecular identification and genetic editing of transcription factors linked to stress in crop improvement. Hence, we review known information (basic structure, and regulatory mechanisms) on four transcription factor families (WRKY, NAC, bZIP, MYB) involved in biotic stresses.

Keywords: Transcription factors; biotic stress; WRKY; NAC; bZIP; MYB; PR protein; Salicylic acid; Jasmonic acid; hypersensitive cell death.

Background:
Plants are continuously exposed to both biotic and abiotic stress from germination throughout the life cycle [1]. Stress affects plant growth and reproduction by altering physiological, molecular, and biochemical processes with low yield [2]. The world population will reach approximately 8 billion by 2050 and food demand is high [3]. Plants are more prone to stress under field condition in response to changing climatic conditions with different types of disease outbreaks[4]. It is known that the rate of *Macrophomina phaseolina* infection in bean plants was found high under drought stress as compared to the control [5]. The primary defence processes constitutively activate the complex signalling

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mechanisms within the plants to balance stress [6]. Plants activate different types of ion channels, kinase cascades, phytohormone signalling pathways like abscisic acid (ABA), jasmonic acid (JA), salicylic acid (SA), and ethylene signalling pathways and sometimes accumulate and reprogram the genetic machinery to adequate defence mechanisms and to increase tolerance against stress [5, 6]. Numerous genes are activated and consequently, various proteins and phytohormones are produced in the cell through signal transduction processes [7, 8]. The activation of such stress-responsive genes and production of various proteins and phytohormones are regulated by different types of specific transcription factors (TFs) [9, 10] as shown in **Figure 1**. Transcription factors are known as regulators of genes and or gene clusters [11][12]. Transcription factors (TFs) bind with the RNA polymerase and recognizes the promoter and transcription process begins [13, 14]. The activation and inactivation are regulated by the transcriptional control mechanism using transcription factors (TFs) [15]. Transcription factors comprise clusters of domains like DNA binding domain, oligomerization site, transcription regulation domain, and nuclear localization domain for the activation and regulation of a gene or clusters of a gene [16]. Sequence analysis shows gene duplication, translocation, exon capture, and mutation are the processes by which transcription factors are identified [17, 18]. During stress condition, the transcription factors help in activating various genes and the synthesis of various proteins [19, 20]. The induced genes help in protecting the cells from stress by synthesizing various functional proteins and also regulates the signal transduction in stress condition by synthesizing regulatory proteins [19, 21]. Different types of transcription factors like WRKY, NAC, MYB, MYC, bZIP etc. have been identified to play a vital role in plants to response against stressed conditions [22][23][24]. Hence, we review known information (basic structure, and regulatory mechanisms) on four transcription factor families (WRKY, NAC, bZIP, MYB) involved in biotic stresses.

WRKY transcription factors and its basic structure:

WRKY is one of the most important transcription factor families of plants. A large number of WRKYs are found in plants. About 109 types of WRKY transcription factor are discovered in rice and approximately 74 in Arabidopsis [25]. WRKY transcription factors comprise ≈60 amino acid long four-stranded β-sheet WRKY DNA binding domains (DBD) and zinc-finger motifs [26]. Based upon the aminoacid sequences it has shown that β1 and β2 are highly conserved, while β3 and β4 show differences in their amino acids. They also comprise basic nuclear localization domain, a kinase domain, leucine zippers, serine-threonine, glutamine, a proline-rich region, and TIR-NBS-LRR domain [27]. Based upon the DBD and zinc-binding motifs WRKYs are divided into three groups [25].

[1] **Group I -** 2 WRKY DNA binding domain.
[2] **Group II** - Single DBD with altered C2H2 zinc finger.
[3] **Group III** - Single DBD with altered C2H zinc finger

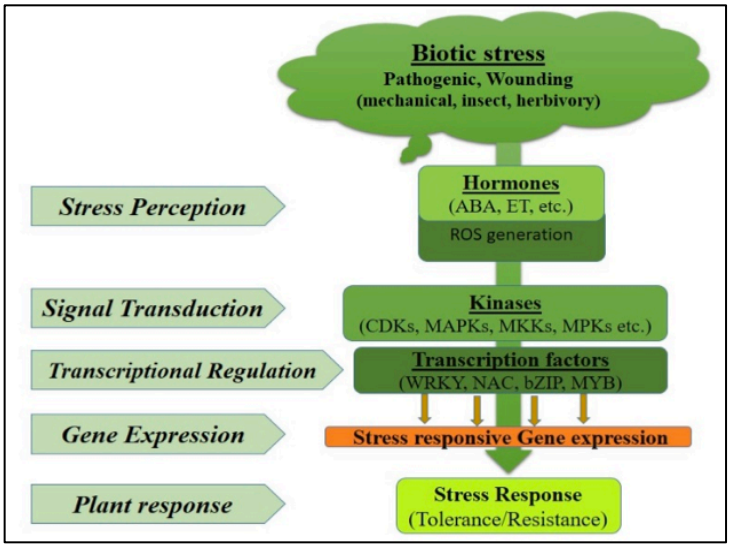

Figure 1: Schematic representation of involvement of transcription factors in plant biotic stress response

Based upon the primary amino acid sequence, Group II is further classified into IIa, IIb, IIc, IId, and IIe [25]. The W- boxes and the clustered W-boxes which are present in the promoter region of the downstream gene interact with the WRKY transcription factors and regulate dynamic web signalling through kinase and other phosphorylation cascades [28]. There are also some WRKYs, which also bind with the non-W-box element, for example, OsWRKY13 (*Oryza sativa* WRKY13) binds to both binds to both W-box and PRE4 element (TGCGCTT). HvWRKY46 (*Hordeum vulgare* WRKY46) is another type of transcription factor which binds both W-box and sugar responsive element (SURE) TAAAGATTACTAATAGGAA, NtWRKY12 (*Nicotiana tabacum* WRKY) can bind to the SURE like element not to the W-box [25]. Instead of WRKYGQK, NtWRKY12 bear a sequence (WRKYGKK) and interact with the WK- box TTTTCCAC. The DNA binding domain of WRKY is highly conserved, under a particular condition the activation of downstream genes helps the conserved region of the DNA binding domain to interact with the W-boxes of cis-motif [25, 29].

The WRKY domain and W-box:

The DBD is the main feature of the WRKY transcription factors. Due to the presence of WRKY signature sequences, it termed as WRKY

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domain [30]. In a few cases, the signature term WRKY is displaced by WRRY, WSKY, WKRY, WVKY or WKKY [31, 32]. The WRKY domain consists of 60 amino acid residues with WRKY signature, and at C-terminus, there is a zinc-finger structure. The pattern of zinc-finger is either $Cx_{4-5}Cx_{22-23}HxH$ or $Cx_7Cx_{23}HxC$. In group I and group II the zinc-finger pattern is C_2H_2 type, while in groupIII the pattern is C_2-HC type [33][34]. As mentioned above that there are three groups of the WRKY transcription factor, and based upon the primary amino acid sequences group II is further classified into five subgroups. The details structure of WRKY transcription factor is shown in **Figure 2**.

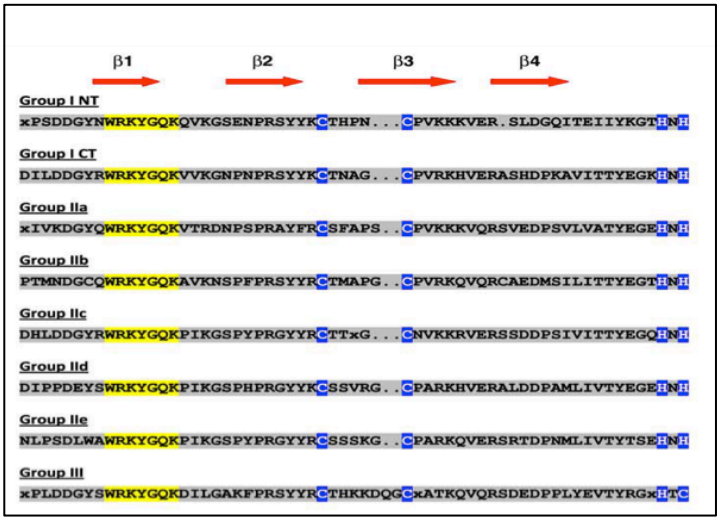

Figure 2: The WRKY domain; the yellow colour highlighted region indicates WRKY signature [34]. While blue colour highlighted region denoted C_2H_2 and C_2-HC zinc finger motifs. The NT and CT represent the N-terminus and C-terminus regions of WRKY protein. The red arrows indicating β strands of WRKY TFs

Molecular mechanisms of WRKY transcription factors regulating plant responses against pathogens

The involvement of WRKY TFs in plants defence responses is mentioned in **Table 1**. Some WRKY TFs are involved in the regulation and expression of other genes in defence mechanism [27]. During the pathogen attack, OsMKK4-OsMPK3/OsMPK6 activates the OsWRKY53, and hence overexpression of OsWRKY53 thereby leading to the activation of defence response against blast disease in rice [35]. OsWRKY28 act as a modulator to maintain responses at the appropriate level by attenuating activation of defence-related gene expression level against blast disease and also plays an efficient role in arsenic stress mitigation [36, 37]. In the case of OsWRKY30, overexpression of TF leads to the activation of Salicylic acid (SA) responsive genes which play a vital function in plant defence mechanism [38]. Panicle blast1 (Pb1) gene interact with OsWRKY45 and activates the SA pathway and mediates blast resistance **figure 3** [39]; OsWRKY77 regulates Pathogen Responsive (PR) gene expressions and basal resistance to the bacterial pathogen. On the other hand, OsWRKY47 upregulate the secondary metabolism which enhances the resistance against the pathogen in transgenic plants [28, 40]. Overexpression of OsWRKY76 down-regulates the induction of activation of specific PR or the gene involved in the phytotoxin synthesis after infection with blast fungus [41].

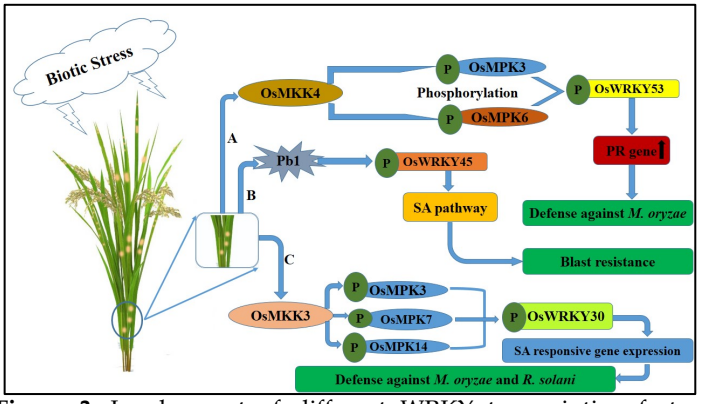

Figure 3: Involvement of different WRKY transcription factors regulated by MKKs, MPKs, and Pb1 in plant biotic stress response. The pathogen attack activates MKKs, MPKs, and Pb1, which further phosphorylates and activates WRKY TFs. A) After the pathogen attack, OsMKK4 activates OsMPK3 and OsMPK6, which further activates and OsWRKY53. The activated OsWRKY53 binds with W-box of PR gene and upregulate the gene expression in rice plants and provides resistance against M. oryzae. B) Pb1 activates OsWRKY45 and provides resistance against blast disease by enhancing SA mediated defence mechanism in rice plants. C) Similarly OsMPK3/7/14 activated by OsMKK3 interact with OsWRKY30 and enhances SA mediated defence against M. oryzae and R. solani.

In another study, it has been observed that after infection with blast fungus, various PR genes are co-expressed along with OsWRKY22 [22]. Overexpression of OsWRKY76 increase the susceptibility to the pathogen *Magnaporthe oryzae* and suppress the activity of some PR genes or the genes involved in the synthesis of phytotoxins after pathogen infection [42]. In transgenic plants, overexpression of OsWRKY30 is associated with jasmonic acid synthesis [43]. A group of WRKY TFs such as OsWRKY28, OsWRKY71, OsWRKY76,

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OsWRKY62 unregulated the PR10 and interact functionally to restrain plant innate immune responses [44]. Overexpression of OsWRKY45-1are is highly susceptible to *Xanthomonas oryzae pv oryzae* (Xoo) and *Xanthomonas oryzae pv oryzicola* (Xoc). While overexpression of OsWRKY45-2 is resistance to *Xanthomonas oryzae pv oryzae* (Xoo) and *Xanthomonas oryzae pv oryzicola* (Xoc) [45, 46]. On the other hand, overexpression of both is highly resistance to *Magnaporthe grisea* [45]. The phosphorylated OsWRKY33 bind to the W-boxes of PR gene promoter and mediates SA dependent defence mechanism [25]. OsWRKY62 negatively regulates the plants immune responses by suppressing the pathogen-related genes [47]. The light-dependent OsWRKY03 along with OsNPR1 and several other pathogen-related genes act as a transcriptional activator of salicylic acid (SA) and jasmonic acid (JA) defence responsive pathway [48].

In transgenic plants, overexpression of AtWRKY8 makes the plant highly susceptible to P. *syringae* but high resistance to *Botrytis cinerea* [22]. In Arabidopsis SA dependent defence mechanism is regulated by AtWRKY25, but AtWRKY25 suppress the JA signalling defence mechanism in Arabidopsis [49]. StWRKY8 plays an important role against the late blight in potato by synthesizing benzylisoquinoline alkaloid [50]. In transgenic poplar, PtrWRKY89 interact with the pathogen-related (PR) gene and activates the SA signalling defence pathway against the pathogen, while in transgenic Arabidopsis, overexpression of PtrWRKY89 reduces plant resistance against pathogen [51]. PtrWRKY18 and PtrWRKY35 are involved in the activation of PR genes, the activated PR genes act against a biotrophic pathogen Melampsora. In this process, the SA-dependent defence mechanism is also involved [52].

Table 1: WRKY transcription factors and their specific role in the plant defence mechanism.

WRKY TFs	Phytopathogen	Regulatory mechanism	Reference
Oryza sativa			
$OsWRKY_{53}$	Magnaporthe grisea	OsMKK4-OsMPK3/OsMPK6 mediated activation of $OsWRKY_{53}$ regulates the defence-related gene expression and protects rice plants against blast diseases.	**[35]**
$OsWRKY_{28}$	Magnaporthe grisea	Negatively regulates the plant immune responses by constricting the defence-related gene expression levels.	**[36]**
$OsWRKY_{30}$	Xanthomonas orizae pv. orizae (Xoo)	Overexpression of $OsWRKY_{30}$ provides resistance against Xoo infection activating of the SA responsive gene.	**[53]**
$OsWRKY_{45}$	Magnaporthe grisea	Pb1 interact with the OsWRKY45 and initiates the SA signalling pathway, and provides resistance against blast.	**[39]**
$OsWRKY_{77}$	Pseudomonas syringae pv.	Upregulates the PR gene expression (PR1, PR2, and PR5) and provide resistance to a bacterial pathogen.	**[40]**
$OsWRKY_{47}$	Magnaporthe grisea	$OsWRKY_{47}$ interacts and up-regulates the expression of genes involved in signaling and secondary metabolism and enhanced resistance against rice blast caused by Magnaporthe grisea.	**[54]**
$OsWRKY_{76}$	Magnaporthe grisea	After infection overexpression of $OsWRKY_{76}$ negatively regulates blast resistance in rice by suppressing the activation of PR genes as well as phytotoxin synthesis genes.	**[55]**
$OsWRKY_{22}$	Magnaporthe grisea	A large number of resistance and defence-related genes are co-expressed with $OsWRKY_{22}$.	**[56]**
$OsWRKY_{30}$	Rhizoctonia solani, Magnaporthe grisea	In transgenic plants, overexpression of OsWRKY30 provides resistance against fungal attack by up-regulating PR genes and enhancing endogenous JA accumulation.	**[57]**
$OsWRKY_{6}$	Xanthomonas campestris pv. campestris (Xcc)	OsWRKY6 enhanced the expression of the reporter gene downstream of OsPR1 promoter, and act as a positive transcriptional regulator in rice plants.	**[58]**
$OsWRKY_{28}$ $OsWRKY_{71}$ $OsWRKY_{76}$ $OsWRKY_{62}$	Xoo	Overexpression of these TFs up-regulates the PR10 gene and interact functionally to modulate plant innate immunity.	**[59]**
$OsWRKY_{67}$	Magnaporthe oryzae and Xanthomonas oryzae pv. oryzae (Xoo)	Overexpression of $OsWRKY_{67}$ provides plants disease resistance by up-regulates the expression of PR genes like PR1a, PR1b, PR4, PR10a, and PR10b.	**[60]**
$OsWRKY_{33}$	Magnaporthe grisea	After pathogen attack OsWRKY33 enhances SA dependent defence responses against the pathogen.	**[61]**
$OsWRKY_{45\text{-}1}$ (japonica)	Xoo and Xoc	Overexpression of OsWRKY45-1 plants is highly susceptible while OsWRKY45-1 knockout plants are resistant to the Xoo and Xoc.	**[45]**

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$OsWRKY_{45-2}$ (indica)	Magnaporthe grisea	Overexpression of OsWRKY45-2 is highly resistance while suppression of OsWRKY45-2 is highly susceptible to Xoo and Xoc. Overexpression of both OsWRKy45-1 and OsWRKY45-2 are highly resistance to M. grisea.	**[45]**
$OsWRKY_{13}$	Xoo	OsWRKY13 play a crucial role in disease resistance mechanism.by regulating several genes involved various signalling pathway.	**[62]**
$OsWRKY_{13}$	Xoo	$OsWRKY_{13}$ positively regulates plants defence mechanisms by interacting with pathogen induced proteins.	**[63]**
$OsWRKY_{71}$	Xanthomonas oryzae	$OsWRKY_{71}$ activates disease-related genes with the help of chitinase.	**[64]**
$OsWRKY_{62}$	Xoo	$OsWRKY_{62}$ negatively regulates the plant's innate immune response by suppressing the activation of defence-related genes.	**[65]**
Other WRKY TFs	Regulatory mechanisms		Reference
$AtWRKY_{8}$	Overexpression of AtWRKY8 in transgenic plants is more susceptible towards P. syringae but high resistance to B. cinerea.		**[66]**
$AtWRKY_{25}$	$AtWRKY_{25}$ is regulates the SA signalling pathway in plant defence mechanism against the pathogen.		**[67]**
$StWRKY_{8}$	$StWRKY_{8}$ provides potato plants resistance against late blight by regulating Benzyl-isoquinoline synthesis.		**[50]**
$PtrWRKY_{89}$	In transgenic Poplar, $PtrWRKY_{89}$ provides plant resistance by regulating the pathogen-related protein genes and activating salicylic acid depending defence pathways. While in transgenic Arabidopsis, overexpression of PtrWRKY89 decreases the resistance against pathogen.		**[51]**
$PtrWRKY_{18}$, $PtrWRKY_{35}$	$PtrWRKY_{18}$, $PtrWRKY_{35}$ regulates the activity of PR genes and SA mediated defence signaling pathways in plants and provides resistance against Melampsora, a biotrophic pathogen.		**[52]**

Involvement of WRKY TFs in SA mediated plants defence mechanisms:

The SA signalling defence mechanism regulates most of the defence mechanisms in plants. WRKY TFs play a vital role in the activation of SA signalling pathways. WRKY TFs mainly binds to the W-boxes of PR genes and regulates them in the defence responses against pathogens [68]. They are also involved in the activation of SA biosynthetic genes Isochorismate synthase (ICS) and PBS3, which helps in the accumulation of SA. Isochorismate synthase1 (ICS1) play a key role in SA biosynthesis, the expression of ICS1 is regulated by WRKY18, WRKY28, WRKY56 [69, 70]. SID2 (SA induction deficient2) is an ICS involved SA biosynthesis as shown in **Figure 4**. The mutant SID2 allele reduces the expression of SA biosynthesis in Arabidopsis and hence plants are more susceptible to the pathogen [71].

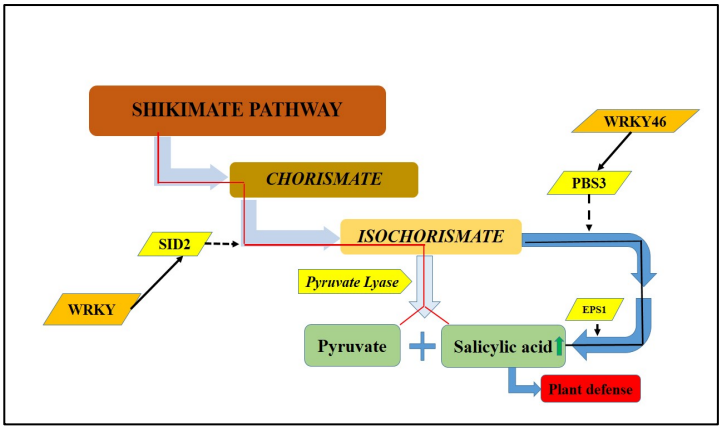

Figure 4: Schematic representation for the involvement of WRKY transcription factors in SA biosynthesis.

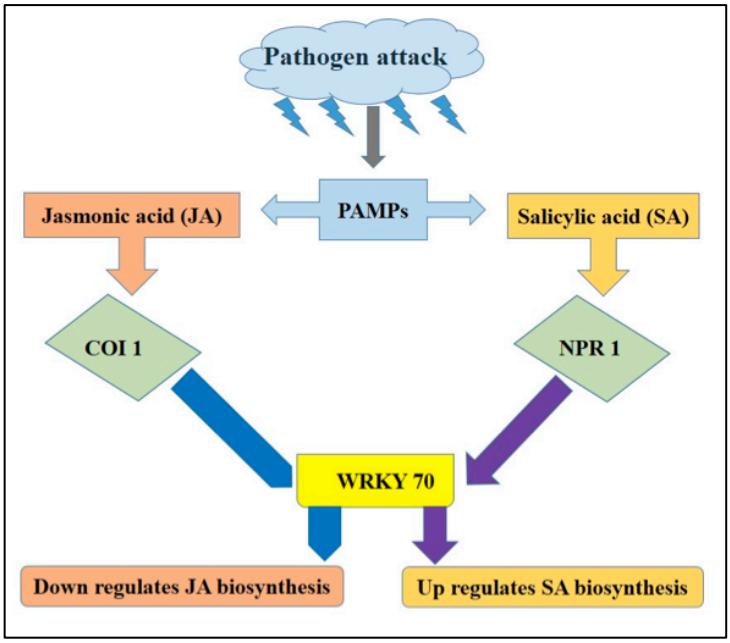

Figure 5: Schematic representation of WRKY70 crosstalk in SA and JA signalling after pathogen attack. Purple arrow box represents positive regulation while the blue box represents negative regulation.

SA-O-β glucoside (SAG) regulates the SA dependent defence mechanism. PBS3 (Auxin responsive GH3 family protein) involved in the pathogen-induced accumulation of SA-O-β glucoside (SAG), the activity of PBS3 is enhanced by WRKY46 [72]. PBS3 is a member of acyl-adenylate/thioester forming enzyme family, which along with EPS proceeds SA biosynthesis **(Figure 4)** [73, 74]**.** Non-

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expresser PR gene 1 (NPR1) is a transcription cofactor, present in the cytosol as high oligomeric complex. Due to the SA accumulation, the oligomeric complexes are converted into NPR1 monomers and translocated into the nucleus upon the activation by SA. Some of the NPR1 homomeric complexes remain in the cytoplasm [75]. Inside the nucleus, they help in the activation of SA responsive PR genes. The remaining SA activated NPR1 in the cytosol are involved in downregulation of JA responsive PR gene expression by delivering the negative regulator of JA-responsive genes in the nucleus [76]. Many transcription factors are involved in the crosstalk between SA and JA signalling pathways. WRKY 70 transcription factor is one of the examples, which involve in the crosstalk between SA and JA signalling pathways. WRKY70 upregulates the SA signalling pathway and downregulates the JA signalling pathways in Arabidopsis [76] **(Figure 5).**

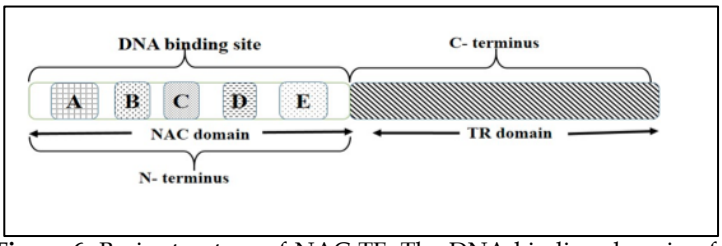

Figure 6: Basic structure of NAC TF. The DNA binding domain of N- terminus consists of five domains (A-E). C-Terminus is occupied by transcriptional regulatory (TR) domain, which interacts with stress-responsive genes either for activation or repression.

NAC transcription factor family

The name NAC is derived from the first letter of the three transcription factors (**N**AM, **A**TAF1-2, **C**UC) [77]. More than 100 NAC transcription factors are identified in rice, Arabidopsis, tobacco, potato, soybeans etc. hence, it is believed that NAC TFs occupies the largest part of the transcription factor. NAC TFs are widely distributed in land plants [78]. The name NAC was derived from the transcription factors NAM (no apical meristem, from Petunia), ATAF1, ATAF2, and CUC (Cup Shaped Cotyledon, from Arabidopsis). Highly conserved N-terminal DNA binding domain was first observed in NAM and CUC2 transcription family. The N-the terminal region is 150 amino acid sequence long [79]. The N-terminus is classified into five sub-class (A-E) shown in **figure 6**. The conserved sub-domains C and D binds to DNA whereas the functional dimer is formed by the subdomain A. The functional diversity of NAC genes is maintained by subclass B and E [80]**.** The C-terminus region of NAC proteins are highly diverse, the diverse C-terminus region does not encode any functional proteins. The C-terminal region functions in transcriptional activation of protein binding domain and transmembrane motif [81]. In model plants as well as in crop plants, it has shown that the NAC TFs play a vital role in responses to biotic as well as abiotic stresses [78, 82]. So it is believed to be the NAC TFs may be used in crop improvement against biotic and abiotic stresses.

Molecular mechanisms of NAC transcription factors regulating plant responses against pathogens:

NAC, the plant-specific transcription factors are believed to be the largest family of transcription factors. They play a vital role in the protection of plant against the pathogen attack [78]. They protect the plant either by activating various phytohormone signalling pathways (like JA, SA, ABA, ET signalling pathways), or interacting with PR genes or by performing hypersensitive cell death. Some of the NAC TFs also acts as a negative regulator and expression of these TFs makes the plant susceptible to pathogen attack [83]. The involvement of NAC TFs as a positive as well as a negative regulator in plants is mentioned in **Table 2**. GhATAF1, ATAF1, ATAF2, ANAC042, CBNAC1, ANAC019, ANAC055, ANAC072 are some of the examples of NAC TFs which act as a negative regulator in plant defence mechanism, while OsNAC6, ONAC122, ONAC131, HvNAC6, VvNAC1 and OsNAC4 are some example of positive regulators in plants. The expression of ATAF1, ATAF2, NTL6, and HvNAC6 in plant defence mechanism is shown in **figure 7**. Several NAC protein positively regulates plant defence by triggering PR genes, or by inducing hypersensitivity or by cell death in the infected cells [84].

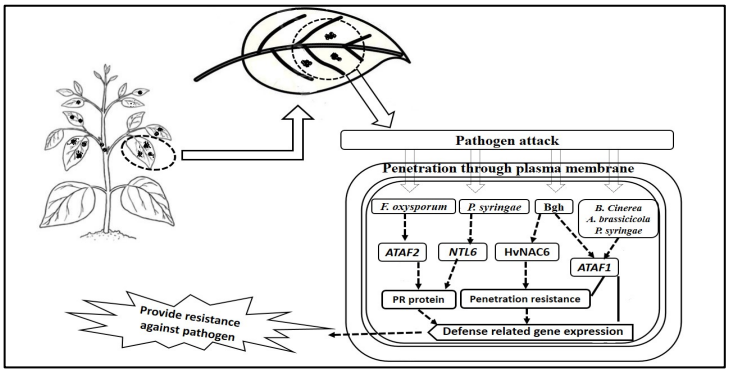

Figure 7: Gene expression regulation by NAC TFs during pathogen attack, dot arrow line showing positive regulatory mechanisms while solid line showing negative regulatory mechanisms.

Expression of GhATAF1 suppress the JA signalling defence mechanism in cotton and increases susceptibility towards fungal infection [85]; ATAF1 and ATAF2 act as a negative regulator in

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plant defence mechanism, ATAF2 suppress the expression of PR genes a result of which the plant becomes more susceptible[7, 86]; CBNAC along with SNI1 acts as a repressor protein which suppress the plant basal defence system [87]. The expression of OsNAC6 in transgenic plants act as a positive regulator in plant defence mechanism against the fungal disease [83]; ONAC122 and ONAC131 play an important function in various phytohormone signalling pathways and protects the plant from pathogen attack [88]; HvNAC6 and VvNAC1 act as a positive regulator in plant defence mechanism against *Blumeria graminis hordei*, *B. cinerea* and *Hyaloperonospora arabidopsidis* [83]. OsNAC4 is one of the important NAC transcription factors, after infection, the expression of OsNAC4 involve in hypersensitive cell death of the infected cell and protect the uninfected cell and the plants [89]
.

Table 2: NAC transcription factors and their specific role in the plant defence mechanism.

bZIP TFs	Phytopathogen	Regulatory mechanisms	Reference
GhATAF1	Verticillium dahliae, Botrytis cinerea.	Act as a negative regulator by suppressing JA signaling pathway.	[90]
ATAF1	Botrytis cinerea.	ATAF1 acts as a negative regulator in Arabidopsis by suppressing defence related regulatory mechanisms.	[91]
ATAF2	Fusarium oxysporum.	ATAF2 suppress the activity of PR genes and makes the plants susceptible to pathogen.	[92]
ANAC042	Alternaria brassiciola.	ANAC042 inhibits camalexin synthesis and makes the plant susceptible to Alternaria brassiciola.	[93]
CBNAC	Pseudomonas syringae.	CBNAC along with SNI1 acts as a repressor protein and suppress the plant basal defence.	[94]
ANAC019, ANAC055, ANAC072	Pseudomonas syringae.	Acts as a negative regulator by helping bacterial propagation.	[95]
OsNAC6	Magnaporthe grisea.	In transgenic rice overexpression of OsNAC6 makes the plant resistant to blast disease.	[96]
ONAC122, ONAC131	Magnaporthe grisea.	ONAC122 and ONAC131 involved in various phytohormone signalling pathways and acts as a positive regulator against M. grisea.	[88]
HvNAC6	Blumeria graminis f.sp. hordei (Bgh)	Act as a positive regulator against the Bgh.	[97]
VvNAC1	Botrytis cinerea.	In Arabidopsis, overexpression of VvNAC1 act as a defence mechanism against B. cinerea and Hyaloperonospora arabidopsidis.	[98]
OsNAC4	Magnaporthe grisea	After infection, the OsNAC4 perform the hypersensitive cell death of the infected cell and protect the plants.	[89, 99]

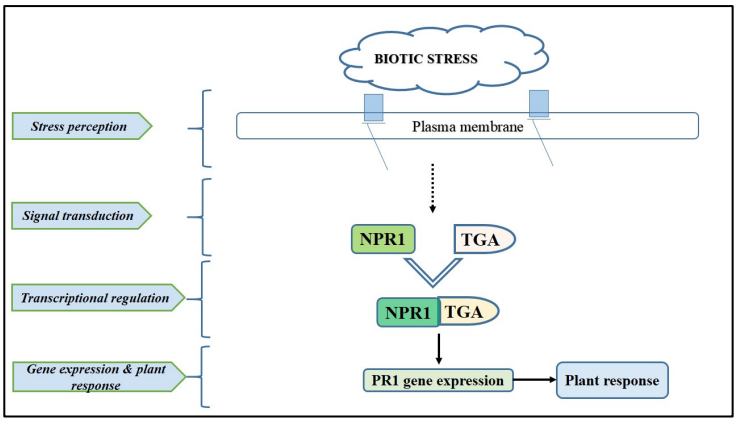

Figure 8: Schematic model for the involvement of bZIP TFs against biotic stress management. NPR1 is a cofactor for TGA TFs of bZIP family, after signal transduction NPR1 interacts with TGA TFs and regulates the expression of PR1 gene and protects the plants from biotic stress. Some of the examples of bZIP transcription factors in plant defence mechanism against biotic stress are mentioned in Table 3.

bZIP transcription factor family:

Basic region/leucine zipper motif (bZIP) is another type of transcription factor involved in the regulation of plant defence mechanism in biotic as well as abiotic stress [100]. Transcription factors are mainly classified based on their DNA binding domains. A basic region is present in the N-terminal region of bZIP TFs which binds to the DNA and a leucine zipper dimerization motif in the C-terminal region[101]. bZIP transcription factors are commonly found in all eukaryotes [102]. The basic region of bZIP is 16 amino acid residues long containing N-X_7-R/K motif, that contact with DNA while the leucine zipper contains seven repeats of leucine or other bulky amino acid residues, which are hydrophobic in nature [102, 103]. The interaction between hydrophobic sides of two subunits of bZIP protein creates a coiled-coil structure. Hence, it is known as a zipper[102]. The bZIP protein of plants mainly interacts with the ACGT core. The A-box (TACGTA), C-box (GACGTC), and G-box (CACGTG) are the region where the bZIP TFs interact [104]. Based upon the similarity of basic DNA binding region the Arabidopsis bZIP (AtbZIP) proteins are subdivided into ten groups: A, B, C, D, E, F, G, H, I, and S. Most of the groups interact with similar cis-elements [103]. TGA transcription factors belong to the member of the bZIP TFs family [105]. In Arabidopsis ten TGA transcription factors were identified, seven out of ten were differentiate based upon interaction

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with NPR1 protein. NPR1 acts as a cofactor of TGA transcription factors and promote binding of TGA to the promoter of the PR gene and transcription of the PR gene starts [75, 106, 107]as shown in **Figure 8**. The bZIP transcription factors play a vital role in the plant defence mechanism against biotic and abiotic stress [108]. During biotic stress (wounding, pathogen attack etc.) the bZIP transcription factor protects the plant by phytohormone signalling pathways or by hypersensitive responsive defence mechanism [109].

Table 3: Role of bZIP transcription factors in plant defence mechanism.

bZIP TFs	Host plant	Regulatory mechanisms	Reference
MebZIP3 and MebZIP5	Manihot esculenta	Overexpression of MebZIP3 and MebZIP5 TFs enhanced plant resistance against Cassava blight disease.	[110]
CabZIP2	Arabidopsis thaliana	Overexpression of CabZIP2 transcription factors enhanced plant resistance against Pseudomonas syringae	[111]
CabZIP1	Capsicum annuum L.	Auxin synthesis, defence against pathogen.	[112, 113]
OBF protein	Arabidopsis thaliana.	Induction of PR gene expression by the SA signalling pathway.	[114]
AtbZIP10	Arabidopsis thaliana	Protect the plant by cell death, a positive regulator of HR associated pathogen recognition.	[115]
TGA	Arabidopsis thaliana	PR gene-mediated defence mechanism.	[75]
rTGA2.1, rTGA2.2, rTGA2.3	Oryza sativa	SA mediated defence mechanism.	[116]
G/HBF1	Glycine max	Regulation of defence-related genes.	[108]
VvbZIP23	Vitis vinifera	Regulation of biotic and abiotic stress responses.	[117]
PPI1	Capsicum chinense	Regulates defence gene expression against the pathogen.	[118]
OsTGAP1	Oryza sativa	Involve in diterpenoid biosynthetic gene regulation.	[119]
SlAREB1	Solanum Lycopersicum	Upregulation of PR proteins.	[120]

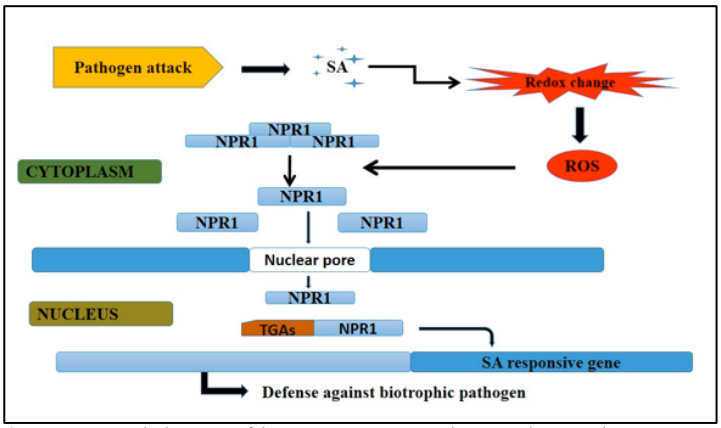

Figure 9: Modulation of bZIP TFs against biotrophic pathogens via the SA signalling pathway. Pathogen attack induces SA synthesis which changes cellular redox potential by the formation of ROS. Change in redox potential breaks the clusters of NPR1 into the cytoplasm and translocate them into the nucleus where it interacts with TGA TFs of bZIP family and regulates the expression of SA responsive genes and promotes defence mechanisms against biotrophic pathogens.

Molecular mechanisms of bZIP transcription factors regulating plant responses against pathogens:

After pathogen attack, plant immune system tries to recognize the pathogen via interaction between resistance (R) gene and the protein produced by pathogen, the process is known as immunity triggered by an immune effector (TSI) [121]. In the absence of the corresponding gene, plants are not able to activate the defence mechanism and become more susceptible to the pathogen [122]. The immunity triggered by Pathogen associated Molecular Pattern (PTI), a recognition system in plants. PTI is activated by SA [108]. Non-expresser to pathogen-related gene1 (NPR1) is an important factor that involves in SA signalling defence mechanism. Under normal condition the most of the NPR1 present in the cytoplasm in oligomeric form. After the pathogen attack, the SA is synthesized and changes the cellular redox state by the formation of ROS. ROS converts the oligomeric NPR1 to monomeric forms. The monomeric NPR1 genes than translocates to the nucleus. The NPR1 interacts with TGA transcription factors of a bZIP protein family and bind to SA-responsive gene promoters and produce defense signal against biotrophic pathogen [75] as shown in **Figure 9**.

MYB transcription factor family

The MYB transcription factors are present in all eukaryotes. The first MYB protein was identified in Avian myeloblastosis virus [123]. C1 MYB was the first identified plant MYB gene, which involves in anthocyanin biosynthesis in *Zea maize* [124]. The MYB TFs has two regions **a)** the N-terminal region which contains highly conserved one or more MYB domains; **b)** the C-terminal region, which performs the regulatory functions. The MYB domain is 52 amino acid residues long and forms helix-turn-helix conformation which interacts with the core DNA sequences [125][126]. Based on the MYB repeats in the N-terminus the MYB transcription factors

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are subdivided into four groups: 1R MYB, 2R MYB (R2R3MYB), 3RMYB (R1R2R3MYB), and 4RMYB. Most of the identified MYB proteins belong to the R2R3MYB group. 4RMYB group is believed to be the smallest MYB group, which contains four R1/R2-like repeats [127]. The MYB transcription factor performs various biological functions like cell cycle, cell wall biosynthesis, growth and development. It also performs a crucial role in biotic as well as abiotic stresses [128].

Molecular mechanisms of MYB transcription factors regulating plant responses against pathogens

MYB protein family protects the plant from biotic as well as abiotic stress by enhancing various related gene expressions [129]. During biotic stress, the MYB proteins protect the plants by hypersensitive responsive cell death of the infected cells or by activating various phytohormone signalling pathways like SA, JA, ET pathways [130, 131]. In sorghum plants, the MYB TFs plays a significant role against *Colletotrichum sublineolum* a fungal pathogen by synthesizing 3-deoxyanthocyanidin phytoalexins an effector molecule. Interestingly, expressing this MYB TF in transgenic Arabidopsis plant induced 3-deoxyanthocyanidin synthesis and provided resistance against leaf blight [19]. In 2016, a group of researchers observed that in wheat plant overexpression of R2R3-MYB Transcription factor gene *TaRIM1* enhanced resistance against *Rhizoctonia cerealis* [126]. Overexpression *CmMYB19* in transgenic plants showed decreased aphid invasion by enhancing lignin accumulation [132]. Some of the examples of MYB transcription factors during biotic stress are mentioned in **Table 4**.

Table 4: MYB transcription factors and their specific role in the plant defence mechanism.

MYB TFs	Organism	Specific Role	Reference
CmMYB19	Chrysanthemum	Overexpression of TF reduced aphid infestation via lignin accumulation into the plants.	[132]
AtMYB30	Arabidopsis thaliana	After the pathogen attack, its function as an activator of hypersensitive cell death.	[133]
OsLTR1	Oryza sativa	JA mediated defence mechanism.	[123]
AtMYB15, AtMYB34, AtMYB51, AtMYB75	Arabidopsis thaliana	Act as a positive regulator in wound healing and resistant to insects.	[134]
AtMYB44	Arabidopsis thaliana	Act as a positive regulator in defence responses against aphids.	[135]
AtMYB96	Arabidopsis thaliana	AtMYB96 mediated ABA signal enhanced pathogen resistance signal by inducing SA signalling	[136]

R2R3 MYB genes are found to involve in various signal transduction pathways like salicylic acid, abscisic acid, gibberellic acid (GA) and JA signalling pathways. These signal transduction pathways are involved in protecting the plants against the pathogen attack and various abiotic stresses [128]. Phenylalanine ammonia-lyase (PAL) is a key enzyme which plays a vital role in the plant defence mechanism against insect diseases [137]. Brown planthopper (BPH) attack in rice plants is one of the destructive insect attack affecting huge rice production. After insect, attack plant perceives signals and transduces the signals in the form of herbivore-associated molecular pattern molecules (HAMPs) or plant-derived damage-associated molecular pattern molecules (DAMPs) as insect releases some elicitors. The transducer molecules phosphorylate and activate different transcription factors which regulate the expression of different defence-related genes as a results plant secretes different types of enzymes which provides resistance against the insect attack [19, 138]. For example, Brown planthopper (BPH) attack on rice plants phosphorylates and activates an R2R3 transcription factor OsMYB30 which enhances the activity of OsPAL6 and OsPAL8 activity and provides resistance against Brown planthopper (BPH) **figure 10** [139]. Abscisic acid is an important phytohormone produced during water deficiency, high salinity and protects the plants[140, 141]. MYB TFs also acts as a negative regulator in plant defence responses. They may act as a transcriptional silencer, silencing of transcription process makes the plants more susceptible towards pathogen attack [136]. Some of the examples of MYB transcription factor as negative regulator are- ZmMYB-31, which is responsible in the reduction of sinapoylmalate and phenylpropanoids in plants which makes the plant more sensitive to UV-irradiation and induce several stress related proteins [142]; ZmMYB-31 also involve in the down regulation of various genes involve in monolignol synthesis, which results in the reduction of lignin content in transgenic plants [143].

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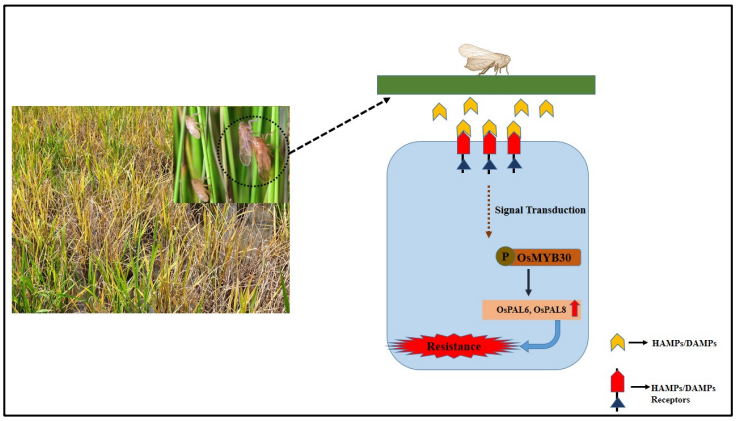

Figure 10: Schematic model for the involvement of MYB transcription factor in the plant defence mechanism after insect attack**.** After insect (brown plant hopper)/ herbivore attack plant perceives the signal through HAMPs/DAMPs and transduces the signal different parts of the cell and activates OsMYB30 TFs in rice plants which further regulates and enhances the activities of OsPAL6 and OsPAL8 and proved resistance against insect-like brown plant hopper attack.

Conclusion:

We report known information (basic structure, and regulatory mechanisms) on four families of transcription factors (WRKY, NAC, bZIP, MYB) involved in biotic stresses. And under field condition, most of the plants experience multi stress conditions as one stress make the plant more susceptible to another form of stress. But, most of the studies were performed for single transcription factors against the single stressed condition. Therefore, in a future study, it is necessary to perform the combinatorial effect of multiple TFs against multi stressed conditions. This will help to understand the crosstalk among the different types of TFs under a stressed condition. In future studies researchers can take the help of powerful gene-editing tool CRISPR/Cas9 system to attain resistance or tolerance against the stressed conditions by assessing the genes and gene functions under stressed conditions.

References:

[1] Tajima Y *et al.* Academic Press. 2020. p. 21–34
[2] Erpen L *et al.* Tissue Organ Cult. 2018;132:1.
[3] Herman RA *et al.* Trends Biotechnol. 2019;37:1272 [PMID: 31307666]
[4] Pandey P *et al.* Front Plant Sci. 2017;8:537 [PMID: 28458674]
[5] Rejeb I *et al.* Plants. 2014;3:458 [PMID: 27135514]
[6] Kaur C *et al.* Singapore: Springer Singapore; 2019. p. 163–86.
[7] Andersen E *et al.* Genes (Basel). 2018;9:339 [PMID: 29973557]
[8] Thakur M *et al.* ISRN Biochem. 2013;2013:1 [PMID: 25969762]
[9] Xie Z *et al.* Front Plant Sci. 2019; 10:228 [PMID: 30873200]
[10] Chattopadhyay A *et al.* Curr Sci. 2019; 117:1598
[11] Sikdar S *et al.* BMC Bioinformatics. 2017;18:79 [PMID: 28148240]
[12] Hong Z *et al.* J. Plant Biol. Crop Res. 2018; 1: 1005
[13] Philips T *et al.* Nature Education [internet]. 2008; 1 (1): 119.
[14] Merkulov VM *et al.* Russian Journal of Genetics: Applied Research. 2014; 4(6):549
[15] Karp G *et al.* John Wiley & Sons; 2020 Feb 19.
[16] Gonzalez DH. Academic Press, 2016; 3
[17] Liu L *et al.* Eur J Biochem. 1999;262:247.
[18] Sharma N *et al.* BMC Genomics. 2013;14:915 [PMID: 24365221]
[19] Baillo EH *et al.* Genes. 2019; 10(10):771 [PMID: 31575043]
[20] Khan S-A *et al.* Int J Mol Sci. 2018;19:1634. [PMID: 29857524]
[21] Hoang XLT *et al.* Curr. Genomics. 2017; 18(6): 483 [PMID: 29204078].
[22] Gao Y *et al.* Physiologia plantarum. 2020; 168(1):98. [PMID: 31017672]
[23] Yuan X *et al.* BMC Plant Biol. 2019; 19(1):278. [PMID: 31238869]
[24] Goel P *et al.* InMolecular Approaches in Plant Biology and Environmental Challenges 2019 (pp. 7-50). Springer, Singapore.
[25] Phukan UJ *et al.* Front Plant Sci. 2016; 7:760. [PMID: 27375634]
[26] Bakshi M *et al.* Plant Signal Behav. 2014; 9: e27700. [PMID: 24492469]
[27] Chen X *et al.* Phytopathology Research. 2019; 1(1):13.
[28] Banerjee A *et al.* ScientificWorldJournal. 2015; 2015: 807560. [PMID: 25879071]
[29] Sun C *et al.* Plant Cell. 2003 Sep; 15(9): 2076. [PMID: 12953112]
[30] Singh A *et al.* Biology (Basel). 2019; 8(4): 83. [PMID: 31690005]
[31] Mohanta TK *et al.* Sci Rep. 2016; 6: 37309. [PMID: 27853303]
[32] Center for Bioinformatics PU. PlantTFDB - Plant Transcription Factor Database @ CBI, PKU. Center for

BIOINFORMATION
Discovery at the interface of physical and biological sciences

Bioinformatics, Peking University. 2019. http://planttfdb.cbi.pku.edu.cn/family.php?fam=WRKY. Accessed 8 Mar 2020.
[33] Nan H *et al.* Front Genet. 2019; 10:363. [PMID: 31191596]
[34] Rushton PJ *et al.* Trends Plant Sci. 2010; 15(5):247. [PMID: 20304701]
[35] Chujo T *et al.* PLoS One. 2014; 9(6):e98737. [PMID: 24892523]
[36] Chujo T *et al.* Plant Mol Biol. 2013; 82(1-2):23. [PMID: 23462973]
[37] Wang P *et al.* Front Plant Sci. 2018; 9:1330. [PMID: 30258455]
[38] Bigeard J *et al.* Front Plant Sci. 2018; 9:469. [PMID: 29696029]
[39] Inoue H *et al.* Rice. 2017; 10(1):36. [PMID: 28766258]
[40] Lan A *et al.* Plant Biol. 2013; 15(3):452. [PMID: 23061987]
[41] Yokotani N *et al.* J Exp Bot. 2013; 64(16):5085. [PMID: 24043853]
[42] Liu J *et al.* Plant Physiol. 2016; 171(2):1427. [PMID: 27208272]
[43] Sun Q *et al.* Plant Gene. 2019; 19:100200.
[44] Gu L *et al.* PLoS One. 2018; 13(1):e0191681. [PMID: 29370286]
[45] Tao Z *et al.* Plant Physiol. 2009; 151(2):936. [PMID: 19700558]
[46] Li B bei *et al.* Journal of Integrative Agriculture. 2019; 18(6):1199.
[47] Birkenbihl RP *et al.* Curr Opin Plant Biol. 2017; 38:1. [PMID: 28458046]
[48] Liu XQ *et al.* Cell Res. 2005; 15(8):593. [PMID: 16117849]
[49] Jiang J *et al.* J Integr Plant Biol. 2017; 59(2):86. [PMID: 27995748]
[50] Yogendra KN *et al.* Plant Sci. 2017; 256:208. [PMID: 28167034]
[51] Jiang Y *et al.* PLoS One. 2016; 11(3):e0149137. [PMID: 27019084]
[52] Jiang Y *et al.* Tree Physiol. 2017; 37(5):665. [PMID: 28338710]
[53] Jalmi SK *et al.* Sci Rep. 2016; 6:37974. [PMID: 27897241]
[54] Wei T *et al.* PLoS One. 2013; 8(3):e59720. [PMID: 23544090]
[55] Liang X *et al.* Sci Rep. 2017; 7: 2474. [PMID: 28559550]
[56] Abbruscato P *et al.* Mol Plant Pathol. 2012; 13(8): 828. [PMID: 22443363]
[57] Yu Y-H *et al.* Scientia Horticulturae. 2019; 256:108640.
[58] Yin J *et al.* Int J Mol Sci. 2019; 20(11): 2807. [PMID: 31181758]
[59] Peng Y *et al.* Rice (N Y). 2010; 3(1): 36. [PMID: 21961049]
[60] Vo KTX *et al.* Front Plant Sci. 2017; 8: 2220. [PMID: 29375598]
[61] Koo SC *et al.* Biochem Biophys Res Commun 2009; 387(2):365. [PMID: 19607808]
[62] Xiao J *et al.* Plant Physiol. 2013 Dec; 163(4):1868. [P 24130197]
[63] Cai M *et al.* Plant Cell Environ. 2008; 31(1):86. [PMI 17986178]
[64] Han Y *et al.* Plant Physiol. 2019; 179(4):1416. [PMII 30696749]
[65] Peng Y *et al.* Mol Plant. 2008; 1(3):446. [PMID: 19825552]
[66] Chen L *et al.* Mol Plant Microbe Interact. 2010; 23(5):558. [PMID: 20367464]
[67] Pandey SP *et al.* Plant Physiol. 2009; 150(4):1648. [PMID: 19420325]
[68] Fukushima S *et al.* Plant Cell Physiol. 2016; 57(12):2541. [PM 27837098]
[69] Huang W *et al.* Mol Plant. 2020; 13(1):31. [PMID: 31863850]
[70] van Verk MC *et al.* BMC Plant Biol. 2011; 11:89. [PMID: 21595875]
[71] Ding Y *et al.* Front Plant Sci. 2015; 5:763. [PMID: 25610446]
[72] Wang F-P *et al.* Hortic Res. 2019; 6:102. [PMID: 31645957]
[73] Ding P *et al.* Trends Plant Sci. 2020; 25(6):549. [PMID: 32407695]
[74] Nobuta K *et al.* Plant Physiol. 2007; 144(2):1144. [PMID: 17468220]
[75] Backer R *et al.* Front Plant Sci. 2019; 10: 102. [PMID: 30815005]
[76] Caarls L *et al.* Front Plant Sci. 2015; 6: 170. [PMID: 25859250]
[77] Mathew IE *et al.* Front Plant Sci. 2016; 7: 1638. [PMID: 27872632]
[78] Nuruzzaman M *et al.* Front Microbiol. 2013; 4: 248. [PMID: 24058359]
[79] Diao W *et al.* Int J Mol Sci. 2018 Apr; 19(4): 1028. [PMID: 29596349]
[80] Khedia J *et al.* Front Plant Sci. 2018; 9: 1522. [PMID: 30420862]
[81] Puranik S *et al.* Trends Plant Sci. 2012; 17(6):369. [PMID: 22445067]

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Discovery at the interface of physical and biological sciences

[82] Shao H *et al.* Front Plant Sci. 2015; 6:902. [PMID: 26579152]
[83] Yuan X *et al.* Phytopathology Research. 2019; 1(1):3.
[84] Wang G *et al.* Physiol Plant. 2016; 158(1):45. [PMID: 26991441]
[85] He X *et al.* Int J Mol Sci. 2018; 19(12):3997. [PMID: 30545043]
[86] Wang B *et al.* J Integr Plant Biol. 2018; 60(5):432. [PMID: 29251427]
[87] Alves M *et al.* Proteomes. 2014; 2(1):85. [PMID: 28250372]
[88] Sun L *et al.* Plant Mol Biol. 2013; 81(1-2):41. [PMID: 23103994]
[89] Lee M-H *et al.* New Phytol. 2017; 214(1):343. [PMID: 28032643]
[90] He X *et al.* Plant Cell Rep. 2016; 35(10):2167. [PMID: 27432176]
[91] Kleinow T *et al.* Plant Science. 2009; 177(4):360.
[92] Delessert C *et al.* Plant J. 2005; 43(5):745. [PMID: 16115070]
[93] Castillo N *et al.* Front Plant Sci. 2019; 10:1162. [PMID: 31611892]
[94] Kim HS *et al.* Nucleic Acids Res. 2012; 40(18):9182. [PMID: 22826500]
[95] Mine A *et al.* Proc Natl Acad Sci U S A. 2017; 114(28):7456. [PMID: 28652328]
[96] Sun L *et al.* Int J Mol Sci. 2015; 16(2):4306. [PMID: 25690040]
[97] Chen Y-J *et al.* Plant Mol Biol. 2013; 83(6):577. [PMID: 23896755]
[98] Le Hénanff G *et al.* J Exp Bot. 2013; 64(16):4877. [PMID: 24043850]
[99] Kaneda T *et al.* EMBO J. 2009; 28(7):926. [PMID: 19229294]
[100] Agarwal P *et al.* Sci Rep. 2019; 9(1):4608. [PMID: 30872683]
[101] Li D *et al.* BMC Genomics. 2015; 16: 771. [PMID: 26459863]
[102] [102] Jakoby M *et al.* Trends Plant Sci. 2002; 7(3):106. [PMID: 11906833]
[103] Yang Z *et al.* BMC Genet. 2019; 20(1):41. [PMID: 31023242]
[104] Wang Y *et al.* PLoS One. 2018; 13(7):e0200850. [PMID: 30011333]
[105] Ullah I *et al.* Sci Rep. 2019; 9(1):11186. [PMID: 31371739]
[106] Gatz C. Mol Plant Microbe Interact. 2013; 26(2):151. [PMID: 23013435]
[107] Kesarwani M *et al.* Plant Physiol. 2007; 144(1):336. [PMID: 17369431]
[108] Alves M *et al.* Int J Mol Sci. 2013; 14(4):7815. [PMID: 23574941]
[109] Tolosa *et al.* Plants (Basel). 2020; 9(1):56. [PMID: 31906447]
[110] Li X *et al.* Front Plant Sci. 2017; 8:2110. [PMID: 29276527]
[111] Lim CW *et al.* Mol Plant Microbe Interact. 2015; 28(7):825. [PMID: 25738319]
[112] Noman A *et al.* Biotechnol Lett. 2017; 39(12):1779. [PMID: 28879532]
[113] Kuhlmann M *et al.* J Biol Chem. 2003; 278(10):8786. [PMID: 12499372]
[114] Zhou J-M *et al.* Mol Plant Microbe Interact. 2000; 13(2):191. [PMID: 10659709]
[115] Kaminaka H *et al.* EMBO J. 2006; 25(18):4400. [PMID: 16957775]
[116] Moon S-J *et al.* PLoS One. 2018; 13(11):e0206910. [PMID: 30444888]
[117] Tak H *et al.* Protoplasma. 2013; 250(1):333. [PMID: 22610648]
[118] Lee SJ *et al.* Mol Plant Microbe Interact. 2002; 15(6):540. [PMID: 12059102]
[119] Okada A et al J Biol Chem. 2009; 284(39):26510. [PMID: 19635799]
[120] ORELLANA S *et al.* Plant Cell Environ. 2010; 33(12):2191. [PMID: 20807374]
[121] Miller RNG *et al.* Ann Bot. 2017; 119(5):681. [PMID: 28375427]
[122] Bigeard J *et al.* Mol Plant. 2015; 8(4):521. [PMID: 25744358]
[123] Ambawat S *et al.* Physiol Mol Biol Plants. 2013; 19(3):307. [PMID: 24431500]
[124] Lloyd A *et al.* Plant Cell Physiol. 2017; 58(9):1431. [PMID: 28575507]
[125] Du H *et al.* Biochemistry (Mosc). 2009; 74(1):1.[PMID: 19232042]
[126] Shan T *et al.* Sci Rep. 2016; 6:28777. [PMID: 27364458]
[127] Chen C *et al.* Critical Reviews in Plant Sciences. 2019; 38(3):159.

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[128] Shen X-J *et al.* Int J Mol Sci. 2018; 19(12):3958. [PMID: 30544851]
[129] Das A *et al.* PLoS One. 2019; 14(2):e0211887. [PMID: 30763346]
[130] Ruan J *et al.* Int J Mol Sci. 2019; 20(10):2479. [PMID: 31137463]
[131] Zhang Y *et al.* Plant Physiol. 2018; 178(1):189. [PMID: 29987002]
[132] Wang Y *et al.* Int J Mol Sci. 2017; 18(3):619. [PMID: 28287502]
[133] Raffaele S *et al.* Front Plant Sci. 2013; 4:98. [PMID: 23596456]
[134] Johnson ET *et al.* J Agric Food Chem. 2004; 52(16):5135. [PMID: 15291486]
[135] Jaradat MR *et al.* BMC Plant Biol. 2013; 13:192. [PMID: 24286353]
[136] Baldoni E *et al.* Int J Mol Sci. 2015; 16(7):15811. [PMID: 26184177]
[137] Kaur H *et al.* Journal of Plant Interactions. 2017; 12(1):304.
[138] Hogenhout SA *et al.* Nat Biotechnol. 2015; 33(3):262-3. [PMID: 25748917]
[139] He J *et al.* Proc Natl Acad Sci U S A. 2020; 117(1):271. [PMID: 31848246]
[140] Vishwakarma K *et al.* Front Plant Sci. 2017; 8:161. [PMID: 28265276]
[141] Yu Y-T *et al.* Journal of Plant Biology. 2016; 59(2):152.
[142] Fornalé S *et al.* Plant J. 2010; 64(4):633. [PMID: 21070416]
[143] Tak H *et al.* PLoS One. 2017; 12(2):e0172695. [PMID: 28234982]

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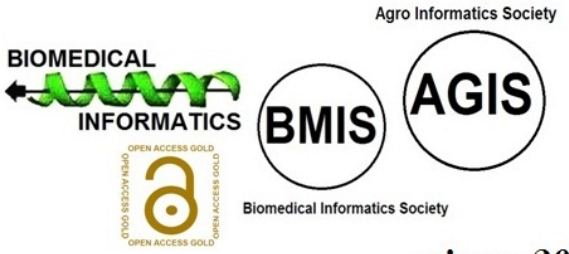

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