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ARABIDOPSIS PATHOSYSTEMS : A MODEL SYSTEM FOR THE STUDY OF HOST-PATHOGEN INTERACTION

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ABSTRACT

The small cruciferous weed *Arabidopsis thaliana* has become an interesting model host for the investigation of outstanding problems in plant pathology. *Arabidopsis thaliana* has been studied intensively as a model host plant for diseases caused by different plant pathogens. *A. thaliana* provides a genetically amenable system which can be used to examine the various component of disease resistance. Many ecotypes of *A. thaliana* differ genetically as a result of selection pressures imposed by their different environment of origins and these differences include resistance to pathogens. The development of Arabidopsis as a model system for plant molecular biologists sparked a search for pathogens for Arabidopsis.

Key words : *Arabidopsis, pathosystems, host, pathogen.*

Arabidopsis thaliana is a small weed belonging to the cruciferous family and it is estimated that Brassica and Arabidopsis lineage diverged 12.2-19.2 million year ago. Brassica and Arabidopsis lineage diverged by triplication followed by extensive deletions and significant change of genome microstructure. Arabidopsis has been embraced by plant researchers as a plant of choice for studying the most aspects of plant biology. It is true diploid and can be readily out crossed or selfed. Its generation time is less than six weeks and individual plant can produce thousands of seeds. The seed can be easily mutagenized by using chemical mutagens or ionizing radiation. The size of the genome is smaller than any of the known flowering plants. The genome of Arabidopsis contains 125 mega bases of sequence, encodes approximately 25,500 genes and has very less repetitive DNA. Arabidopsis is particularly suited for positional cloning approach and can be readily transformed by *Agrobacterium tumefaciens*. The development of Arabidopsis as a model system for plant molecular biologists sparked a search for pathogens for Arabidopsis. Such pathogens were identified either by identifying naturally occurring infections on Arabidopsis or by screening pathogens collected from other plants, especially pathogens of other crucifers. Out of 25,500 genes, 2055 are denoted as involved in plant defense.

Arabidopsis Pathosystems : *A. thaliana* has been studied intensively as a model host plant for diseases caused by viral, bacterial and fungal pathogens (Table-1). The small cruciferous weed *Arabidopsis thaliana* has become an interesting model host for the investigation of outstanding problems in plant pathology (Mauch-mani and Slusarenko, 1994). *A. thaliana* provides a genetically amenable system which can be used to examine the various component of disease resistance (Koch and Slusarenko, 1990b). Many ecotypes of *A. thaliana* differ genetically as a result of selection pressures imposed by their different environment of origins (Kagan and Hammerschmidt, 2002). These differences include resistance to pathogens. Different ecotypes of *A. thaliana* differ in their ability to resist infection by club root pathogen *Plasmodiophora brassicae* (Fuchs and Sacristan, 1996), Cauliflower mosaic virus (Leisener and Howell, 1992; Callaway *et al*., 1996), Turnip crinkle virus (Dempsey et al., 1997), *Xanthomonas campestris pv. campestris* (Tsuji *et al*., 1991), *Albugo candida* (Holub *et al*., 1995), *Hyaloperonospora parasitica* (Mauch-mani *et al*., 1993a). Resistance to these pathogens is due to presence of R gene(s). The types of R genes vary among ecotypes, which may be a reflection of variations in disease pressure in the locations from where ecotypes were collected (Kagan and Hammerschmidt, 2002).

The first pioneering work to establish the Arabidopsis as a useful model in plant pathology was the activation of classical defense responses, such as the accumulation of transcripts for phenylalanine ammonia-lyase (PAL) and chalcone synthase (CHS) and which also demonstrated the induction of peroxidase activity in elicitor treated cell suspension

culture (Davis and Ausubel, 1989). Two approaches were used to develop pathosystems with Arabidopsis. The first approach was to take pathogens of related hosts, inoculate them into Arabidopsis and observe multiplication of the pathogens and disease symptoms and the other approach was to search for naturally infected plants in the field (Debner *et al*., 1991; Whalen *et al*., 1991). Several pathosystems involving viral (Li and Simon, 1990; Sosnova and Polak, 1975), bacterial (Davis *et al*., 1991; Debner *et al*., 1991; Whalen *et al*., 1991; Tsuji *et al*., 1991; Parker *et al*., 1993a), fungal (Koch and Slusarenko, 1990a; Crute *et al*., 1993; O'Connell *et al*., 2004) and nematode (Sijmons *et al*., 1991) plant pathogens have been developed for study.

Arabidopsis-Turnip crinkle virus pathosystem : Simons *et al*., (1992) first reported the phenotypic variation among Arabidopsis ecotypes to virus challenge and showed Di-0 (Dijon) was resistant to Turnip crinkle virus and Col-0 and other ecotypes were susceptible.

Arabidopsis *Pseudomonas syringae pv. Tomato* Pathosystem : In order to investigate natural variation of partial resistance to *Pseudomonas syringae pv. tomato* DC3000 (*Pst*) in *Arabidopsis thaliana*, 27 Arabidopsis accessions were tested for their response to the virulent *Pst*. Phenotypic analysis of a set of Arabidopsis accessions, based on evaluation of *plant* pathogen growth revealed extensive quantitative variation for partial resistance to *Pst (*Perchepied *et al*., 2006).

Arabidopsis-Albugo Pathosystam : Infection of the foliage of Arabidopsis with *Albugo candida* showed the occurrence of pale-green patches on the upper surface which eventually turn chlorotic and develop red borders. On the under surface, fungus produces white pustules. Isolate-specific interaction phenotypes have been examined for more than 30 ecotypes of Arabidopsis following inoculation of seedlings at the cotyledon stage with two isolates (Acem1 and Acks 1) of *A. candida* (Crute *et al*., 1993).

Arabidopsis Hyaloperonospora pathosystem : *Hyaloperonospora parasitica* isolate, Noco2 showed the resistant reaction to Ler and susceptible reaction to Col-0. Macroscopic and microscopic examinations of inoculated Ler and Col-0 cotyledons showed that restriction of fungal growth in Ler was accompanied by massive callose accumulation and death of plant cells in direct contact with points of attempted fungal penetration (Parker *et al*., 1993b). The ecotypes Weiningen (Wei-0) and Ler were susceptible against the WELA isolate of *Hyaloperonospora parasitica*, whereas RLD and Col-0 were resistant (Joos *et al*., 1996).

Arabidopsis-Erysiphe pathosystem : Six Arabidopsis ecotypes showed resistance to wild isolate of the powdery pathogens. Three ecotypes Wa-1, Kas-1 and Sl-0 were highly resistant, while other ecotypes permitted some fungal growth. The resistance phenotypes in Wa-1 and Kas-1 were characterized by hypersensitive necrosis as evidenced by rapid (24-48 h) appearance of small necrotic flecks at the inoculation sites (Adam and Somerville, 1996). *A. thaliana* ecotypes Ler was susceptible and Ms-0 was resistant to powdery mildew disease caused by UEA1 (*Erysiphe cruciferarum*) and UCSC1 (*Erysiphe cichoracearum*). Growth of UEA1 on Ms-0 leaves was arrested after formation of first appressorium resulting the underlying host epidermal cells collapsed and occasionally there was necrosis of one or two host mesophyll cells, while the growth UCSC1 on Ms-0 leaves was arrested after emergence of several germ tubes from the conidium and there was necrosis of host mesophyll cells at the site of infection (Xiao *et al*., 1997). Interactions of the two Erysiphe isolates UEA1 (*E. cruciferarum*) and UCSC1 (*E. cichoracearum*) with 360 *A. thaliana* ecotypes were examined to provide a comprehensive profile of naturally occurring powdery mildew resistance. The majority of *A. thaliana* ecotypes (213) were susceptible to both isolates. Among the ecotypes exhibiting some degree of resistance, 84 ecotypes responded differentially to UEA1 and UCSC1 and the rest were resistant to both isolates (Adam *et al*., 1999).

Arabidopsis-Phytophthora pathosystem : Roetschi *et al*., (2001) established the Arabidopsis-Phytophthora pathosystem and showed facultative biotrophic interaction. In susceptible ecotypes, extensive colonization of host the host tissue occurred and sexual and asexual spores are formed. Hypersensitive response and papilla formation found at the attemped penetration sites of plant in incompatible interaction between Arabidopsis and *Phytophthora porri*.

Arabidopsis-Plasmodiophora pathosystem : The observation of ecotypes of worldwide origin has revealed that there is natural variation in the responses of *A. thaliana* to club root disease (*Plasmodiophora brassicae*) (Alix *et al*., 2007; Fuchs and Sacristan, 1996; Siemens *et al*., 2002). Ten to twenty days after

Table-1 : Pathogens reported in *Arabidopsis thaliana.*

Pathogen	References
Cauliflower Mosaic Virus	Leisener and Howell, (1992); Callaway *et al*., (1996)
Turnip Crinkle Virus	Simon *et al*., (1992); Dempsey *et al*., (1993)
Alfalfa Mosaic Virus	Balasubramanium *et al*., (2006)
Cherry leaf roll virus	Rumbou *et al*., (2009)
Xanthomonas campestris pv. campestris	Tsuji and Somerville, (1988, 1992); Simpson and Johnson, (1990); Davis *et al*., (1991); Tsuji *et al*., (1991); Parker *et al*., (1993a)
Xanthomonas campestris pv. amoraciae	Davis *et al*., (1991)
Pseudomonas syringae pv. tomato	Davis *et al*., (1991); Whalen *et al*., (1991)
Pseudomonas syringae pv. maculicola	Davis *et al*., (1991); Debner *et al*., (1991)
Pseudomonas syringae pv. pisi	Davis *et al*., (1991)
Pseudomonas cichorii	Davis *et al*., (1991)
Spiroplasma citri	Fletcher and Eastman, (1992)
Hyaloperonospora parasitica (*Peronospora parasitica)*	Koch and Slusarenko, (1990a); Holub *et al*., (1991, 1994) Mauch-mani and Slusarenko, (1993a)
Albugo candida	Holub *et al*., (1991)
Pythium spp.	Mauch-mani and Slusarenko (1993b)
Phytophthora porri	Roetschi *et al*., (2001)
Alternaria brassicae	Berger, (1965)
Alternaria brassicicola	Ellis, (1968); Kagan and Hammerschmidt, (2002); Penninckx *et al*., (1996)
Sclerotinia sclerotiorum	Morgan, (1971); Dickman and Mitra, (1992)
Leptosphaeria maculans (Phoma lingam)	Sjodin and Glimelius, (1988)
Erysiphe cruciferarum	Koch and Slusarenko, (1990a); Xiao *et al*., (1997)
Erysiphe cichoracearum	Adam and Somerville, (1996); Adam *et al*., (1999)
Botrytis cinerea	Koch and Slusarenko, (1990a); Rowe and Kliebenstein, (2008)
Fusarium oxysporum	Mauch-mani and Slusarenko, (1993b, 1994)
Cladosporium spp.	Mauch-mani and Slusarenko, (1993b)
Colletotrichum destructivum	O'Connell *et al*., (2004)
Colletotrichum higginsianum	Narusaka *et al*., (2004)
Rhizoctonia solani	Koch and Slusarenko, (1990b)

inoculation, susceptible ecotypes showed severe swelling of hypocotyls, main root and lateral roots. Infected plants often were stunted and delayed in flowering and some of them died before flowering (Koch and Slusarenko, 1990a; Fuchs and Sacristan, 1996). Ze-0 and tsu-0 ecotypes were identified resistant to *P. brassicae* isolate 'e' (Fuchs and Sacristan, 1996). Alix *et al*., (2007) identified the ecotype Burren (Bur-0) as partially resistant to the *P. brassicae* isolate 'eH'.

Arabidopsis-Leptosphaeria pathosystem : To investigate the variability in resistance against *Leptosphaeria maculans,* 168 Arabidopsis ecotypes were screened with the three different fungal isolates. Non spreading lesions were formed, and no fungal growth could be observed at the infection sites of 163 ecotypes including Columbia (Col-0) and Landsberg erecta (Ler-0). Scanning electron microscopy (SEM) revealed that inoculation sites were surrounded by a few layers of dead cells. Blackmount (Ba-1), Leiden (Le-0), Niederlauken (Nie-0) and Ovelgoenne (Ove-0) showed moderate symptoms, whereas An-1 developed clear disease symptoms (Bohman *et al*., 2004).

Arabidopsis-Verticillium pathosystem: A total of 169 ecotypes of *A. thaliana* were screened against *Verticillium longisporum* and score on the basis of number of days until the opening of the first flower, plant height and typical disease symptoms such as discolouration, chlorosis and microsclerotia formation. A total of 54 ecotypes were scored as resistant, whereas 115 ecotypes exhibited various degree of susceptibility. The ecotype Shahdara was most resistant, whereas Che-0 and Je-0 displayed high degree of susceptibility (Johansson *et al*., 2006).

Arabidopsis-Colletotrichum pathosystem : In compatible interactions, *Colletotrichum destructivum* showed two stage hemibiotrophic infections. The initial biotrophic phase was associated with large,

intracellular primary hyphae and was confined to the one epidermal cell, whereas in the subsequent necrotic phase, narrow secondary hyphae extensively colonized the tissue and conidia were produced in acervulii (O'Connell *et al*., 2004).

Arabidopsis-Alternaria pathosystem : All tested accessions of Arabidopsis are resistant to the necrotrophic fungal pathogen *Alternaria brassicicola*. Resistance is compromised by pad3 or coi1 mutations, which requires the Arabidopsis phytoalexin camelexin and jasmonic acid dependent signaling respectively. This contrasts with most well suited Arabidopsis pathogens, which are controlled by salicylic acid-dependent responses and do not benefit from absence of camelexin or jasmonic acid. Mutants with defects in camelexin synthesis (pad1, pad2, pad3 and pad5) or in jasmonic acid signaling (pad1, coi1) were found to be more susceptible than wild types. Mutants with defects in salicylic acid (pad4 and sid2) or ethylene (ein2) signaling remain constant (Van Wees *et al*., 2003). A total of five wild type ecotypes, Col-0, Col-6, DiG (Dijon G), Ler (Landsberg erecta), Ws (Wassilewskija) and three mutants (glip1-1, glip1-2 and acd1) were screened against *Alternaria brassicicola*. Lesion diameter and spore production were measured to assess the disease progression. The col-0 ecotype showed an incompatible response and lesions did not progress beyond the boundaries of the inoculation site. DiG was most susceptible, showing larger lesions than either of the glip1 mutants or Col-6, which are relatively susceptible as compared to Col-0. The lesion-mimic mutant acd1 was no more susceptible to the pathogen than was Col-0. The lesions on DiG leaves continued to spread and often showed concentric rings as seen in the interaction with the compatible host *Brassica oleracea* (Oh *et al*., 2005; Mukherjee *et al*., 2009).

Arabidopsis-Sclerotinia pathosystem : The necrotrophic pathogen *S. sclerotiorum* rapidly infected the Arabidopsis plants, probably through ball- or cushion-like infection structures. Visible symptoms developed within 24 h and plants were killed 72 h after inoculation. Cellulase, the main enzyme that caused host tissues to rot, was secreted by *S. sclerotiorum* in a pH-dependent manner. Oxalic acid, another pathogenic factor secreted by the fungus, induced necrotic lesions on the leaves (Dai et al., 2006). The defense against *S. sclerotiorum* in Arabidopsis is dependent on jasmonic acid, salicylic acid and ethylene signaling (Guo and Stotz, 2007).

CONCLUSIONS

Arabidopsis has been embraced by plant researchers as a plant of choice for studying the most aspects of plant biology. *A. thaliana* has become an important model host for studying plant-pathogen interactions due to the availability of a complete genome sequence, molecular markers and extensive collection of mutational analysis, and the possibility of using microarrays for gene expression analysis.

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