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Genetic Basis of Host Susceptibility to Plant Pathogens

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Abstract
It is assumed that the bulk of the genes that confer resistance to plant diseases at the R level have a molecular foundation. The biggest collection of these genes is known as nucleotide-binding site-leucine-rich repeat (NBS-LRR), and it is responsible for encoding proteins that are structurally similar to innate immunity proteins that are present in mammals. In contrast, there are not a great number of genes that are responsible for making individuals more prone to certain illnesses. It was recently discovered that Arabidopsis thaliana is susceptible to the fungus Cochliobolus victoriae, which made it possible to clone LOV1, a disease susceptibility gene that, strangely, is a member of the NBS-LRR resistance gene family. Our research revealed that LOV1 is responsible for modulating defense-related responses, despite the fact that mutations in well-known defensive response pathways do not provide protection from C. victoriae susceptibility. The results of this study reveal that NBS-LRR genes may have an effect on disease resistance and susceptibility, and they may also have ramifications for the process of using R genes.

Keywords-: *Genetic, Pathogens, Plant, Susceptibility.*

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I.INTRODUCTION

Up until very recently, the majority of the research that has been conducted on plant infection resistance has been from studies that have focused on crop species selection. There has been a significant amount of research conducted on resistance at the molecular, cellular, and population levels via the use of genetics, histology, and related biochemistry (for reviews, see Alfano and Colher, 1996; Bent, 1996; Dangl et al., 1996; Hammond-Kosack and Jones, 1996 in this issue). However, it wasn't until relatively recently that the mechanisms of plant-mediated pathogen resistance could be completely understood. This was made possible by the isolation and sequencing of a large number of genes that are suspected to interact with pathogens. Whatever the case may be, plant resistance genes have been put to good use in agriculture for a considerable amount of time, even if the benefits they provide are not always lasting. This article provides a cursory overview of the genetics of pathotype-specific resistance in plants, as well as discussion of its historical use in crop improvement and some recommendations for potential implications for the future. On the basis of the findings of research conducted with a limited number of host-pathogen combinations that have been thoroughly investigated, some overarching features have been found.

It has been shown beyond a reasonable doubt that a significant number of genes that are present in plant genomes play a part in the categorization and identification of diseases. Additionally, these genes have a tendency to congregate in complex loci, which may comprise genes that are involved in disease resistance but are not necessarily linked to each other taxonomically (examples of this will be supplied in the near future). It is necessary to take into consideration the intricate genetics in order to diagnose some illnesses. As an instance, wheat has approximately 90 genes that determine isolate-specific resistance to many different species of rust, including Puccinia *striiformis,* P recondita, and P graminis, as well as powdery mildew, which is caused by Frysiphe graminis. It is believed that Lr20/Sr75 is the only one of these genes that is capable of assisting in the identification of a broad range of infections (Crute, 1985...). In addition to the occurrence of genes that express the same specificity at various loci in distinct species, it has also been shown that these genes may be found within the same plant species. A number of genes have alleles that are specific to certain pathotypes. These alleles are shared by a number of genes. In light of this, the hypothesis that genes responsible for resistance are members of enormous families that may be highly conserved across a variety of animals is not completely out of the question anymore. According to the growing body of data, the capacity of Nove1 to differentiate between different types of cells may be the consequence of gene conversion or recombination at these complicated locations.

Shortly after the uncovering of Mendel's key work on heredity, it was shown that genes in plants had the potential to provide disease resistance to the plant. According to Biffen (1905), there was only one locus that was responsible for the resistance of certain wheat cultivars to yellow rust caused by I. striiformis. They were resistant

to the disease. The discovery that a great number of plant species possess hundreds of genes that are connected with disease resistance has recently been made. Early research on the interaction between flax and rust fungus (McRostie, 1919) suggests that resistance to diverse pathogenic variants (also known as pathotypes) may be produced by genes located at separate loci. The significance of this discovery, on the other hand, was not completely understood until Flor's forty years of research (1956, 1971) clarified the gene-for-gene relationship within the population. Following Flor's revolutionary research, it became clear that the result of interactions between various combinations of host and parasite genotypes in a variety of host-parasite relationships was regulated by matching gene pairs, namely avirulence (Avr) and resistance (RI) genes, respectively (Crute, 1985).

II. LITERATURE REVIEW

Zhang, Y., Lubberstedt, T., & Xu, M. (2013). Plants have evolved several complex defenses against harmful diseases. Two-tiered immune systems, chemical and physical barriers, and more are examples. Several ways have been developed to harvest nutrients from plant disease hosts. From a genetic standpoint, a plant's ability to survive infection might be quantitative or qualitative. This categorization is based on gene significance. Quantitative disease resistance protects plants against hemibiotrophic, necrotrophic, and biotrophic infections. Only plant defense against biotrophic diseases has been shown to be qualitative disease resistance. Plants may acquire resistance by preventing pathogen effectors and activating a defense response. Over the last several years, our understanding of host-pathogen interactions has grown. Our study aims to modify the molecular and genetic components that contribute to plant disease resistance.

Lapin, D., & Van den Ackerveken, G. (2013). Most plants are susceptible to infectious diseases that are caused by pathogens that are present in their natural environment. These diseases have a negative impact on agricultural productivity. Due to the powerful immune systems that plants possess, they are able to successfully repel the majority of the invaders. Plants are vulnerable to these invaders because adaptable diseases have the ability to escape or circumvent the protection provided by the host. Failure of the immune system is only one of numerous host mechanisms that might alter the susceptibility of a plant to the presence of disease. Not only does this study concentrate mostly on the biotrophic stages of infection, but it also briefly discusses current research that indicates the host may be actively contributing to the development of sickness. Due to the fact that plants are attractive to infections, it is easier for pathogens to find their way into plants, stay there, and provide nutrients.

Urban, M., Pant, R., Raghunath, A., Irvine, A. G., Pedro, H., & Hammond-Kosack, K. E. (2015). Food security, agricultural production, and the health of humans, animals, and ecosystems are threatened by fast growing pathogen-caused diseases and outbreaks. To fight diseases, one must understand the pathogenic process in diverse species. Pathogen-Host Interactions database (PHI-base) covers bacteria, fungi, and protists with effector genes, virulence, and pathogenicity. All mutant oddities are genetic. Infectious illnesses may spread between humans, animals, plants, insects, fish, and fungi. The latest PHI-base, 3.6, is available at http://www.phi-base.org. It contains 28,75 genes, 4,102 interactions, 110 host species, 160 pathogenic species (103 plant, 3 fungus, and 54 animal species that may infect), and 181 illnesses from 1243 references. We collected phenotypic and gene function data by manually cultivating expert-reviewed literature. A controlled vocabulary's nine high-level phenotypic concepts simplify data interpretation and taxonomy categorization comparisons. PHI-base phenotypes were mapped to Ensembl Genomes reference genomes. Gene information associated with phenotypes was used for this mapping. Genome browsers provide real-time viewing of virulence genes and hotspots. The PHI-upcoming base will build tools for community-led curation and add relevant host targets.

Urban, M., Cuzick, A., Seager, J., Wood, V., Rutherford, K., Venkatesh, S. Y., ... & Hammond-Kosack, K. E. (2020). PHI-base, the Pathogen-Host Interactions Database, may be accessed at www.phi-base.org. PHI-base contains biological and molecular data on genes that affect pathogen-host interactions in scientific studies. PHI-base also collects information on gene mutations that did not affect disease interaction. It does this to complete its comparative research databases with more data. Since viruses are widely documented elsewhere, this database does not contain them. This website also discusses PHI-base's enlarged data content, new features, and better integration with other databases. As of September 2019, PHI-base version 4.8 contains 13,801 interactions and 6780 genes from 268 pathogens tested on 210 hosts. It also contains 3454 hand-picked references. Prokaryotic pathogens are almost as abundant as eukaryotic ones. Sixty percent of the host species are plants, with 50% cereal and 50% non-cereal. The remaining 40% of host species are ecological or medically important species. More entries for diseases that infect agricultural species of global relevance have been added, and pathogen effector data is now about a third more extensive. We also briefly explore the project's future and PHI-based curation difficulties.

III.RESEARCH METHODOLOGY

Plant Material, Growth Conditions, and Victorin Sensitivity Assays.

The Arabidopsis Biological Resource Center at Ohio State University in Columbus, Ohio, provided us with the seeds of the A. thaliana plant. Additionally, the mutant lines jar1-1 (39), npr1-1 (40), ein2-1 (41), pad3 (42), and NahG (23), all of which were developed in Col-0, were included to the collection. The LOV1 (16) and Col-LOV (36) were both produced by us. An additional collection of seeds was made available by the following individuals: A. Bent from the University of Wisconsin in Madison, Wisconsin; B. Staskawicz from the University of California in Berkeley, California; B. Kunkel from Washington University in St. Louis, Missouri; Sainsbury Laboratory; and J. Chang. Following a period of five days at a temperature of four degrees Celsius in 0.1% agar, the seeds were planted in soil and then treated to a protracted photoperiod consisting of sixteen hours of light and eight hours of darkness. Over the course of their development, the temperature was kept at 22 degrees Celsius. The victorin sensitivity experiments used Victorin C, which was extracted in accordance with reference 46. With doses ranging from 1 to 20 µg/ml, the effectiveness of the victorin was evaluated.

Fine-Mapping and Cloning of LOV1.

Reference number 16 provides a description of the process by which LOV1 was first mapped to the Nga63 and NCCI interval on Chromosome 1. For the purpose of fine-mapping, polymerase chain reaction (PCR) was used to analyze 200 more F2 offspring of a hybrid of LOV1 and Col-4 that exhibited insensitivity to victorin. According to the information that was provided by Edwards et al. (47), the DNA that was used for the polymerase chain reaction was isolated from the leaves.

For the purpose of generating a genomic library, the binary vector pCLD04541 (18) was used. For the purpose of purifying the DNA that was isolated from LOV1 by the CTAB technique (48), ultracentrifugation in cesium chloride was used. Sau3A was used to partially digest the DNA in order to facilitate the process of ligating it into the pCLD04541 BamHI site. Gigapack III XL extracts were used to pack the ligations after they had been turned into XL1-Blue MR cells which were manufactured by Stratagene in La Jolla, California. The 32P-labeled DNA of BAC clone T19D16, as well as specific portions of BAC clones T16B5 and T28P6, both of which were acquired from ABRC, were used in the screening process of the genomic library that was created. Following the completion of end-sequencing, contig construction, and triparental mating, the positive clones that were obtained from this screen were later converted into the strain GV3101 of Agrobacterium tumefaciens (49). There is no evidence that electroporation is an effective technique for pCLD04541.

Col-4 plants were transformed by the use of the floral dip technique (50). Following the completion of the soaking process, the plant seed was placed onto nutritional agar (16) that had been augmented with 100 µg/ml of cefatoxamine and 100 µg/ml of kanamycin. After a week had passed, transgenic seedlings were planted in the ground. The procedure that was provided was used in order to examine the victorin sensitivity of transgenic plants. A 6.5-kilobase XbaI fragment that included LOV1 was electroporated into A. tumefaciens strain GV3101 (51) after it had been subcloned from pCL26A into pCB302 after the preceding step. The processes that were mentioned before were used in order to create transgenic plants; however, the seeds that were meant to be transgenic were planted immediately in soil that had been soaked with 0.02% glufosinate-ammonium.

Isolation of LOV1 Signal Transduction Mutants.

In order to determine victorin sensitivity and PCR markers that are connected to the various loci of interest, the progeny of LOV1 crosses with signal transduction mutant lines (NahG, npr1-1, ndr1-1, ein2, coi1-35, jar1, pad3, and dnd1, respectively) were examined. PCR markers to follow the segregation of the aforementioned loci, or gene-specific primers for NDR1-1 (forward, AATCTACTACGACGATGTCCAC; reverse, GTAACCGATGGCAACTTTCAC) and NahG (forward, CAGAAGGTATCGCCCAATTC; reverse, ACCTTCCAGCACATGGCTAC), according to sequence data available at The Arabidopsis Information. Some of the obvious symptoms that may indicate the existence of multiple mutations are dwarfism in dnd1, leaky male-sterility in coi1-35, and a triple-mutant response in ein2-1 cultured on MS agar with 0.5 mM ACC. In addition, there are a number of other evident symptoms that may disclose the presence of multiple mutations. Toxin-sensitive and capable of being directly tested for victorin sensitivity, ecotype Ws is comprised of eds1-1 and rar1, and it is also resistant to toxins.

Approximately one hundred F2 plants were utilized for PCR and victorin sensitivity tests. These plants were derived from each of the aforementioned crossings. In the event that plants were homozygous for all mutant alleles, either heterozygous or homozygous for LOV1, they were permitted to self-fertilize. Additionally, an attempt was made to identify victorin sensitivity as well as relevant PCR markers in the F3 generation.

C. victoriae Susceptibility Assays.

In reference 16, the procedure for the production of C. victoriae spores is described in full. There were 32 plants that were between three and four weeks old when they were sprayed with the following strains of C.

victoriae: Col-0, LOV1, ein2-1, ein2LOV1, NahG, NahGLOV1, npr1-1, npr1LOV1, and LOV1 are the names of the individuals. There were 106 spores per milliliter; this was the concentration. Spores stayed on the plants until runoff was able to remove them. The plants were then kept in a growing environment at a temperature of 22 degrees Celsius, with a humidity level of one hundred percent, and a protracted photoperiod consisting of sixteen hours of light and eight hours of darkness until the onset of symptoms. We utilized a more straightforward detached leaf experiment when we discovered that no LOV1-deficient plant had any disease lesions and that all LOV1-containing plants (LOV1, ein2LOV1, NahGLOV1, and npr1LOV1) seemed to be equally sensitive to the illness.

It was decided to insert the third through sixth true leaves, or six detached leaves for each genotype, in a Petri dish that had been sealed and lined with moist filter paper. A time-course experiment was conducted in which plants were positioned with their leaves on top of a covered surface in order to preserve humidity. The findings of this experiment are shown in Figure 4. After washing the leaves in accordance with the instructions and resuspending them to a concentration of 105 spores per milliliter, 10 microliters of C. victoriae spores were pipetted into the center of each leaf. We examined the leaves on a daily basis for a period of no more than ten days, trying to identify any evidence of fungal growth and symptoms. Once again, the lack of symptoms was seen in the leaves of plant genotypes that did not contain LOV1. In the sequence listed above, chlorosis, necrosis, hyphae, and sporulation were seen on the majority of the leaves that were caused by the LOV1 genotype. With regard to the initial infection location, the age of the leaves and the form of the leaves were variables that impacted aspects such as the dispersion of the fungi and the amount of moisture. As a consequence of this, the infection was not uniform throughout all of the leaves; for example, the places where the leaves curled up from the surface were less wet than the parts where moisture collected. Given this heterogeneity and the fact that the only discernible differences in disease development were between genotypes that included LOV1 and those that did not, we simply classified the plants as either susceptible or resistant. This was done since the only evident differences in disease development were between the two groups. The fact that Col-4 transgenic for LOV1 frequently demonstrated higher susceptibility to C. victoriae and victorin sensitivity was an intriguing exception from this norm. It is possible that this was due to variances in the number of copies of the gene. In accordance with the findings shown in SI Table 1, ecotypes Ws and Ws-0 were also less sensitive to victorin and C. victoriae. According to reports (25), it was discovered that Ws-0 has the rar1-1 mutation. We identified polymorphisms for seven SSLP markers that were scattered across the genome and compared the LOV1 sequences of Ws, Ws-0, and rar-1. This was done because of the resemblance that exists between the rar1-1 plants and the Ws ecotype. When compared to plants with the Ws-0 genotype, which had a slightly different LOV1 sequence and four SSLP markers, plants with the rar1-1 genotype had the same LOV1 sequence and SSLP profile as Ws. In the rar1-1 trials that we conducted, we saw comparable outcomes whether we used Ws and Ws-0 as controls. Please find the results of the Ws shown here.

For the purpose of visualizing fungus, Lorang et al. (16) used the approach developed by Keogh et al. (52). For the purpose of investigating the specimens, a stereoscope and a Nomarski optical microscope were used.

IV.DATA ANALYSIS

The RPP8 resistance gene family is remarkably similar to the CC-NBS-LRR protein that is encoded by the Lov1 region of the genome.

The Arabidopsis LOV1 locus, which results in the plant being susceptible to the C. victoriae disease, was discovered on chromosome 1 in the region that is located between the Nga63 and NCCI loci (16). With the help of an additional two hundred victorin-insensitive F2 offspring, we were able to effectively fine-map LOV1. This was accomplished by crossing a victorin-sensitive line, LOV1 (ecotype Cl-0), with a victorin-insensitive line, Col-4. Following the construction of primers that flanked each insertion and deletion, we searched for polymorphisms between LOV1 and Col-4. In addition, we were able to identify polymorphic small insertions and deletions between Ler and Col-0 (17), which allowed us to construct PCR identification markers. Following that, a region of 193 kilobases (kb) employing polymorphic markers was used to map LOV1 (Fig. 1A). This region was located between markers 3571 and 3764.

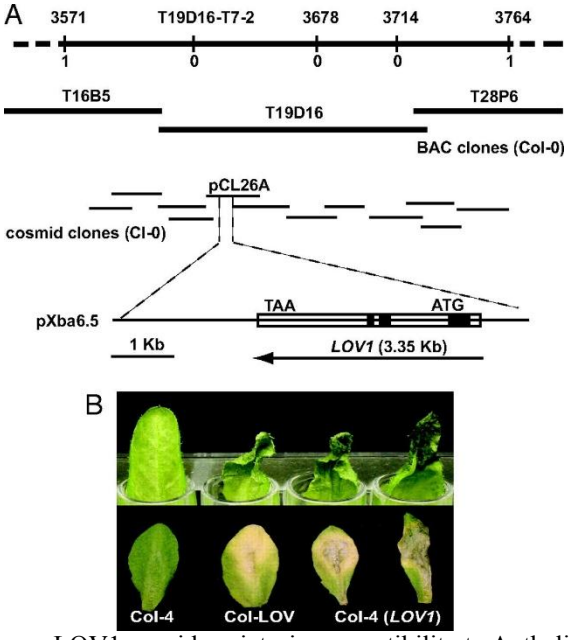

Figure 1: Recently identified gene LOV1 provides victorin susceptibility to A. thaliana and Candida victoriae. A. thaliana Cl-0 LOV1 locus map-based cloning. SSLP markers for LOV1 mapping are provided above the line based on chromosome 1 kilobase position and CAPS marker T19D16-T7-2. Recombinant numbers between markers and LOV1 are below the line. Over time, BAC clones were obtained from the Arabidopsis Information Resource and cosmid clones from a Cl-0 genomic library. LOV1-carrying clones pCL26A and pXba6.5 LOV1 ORF is visible in the open region. Black represents introns. (B) After 36 hours and 5 days, Arabidopsis leaves were treated with 10 µg/ml victorin (Upper) and injected with 1 × 105/ml C. victoriae spores (Lower). The LOV1 gene is expressed in transgenic Col-4 (LOV1), which is virtually isogenic to LOV1, and victorin has no impact on Col-4.

After LOV1 (Cl-0) DNA was produced in a genomic library by using the binary vector pCLD04541 (18), victorin-insensitive Arabidopsis (Col-4) were injected with clones that contained a contig of the 193-kb gap. This is seen in Figure 1B. When exposed to the cosmid clones pCL26A and pXba6.5, it was discovered that C. victoriae was sensitive to victorin and susceptible to Col-4 (Fig. 1 A and B). via the process of DNA sequencing, it was discovered that the annotated genome of Arabidopsis ecotype Col-0 contains a single open reading frame (ORF) for a pseudogene known as At1g10920. This was discovered via the sequencing of clone pXba6.5. There are six polymorphisms that may be found between At1g10920 (Col-0) and LOV1 (LOV1, Cl-0). One of these polymorphisms is a frameshift insertion in LOV1, while the other two are base pair modifications that delete stop codons (GenBank accession number EF472599). LOV1 now codes for an ORF that corresponds to a full CC-NBS-LRR protein. This protein is 86% similar to members of the RPP8 resistance gene family and 70% comparable to them. These changes have occurred as a result of the implementation of these alterations. In addition to combating Hyaloperonospora (Peronospora) parasitica Emco5 (19), turnip crinkle virus (20), and cucumber mosaic virus (CMV-Y) (21) it is a member of the RPP8 family. On the other hand, the susceptibility of C. victoriae is induced by LOV1 (Fig. 1B).

LOV1 Conditions Victorin-Dependent Induction of Defense-Associated Proteins.

Due to the fact that victorin induces a resistance-like physiology in oats that is distinct to a particular genotype, as well as the fact that LOV1 confers a victorin sensitivity that is specific to a certain genotype in Arabidopsis and encodes an R-like protein, we explored defensive responses in Arabidopsis after experiencing victorin treatment. As can be seen in Figure 2, the expression patterns of casalexin and PR-1 are very similar. Despite the fact that Victorin had no impact on Col-4, it soon enhanced PR-1 expression and camalexin synthesis in the Arabidopsis line LOV1 (Cl-0). Other necrotrophic fungi are responsible for activating resistance-associated genes, but neither plant genotype was responsible for producing these genes (2002). Considering that NahG plants, which are responsible for the degradation of SA, could not generate PR-1, it may be deduced that SA is essential for the induction of PR-1 (23).

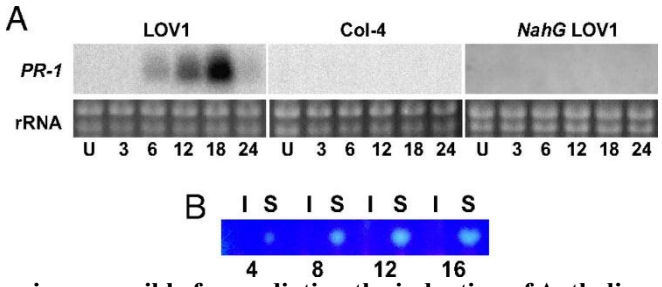

Figure 2: The LOV1 gene is responsible for mediating the induction of A. thaliana defense by Victorin. In Arabidopsis leaves, camalexin accumulation (B) and PR-1 gene expression (A) were detected after infiltration with 30 µg/ml victorin (these quantities are shown below the panels). I is for insensitivity; U stands for untreated; S stands for victorin-sensitive LOV1. Section 4.

Multiple Defense Signaling Pathways Are Dispensable for C. victoriae Susceptibility.

In LOV1 genotypes, mutations in SA-, jasmonic acid-, and ethylene-mediated pathways, as well as in the phytoalexin production pathway, camalexin (PAD3), and the defense-related gene DND1, which is required for HR cell death, were studied (24). These mutations were shown to be essential for HR cell death. These alterations had no impact whatsoever on the susceptibility of victorin-sensitive Arabidopsis to C. victoriae. Due to the fact that these mutations did not have any impact on the illness, we used these signaling pathways in order to evaluate victorin exposure.

The victorin sensitivity receptor is not completely controlled by Lov1. When compared to homozygous plants, LOV1 heterozygous plants exhibit a modest reduction in their sensitivity to victorin. By assessing diluted toxin concentrations on homozygous and heterozygous LOV1 Arabidopsis genotypes, it is feasible to discover small impacts of signaling pathways on victorin sensitivity. If this is done, victorin sensitivity may be improved. There was a little decrease in victorin sensitivity in ein2 mutants when they were exposed to 5 µg/ml victorin in plants that had LOV1 heterozygosity in different mutant contexts, as seen in Figure 3. However, there was no discernible change observed in other mutant settings. In light of these findings, it seems that ethylene may have a very limited role in the susceptibility to sickness caused by victorin. It is not likely that ethylene has a significant impact in the resistance of cucumber mosaic virus to RCY1. Twenty percent of Ein2 Arabidopsis showed a reduction in their resistance to the cucumber mosaic virus.

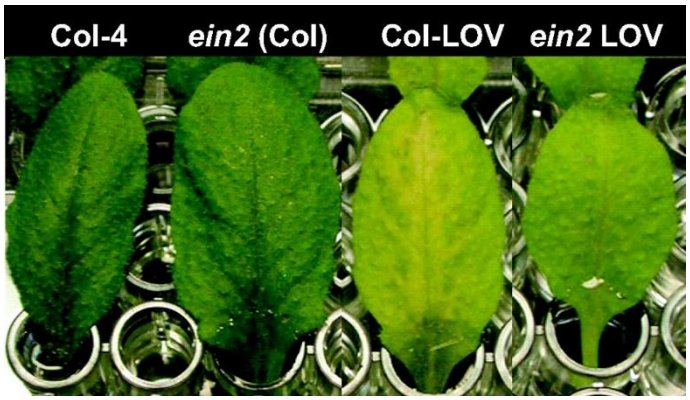

Figure 3: The presence of LOV1 mutations in ein2-1 results in an inhibition of victorin sensitivity. Victorin was given to A. thaliana thirty-six hours after the therapy had been completed.

For steady-state levels of NBS-LRR to be maintained, it is necessary for many R genes to interact with one another in order to offer complete resistance (25-30). It is possible that RAR1 and HSP90, in their capacity as cochaperones, have a beneficial effect on the levels of NBS-LRR (26, 28-30). Through the degradation of cellular proteins, SGT1b may be able to interfere with RAR1 in disease resistance that is conditioned by RPP8 (25, 28). The HSP90 and SGT1b proteins are directly involved in the physical interactions of a variety of R proteins (26, 31). By using RAR1, SGT1b, and HSP90, we were able to study victorin sensitivity that was mediated by LOV1. Both rar1-1 and sgt1b mutations had an effect on the sensitivity of Victorin when it was present in a LOV1 context. For the purpose of determining whether or not victorin sensitivity is required for HSP90, the HSP90-specific inhibitor geldanamycin was used. After 36 hours of treatment, there was no discernible decrease in victorin sensitivity in rar1 (Ws) plants that had been treated with victorin. Within the time frame of 48 to 72 hours, there was no discernable phenotype. The victorin sensitivity of Arabidopsis was not altered by either Sgt1b or geldanamycin, as shown by the results of the study (SI Table 1 and details not shown).

Because RPP8-mediated sickness resistance does not need functional SGT1b or HSP90, mutations in RAR1 have only a moderate influence on the disease resistance that it confers (28). Finally, proteins that influence steady-state levels of certain R proteins (RAR1, SGT1b, and HSP90) are not required to maintain LOV1 at a presumed threshold level. Furthermore, neither the LOV1-conditioned disease susceptibility response nor the RPP8-conditioned disease resistance response require the presence of known defense response signaling pathways according to research (28).

V.CONCLUSION

In spite of the fact that NBS-LRR genes are often associated with plant disease resistance, our research indicates that they may also have an effect on disease susceptibility by themselves. The NBS-LRR gene LOV1 of Arabidopsis thaliana is the cause of the plant's susceptibility to the infections induced by Cochliobolus victoriae. The role of NBS-LRR genes, which are assumed to be implicated in both disease resistance and susceptibility, is made more complicated as a result of this. The extent to which we comprehend the use of R genes in the management of plant diseases is significantly impacted as a result of this.

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