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INSIGHTS INTO CHRYSANTHEMUM WHITE RUST: LIFE CYCLE, DISEASE INCIDENCE, SEVERITY, SYMPTOMS, DISPERSAL, SURVIVAL AND HOST-PATHOGEN INTERACTIONS

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ABSTRACT

Chrysanthemum white rust is a major fungal disease caused by *Puccinia horiana* Henn. This disease severely infects the chrysanthemum nurseries, crop growing in green house and also in open field conditions. Better understanding about pathogen with respect to its life cycle, disease incidence and disease severity, symptoms, disease dispersal and survival, will lead to awareness and adoption of various management practices accordingly. The detail study of various processes involved in molecular signalling, which leads to compatibility and incompatibility, help to isolate and characterize the resistance gene of host and avirulent genes of the pathogen. Identification of resistant/ tolerant germplasm will help the growers to choose the variety for commercial production and also will be effective to expand the cultivation area.
Keywords **:** White rust, Chrysanthemum, pustules, genotype, resistant and susceptible

Introduction

Chrysanthemum (*Dendranthema x grandiflora* Tzvelev.) is an important flower crop, which is used as cut flower, potted plant and for garden display. The quality and quantity of produce in chrysanthemum is affected by a number of pests and diseases. Chrysanthemum white rust is a major fungal disease caused by *Puccinia horiana* Henn. This disease severely infects the chrysanthemum nurseries, crop growing in green house and also in open field conditions. As a biotrophic plant pathogen, this does not cause mortality in plants but is listed as a pest of quarantine importance. Consequently, the noticeable symptoms of this rust resulted in regulatory action. The European Union follows strict quarantine actions to prevent occurrence of this disease in new cropping areas. The consignment (the cuttings or cut flowers) is inspected officially prior to transportation and there should be no symptoms of this disease. If the consignment is found infected with the chrysanthemum white rust, this will lead to rejection of consignment causing huge monetary loss to farmers (EFSA Panel on PLH, 2013).

History of chrysanthemum white rust

Chrysanthemum white rust was first reported in Japan, and then in China, afterwards it was reported in European countries. In present scenario, this disease has been found in major chrysanthemum growing areas (Firman and Martin, 1968). The prevalence of this disease is not reported in chrysanthemum growing areas in Northern India, however in Sothern India, this rust disease was reported a few years back in Bengaluru and Tamil Nadu in 2012. The appearance of this disease is noticed in chrysanthemum growing areas mostly during the winter months. This rust requires low temperatures which is available during the winter months. However, the appearance of this rust was also reported in monsoon season in Bengaluru during 2018 because long duration of heavy rains lowers down the temperature and makes the environmental condition conducive for rust initiation and development. The chrysanthemum white rust affects most commonly grown chrysanthemum species i.e. *Dendranthema grandiflora*. The incidence of disease varied among different genotypes in chrysanthemum. Some genotypes are observed as highly susceptible, while a few are moderately resistant and others are resistant during the same period of disease incidence in the growing chrysanthemum crop (Sriram *et al*., 2015).

Symptoms and sign

Symptoms of white rust are observed mostly on the leaves, however, in severely infected stage, these symptoms can also be seen on buds and stem. The symptom of rust initiates as a very minute yellow spot on the upper side of the leaf and after a few days pink or buff colored pustule start developing on the exact lower side of the yellow spot. The pustule become raised with time and become waxy white. At the latter stages, these pustules become brownish from centre and dry up (Baker, 1967). When the disease incidence is very high, the pustules get coalesced over the leaf surface and reduce the area for photosynthesis, thus, affecting the plant growth and the flower produced are small in size and of poor quality. This disease is causing major monetary loss to chrysanthemum growing farmers.

Anmol **Vijay Kiran** **Pusa Aditya**

Fig. 1 : White rust symptoms in chrysanthemum genotypes under pot culture (Thakur *et al*., 2019)

Dispersal and survival

Puccinia horiana Henn. survive for a longer duration with the host plant. The wind and rains disperse the Basidiospores in the chrysanthemum growing areas. Basidiosopre survive under high humidity and cool temperature for an hour whereas basidiospores get rapidly desiccated under hot wind currents in less than five minutes. Teliospore could survive in previous season plant debris for several months even if it is deeply buried in soil (Kapooria and Zadoks, 1973).

Disease cycle

Puccinia horiana Henn. is an autoecious micro cyclic rust fungus. The pustules scrapped from the lower side of the chrysanthemum leaf can be observed under microscope for the two celled teliospores with a pedicel. Each teliospore further release two or three basidiospore on favorable conditions. This basidiospore is only infection initiating and disease spreading agent. The very high relative humidity, cool temperature (less than 23^{O}C) and a thin water film on leaf is considered favorable condition for disease initiation and disease severity. After the inoculum lands on the chrysanthemum plants, the fungus may remain latent for one or two weeks until the favorable conditions and also depends on different chrysanthemum genotype. Afterwards, a minute yellow spot is initiated on upper side of the leaves and subsequently on lower side of that spot, the pustules are developed. The teliospores in the pustule form the basidiospore for further infecting the new healthy plants (Dickens, 1970).

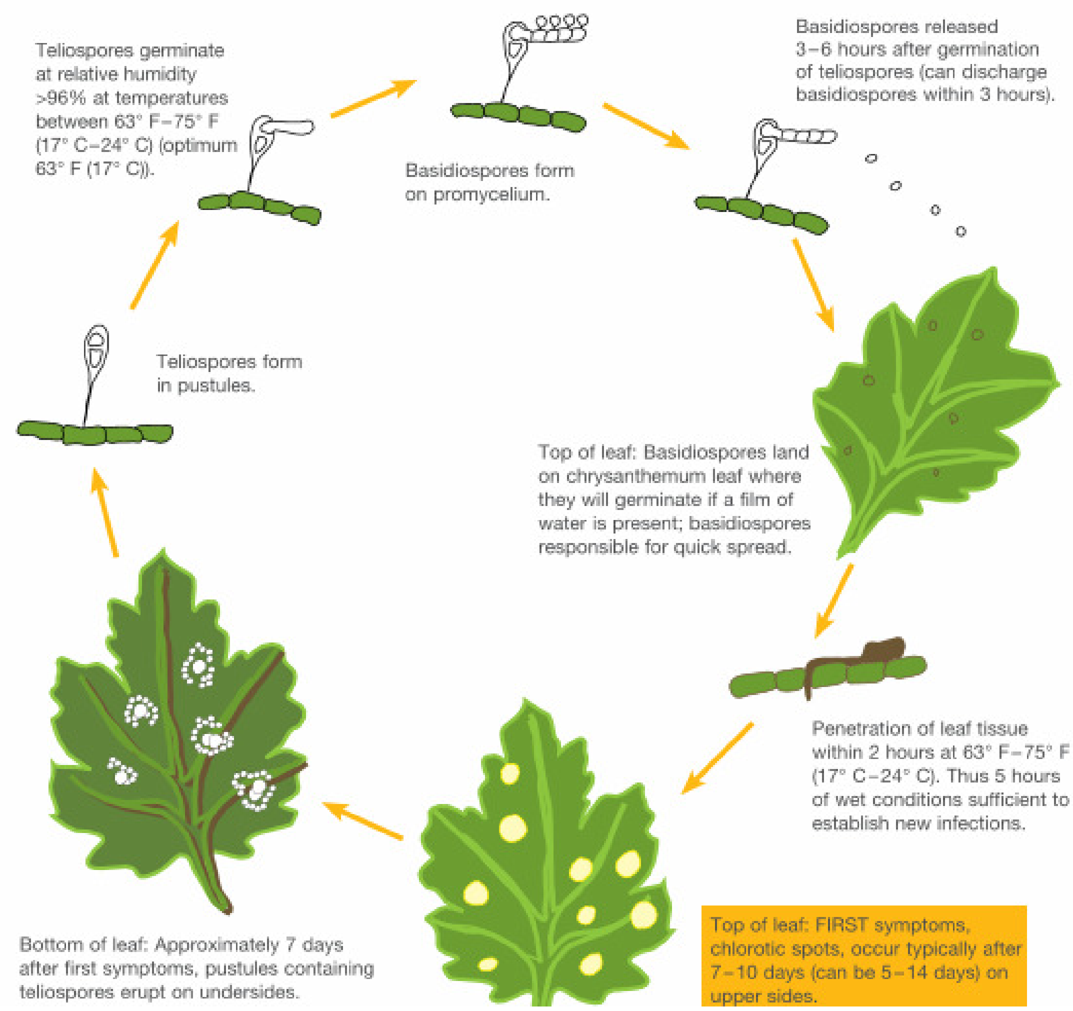

Fig. 2 : Life cycle of *Puccinia horiana* Henn. (Firman and Martin, 1968)

Management of white rust

This rust disease can be managed with Integrated Disease Management which includes use of healthy planting material, avoid closer spacing, proper control of the humidity, avoiding use of overhead irrigation on plant (to avoid the leaf wetness), use of biocontrol agent like *Lecanicillium lecanii* and use of fungicides in recommended doses (Whipps, 1993). When the environmental conditions are favorable for the disease initiation, the planned prophylactic spraying of contact fungicide such as Chlorothalanil is done at regular time intervals. When the disease severity is very high, the systematic fungicide spraying is done. For the management of white rust disease that propiconazole (0.1%) and Tebuconazole + Trifloxystrobin (0.1%) was found effective (Somasekhara and Chandramohan, 2016). The sustainable, environment friendly strategy alternative to chemical use is use of disease resistant varieties and use of healthy planting stock from certified nurseries.

Host-pathogen interaction in chrysanthemum white rust

Host pathogen interaction is important for establishing successful infection. When the inoculum lands on leaf surface, a series of events is produced in the plant in defense to this. The success of the disease development depends upon the how much the pathogen is able to overcome the hindrance provided by the plant to establish the suitable environment for growth and further reproduction. The main difference between susceptible genotype and resistant genotype is that the resistant genotypes are able to recognize the invading pathogen on time and quickly develop the defending response. Whereas, the susceptible genotypes are either not able to recognize the invading pathogen on time or the defending responses elicited are much delayed that the infection is already established (Zheng *et al.*, 2000).

Bonde *et al.* (2015) performed transmission and electron microscopic studies with respect to chrysanthemum white rust caused by *Puccinia horiana* to see how it establishes and cause infection. He found the deformation lines in the mesophyll cell walls of the host, as the pathogen entered systematically by the enzymatic digestion of host cell walls. He also examined the infected crown of the host plant and reported the fungus in vascular tissue. In the severely infected vascular tissue, the fungus has replaced the whole contents of tracheid cell walls.

De Jong and Rademaker (1986) described following four types of reactions with respect to white rust. Complete resistance is reported when no macroscopic symptoms were visible. It is controlled by a single dominant gene. It was reported that the rust spores germinate and penetrate plant via the stomata, but subsequently, the invaded cells die so rapidly that there is no macroscopic evidence of infection. The second type of resistance reported in chrysanthemum was incomplete resistance as few pustules developed over the time. The plants evidently carry a number of barriers that inhibit growth and multiplication of the rust and the penetration into the leaf are more difficult. A high spore-pressure and stringent environmental conditions are needed for successful infection and even the number of pustules per leaf is small. Incomplete resistance superimposed on complete resistance gives additional protection against breakdown and a high frequency of resistance in the progeny. Necrosis occurs when necrotic areas develop around the growing rust colonies. The hypersensitive response to the invading pathogen is usually too slow to prevent sporulation completely. If the numbers of lesions are high, the whole leaf may die. Susceptible cultivars may show large or small pustules with abundant sporulation. The progeny is completely susceptible.

Primary and secondary plant defense responses

The interaction of receptors of the host with elicitors of the pathogen result in cascade of primary defense events such as reactive oxygen species (ROS), protein phosphorylation, ion fluxes and hypersensitive reaction, *etc*. Secondary plant signal molecules such as salicylic acid, etc., intensified the initiated primary defense responses. This leads to activation of a number of plant defense related genes, encoded peroxidases, glutathione S-transferees (GST), proteinase inhibitors, pathogenesis-related (PR) proteins, cell wall proteins, phytoalexin biosynthetic enzymes and hydrolytic enzymes (Zheng *et al*., 2000).

In addition to eliciting primary defense responses, pathogen signals may be amplified through the generation of secondary plant signal molecules such as salicylic acid. At the macroscopic level, induced defense responses are frequently manifested in plant part as a hypersensitive response. In addition to the localized HR, plants may respond to pathogen infection by activating defense responses in uninfected parts of the plant, expressing so called systemic acquired resistance (SAR) that can be long-lasting and often confers broad-based resistance (Ling *et al*., 2007).

Better understanding of processes involved in molecular signalling, which leads to compatibility and incompatibility, help to isolate and characterized the resistance gene of host and avirulence genes of the pathogen. Isolation of effectors proteins of rust will be useful for screening of chrysanthemum germplasm to discover new, potentially durable, resistance genes. This strategy can be used to screen non-host species for new R genes and pyramid those genes for improving white rust resistance in chrysanthemum. The information about involvement of secondary metabolite in resisting the white rust infection will be useful to get resistant varieties.

White rust screening in different chrysanthemum genotypes

The different genotypes of chrysanthemum showed varied degrees of reactions either as resistance or susceptible to white rust (*Puccinia horiana* Henn.). Whole plant methods of screening was used by Martin and Firman (1970) to screen two hundred and seventy cultivars of the *Chrysanthemum sinense* of which only different extent of reaction was observed on ninety three cultivars. The others remaining two-thirds cultivars were categorized as practically immune or immune. Among the ninety-three, forty cultivars were categorized as susceptible as their infected leaves were completely covered with active telia. Others twenty-two cultivars also showed infection in lesser extent with numerously smaller telia and categorized as moderately susceptible. Twenty-five cultivars were categorized as resistant with a very few discrete telia. Six cultivars, instead of showing the chlorotic spot, observed with a very strong necrotic reaction were rated as very resistant. The twenty-six genotypes were observed with the similar necrotic areas but with advances in time these necrotic areas form hollow spots and there is no further teliospores development. These are rated as practically immune. Remaining 151 cultivars showed no symptoms over the crop growing period with any of the three artificial screening tests and were categorized as immune.

Yamaguchi (1981) screened the 250 chrysanthemum cultivars with multiple isolates of chrysanthemum rust. He used the six Japanese isolates of white rust for inoculation of different chrysanthemum cultivars and discovered differential interaction phenotype profiles for several of the cultivars used. The presence of pathotypes (*i. e.* physiological races) is the resultant of the gene-for-gene type of interactions.

The mycoparasitic ability of *Verticillium lecanii* was assessed by Whipps, (1993) for the management of white rust in greenhouse-grown all-year-round chrysanthemums. He reported the *V. lecanii* parasitizes the spores of this rust fungi. Therefore, it has the potential for integrated management of white rust and more research involving various aspects of bio control agents is required for sustainable management of this rust.

Zeng *et al.* (2013) screened the commercial chrysanthemum near-relatives (11 Chrysanthemum species including five accessions of *Chrysanthemum indicum* and four *Ajania* species for white rust under greenhouse at two different locations in eastern China. The *C. shimotomaii, C. japonense, and A. przewalskii* were observed as highly resistant and *C. nankingense, C. crissum, C. vestitum, Chrysanthemum indicum, C. yoshinaganthum, C. makinoi var. wakasaense, C. lavandulifolium and Ajania tripinnatisecta were reported as immune.*

Park *et al.* (2014) screened 179 commercial genotypes and 11 accessions of six wild species for resistance to white rust. The species which were found resistance were *Chrysanthemum boreale*, *C. yashinaganthum,* and *C. zowadaskii*. Other than these three species, 41 spray types of chrysanthemum genotypes were also reported resistant. Twenty-eight spray and nine standard type of chrysanthemum genotypes were rates as moderately resistant. The remaining 18 standard, 83 spray cultivars and three wild species (*Aster spathulifolius*, *C. indicum*, and *C. pacificum*) were highly susceptible.

Deepa et al. (2016) surveyed the fifteen chrysanthemum varieties growing commercially in Yercaud hills and Kothagiri hills, Tamil Nadu for white rust incidence during the winter and summer season. They reported that mean percent disease index was more in winter season with 62.72 per cent at Yercaud and 65.81 at Kothagiri, whereas during summer season, the mean per cent disease index was 39.19 per cent at Yercaud and 42.34 at Kothagiri. This survey revealed that environmental condition during winter season is extremely favourable for the initiation and outbreak of the disease. Among these varieties, irrespective of the season, Saffin Pink was highly susceptible to chrysanthemum white rust (CWR).

Thakur *et al.* (2019) screened forty-nine chrysanthemum genotypes for white rust resistance and categorized according to the disease severity. Among 49 chrysanthemum genotypes, twenty-seven genotypes were identified as resistant with disease severity index of 0.0 (no visible symptom); five genotypes *viz.* Arka Ravikiran, Rekha, Flirt, CO.1 and Fitonia, and as moderately resistant with disease severity index of 0.1-1.0 (few pustules developed slowly and sporulate limitedly); eight chrysanthemum genotypes *viz.* Vasanthik, Pusa Aditya, Ratlam Selection, Anmol, Sunil Marigold, Shyamal and Vijay Kiran as highly susceptible with disease severity index of 1.1-3.0 (many pustules developed), nine genotypes *viz.* Arka Chandrika, Kargil, Rajat, Roopanjali, Sharadmala, Coffee, Winter Queen, Arka Yellow Star and Sadbhavana and as susceptible with disease severity index of 3.1-4.0 (many pustules developed quickly and coalesced over major portion of the leaf and sporulated abundantly).

Wang *et al.* (2007) found differences in accumulation of ROS (O_2^- and H_2O_2) and cell death responses in compatible and incompatible interactions between wheat and its stripe rust fungus. These responses were expressed more rapidly and extensively in the incompatible than in the compatible interaction.

Conclusion

Although the incidence of white rust is not very prevalent in India but our southern growers are facing this problem on large scale. Many methods have been advised and devised from time to time to combat the problem, but due to unawareness and least preference the research in this field has not taken pace. To tackle this effectively we need to focus on crop and disease specific research which could come up with the long-term solution. Researchers have led our understanding to host pathogen interaction which results in infection. Many molecular signalling pathways have been underlined through molecular research on this aspect but still many hidden molecular aspects need to be thoroughly studied. Better understanding of processes involved in molecular signalling, which leads to compatibility and incompatibility, help to isolate and characterize the resistance gene of host and avirulent genes of the pathogen. Isolation of effector proteins of rust will be useful for screening of chrysanthemum germplasm to discover new, potentially durable, resistance genes to white rust. There is also need to study the correlation of various phenols and flavonoides with the disease susceptibility and resistance which will help the researchers to screen the resistant and susceptible at seedling stage.

Identification of resistant/ tolerant germplasm will help the growers to choose the variety for commercial production and also will be a tool to expand the cultivation area.

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