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PATHOGENIC VARIATION IN *PSEUDOMONAS SYRINGAE* AND *XANTHOMONAS CAMPESTRIS* PV. *SESAMI* ASSOCIATED WITH BLIGHT OF SESAME

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Abstract

The role of *Pseudomonas syringae* pv. *sesami* and *Xanthomonas campestris* pv. *sesami*, alone and combination, was studied in symptoms development of bacterial blight in sesame. Highest leaf infection of 80.6 % occurred in plants inoculated with both the pathogens together as compared to individual inoculations (*P. syringae sesami* 75.6%) and (*X. campestris* pv *sesami* 50%). The control plants remained asymptomatic and continued to grow healthier. Significant variability among the two pathogens was noted on defoliation (5%) and stem infection (47.16%) respectively, in case of combined inoculation as against 38 % and 36.66 % in individual inoculations. Responses in stem infection were similar, although in some cases stem tended to be more susceptible. Highest stem infection (47.16%) was observed for P+X, followed by *X. campestris* and *P. syringae* inoculations showing 43.16 and 26.66% infections respectively. Disease progress was initially slow and the plants treated with *P. syringae* and *X. campestris* developed small chlorotic and necrotic areas, but it was severe after two weeks when mixture of P+X was used as inoculum. Initially necrotic spots produced by *P. syringae* were small in size (1-3 mm in length) as compared to by *X. campestris* (2-4mm in length) but after 4 weeks of inoculation, the necrotic spots coalesced and caused defoliation in both cases.

Introduction

Sesame (*Sesamum indicum* L.) is one of the oldest crops domesticated and cultivated by man for cooking and medicinal purposes for more than 5000 years. Now the crop is mostly grown in the tropical and subtropical regions of the world, with maximum concentration and production in Asia. In Pakistan, the sesame is grown on about 102,000 hectares with a total production of about 51,000 tons and yielding only 504 kg/h. The low yields in the country are attributed to prevalence of several parasitic diseases. As far as bacterial diseases are concerned, leaf spot or blight caused by *Pseudomonas syringae* van Hall. pv. *sesami* and *Xanthomonas campestris* (Pammel) Dawson pv. *sesami* is most common, wide spread and inflict heavy losses in production.

Bacterial blight of sesame was first recorded in Sudan (Sabet & Dowson, 1960) and then reported from India, Venezuela (Rao, 1962; Malaguti, 1971) and from Pakistan (Mirza & Akhtar, 1987). The disease mainly develops in the rainy season or with high relative humidity, at night. The disease affects the plant at any age and under severe conditions, producing extensive blight of the foliage, invading petioles, flowers and stems, and causing defoliation and sterility. It is a destructive disease and reported to cause complete loss of crop particularly under rain fed conditions in Sudan (Sabet & Dowson, 1960). Vijayat & Chakravarti (1977) reported 60 % loss in the capsules due to blight under field conditions in Turkey while through artificial inoculation in the field, the disease caused 21-27% loss of yield in India. Approximately 20% loss in yield has been reported from Jalapur area in Madhya Pradesh by Shukla *et al*., (1972).

In Pakistan, the bacterial blight of sesame caused by *P. syringae* pv. *sesami* is generally observed during Kharif season (July-September). Currently this disease has become more devastating and is posing great threat to sesame production in Pakistan. No serious and systematic study has been conducted on this problem except some sporadic records on association of *Pseudomonas syringae* and *Xanthomonas campestris* with the disease. Keeping in view its significance and potential threat to sesame production, the present study was carried out to ascertain the pathogenic role of both the organisms in the development of disease.

Materials and Methods

For the preparation of inocula of *P. syringae* and *X. campestris*, 24-48 hour old slant agar cultures were used. A loopful of bacterial growth was re-suspended in sterile water and the bacterial concentrations were adjusted to 10^8 cfu/ml (through Gennie spectrophotometer at 550 nm).

In order to establish the role of *P. syringae* and *X. campestris* in blight symptoms development of sesame, the most susceptible variety i.e., GP-9, was grown in pots containing sterilized potting mixture consisting of sand, farmyard manure and clay (1:1:2). The mixture was sterilized with 37% formalin. Treated soil was covered with a polythene sheet in sun light for 3-4 days. The sheet was removed and soil was exposed for 5 days to release fumes of formaline. The trial was conducted in the greenhouse of the University of Arid Agriculture, Rawalpindi during growing season 2006. The treatments were as follows:

T_1 = Pathogen inoculation with the mixture of *P. syringae* pv. *sesami* and *X. campestris* pv. *sesami* (P+X).
T_2 = Pathogen inoculation with *X. campestris* alone and
T_3 = Pathogen inoculation with *P. syringae*.

One-month-old seedlings were inoculated by spraying (10^6 CFUs/ml) and stem puncture technique (Carmen, 1995). The appearance of symptoms and development of disease were recorded at weekly intervals, starting from the 7^{th} day after inoculation as percentage of leaf and stem infection, defoliation and lesion size.

Results and Discussion

Highest leaf infection of 80.6%, corresponding to disease severity score (4.03), was observed in the plants inoculated with both the pathogens, *X. campestris* and *P. syringae* while individual inoculations caused 70.6% (3.78) and 50% (2.50) leaf infections, respectively (Fig. 1). The plants kept as control showed no symptoms and continued to grow healthier.

Significant variability was also noted among the pathogens on defoliation and stem infection. Maximum defoliation (50%) was recorded in case of P+X, followed by *P. syringae* and *X. campestris* showing 38% and 36.66% defoliations, respectively (Fig. 2). Responses in stem infection were similar, although in some cases stem tended to be more susceptible. Highest stem infection (47.16 %) was observed for P+X, followed by *X. campestris* and *P. syringae* inoculations showing 43.16 and 26.66 % infections (Fig. 3).

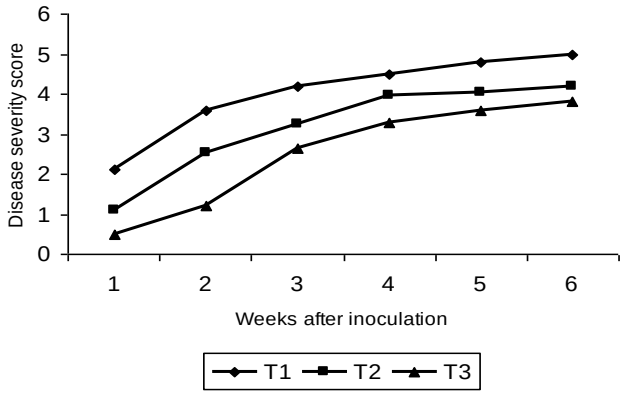

Fig. 1. Effects of *Pseudomonas syringae* pv. *sesami* (T_1) and *Xanthomonas campestris* pv. *sesami* (T_2) alone and in combination (T_3) on leaf infection.

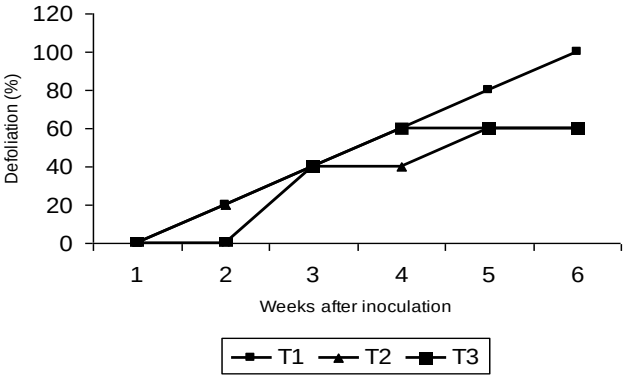

Fig. 2. Effects of *P.syringae* pv. *sesami* (T_1) and *X. campestris* pv. *sesami* (T_2) alone and in combination (T_3) on defoliation.

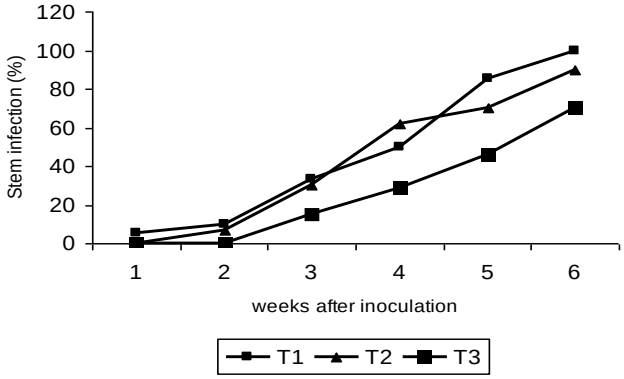

Fig. 3. Effects of *P. syringae* pv. *sesami* (T_1) and *X. campestris* pv. *sesami* (T_2) alone and in combination (T_3) on Stem Infection.

Disease progress was initially slow and the plants inoculated with *P. syringae* and *X. campestris* developed small chlorotic and necrotic areas. Two weeks after inoculation, disease development was severe when mixture of P+X was used. Initially necrotic spots produced by *P. syringae* were small in size (1-3 mm in length) as compared to *X. compestris* (2-4mm in length), but after 4 weeks of inoculation as the disease progressed, the necrotic spots coalesced and defoliation occurred in both cases. Similar results were reported by Hayward & Waterson (1998).

In vitro studies on the interaction between *P. syringae* and *X. campestris* and combination of both the bacteria showed that the fastest occurrence and the highest overall mean severity of the disease symptoms were observed in sesame genotypes sequentially inoculated, except for defoliation which was higher in plants inoculated with the *P. syringae* first then the *X. campestris* on the same day (Young *et al*., 1978). Disease symptoms were less severe in plants inoculated with the *P. syringae* alone. This study showed that both bacteria have a higher significant impact on disease severity. A synergistic relationship seems to exist between *X. campestris* and *P. syringae*, which results in severe disease infection during wet conditions. The disease complex at this stage creates difficulty in distinguishing between the symptoms produced by the pathogens in the field as already reported by Malaguti (1971). Dye (1978) described that *P. syringae* causes dark olive green leaf spots, which increase in size to 2-3 mm and become red-brown to black. The spots coalesce to cover large portions of a leaf. Stem and capsules may also be affected, with oval, slightly raised, dark red-brown lesions. Wasnikar *et al.* (1991) found that when plants were sprayed with inoculum of *X. campestris* in greenhouse experiments, multiplication of pathogen occurred on the inoculated leaves, but not on uninjured leaf surfaces. Blight lesions developed on leaves after seed inoculation in susceptible interactions but not on comparable plants inoculated with a race to which they were resistant. Bacteria increased on these plants, and then diminished more rapidly on the surface of leaves with intermediate reactions as compared to more susceptible leaves. Race specificity of *X. campestris* pv. *sesami* correlated with the resident phase of the bacteria on the leaf surface. Hayward & Waterson (1998) reported that *P. syringae* pv. *sesami* produced blackish-brown spots, which extended along whole length of stem. Veins delimit angular spots on leaves. Infected capsules may blacken and transmit presumably by wind driven rain.

On the basis of results obtained, it was assessed that the severe symptoms were expressed when the two pathogens were inoculated simultaneously. The mechanism involved in disease expression through toxin production was complex for which molecular techniques are required to explain a host-pathogen relationship (Dangl, 1994).

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