Studies of *Albugo candida* on Rapeseed: Yield Effects, Disease Development on Cultivar Mixtures, and Infection Dynamics

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Ronald Steven Pidskalny

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The support and encouragement of my family throughout the course of the programme has made this all possible.

FOREWORD

The format of this thesis follows the manuscript style which has been outlined by the Council of the Faculty of Graduate Studies and the Department of Plant Science of the University of Manitoba. Three manuscripts are presented, including abstract, introduction, materials and methods, results, and discussion. A general abstract, a general introduction, and a review of literature precede the manuscripts. A bibliography and appendices terminate the thesis.

ABSTRACT

PIDSKALNY, RONALD STEVEN. M.Sc., The University of Manitoba, February 1984. Studies of Albugo candida on Rapeseed: Yield Effects, Disease Development on Cultivar Mixtures, and Infection Dynamics. Major Professor; S. Roger Rimmer.

The development of symptoms incited by Albugo candida on Brassica campestris and the effect of infection on yield was examined at two locations in 1982 and at one location in 1983. Foliar infection on the first and second leaves followed similar developmental patterns but comparisons could not be made between sites or between years. Staghead and partial staghead developmental patterns were unique at each location and in each year. Conditions in 1983 encouraged the development of partial stagheads rather than stagheads. The reverse was true in 1982. The application of various fungicide treatments resulted in significantly different levels of white rust infection in B. campestris plots. Disease incidence and severity, however, were not sufficient to result in detectable yield losses.

In 1982 and 1983, the development of disease symptoms incited by Albugo candida was examined in various mixtures of two Brassica campestris cultivars: Torch (susceptible to race seven) and Tobin (moderately resistant to race seven).

Torch and Tobin were mixed by seed weight in four ratios: 1:0, 3:1, 1:1, and 1:3, representing 100%, 75%, 50%, and 25% susceptible plant tissue per treatment, respectively. Early in the season, there were no significant differences in disease levels between treatments. In the treatment containing 25% susceptible plant tissue, foliar and floral symptoms lagged behind those in the pure, susceptible treatment by six and four days, respectively, in 1983 though not in 1982. Pathogen dissemination appeared to be slower in the mixtures than in the pure, susceptible stand. There was a negative correlation between the percentage of Tobin in each mixture and disease levels observed at the end of each season. Despite significantly lower disease levels in the 25% susceptible treatment than in the 100% susceptible treatment, no significant differences in yield were observed.

The inoculation of seven differential test hosts with sporangia of Albugo candida obtained from Brassica campestris and B. juncea indicated that these isolates were races seven and two, respectively. Of eleven cruciferous hosts inoculated with race seven, the B. campestris cultivars Torch and Candle were equally susceptible, followed by the less susceptible B. napus cultivar Triumph, and the moderately resistant B. campestris cultivar Tobin. Raphanus sativus cultivar Raoula and B. oleracea were even less susceptible than Tobin and developed significantly

smaller pustules than the aforementioned cultivars. The B. juncea cultivars Domo and Common Brown were equally susceptible to infection by race two. One race two pustule was found on Tobin. It was similar in size to those initiated by race seven of A. candida.

The development of A. candida race seven was also observed on cotyledons of Torch and Tobin using Epifluorescent and Differential Interference Contrast microscopy. Encysted zoospores with germ tubes were observed on the surface of Torch cotyledons up to two days following inoculation. Colony length and width increased significantly between days three and four though not between days four and five. Sporulation occurred nine days after inoculation. Infection sites were rarely observed on Tobin cotyledons. When observed, however, it appeared that zoospores had encysted in the stomatal cavity, below one of the guard cells. From five days following inoculation until sporulation, colony development on Tobin was similar to that on Torch.

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INTRODUCTION

Rapeseed or canola, an oilseed crop, is grown extensively in western Canada. Two species of oilseed rape are grown, Brassica napus L. and B. campestris L. B. napus (summer rape), with an average of 93 days to maturity is higher yielding than B. campestris (summer turnip rape), a species which requires approximately 79 days to mature (Anon., 1983b). B. campestris is recommended in areas with short frost-free seasons or where seeding has been delayed.

A total of 1.66 million hectares were planted in the prairie provinces in 1982 (Anon., 1983a). The average yield was approximately 1,200 kg/ha. Total production was 2.057 M tonnes.

Albugo candida (Pers. ex. Lev.) Ktze., a member of the Oomycetes, is an obligate parasite. It is the causal agent of white rust or staghead, one of the more prominent diseases of B. campestris on the Canadian prairies. Other members of the Cruciferae as well as some members of the Capparidaceae are also susceptible to infection by A. candida (Mukerji, 1975). Biga (1955) has listed 241 species in 63 genera of the Cruciferae which are attacked by the white rust fungus.

Symptoms produced by the pathogen are first noticed when white coalescing lesions begin to form on the abaxial leaf surface. These pustules are thought to have little effect on yield (Harper and Pittman, 1974). Most losses due to white rust have been attributed to the infection of floral tissues with resultant hypertrophy or staghead formation. A linear relationship has been proposed relating yield loss to the percentage of B. campestris plants systemically infected with white rust.

White rust was the only disease of any consequence in rape in Saskatchewan in 1958 (Vanterpool, 1958). Yield reductions in Manitoba in 1971 were estimated at between 30% and 60% in severely infected fields (Bernier, 1972). In Saskatchewan, Petrie (1973) reported yield losses of approximately 3%, 6%, and 9% in 1970, 1971, and 1972, respectively. Yield losses were approximated at 1.2% in northern and central Alberta in 1971 (Berkenkamp, 1971). In India, white rust was found to be a serious disease of mustard if humidity was high during flowering (Kumari et al., 1970).

Canadian varieties of B. napus have been shown to be immune to isolates of A. candida from B. campestris in both field and laboratory studies (Petrie, 1975a, Petrie and Dueck, 1979). In China, however, some B. napus cultivars are quite susceptible to white rust (Fan et al., 1983). Recently, a moderately resistant variety of B. campestris,

Tobin, has been introduced. Commercial mustard (B. juncea Coss.) cultivars though moderately resistant to isolates from B. campestris are susceptible to isolates from B. juncea. With these facts in mind, the objectives of this study are (a) to examine the race concept in A. candida using various cruciferous species and two isolates of A. candida, (b) to evaluate the resistance of two B. campestris cultivars, Torch and Tobin, to A. candida in terms of infection and colony development, (c) to evaluate the effects of A. candida on yield of B. campestris, and (d) to evaluate disease development incited by A. candida in various mixed stands of the susceptible cultivar, Torch, and the moderately resistant cultivar, Tobin.

LITERATURE REVIEW

1. Nature of the pathogen

1.1 The pathogen

Albugo candida (Pers. ex Lev.) Ktze., an obligate parasite, is one species in the only genus of the family Albuginaceae (Webster, 1980) of the order Peronosporales (Talbot, 1971). A. candida has several synonyms: Aecidium candidum Pers., Uredo cheiranthi Pers., Cystopus candidus Lev., Uredo candida Pers. (Walker, 1957), Uredo candida thlaspeus Pers. (Mukerji, 1975), and Albugo cruciferarum S. F. Gray (Connors, 1967). The Albuginaceae cause white rust of the leaves and stems, and hypertrophy of floral structures of the Cruciferae and some species of the Caparidaceae (Walker, 1957). Some economically important crops are susceptible to infection by several species of Albugo: A. occidentalis G. W. Wilson causes white rust of spinach (Walker, 1957); A. ipomoeae-panduraneae (Schw.) Swing. or A. minor (Speg.) Cif., white rust of sweet potato; A. tragopogonis (DC.) S. F. Gray, white rust of salsify (Tragopogon porrifolius), goatbeard (T. pratensis) (Webster, 1980), and Senecio squalidus and A. candida, white rust of a number of cruciferous hosts (Biga, 1955).

1.2 Races of the pathogen

Host specialization has been exhibited in A. candida (Melhus, 1911; Hiura, 1930; Napper, 1933; Togashi and Shibasaki, 1934). Six biological races were later established (Pound and Williams, 1963). Three races are of some economic importance: race one infects radish (Raphanus sativus L.); race two, leafy mustard (B. juncea (L.) Coss.); and race three, horseradish (Armoracea rusticana Gaertn. Mey & Scherb.). The three races of little economic importance, races four, five, and six, are limited to the host weeds on which they are found; shepherd's purse (Capsella bursa-pastoris (L.) Medik.), hedge mustard (Sisymbrium officinale (L.) Scop.), and marsh yellow cress (Rorippa islandica (L.) Bess), respectively. Two other isolates have since been described. Williams designated an isolate obtained from B. campestris L. as race seven (Verma et al., 1975) and an isolate from B. nigra (L.) Koch. has been designated race eight. (Delwiche and Williams, 1977).

1.3 Life cycle

Intercellular mycelia of A. candida mass beneath the abaxial host epidermis producing a layer of cylindrical sporangiophores from which sporangia arise, one at a time, as buds from the apices (Khan, 1977). The lengthening chains eventually produce sufficient pressure to rupture the host epidermis and release the wind disseminated sporangia

(Fraymouth, 1956). Germination occurs most readily shortly after spore release (Eberhardt, 1904) but only after a period of chilling temperatures (Melhus, 1911). Napper (1933) has determined that the water content of spores must be reduced to approximately 30% before germination of sporangia is possible. At temperatures between 1 and 20 C, sporangia germinate within a few hours in thin films of water on host leaves (Melhus, 1911). About eight biflagellate zoospores are released (Webster, 1980). The zoospores swim for a period of time, encyst, and then develop germ tubes which enter through the adaxial stomata (Walker, 1957). In susceptible hosts, haustoria soon develop and the mycelium proliferates. Sporangia develop within 10 days (Webster, 1980) and the cycle may be repeated several times during a single growing season. In resistant hosts, pathogen growth is arrested in the substomatal chamber (Walker, 1957).

Stagheads form towards the end of each growing season and it is within these hypertrophied tissues that A. candida reproduces sexually. Multinucleate oogonia and antheridia are formed in the intercellular spaces of the stem (Webster, 1980). The gametes fuse and the zygote nucleus begins to divide repeatedly forming a mature oospore. Circumstantial evidence suggests that A. candida is heterothallic (Sansome and Sansome, 1974). Oospores probably provide the principle means of overwintering for A. candida (Walker, 1957).

Mycelium has been known, however, to survive in the crown and roots of perennial hosts (Kadow and Anderson, 1940; Takeshita and Linn, 1953). In the spring, the outer covering of the oospore bursts and the endospore is extruded (Webster, 1980). Within the endospore, 40 to 60 zoospores are differentiated, released, and may incite disease in suitable hosts.

Oospores are considered to be a primary source of spring inoculum (Vanterpool, 1959; Verma and Petrie, 1975a; Petrie, 1975b). They may remain viable for extended periods of time, germinating even after 20 years in dry storage under laboratory conditions (Verma and Petrie, 1975b). In addition to a thick and highly differentiated cell wall, an oogonial wall, and a persistent material in the periplasmic space may play a role in keeping the oospores viable (Tewari and Skoropad, 1977). That oospores of A. candida were found in 468 of 585 seed samples of B. campestris in western Canada indicates that large quantities of primary inoculum would be present in most turnip rape fields every spring. It has also been suggested that spring rains may cause a leaching effect comparable to conditions which have stimulated oospore germination in the laboratory (Verma and Petrie, 1975a).

1.4 Spore germination in vitro

Vanterpool (1959) found oospore germination of Albugo candida to be irregular and uncertain. Though 1 to 4% germination was sometimes possible, only a trace was usually obtained. Using an improved technique, Petrie and Verma (1974) reported up to approximately 70% germination and later up to 88% germination (Verma and Petrie, 1975). Germination has been reported at temperatures between 10 and 20 C (Verma and Petrie, 1975a), between 10 and 12 C (Vanterpool, 1959), and at 13 C (Petrie and Verma, 1974). Maximum germ tube growth was attained at 15 C. Free moisture is required for oospore germination to occur. Sterile distilled water (Vanterpool, 1959), sterile tap water (Petrie and Verma, 1975; Verma and Petrie, 1974; Vanterpool, 1959), non-sterile tap water, and sterile deionized water (Petrie and Verma, 1975) have been used.

Sporangia of A. candida germinate at temperatures between 1 and 18 C with maximum germination occurring between 10 and 15 C (Melhus, 1911; Napper, 1933; Pound and Williams, 1963). As with oospores, sporangia also require free moisture for germination (Endo and Linn, 1960). Good germination occurs in rain water, deionized water, distilled, and double distilled water (Endo and Linn, 1960). Light or dark conditions have no affect on the germination of sporangia in vitro (Endo and Linn, 1960; Melhus, 1911).

2. Nature of the disease

2.1 Symptoms of white rust

A. candida infection may be detected by the appearance of distorted stems and floral structures (stagheads), and white or cream coloured blisters on the leaves and stem (Webster, 1980). Leaf and stem lesions have been referred to as local infections whereas the term systemic infection has been used to describe staghead formation (Walker, 1957).

Local infection is characterized by raised, shiny pustules or sori one to two mm in diameter which commonly develop on abaxial leaf surfaces (Walker, 1957) and later on green hypertrophied tissue (Petrie, 1975a). Pustules have also been described on adaxial leaf surfaces (Williams, personal communication, 1983). Pustules may appear quite small and in close proximity or may coalesce and form large spreading lesions (Walker 1957). After the pustule has matured, the host epidermis ruptures releasing powdery, wind dispersed sporangia (Petrie, 1975a). As the season progresses the fungus becomes systemic, stimulating hypertrophy and hyperplasia in the host tissue (Walker, 1957). Sepals become enlarged, petals enlarge and produce chlorophyll rather than typical flower pigments (Walker, 1957), stems and pods begin to swell and inflorescences may be partially or completely replaced by sterile structures which have the appearance of stag antlers (Petrie, 1975a). These stagheads consist almost entirely of thick walled, overwintering oospores (Verma and Petrie, 1975a).

2.2 Staghead formation

The auxin concentration in host plants is commonly increased after infection by obligate fungal pathogens (Shaw and Hawkins, 1958; Daly and Inman, 1958). Aberrant tissue growth as a result of fungal infection has also been associated with an increase in the auxin levels of host plants (Gruen, 1959). Kiermayer (1958) found that hyperplasia and hypertrophy of floral parts could be induced in Capsella bursa-pastoris and B. campestris by A. candida. Diffusible auxin levels were found to increase in the host tissue (Hirata, 1954). Before the terminal stages of gall formation, however, a decrease in diffusable auxin was observed. It was suggested that the pathogen was responsible for auxin production (Hirata, 1956). Srivastava et al. (1962) found a lowered auxin concentration in Albugo infected floral parts of Brassica. Petrie (1975a) interpreted this finding as an indication that auxin levels may fall as hypertrophied tissue ages. The production of tryptophan in the hypertrophied peduncles of mustard and the disappearance of tryptophan from normal mustard flowers excited to hypertrophy has been reported (Kumari et al., 1970). In the peduncle, tryptophan formed during hypertrophied growth was thought to react with endogenous phenolic acids to produce IAA, which in turn causes hypertrophied growth. The same process was postulated in the flowers except that endogenous tryptophan is used to produce IAA.

3. Host-pathogen interactions

3.1 Infection process and the host-pathogen interface

The development of A. candida on Capsella bursa-pastoris, Cardamine hirsuta, Sisymbrium officinale, Alyssum saxatible, Aubretia spp. and Arabis spp. was characterized along with other interactions between members of the Peronosporales and their respective hosts (Fraymouth, 1956). After penetration of a susceptible host, hyphae grew intercellularly, branching in all directions and kept in intimate contact with the host cell walls. They took the shape of the intercellular spaces in which they occurred, not uncommonly reaching a size of six or seven times the width of a typical hyphal element. Where the leaf structure was firm, as it appears to be in B. campestris and B. napus, hyphae remained quite narrow and did not appear to disrupt host cells to any great extent.

Intracellular haustoria soon developed, connected by a narrow neck to larger hyphal mother cells (Coffey, 1975). Penetration of the host cell was achieved through the cell wall (Fraymouth, 1956). Haustoria often penetrated near the junction of adjacent host mesophyll cells and were usually found in the vicinity of chloroplasts (Coffey, 1975). They invaginated but did not penetrate the host protoplast. A series of tubular and vesicular elements extended from the invaginated host plasmalemma into the host cytoplasm in the vicinity of the haustorium and were evidence of a secretory

process induced in the host by the parasite (Berlin and Bowen, 1964).

3.2 Infection studies

There has been some disagreement regarding the pattern of "green island" formation as well as the photosynthetic capacity of "green island" tissue in various host-pathogen combinations (Allen, 1942; Wang, 1961). The "green island" effect has also been investigated using detached cotyledons of B. juncea infected with race two of A. candida (Harding et al., 1968). More labelled carbon dioxide was fixed in "green island" than in non-infected tissue in light while approximately equal amounts were fixed in the dark. Prolonged photosynthetic activity and the maintainance of chlorophyll content in "green islands" was due to the delayed breakdown of chloroplasts. Kinetin treated tissue reacted in a similar manner as "green island" tissue supporting strong circumstantial evidence for cytokinin involvement in delaying leaf senescence.

The infection and development of race seven of A. candida was later investigated in cotyledons of four Brassica species: B. campestris, susceptible; B. hirta, moderately susceptible; and B. napus, resistant (Verma et al., 1975). Race two was observed on B. juncea, a mustard cultivar susceptible to race two of A. candida. No apparent differences were noted in the infection processes in the

various hosts up to the time of formation of the first haustorium. Mycelial growth ceased in B. napus two to three days after inoculation and an encapsulation formed around each haustorium. Mycelial growth accelerated after haustorium formation in the susceptible hosts.

Two types of resistance to race one of A. candida were found in two varieties of Raphanus sativus (Williams and Pound, 1963). A hypersensitive response in China Rose Winter was initiated at the time of fungal invasion of host tissue. As haustoria were produced, the death and suberization of cells bordering intercellular hyphae resulted in destruction of the fungus. That there was no evidence of penetration of the variety Round Black Spanish is indicative of manifestation of the resistance mechanism outside of the host tissue.

4. Disease control

4.1 Host resistance

Resistance to various races of A. candida has been reported in a number of cruciferous hosts. Resistance to race one was found in two varieties of radish, China Rose Winter (CRW) and Round Black Spanish (RBS) (Williams and Pound, 1963). Crosses with the susceptible variety Red Prince indicated that resistance in CRW and RBS was controlled by one dominant gene. Manifestations of resistance, however, occurred by means of different

mechanisms. Also, RBS resistance was not environmentally sensitive whereas the reaction of CRW to infection ranged from resistance to tolerance depending on growing conditions. This suggested the presence of minor genes in CRW capable of modifying the expression of the major resistance gene. The major resistance gene has been designated Acl (Humaydan and Williams, 1976).

Resistance to A. candida race two has also been reported. Ebrahini et al. (1976) found resistance to the white rust pathogen in accession P.I. 347618. Fl progenies from crosses of resistant and susceptible parents resulted in reactions resembling the resistant parent. Parui and Bandyopadhay (1973) found a collection of yellow rai T-4 which was virtually immune to infection by A. candida. Resistance of B. nigra (susceptible to A. candida race eight) to race two of A. candida has been reported as under the control of a dominant gene (Delwiche and Williams, 1976).

Hougas et al. (1952) presented evidence of resistance of horseradish to race three of A. candida. Successful matings of the resistant Bohemian strain and the susceptible Common strain of horseradish were found to have a wide range of susceptiblilty to white rust race three. Three types of host-parasite reaction were observed: abundant sporulation (more susceptible than Bohemian), limited sporulation (as susceptible as Bohemian), and no sporulation (more resistant than Bohemian).

Resistance of B. napus to race seven of A. candida was thought to be conditioned by three independent dominant genes: Ac7-1, Ac7-2, and Ac7-3 (Fan et al., 1983). Dominance at any one locus would confer resistance whereas trigenic recessives were susceptible. The idea that minor genes may modify the disease reaction has been suggested by the variation in the susceptibility of two Chinese lines, 2282-9 and Green Cup Leaf, and their F1 and F2 progenies.

4.2 Mixed cutivars

Consideration has been given to cultivar mixtures as a means of controlling plant disease epidemics. Mixtures would be most useful in situations where newly introduced, resistant cultivars were lower yielding than older, susceptible cultivars. Through field experimentation it would be possible to determine the proportion of resistant to susceptible plants in a crop stand which would optimize yield. Factors such as the effect of disease on yield loss and the severity of the disease in any given season, as well as the difference in yield between the resistant and the susceptible cultivars would play a role in determining the optimum proportion of resistant and susceptible plants.

The effect of cultivar mixtures on epidemic progress has been outlined by Wolfe and Barrett (1980). Mixtures had little advantage at the beginning of the season when inoculum was predominantly from external sources. When

disease increase became dependent upon inoculum buildup within the crop, pathogen dissemination was slower in mixtures than in the component pure stands. Towards the end of the season, the progress of the epidemic seemed to accelerate in the mixtures because the infection, still increasing in the healthier mixtures, had reached a saturation level and had stopped developing in the pure stands.

Cultivar mixtures, often higher yielding than any of their components could be recommended for other than epidemiological reasons (Simmonds, 1962). Jensen (1952) reported that yields of field blends of oat varieties exceeded the average yield of all components included in the mixture. Though these data were not statistically significant, there was no reason to rule against the use of a mixture on the basis of lowered efficiency. At the highest seeding rate examined, a 1:1 mixture of two spring wheat cultivars yielded higher than pure stands of either component cultivar in one year, but lower in another (Baker, 1977). It was suggested that various environmental conditions, as well as the seeding rate, could have been responsible for this discrepancy.

Pfahler (1965) proposed that heterogeneous populations composed of a number of homozygous varieties could be utilized not only to maximize grain yield, but to minimize variability in production. Varietal interactions, however,

necessitated the actual testing of the composite. A prediction of composite response based solely on the performance of component cultivars in pure stands was considered unreliable. Baker (1977) did not think that it was possible to determine the yield of a composite variety based on component variety yield and yield component response in pure stands.

Using two mathematical models, Jeger et al. (1981a) examined the progress of an unspecialized pathogen in pure and mixed stands of cereal cultivars. In most situations, the simple discrete model predicted that in mixtures the amount of disease would be less than or equal to the arithmetic means of the component pure stands. An increase in disease was predicted when the infection frequency and sporulation rates of the component cultivars were opposed in rank. Solutions to the model employing differential equations supported the conclusions of the discrete model.

These models also proved useful in field situations (Jeger et al., 1981b). Mixtures of two spring wheat and two winter wheat cultivars with different levels of resistance to Septoria nodorum and two winter barley cultivars with different levels of resistance to Rhynchosporium secalis reduced disease levels almost to those of the more resistant pure stand. There was no significant effect on grain yield due to low disease levels.

Though net blotch was severe and persistant on barley, Suneson (1949) noted that the susceptibility of mixture components was not correlated with competitive ability as measured by a plant census the following year. Disease data for scald and mildew, as well as net blotch, were closely related to yield.

Two oat varieties, one resistant and one susceptible to race seven of oat stem rust, were grown in a 1:1 mixture and in pure stands (Browning, 1957). In subsequent seasons, after being subjected to an induced epiphytotic of race seven, the mixture yielded seven bushels/acre and 14.5 bushels/acre above the average yields of the component varieties grown in pure stands. The susceptible variety supported much less rust in the field blend than in the pure stand.

Leonard (1969) observed the disease progress in cultivar mixtures under conditions of minimum racial interaction, where compatible type pustules were produced on different host plants, and maximum racial interaction, where both races produced compatible type pustules on the same cultivar. When minimal racial interaction occurred, the amount of rust produced was almost exactly equal to the sum of the amounts produced in similar mixtures where each race increased alone. Considerably less rust was produced under conditions of maximum racial interaction indicating that competitive inhibition occurred when both races were

virulent on the same variety. In these mixtures, resistant plants seemed to play a role in reducing the rate of increase of the pathogen by reducing the quantity of effective spore dispersals. Autoinfecting spores were not deemed to be subject to the effects of the resistant plants in the field mixtures.

During an epidemic, the average estimated incidence of Cercospora apii on a tolerant celery cultivar lagged behind that on the susceptible cultivar by about three weeks (Berger, 1973). Following the initial infection, there was little difference between the cultivars in calculated periodic infection rates. Later, a major flush of new foliage in the tolerant cultivar had the effect of diluting the increase in the amount of infected foliage. That the susceptible cultivar did not display such a flush was reflected in its higher infection rate for that period. The protective effect of tolerant plants in the mixed stand was observed as long as the disease incidence remained below 25%. There was a positive relationship between the percentage of tolerant plants in the population and the protective effect afforded by these plants.

The development of Erysiphe polygoni was evaluated on several mixtures of Ne Plus Ultra (NPU), a highly susceptible swede cultivar (B. napus), and Ruta Otofte (RO), a partially resistant cultivar (Stitch and Wittington, 1983). Early in the season, population composition

significantly affected infection rate; disease on NPU increased less rapidly as the percentage of RO was raised. Soon, early disease control was lost with NPU ultimately supporting similar disease levels in all treatments. Averaged over all assessment dates, though, an increasing proportion of RO was correlated with decreasing disease on the NPU component of each mixture. Whereas consistent yield deviations from expected values were not exhibited in RO, NPU outyielded expected values in mixtures by 7 to 21%, conceivably reflecting the disease control offered by cultivar mixtures.

4.3 Chemical control

Fungicidal control of white rust has been achieved in both B. campestris and B. juncea. In the field, effective control of foliar infection as well as increased yield was reported with Polyram (zinc activated polyethylene thiram disulfide) on B. campestris cultivar Sarson (Perwaiz et al., 1969). Staghead incidence was significantly reduced in B. campestris cultivar Torch with three foliar applications of the fungicide CGA 48988 [methyl N-(2,6-dimethylphenyl)-N-methoxyacetyl-2-alaninate] (Metalaxyl) (Altman and Campbell, 1977). Seed treatment proved ineffective for white rust control. Foliar infection of B. campestris cultivar Torch was reduced with two applications of three protectant fungicides: chlorothalonil

,mancozeb, and DPX 164 (MBC 10% + Maneb 64%) (Dueck and Stone, 1979). Staghead formation, however, was not inhibited. The acylalanine fungicide CGA 29212 [methyl N-(2,6-dimethylphenyl)-N-chloroacetyl-2-alaninate] significantly decreased staghead incidence.

In the greenhouse, foliar infection was controlled on B. campestris cultivar Torch with applications of chlorothalonil or mancozeb ([ethylene-bis(dithiocarbamate)] manganese) prior to inoculation and at seven day intervals (Verma and Petrie, 1975b). CGA 29212 and Metalaxyl proved to be excellent eradicants of foliar infection of Torch caused by A. candida in the growth room (Dueck and Stone, 1979).

Of 11 fungicides tested, only Miltex (co-ordinated product of Zineb and copper oxychloride) and Brestan (tin triphenyl-tin acetate) provided effective control of white rust of B. juncea cultivar RL-18 while at the same time demonstrating significantly higher yields over the control plots (Gupta et al., 1977). Blitox (copper oxychloride) and Dithane Z-78 (zinc ethylene bisdithiocarbamate) were found to be the most effective fungicides in controlling white rust of B. juncea cultivar RL-18 (Bains and Jhooty, 1979).

4.4 Relationship between disease incidence and yield loss

Harper and Pitman (1973) claimed a relationship between yield loss and systemic infection of B. campestris cultivar Span by A. candida. The equation

$$Y = 100 - 0.952X$$

where Y= yield as a percentage of the potential and X= percentage of stems systemically infected was offered for use in disease loss assessment. Plants had been divided up by disease category and yield was evaluated for each category.

INFECTION OF VARIOUS CRUCIFEROUS SPECIES WITH WILD TYPE ISOLATES OF WHITE RUST FROM RAPESEED AND MUSTARD

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Abstract

The inoculation of seven differential test hosts with sporangia of Albugo candida obtained from Brassica campestris and B. juncea indicated that these isolates were races seven and two, respectively. Of eleven cruciferous hosts inoculated with race seven, the B. campestris cultivars Torch and Candle were equally susceptible, followed by the less susceptible B. napus cultivar Triumph, and the moderately resistant B. campestris cultivar Tobin. Raphanus sativus cultivar Raoula and B. oleracea were even less susceptible than Tobin and developed significantly smaller pustules than the aforementioned cultivars. The B. juncea cultivars Domo and Common Brown were equally susceptible to infection by race two. One race two pustule was found on Tobin. It was similar in size to those initiated by race seven of A. candida.

The development of A. candida race seven was also observed on cotyledons of Torch and Tobin using Epifluorescent and Differential Interference Contrast microscopy. Encysted zoospores with germ tubes were observed on the surface of Torch cotyledons up to two days following inoculation. Colony length and width increased significantly between days three and four though not between days four and five. Sporulation occurred nine days after inoculation. Infection sites were rarely observed on Tobin cotyledons. When observed, however, it appeared that

zoospores had encysted in the stomatal cavity, below one of the guard cells. From five days following inoculation until sporulation, colony development on Tobin was similar to that on Torch.

Introduction

The white rust or staghead fungus Albugo candida (Pers. ex. Lev.) Ktze., is the causal agent of one of the more prominent diseases of Brassica campestris L. (summer turnip rape) on the Canadian prairies. Average yield losses have been reported at between 1.2 and 9% (Berkenkamp, 1971; Petrie, 1973). Yield reductions of 30 to 60% have occurred in severely infected fields (Bernier, 1972).

Two isolates of A. candida are of some economic importance on the prairies. One isolate obtained from B. campestris has been designated race seven (pers. comm. Williams - see ref: Verma et al., 1975). The other isolate designated race two was procured from B. juncea (L.) Coss. (leafy mustard) (Pound and Williams, 1963).

The development of A. candida on Capsella bursa-pastoris, Cardamine hirsuta, Sisymbrium officinale, Alyssum saxatile, Aubretia spp. and Arabis spp. has been characterized along with other interactions between members of the Peronosporales and their respective hosts (Fraymouth, 1956). After penetration of a susceptible host, hyphae grew intercellularly, branching in all directions and kept in intimate contact with the host cell walls. They took the shape of the intercellular spaces in which they occurred, not uncommonly reaching a size of six or seven times the width of a typical hyphal element. Where the leaf structure was firm, as it appears to be in B. campestris and B. napus,

hyphae remained quite narrow and did not appear to disrupt host cells to any great degree.

Intracellular haustoria soon developed, connected by a narrow neck to larger hyphal mother cells (Coffey, 1975). Penetration of the host cell was achieved through the cell wall (Fraymouth, 1956). Haustoria often penetrated near the junction of adjacent host mesophyll cells and were usually found in the vicinity of chloroplasts (Coffey, 1975). They invaginated but did not penetrate the host protoplast.

The infection and development of race seven of A. candida was investigated in cotyledons of four Brassica species: B. campestris, susceptible; B. hirta, moderately susceptible; and B. napus, immune (Verma et al., 1975). B. juncea was inoculated with race two of A. candida. No apparent differences were noted in the infection processes in the various hosts up to the time of formation of the first haustorium. Mycelial growth ceased in B. napus two to three days after inoculation and an encapsulation formed around each haustorium. Mycelial growth accelerated after haustorium formation in the susceptible hosts.

Resistance of B. napus to race seven of A. candida was thought to be conditioned by three independent dominant genes: Ac7-1, Ac7-2, and Ac7-3 (Fan et al., 1983). Dominance at any one locus would confer resistance whereas trigenic recessives were susceptible. The idea that minor genes may modify the disease reaction has been suggested by

the variation in the susceptibility of two Chinese lines, 2282-9 and Green Cup Leaf, and their F1 and F2 progenies.

In evaluating resistance to A. candida it would be desirable to understand the nature of the stability of pathogenic isolates. Though isolates of the white rust fungus cause the most severe disease symptoms on the host from which they were obtained, they may also infect a variety of other cruciferous species (Pound and Williams, 1963). It may therefore not be effective to use only race seven in screening B. campestris for resistance to A. candida. Race two is of particular interest, having a wide host range on various Brassica species including turnip (B. campestris var. rapa), collard, and several seed mustards (Pound and Williams, 1963).

The infection process of A. candida in the newly introduced B. campestris cultivar, Tobin, with moderate resistance to A. candida and the susceptible B. campestris cultivar Torch was also investigated.

Materials and Methods

A relationship concerning the genomes of related Brassicas provides the basis for cultivar selection in this study (Tsunoda, 1980).

B. nigra (n=8), B. oleracea (n=9), and B. campestris (n=10) are primary species containing a single genome each, bb, cc, and aa, respectively. B. juncea (aabb), B. carinata

(bbcc), and B. napus (aacc) are amphidiploids derived from these primary genomes. B. carinata (n=17) is the product of hybridization of B. nigra and B. oleracea. B. juncea (n=18) is the amphidiploid from B. nigra and B. campestris. B. napus (n=19) is the amphidiploid of B. oleracea and B. campestris.

Disease incited by races two and seven of A. candida was evaluated macroscopically on 11 cruciferous cultivars: B. napus L. (summer rape) cv. Regent (resistant to race seven), and Triumph (susceptible to race seven); B. campestris cv. Tobin (moderately resistant to race seven), Candle (susceptible to race seven), and Torch (very susceptible to race seven); B. juncea cv. Domo (moderately resistant to race two), Common Brown Mustard, and Pusa Bold (both susceptible to race two); B. carinata A. Braun, B. oleracea L., and Raphanus sativus L. (n=9) (susceptible to race one).

In order to confirm that the two isolates used in this study were indeed races two and seven, five of six original differential test hosts (Pound and Williams, 1963) were inoculated: R. sativus cv. Raoula; B. juncea cv. Southern Giant Curled; Armoracia rusticana (susceptible to race three); Capsella bursa-pastoris (susceptible to race four); and Rorippa islandica (susceptible to race six). Two additional species, B. campestris cv. Torch, and B. nigra (susceptible to race eight) (Delwiche and Williams, 1977) were included in the test. A microscopic examination of the

infection process of A. candida was carried out on Torch and Tobin.

Seeds were germinated on moistened filter paper in covered Petrie dishes. When the cotyledons had expanded, seedlings were transplanted into peat cups containing a 2:1:1 mixture of soil: sand: peat fertilized with 11-48-0 and were placed in a growthroom. Day/night temperatures were 20 C and 15 C, respectively, and daylength was 16 hours for the duration of the experiment.

For evaluation of macroscopic white rust symptoms, seedlings were transferred to a misting chamber at the four leaf stage and were inoculated after 24 hours at 100% RH. For the microscopic infection study, plants were transferred to the misting chamber when cotyledons reached an average diameter of one cm.

To obtain zoospores for inoculation, approximately 100 mg of A. candida sporangia stored in 00 gelatin capsules at a temperature below -10 C were added to 10 ml of distilled water and centrifuged at 3000 RPM for two minutes. The supernatant was poured off and the pellet of sporangia was resuspended in another 10 ml of distilled water. The procedure was repeated four more times. The final time the supernatant was poured off, the pellet of sporangia was resuspended in five ml of double distilled water in a test tube and placed in an incubator at 12 C for eight hours. Following incubation, the sporangia were resuspended in the double distilled water by gently swirling the test tube. For

macroscopic observations, the incubated sporangia were then added to 150 ml of distilled water and sprayed uniformly onto approximately 50 plants using a plant mister. For the inoculation of cotyledons for macroscopic analysis, incubated sporangia were gently swirled in the test tube and using a 100 ul Eppendorf micropipette the largest droplet size possible was placed on each half cotyledon. Average droplet size was 50 to 70 ul. Seedlings and cotyledons were returned to the growthroom 48 hours after inoculation.

Ten days following inoculation, macroscopic disease symptoms were evaluated on five plants of each cultivar with the exception of C. bursa-pastoris, A. rusticana, and B. nigra where only one, three, and four plants were available, respectively, for the testing of each isolate. The number of pustules and the average size of pustules (in mm) were recorded for the five bottom leaves of each plant.

Microscopic colony development

At three, four, and five days after inoculation, ten to fifteen cotyledons were removed randomly and were fixed and cleared by boiling for three to five minutes in a 1:2 v/v mixture of lactophenol/95% ethanol. Cotyledons were stained with Calcofluor M-1 following the procedure outlined by Rohringer et al. (1977) and mounted in glycerol. The lengths and widths of 25 colonies were recorded at day three and four. At day five, fifty-two colonies were evaluated. A

Zeiss Research Microscope set up for incident-light fluorescence microscopy was equipped with a Mercury high pressure lamp, X16, X25, and X40 Neofluar objectives, and X10 kpl ocular. A number 18 filter set was used for UV-violet excitation which included exciter filter BP 395-425 with maximum transmission between 395 and 425 nm; dichromatic beam splitter FT 425; and barrier filter 450.

Photomicrography

At one, two, five, seven, and nine days after inoculation, ten to fifteen cotyledons were removed randomly and were fixed and cleared in a 1:1:1 mixture of lactophenol:methanol:chloroform. Cotyledons were stained in 0.01 w/v Trypan blue/lactophenol for five hours and mounted in glycerol with a trace of lactophenol as a preservative. Observations were made with a Zeiss Research Microscope equipped with X16 and X40 Neofluar Differential Interference Contrast objectives and X10 kpl ocular.

Statistical analysis

Data were entered into the University of Manitoba Amdahl 5850 computer system using MANTES (Ferch et al., 1978). Statistical analysis was carried out using the SAS package (Ray et al., 1982). Because the data of macroscopic leaf symptoms could not be assumed to be normally distributed, the Kruskal-Wallis test, the non-parametric equivalent of a

one-way analysis of variance, was used for analytical purposes. The lsd multiple comparison procedure was performed on each dependent variable. For the microscopic infection study, an analysis of variance procedure was used for analytical purposes. Paired T-tests were used to test for differences between colony lengths and colony widths on Torch. Comparisons were made between days three and four, days three and five, and days four and five. All statistical tests were performed at an alpha level of 5%.

Results

Inoculation with rapeseed isolate

Pustule size

Kruskal-Wallis tests for average leaf pustule size indicated a significant block by cultivar interaction and highly significant differences in responses of 11 crucifers to infection by A. candida (Appendix table 1). Highly significant differences were also noted between the seven differential test hosts.

A lsd test grouped all B. campestris cultivars, including Torch, Tobin, and Candle, as well as the B. napus cultivar Triumph in the same category (Table 1). There was no significant difference between average pustule sizes in the range of 0.9 to 1.2 mm. Raoula radish and B. oleracea pustules were equal in size with an average diameter of 0.6 mm and were grouped separately from the B. napus and B.

campestris cultivars in the lsd test. B. carinata, B. napus cultivar Regent, and the B. juncea cultivars Pusa Bold, Common Brown Mustard, and Domo were not infected by race seven.

With the inoculation of seven differential test hosts, symptoms were only observed on B. campestris. Average pustule size was 0.6 mm (Table 3).

Number of pustules per leaf

Main effects and interactions in the Kruskal-Wallis tests for average number of pustules/leaf were identical to those for average pustule size (Appendix table 2). The lsd test indicated that there were no significant differences between Torch and Candle with averages of 27 and 23 pustules/leaf, respectively (Table 2). Triumph was significantly less susceptible than Torch or Candle according to the lsd test with an average of 10 pustules/leaf. Tobin, in the third lsd group had an average of six pustules/leaf. Raoula radish and B. oleracea with a very low level of infection, did not have significantly more pustules on each leaf than did the uninfected crucifers.

B. campestris, when inoculated along with six other differential test hosts, had an average of three pustules/leaf (Table 4).

Inoculation with mustard isolate

Pustule size

Highly significant differences between cultivars were found in Kruskal-Wallis tests for the inoculation of 11 cruciferous hosts as well as for the inoculation of seven differential test hosts (Appendix table 1).

Average pustule size on Tobin was 1.0 mm and was significantly different from the average pustule size on the B. juncea cultivars Pusa Bold and Common Brown Mustard with average pustule diameters of 0.5 and 0.4 mm, respectively (Table 1). The remaining cultivars did not exhibit disease symptoms.

In the inoculation of seven differential test hosts, average pustule size was 0.4 mm on the B. juncea cultivar Southern Giant Curled. The other six hosts were not infected.

Number of pustules per leaf

Kruskal-Wallis test main effects and interactions were identical to those for average pustule size (Appendix table 2).

Common Brown Mustard and Pusa Bold with averages of twenty-nine and eight pustules/leaf, respectively, were not significantly different according to the lsd test. Tobin, on which only one pustule was observed was in another lsd group and was not significantly different from the uninfected cultivars.

In the inoculation of the differential test hosts, an average of three pustules were observed on leaves of the B. juncea cultivar Southern Giant Curled. None of the other differential test hosts displayed white rust symptoms.

Microscopic colony development

Analysis of variance indicated that there were highly significant differences between colony length and width in Torch cotyledons at three, four, and five days after inoculation with race seven of A. candida (Appendix tables 38 and 39). Average colony lengths at three, four, and five days after inoculation were 112, 255, and 346 um, respectively (Table 5). At three, four, and five days following inoculation, colony widths were 55, 162, and 202 um, respectively. For both colony length and width, T-tests indicated that comparisons were significant between days three and four, and days three and five, but not between days four and five (Tables 6 and 7).

Microscopic observations

On the B. campestris cultivar Torch, only encysted zoospores were observed at one day following inoculation with race seven of A. candida. Germ tubes were not seen. At two days after inoculation, germ tubes were scarce (Figures 2 and 4). Occasionally encysted zoospores germinated away from the nearest stoma (Figure 4). The germ tubes, however,

then grew around the encysted zoospore and towards the stomatal opening. Five days following inoculation Albugo colonies were quite extensive (Figure 1), and two or three haustoria could be found in pallisade mesophyll cells (Figure 5). By seven days after inoculation, hyphae had penetrated into the spongy mesophyll. Sporulation occurred by nine days following inoculation.

Nothing resembling infection by A. candida was observed on Tobin cotyledons at one or two days following inoculation. By five days following inoculation, however, well developed haustoria were observed (Figures 8, 11, and 12), and the infection on Tobin was indistinguishable from that on Torch. As with Torch, hyphae penetrated into the spongy mesophyll by seven days following inoculation and sporulation occurred at nine days after inoculation. Infection sites were only rarely found on Tobin as compared to infection sites on Torch. Also, infection on Tobin was only observed when zoospores had encysted in the stomatal cavity, below one of the guard cells (Figures 7, 8, 9, and 11).

Due to the difficulty in finding infection sites in Tobin prior to five days following inoculation, the sequence of events from zoospore encystment to formation of the first haustorium could not be ascertained. With the few infection sites observed in Tobin, however, it was apparent that infection only occurred when zoospores encysted within a

stoma, under one of the guard cells (Figures 7, 8, 9, and 11). It is not unlikely that zoospores would have access to the substomatal cavity by way of the stomata as even sporangia, structures substantially larger than zoospores (Figure 10), have been observed in stomatal openings (Figure 3).

Infections on Torch appear to be initiated by means of a germ tube growing through a stoma (Figures 6 and 13). A haustorium soon develops within a palisade mesophyll cell (Figure 14). The colony continues to grow in size (Figure 1) with several haustoria developing in each mesophyll cell (Figure 15).

Discussion

The block by cultivar interaction with respect to size and number of pustules on leaves of 11 cruciferous hosts when inoculated with race seven of A. candida may be accounted for by conditions in the misting chamber. With the placement of the humidifier in the lower level of the misting chamber and two intake fans on the upper level, a film of water tended to accumulate on the flat of plants furthest from the fans. As distance from the fans decreased, the length of time water was retained on the leaves decreased, thus reducing the length of time motile zoospores were exposed to free water.

That the isolates from B. campestris and B. juncea only infected the hosts from which they were obtained would indicate that these isolates are indeed races seven and race two, respectively.

For the inoculation of seven differential test hosts, the pustule size and number on Torch were significantly lower than when 11 cruciferous hosts were inoculated. Unfortunately, a breakdown in growthroom facilities necessitated the transfer of differential test host flats to a lower temperature regime, resulting in a slower fungal growth rate and a lower pustule size and number after ten days of growth.

Epifluorescent equipment, because of the great depth of field afforded, facilitated the simple and accurate measurement of colony length and width (Figure 1). Problems were encountered, however, prior to three days following inoculation. Because structural details of both the fungus and the host epidermis were obscured, it was difficult to determine whether stained structures were actually fungal in nature or were merely densely staining areas in the host epidermal cells.

Differential Interference Contrast (DIC) optics allowed for observations of details of fungal structures such as haustoria (Figures 5, 8, 11, and 12), but depth of field was reduced to the point that the extent of colony development could not easily be determined. DIC microscopy, however, did

allow for observations to be made quite early in the infection process (Figures 2 and 4).

Verma et al. (1975) observed formation of the initial haustorium in B. campestris cultivar Span 48 h following inoculation. Though only germ tubes were noted in this experiment two days after inoculation (Figures 2 and 4), 1 C lower day and night temperatures, shorter day length, and the infection of a less susceptible cultivar (unpublished data) may account for this discrepancy. Verma et al. (1975) also reported signs of a developing pustule at about seven days. Pustule development was commonly observed nine days following inoculation under conditions similar to those reported in this experiment.

Conclusion

Inoculation of seven of eight differential test hosts indicated that the A. candida isolates obtained from B. campestris and B. juncea were races seven and two, respectively. Of eleven cruciferous hosts inoculated with race seven of A. candida, six displayed typical white rust symptoms. B. oleracea and R. sativus cultivar Raoula had the fewest leaf pustules followed by B. campestris cultivar Tobin, B. napus cultivar Triumph, and the two B. campestris cultivars Candle and Torch. R. sativus and B. oleracea had significantly smaller pustules than the other four infected cultivars. The B. juncea cultivars Common Brown and Pusa

Bold did not display significantly different leaf symptoms after inoculation with race two. One race two pustule, approximately the same size as those incited by race seven, was noted on Tobin.

Up to two days following inoculation, only encysted zoospores with germ tubes were observed on the surface of Torch cotyledons. By three days, colonies had developed. Significant increases in colony length and width were noted between days three and four. There were no significant differences in colony size between days four and five. Several haustoria were noted in palisade mesophyll cells by five days. Sporulation occurred nine days following inoculation. Infection sites were only rarely found on Tobin. From five days after inoculation onward, the infection process on Tobin was indistinguishable from that on Torch. Infection on Tobin was only observed where zoospores had encysted in the stomatal cavity, below one of the guard cells.

TABLE 1. Average pustule size and lsd grouping of a series of crucifers inoculated with isolates of *A. candida* obtained from *B. campestris* and *B. juncea*.

Isolate¬	Cultivar	lsd # Grouping	Average * Pustule Size (in mm)
B.campestris	Torch	A	1.2
	Tobin	A	1.1
	Triumph	A	1.1
	Candle	A	0.9
	Raoula Radish	B	0.6
	B. oleracea	B	0.6
	Pusa Bold	C	0.0
	Regent	C	0.0
	B. carinata	C	0.0
	Common Brown Mustard	C	0.0
	Domo	C	0.0
B. juncea	Tobin	A	1.0
	Pusa Bold	B	0.5
	Common Brown Mustard	B	0.4
	Candle	B	0.0
	Domo	B	0.0
	B. oleracea	B	0.0
	Raoula Radish	B	0.0
	Regent	B	0.0
	B. carinata	B	0.0
	Torch	B	0.0
	Triumph	B	0.0

\# lsd grouping based on ranked data, alpha 0.05.
* Averages calculated from untransformed data.

¬ *B. campestris* CV= 15.88%
B. juncea CV= 18.94%

TABLE 2. Average pustule number/leaf and lsd grouping of a series of crucifers inoculated with isolates of A. candida obtained from B. campestris and B. juncea.

Isolate¬	Cultivar	lsd # Grouping	Average * Pustule Number/ Leaf
B.campestris	Torch	A	27
	Candle	A	23
	Triumph	B	10
	Tobin	C	6
	Raoula Radish	D	0
	B. oleracea	D	0
	Pusa Bold	D	0
	Regent	D	0
	B. carinata	D	0
	Common Brown Mustard	D	0
	Domo	D	0
B. juncea	Common Brown Mustard	A	29
	Pusa Bold	A	8
	Tobin	B	0
	Candle	B	0
	Domo	B	0
	B. oleracea	B	0
	Raoula Radish	B	0
	Regent	B	0
	B. carinata	B	0
	Torch	B	0
	Triumph	B	0

\# lsd grouping based on ranked data, alpha 0.05.
* Averages calculated from untransformed data.

¬ B. campestris CV= 15.18%
B. juncea CV= 18.92%

TABLE 3. Average pustule size and lsd grouping of seven differential test hosts inoculated with isolates of *A. candida* obtained from *B. campestris* and *B. juncea*.

Isolate¬	Cultivar	lsd # Grouping	Average * Pustule Size (in mm)
B. campestris	*B. campestris*	A	0.6
	R. sativus	B	0.0
	Capsella bursa-pastoris	B	0.0
	B. juncea	B	0.0
	Rorippa islandica	B	0.0
	Armoracia rusticana	B	0.0
	B. nigra	B	0.0
B. juncea	*B. juncea*	A	0.3
	R. sativus	B	0.0
	Capsella bursa-pastoris	B	0.0
	B. campestris	B	0.0
	Rorippa islandica	B	0.0
	Armoracia rusticana	B	0.0
	B. nigra	B	0.0

\# lsd grouping based on ranked data, alpha 0.05.
* Averages calculated from untransformed data.

¬ *B. campestris* CV= 14.06%
B. juncea CV= 17.23%

TABLE 4. Average pustule number/leaf and lsd grouping of seven differential test hosts inoculated with isolates of A. candida obtained from B. campestris and B. juncea.

Isolate¬	Cultivar	lsd # Grouping	Average * Pustule Number/ Leaf
B. campestris	B. campestris	A	3
	R. sativus	B	0
	Capsella bursa-pastoris	B	0
	B. juncea	B	0
	Rorippa islandica	B	0
	Armoracia rusticana	B	0
	B. nigra	B	0
B. juncea	B. juncea	A	3
	R. sativus	B	0
	Capsella bursa-pastoris	B	0
	B. campestris	B	0
	Rorippa islandica	B	0
	Armoracia rusticana	B	0
	B. nigra	B	0

\# lsd grouping based on ranked data, alpha 0.05.
* Averages calculated from untransformed data.

¬ B. campestris CV= 14.06%
B. juncea CV= 17.23%

TABLE 5. Average length and width (in um) of race seven colonies of A. candida on Torch cotyledons at three, four, and five days after inoculation.

Day	Length	Width
3	112	56
4	256	162
5	346	203

TABLE 6. T-tests for colony length (in um) of race seven of *A. candida* on Torch cotyledons at three, four, and five days after inoculation.

Day comparison	Lower confidence limit	Difference between means	Upper confidence limit
3 to 4	27	143	259 *
3 to 5	135	234	332 *
4 to 5	-8	90	189

* Comparisons significant at the 0.05 level

TABLE 7. T-tests for colony width (in um) of race seven of A. candida on Torch cotyledons at three, four, and five days after inoculation.

Day comparison	Lower confidence limit	Difference between means	Upper confidence limit
3 to 4	39	106	173 *
3 to 5	90	147	203 *
4 to 5	-15	41	97

* Comparisons significant at the 0.05 level

FIGURES 1-12. Development of A. candida in cotyledons of Torch and Tobin.

Fig. 1. Hyphae in Torch cotyledons growing around palisade mesophyll cells 5 days after inoculation (epifluorescent). x240.

Fig. 2. Torch 2 days. x380. Germ tube arrowed.

Fig. 3. Sporangium in stoma. x380.

Fig. 4. Torch 2 days. x380. Note germ tube growing around towards stoma (arrowed).

Fig. 5. Torch 5 days. x380. Haustorium arrowed.

Fig. 6. Torch 5 days. x150. Note germ tube growing from encysted zoospore (arrowed) and penetration of host through stoma.

Fig. 7. Fungal colony under epidermis of Tobin 5 days after inoculation. x380.

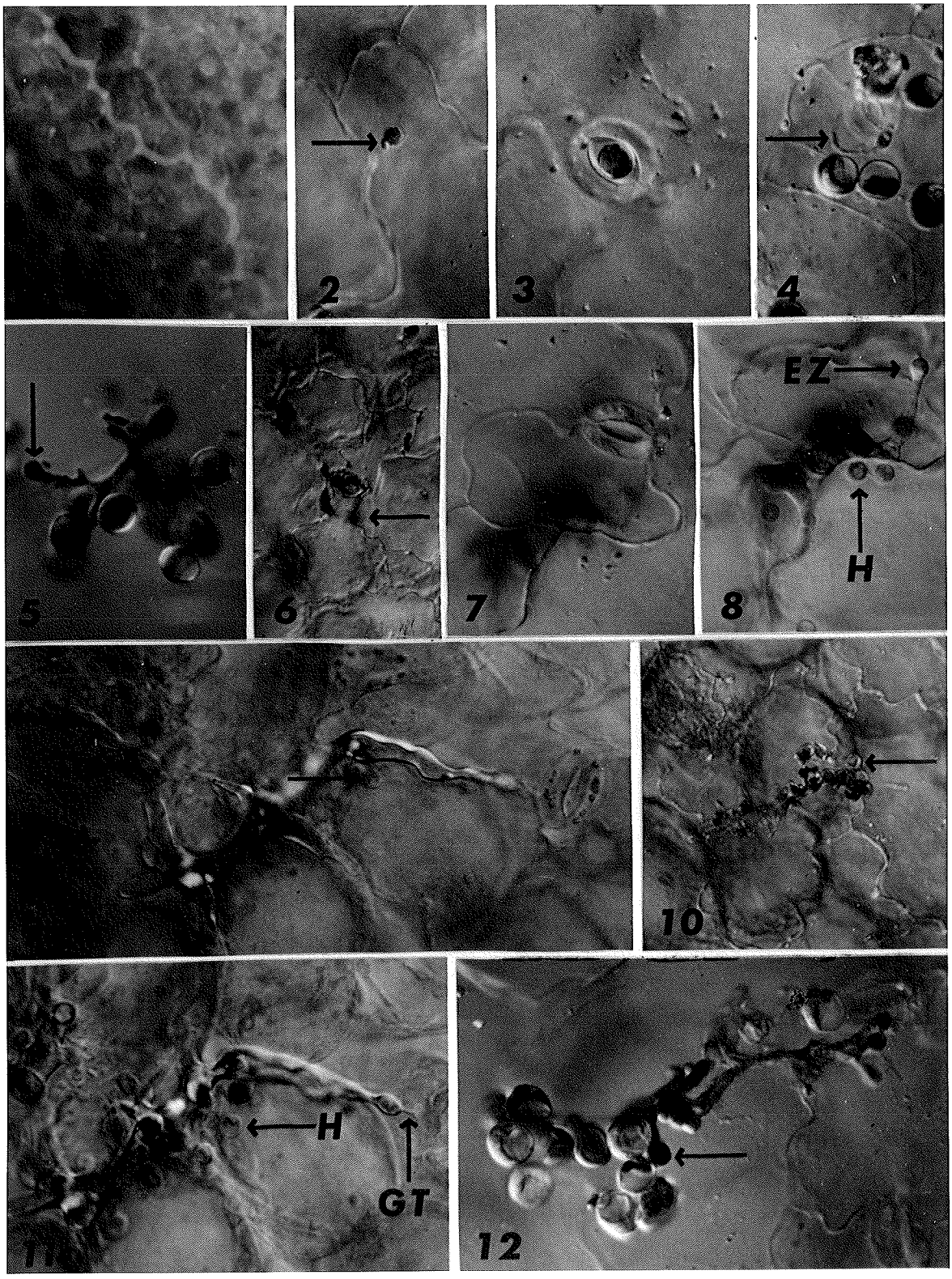
Fig. 8. Same colony as figure 7. x380. Note encysted zoospore (EZ) in stoma and haustorium (H).

Fig. 9. Tobin 7 days. x380. Haustorium arrowed.

Fig. 10. Tobin 5 days. x150. Sporangium arrowed.

Fig. 11. Same colony as figure 9. x380. Note germ tube (GT) in vicinity of stoma and haustorium (H).

Fig. 12. Same colony as figure 10. x380. Haustorium arrowed.


2
3
4
5
6
7
EZ
H
8
10
H
GT
11
12

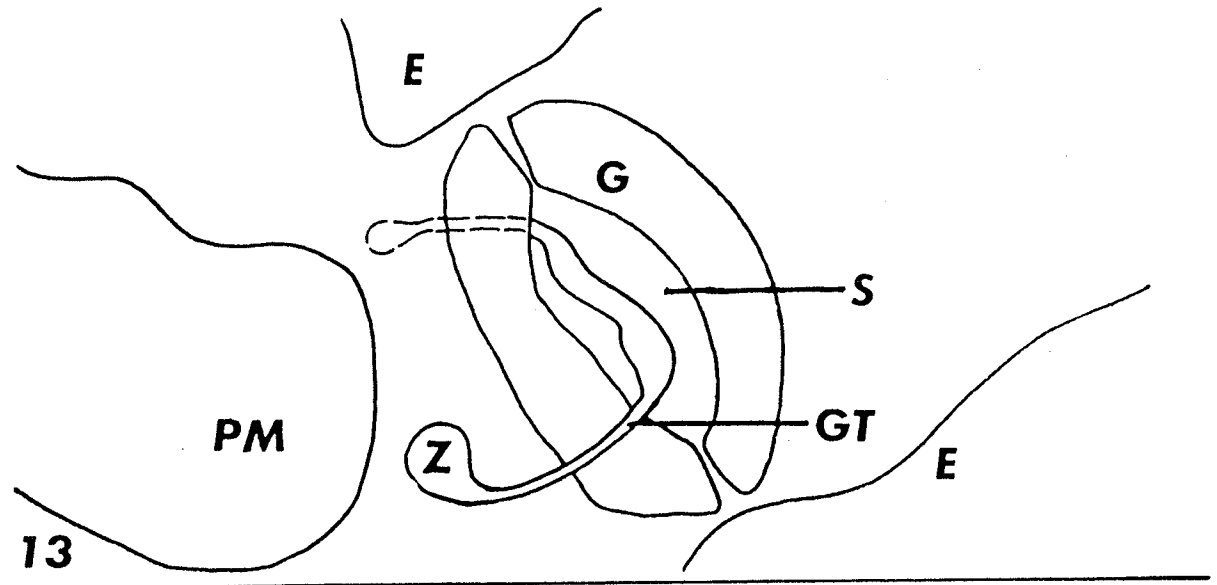
FIGURES 13-15. Development of A. candida in cotyledons of Torch.

Fig. 13. Germ tube (GT) elongation and penetration of stoma (S).

E=Epidermis, G=Guard cell, PM=Palisade mesophyll, Z=Encysted zoospore.

Fig. 14. Initiation of first haustorium (H).

Fig. 15. Colony development with several haustoria in palisade mesophyll cells.



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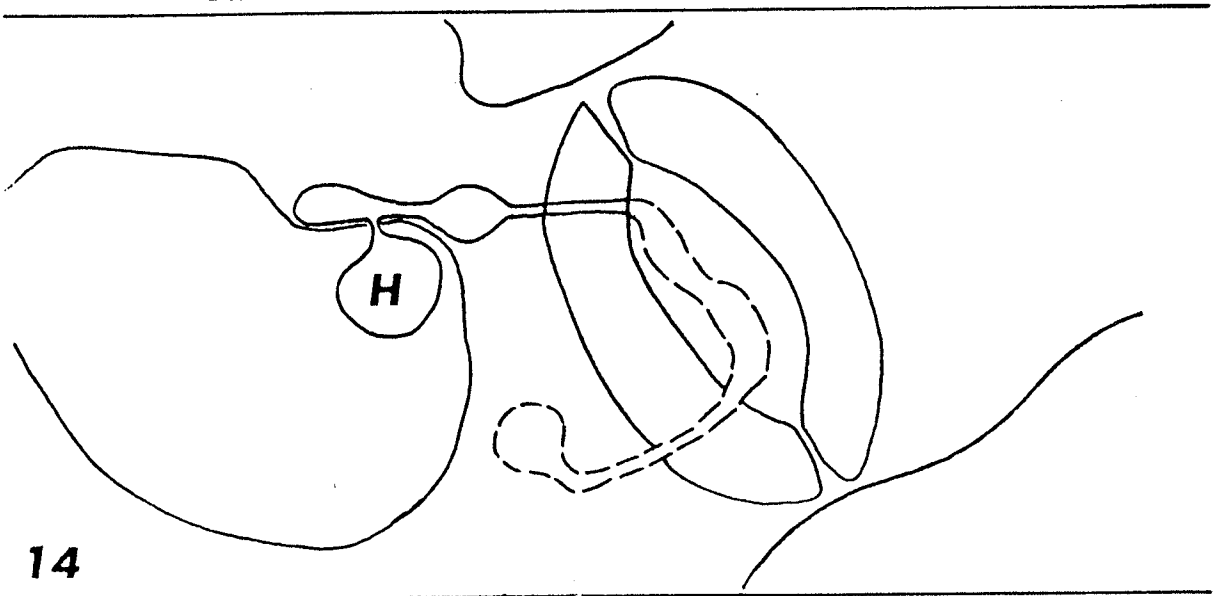

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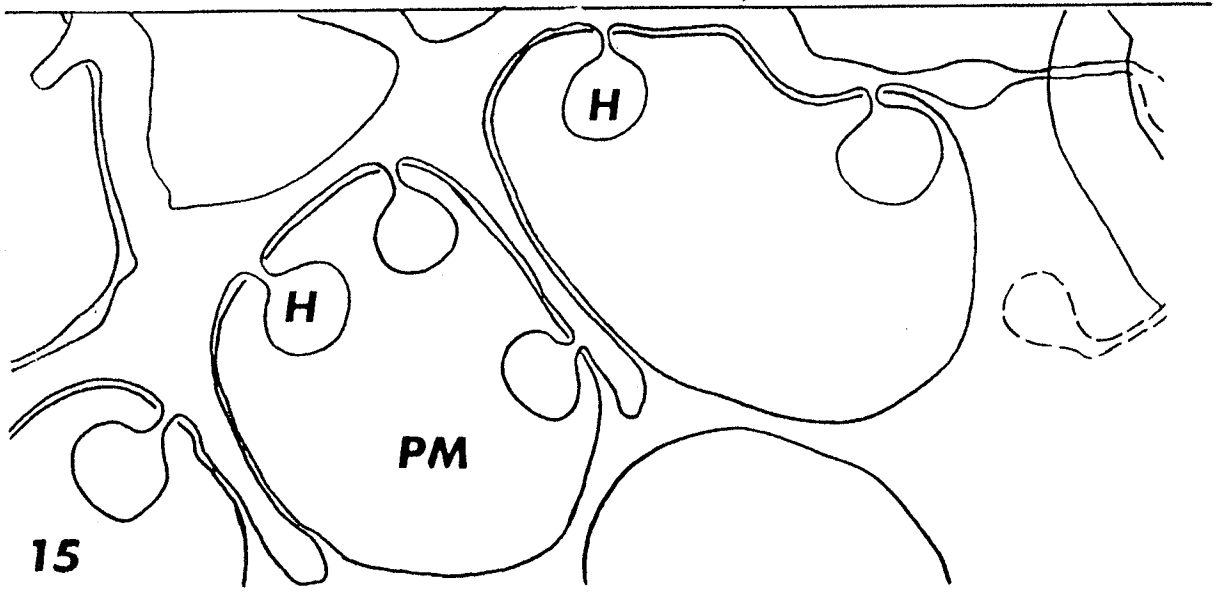

15

EFFECT OF WHITE RUST INFECTION ON YIELD OF TURNIP RAPE

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Abstract

The development of symptoms incited by *Albugo candida* on *Brassica campestris* and the effect of infection on yield was examined at two locations in 1982 and at one location in 1983. Foliar infection on the first and second leaves followed similar developmental patterns but comparisons could not be made between sites or between years. Staghead and partial staghead developmental patterns were unique at each location and in each year. Conditions in 1983 encouraged the development of partial stagheads rather than stagheads. The reverse was true in 1982. The application of various fungicide treatments resulted in significantly different levels of white rust infection in *B. campestris* plots. Disease incidence and severity, however, were not sufficient to result in detectable yield losses.

Introduction

Harper and Pittman (1974) claimed a relationship between yield loss and systemic infection of Brassica campestris cultivar Span by Albugo candida. The equation

$$\text{Yield loss (\%)} = 100 \times \frac{\text{number of stems systemically infected}}{\text{total number of stems examined}}$$

was suggested for use in disease loss assessment. Plants had been divided up by disease category and yield was evaluated for each category. The occurrence of "systemically infected, discrete pods" was ignored (Harper and Pittman, 1974).

In the field, effective control of foliar infection as well as increased yield was reported with Zineb (zinc activated polyethylene thiram disulfide) on B. campestris cultivar Sarson (Perwaiz et al., 1969). Staghead incidence was significantly reduced in B. campestris cultivar Torch with three foliar applications of the fungicide CGA 48988 (Metalaxyl) [methyl N-(2,6-dimethylphenyl)-N-methoxyacetyl-2-alaninate] (Altman and Campbell, 1977). Seed treatment proved ineffective for white rust control. Foliar infection of B. campestris cultivar Torch was reduced with two applications of three protectant fungicides: Chlorothalonil, Mancozeb ([ethylene-bis(dithiocarbamate)]manganese), and DPX 164 (MBC 10% + Maneb 64%) (Dueck and Stone, 1979). Staghead formation, however, was not inhibited. The acylalanine

fungicide CGA 29212 [methyl-N-(2,6-dimethylphenyl)-N-chloroacetyl-2-alaninate] significantly decreased staghead incidence.

In the greenhouse, foliar infection was controlled on B. campestris cultivar Torch with applications of Chlorothalonil or Mancozeb prior to inoculation and at seven day intervals (Verma and Petrie, 1975b). CGA 29212 and CGA 48988 proved to be excellent eradicants of foliar infection of Torch caused by A. candida in the growthroom (Dueck and Stone, 1979).

The objective of this study was to evaluate the yield loss-plant disease relationship at the plot level and to allow for an interpretation of the interactions between diseased and non-diseased plants within a plot. Systemic and non-systemic fungicides applied at various intervals as well as varieties which were not equally susceptible to the white rust fungus were used to provide a range of disease levels between plots.

Materials and Methods

Field studies were conducted at Portage la Prairie, Manitoba and the University of Manitoba Arboretum in 1982 but only at the Arboretum in 1983. The Arboretum experiments were carried out on a Fort Garry clay with poor subsurface drainage. Experiments at Portage la Prairie were conducted on a Gnadenthal loam soil. Arboretum plot areas

were seeded to two lines of B. campestris and to the B. juncea cultivars Domo and Burgonde-A in 1981 and 1982, respectively. The Portage la Prairie experimental site was seeded to B. napus cultivar Regent in 1981. The Arboretum was fertilized with 16-20-0 at a rate of 100 Kg actual nitrogen/ha. In the fall, Trifluralin (Treflan) granular herbicide was applied at a rate of 28 kg/ha and was incorporated. Liquid Trifluralin was spring applied at the Portage la Prairie site at 1.5 L ai/ha in 100 L water, incorporated twice at right angles with a tandem disc, harrowed, and packed.

Seeding in 1982 was on June 2 and June 17 (day of the year 153 and 168) at the Arboretum and Portage la Prairie, respectively. The Arboretum was seeded on May 25 (day 145) in 1983. A four row belt seeder and packer were used to seed to a depth of 2.5 to 4.0 cm. Plot size at the Arboretum was 3.7 x 6.1 m and 3.7 x 4.6 m at Portage la Prairie resulting in seeding rates of five kg/ha and seven kg/ha, respectively. Carbofuran (Furadan 10G) insecticide was applied with the seed at 2.8 kg/ha for flea beetle control. At the 1982 Arboretum location, malathion was applied at rapeseed growth stage 2.1 at 2.2 L/ha for additional flea beetle control. The herbicide 3,6-dichloropicolinic acid (Lontrel) was applied on day 178 (gs 4.1), 1983 in the Arboretum at 850 ml product/ha for thistle control.

In 1982, each block included the B. campestris cultivars Span and Torch. Only Torch was seeded in 1983. Each plot contained 12 rows, the outer two rows on each side being seeded to B. juncea cultivar Domo.

The experiment was set up as a randomised complete block design (RCBD) with six blocks and twelve blocks in 1982 and 1983, respectively. In 1982, all blocks had three of five treatments in common: in two plots, no attempt was made to control white rust and in the other plot Metalaxyl (Ridomil) was applied as a seed treatment at 30 g ai/100 kg seed. In addition to the common treatments, the 1982 Arboretum location included two plots where Metalaxyl was applied to the foliage at 0.25 g ai/ha in 170 L water (Metalaxyl foliar treatment). One plot was sprayed with Metalaxyl on day 196 and 207 when the plants were in growth stages (gs) 4.2 and 5.2, respectively; the other on day 200 (gs 4.2) and 207. Growth stages were evaluated based on Harper and Berkenkamp's (1975) revised growth stage key for B. campestris and B. napus. Blocks at Portage la Prairie included the three common treatments, one Metalaxyl foliar treatment (Arboretum rate and dates of application) and one plot where Captafol (Difolatan) was applied to the foliage at 1.14 L ai/ha in 170 L water on day 196 (growth stage 2.4) followed by Metalaxyl at 0.25 g ai/ha in 170 L water on day 207 (growth stage 4.3) (Captafol/Metalaxyl treatment). In 1983, each block included one control treatment where

white rust was permitted to develop naturally and one Metalaxyl seed treatment, as in 1982, and two plots where Metalaxyl was applied at a rate of 0.25 kg ai/ha in 170 L water. One Metalaxyl foliar treatment was applied on day 179 (gs 4.1) and the other on days 169 (gs 2.4), 179 (gs 4.1), and 191 (gs 4.4).

White rust symptoms were evaluated periodically throughout each growing season. Disease severity, or percent leaf area infected with white rust pustules was evaluated on the first and second non-senescent leaves of 10 randomly selected plants from the middle four rows of each plot. For analytical purposes leaf area ratings were expressed as average percent leaf area infected per plot. Staghead incidence and partial staghead incidence were also evaluated for each plot.

A staghead was considered formed when 50% or more of an inflorescence had been replaced with hypertrophied tissue. A partial staghead was considered as one which less than 50% of the inflorescence was hypertrophied.

Because root rot infection could possibly have confounded the analysis of yield at the 1983 Arboretum location, root rot was rated in each plot. Ten plants were rated per plot and the variable was expressed as average root rot severity per plot.

At maturity, the centre three meters of the middle four rows of each plot in the Arboretum and the middle two rows

of each plot at Portage la Prairie were hand harvested when 60 to 75% of the seed in the pods had turned from green to brown. Plants were cut just above ground level, placed in burlap sacks, and allowed to air dry for one to two weeks before threshing. Seed yields were determined and for analytical purposes were expressed as grams of seed per plot.

Data were entered into the University of Manitoba Amdahl 5850 computer system using MANTES (Ferch et al., 1978). Statistical analyses were carried out using the SAS package (Ray et al., 1982). Because population distributions could not be assumed normal, the Kruskal-Wallis test, the non-parametric equivalent of a one-way analysis of variance, was used for analytical purposes. The lsd multiple comparison procedure was performed on each independent variable in which treatments were significant. Where dependent variables were rated more than once, data were pooled to permit an analysis of the independent variable, date. These variables were then sorted by date and the Kruskal-Wallis test was performed on each dependent variable at each date. In 1982, block (B), variety (V), treatment (T), and date (D) main effects and V*T, V*D, T*D, B*V, and V*T*D interactions were analysed for each dependent variable. The main effects and interactions in the model in 1983 included block, treatment, and date , and T*D, and B*T, respectively. All statistical tests were performed using alpha level of 5%.

Results

Pooled data of disease parameters did not exhibit significant block effects at the Portage la Prairie site. Significant staghead block effects were observed, however, at the second and third rating dates when first leaf foliar infection was analysed using the Kruskal-Wallis test (Kruskal-Wallis tests in appendix tables 3 to 18). Block effects were significant at the Arboretum in 1982 for pooled data of foliar infection on the first and second leaves and for staghead incidence. At the 1983 Arboretum location, block effects for pooled data were highly significant for first and second leaf foliar infection. With respect to yield, significant block effects were apparent in both 1982 and 1983 in the Arboretum.

A highly significant V*T interaction was observed for pooled data in the Arboretum in 1982 with respect to the dependent variable stagheads. A significant B*V interaction for pooled data was noted for partial stagheads at the same location.

Development of white rust

Pooling the data for each location allowed an analysis of the development of white rust disease and of the relationship between the various disease parameters. In 1983, white rust foliar symptoms were detected initially on day 178 (Figure 1). Disease severity increased more rapidly

and to a greater degree on the first than on the second leaf. A peak was observed on day 193. Disease severity was first rated on day 200 in 1982 at which time foliar severity was decreasing. White rust severity was always lower on the second than on the first leaf.

In 1983, stagheads were detected four days after the foliar severity of white rust peaked. Though staghead production was initiated at a later date at Portage la Prairie, staghead incidence reached a plateau at approximately the same level as in the Arboretum in 1983. Staghead production in the Arboretum in 1982 did not reach a plateau though the rate of increase of stagheads decreased substantially between day 209 and 213.

Partial stagheads were detected and reached a plateau at the same dates as stagheads at the 1983 Arboretum site. In 1982, partial stagheads were detected quite late in the season. Only at the Arboretum in 1982 did the maximum staghead incidence surpass the maximum partial staghead incidence.

Foliar infection of the first leaf

At Portage la Prairie, highly significant date and significant treatment effects were observed. Average infection per plot was declining in all treatments (Figure 2). The control had significantly different disease levels as opposed to the seed treatment and the Captafol/Metalaxyl

treatment (Table 1). Foliar application of Metalaxyl alone resulted in infection levels which were not significantly different from either of the lsd groups. No treatment effects were significant in the Arboretum in 1982.

Highly significant date and treatment effects as well as a highly significant T*D interaction were observed in the Arboretum in 1983. Foliar infection was noted on the first leaf by day 187 in the Metalaxyl seed treatment, the diseased control, and in the treatment where Metalaxyl was applied once (Figure 2). Pustules were not observed until day 193 where Metalaxyl was applied three times. In all plots except the triple application of Metalaxyl, the average percent leaf area infected peaked at day 193 and then dropped off.

The T*D interaction may be accounted for by the three patterns of white rust development apparent on the first leaf (Figure 2). Control and seed treatment plots had similar slopes between day 187 and 193, and between day 193 and 197. Patterns of pustule development where Metalaxyl was applied once and three times were unique.

Foliar infection of the second leaf

With the exception of a significant date effect and a V*D interaction at the 1982 Arboretum location and the treatment effect at Portage la Prairie being highly significant rather than significant, the second leaf responded the same as the

first to white rust infection with respect to main effects and interactions in the Kruskal-Wallis test.

For the Arboretum in 1983, disease curves for the second leaf followed similar patterns to those for the first leaf except that between day 193 and 197, the average percent leaf area infected declined more rapidly in the seed treatment than in the control plots (Figure 3). Also, a peak which did not occur in 1982 for average first leaf area infected was noted at day 193.

Significantly different disease levels occurred on the second leaf at Portage la Prairie. The Captafol/Metalaxyl treatment had significantly lower levels of disease than the other three treatments (Table 1). At the 1983 Arboretum site a highly significant T*D interaction was observed. The highest disease levels were found in the control plot and in the seed treatment, followed by one application of Metalaxyl and three applications of Metalaxyl.

Staghead infection

At Portage la Prairie, the Kruskal-Wallis test indicated a highly significant T*D interaction. Stagheads were first recorded on day 203 and increased at about the same rate between day 203 and 211 (Figure 4). Between day 211 and 217, stagheads increased less rapidly in the Captafol/Metalaxyl treatment than in the other three treatments and reached a final average incidence of approximately one staghead per

plot, less than one-half the level of the other treatments. A Kruskal-Wallis test by date indicated that treatments were not significantly different until the last rating date, day 217.

Treatments were not significant at the Arboretum in 1982 or in 1983. All treatments in 1983 supported an average of less than one staghead per plot (Figure 4). More stagheads were produced in the Metalaxyl foliar treatment in 1982 than in the control or in the Metalaxyl seed treatment, however, these differences were not statistically significant.

Partial staghead infection

Partial stagheads were not detected in Portage la Prairie until day 217 (Figure 5). The lsd test indicated that the seed treatment, Metalaxyl foliar treatment, and the control were significantly different from the Captafol/Metalaxyl treatment (Table 1). Partial stagheads did not occur in Captafol/Metalaxyl treated plots. There were no significant differences between treatments in the Arboretum in 1982.

At the 1983 Arboretum site, partial stagheads were detected on day 197 (Figure 5). The T*D interaction may be explained in that the rate of increase of partial stagheads was less rapid in plots where Metalaxyl was applied one and three times than in the seed treatment and control plots. The incidence of partial stagheads did not increase between day 201 and 213.

Yield of turnip rape

There were no significant differences in the yield response of the cultivars Torch and Span in 1982. Though the various treatments incited significantly different disease levels, there was no significant effect on yield in either 1982 or 1983. Interactions were insignificant in 1982.

Root rot infection

Though root rot was moderate to severe throughout most of the experimental area in 1983, there were no significant block or treatment effects.

Discussion

At the Arboretum site, block effects may be attributable to fertility gradients. In 1980, the northern portion of the experimental area was left fallow. Mustard plants in this area in 1981 were 30 to 40 cm taller than adjacent plants seeded on stubble.

The proximity of the 1983 Arboretum experiment to a *Rhizoctonia* root rot nursery could have resulted in a gradient across the yield trial, and a significant block effect would have been observed in the Kruskal-Wallis test. A treatment effect with respect to root rot rating would have indicated that disease incited by *A. candida* was not solely responsible for the variability in yield.

The lower severity of white rust infection on the first as opposed to the second leaves of turnip rape may be accounted for by the time of exposure to the initial inoculum source and leaf height within the crop canopy. A. candida could have established itself and proliferated within the host tissue of the first leaf before the second leaf emerged and expanded. Also, the second leaf, being higher in the crop canopy than the first leaf would have been exposed to free moisture for shorter periods of time.

The senescence of lower leaves may in part contribute to the peak and decline in pustule area. This would have had the effect of reducing the density of the crop canopy, thus decreasing the extent of microclimate modification.

The years 1982 and 1983 were examples of both optimal and sub-optimal conditions for white rust disease development,respectively. In 1982, precipitation during the period of disease development was higher than average with near normal temperatures whereas precipitation was rare and daily temperatures were well above normal in this same period in 1983. Plateaus in staghead and partial staghead incidence may be attributed to the absence of the initiation of new infection sites in the inflorescence or to the lack of substantial systemic development of the white rust fungus within host tissue.

The occurrence of partial stagheads in both years suggested that the Harper and Pittman (1974) equation for

yield loss assessment may not be completely valid. Partial stagheads represented a considerable proportion of the total number of floral infections in 1982 and accounted for most of the observed hypertrophy of floral tissue in 1983. Rating yield loss at 100% for systemically infected stems may tend to overestimate disease losses due to A. candida as partial stagheads are found on stems on which at least half of the pods have produced seed. Though the definition of partial stagheads would include hypertrophy of discrete pods, these symptoms were more typical of floral infection of B. juncea and were less commonly observed on B. campestris (unpublished data).

Though some fungicide treatments were ineffective for the control of either foliar or floral infection of turnip rape by A. candida, significantly different levels of white rust were observed in both years. The various levels of white rust observed, however, were not reflected in the treatment yields. Low incidence of floral symptoms may have contributed to this lack of correlation.

Conclusion

Significantly different levels of foliar and floral white rust infection were observed in B. campestris when various fungicide treatments were applied. On an experimental plot scale, however, disease incidence and severity incited by A. candida were not sufficient to result in detectable yield

losses. Metalaxyl and Captafol did control disease, however.

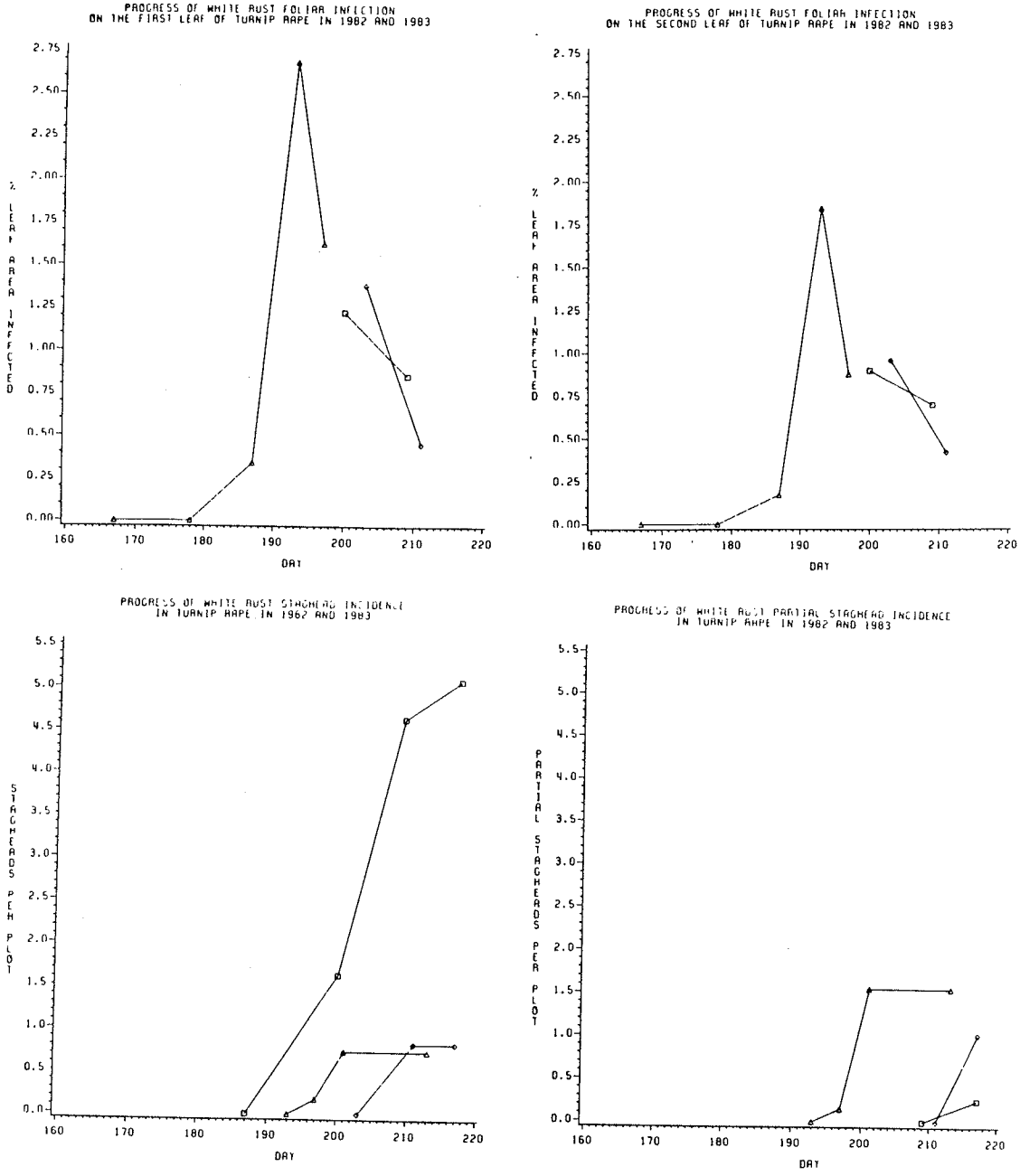
TABLE 1. Response of various white rust disease parameters to fungicide treatment of turnip rape in the 1982 Portage la Prairie yield experiment.

Variable	Treatment	lsd ¬ Grouping		Average * Infection
First Leaf#	Diseased control	A		1.2
	Metalaxyl foliar	A	B	0.9
	Metalaxyl seed treatment		B	0.8
	Captafol/Metalaxyl		B	0.5
Second Leaf&	Diseased control	A		0.9
	Metalaxyl seed treatment	A		0.7
	Metalaxyl Foliar	A		0.7
	Captafol/Metalaxyl		B	0.3
Partial Stagheads$	Metalaxyl seed treatment	A		2
	Metalaxyl foliar	A		2
	Diseased control	A		1
	Captafol/Metalaxyl		B	0

¬ lsd grouping based on ranked data, alpha=0.05.
* Averages calculated from untransformed data.
* First and second leaf variables expressed as average % leaf area infected/plot.
* Partial stagheads expressed as average number/plot.

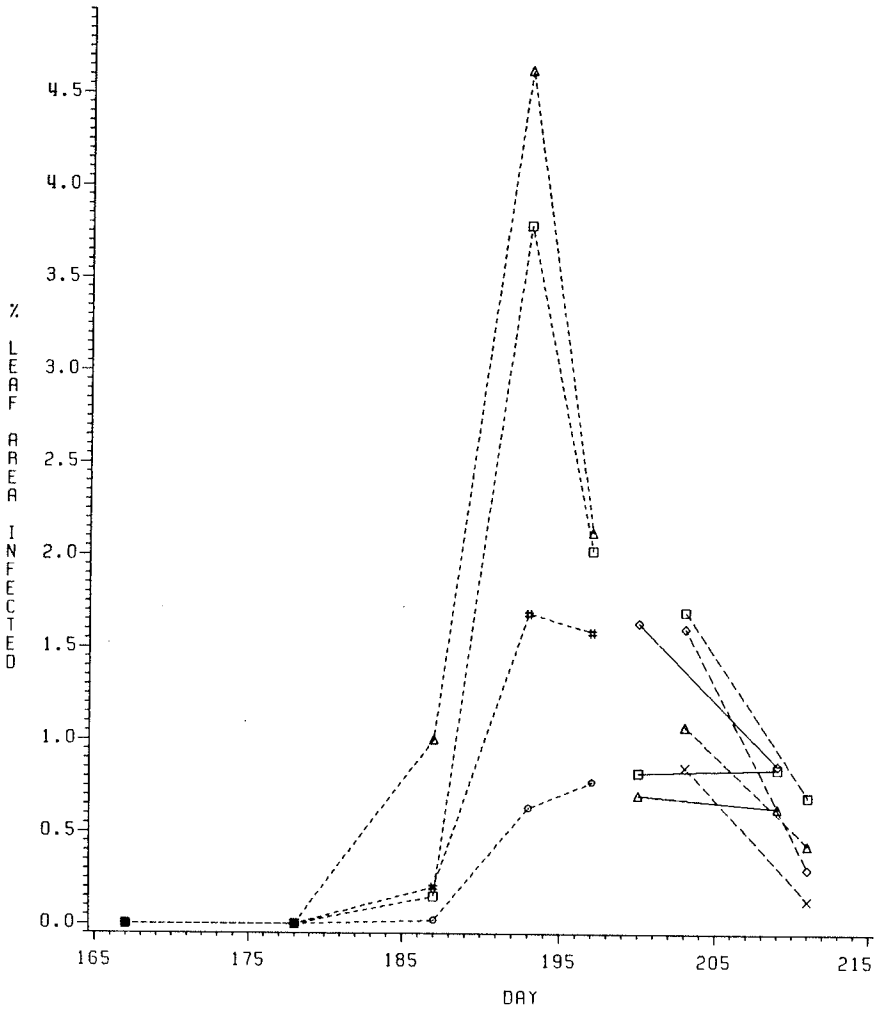
CV= 47.49%
& CV= 50.26%
$ CV= 45.64%

FIGURE 1. Development of white rust infection in 1982 and 1983.



U. of M. Arboretum 1982
U. of M. field station, Portage la Prairie 1982
U. of M. Arboretum 1983

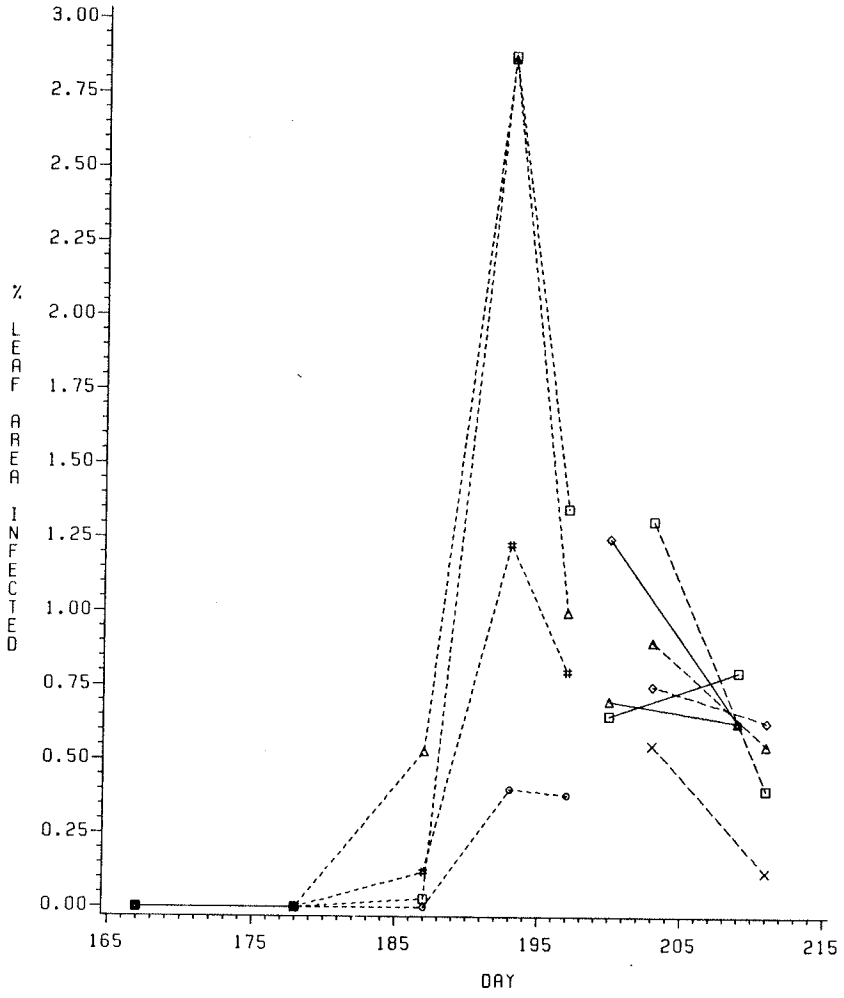
FIGURE 2. Effect of fungicide treatment on the development of white rust infection on the first leaf of turnip rape in 1982 and 1983.



Diseased control (A 1982)
Diseased control (P 1982)
Diseased control (A 1983)
Metalaxyl (0.25 kg ai/ha) every 20 days (A 1983)
Metalaxyl (0.25 kg ai/ha) every 10 days (A 1983)
Captafol (1.14 L ai/ha)/Metalaxyl (P 1982)
Metalaxyl (0.25 g ai/ha) twice (A 1982)
Metalaxyl (0.25 g ai/ha) twice (P 1982)
Metalaxyl seed treatment (A 1982)
Metalaxyl seed treatment (P 1982)
Metalaxyl seed treatment (A 1983)

A = Arboretum
P = Portage la Prairie

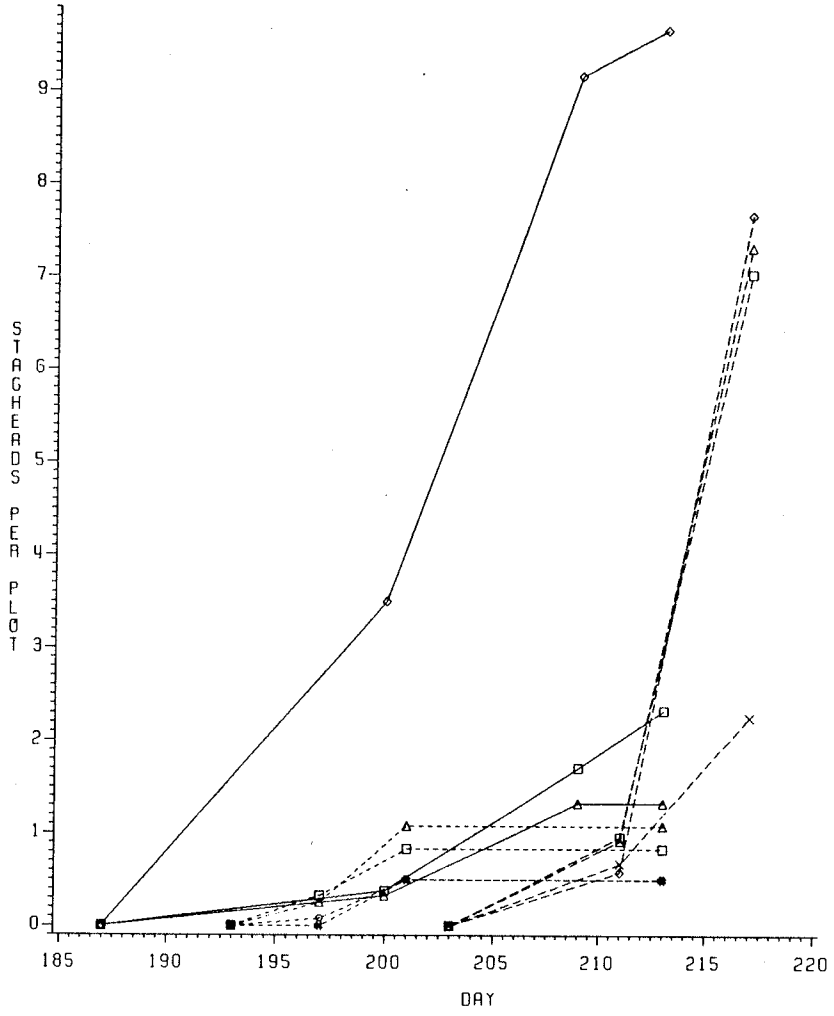
FIGURE 3. Effect of fungicide treatment on the development of white rust infection on the second leaf of turnip rape in 1982 and 1983.



Diseased control (A 1982)
Diseased control (P 1982)
Diseased control (A 1983)
Metalaxyl (0.25 kg ai/ha) every 20 days (A 1983)
Metalaxyl (0.25 kg ai/ha) every 10 days (A 1983)
Captafol (1.14 L ai/ha)/Metalaxyl (P 1982)
Metalaxyl (0.25 g ai/ha) twice (A 1982)
Metalaxyl (0.25 g ai/ha) twice (P 1982)
Metalaxyl seed treatment (A 1982)
Metalaxyl seed treatment (P 1982)
Metalaxyl seed treatment (A 1983)

A = Arboretum
P = Portage la Prairie

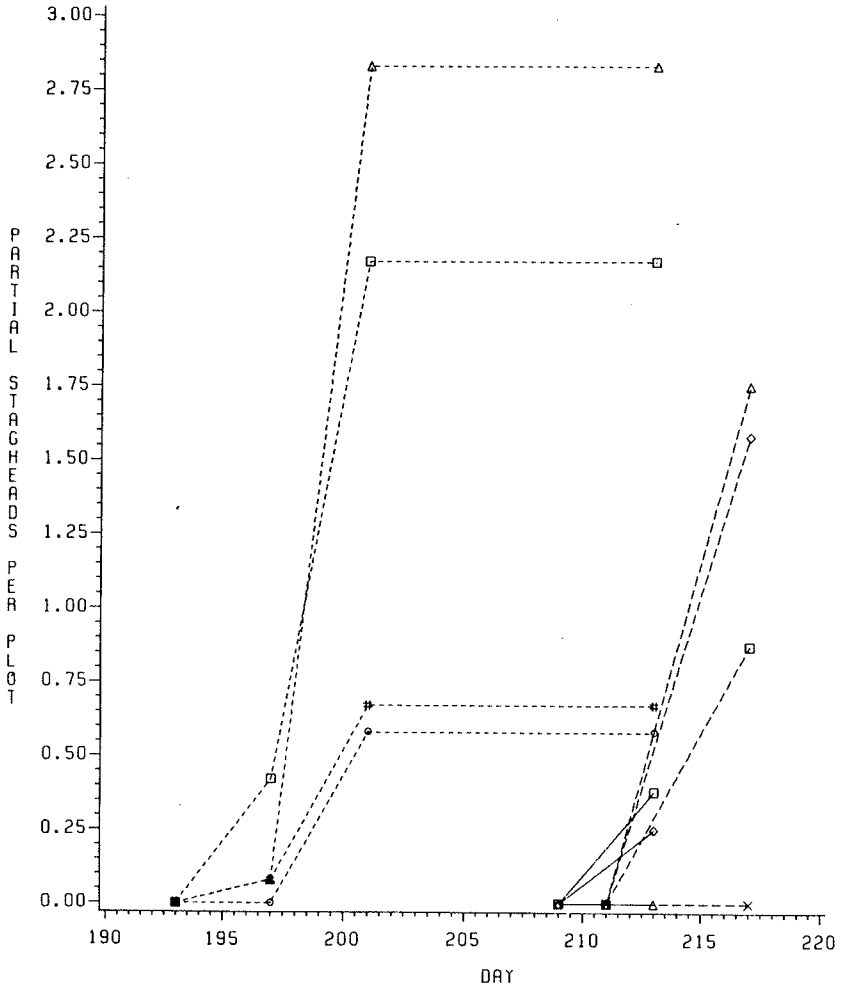
FIGURE 4. Effect of fungicide treatment on staghead incidence in turnip rape in 1982 and 1983.



Diseased control (A 1982)
Diseased control (P 1982)
Diseased control (A 1983)
Metalaxyl (0.25 kg ai/ha) every 20 days (A 1983)
Metalaxyl (0.25 kg ai/ha) every 10 days (A 1983)
Captafol (1.14 L ai/ha)/Metalaxyl (P 1982)
Metalaxyl (0.25 g ai/ha) twice (A 1982)
Metalaxyl (0.25 g ai/ha) twice (P 1982)
Metalaxyl seed treatment (A 1982)
Metalaxyl seed treatment (P 1982)
Metalaxyl seed treatment (A 1983)

A = Arboretum
P = Portage la Prairie

FIGURE 5. Effect of fungicide treatment on partial staghead incidence in turnip rape in 1982 and 1983.



Diseased control (A 1982)
Diseased control (P 1982)
Diseased control (A 1983)
Metalaxyl (0.25 kg ai/ha) every 20 days (A 1983)
Metalaxyl (0.25 kg ai/ha) every 10 days (A 1983)
Captafol (1.14 L ai/ha)/Metalaxyl (P 1982)
Metalaxyl (0.25 g ai/ha) twice (A 1982)
Metalaxyl (0.25 g ai/ha) twice (P 1982)
Metalaxyl seed treatment (A 1982)
Metalaxyl seed treatment (P 1982)
Metalaxyl seed treatment (A 1983)

A = Arboretum
P = Portage la Prairie

EFFECT OF TURNIP RAPE MIXTURES ON WHITE RUST DEVELOPMENT

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Abstract

In 1982 and 1983, the development of disease symptoms incited by Albugo candida was examined in various mixtures of two Brassica campestris cultivars: Torch (susceptible to race seven) and Tobin (moderately resistant to race seven). Torch and Tobin were mixed by seed weight in four ratios: 1:0, 3:1, 1:1, and 1:3, representing 100%, 75%, 50%, and 25% susceptible plant tissue per treatment, respectively. Early in the season, there were no significant differences in disease levels between treatments. In the treatment containing 25% susceptible plant tissue, foliar and floral symptoms lagged behind those in the 100% susceptible treatment by six and four days, respectively, in 1983 though not in 1982. Pathogen dissemination appeared to be slower in the mixtures than in the susceptible stand. There was a negative correlation between the percentage of Tobin in each mixture and disease levels observed at the end of each season. Despite significantly lower disease levels in the 25% susceptible treatment than in the 100% susceptible treatment, no significant differences in yield were observed.

Introduction

Consideration has been given to cultivar mixtures as a means of controlling plant disease epidemics. Mixtures would be most useful in situations where newly introduced, resistant cultivars were lower yielding than older, susceptible cultivars. Through field experimentation it would be possible to determine the proportion of resistant to susceptible plants in a crop stand which would optimize yield. Factors such as the effect of disease on yield loss and the severity of the disease in any given season, as well as the difference in yield between the resistant and the susceptible cultivars would play a role in determining the optimum proportion of resistant and susceptible plants.

The effect of cultivar mixtures on epidemic progress has been outlined by Wolfe and Barrett (1980). Mixtures had little advantage at the beginning of the season when inoculum was predominantly from external sources. When disease increase became dependent upon inoculum buildup within the crop, pathogen dissemination was slower in mixtures than in the susceptible stands. Towards the end of the season, the progress of the epidemic seemed to accelerate in the mixtures because the infection, still increasing in the healthier mixtures, had reached a saturation level and had stopped developing in the pure stands.

During an epidemic, the average estimated incidence of Cercospora apii on a tolerant celery cultivar lagged behind that on the susceptible cultivar by about three weeks (Berger, 1973). Following the initial infection, there was little difference between the cultivars in calculated periodic infection rates. Later, a major flush of new foliage in the tolerant cultivar had the effect of diluting the increase in the amount of infected foliage. That the susceptible cultivar did not display such a flush was reflected in its higher infection rate for that period. The protective effect of tolerant plants in the mixed stand was observed as long as the disease incidence remained below 25%. There was a positive relationship between the percentage of tolerant plants in the population and the protective effect afforded by these plants.

The development of Erysiphe polygoni was evaluated on several mixtures of Ne Plus Ultra (NPU), a highly susceptible swede cultivar (B. napus), and Ruta Otofte (RO), a partially resistant cultivar (Stitch and Wittington, 1983). Early in the season, population composition significantly affected infection rate; disease on NPU increased less rapidly as the percentage of RO was raised. Soon, early disease control was lost with NPU ultimately supporting similar disease levels in all treatments. Averaged over all assessment dates, though, an increasing proportion of RO was correlated with decreasing disease on

the NPU component of each mixture. Whereas consistent yield deviations from expected values were not exhibited in RO, NPU outyielded expected values in mixtures by seven to 21%, conceivably reflecting the disease control offered by cultivar mixtures.

A newly introduced cultivar of B. campestris, Tobin, has moderate resistance to Albugo candida but is lower yielding than two susceptible cultivars, Torch and Candle. The purpose of this experiment was to examine the effect of various mixtures of Torch and Tobin on the development of white rust incited by A. candida. The effect of cultivar mixtures on yield of B. campestris was also to be investigated.

Materials and Methods

Field studies were conducted at Portage la Prairie, Manitoba in 1982 and at the University of Manitoba Arboretum in 1983. The Arboretum experiments were carried out on a Fort Garry clay with poor subsurface drainage. Experiments at Portage la Prairie were conducted on a Gnadenthal loam soil. In 1981, the Portage la Prairie location was seeded to B. napus cultivar Regent. The Arboretum site had been seeded to two lines of B. campestris the previous year.In the fall, Trifluralin (Treflan) granular herbicide was applied to the Arboretum at a rate of 28 kg/ha and was incorporated. Liquid Trifluralin was spring applied at the Portage la

Prairie site at 1.5 L/ha in 100 L water, incorporated twice at right angles with a discer, harrowed, and packed. The Arboretum was fertilized in the spring of 1983 with 16-20-0 at a rate of 100 Kg actual nitrogen/ha.

Plots were seeded on June 17 and May 25 (day of the year 168 and 145) in 1982 and 1983,respectively. A four row belt seeder and packer were used to seed to a depth of 2.5 to 4.0 cm. Plot size was 4.6 x 4.9 m resulting in a seeding rate of seven kg/ha. Carbofuran (Furadan 10G) insecticide was applied with the seed at 2.8 kg/ha for flea beetle control. The herbicide 3,6-dichloropicolinic acid (Lontrel) was applied on day 178 (gs 4.1), 1983 in the Arboretum at 850 ml product/ha for thistle control.

A randomised complete block design (RCBD) with four blocks was used in both years. Each block included four plots seeded to various ratios (by seed weight) of Torch to Tobin: 1:0, 3:1, 1:1, and 1:3. Every second plot within each block was seeded to *B. napus* cultivar Regent (immune to *Albugo candida*) to decrease the interplot dispersal of propagules. This resulted in eight plots per block, four of which were not employed in the experiment. Growth stages were evaluated based on Harper and Berkenkamp's (1975) revised growth stage key for *B. campestris* and *B. napus*.

White rust symptoms were evaluated periodically throughout each growing season. Disease severity, or percent leaf area infected with white rust pustules was evaluated on

the first and second non-senescent leaves of ten randomly selected plants from the middle four rows of each plot. For analytical purposes leaf area ratings were expressed as average percent leaf area infected per plot. Staghead incidence and partial staghead incidence were also evaluated for each plot.

A staghead was considered formed when 50% or more of an inflorescence had been replaced with hypertrophied tissue. A partial staghead was considered as one which less than 50% of the inflorescence was hypertrophied.

Two additional variables were evaluated in 1983. From the same 10 plants selected for disease severity ratings, the foliar incidence of white rust was recorded for the first and second non-senescent leaves of each plant and was expressed as the percentage of plants with foliar symptoms per plot.

Because Sclerotinia and root rot infection might have confounded the analysis of yield in 1982 and 1983, respectively, these diseases were rated at their respective locations at the end of each season. Root rot was evaluated on 10 plants per plot and the variable was expressed as average root rot severity per plot. Twenty plants per plot were evaluated for Sclerotinia incidence per plot.

At maturity, the centre three metres of the middle four rows of each plot were hand harvested when 60 to 75% of the seed in the pods had turned from green to brown. Plants were

cut just above ground level, placed in burlap sacks, and allowed to air dry for one to two weeks before threshing. Seed yields were determined and for analytical purposes were expressed as grams of seed per plot.

Data were entered into the University of Manitoba Amdahl 5850 computer system using MANTES (Ferch et al., 1978). Statistical analysis was carried out using the SAS package (Ray et al., 1982). Because the populations could not be assumed normal, the Kruskal-Wallis test, the non-parametric equivalent of a one-way analysis of variance, was used for analytical purposes. The lsd multiple comparison procedure was performed on each significant dependent variable in which treatments were significant. Where dependent variables were rated more than once, data were pooled to permit an analysis of the independent variable, date. These variables were then sorted by date and the Kruskal-Wallis test was performed on each dependent variable at each date. Block (B), treatment (T), and date (D) main effects, and T*D and B*T interactions were analysed for each dependent variable. All statistical tests were performed using an alpha level of 5%.

Results

Foliar infection of the first leaf

In both 1982 and 1983 date effects were highly significant according to the Kruskal-Wallis test (Kruskal-Wallis test in appendix tables 19 to 37). There was no other significant independent variable in 1982. In 1983, a significant T*D interaction and a highly significant treatment effect were observed. The T*D interaction was apparent between day 187 and 197 where the rate of increase in percent leaf area infected followed a similar pattern in the 100% and 75% Torch treatments and in the 50% and 25% Torch treatments (Figure 1).

In 1983, analysis by date indicated that treatments were insignificant at day 167, 178, and 187, significant at day 193, and highly significant at day 197. White rust symptoms were observed at day 187 in the 100% and 75% Torch treatments. Disease symptoms in the 50% and 25% Torch treatments were not noted until day 193.

Compared to the 100% Torch treatment, the 75% Torch treatment, with 75% of the susceptible tissue of the 100% Torch treatment, supported 77% as much white rust disease in 1983. The 50% and 25% Torch treatments supported 33% and 16% as much white rust disease, respectively. Compared to the pure Torch plots in 1982, only the 25% Torch treatment supported less white rust than the proportion of susceptible tissue in the crop stand.

Foliar infection of the second leaf

With the exception of a highly significant rather than a significant date effect at day 193 and no significant T*D interaction at the Arboretum location, the data for the second leaf were similar to those on white rust infection of the first leaf with respect to main effects and interactions in the Kruskal-Wallis test. Though the T*D interaction was insignificant at the Arboretum, patterns of white rust development on the first leaf of each treatment were similar to those on the second leaf (Figure 2). Disease symptoms were first observed on day 193 in the 25% Torch treatment and on day 187 in the other three treatments.

A lsd test indicated that there were significant differences in average second leaf infection per plot between the 100% and the 75% Torch treatments (Table 1). The 50% and the 25% Torch treatments were in a third lsd group. The 75%, 50%, and 25% Torch treatments supported 70%, 22%, and 14% as much white rust disease on the second leaf, respectively, as the 100% Torch treatment. Similar trends were observed in 1982.

White rust foliar incidence

In all treatments, white rust foliar incidence developed in a similar manner as foliar infection with the exception of foliar incidence on the second leaf in the 100% Torch treatment where no peak in disease incidence was observed

(Figures 3 and 4). Significant differences in disease incidence were only detected with respect to the first leaf and were apparent as a T*D interaction. In the 100% Torch treatment, the average percent white rust incidence per plot on the first leaf was 41%. The 75%, 50%, and 25% Torch treatments supported 78%, 44%, and 20% as much white rust disease, respectively, as the 100% Torch treatment. Symptoms were first observed on the first leaf at day 187 in the 100% and 75% Torch treatments and at day 193 in the 50% and 25% Torch treatments. On the second leaf, symptoms were observed in the 25% Torch treatment on day 193 and on day 187 in the other three treatments.

Analysis by date indicated that with respect to disease incidence on the first leaf, treatments were not significant at day 167, 178, or 187 but were highly significant at day 193 and 197. Though treatments were not significant with respect to pooled data of white rust incidence on the second leaf, the Kruskal-Wallis analysis by date showed significant differences between treatments at day 197.

Staghead incidence

Highly significant date effects and B*T interactions were apparent in both years. In 1982, a significant treatment effect and a highly significant T*D interaction were observed. When analysed by date, the effects of the various treatments were only significant at day 217, the third of three rating dates.

Stagheads were first observed at day 211 in 1982 (Figure 5). The rate of staghead production was rapid up to this point and then levelled off slightly in all treatments. In 1983, stagheads were first observed at day 193 in the 100% Torch treatment but not until day 197 in the other three treatments. The number of stagheads in each treatment reached a peak at day 201 and remained constant to the termination of the experiment on day 213. The maximum average number of stagheads per treatment was approximately thirty-nine in 1982 but only about eight in 1983.

In 1982, as with the first leaf, only in the 25% Torch treatment was the staghead incidence lower than the proportion of susceptible tissue in the crop stand compared to the 100% susceptible plots at the last rating date.

Partial staghead incidence

Highly significant date effects and B*T interactions were observed in both years. A significant treatment effect was apparent in 1983. Analysis by date indicated that treatments were only significant on day 197 and 201, the second and third of four rating dates.

Partial stagheads were first observed at day 211 in 1982 (Figure 6). The rate of partial staghead production was high between day 203 and 211 and then levelled off slightly in all treatments between day 211 and the termination of the experiment on day 217. In 1983, partial stagheads were

initially observed at day 193 in the 100% Torch treatment, at day 197 in the 75% and 25% Torch treatments, but not until day 201 in the 50% Torch treatment. The number of partial stagheads reached a peak on day 201 and remained at this level to the end of the experiment on day 213.

In 1982, none of the mixtures supported fewer partial stagheads than the percentage susceptible tissue in the mixture when compared with the 100% Torch plots.

Yield of turnip rape

There were no significant block effects in either year. Though the various ratios of resistant to susceptible cultivars in the treatments incited significantly different disease levels with respect to one or more of the dependent variables, there was no significant effect on yield.

Sclerotinia infection

Though the incidence of Sclerotinia sclerotiorum in the turnip rape plots ranged from 48% to 85% throughout the experimental area, there were no significant block or treatment effects.

Root rot infection

Rhizoctonia root rot was moderate to severe throughout most of the experimental area in 1983. A significant block effect was observed in the Kruskal-Wallis test, however,

there was no obvious correlation between root rot severity and yield.

Discussion

Wolfe and Barrett (1980) have noted that cultivar mixtures have little advantage over pure, susceptible stands at the beginning of the season when inoculum is from "external" sources. This statement was supported by the 1983 data for all dependent disease variables in that treatments were not significantly different initially but became significant or even highly significant as the epidemic progressed. Though differences between treatments were not significant early in the season, foliar and floral symptoms in the 25% Torch treatment did lag behind those of the 100% Torch treatment by six and four days, respectively. That symptoms were not observed as early in the more resistant as opposed to the susceptible stands in 1983 may only be a reflection of the insensitivity of the sampling technique. These trends were not observed in 1982 with respect to foliar and floral variables, reflecting an absence of early season ratings and a rapid increase in floral hypertrophy, respectively.

Pathogen dissemination appeared slower in the cultivar mixtures than in the pure stand of Torch. In general, the rate of disease development between rating dates decreased as the percentage of resistant plants in the cultivar mixture increased.

There appeared to be a correlation between the percentage of Tobin in each mixture and the protective effect afforded by these plants. In 1983, each mixture generally appeared to support a lower percentage of white rust than the proportion of susceptible plants in the mixture compared to disease levels in the pure Torch stands. In 1982, with the exception of the dependent variables second leaf and partial stagheads, the 25% Torch treatment appeared to support considerably less white rust infection when compared with the 50%, 75%, and 100% Torch treatments as a group. The 25% Torch treatment conceivably represented a critical proportion of resistant tissue which significantly slowed the dissemination of the pathogen within the plot.

Though the more resistant cultivar mixtures significantly decreased the incidence and severity of white rust symptoms, there were no significant differences in the yields of the four treatments. Low incidence of floral symptoms may have contributed to this lack of correlation.

Conclusion

There were no significant differences in disease incidence and severity between cultivar mixtures and the susceptible stand early in the season. Pathogen dissemination appeared slower in the mixtures than in the pure stands and there was a negative correlation between the percentage of Tobin in each mixture and disease incidence

and severity in the crop stand at the end of each season. Despite significantly lower disease levels in the 25% Torch treatment than in the 100% Torch treatment, no significant differences in yield were observed.

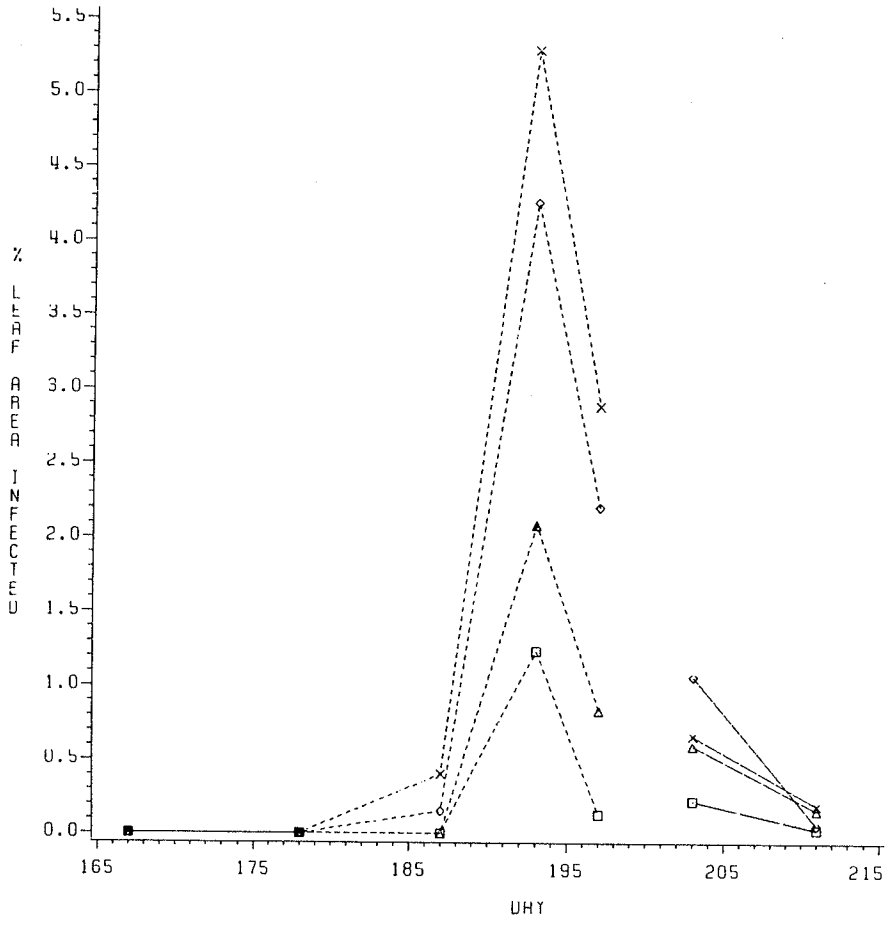
TABLE 1. Response of various white rust disease parameters to four ratios of resistant to susceptible turinip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Variable	Treatment	lsd # Grouping	Average * Infection
Second Leaf&	100% Torch	A	1.1
	75% Torch	B	0.8
	50% Torch	C	0.2
	25% Torch	C	0.2

\# lsd grouping based on ranked data, alpha=0.05.
* Averages calculated from untransformed data.
* Second leaf variables expressed as average % leaf area infected/plot.

& CV= 24.63%

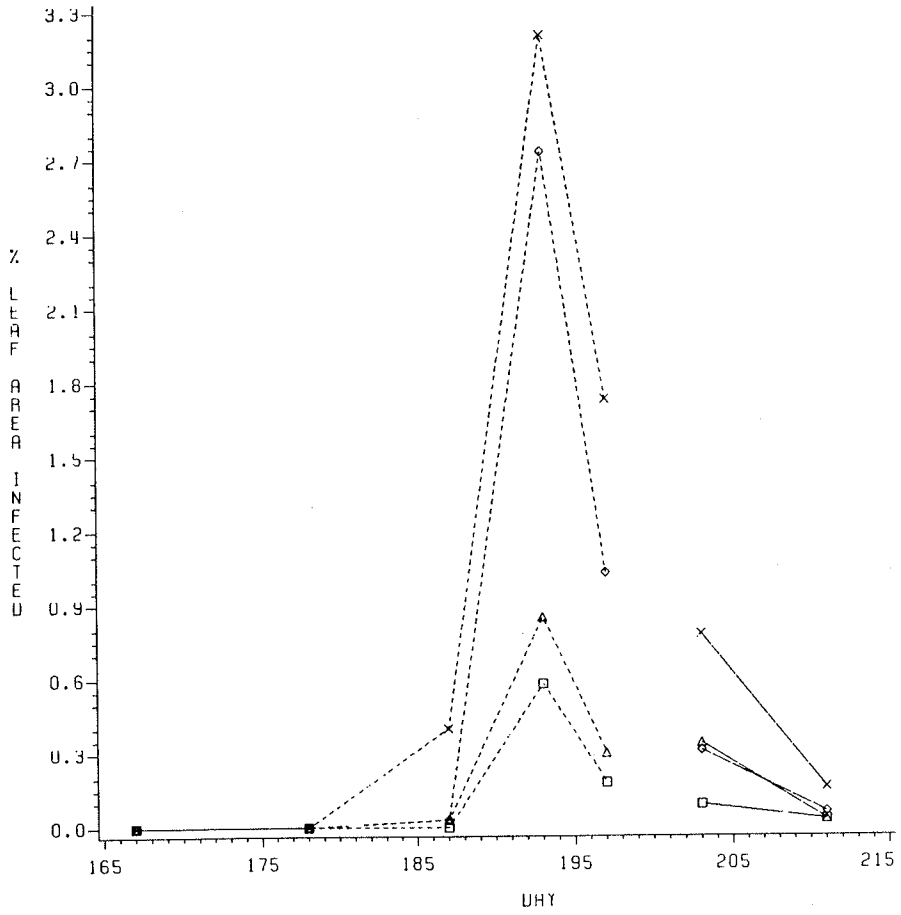
FIGURE 1. Effect of proportion of susceptible plants in a cultivar mixture on the development of white rust infection on the first leaf of turnip rape in 1982 and 1983.



100% Torch (P 1982)
75% Torch (P 1982)
50% Torch (P 1982)
25% Torch (P 1982)
100% Torch (A 1983)
75% Torch (A 1983)
50% Torch (A 1983)
25% Torch (A 1983)

A = Arboretum
P = Portage la Prairie

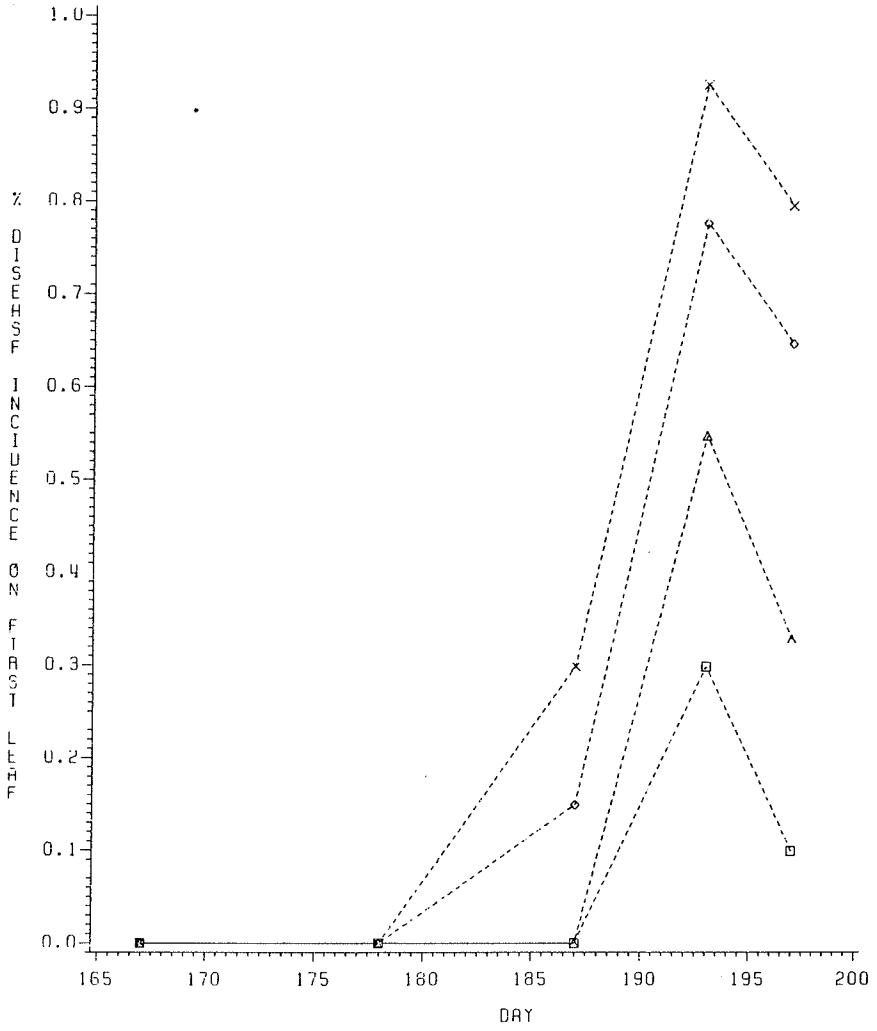
FIGURE 2. Effect of proportion of susceptible plants in a cultivar mixture on the development of white rust infection on the second leaf of turnip rape in 1982 and 1983.



100% Torch (P 1982)
75% Torch (P 1982)
50% Torch (P 1982)
25% Torch (P 1982)
100% Torch (A 1983)
75% Torch (A 1983)
50% Torch (A 1983)
25% Torch (A 1983)

A = Arboretum
P = Portage la Prairie

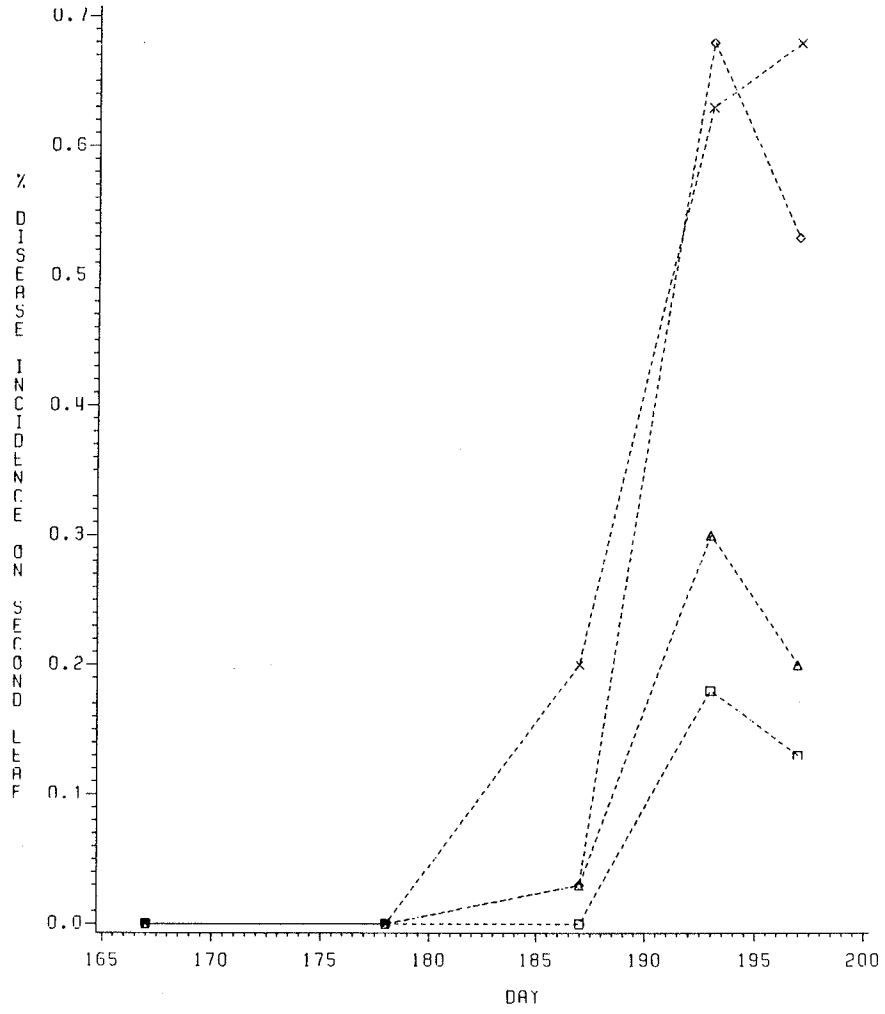
FIGURE 3. Effect of proportion of susceptible plants in a cultivar mixture on white rust incidence on the first leaf of turnip rape in 1983.



100% Torch (A 1983)
75% Torch (A 1983)
50% Torch (A 1983)
25% Torch (A 1983)

A = Arboretum

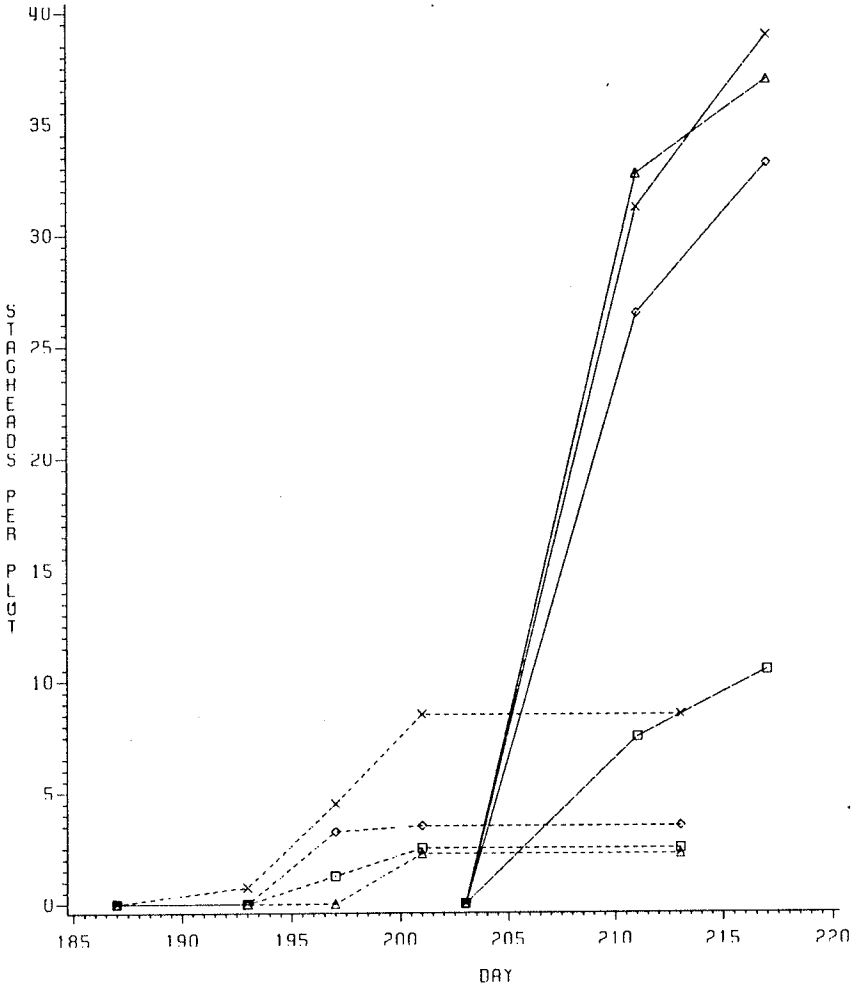
FIGURE 4. Effect of proportion of susceptible plants in a cultivar mixture on white rust incidence on the second leaf of turnip rape in 1983.



100% Torch (A 1983)
75% Torch (A 1983)
50% Torch (A 1983)
25% Torch (A 1983)

A = Arboretum

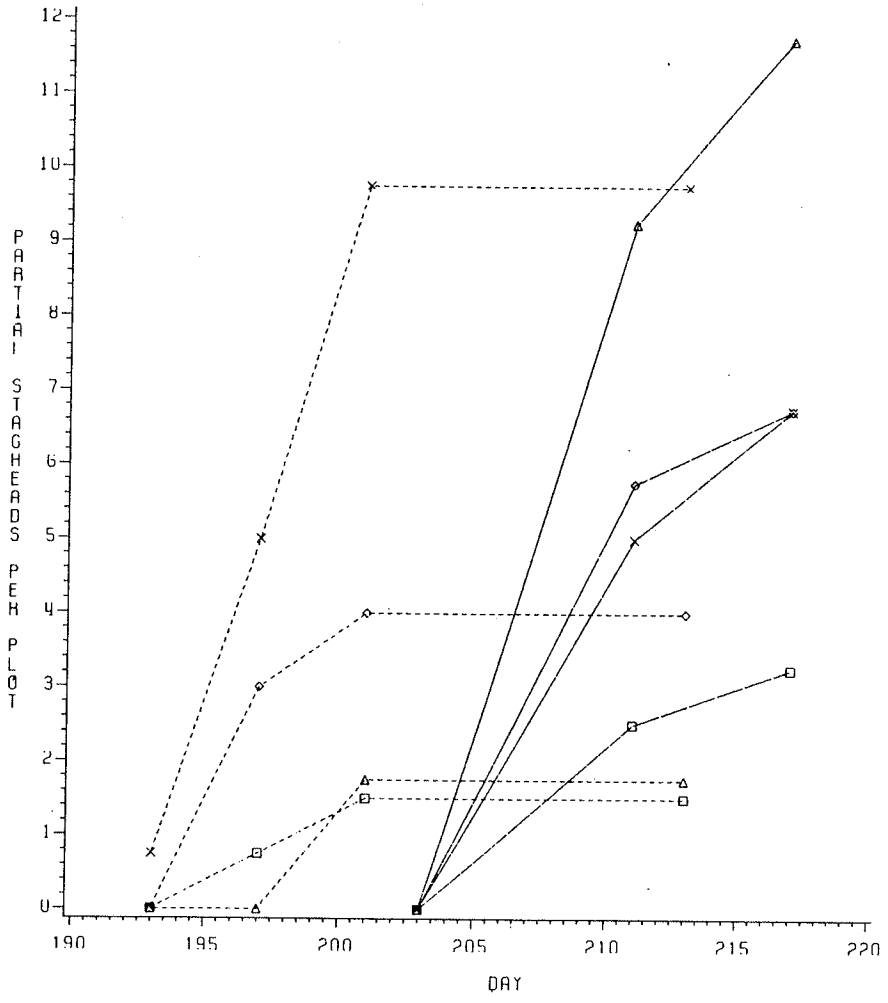
FIGURE 5. Effect of propertion of susceptible plants in a cultivar mixture on staghead incidence in turnip rape in 1982 and 1983.



100% Torch (P 1982)
75% Torch (P 1982)
50% Torch (P 1982)
25% Torch (P 1982)
100% Torch (A 1983)
75% Torch (A 1983)
50% Torch (A 1983)
25% Torch (A 1983)

A = Arboretum
P = Portage la Prairie

FIGURE 6. Effect of propertion of susceptible plants in a cultivar mixture on partial staghead incidence in turnip rape in 1982 and 1983.



100% Torch (P 1982)
75% Torch (P 1982)
50% Torch (P 1982)
25% Torch (P 1982)
100% Torch (A 1983)
75% Torch (A 1983)
50% Torch (A 1983)
25% Torch (A 1983)

A = Arboretum
P = Portage la Prairie

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APPENDIX

TABLE 1. Kruskal-Wallis test for average leaf pustule size.

Isolate	Source	DF	MS	F-value
B. campestris#	Block	1	121.11	3.82
	Cultivar	10	454.75	14.33**
	Block*Cultivar	10	31.74	2.48*
	Error	22	12.77	
	Total	43		
B. campestris&	Cultivar	6	700.00	4.57**
	Error	168	153.15	
	Total	174		
B. juncea¬	Cultivar	10	3673.60	5.38**
	Error	264	683.18	
	Total	274		
B. juncea‡	Cultivar	6	2143.75	9.33**
	Error	168	229.79	
	Total	174		

\# CV= 15.88%
Inoculation of 11 cruciferous hosts

& CV= 14.06%
Inoculation of 7 differential test hosts

¬ CV= 18.94%
Inoculation of 11 cruciferous hosts

‡ CV= 17.23%
Inoculation of 7 differential test hosts

* Significant
** Highly significant

TABLE 2. Kruskal-Wallis test for average number of pustules/leaf.

Isolate	Source	DF	MS	F-value
B. campestris#	Block	1	76.45	1.52
	Cultivar	10	443.13	8.82**
	Block*Cultivar	10	50.27	4.31*
	Error	22	238.63	
	Total	43		
B. campestris&	Cultivar	6	700.00	4.57**
	Error	168	153.15	
	Total	174		
B. juncea¬	Cultivar	10	3707.08	5.44**
	Error	264	681.99	
	Total	274		
B. juncea‡	Cultivar	6	2143.75	9.33**
	Error	168	229.85	
	Total	174		

\# CV= 15.18%
Inoculation of 11 cruciferous hosts

& CV= 14.06%
Inoculation of 7 differential test hosts

¬ CV= 18.92%
Inoculation of 11 cruciferous hosts

‡ CV= 17.23%
Inoculation of 7 differential test hosts

* Significant
** Highly significant

TABLE 3. Kruskal-Wallis test of white rust foliar infection of the first leaf of turnip rape in the 1982 and 1983 yield experiments.

Location and year	Source	DF	MS	F-value
Arboretum 1982¬	Block (B)	5	9115.42	7.51*
	Variety (V)	1	210.68	0.17
	Treatment (T)	4	475.51	0.52
	Date (D)	1	3445.41	3.77
	V*T	4	255.79	0.28
	V*D	1	83.33	0.09
	T*D	4	210.61	0.23
	B*V	5	1214.15	1.33
	V*T*D	4	391.67	0.43
	Error	90	913.06	
	Total	119		
Portage 1982&	Block (B)	5	643.20	0.65
	Variety (V)	1	54.68	0.06
	Treatment (T)	4	2791.53	3.38*
	Date (D)	1	38485.01	46.63**
	V*T	4	901.89	1.09
	V*D	1	644.03	0.78
	T*D	4	828.26	1.00
	B*V	5	986.93	1.20
	V*T*D	4	470.72	0.57
	Error	90	825.38	
	Total	119		
Arboretum 1983+	Block (B)	11	2467.51	3.01**
	Treatment (T)	3	18524.62	22.56**
	Date (D)	4	158011.94	194.92**
	T*D	12	5909.32	7.29**
	B*T	33	821.00	1.01
	Error	176	810.67	
	Total	239		

¬ CV= 49.95%
& CV= 47.49%
+ CV= 23.63%

* Significant
** Highly significant

TABLE 4. Kruskal-Wallis test by day of the year of white rust foliar infection of the first leaf of turnip rape in the 1982 Portage la Prairie yield experiment.

Day	Source	DF	MS	F-value
203¬	Block (B)	5	345.22	1.18
	Variety (V)	1	20.42	0.07
	Treatment (T)	4	412.43	1.41
	V*T	4	304.36	1.04
	Error	45	293.23	
	Total	59		
211+	Block (B)	5	74.97	0.26
	Variety (V)	1	62.02	0.22
	Treatment (T)	4	726.64	2.53
	V*T	4	113.10	0.39
	Error	45	287.43	
	Total	59		

¬ CV= 56.14%
\+ CV= 55.59%

* Significant
** Highly significant

TABLE 5. Kruskal-Wallis test by day of the year of white fust foliar infection of the first leaf of turnip rape in the 1982 Winnipeg Arboretum yield experiment.

Day	Source	DF	MS	F-value
200¬	Block (B)	5	1513.70	7.30**
	Variety (V)	1	2.82	0.01
	Treatment (T)	4	108.18	0.52
	V*T	4	62.11	0.30
	Error	45	207.22	
	Total	59		
209+	Block (B)	5	1381.04	5.89**
	Variety (V)	1	20.42	0.07
	Treatment (T)	4	412.43	1.41
	V*T	4	304.36	1.04
	Error	45	293.23	
	Total	59		

¬ CV= 47.20%
\+ CV= 50.19%

* Significant
** Highly significant

TABLE 6. Kruskal-Wallis test by day of the year of white rust foliar infection of the first leaf of turnip rape in the 1983 Winnipeg Arboretum yield experiment.

Day	Source	DF	MS	F-value
187+	Block (B)	11	186.90	2.17*
	Treatment (T)	3	863.24	10.02**
	Error	33	86.15	
	Total	47		
193&	Block (B)	11	160.10	2.68**
	Treatment (T)	3	1808.32	30.25**
	Error	33	59.77	
	Total	47		
197#	Block (B)	11	389.45	5.60**
	Treatment (T)	3	856.31	12.31**
	Error	33	69.55	
	Total	47		

\+ CV= 37.88%
& CV= 31.56%
# CV= 34.04%

* Significant
** Highly significant

TABLE 7. Kruskal-Wallis test of white rust foliar infection of the second leaf of turnip rape in the 1982 and 1983 yield experiments.

Location and year	Source	DF	MS	F-value
Arboretum 1982¬	Block (B)	5	9414.67	8.12*
	Variety (V)	1	760.03	0.66
	Treatment (T)	4	277.91	0.28
	Date (D)	1	4236.41	5.21*
	V*T	4	186.71	0.23
	V*D	1	4380.21	5.38*
	T*D	4	282.73	0.35
	B*V	5	1159.70	1.43
	V*T*D	4	1055.31	1.30
	Error	90	813.83	
	Total	119		
Portage 1982&	Block (B)	5	641.15	1.07
	Variety (V)	1	2.70	0.00
	Treatment (T)	4	214.53	2.94**
	Date (D)	1	26048.53	28.17**
	V*T	4	700.98	0.76
	V*D	1	1672.53	1.81
	T*D	4	2203.34	2.38
	B*V	5	597.73	0.65
	V*T*D	4	365.21	0.39
	Error	90	924.68	
	Total	119		
Arboretum 1983+	Block (B)	11	2655.99	2.37**
	Treatment (T)	3	17396.14	15.55**
	Date (D)	4	128545.96	144.49**
	T*D	12	5337.51	6.00**
	B*T	33	1118.60	1.26
	Error	176	889.66	
	Total	239		

¬ CV= 47.15%
& CV= 50.26%
+ CV= 24.75%

* Significant
** Highly significant

TABLE 8. Kruskal-Wallis test by day of the year of white rust foliar infection of the second leaf of turnip rape in the 1982 Portage la Prairie yield experiment.

Day	Source	DF	MS	F-value
203¬	Block (B)	5	568.39	2.16
	Variety (V)	1	336.07	1.28
	Treatment (T)	4	559.29	2.13
	V*T	4	105.09	0.40
	Error	45	262.94	
	Total	59		
211+	Block (B)	5	174.86	0.71
	Variety (V)	1	141.07	0.58
	Treatment (T)	4	935.79	3.82**
	V*T	4	253.71	1.04
	Error	45	244.98	
	Total	59		

¬ CV= 53.17%
\+ CV= 51.32%

* Significant
** Highly significant

TABLE 9. Kruskal-Wallis test by day of the year of white rust foliar infection of the second leaf of turnip rape in the 1982 Winnipeg Arboretum yield experiment.

Day	Source	DF	MS	F-value
200¬	Block (B)	5	1429.40	7.34**
	Variety (V)	1	1085.40	5.44*
	Treatment (T)	4	15.82	0.08
	V*T	4	127.39	0.65
	Error	45	194.67	
	Total	59		
209+	Block (B)	5	1296.03	5.72**
	Variety (V)	1	160.07	0.71
	Treatment (T)	4	125.41	0.55
	V*T	4	127.14	0.56
	Error	45	226.62	
	Total	59		

¬ CV= 45.74%
\+ CV= 49.36%

* Significant
** Highly significant

TABLE 10. Kruskal-Wallis test by day of the year of white rust foliar infection of the second leaf of turnip rape in the 1983 Winnipeg Arboretum yield experiment.

Day	Source	DF	MS	F-value
187+	Block (B)	11	161.88	3.06**
	Treatment (T)	3	248.38	4.70**
	Error	33	52.83	
	Total	47		
193&	Block (B)	11	161.92	2.12*
	Treatment (T)	3	1611.14	21.10**
	Error	33	76.36	
	Total	47		
197#	Block (B)	11	310.20	2.75**
	Treatment (T)	3	1956.71	5.79**
	Error	33	112.64	
	Total	47		

\+ CV= 29.67%
& CV= 35.67%
# CV= 43.32%

* Significant
** Highly significant

TABLE 11. Kruskal-Wallis test of staghead incidence in turnip rape in the 1982 and 1983 yield experiments.

Location and year	Source	DF	MS	F-value
Arboretum 1982¬	Block (B)	5	31625.58	5.18*
	Variety (V)	1	897.80	0.15
	Treatment (T)	4	931.10	0.85
	Date (D)	2	17819.71	16.25**
	V*T	4	4451.85	4.06**
	V*D	2	22.40	0.02
	T*D	8	1019.92	0.93
	B*V	5	6107.16	5.57**
	V*T*D	8	295.82	0.27
	Error	140	1096.55	
	Total	179		
Portage 1982&	Block (B)	5	643.20	0.65
	Variety (V)	1	0.67	0.00
	Treatment (T)	4	1683.36	3.07*
	Date (D)	2	143602.81	261.50**
	V*T	4	178.21	0.32
	V*D	2	59.17	0.11
	T*D	8	1415.39	2.58*
	B*V	5	1230.72	2.24
	V*T*D	8	142.93	0.26
	Error	140	549.14	
	Total	179		
Arboretum 1983+	Block (B)	11	3309.53	1.54
	Treatment (T)	3	1319.06	0.62
	Date (D)	2	8906.64	22.86**
	T*D	6	71.05	0.18
	B*T	33	2142.80	5.50**
	Error	88	389.67	
	Total	143		

¬ CV= 36.59%
& CV= 25.89%
+ CV= 27.23%

* Significant
** Highly significant

TABLE 12. Kruskal-Wallis test by day of the year of staghead incidence in turnip rape in the 1982 Portage la Prairie yield experiment.

Day	Source	DF	MS	F-value
211+	Block (B)	5	1176.34	6.67**
	Variety (V)	1	6.67	0.04
	Treatment (T)	4	69.84	0.40
	V*T	4	51.61	0.29
	Error	45	176.35	
	Total	59		
217&	Block (B)	5	924.90	5.14**
	Variety (V)	1	36.82	0.20
	Treatment (T)	4	1177.11	6.54**
	V*T	4	96.82	0.54
	Error	45	179.88	
	Total	59		

\+ CV= 43.54%
& CV= 43.97%

* Significant
** Highly significant

TABLE 13. Kruskal-Wallis test by day of the year of staghead incidence in turnip rape in the 1982 Winnipeg Arboretum yield experiment.

Day	Source	DF	MS	F-value
200¬	Block (B)	5	652.33	4.65**
	Variety (V)	1	33.75	0.24
	Treatment (T)	4	115.29	0.82
	V*T	4	200.94	1.43
	Error	45	140.25	
	Total	59		
209+	Block (B)	5	1387.95	8.59**
	Variety (V)	1	62.02	0.38
	Treatment (T)	4	119.13	0.74
	V*T	4	257.06	1.59
	Error	45	161.60	
	Total	59		
213&	Block (B)	5	1948.70	14.42**
	Variety (V)	1	62.02	0.46
	Treatment (T)	4	83.74	0.62
	V*T	4	146.76	1.09
	Error	45	135.13	
	Total	59		

¬ CV= 38.83%
\+ CV= 41.68%
& CV= 38.11%

* Significant
** Highly significant

TABLE 14. Kruskal-Wallis test by day of the year of staghead incidence in turnip rape in the 1983 Winnipeg Arboretum yield experiment.

Day	Source	DF	MS	F-value
197¬	Block (B)	11	35.01	0.71
	Treatment (T)	3	31.43	0.63
	Error	33	49.61	
	Total	47		
201+	Block (B)	11	35.01	0.71
	Treatment (T)	3	31.43	0.63
	Error	33	49.61	
	Total	47		
213&	Block (B)	11	244.53	1.87
	Treatment (T)	3	64.51	0.49
	Error	33	130.74	
	Total	47		

¬ CV= 28.75%
\+ CV= 28.75%
& CV= 46.67%

* Significant
** Highly significant

TABLE 15. Kruskal-Wallis test of partial staghead incidence in turnip rape in the 1982 and 1983 yield experiments.

Location and year	Source	DF	MS	F-value
Arboretum 1982¬	Block (B)	5	59.24	0.20
	Variety (V)	1	29.40	0.10
	Treatment (T)	4	125.51	1.15
	V*T	4	64.41	0.59
	B*V	5	296.36	2.71*
	Error	40	109.20	
	Total	59		
Portage 1982&	Block (B)	5	309.91	2.88
	Variety (V)	1	81.82	0.83
	Treatment (T)	4	770.09	3.97**
	V*T	4	326.29	1.68
	B*V	5	107.45	0.55
	Error	40	193.80	
	Total	59		
Arboretum 1983+	Block (B)	11	1390.88	0.69
	Treatment (T)	3	9787.14	4.82**
	Date (D)	2	24025.00	55.96**
	T*D	6	1545.98	3.60**
	B*T	33	2029.90	4.73**
	Error	88	429.32	
	Total	143		

¬ CV= 34.26%
& CV= 45.64%
+ CV= 28.58%

* Significant
** Highly significant

TABLE 16. Kruskal-Wallis test by day of the year of partial staghead incidence in turnip rape in the 1983 Winnipeg Arboretum yield experiment.

Day	Source	DF	MS	F-value
197¬	Block (B)	11	64.55	1.13
	Treatment (T)	3	148.04	2.60
	Error	33	56.89	
	Total	47		
201+	Block (B)	11	64.55	1.13
	Treatment (T)	3	148.04	2.60
	Error	33	56.89	
	Total	47		
213&	Block (B)	11	123.44	0.83
	Treatment (T)	3	148.04	2.60
	Error	33	56.89	
	Total	47		

¬ CV= 30.79%
+ CV= 30.79%
& CV= 49.92%

* Significant
** Highly significant

TABLE 17. Kruskal-Wallis test of yield in turnip rape in the 1982 and 1983 yield experiments.

Location and year	Source	DF	MS	F-value
Arboretum 1982¬	Block (B)	5	1591.48	6.22*
	Variety (V)	1	60.00	0.23
	Treatment (T)	4	153.06	0.86
	V*T	4	235.50	1.32
	B*V	5	255.86	1.43
	Error	40	178.53	
	Total	59		
Portage 1982&	Block (B)	5	434.14	1.10
	Variety (V)	1	16.02	0.04
	Treatment (T)	4	287.89	0.92
	V*T	4	24.40	0.08
	B*V	5	393.30	1.25
	Error	40	314.48	
	Total	59		
Arboretum 1983+	Block (B)	11	359.32	2.35*
	Treatment (T)	3	72.17	0.47
	Error	33	152.82	
	Total	47		

¬ CV= 43.81%
& CV= 58.14%
+ CV= 50.46%

* Significant
** Highly significant

TABLE 18. Kruskal-Wallis test of root rot rating of turnip rape in the 1983 Winnipeg Arboretum yield experiment.

Source	DF	MS	F-value
Block (B)	11	309.60	1.94
Treatment (T)	3	43.63	0.27
Error	33	159.83	
Total	47		

CV= 51.60%

* Significant
** Highly significant

TABLE 19. Kruskal-Wallis test of white rust foliar infection on the first leaf of turnip rape in the 1982 and 1983 mixed cultivar experiments.

Location and year	Source	DF	MS	F-value
Portage 1982¬	Block (B)	3	92.15	1.18
	Treatment (T)	3	72.27	0.93
	Date (D)	1	684.50	12.47**
	T*D	3	4.52	0.08
	B*T	9	78.08	1.42
	Error	12	54.87	
	Total	31		
Arboretum 1983+	Block (B)	3	121.43	1.15
	Treatment (T)	3	850.89	8.09**
	Date (D)	4	6496.51	120.46**
	T*D	12	175.56	3.26*
	B*T	9	105.24	1.95
	Error	48	53.93	
	Total	79		

¬ CV= 44.89%
\+ CV= 18.13%

* Significant
** Highly significant

TABLE 20. Kruskal-Wallis test by day of the year of white rust foliar infection of the first leaf of turnip rape in the 1982 Portage la Prairie mixed cultivar experiment.

Day	Source	DF	MS	F-value
203¬	Block (B)	3	2.13	0.46
	Treatment (T)	3	20.50	0.78
	Error	9	26.18	
	Total	15		
211+	Block (B)	3	29.17	1.60
	Treatment (T)	3	7.88	0.43
	Error	9	26.18	
	Total	15		

¬ CV= 60.20%
\+ CV= 50.20%

* Significant
** Highly significant

TABLE 21. Kruskal-Wallis test by day of the year of white rust foliar infection of the first leaf of turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Day	Source	DF	MS	F-value
187+	Block (B)	3	2.79	0.22
	Treatment (T)	3	34.83	2.69
	Error	9	12.96	
	Total	15		
193&	Block (B)	3	4.88	0.38
	Treatment (T)	3	69.50	5.42*
	Error	9	12.82	
	Total	15		
197#	Block (B)	3	16.50	3.30
	Treatment (T)	3	81.16	16.23**
	Error	9	5.00	
	Total	15		

\+ CV= 42.35%
& CV= 42.12%
# CV= 26.31%

* Significant
** Highly significant

TABLE 22. Kruskal-Wallis test of white rust foliar infection on the second leaf of turnip rape in the 1982 and 1983 mixed cultivar experiments.

Location and year	Source	DF	MS	F-value
Portage 1982¬	Block (B)	3	38.90	0.86
	Treatment (T)	3	148.71	3.28
	Date (D)	1	750.78	13.85**
	T*D	3	35.53	0.66
	B*T	9	45.33	0.84
	Error	12	54.20	
	Total	31		
Arboretum 1983+	Block (B)	3	238.88	2.05
	Treatment (T)	3	801.03	5.78*
	Date (D)	4	5343.54	53.72**
	T*D	12	183.31	1.84
	B*T	9	138.58	1.39
	Error	48	99.47	
	Total	79		

¬ CV= 44.62%
\+ CV= 24.63%

* Significant
** Highly significant

TABLE 23. Kruskal-Wallis test by day of the year of white rust foliar infection of the second leaf of turnip rape in the 1982 Portage la Prairie mixed cultivar experiment.

Day	Source	DF	MS	F-value
203¬	Block (B)	3	4.04	0.21
	Treatment (T)	3	49.79	2.63
	Error	9	18.94	
	Total	15		
211+	Block (B)	3	17.46	1.03
	Treatment (T)	3	8.25	0.49
	Error	9	16.88	
	Total	15		

¬ CV= 51.20%
\+ CV= 48.33%

* Significant
** Highly significant

TABLE 24. Kruskal-Wallis test by day of the year of white rust foliar infection of the second leaf of turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Day	Source	DF	MS	F-value
187+	Block (B)	3	9.63	0.67
	Treatment (T)	3	12.13	0.84
	Error	9	14.42	
	Total	15		
193&	Block (B)	3	3.04	0.40
	Treatment (T)	3	87.21	11.42**
	Error	9	7.64	
	Total	15		
197#	Block (B)	3	30.54	6.07*
	Treatment (T)	3	66.54	13.23**
	Error	9	5.03	
	Total	15		

\+ CV= 44.67%
& CV= 32.52%
# CV= 26.38%

* Significant
** Highly significant

TABLE 25. Kruskal-Wallis test of foliar incidence of white rust of the first leaf of turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Source	DF	MS	F-value
Block (B)	3	97.31	0.90
Treatment (T)	3	1109.87	10.27**
Date (D)	4	6010.41	88.57**
T*D	12	218.52	3.22**
B*T	9	108.10	1.59
Error	48	67.86	
Total	79		

CV= 20.34%

* Significant
** Highly significant

TABLE 26. Kruskal-Wallis test by day of the year of white rust foliar incidence on the first leaf of turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Day	Source	DF	MS	F-value
187+	Block (B)	3	2.79	0.22
	Treatment (T)	3	34.83	2.69
	Error	9	12.96	
	Total	15		
193&	Block (B)	3	0.83	0.38
	Treatment (T)	3	104.04	15.11**
	Error	9	2.21	
	Total	15		
197#	Block (B)	3	10.16	1.66
	Treatment (T)	3	82.50	13.50**
	Error	9	6.11	
	Total	15		

\+ CV= 42.35%
& CV= 17.48%
# CV= 29.08%

* Significant
** Highly significant

TABLE 27. Kruskal-Wallis test of foliar incidence of white rust on the second leaf of turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Source	DF	MS	F-value
Block (B)	3	254.58	0.98
Treatment (T)	3	693.70	2.67
Date (D)	4	4714.72	38.41**
T*D	12	189.33	1.54
B*T	9	260.06	2.12*
Error	48	122.75	
Total	79		

CV= 27.36%

* Significant
** Highly significant

TABLE 28. Kruskal-Wallis test by day of the year of white rust foliar incidence on the second leaf of turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Day	Source	DF	MS	F-value
187+	Block (B)	3	9.63	0.67
	Treatment (T)	3	12.13	0.84
	Error	9	14.42	
	Total	15		
193&	Block (B)	3	4.83	0.26
	Treatment (T)	3	50.63	2.69
	Error	9	18.79	
	Total	15		
197#	Block (B)	3	17.42	3.79
	Treatment (T)	3	78.79	17.14**
	Error	9	4.60	
	Total	15		

\+ CV= 44.67%
& CV= 51.00%
# CV= 25.22%

* Significant
** Highly significant

TABLE 29. Kruskal-Wallis test of staghead incidence in turnip rape in the 1982 and 1983 mixed cultivar experiments.

Location and year	Source	DF	MS	F-value
Portage 1982¬	Block (B)	3	50.29	0.71
	Treatment (T)	3	301.88	4.29*
	Date (D)	2	3314.25	158.45**
	T*D	6	78.85	4.01**
	B*T	9	70.41	3.58**
	Error	24	19.65	
	Total	47		
Arboretum 1983+	Block (B)	3	833.86	2.60
	Treatment (T)	3	847.89	2.64
	Date (D)	3	2588.73	30.49**
	T*D	9	120.88	1.42
	B*T	9	321.08	3.78**
	Error	36	84.91	
	Total	63		

¬ CV= 18.10%
\+ CV= 28.35%

* Significant
** Highly significant

TABLE 30. Kruskal-Wallis test by day of the year of staghead incidence in turnip rape in the 1982 Portage la Prairie mixed cultivar experiment.

Day	Source	DF	MS	F-value
211+	Block (B)	3	10.08	0.62
	Treatment (T)	3	54.46	3.37
	Error	9	16.15	
	Total	15		
217&	Block (B)	3	10.21	0.73
	Treatment (T)	3	61.17	4.39*
	Error	9	13.93	
	Total	15		

\+ CV= 47.28%
& CV= 43.91%

* Significant
** Highly significant

TABLE 31. Kruskal-Wallis test by day of the year of staghead incidence in turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Day	Source	DF	MS	F-value
193¬	Block (B)	3	4.00	1.00
	Treatment (T)	3	4.00	1.00
	Error	9	4.00	
	Total	15		
197+	Block (B)	3	24.54	2.40
	Treatment (T)	3	37.79	3.70
	Error	9	10.22	
	Total	15		
201&	Block (B)	3	26.79	2.66
	Treatment (T)	3	36.04	3.58
	Error	9	10.05	
	Total	15		
213#	Block (B)	3	24.17	1.28
	Treatment (T)	3	28.54	1.51
	Error	9	18.88	
	Total	15		

¬ CV= 23.53%
\+ CV= 37.61%
& CV= 37.31%
# CV= 51.11%

* Significant
** Highly significant

TABLE 32. Kruskal-Wallis test of partial staghead in turnip rape in the 1982 and 1983 mixed cultivar experiments.

Location and year	Source	DF	MS	F-value
Portage 1982¬	Block (B)	3	101.90	0.75
	Treatment (T)	3	303.06	2.24
	Date (D)	1	112.50	9.83**
	T*D	3	4.65	0.41
	B*T	9	135.26	11.82**
	Error	12	11.44	
	Total	31		
Arboretum 1983+	Block (B)	3	853.14	2.45
	Treatment (T)	3	1621.41	4.65*
	Date (D)	3	1179.73	23.60**
	T*D	9	101.65	1.35
	B*T	9	348.38	4.62**
	Error	36	75.42	
	Total	63		

¬ CV= 20.50%
\+ CV= 26.72%

* Significant
** Highly significant

TABLE 33. Kruskal-Wallis test by day of the year of partial staghead incidence in turnip rape in the 1982 Portage la Prairie mixed cultivar experiment.

Day	Source	DF	MS	F-value
211+	Block (B)	3	23.50	1.06
	Treatment (T)	3	21.63	0.98
	Error	9	22.13	
	Total	15		
217&	Block (B)	3	6.71	0.46
	Treatment (T)	3	61.88	4.28*
	Error	9	14.47	
	Total	15		

\+ CV= 55.34%
& CV= 44.76%

* Significant
** Highly significant

TABLE 34. Kruskal-Wallis test by day of the year of partial staghead incidence in turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Day	Source	DF	MS	F-value
193¬	Block (B)	3	5.38	1.00
	Treatment (T)	3	16.00	2.98
	Error	9	5.38	
	Total	15		
197+	Block (B)	3	12.79	1.08
	Treatment (T)	3	50.83	4.31*
	Error	9	11.79	
	Total	15		
201&	Block (B)	3	14.04	1.30
	Treatment (T)	3	52.63	4.86*
	Error	9	10.83	
	Total	15		
213#	Block (B)	3	33.42	2.64
	Treatment (T)	3	39.71	3.13
	Error	9	12.68	
	Total	15		

¬ CV= 27.28%
\+ CV= 40.40%
& CV= 38.72%
# CV= 41.89%

* Significant
** Highly significant

TABLE 35. Kruskal-Wallis test of yield in turnip rape in the 1982 and 1983 mixed cultivar experiments.

Location and year	Source	DF	MS	F-value
Portage 1982¬	Block (B)	3	7.38	0.28
	Treatment (T)	3	27.04	1.03
	Error	9	24.28	
	Total	15		
Arboretum 1983+	Block (B)	3	29.50	2.00
	Treatment (T)	3	39.50	2.67
	Error	9	14.78	
	Total	15		

¬ CV= 60.28%
\+ CV= 45.23%

* Significant
** Highly significant

TABLE 36. Kruskal-Wallis test of sclerotinia rating of turnip rape in the 1982 Portage la Prairie mixed cultivar experiment.

Source	DF	MS	F-value
Block (B)	3	37.63	1.55
Treatment (T)	3	1.71	0.07
Error	9	24.28	
Total	15		

CV= 57.97%

* Significant
** Highly significant

TABLE 37. Kruskal-Wallis test of root rot rating of turnip rape in the 1983 Winnipeg Arboretum mixed cultivar experiment.

Source	DF	MS	F-value
Block (B)	3	66.63	7.16**
Treatment (T)	3	14.96	1.61
Error	9	9.31	
Total	15		

CV= 38.89%

* Significant
** Highly significant

TABLE 38. ANOVA for colony length.

Source	DF	MS	F-value
Day	2	477493.80	11.14**
Error	104	42848.29	
Total	106		

CV= 76.52%

* Significant
** Highly significant

TABLE 39. ANOVA for colony width.

Source	DF	MS	F-value
Day	2	187225.15	13.26**
Error	104	14114.65	
Total	106		

CV= 74.77%

* Significant
** Highly significant