INHERITANCE OF RESISTANCE TO *ALBUGO CANDIDA* IN RAPE (*BRASSICA NAPUS L.*)

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Zhegong Fan

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ABSTRACT

Fan Zhegong, M.Sc., The University of Manitoba, February, 1983. The inheritance of resistance to Albugo candida in rape (Brassica napus L.). Major Professor, B.R. Stefansson.

Several lines of B.napus obtained from China were susceptible to white rust caused by A.candida. Two Chinese lines, 2282-9 and Green Cup Leaf (GCL), and one Canadian cultivar, Regent, were chosen for the genetic study. From the results, inheritance of white rust resistance is conditioned by independent dominant genes at three loci, which are designated Ac7-1, Ac7-2 and Ac7-3. The resistance is conferred by dominance at any one of the three loci, and trigenic recessives are susceptible.

Since different F2 and BC ratios were obtained for populations from different individual plants from Regent, this Regent population is not homogeneous for resistance to white rust. All Regent plants appear to be homogeneous for resistance at two loci while others may also carry resistance at a third locus.

Variation in susceptibility of 2282-9 and GCL, as well as their F1 and F2 progenies suggests the existence of minor genes modifying the disease reaction. The major recessive genes in 2282-9 and GCL are allelic.

INTRODUCTION

White rust is caused by the fungus Albugo candida (Pers. ex Chev.) Kuntze. and occurs commonly on many species of the Cruciferae and some species of the Capparidaceae. It has been reported that at least 241 species in 63 genera in the Cruciferae are affected by the disease (Biga,1955). Most of these species are cruciferous weeds. Cultivated crops, such as horseradish, radish, cabbage, mustard, turnip rape, etc, also suffer from the disease to a certain extent.

In proportion to its widespead occurrence, white rust is generally considered to be an economical disease of limited importance. In some cases, however, it may become severe on horseradish (Armoracia rusticana G.) in northern states of U.S.A., due to heavy infection of the leaves and roots (Endo & linn, 1960). The production of radish (Raphanus sativus L.) has never been reduced substantially by the disease, but the seed crop of radish may be substantially reduced by the distortion of branches and flower parts (Williams & Pound, 1963). In India, white rust was found to be a serious disease on mustard (Brassica juncea Coss.) if conditions during the flowering time favored the disease (Kumari et al, 1970). Considerable yield losses of turnip rape (Brassica campestris L.) grown in western Canada were

reported. In Saskatchewan, the losses were approximately 3%. 6%, 9%, respectively, for 1970,1971 and 1972 (Petrie, 1973). In central and northern Alberta, there was a 1.2% loss in 1971 (Berkenkamp, 1972). In Manitoba, the yield reductions in severely affected fields were estimated to range from 30 to 60% in 1971 (Bernier, 1972). Harper and Pittman (1974) have proposed a formula which can be used to assess accurately the yield loss from the meristematic infection.

Rape (Brassica napus L.) is highly resistant or immune to white rust in Canada. This has been demonstrated by lack of both natural infection in the field and artificial infection in the greenhouse (Petrie, 1975a, Petrie and Dueck, 1979). However, in central China some cultivars grown are quite susceptible to white rust. Stagheads (inflorescence galls) occur frequently in the field every year. To date losses from the disease have not been estimated accurately.

Genetic studies of white rust resistance and breeding of resistant varieties have been conducted with horseradish, radish, and mustard (Hougas et al, 1952, Williams and Pound, 1963, Ebrahimi et al, 1976). Little information is available for rape. Therefore the present study was undertaken to investigate the inheritance of white rust resistance in rape.

WHITE RUST AND THE CAUSAL AGENT

Albugo candida is an obligate parasite. It causes local and meristematic infections on host plants. The local infection results in the production of white pustules on leaves and stems. The pustules in close proximity usually merge to form large patches. Release of sporangia from the pustules occurs when the host epidermis bursts. In the meristematic infection, infected inflorescences are distorted and hypertrophied, and seed development is prevented. This stage is often called "staghead".

Pathogen

Albugo is the only genus in the family Albuginaceae. In the early literature, synonyms of A.candida are Aecidium candidum Pers., A.cruciferarum., Uredo candida Pers., Uredo cheiranthi Pers., and Cystopus candidus Lev. (Walker, 1969). Descriptions of the fungal characteristics and life cycle can be found in many books of plant pathology and mycology (Walker, 1969, Webster, 1980,).

The mycelium of A.candida in host tissues is intercellular with intracellular haustoria. The fine structure of the haustorium has been studied by Berlin and Bowen (1964) on radish and Verma et al (1975) on four

Brassica species. The haustorium is a small, spherical body which originates from the mycelium and projects inside the host cell.

A typical feature of the fungus is that the mycelium forms a compacted palisade of cylindrical sporangiophores beneath the host epidermis. From these sporangiophores chains of zoosporangia arise in such a fashion that the newly formed zoosporangium is at the base. When the zoosporangia are fully mature, white pustules are externally visible. The epidermis is eventually ruptured by the pressure of developing zoosporangia which results in the release of white powdery zoosporangia into the air currents.

The presence of a water film is essential for the germination of the zoosporangium which gives rise to biflagellate zoospores. The zoospore, if on the leaf or the stem, swims for a while, encysts, and then penetrates the epidermis by forming a germ tube.

In the meristematic infected tissue, development of oogonia and antheridia takes place in the intercellular spaces. Both the oogonium and the antheridium are multinucleate, but only one nucleus in each of them is functional. Meiotic division occurs within the gametangia (Sansome and Sansome, 1974). Fertilization is accomplished by the penetration of a fertilization tube from the antheridium into the oosphere formed at the center of the oogonium. The nucleus discharged from the fertilization

tube fuses with the one in the oosphere from which an oospore develops. The development of the oospore in B.campestris has been studied by Tewari and Skoropad (1977). The fusion nucleus later divides and the division repeats so that the mature oospore may contain as many as thirty-two nuclei. The oospores are thick-walled resting spores.

A period of dormancy is not necessary before the germination of the oospores can occur (Petrie and Verma, 1974). Three different ways of the oospore germination have been reported: the production of a sessile vesicle which contains zoospores (De Bary, 1863), the formation of a discharge tube with a terminal zoosporangium (Vanterpool, 1959), and the formation of a germ tube which directly gives rise to the mycelium (Petrie and Verma, 1974).

The disease cycle

A detailed description of the disease cycle of white rust has been presented by Petrie (1975a). A.candida overwinters by means of the oospores in annual hosts, whereas, the mycelium persisting in the crown and lateral roots may also function as an overwintering form in perennial hosts (Takeshita and Linn, 1953). The oospores may persist for long periods as they have been shown to be viable for more than 20 years after dry storage (Verma and Petrie, 1975). They are either present in the soil or mixed with seeds. From the observation of a high infection of the disease

resulting from the continuous cultivation of rape (B.napus) on the same a piece of land (Vanterpool, 1959) and the finding of a considerable number of oospores in seed samples of B.campestris (Petrie, 1975b), it has been suggested that the oospores are important as the primary inoculum in nature.

Petrie and Verma (1974) pointed out that leaching of the oospores by spring rains was essential for their germination. They also found that the germination was primarily by means of producing a sessile vesicle in which 40-60 zoospores were formed, and the other two types of the germination were probably adopted in stress conditions. The zoospores are released by the time that the membrane of the sessile vesicle is ruptured. They are mobile and capable of swimming in water. Once they reach a suitable host, the invasion takes place in which the germ tubes produced by them pass through stomata into the host tissues.

As a result of the infection, white blisters appear on leaves and stems about ten days after penetration. The shiny, smooth pustules contain a large number of zoosporangia which serve as effective secondary inoculum soon after their dispersion by wind. The zoosporangia alighting on healthy leaves are ready to germinate in the presence of water (Eberhardt,1904). It has also been reported that germination requires a chilling treatment of the zoosporangia (Melhus, 1911,) and dehydration of the

zoosporangia to about 30 percent (Napper, 1933). About eight zoospores are produced from each zoosporangium. The secondary infection may proceed several times in a season.

The meristematic infection which occurs late in the season results in oospore development in the hypertrophied inflorescences. At harvest, crushing stagheads leads to the release of the oospores in the soil or the distribution of the oospores among seeds.

The hypertrophy of the stem and the occurrence of pustules on the leaf also provide an entrance of secondary microorganisms into the host. The prominant association of Peronospora parasitica with A.candida on the stagheads has been well documented by Vanterpool (1960). Later, Petrie and Vanterpool (1974) found that over 20 species of fungi were in such association with A.candida on distorted stems and pod blisters of turnip rape (B. campestris), wild mustard (B.kaber) and false flax (Camelina microcarpa).

Physiological studies

Metabolic changes induced by A.candida on radish cotyledons have been studied by Williams and Pound (1964). They found that most of these changes were similar to those in hosts infected by pathogens of cereal rusts and powdery mildews (Allen, 1959, Millerd and Scott, 1962). Respiration was stimulated at the infection site, and increased up to 2-3 times that of healthy tissue before spore discharge. The accumulation of starch around fungal colonies and the

significant dropping of the ratio of carbon dioxide in nitrogen atmosphere and in air were observed. The only difference was that the C6/C1 ratio was high, around 0.4, and remained constant throughout the disease development of white rust, whereas, in cases of cereal rusts and powdery mildews, the ratio usually decreased considerably. Germinating zoosporangia of A.candida also had high C6/C1 ratio which was between 0.47 and 0.75. These results suggested that increases in respiration and synthesis were mediated through pathways operating in healthy plants or the contribution of the parasite itself.

In many plant diseases, retention of green tissue at infection sites after surrounding tissue becomes chlorotic is called the "green island" effect (Bushnell and Allen, 1962, Wang, 1961,). This phenomenon has been investigated on detached B.juncea cotyledons invaded by A.candida (Harding, et al, 1968,). Autoradiographic and electron microscopic studies revealed that photosynthetic activities were maintained in green island tissues and that delayed breakdown of chloroplast grana was responsible for its formation. In addition, the green island effect was also induced on tissue treated with kinetin.

Alteration of plant growth patterns with infection is a characteristic of plant disease development and is associated with changes of auxin level in host tissue (Gruen, 1959,). The most conspicuous symptom of white rust

is hyperplasia and hypertrophy of floral parts, which become leaf-like. Hirata (1954) found that the stem and leaf tissues of rape plant (B.napus) infected by A.candida contained more diffusible auxin than healthy tissue. A similar result was also reported by kiermayer (1958) for A.candida on Capsella bursa-pastoris where more indole-3-acetonitrile was found in infected leaves and stems. Srivastava et al (1962) reported lower concentrations of auxins and related compounds in extracts from hypertrophied inflorescences of B.napus. They explained that the low levels of auxins prevented normal development of flowers since healthy flowers were rich in auxins. Kumari et al (1970) analysed amino acids contents in distorted peduncles, buds and flowers of B.juncea and suggested that the infection caused the breakdown of plant proteins, resulting in the release of small quantities of tryptophan which was probably used for synthesis of indole-3-acetic acid.

An understanding of the resistant nature of hosts to white rust is provided by histological studies of the sequence of infection events. Williams and Pound (1963) found two types of resistance responses in radish. A variety, China Rose Winter, displayed a hypersensitive reaction in which the initiation of resistant mechanisms was induced by the penetration of the fungus. Histological examinations showed that as the hyphae grew in intercellular spaces and the haustoria were produced, cells surrounding

the hyphae were dead and suberized in response to the invasion. This subsequently brought about destruction of the fungus. In another variety, Round Black Spanish, no evidence was found that the fungus had actually entered the host, thus, the resistant mechanisms probably occurred on the outside of the host.

Verma et al (1975) also studied the infection process of A.candida in cotyledons of four Brassica species, B.napus, B.hirta, B.juncea and B.campestris. They found that initial stages of infection from zoospore encystment to formation of the first haustorium was the same in both resistant and susceptible species, and no morphological barrier was observed to interfere with these events. In the resistant B.napus the hyphae ceased to elongate when the first haustorium was formed. This haustorium was later enclosed by a thick encapsulation in the host cell, and subsequently, the death of the hyphae and haustorium occurred.

Races of the pathogen

Biological and morphological specializations have long been noted in A.candida and different classifications of races of the fungus have been proposed (Togashi and Shibasaki, 1934, Hiura, 1930, Napper, 1933). The classification established by Pound and Williams (1963) was based on host specificity, and has been accepted by most pathologists working in this area. They defined six races

according to the hosts on which the isolates were collected (Table 1). Race 1, 2 and 3 are to some extent economically important. Races 4, 5 and 6 attack their weed hosts. Race 7, designated by Williams, occurs predominantly on turnip rape (B.campestris) in western Canada (Petrie, 1975a). Delwiche and Williams (1977) found another isolate differing from the above seven races and designated it race 8.

TABLE 1

Classification of races of Albugo candida

Race	Host	Reference
1	Raphanus sativus	Pound and Williams
2	Brassica juncea	Pound and Williams
3	Armoracia rusticana	Pound and Williams
4	Capsella bursa-pastoris	Pound and Williams
5	Sisymbrium officinale	Pound and Williams
6	Rorippa islandica	Pound and Williams
7	Brassica campestris	Williams
8	Brassica nigra	Delwiche and Williams

Genetic studies of resistance of Brassica spp. to A.candida

Inheritance of resistance to A.candida has been investigated in several Brassica species. Hougas et al (1952) provided the first evidence of genetic control of white rust resistance in horseradish (A.rusticana). However, elucidation of the precise pattern of the inheritance was not possible because of male sterility in Common horseradish. The resistant variety, Bohemian horseradish, did occasionally produce some functional pollen, which offered a possibility of making crosses with susceptible Common horseradish and of self-pollination. Reactions of F1 which segregated and of selfed progenies to white rust were divided into three categories, none, restricted and abundant sporulation. Since the progenies withstood the disease, the dominant nature of the resistance was suggested.

In radish, Williams and Pound (1963) found two resistant varieties, China Rose Winter (CRW) and Round Black Spanish (RBS), from 14 commercial varieties and 283 accessions in a screening program for the resistance to race 1. Crosses of both varieties were made to a susceptible one, Red Prince. Although genetic data revealed that the resistance was controlled by a single dominant gene in both CRW and RBS, their resistant reactions were different. Histological examinations of these two types of resistance has been mentioned (section on physiological studies). Furthermore,

the expression of the resistant gene in RBS was stable and was not influenced by environment. Shifts from resistance to tolerance defined as limited sporulation appeared in CRW as conditions in which plants were grown changed. This suggested the existence of some environmentally sensitive minor genes that may modify the action of the major resistant gene in CRW.

The same result of monogenic dominant resistance in CRW was also obtained by Humaydan and Williams (1976). This gene was symbolized by Acl which are the initials of Albugo candida race 1. Acl is closely linked to the gene, Pi, that conditions the expression of pink pigmentation in the radish plant.

A.candida race 2 infects mainly the species B.juncea and screening for resistant sources has been carried out. Parul and Bandyopadhyay (1973) found that a strain from their collection, Yellow rai T-4, was virtually immune to white rust under natural conditions. Ebrahimi, et al, (1976) reported that the accession, P.I.347618, was resistant to race 2, and after selfing it for two generations, true breeding resistant lines were obtained. F1 progenies of crosses between resistant and susceptible plants gave reactions similar to those of the resistant parents. However they did not provide the results of F2 progeny tests. Failures to find resistance in B. juncea were also reported (Delwiche and Williams, 1974, Bains and JHooty, 1979,).

B.nigra, B.campestris and B.carinata are also infected by race 2. Genetic studies showed that a dominant gene was involved in the resistance in these species (Delwiche and Williams, 1974, 1976,).

Breeding for white rust resistance

Although the demage caused by white rust can be substantially reduced by means of chemical control (Dueck and Stone, 1979), incorporation of white rust resistance into desirable commercial cultivars is apparently a practical measure for control of this disease. Previously described genetic studies of white rust resistance have provided information for breeding in this area .

In B.juncea, the limited resistant germplasm is a great barrier to using this approach within the species. Thus, introduction of the resistance from its related species becomes necessary (Delwiche and Williams, 1974). Williams (1978) reported that resistance to race 2 had been transferred from B.nigra and B.campestris to B.juncea.

Instead of transferring resistance by hybridization, Delwiche and Williams (1976) used mutants, either naturally occurring or induced by ethyl methanesulfonate treatment, for the selection, and resistant plants of B.nigra which could be used in breeding programs were found.

MATERIALS AND METHODS

Parental strains

B.napus is an amphidiploid species. Its genomes are derived from *B.campestris* and *B.oleracea* (Olsson and Ellerstrom, 1980).

The resistance to the white rust is well known in *B.napus* cultivated in Canada. Regent, a variety currently grown in western Canada, was chosen as a resistant cultivar in the study. It is a summer form of rape and symptoms of the white rust were never observed on it during the period of cultivation.

Several rape strains obtained from China were planted in the field nursery and in the greenhouse in 1981. They were all susceptible to the white rust under natural or artificial inoculation. From these collections, 2282-9 and Green cup Leaf (GCL) were selected for study. 2282-9 was a breeding line originally selected for yellow seed and had been self-pollinated for four generations. GCL was a variety grown in eastern China. The reactions of 2282-9 and GCL to white rust were somewhat different. Pustules occurring on GCL were of small size, usually surrounded by chlorotic rings, and seldom merged to form patches. However, 2282-9 had pustules coalesceing frequently into patches, and chlorotic rings sometimes occurred. However,

these differences sometimes were vague, since two reactions could appear on different leaves of same plant, or even on the same leaf, especially in GCL.

Hybrid populations

The experiment was conducted in the greenhouse during the period of 1981-1982. Crosses and reciprocals of Regent with 2282-9 and GCL respectively were made on five plants of each entry. Vernalization was applied to 2282-9 and GCL because they were winter forms of rape. To match their flowering time with that of Regent, they were planted one month earlier than Regent. After testing for white rust resistance at approximately the 3-4 leaf stage, the plants were placed in the cold room at 4° C for four weeks, then transfered to the greenhouse. The crosses were made by hand. The inflorescences on which the crosses were made were bagged to prevent contamination with any foreign pollen. The bagging was also used to self-pollinate.

Approximately fifty F1 plants of each cross were backcrossed to their susceptible parents, and F1 plants were self-pollinated to obtain F2 progenies.

The cross and reciprocal between 2282-9 and GCL were also made to determine whether the genes they carried were allelic.

Inoculation

The inoculum used in the study were zoosporangia of A.candida collected from Torch (B.campestris) grown in the field in the summers of 1981 and 1982. The zoosporangia were kept in capsules and stored in a freezer at temperature below -10°C for later use.

Suspensions of zoospores were prepared according to the method of Pound and Williams (1963). Sufficient zoosporangia were mixed with 150 ml sterile distilled water in a 250 ml flask. The flask was shaken vigorously to obtain an even suspension of the zoosporangia, then placed in an incubator at 12°C for 6-8 hours. The suspension had a high concentration of the zoospores ready for inoculation.

Plants to be tested were grown in flats with the mixture of soil, sand and peat in 2:1:1 ratio. When the second leaf expanded, approximately three weeks after seeding, the plants were inoculated with the zoospore suspension by spraying. Immediately after the inoculation, the flat was transferred to the mist chamber at 100% humidity with illumination for 16 hours. The temperature was 20°C during daytime and 15°C at night. The plants were maintained in this environment for three days, then moved to the growth chamber where they remained for 8-10 days until pustules appeared on leaves.

F1, F2 and backcross progenies were tested for white rust resistance. The parents were also grown in each flat as

controls. In addition, Torch was randomly planted in the flats to ensure that inoculation was satisfactory.

Data analysis

Resistance was defined as the absence of pustules, and susceptibility as the presence of pustules. The Chi-square test was used to evaluate the data from segregating generations.

RESULTS

F1 plants of crosses and reciprocals of 2282-9 X Regent and GCL X Regent gave the same reaction to white rust as their resistant parent (Table 2). Thus resistance in Regent is dominant and this characteristic is controlled by the nuclear genes. In the 2282-9 X GCL cross and its reciprocal, F1 progenies were all susceptible. Although an attempt was made to identify the reaction of F1 plants in relation to that of their parents, difficulty was experienced because variations in the response to white rust were found in 2282-9, in GCL as well as in F1 plants. Thus objective classification was not realized.

In the 2282-9 X Regent cross, F2 progenies segregated for white rust resistance in the ratio of 15 resistant to 1 susceptible (Table 3). The Chi-square test indicates satisfactory fit of the observed ratio to the expected ratio (P=.50-.70). From these data, a two gene system with dominance for each allele is involved in this cross. With this model, resistant plants would result from the presence of a dominant allele at any one of the two loci, and the susceptibility would be expressed when both alleles at both loci were homozygous recessive.

TABLE 2

Reaction of the F1 from crosses and reciprocals of 2282-9 X Regent, GCL X Regent, and 2282-9 X GCL to A.candida race 7

Cross	Reaction*	
	R	S
2282-9 X Regent	95	0
Regent X 2282-9	55	0
GCL X Regent	66	0
Regent X GCL	97	0
2282-9 X GCL	0	54
GCL X 2282-9	0	44

* R = Resistant, S = Susceptible.

TABLE 3

Observed segregation and Chi-square tests for F2 data from the cross 2282-8 X Regent involving resistance (R) and susceptibility (S) to race 7 of A.candida

	Reaction				
Accession*	R	S	Ratio	X^2	P
1	101	2	15:1	3.263	.05-.10
2	92	8	15:1	0.523	.30-.50
3	100	5	15:1	0.397	.50-.70
4	97	10	15:1	1.750	.10-.20
5	98	9	15:1	0.853	.30-.50
6	95	6	15:1	0.017	.70-.90
7	100	5	15:1	0.397	.50-.70
8	300	21	15:1	0.047	.70-.90
9	101	10	15:1	1.441	.20-.30
total	1084	76	15:1	8.689	
Deviation X^2				0.180	.50-.70
Heterogeneity X^2				8.509	.30-.50

*Each F2 population was derived from a single identified F1 plant, e.g. 101 R and 2 S plants were derived from the F1 Accession 1.

Progenies from the backcross (2282-9 X Regent) X 2282-9 segregated in the ratio of 3 resistant to 1 susceptible (P=.30-.50) (Table 4), and thus confirmed the digenic model with dominance.

In addition to the 15 : 1 ratio, some of the F2 progenies from the GCL X Regent cross segregated into a ratio of 63 resistant to 1 susceptible (Table 5). This indicates the existence of a third dominant gene conferring the resistance. In the GCL-4 X Regent-4 family, both ratios were found. This suggests a heterozygous state for Regent-4 at one of the three loci.

The identity of the F1 plants (Accession numbers 1 to 12) used to produce F2 and backcross seed was preserved so that F2 and backcross data could be traced to a particular F1 plant. In the cross involving GCL and Regent, progenies from eight F1 plants segregated in a 15:1 F2 ratio and a 3:1 backcross ratio while the progenies from four F1 plants segregated in a 63:1 F2 ratio and a 7:1 backcross ratio (Table 5 and 6). Since no resistant plants were recovered from the parents and progeny of cross between GCL and 2282-9, this result indicates that all Regent plants were homozygous and homogeneous for alleles for resistance to white rust at two loci and that some plants also have resistance genes at a third locus. Since genes for resistance at one locus are sufficient to confer resistance to the plant, variability at a third locus within the

cultivar Regent could not be detected until the progeny of appropriate crosses had been examined.

Seven hundred and seventy F2 plants from the 2282-9 X GCL cross were tested. They were all susceptible to white rust. In general, the F2 seemed to be more susceptible than the F1 and their parents in respect to leaf area infected, Variation in the disease reaction were also observed among the F2 plants.

TABLE 4

Observed segregation and Chi-square tests for backcross data from (2282-9 X Regent) X 2282-9 involving resistance (R) and susceptibility (S) to race 7 of A.candida

	Reaction				
Accession*	R	S	Ratio	X^2	P
1	87	20	3:1	2.271	.10-.20
2	78	29	3:1	0.252	.50-.70
3	80	22	3:1	0.641	.30-.50
4	88	26	3:1	0.292	.50-.70
5	76	30	3:1	0.616	.30-.50
6	77	29	3:1	0.314	.50-.70
7	80	25	3:1	0.079	.70-.90
8	244	75	3:1	0.377	.50-.70
9	80	25	3:1	0.079	.70-.90
Total	890	281		4.921	
Deviation X^2				0.629	.30-.50
Heterogeneity X^2				4.292	.70-.90

* The accession numbers correspond to those in F2, indicating that both populations were derived from the same maternal parent.

TABLE 5

Observed segregation and Chi-square tests for F2 data from the the cross GCL X Regent involving resistance (R) and susceptibility (S) to race 7 of A.candida

Accession	Reaction R	Reaction S	Ratio	X^2	P
(GCL-1XRegent-1)					
1	80	9	15:1	2.266	.10-.20
2	91	3	15:1	1.501	.20-.30
3	74	7	15:1	0.791	.30-.50
4	96	4	15:1	0.864	.30-.50
5	96	5	15:1	0.291	.50-.70
6	92	10	15:1	2.199	.10-.20
(GCL-4XRegent-4)					
7	93	3	15:1	1.600	.20-.30
8	98	7	15:1	0.031	.70-.90
9	91	1	63:1	0.135	.70-.90
10	103	2	63:1	0.080	.70-.90
(GCL-5XRegent-5)					
11	153	2	63:1	0.075	.70-.90
12	160	5	63:1	2.311	.10-.20

For 15:1 ratio, deviation X^2= 0.000 (P =.95-1.00), heterogeneity X^2= 9.543 (P =.20-.30). For 63:1 ratio, deviation X^2= 0.464 (P =.30-40), heterogeneity X^2= 2.037 (P =.50-.70).

TABLE 6

Observed segregation and Chi-square tests for backcross data from (GCL X Regent) X GCL involving resistance (R) and susceptibility (S) to race 7 of A.candida

	Reaction				
Accession*	R	S	Ratio	X^2	p
1	35	14	3:1	0.333	.50-.70
2	80	28	3:1	0.049	.70-.90
3	73	21	3:1	0.355	.50-.70
4	75	29	3:1	0.462	.30-.50
5	60	22	3:1	0.146	.70-.90
6	83	20	3:1	1.712	.10-.20
7	85	18	3:1	3.110	.05-.10
8	81	23	3:1	0.462	.30-.50
9	92	10	7:1	0.678	.30-.50
10	91	13	7:1	0.000	.95-1.00
11	89	18	7:1	1.828	.10-.20
12	95	16	7:1	0.372	.50-.70

* The accession numbers correspond to those in F2, indicating that both populations were derived from the same maternal parent.

For 3:1 ratio, deviation X^2= 0.986 (P =.30-.50), heterogeneity X^2= 5.643 (P =.50-.70). For 7:1 ratio, deviation X^2= 0.345 (P =.50-.70), heterogeneity X^2= 2.533 (P =.30-.50).

DISCUSSION

Zoosporangia used as inoculum were collected from Torch grown in the field in the summers of 1981 and 1982. Ideally, zoosporangia from single pustules should be kept seperately and propagated in suitable conditions to produce a large amount of homogeneous zoosporangia for inoculation. However, the zoosporangia propagated in the growth chamber are usually immature and their germination is poor. A large quantity of homogeneous inoculum cannot be obtained by means of propagation of zoosporangia from a single pustule in the growth chamber. In the experiment, plants of Torch were equally susceptible to the inoculum collected in 1981 and 1982, and no difference in pathogenicity between the inoculum was observed. Differentiation in pathogenicity within a race of A.candida has not been reported. Thus, homogeneity of A.candida race 7 is assumed.

Throughout the experiment, Torch (B.campestris) used as an additional control was more susceptible than 2292-9 and GCL (B.napus) since a larger leaf area was infected in Torch plants than in 2282-9 and GCL. The two species may have different responses to white rust. One factor that may be involved in this difference is leaf wax deposits which reduce the adhesion of water droplets containing zoospores to the leaf surface. In 2282-9 and GCL, when the inoculum

suspension was spread on the leaf, the water was held in the areas at the edge of the leaf and in the proximity of the central vein. Pustules occurred later and mostly in these areas. Leaves of Torch could be completely covered by the inoculum suspension, and consequently, pustules could appear anywhere on the leaf. Tewari and Skoropad (1976) have provided evidence that epidermal cells of Torch have a very limited amount of cylindrical and plate-like wax crystals. They considered this as one of the reasons why Torch was more susceptible to Alternaria brassicae than Midas. Wilson and Jarvis (1963) also reported that susceptibility to A.candida of a glossy-leaved mutant of brussels sprout (B.oleracea var. Gemmifera) may be associated with the absence of wax extrusions. The difference in leaf surface characteristics between the two species may thus in part account for the difference in leaf area infected.

Based on their different reactions to white rust, 2282-9 and GCL were initially chosen in the study. It was later found that the dissimilarities between the two reactions that 2282-9 and GCL possessed were not conspicuous, and that variation in the disease reaction occurred among the plants. Thus, difficulty was encountered in classifying the reactions of F1 and F2 of 2282-9 X GCL cross according to those of their parents. The F1 and F2 progenies also varied in the disease reaction. In general, the F2 plants were more susceptible than the F1 as well as their parents as measured by leaf area infected. There may be some minor

genes modifying the susceptibility and sensitivity to changes of environment. While there were differences in the disease responses between 2282-9 and GC1, they and their F1 and F2 progenies were susceptible. This suggests that the genes carried by 2282-9 and GCL are allelic.

The resistance of Regent to white rust is conditioned by two or three gene loci and the genes for resistance are dominant. These genes segregated independently, and presence of a dominant allele at any one of the three loci results in the resistance. Homozygous recessives at the three loci lead to susceptibility. According to the proposal of Humaydan and Williams (1976), these three genes are designated Ac7-1, Ac7-2 and Ac7-3.

The heterogeneity of the Regent population at certain white rust resistance loci is interesting. As seen in the Table 5 and 6, some plants carried resistant genes at two loci , but others resistant genes at three loci. Due to the limited number of plants used in the study, it is not possible to determine whether Regent is heterozygous at only Ac7-3 or at both Ac7-2 and Ac7-3. Ac7-1 must be homozygous in the population. If Ac7-1 is not, homozygous recessive susceptible plants would appear in the population since B.napus is a partially cross-pollinated species. However, this has never been observed. If Ac7-2 and Ac7-3 are both heterozygous, the genotype Ac7-1 Ac7-1,ac7-2 ac7-2, ac7-3 ac7-3 would be present in the population. When a plant of

this genotype is crossed to a susceptible one, ratios of three resistant to one susceptible and one resistant to one susceptible would be expected for F2 and backcross, respectively. In the present study, these segregating ratios have not been found. But the possibility is not excluded. It is most likely that Ac7-1 and Ac7-2 are homogeneous and mask the heterozygosity or heterogeneity of Ac7-3 in the population.

Since most cultivars of B.napus grown in Canada and Europe are highly resistant to white rust, it is surprising that many cultivars of this species in China are susceptible. Since B.campestris has been used in many programs to transfer earliness to B.napus cultivars in China, white rust susceptibility may also have been transferred from B.campestris to B.napus.

Rapeseed bas been cultivated in Canada for approximately 40 years. During this time, cultivars of B.campestris have been susceptible to white rust while the resistance in B.napus has remained effective. This study provides information on the genetic basis of this resistance which has not broken down during 40 years of exposure to the pathogen. The number of genes for resistance in the Canadian cultivars probably has helped to prevent the pathogen from overcoming the resistance. For this reason, Canadian breeders probably should be cautious about using cultivars of B.napus from the Orient or crosses with B.campestris in their breeding programs. Plant breeders in

China probably should also endeavor to introduce white rust resistance genes at more than one locus to ensure that the resistance will be as stable as possible.

SUMMARY

The inheritance of reaction to white rust (A.candida) was investigated in the progeny of crosses involving the B.napus cultivars, Regent, 2282-9 and GCL. The Canadian cultivar, Regent, was resistant and the Chinese strains, 2282-9 and GCL, were susceptible. The white rust resistance was controlled by three independent dominant genes, Ac7-1,Ac7-2 and Ac7-3. A dominant allele at any one of the three loci results in the resistance. The susceptible has recessives at the three loci.

The population of Regent was heterogeneous at one or more of the loci involved in white rust resistance. It is suggested that Ac7-1 and Ac7-2 are homogeneous, and mask the heterogeneity of Ac7-3 in the population.

The Chinese strains 2282-9 and GCl were consistently less susceptible than B.campestris cultivar Torch. This may partially be explained by greater deposits of wax on the leaf surface in B.napus.

Minor differences in the disease reaction were observed between 2282-9 and GCL. And variations in the disease reaction were also found within 2282-9 and GCL, as well as their F1 and F2 progenies. There may be some minor genes modifying the susceptibility. However, the major recessive genes in 2282-9 and GCL are allelic.

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