

EVALUATION OF RESISTANCE TO RUST
(Uromyces viciae-fabae) IN FABA BEAN
(Vicia faba) AND PATHOGENIC VARIABILITY
IN Uromyces viciae-fabae

A Thesis
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Robert Lorne Conner

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FOREWORD

The format adopted for this thesis deviates from the conventional in that the materials, methods and results are presented in the form of four publications, the style of which complies with the requirements of either the Canadian Journal of Plant Pathology or Phytopathology. A general discussion follows and the thesis terminates with the bibliography and appendices. This format has been approved by the Council of the Faculty of Graduate Studies of the University of Manitoba.

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GENERAL ABSTRACT

Conner, Robert Lorne. Ph.D., The University of Manitoba, February, 1981.

Evaluation of Resistance to Rust (*Uromyces viciae-fabae*) in Faba Bean (*Vicia faba*) and Pathogenic Variability in *Uromyces viciae-fabae*.

Major Professor: C.C. Bernier.

Rust [*Uromyces viciae-fabae* (Pers.) Schroet.] isolates from species of *Vicia*, *Lathyrus* and *Pisum* were used in cross-inoculation studies on a total of 20 *Vicia* spp., 10 *Lathyrus* spp., one *Lens* sp. and one *Pisum* sp. The host ranges of these isolates were much larger than previous studies of isolates from Japan and Europe had indicated. Rust isolates with distinct host ranges were found to share so many hosts in common that it was not possible to separate them into discreet formae speciales. Isolates from native species of *Vicia* and *Lathyrus* were able to infect one or more of the crop species *Vicia faba* L., *Pisum sativum* L. and *Lens culinaris* Medic., and thus could play an important role in the epidemiology of this disease.

A number of rust isolates were tested for differences in virulence patterns on nine faba bean inbred lines and on 16 pea cultivars. Rust isolates from *V. faba* could be divided into two races on the basis of their reactions on the inbred lines of *V. faba* and pea cultivars. Isolates from *Vicia americana* Muhl. reacted the same on the inbred lines as race 1 of the faba bean rust isolates. Pea rust isolates behaved as a third race on the pea cultivars. Several races were detected

amongst isolates of Lathyrus venosus Muhl. and an isolate from Lathyrus ochroleucus Hook. was avirulent on all the faba bean inbred lines.

Several faba bean cultivars were found to be almost uniformly susceptible to race 2 and quite heterogeneous in their reaction to race 1. With a few exceptions the pea cultivars reacted uniformly to most rust isolates.

F₂ and backcross data from crosses between nine inbred faba bean lines revealed the presence of three different dominant genes for resistance. The gene Fr1 found in lines Ack-11 and 2N12-2 provided resistance only to race 1 (isolate SS3). A second gene, Fr2, for resistance to race 1 was identified in lines Erf-1-4 and MB-1-2. An additional homozygous gene, Fr3 in line MB-1-2, conditioned resistance only to race 2 (isolate SP51). Heterozygous resistance genes in Erf-1-4 and MB-1-2 had their segregation masked by the homozygous resistance genes. Resistance genes controlling resistance to both isolates were still heterozygous in lines D-50-7 and HF-13-4 after three generations of inbreeding and selection for resistance.

Twenty-five open pollinated faba bean lines were evaluated in the field for differences in the ability to retard rust development. The incidence and severity of rust were assessed at weekly intervals in replicated plots with rust spreader rows at each end. In all three years of the study, significant differences were found between lines for values of area under the disease progress curve (AUDPC), even though within-line variation was high. Three lines consistently had low AUDPC values and another six lines had AUDPC values that shifted from low to intermediate. The other lines either behaved inconsistently or were

fast rusters.

Final rust severity was well correlated to AUDPC in each year of this study. Measurements summarizing the incidence of infected plants over time and the percentage of infected plants did not appear to be related to AUDPC.

GENERAL INTRODUCTION

The occurrence of Uromyces viciae-fabae (Pers.) Shroet. is known to be world wide (Guyot 1957). In certain areas this rust is considered one of the most serious fungal diseases of faba beans (Vicia faba L.) (El Helaly 1939). Williams (1978) found that severe early rust infestations could result in yield losses as high as 45%. Since the introduction of faba beans to Western Canada in 1968, rust has been reported at several locations in Manitoba and Saskatchewan (Bernier 1975, McKenzie and Morrall 1975).

This rust also infects crops such as peas (Pisum sativum L.) and lentils (Lens culinaris Medic.) as well as certain wild and cultivated species of Vicia and Lathyrus (Connors 1967, Guyot 1957). Studies in Europe and Japan indicate several formae speciales exist within this rust species (Gaumann 1934, Hiratsuka 1933). Similar studies with isolates from hosts native to North America have never been conducted.

A lack of uniformity within faba bean cultivars has hampered efforts to identify races of Uromyces viciae-fabae and sources of resistance in V. faba (Kispatic 1944 and 1949).

This study was initiated to gain a better understanding of the variability that exists for host range and race composition within this rust in Manitoba. The genetic basis of rust resistance in selected inbred lines of V. faba was examined and methods for the detection of slow rusting resistance in faba bean lines were also evaluated.

LITERATURE REVIEW

1. Nature of the Host

1.1 Center of Origin and History in Western Canada

Vicia faba L. is thought to have originated in the Near East (Cubero 1974, Zohary and Hopf 1973). From this center of origin it is believed to have spread throughout Europe, Northern Africa and into Asia. Areas such as Ethiopia and Afghanistan are considered by some to be secondary centers of diversity (Bond 1976).

The ancestry of V. faba is unknown; Vicia narbonensis L. was once thought to be the ancestor of this crop but cytogenetic evidence has shown these two species to be widely divergent (Zohary and Hopf 1973). Vicia galilea Plitm. et Zoh. has also been proposed as an ancestor of V. faba because of its resemblance to V. faba. However, Ladizinsky (1975) found the albumin profiles of seed protein from V. galilea, V. narbonensis and Vicia hyaeniscyamus Mout. had little in common with the profile of V. faba. This, along with the fact that these species are cross incompatible with V. faba all suggests that these species are not the ancestors of faba beans.

Cultivation of V. faba is thought to have started in the late Neolithic era (Zohary and Hopf 1973). Its use has been largely confined to temperate zones and to the cool season in subtropical areas. This crop is grown extensively throughout the Middle East and China, also

to a lesser extent in Southern Europe (Hawtin and Stewart 1979).

V. faba has been divided into three subspecies on the basis of seed size (Evans and Slinkard 1975, Robinson 1968). The subspecies major, equina and minor include large, medium and small seed types respectively. Subspecies major is used primarily for human consumption while the two smaller seed types are grown chiefly as grain legumes for livestock feed.

Faba beans were first grown commercially in Western Canada in 1970 with the aim of providing a regional source of protein for livestock (Evans and Slinkard 1975). More recently, it has been found to be an excellent silage crop and its ability in combination with suitable rhizobia to fix nitrogen also makes it a good green manure crop. The subspecies equina and minor have been used exclusively because their small seed size make them better suited for handling with conventional farm machinery. Field trials have shown this crop to be well adapted to the moister growing areas of the prairies (Evans et al. 1972). Current research programs are aimed at improving traits such as shatter resistance, earlier maturity and disease resistance (Furgal and Evans 1976).

1.2 Faba Bean Uniformity

Studies on the breeding system of V. faba indicate that quite a high degree of cross-fertilization can occur. Estimates on the percentage of cross-fertilization have ranged between 19-79% (Poulsen 1975, Toynbee-Clarke 1974).

A number of explanations have been advanced to account for the large disparity found in outcrossing values. Factors such as the amount of bee activity, flower colour and flowering time have all been

found to influence the level of cross-fertilization (de Vries 1978, Poulsen 1975). Plant density may also have an effect on the degree of cross-fertilization, although the evidence is conflicting (Holden and Bond 1960). Poulsen (1975) reported that earlier formed flowers tend to outcross more than later ones, the latter ensuring seed production if bee-activity is low.

The plant may have several mechanisms which favor cross-pollination, such as floral tripping requirement (Rowlands 1960, Toynbee-Clarke 1974). Rowlands (1960) also proposed that V. faba may have some type of self-incompatability system controlled by a number of deleterious recessive genes. There is little evidence to support this theory although it is generally recognized that fertility declines after several generations of inbreeding even when there is selection for self-fertility (Holden and Bond 1960). Contrary to this view, Poulsen (1975) suggests that fertility declines for the first few generations of selfing and later levels off as the genes controlling auto-fertility become fixed.

Most estimates on the level of cross-fertilization within V. faba are based on the results of studies using inbred lines with recessive markers. This tends to exaggerate the amount of outcrossing since inbred lines would outcross more and inbred progeny may have a higher mortality rate than outcrossed progeny (Holden and Bond 1960). A better approach to estimate the level of outcrossing has been to study changes in the frequency of some simply inherited trait within a population surrounded by another population where the frequency of this trait is vastly different.

In order to maintain faba bean selections, an understanding is required of the isolation distances needed to limit the amount of

cross-fertilization. Bond and Pope (1975) found that contamination decreased rapidly over short distances. An isolation distance of 134 m reduced cross-fertilization to 1-2% but low levels of contamination could still occur at distances of 1.1 Km or more.

1.3 Breeding Strategies

Most faba bean breeding programs are based on variations of either pure line selection, mass selection or recurrent selection.

The pure line and mass selection methods may be useful for the improvement of simply inherited traits but have not been successful for the improvement of quantitative traits such as yield (Nassib et al. 1978). This failure is likely due to a combination of inadequate pollination control and inbreeding depression (Hawtin 1978). Hawtin (1978) suggests that active selection for auto-fertility might overcome the problem of inbreeding depression, whereas Nassib et al. (1978) feel that this is not likely to be effective. Bond (1979) found inbreds to be influenced more by environment than were synthetics. This is in agreement with Byth and Weber (1978) who found a greater host-environment interaction for homogeneous populations of soybean Glycine max (L.) Merr. than for heterogeneous populations. This greater sensitivity to the environment could cause the yields of pure lines to fluctuate much more than that of synthetics (Bond 1979). The pure line or mass selection breeding schemes could narrow the genetic base of faba beans (Nassib et al. 1978) and may result in greater vulnerability to disease (Horsfall et al. 1972). Demands for better uniformity for agronomic traits such as tannin free seeds and shattering resistance could also lead to increased disease vulnerability.

Recurrent selection has been very successful in developing several faba bean cultivars in the United Kingdom (Bond 1979). The advantage of recurrent selection is that it permits the rearrangement of genes within linkage groups, allowing better combinations to be accumulated. Recurrent selection appears to be a logical approach in an outcrossing crop. However, use of such a program to improve disease resistance has so far not been examined. Presently, attempts are being made to compare the ability of these breeding systems to improve disease resistance and certain agronomic traits in faba beans (Hawtin 1978).

2. Nature of the Pathogen

2.1 Rust Taxonomy

There has been considerable disagreement on the naming of faba bean rust; most early works refer to it as Uromyces fabae (Pers.) de Bary based on a description of Uredo fabae by Persoon in 1794 (Laundon and Waterston 1965).

Most recently the most widely accepted name of this rust has been Uromyces viciae-fabae (Pers.) Schroet. based on Uredo viciae-fabae Pers. described in 1801 (Personal communication of G.B. Cummins to C.C. Bernier, Cummins 1978).

Boerema and Verhoeven (1979) feel this is incorrect since Persoon only described the uredia and not the telia. They suggest the name Uromyces fabae (Grev.) de Bary ex Fuckel based on Puccinia fabae Grev. 1821 has priority. Cummins and Stevenson (1956) have also used this name to describe this rust.

Laundon (1968) puts forth an argument in favor of the name Uromyces viciae-fabae (Pers.) Schroet. He agrees that Persoon never described the

teliospores but believed that he did in fact describe the telia. He felt that Persoon's reference to the dark colour of certain sori must have been describing sori containing teliospores. Jorstad (1958) found uredia and telia in the same type specimen that Persoon had used in his description of Uredo viciae-fabae (Boerema and Verhoever 1979). For these reasons Laundon (1968) felt that the possibility could not be excluded that Persoon had actually seen and described the telia (in agreement with article 59 of the International Code of Botanical Nomenclature). Since Uromyces viciae-fabae is the oldest validly published name based on telia after 1801, it takes precedence (Article 13 I.C.B.N.). This name has generally been accepted and is presently used almost exclusively (Cummins 1978, Cummins 1962, Laundon and Waterston 1965, Wilson and Henderson 1961).

2.2 Importance of Faba Bean Rust

Uromyces viciae-fabae is considered by Wilson and Henderson (1961) to be one of the most geographically widespread members of the Uredinales. It is known to infect a large number of members in the tribe Viciae (Guyot 1957).

El Helaly (1939) reported that rust is the most destructive fungal disease of faba beans in Egypt. It has also been found to be very damaging on lentil (L. culinaris) (Gupta 1974, Nene et al. 1975). Rust attacks the leaves and stems and when severe enough, can result in partial defoliation, stem distortion and even death (Gupta 1974). Rust occurs in many areas on a regular basis, being most destructive where fields are irrigated or rainfall is high (El Helaly 1939, Gerard 1960, Kaiser 1967, Money 1965, Williams 1978).

Williams (1978) studied the influence of different levels of rust

severity on the yield of two broadbean cultivars. He found a linear relationship between yield loss and rust severity at the pod filling stage. When rust was most severe, yields were reduced by as much as 45%. These yield losses appeared to be almost entirely due to a reduction in weight of seeds per pod, an observation which is similar to those made by El Helaly (1939).

The incidence of rust on faba beans in Western Canada has been quite low. McKenzie and Morrall (1975) found rust in only one out of 28 fields surveyed. Several introduced faba bean lines which have only been grown on an experimental basis were severely attacked by rust (Bernier 1975, McKenzie and Morrall 1975). At present, rust is generally regarded as a potential problem for faba bean production in Western Canada (McKenzie and Morrall 1975).

2.3 Biology and Life Cycle of Rust

Uromyces viciae-fabae is a eu-autoecious, macrocyclic rust.

Descriptions of the life cycle and various spore types have been given in numerous sources (Arthur 1934, Butler and Jones 1961, Cummins 1978, Laundon and Waterston 1965, Wilson and Henderson 1966). The thick-walled, single-celled teliospores allow the rust to survive periods of adverse weather conditions such as extremely low or high temperatures (Gupta 1974, Kispatic 1949, Money 1965, Prasada and Verma 1948). In some parts of the world, such as the mediterranean area, this rust has been reported to persist the year round in the uredial stage (Kispatic 1949). This rust is also known to produce amphispores (McKenzie and Morrall 1975) which allow it to survive periods of high temperatures. These dark-coloured, thick-walled uredospores are similar in appearance to teliospores

differing only for germ pore numbers (McAlpine 1906).

Soon after teliospores are produced they can germinate and produce basidiospores with no evidence of any dormancy (Kapooria 1971, Kispatic 1949, Prasada and Verma 1948).

The pycnial and aecial stages are found only infrequently on faba beans and peas (Butler and Jones 1961, Gupta 1974). Studies on the sexual behavior of this rust have shown it to be heterothallic (Brown 1940, Prasada and Singh 1975). Aecia form only when pycniospores from a pycnium of opposite mating type are applied to the pycnium or when adjacent pycnia coalesce (Brown 1940, Prasada and Singh 1975). This rust has a short-cycling tendency which allows uredia to form on aecial mycelium without aecia ever being produced (Brown 1940).

Generally, aecia are produced only early in the season just prior to the uredial stage (Gupta 1974, Kispatic 1949) but repeating aecia have been reported. Prasada and Verma (1948) found that the production of secondary aecia continues throughout most of the season in India, causing severe damage on lentils. It may be that the rust Prasada and Verma (1948) described was Uromyces ervi West. which infects certain species of Lens and Vicia and is known to produce secondary aecia (Hiratsuka 1933, Laundon and Waterston 1965).

The uredial stage is the most destructive phase, attacking the leaves, stems and also the pods of certain hosts. Kispatic (1949) found that depending on the temperature, between six to nine uredospore generations could occur per season in Yugoslavia.

The temperature requirements of this rust has been studied in both Japan and Yugoslavia. The reported results were not always in agreement, possibly because of differences in local adaptation of

rust isolates (Hiratsuka 1934, Kispatic 1949). The optimum temperature range for germination and infection of uredospores was reported by Kispatic as 16-20°C and as 14-24°C by Hiratsuka. Kispatic found germination of aeciospores had a lower optimum temperature that ranged from 11-16%. Temperatures above 28°C prevented both germination and infection. Both workers found that a small amount of infection could occur at 0°C and rust mycelium in infected leaves could withstand temperatures of several degrees below 0°C.

3. Host Species - Pathogen Interactions

3.1 Formae speciales Concept

The term formae speciales is defined as a subdivision of a species of a parasitic or symbiotic micro-organism distinguished primarily by their adaptation to different hosts (Talboys et al. 1973). This concept has been used primarily for obligate parasites since obligate biotrophy demands a high degree of host specificity (Brian 1975). Formae speciales are thought to have resulted from a parallel evolution of host and pathogen, which has resulted in rusts which are more specialized and ecologically isolated (Brian 1975, Green 1971).

Host range studies within centers of diversity for host-parasite systems generally indicate a much wider scope of host specialization than do similar studies outside these areas. Studies with Erysiphe graminis D.C. in Israel, an area which is part of its center of diversity, indicate that these isolates have very large host ranges (Wahl et al. 1978). Certain isolates were able to infect as many as 30 genera representing several tribes. The native grasses of Israel were found to be quite important in the epidemiology of powdery mildew.

A similar study in North America showed that there was some overlap in the host range of Puccinia helianthi Schw. isolates taken from seven different Helianthus spp. (Zimmer and Rehder 1976). All the P. helianthi isolates shared one or more hosts in common with at least one other isolate and this would permit an exchange of virulence genes between the various isolates. It was concluded that the inability of host specificity to restrict gene flow could allow wild sunflower species to serve as breeding sanctuaries for new virulent races.

The ability of certain formae speciales to intercross has been demonstrated in several systems. Hiura (1978) found that while not all formae speciales of Erysiphe graminis intercross, at least three could. In less compatible combinations, hybrid cleistothecia formed but they produced few ascospores and often these ascospores failed to germinate. Where crossing did take place, cross compatibility was controlled by a single pair of genes which is typical of the heterothallic mating systems of Erysiphe graminis. From crosses of f. sp. tritici and f. sp. agropyri, hybrids were produced that were virulent on wheat or wheatgrass. Cultures that could infect both hosts were often low or intermediate in aggressiveness.

There is evidence of intercrossing between formae speciales of Puccinia graminis Pers. Watson and Luig (1959) found that somatic hybrids could form between f. spp. secalis and tritici. Usually the hybrids were intermediate in pathogenicity but some were more virulent on rye than their secalis parent and a number were virulent on several of the standard wheat differentials. They also found that by inbreeding f. sp. secalis, isolates were obtained that could infect wheat (Watson and Luig 1962).

Luig and Watson (1972) later concluded that the inability of hybrids of two formae speciales to compete successfully with the better adapted formae speciales would effectively limit their importance on cereals. However, on the native grasses of Australia, hybrids of the formae speciales tritici and secalis were found to be very common.

Green (1971) found a low rate of interfertility in crosses of Puccinia graminis f. spp. tritici and secalis. All the hybrids were low in virulence on both rye and wheat but could readily infect barley, a host common to both formae speciales. Based on these results he concluded that virulence was controlled by such a large number of genes that hybrids would likely be of no importance.

Hiura (1978) drew similar conclusions from his studies on formae speciales of Erysiphe graminis. He suggested that virulence and aggressiveness were controlled by more than five genes but unlike Green (1971), he found hybrids which could attack either wheat or wheatgrass as well as the parent cultures. The ability to infect both hosts was associated with low infection types and poor survival. Hiura speculates that this is due to the breakup of linkage groups needed for aggressiveness on wheat or wheatgrass.

3.2 Host Range of Uromyces viciae-fabae

Plowright (1889) was the first to examine the host specialization of Uromyces viciae-fabae. His work, and the work of several others, was later summarized by Gaumann (1934) (Appendix 1). Gaumann proposed six formae speciales in Uromyces viciae-fabae and three formae speciales in Uromyces orobi (Pers.) Lev. Uromyces orobi was considered to be a different species from U. viciae-fabae based on differences in thickness

of the uredospore wall. This was later judged to be a poor taxonomic trait so U. orobi is now considered to be synonymous to U. viciae-fabae. So, according to Gaumann's system, there are at least nine different formae speciales in Uromyces viciae-fabae (Appendix 1).

Later work by Kispatic (1949) in Yugoslavia showed that what Gaumann had designated f. sp. viciae-fabae had a much wider host range than had been shown previously (Appendix 2). In India, rust isolates were found which shared hosts of several formae speciales of both U. viciae-fabae and U. ervi described by Gaumann (Kapooria and Sinha 1966, Kapooria and Sinha 1971). Unfortunately, the original host(s) of these isolates were not mentioned.

With the exception of the study conducted by Hiratsuka (1933), all the reports on host range have been based only on one or two collections of rust. Even in the case of Hiratsuka (1933), most collections were made in the Tottori area of Japan and often only one collection was made from each host. Isolates were usually not tested in a systematic fashion, so many of the hosts of one formae speciales were never tested against isolates of supposedly different formae speciales.

The possibility of heterogeneity within host species was not considered in studies with this rust but recent studies on Erysiphe graminis (Wahl et al. 1978) and Puccinia helianthi (Zimmer and Rehder 1976) have shown that host populations often do not react uniformly.

In North America, U. viciae-fabae has been reported on nine species of Vicia, four species of Lathyrus and on peas (Anon. Index of Pl. Dis. 1960, Connors 1967). The relationships of the rust isolates from various hosts has never been examined in North America.

4. Host Cultivar - Pathogen Interactions

4.1 Resistance in Outcrossing Crops

A considerable amount of work has been done on the study of rust resistance in several outcrossing crops, the most important ones being corn and rye.

Early work on race identification for stem rust on rye was based on differential behavior of rust isolates on open pollinated rye cultivars. This approach allowed two or three races to be identified (Levine and Stakman 1923). However, this work was found to be difficult to repeat because of the heterogeneity within open pollinated cultivars (Tan et al. 1975). More recently, race identification has been based on reaction of selected inbred lines of rye which carry homozygous genes for rust resistance (Tan et al. 1975). Once problems with self incompatibility were overcome, it was found this method allowed detection of more races.

Rye inbred lines have been used in inheritance studies of resistance to rye stem rust (Tan et al. 1976). Crosses involving six inbred lines tested against three strains of rye stem rust, indicated the presence of at least six different resistance genes. A similar study using the same lines against two cultures of wheat stem rust allowed the identification of eight different genes for resistance (Tan et al. 1977). In both studies it was found that several lines with two or more resistance genes still had heterozygous resistance genes, even after 15 generations of inbreeding.

The same procedure has been used in studies on corn rusts Puccinia sorghi Schw. and Puccinia polysora Underw. (Futrell et al. 1975,

Wilkinson and Hooker 1968). The use of inbred lines in inheritance studies has allowed identification of at least six different resistance loci (Hooker 1977) and has enabled the study of multiple allelic systems (Saxena and Hooker 1974, Wilkinson and Hooker 1968).

4.2 Resistance to Uromyces viciae-fabae

Attempts have been made to identify races of U. viciae-fabae using faba beans, peas and lentils. Hiratsuka (1934) tested nine broad bean cultivars against five sources of rust but found no resistance. Kispatic (1944) proposed a scheme for rating infection types of rust reactions on V. faba. He tested 14 cultivars against single spore isolates from three locations but found that the lack of uniformity for rust reaction in the cultivars made it impossible to distinguish races.

Race identification has been more successful in self pollinated crops such as peas and lentils. Kispatic (1949) differentiated nine rust races based on their reaction on a number of pea cultivars. Sohi et al. (1974) tested 35 lines of peas to the natural inoculum in the area near Bangalore, India. Certain lines were less severely attacked than others suggesting they had some type of resistance.

Nene et al. (1975) tested between 129-157 lines of lentils to rust over two seasons and a few lines were found to be either immune or to have low terminal rust severity.

5. Breeding Strategies for Disease Control

5.1 Monogenic Resistance

Since the turn of the century, man has attempted to improve crop

production, largely through the use of pure line cultivars. The replacement of land races by pure line cultivars has resulted in a dramatic reduction in crop diversity (Marshall 1977, Knott 1972, Leakey 1970). Cultivars within a species are often closely related, each carrying the same gene or genes for disease resistance. In many instances, disease resistance over vast acreages is effectively controlled by only a single resistance gene conditioning an immune or hypersensitive response.

Monogenic resistance has allowed long term control of many diseases caused by slow spreading facultative parasites (Knott 1972). The effectiveness of such resistance has often been short lived for the control of diseases caused by obligate parasites (Johnson 1961). Single gene resistance disrupts the evolutionary balance between the host and pathogen, placing strong selection pressure on the pathogen population (Johnson 1961, Knott 1972). New races, produced by mutation and either sexual or somatic hybridization, are selected which overcome the host's resistance. In many host-pathogen systems, the repeated release of resistance genes has resulted in a step-wise shift in the pathogen population to more virulent races (Marshall 1977).

A lack of stable resistance, coupled with the monocultural system of crop production, has left many of the world's major crops very vulnerable to new disease outbreaks (Horsfall et al. 1972).

5.2 Gene Pyramiding

The repeated failure of monogenic resistance to provide long lasting disease control has led to the use of more complex forms of resistance. Multigene resistance is thought to extend the useful life

of the resistance genes, since it is more difficult for a single biotype of a pathogen to simultaneously acquire virulence against several genes (Coyne and Schuster 1975, Green 1975, Marshall 1977). Resistance in multigene lines might also be enhanced by the interaction of various resistance genes (Marshall 1977).

To be effective, the genes used in such complexes should not be released singly in other cultivars; this prevents stepwise increases in virulence which could destroy multigene resistance (Knott 1972).

The success of this type of gene management is dependent on the number of genes employed and the sources of variation in the pathogen population. Resistance would likely be less stable if the pathogens were able to recombine sexually or asexually, than if change was entirely due to mutation (Green 1975, Marshall 1977).

5.3 Multilines

The multiline concept has been proposed as an alternative to the traditional monoculture system, in an attempt to reintroduce diversity into crop cultivars (Browning et al. 1977). Multilines are composed of a number of host lines each carrying different resistance genes or combinations of resistance genes.

There is some disagreement on the actual number of effective resistance genes that should be included in any single component of a multiline cultivar. The "Clean crop" approach uses several resistance genes in each line to prevent any of the components from being attacked. If the pathogen population shifts, the susceptible components are removed and new ones are added. Losses caused by changes in race composition in any single year would not be great, since the remaining

resistant components would compensate for the diseased plants (Marshall 1977). This approach has been criticized as being gene wasteful, since it may not extend the useful life of any of the resistance genes.

The "Dirty crop" approach has been suggested as an alternative which might overcome some of the problems associated with the "Clean crop" approach (Knott 1972, Marshall 1977). In this case, only a single gene is used in each component and some disease will always be present. This system works by keeping the predominant races simple, since less fit complex races would compete poorly with simpler races. This could result in an extension of the useful life of the resistance genes.

In order for these strategies to be effective, the level of stabilizing selection must be high, otherwise complex races able to overcome all the resistant lines would eventually develop (Coyne and Schuster 1975). It has been pointed out that little work has been done to determine the actual level of stabilizing selection that exists in the field (Groth 1976). It has been proposed that the level of stabilizing selection must be quite high to offset the reproductive advantage associated with the ability of complex races to attack an increased number of components. Wolfe and Barrett (1980) disagree with this view and suggest that the effectiveness of multilines will gradually diminish only after beneficial modifying genes have been selected for in the more complex races.

There is also disagreement over how uniform the components should be. Wolfe and Barrett (1980) feel the production of isogenic lines is too time consuming and that only two or three generations of back-crossing are necessary to produce suitable cultivars. Others feel isogenic lines should be used to reduce the problem of certain components

predominating over time (Browning et al. 1977, Viera and Wilkinson 1972).

In theory, multiline cultivars act in two ways to delay epiphytotics. First, multilines reduce the initial amount of effective inoculum, since much of the inoculum lands on resistant components (Browning et al. 1977). Secondly, multilines reduce the rate of build up because most of the inoculum produced on multilines lands on adjacent resistant hosts. Tests with wheat multilines against Erysiphe graminis f. sp. tritici, found that only the initial inoculum was reduced (Fried et al. 1979).

Multilines may also reduce disease build up through the mechanism of induced resistance (Johnson and Allen 1975). Field tests have shown that prior inoculation with avirulent races can cause a slight reduction in subsequent infection by virulent races of stripe rust (Puccinia striiformis) on wheat.

The use of multilines has been criticized as being conservative, since it does not allow rapid improvement of agronomic traits and may result in a lack of uniformity for characteristics such as quality (Coyne and Schuster 1975, Knott 1972).

6. Slow Rusting

6.1 Terminology

A great deal of confusion and controversy has been associated with the terminology used in the description of this disease retarding resistance (Mortensen and Green 1978, Nelson 1978). As our understanding of the properties of this type of resistance has evolved, the use of certain terms has increased while other terms have fallen into disuse.

Terms such as horizontal resistance, generalized resistance, non-specific resistance and uniform resistance are now regarded as unsuitable since it is known that not all races are affected equally (Browning et al. 1977, Nelson 1978). Nonhypersensitive resistance, polygenic resistance and minor gene resistance are considered inappropriate since they suggest certain attributes of the mechanisms and genetics of this type of resistance, which have not yet been clearly demonstrated (Nelson 1978). The terms stable resistance and durable resistance infer that such resistance is long lasting and effective under all conditions. Johnson (1979) has pointed out that it is not possible to have absolute proof that any type of resistance will be long lasting. Cases are also known where the effectiveness of such resistance has been effectively diminished in different environments or by the appearance of new races (Johnson 1979).

The most widely accepted terminology for this type of resistance is based on its effect on disease epidemiology. The term slow rusting resistance imply only that such resistance retards rust development which is a trait that can be seen and measured.

6.2 Measurements

Slow rusting resistance acts not by reducing the amount of initial inoculum but rather by retarding epidemic development (Browning et al. (1977). Detection of this type of resistance usually requires that changes in rust severity be monitored over time.

Severity values over time are often summarized as infection rate (r) values (Vanderplank 1963) or as area under the disease progress curve (AUDPC) values (Wilcoxson et al. 1975).

Statler et al. (1977) found that significant differences for infection rates could be detected between durum wheat varieties exposed to Puccinia recondita Rob. ex. Desm. f. sp. tritici Eriks. and Henn. Infection rate has also been found to be correlated to yield losses (Burleigh et al. 1972, Statler et al. 1977).

Apparent infection rate is not considered to be the most reliable measure for comparing disease development on different host lines. Wilcoxson et al. (1975) found infection rate to be less consistent than AUDPC values, when comparing the ability of wheat cultivars to slow down the buildup of Puccinia graminis f. sp. tritici. Shaner and Finney (1977) found that infection rate values were very strongly influenced by minor differences in severity early in the season. Infection rate has also been found to vary with stage of plant development; however, high levels of variation make it unclear whether or not these differences are significant (Verma et al. 1975).

AUDPC is presently one of the most widely used values for comparing disease build up (Shaner and Finney 1977, Wilcoxson et al. 1975, Skovmand et al. 1978, Johnson and Wilcoxson 1979). AUDPC summarizes rust severity over time, allowing relative comparisons to be made between varieties. Such values can distinguish between lines with similar terminal rust severity values but which differ in their ability to retard rust development (Main 1977). Buchenau (1975) found that AUDPC correlated well with yield losses.

The advantages of AUDPC over infection rate are that it is not strongly influenced by minor differences in severity early in the season and that it also reflects variation in the rate of disease development

Shaner and Finney 1977). Since AUDPC and r are relative values, direct comparison cannot be made for different locations or for epidemics with different dates of disease onset.

The ability of the host to slow down disease development has also been studied by comparing rust severity values at some critical stage (Mortensen and Green 1978, Luke et al. 1975, Hooker 1967, Kim and Brewbaker 1976). Such measurements often do not correlate well with yield losses (Burleigh et al. 1972, Main 1977).

A modification of this approach has been to compare the time needed to reach a specific severity value (Shaner and Finney 1977). They feel such a value is superior to infection rate since it embodies both position and slope of the disease progress curve.

6.3 Components of Slow Rusting

A number of possible mechanisms have been suggested which either singly or in combination, might account for the slow rusting phenomenon. A list of these mechanisms includes decreased infection frequency, longer latent period, smaller pustules, lower sporulation rate and shorter infectious period (Johnson and Wilcoxson 1978, Kuhn et al. 1978, Milus and Line 1980, Neervort and Parlevliet 1978, Parlevliet 1978, Sztejnberg and Wahl 1978). It seems likely that some of these mechanisms are manifestations of the same phenomena. Certain of these traits appear to be associated but this may be only a partial association since they can act independently (Johnson and Wilcoxson 1978, Neervort and Parlevliet 1978).

Latent period has been studied extensively because it can be measured most reliably (Neervort and Parlevliet 1978). Latent period

was found to correlate well with performance in the field (Parlevliet 1975). Generally a better correlation with field performance was found when latent period was tested on adult plants rather than seedlings. While latent period was not found to be influenced by factors such as light intensity or inoculum dose, it was influenced by temperature and leaf age (Parlevliet 1975, Shaner et al. 1978). Latent period was found to decrease with leaf age, although it was not clear if these differences were significant (Parlevliet 1975). Other studies have not been able to detect significant differences in latent period between lines (Shaner et al. 1978, Sztejnberg and Wahl 1976).

Infection frequency, measured as pustule number per unit area, is a less reliable measure which is often associated with high coefficients of variation (Johnson and Wilcoxson 1979, Mortensen and Green 1978, Parlevliet 1976). This variability is due to difficulties in obtaining even inoculum deposition and uniform dew formation. Also, it would seem that tissue age should also be considered in such studies. These difficulties make it impractical for use in a breeding program (Kuhn et al. 1978, Mortensen and Green 1978).

Traits such as pustule size and amount of spore production tend to be related (Clifford 1972, Shaner et al. 1978). Krupinsky (1978) feels pustule size is under the control of a number of minor, additive genes. Both spore production and pustule size are influenced by infection density (Neervort and Parlevliet 1978, Shaner et al. 1978). Martin et al. (1979) disagree since they found that pustule density produced by different spore loads did not affect infection type. The relationship of these factors to the low infection types associated with specific resistance is unclear.

Little work has been done on examining the importance of length

of infectious period (Neervort and Parlevliet 1978). They found that where the infectious period was shortened, it was due to an increased latent period and earlier termination of spore production. High pustule densities were also found to result in a reduced infectious period. Coyne and Schuster (1975) suggest that earlier teliospore formation could be an important means of terminating spore production.

6.4 Influence of Environment on Slow Rusting Resistance

Ballantyne (1973) proposed that semitropical or tropical conditions may so favor the pathogen that the effect of slow rusting resistance may be nullified. Kim and Brewbaker (1976) found that corn lines considered to have adequate levels of slow rusting resistance when tested in the corn belt area of the U.S., had no effective resistance when tested in the severe epiphytotics of corn rust (Puccinia sorghi Schw.) that occur in Hawaii. However, certain other corn lines were still able to effectively reduce rust severity.

Southern (1978) found that wheat cultivars rusted much more rapidly under conditions of high rainfall and high levels of inoculum. Yet under these conditions, certain lines still significantly reduced rust severity.

6.5 Influence of Race Composition

The importance of the choice of rust isolates is often ignored in many slow rusting studies (Gavinlertvatana and Wilcoxson 1978, Luke et al. 1975, Statler et al. 1977, Verma et al. 1975). Frequently, naturally occurring inoculum or races of historical importance are used in such studies (Johnson and Wilcoxson 1979, Mortensen and Green

1978). This type of an approach fails to consider whether such rust sources can effectively overcome all the known resistance genes in the cultivars tested (Southern 1978).

Southern (1978) found that certain wheat cultivars carrying the resistance gene Sr 6 behaved as slow rusters in the presence of certain races, and as fast rusters when exposed to other races. This race specific response was due to the temperature sensitive nature of the Sr 6 gene, which allowed infection of avirulent races at high temperatures. The heat inactivation of the Sr 6 gene made lines carrying it appear to behave as slow rusters when exposed to races that normally are unable to attack them and as fast rusters when exposed to races which could readily overcome Sr 6.

In order to avoid complications caused by the presence of specific resistance genes in potential slow rusters, only races able to overcome all the known resistance genes should be used (Wilcoxson *et al.* 1975).

Little work has been done in comparing slow rusting responses to race mixtures as opposed to single isolates. Wilcoxson *et al.* (1975) found ranking of AUDPC values for cultivars to be more consistent when isolates were used singly rather than as mixtures.

6.6 Inheritance of Slow Rusting Resistance

It has commonly been suggested that slow rusting resistance is under polygenic control (Marshall 1977). It is also often stated that the minor genes controlling this trait have no relationship to genes conferring specific resistance. Others disagree, stating that the slow rusting phenomenon can be attributed to the same genes controlling specific resistance (Nelson 1978).

Inheritance studies have shown that the slow rusting trait has a high narrow sense heritability which ranges from 55 to 91%, depending on the degree of dissimilarity between the parents (Skovmond et al. 1978b, Luke et al. 1975, Krupinsky and Sharp 1978, Kim and Brewbaker 1977, Johnson and Wilcoxson 1979).

Estimates of the number of genes controlling slow rusting in various host-parasite systems have varied greatly. Gavinlertvatana and Wilcoxson (1978) estimated that between three to 21 genes controlled slow rusting to isolates of Puccinia recondita f. sp. tritici in the spring wheat (T. aestivum L.) cultivars tested. Luke et al. (1975) concluded that slow rusting in Red rustproof oats to Puccinia coronata was controlled by 2.16 genes; however, it is not clear if he was truly studying slow rusting resistance. Kim and Brewbaker (1977) reported that slow rusting resistance in lines of corn (Zea mays) to Puccinia sorghi Schw. appeared to be controlled by two genes. Skovmand et al. (1978b) estimated that slow rusting in spring wheat cultivars to stem rust isolates (Puccinia graminis f. sp. tritici) was controlled by three genes in some crosses and by as many as 12 gene pairs in other crosses.

The standard methods of gene number estimation give a very rough measure of gene number, which is considered by some to be only slightly better than no estimate at all (Falconer 1976). These estimates are regarded as very imprecise because it is unrealistically assumed that the effects of all loci are equal and no linkage is present.

Krupinsky and Sharp (1978) studied the genetic control of pustule size of isolates of Puccinia striiformis in wheat. The level of general combining ability was found to be quite high, so it was concluded that

the genetic control of this resistance was largely additive in nature. Unlike the previously described studies, the inheritance of this resistance was not studied in the field. It is not clear how this resistance differs from that of specific resistance which is known to produce low infection type. Other factors known to influence pustule size, such as inoculum dose, do not appear to have been considered in this study.

Nelson (1978) has suggested that specific resistance and slow rusting resistance are different expressions of the same resistance genes. When these genes are present alone or in small numbers, they confer resistance typical of specific resistance. When large numbers of these same types of genes are present, they produce a slow rusting response to races with virulence against all the accumulated resistance genes.

Martin and Ellingboe (1976) found that even when the powdery mildew resistance gene PM-4, in wheat, was overcome it still prevented the expression of a completely compatible relationship. The response was similar to mechanisms proposed for slow mildewing, thus causing reduced infection efficiency and a slower rate of development. This test included only a small number of isolates and it is not clear if these isolates are representative of all isolates that can overcome the PM-4 gene.

Skovmand et al. (1978a) was unable to find any clear relationship between slow rusting resistance in wheat and specific genes for resistance to stem rust. Certain genes such as Sr 6 and SrTt1 appeared to be linked to slow rusting genes but this relationship was not always consistent, suggesting the slow rusting character was not controlled by these genes. Other genes studied appeared to not be clearly

associated with slow rusting resistance, however, it was acknowledged that not all the genes for specific resistance present in the lines had been studied. Only genes expressed in the seedling stage were studied; the possible involvement of adult plant resistance was not considered.

More recently Sunderwirth and Roelfs (1980) have proposed that the slow rusting resistance reported in many of these wheat cultivars to stem rust, might be due to the adult plant resistance gene Sr 2. This gene is not expressed until around anthesis, and has been found to be effective against all known races. Cultivars carrying the Sr 2 gene acted like slow rusters by consistently producing fewer and smaller pustules. The relationship of these responses to performance in the field was never demonstrated.

6.7 Isolate Aggressiveness

Interest in slow rusting resistance is largely due to the assumption that such resistance is long lasting because it is race non-specific (Marshall 1977). However, in at least one case the effectiveness of a slow rusting cultivar was short lived (Johnson 1979). This and other studies, have led to the proposal that the pathogen might have the ability to slowly erode such resistance (Clifford and Clothier 1974, Parlevliet 1978, Priestley and Doling 1974).

Cultivar-race interactions have been found in several studies for traits such as latent period and spore production (Clifford and Clothier 1974, Milus and Line 1980, Mortensen and Green 1978, Parlevliet 1976, Parlevliet 1978). This has led to the suggestion that slow rusting resistance might be governed by a gene-for-gene system (Parlevliet 1978).

The existence of such a system could ultimately lead to selection for more aggressive isolates, if slow rusting cultivars were used on a large scale.

RESULTS OF RESEARCH

1. Host Range of *Uromyces viciae-fabae*

ABSTRACT

Rust [*Uromyces viciae-fabae* (Pers.) Schroet.] isolates collected from species of *Vicia*, *Lathyrus* and *Pisum* were used in cross-inoculation studies on a total of 20 *Vicia* spp., 10 *Lathyrus* spp., one *Lens* sp. and one *Pisum* sp. The isolates were found to have much larger host ranges than had previously been indicated for this rust. Rust isolates share so many hosts in common, that it was not possible to group them into discreet biological forms. Isolates found on native species of *Vicia* and *Lathyrus* could infect one or more of the cultivated crop species, *Vicia faba* L., *Pisum sativum* L. and *Lens culinaris* Medic. and so could play an important role in the epidemiology of this disease in Manitoba.

INTRODUCTION

Faba bean rust [Uromyces viciae-fabae (Pers.) Schroet.] attacks several important crops including faba beans (Vicia faba L.), peas (Pisum sativum L.) and lentils (Lens culinaris Medic.). Several species of Vicia and Lathyrus grown as ornamentals or as forage crops, as well as many wild species, are also infected by this rust.

This rust has been reported to infect a total of 52 species of Vicia and 22 species of Lathyrus (Guyot 1957). In Canada and the United States it has been identified on six species of Vicia (V. americana Muhl., V. angustifolia Reichard, V. caroliniana Walt., V. cracca L., V. faba and V. sativa L.) and on six species of Lathyrus (L. japonicus Willd., L. nuttallii Wats., L. ochroleucus Hook., L. ornatus Nutt., L. palustris L. and L. venosus Muhl.) (Anon. 1960, Connors 1967).

The host specialization of isolates of Uromyces viciae-fabae has been studied in Europe and Japan (Gaumann 1934, Hiratsuka 1933, Plowright 1889). Gaumann (1934) summarized most of the work done prior to his time and divided the rust into nine different formae speciales, each with a host range limited to two or three species (Appendix 1).

The host range of f. sp. viciae-fabae was found to be much larger when additional species of Vicia and Lathyrus were tested (Kispatic 1949) (Appendix 2). More recently, Kapooria and Sinha (1966, 1971) described isolates which were capable of infecting hosts included in the

host range of several formae speciales.

This study was undertaken to compare the relationship of rust isolates from native and cultivated hosts in order to determine the importance of native Vicia and Lathyrus spp. in the epidemiology of this disease in Canada. The results were related to previous works and to the formae speciales concept.

MATERIALS AND METHODS

Rust collections were made from P. sativum and several species of Vicia and Lathyrus at 10 widely separated locations in Manitoba and from infected peas received from Normandin, Quebec (Appendix 3). Thirteen single pustule isolates of rust from V. faba and four single pustule isolates of rust from P. sativum were used. All other isolates were handled as field collections. Isolates were increased on their original host, following standard procedures to prevent contamination (Appendix 4). Inoculum was collected with a cyclone spore collector (Browder 1971) and was then stored at 5°C for no longer than one month before use.

A total of 20 Vicia spp., 10 Lathyrus spp., one Pisum sp. and one Lens sp. were included in the host range tests. Two different selections for each species were used when seed was available. The sources of these selections are listed in Appendix 5.

Seed from these selections was scarified and placed in moist perlite. One week after emergence, seedlings were transferred into 7.5 cm paper jiffy pots containing a 2:1:1 mixture of clay loam: sand:peat. The jiffy pots were arranged in flats of 20 pots, each pot containing a different selection. Two weeks later, plants were sprayed with uredospores of a specific isolate suspended in a light oil (Soltrol 170, Philips Petroleum Co., Special Products Div.) using a spore-oil suspension atomizer (Browder 1971). After drying, the plants were placed in a chamber at 100% relative humidity for 24 h. Plants

were then transferred to a growth room with a 16 h photoperiod and a 16-20°C diurnal temperature cycle. Two weeks after inoculation plants were scored for the presence or absence of sporulation. All isolates were tested at least twice.

RESULTS

The faba bean single pustule isolates that shared the same host range are grouped together in Table 1. Faba bean isolates could be categorized into two major groups based on their ability to infect V. americana (Table 1). Group 1 included single pustule isolates 3, 11, 15, 17, 34, 36, 48 and 80 which were all able to infect V. americana, V. calcarata Desf., V. disperma DC., V. faba, L. japonicus and Lens culinaris. Isolates 34, 48 and 15 had the least extensive host range of this group. Isolates 3, 11, 36 and 80 differ from isolate SP17 in their ability to infect V. ervilia (L.) Willd., V. monantha Retz. and V. pisiformis L. and by their inability to infect L. ochroleucus, L. odoratus L. and L. tuberosus L.

Groups 2 of the faba bean rust isolates includes isolates 50, 51, 54, 56 and 61, none of which infect V. americana. These isolates are all able to infect V. calcarata, V. faba, V. monantha, Lens culinaris, P. sativum, L. japonicus, L. latifolius L. and L. sativus L. Differences were present between isolates in group 2 for the ability to infect V. ervilia, V. villosa Roth., L. ochroleucus and L. odoratus.

Rust isolates collected from V. americana had a host range that was very similar to that of V. faba isolates in group 1.

The pea isolates from Quebec have a considerably less extensive host range than isolates collected from peas in Manitoba. The Manitoba isolates infect all of the hosts infected by the Quebec isolates and also several others. Cultivated species such as V. faba and Lens

TABLE 1. Host range of isolates of Uromyces viciae-fabae collected from various hosts.

Host	V. amer. Isolates	L. ochrol. Isolates	L. venosus Isolates	Quebec P. sativum isolates	Manitoba P. sativum isolates	V. faba Isol. no. ^a 3,11,36,80	V. faba Isol. no. ^a 17	V. faba Isol. no. ^a 34,48,15	V. faba Isol. no. ^b 50,51	V. faba Isol. no. ^b 54,61	V. faba Isol. no. ^b 56
<u>V. americana</u> Muhl. †	+	-	-	-	-	+	+	+	-	-	-
<u>V. angustifolia</u> Reichard	-	-	-	-	-	-	-	-	-	-	-
<u>V. bithynica</u> (L.) L.			-	-	-	-	-	-	-	-	-
<u>V. calcarata</u> Desf.	+	-	-	+	+	+	+	+	+	+	+
<u>V. cracca</u> L. †	-	-	-	+	+	-	-	-	-	-	-
<u>V. disperma</u> DC.	+	-	-	+	+	+	+	+	+	+	+
<u>V. ervilia</u> (L.) Willd.	-	-	-	+	+	+	+	+	+	+	+
<u>V. faba</u> L.	+	*	*	-	*	+	+	+	+	+	+
<u>V. hirsuta</u> (L.) S.F. Gray.	-	-	-	-	-	-	-	-	-	-	-
<u>V. lutea</u> L.	-	-	-	-	-	-	-	-	-	-	-
<u>V. monantha</u> Retz.	-	+	+	+	+	+	+	+	+	+	+
<u>V. narbonensis</u> L.	-	-	-	-	-	-	-	-	-	-	-
<u>V. pisiformis</u> L.	+	-	+	-	-	+	-	-	-	-	-
<u>V. sativa</u> L.	-	-	-	-	-	-	-	-	-	-	-
<u>V. sepium</u> L.	-	-	-	-	-	-	-	-	-	-	-

† = Native to Manitoba.

+ = Not all plants in a species infected.

* = Smaller than normal pustule.

a = V. faba rust Group #1.b = V. faba rust Group #2.

TABLE 1. (continued)

Host	V. amer. Isolates	L. ochrol. Isolates	L. venosus Isolates	Quebec P. sativum isolates	Manitoba P. sativum isolates	V. faba Isol. no. a 3,11,36,80	V. faba Isol. no. a 17	V. faba Isol. no. b 34,48,15	V. faba Isol. no. b 50,51	V. faba Isol. no. b 54,61	V. faba Isol. no. b 56
<u>V. tetrasperma</u> (L.) Schreber.	-	-	-	-	-	-	-	-	-	-	-
<u>V. unifluga</u> A.Br.	-	-	-	-	-	-	-	-	-	-	-
<u>V. villosa</u> Roth.	-	-	-	-	-	-	-	-	+	-	-
<u>Lens culinaris</u> Medic.	-	+	+	+	+	+	+	+	+	+	+
<u>Pisum sativum</u> L.	+	+	+	+	+	+	+	-	+	+	+
<u>Lathyrus hirsutus</u> L.	-	-	-	-	-	-	-	-	-	-	-
<u>L. japonicus</u> Willd.†	+	+	+	+	+	+	+	+	+	+	+
<u>L. latifolius</u> L.	-	+	+	-	-	-	-	-	+	+	+
<u>L. montanus</u> Bernh.	-	-	-	-	-	-	-	-	-	-	-
<u>L. ochroleucus</u> Hook.†	-	+	+	-	+	-	+	-	-	+	-
<u>L. odoratus</u> L.	-	-	+	-	-	-	+	-	+	+	-
<u>L. pratensis</u> L.	-	-	-	-	-	-	-	-	-	-	-
<u>L. sativus</u> L.	+	+	+	-	+	+	+	-	+	+	+
<u>L. tuberosus</u> L.	-	-	+	-	-	-	+	-	-	-	-
<u>L. venosus</u> Muhl. †	-	-	+	-	+	-	-	-	-	-	-

† = Native to Manitoba.

+ = Not all plants in a species infected.

* = Smaller than normal pustule.

a = V. faba rust Group #1.b = V. faba rust Group #2.

culinaris were not infected by the Quebec isolates but were infected by the Manitoba isolates. However, the Manitoba pea isolates produced smaller than susceptible pustules on V. faba. Lathyrus species native to Manitoba and L. sativus were also infected by the Manitoban pea isolates.

The host ranges of rust collected from L. ochroleucus and L. venosus were very different from that of other isolates in this study. The host range of the L. venosus rust isolates includes all the hosts infected by the L. ochroleucus rust isolates and additional hosts such as V. pisiformis, L. odoratus, L. tuberosus and L. venosus. Rust from both L. ochroleucus and L. venosus infected P. sativum, Lens culinaris and produced smaller than susceptible pustules on V. faba.

Consistent differences in response to certain rust isolates were found between lines within the same species. This differential effect was observed between lines of V. calcarata, L. sativum and Lens culinaris. In addition, the reactions within selections of other host species were often not uniform.

DISCUSSION

The results of this study clearly demonstrate the existence of host specificity within isolates of Uromyces viciae-fabae. The host ranges of rust isolates were found to be more extensive than previous work had indicated. Frequently, an isolate could infect hosts included in the host ranges of several formae speciales, as described by Gaumann (1934). This can be seen in both groups 1 and 2 of the V. faba isolates, which infect hosts included in the host ranges of f. sp. viciae-fabae, f. sp. viciae-sepium and f. sp. lathyri-maritimi. On the other hand, these isolates behave similarly to isolates of the f. sp. viciae-fabae described by Kispatic (1949), except that they infect V. monantha and are unable to infect V. bithynica and V. narbonensis.

The P. sativum rust isolates from Quebec correspond to Gaumann's f. sp. craccae, since they infect P. sativum and V. cracca and are unable to infect V. faba. However, these isolates also infect V. ervilia and V. monantha, which f. sp. craccae could not infect.

The Manitoba P. sativum isolates infect hosts of f. sp. viciae-fabae, f. sp. craccae and f. sp. lathyri-maritimi. They differ from the V. faba isolates mainly in their ability to infect V. cracca and L. venosus.

Isolates collected on L. ochroleucus and L. venosus differ in host range from any isolate described in this study or any previous studies. Although isolates from L. venosus infect all the hosts infected by the L. ochroleucus isolates, the ability of the former to infect additional

hosts serves to distinguish the two groups.

The consistent differences in rust susceptibility observed between as well as within lines of a given species, make it difficult to recognize major groupings on the basis of host specificity. The heterogeneity found within certain species might be expected, since many of these species were collected in the wild. This lack of uniformity within certain hosts makes it unclear whether or not small deviations in host range are entirely due to differences in host specificity.

Recent studies using peas and faba bean lines have shown that distinct races of this rust do exist (Thesis Section 2). It is possible that race effects could also be confounding host range studies.

The results also demonstrate the importance of wild species of Vicia and Lathyrus in the epidemiology of this disease. The close similarities between the host ranges of V. americana rust isolates and group 1 of the faba bean isolates, suggests that all these isolates may have originally come from V. americana. This host and others such as L. ochroleucus and L. venosus could serve as a source of rust for crops such as peas and/or lentils.

The original source of inoculum for group 2 of the faba bean isolates remains unknown. The inability of isolates from this group to consistently infect native species of either Vicia or Lathyrus on which this rust is commonly found suggests that it did not come from these sources. The numerous differences in host range between the pea rust isolates and group 2 isolates, would seem to rule out peas as the original source. It could be that this group of isolates was introduced; in fact, there is evidence to suggest faba bean rust isolates may have been introduced into other parts of Canada (McKenzie and Morrall 1975).

The formae speciales described by Gaumann show little relationship to the isolates of Uromyces viciae-fabae found in Manitoba. The formae speciales concept seems inapplicable where so many hosts are shared in common by isolates from different host sources. Hybridization between rust isolates could occur on native and cultivated hosts shared by isolates. This type of gene flow might account for the broad spectrum of host specificity found in this study.

2. Pathogenic Variability in *Uromyces viciae-fabae*

ABSTRACT

Two races of *Uromyces viciae-fabae* (Pers.) Schroet. were identified on faba bean (*Vicia faba* L.) inbred lines, from amongst four isolates tested. Faba bean cultivars were variable in their reaction to isolate SS3 (race 1) but were almost uniformly susceptible to a second isolate, SP51 (race 2).

Rust isolates obtained from *Vicia americana* Muhl. behaved the same as the less virulent race 1 on the faba bean inbred lines. Additional races were detected among rust isolates from *Lathyrus venosus* Muhl. An isolate from *Lathyrus ochroleucus* Hook. was avirulent on all inbred faba bean lines.

Reactions of 16 pea (*Pisum sativum* L.) cultivars identified at least three races, two of which correspond to races 1 and 2. With a few exceptions the pea cultivars reacted uniformly to individual races. Field pea cultivars and their parental lines reacted similarly to rust isolates, indicating rust resistance had not been lost in the new cultivars.



INTRODUCTION

Faba bean (Vicia faba L.) and peas (Pisum sativum L.) are both known to be hosts of Uromyces viciae-fabae (Pers.) Schroet. (Conners 1967, Guyot 1957, Laundon 1964). Host range studies have shown that isolates exist which can infect one or both of these hosts (Gaumann 1934, Kispatic 1949).

Previous efforts to identify sources of rust resistance and differentiate rust races using faba bean cultivars have not succeeded. This failure was caused by either an absence of resistance (Hiratsuka 1934) or a lack of cultivar uniformity (Kispatic 1944, Kispatic 1949). Cultivar variability is largely due to the outcrossing nature of this crop, which has been found to range from 17-79% (Toynbee-Clark 1974).

The self-pollinating nature of peas tends to make cultivars quite uniform (Evans and Slinkard 1975), allowing sources of resistance to be identified readily. Differences in rust resistance between a number of pea lines have been reported in preliminary field trials using natural sources of rust inoculum (Sohi et al. 1974). Kispatic (1949) tested five pea cultivars to a number of single spore rust isolates and claimed to have identified nine different races. Closer examination of his results indicates only four races can be identified; the other five are biotypes of one of these four races differing only slightly for resistant infection type induced on one or more hosts.

This study was conducted to examine the extent of variability

for rust resistance in faba bean cultivars presently grown in Manitoba. Inbred faba bean lines selected for their uniform reactions to two specific rust isolates as well as several field and garden pea cultivars were used in an attempt to identify races of rust from wild and cultivated hosts of Uromyces viciae-fabae in Manitoba.

MATERIALS AND METHODS

Plants were grown in a 2:1:1 mixture of soil (clay loam), sand and peat in 10 or 15 cm clay pots in a rust free area of the greenhouse at 16-20°C under a 16 h photoperiod.

Most rust isolates used in this study were increased from single pustules on their original host. The single pustule isolates SP55, 62 and 63 were collected from peas in the Winnipeg area while a fourth isolate SP24 came from peas in Quebec. Faba bean single pustule rust isolates included SS3, SP17, 51 and 80. All other isolates used in this study were increased as field collections from specific localities. Standard procedures to prevent contamination of isolates were used (Appendix 4) and inoculum was stored at 5°C for no longer than a month before use.

Infection types were classified on the basis of pustule size, 11 days after inoculation (Table 2). This effectively limited the spread of rust and allowed the sequential testing of isolates on the same plants.

Heterogeneity of Cultivars

Four licensed cultivars presently grown in Manitoba and the rust susceptible line PI222128 were arranged in a randomized complete block design with 10 replications, each containing two pots of each line or cultivar, with five plants per pot. Three weeks after emergence, plants in one pot of each cultivar or line were inoculated with rust isolate SS3 and plants in the second pot were inoculated with isolate SP51.

TABLE 2. Infection types of Uromyces viciae-fabae
on Vicia faba and Pisum sativum.

Infection type	Cultivar Reaction	Reaction class
0	No sign of infection	Immune
1	Uredia minute, barely sporulating	Very resistant
2	Uredia small (about .5 mm in diameter)	Resistant
3	Uredia large (about 1.0 mm in diameter)	Susceptible
4	Uredia very large (>1.0 mm in diameter)	Very susceptible

Uredospores suspended in a light oil (Soltrol 170 , Philips Petroleum Co., Special Products Div., Borger TX 79007) were applied to the two youngest sets of fully expanded leaves using a fine nylon brush. After drying, the plants were placed in a chamber at 100% relative humidity for 24 h. After 11 days, plants were scored for infection type and infected leaves were removed. Plants were then inoculated with the second isolate (SS3 or SP51 depending on which isolate had been used previously) and treated as before.

Race Identification

Selections from licensed cultivars and introduced lines of V. faba with small to medium seed size, were chosen on the basis of their reaction to rust isolate SS3. After two to four generations of inbreeding and selection, eight of 17 lines, reacted uniformly to rust isolate SS3. Seven of these eight lines and an additional line that did not react uniformly to SS3, were found to react uniformly to a second rust isolate, SP51. These nine inbred lines were used as differentials in race identification.

Sixteen pea lines and cultivars were tested as potential differentials. These included the four field pea cultivars Century, Tara, Trapper and Triumph, presently grown in Manitoba. Certain parental lines of these field pea cultivars including Chancellor, Stirling, PI206852 and PI162567 were also used (Appendix 7). Garden pea cultivars tested in this study included the powdery mildew resistant cultivars Aurora and Melody, the semileafless cultivar Eaton, the leafless cultivar Filby and also the cultivars Thomas Laxton, Little Marvel, Edible Pod and Green Arrow.

Faba bean lines were inoculated as outlined in the heterogeneity study. Pea cultivars were spray inoculated with uredospores suspended in Soltrol 170 and were then treated as described in Thesis Section 1. Isolates were tested on all lines and cultivars at least twice except for the three Lathyrus venosus isolates which were each tested once on the faba bean lines.

RESULTS

The results of the heterogeneity study indicate that, with the exception of PI222128 which is uniformly susceptible to both rust isolates, there is a definite race effect for all cultivars with a significantly greater percentage of plants being susceptible to SP51 (Table 3). The heterogeneity within the cultivars is clearly demonstrated in their response to SS3. This is not the case for SP51, where only a few plants within any cultivar show resistance and where none of the cultivars were found to be significantly different in frequency of susceptible plants from the rust susceptible line PI222128 (Table 3, Appendix 6).

Faba bean rust isolates could be divided into two races based on their reactions to the inbred faba bean lines (Table 4). The less virulent race 1, which corresponds to isolates SS3 and SP80, is characterized by its ability to produce susceptible infection types on only lines 2N447-7, 2N40-3-5 and 2N62-6. The more virulent race 2, to which isolates SP17 and SP51 belong, can successfully infect all the lines susceptible to race 1 and also lines 2N12-2, Ack-11 and Erf-1-4.

In addition, rust isolate SP51 differed from isolate SS3 in its ability to consistently produce concentric rings of uredia around the initial pustule about 14 days after inoculation (Fig. 1). Additional rings continued to appear until the leaf dies.

Isolates collected on Vicia americana behaved the same as race 1 isolates from *V. faba*. The single rust isolate from Lathyrus ochroleucus

TABLE 3. Percentage of susceptible plants within faba bean cultivars infected by rust isolates SS3 and SP51.

Host	Percent plants infected		
	SS3	SP51	SS3 and SP51
Diana	7.0 a ¹	96.0 c	7.0 a*
Ackerperle	24.0 ab	84.0 c	23.3 ab
Herz Freya	24.0 ab	94.0 c	23.0 ab
Erfordia	40.0 b	96.0 c	31.0 b
PI222128	98.0 c	100.0 c	98.0 c

¹Tukey's w procedure at 5% level of significance. Values followed by the same letter are not significantly different.

*Tukey values calculated separately from those of individual isolates.

Table 4. Reactions of rust isolates on inbred faba bean lines.

Rust isolate	Inbred lines									
	2N 447-7	2N 40-3-5	2N 62-6	2N 12-2	Ack -11	Erf -1-4	D -50-7	HF -13-4	MB -1-2	
<u>From V. faba</u>										
SS3	+	+	+	-	-	-	-	+	-	
SP80	+	+	+	-	-	-	-	-	-	
SP51	+	+	+	+	+	+	+	-	-	
SP17	+	+	+	+	+	+	-	-	-	
<u>Rust from</u>										
<u>V. americana</u> Steinbach	+	+	+	-	-	-	-	-	-	
<u>V. americana</u> Falcon Lk.	+	+	+	-	-	-	-	-	-	
<u>L. ochroleucus</u> Oak Point	*	-	-	-	-	-	-	-	-	
<u>L. venosus</u> Haddashville	*	*	+	*	-	-	-	*	*	
<u>L. venosus</u> Killarney	+	*	*	*	-	-	-	*	*	
<u>L. venosus</u> Brokenhead Rv.	+	+	+	*	*	*	*	*	*	

+ = Infection type 3-4.

* = Infection type 2.

- = Infection type 0-1.



FIGURE 1. Infection types of rust isolates (left to right) SP51 and SS3 on faba bean line PI222128 at fourteen days after inoculation.

was avirulent on all the faba bean lines, producing a type 2 infection on only some plants of line 2N447-7. Rust isolates from the three sources of Lathyrus venosus differed from each other for virulence on the inbred faba bean lines. These isolates were also able to produce low infection types on HF-13-4 and MB-1-2 which were immune to all the other isolates in this study.

The faba bean isolates SP17 and SP51, which represent race 2 on faba bean lines, behaved the same on all pea cultivars (Table 5). These isolates were virulent on all garden pea cultivars used in this study but were only able to infect a very small percentage of the field pea cultivars or their parents.

Isolates SS3 and SP80 which correspond to race 1 had an even more limited range of virulence, differing from race 2 by their inability to infect Thomas Laxton.

Rust isolates SP24 and SP62 were different than either of the previous two races since they were virulent on all the pea lines and cultivars. Although isolates SP55 and SP63 infected fewer plants of some cultivars, these two isolates could still belong to this third race. The single Lathyrus venosus rust isolate tested behaved the same as SP55 and SP63 on all the pea lines and cultivars.

TABLE 5. Reaction of rust isolates on lines and cultivars of Pisum sativum.

Host	Rust isolates						L. venosus rust Brokenhead Rv.
	SP24 + 62	SP55 + 63	SPI7 + 51	SS3 + 80			
Field Pea							
Century	15/18a 3 ^b	13/13 2-3	0/31	0/8	6/9	2-3	
Trapper	24/24 3	11/15 2-3	1/34 3	2/8 2-4	8/15	2-3	
Triumph	16/19 3	10/10 3	0/30	0/8	14/15	3-4	
Tara	14/15 2-3	13/13 2-3	0/21 0-2	0/8	11/14	1-3	
Chancellor	14/14 3	6/6 3	0/16	0/3	4/7	3	
Stirling	15/15 3-4	6/6 3-4	1/25 3	0/8	5/7	1-4	
PI206852	9/9 3-4	5/6 3-4	1/14 3	0/2	5/5	3	
PI162567	4/4 3	6/6 3	2/9 3	0/2	2/2	3	
Garden Pea							
Thomas Laxton	10/10 3	9/14 3	24/30 2-3	0/4 1-2	4/6	2-3	
Little Marvel	8/8 3	10/10 3	11/11 3	3/3 3	3/3	3	
Edible Pod	9/11 3	2/4 3	2/13 3	3/3 3-4	2/2	3	
Green Arrow	10/10 3	6/6 3	6/6 3		3/3	3	
Aurora	9/9 2-3	15/15 3	11/13 3		8/8	2-3	
Melody	11/11 3	4/4 3	13/13 3-4	4/4 2-3	6/6	3-4	
Eaton	5/6 3	9/10 2-3	8/11 3				
Filby	8/9 3	14/14 2-3	10/12 3		4/4	3-4	

a = No. susceptible plants/inoculated ; b = infection type

DISCUSSION

The faba bean cultivars were found to be variable in their resistance to rust isolate SS3 whereas they were almost totally susceptible to isolate SP51 (Table 3). It would seem that if isolates similar to SP51 ever became predominant, they could be very damaging to the presently grown cultivars.

This study demonstrates that there is a considerable degree of pathogenic variability within populations of Uromyces viciae-fabae in Manitoba. The reactions of both cultivars and inbred lines clearly show that the faba bean isolates in this study can be separated into two different races (Tables 3 and 4). These results support the suggestion of Tan et al. (1975) that inbred lines would be superior to open pollinated cultivars for race identification.

The ability of rust isolates from V. americana to behave consistently like race 1 (SS3 and SP80) supports the results of a concurrent study, whereby rust isolates from V. americana had a host range that was similar to that of certain V. faba isolates, thus indicating V. americana could have an important role in the epidemiology of this disease (Thesis Section 1). In contrast, L. ochroleucus does not appear to be an important source of rust inoculum on faba beans, although additional isolates should be tested to confirm this.

Several races differing in their ability to infect certain inbred lines of V. faba may exist among isolates of L. venosus although

additional tests are needed to confirm this. The ability of L. venosus isolates to produce type 2 infections on HF-13-4 and MB-1-2, distinguishes them from all the other isolates which were only able to induce an immune response on these lines. This suggests that a different resistance mechanism is operating against these isolates.

Reactions of pea lines and cultivars indicate at least three and possibly, more races exist amongst the isolates tested (Table 5). The virulence patterns of races 1 (SS3 and SP80) and 2 (SP17 and SP51) differ on the pea cultivars but the results do not allow isolates belonging to either race to be further partitioned. A third race to which isolates SP24 and SP62 belong could attack all the cultivars in this test. Isolates SP55, SP63 and the L. venosus isolate from the Brokenhead Rv. may belong to this race, although certain cultivars are not uniformly susceptible to them.

In general, the pea lines and cultivars used in this study appear to be uniform enough to allow race differentiation. Although certain cultivars, most notably Trapper, Thomas Laxton and Edible Pod are not completely homogeneous. This lack of uniformity within cultivars of a self pollinated species is not unexpected since there has never been active selection for rust resistance in these cultivars.

The reactions of the field pea cultivars were identical to that of their parents indicating that the resistance of the parents has not been lost in the development of the new cultivars.

The garden pea cultivars lack the resistance to race 2 (SP17 and 51) that is present in the field pea cultivars. The performance of Thomas Laxton against race 1 (SS3 and SP80) demonstrates that it differs from the other garden pea cultivars.

Faba bean inbred lines and pea cultivars performed equally well in the differentiation of rust races. The choice of systems would depend largely on whether interest lies with a single crop or in an appreciation of the wider scope of race development. The use of faba bean lines has the advantage that at least some of the resistance genes in these lines have been identified (Thesis Section 3). The best system for race identification might be one that supplements the faba bean inbred lines with certain pea cultivars. This is similar to the use of barley cultivars to supplement wheat differentials in the early work on Puccinia graminis f. sp. tritici (Stakman and Christensen 1953).

3. Inheritance of Rust Resistance in Inbred Lines of *Vicia faba*

ABSTRACT

Crosses between nine inbred faba bean (*Vicia faba* L.) lines selected for uniform reaction against rust [*Uromyces viciae-fabae* (Pers.) Schroet.] isolates SS3 and SP51, revealed the presence of three different dominant genes for resistance. The gene Fr1 found in lines Ack-11 and 2N12-2 provides resistance only to SS3. A second gene, Fr2, which also conferred resistance to rust isolate SS3 was detected in lines Erf-1-4 and MB-1-2. An additional homozygous gene Fr3 in MB-1-2 conditioned resistance only to isolate SP51. Heterozygous resistance genes found in Erf-1-4 and MB-1-2 had their segregation masked by other homozygous resistance genes. Resistance in D-50-7 and HF-13-4 was still not homozygous after three generations of inbreeding and selection for resistance.

INTRODUCTION

Faba bean rust caused by Uromyces viciae-fabae (Pers.) Schroet. is present in almost every area of the world where faba beans (Vicia faba L.) are grown (Guyot 1957). Since the recent introduction of this crop to Western Canada, rust on faba beans has been reported in Manitoba and Saskatchewan (Bernier 1975, McKenzie and Morrall 1975). Williams (1978) has shown that this disease can seriously reduce yield if infection occurs early and is severe.

Studies have shown that faba bean lines and cultivars do not respond uniformly when tested against specific rust isolates (Thesis Section 2, Kispatic 1944, 1949).

The inheritance of rust resistance in faba beans has never been studied previously. However, similar studies have been conducted with outcrossing crops such as rye (Secale cereale L.) against Puccinia graminis f. sp. tritici Eriks. and E. Henn. and f. sp. secalis Eriks. and E. Henn. (Tan et al. 1975, 1976, 1977) and in corn (Zea mays L.) to Puccinia sorghi Schw. (Wilkinson and Hooker 1968). In these studies inbreds with uniform rust reactions were developed by repeatedly selfing and testing lines against specific rust isolates.

This study was undertaken to estimate the number of resistance genes in nine inbred faba bean lines that are known to react uniformly to at least one of two rust isolates (Thesis Section 2). The relationship of the resistance gene(s) in each line was also determined.

MATERIALS AND METHODS

The nine inbred lines used in this study, as well as the two rust isolates used to establish their reactions, have been described previously (Thesis Section 2). Three inbred lines were susceptible to both rust isolates, four were resistant only to isolate SS3, one was uniformly resistant only to isolate SP51 and the remaining line was resistant to both isolates.

The rust isolates were increased on the rust susceptible line PI222128, using standard procedure to prevent contamination (Appendix 4) Rust inoculum was collected and stored as described previously (Thesis Section 1).

Plants were grown in a rust-free area of the greenhouse at 16-20°C under a 16 h photoperiod. Reciprocal crosses were made for all combinations of these lines, with the exception of Ack-11 and 2N447-7 which were not included in all crosses because of poor seed production.

All F_1 , F_2 and backcross populations were tested sequentially against both rust isolates as outlined previously (Thesis Section 2), with the exception that the less virulent rust isolate SS3 was always used in the initial inoculation. Parental lines were included in all tests to ensure that rust isolates were free of rust contaminants. Plants were scored as resistant or susceptible based on infection type (Thesis Section 2) 11 days after inoculation.

The progenies of all crosses were advanced to the F_2 generation

by single seed descent. Results from F_2 families of the same crosses were pooled only after chi square tests for homogeneity had shown them to behave the same. Chi square tests for goodness of fit were used to analyze all F_2 and backcross data.

RESULTS

No significant differences were detected between reciprocal crosses so F_2 data from reciprocal crosses were pooled. The F_1 and F_2 data indicated that in all cases resistance was a dominant trait (Appendix 9) (Tables 6 and 8).

Segregation for resistance to isolate SS3 in the F_2 generations for both Ack-11 and 2N12-2 crossed to susceptible lines best fits a 3:1 ratio. This, along with the results of backcrosses, suggests that Ack-11 and 2N12-2 each carry a single resistance gene against rust isolate SS3 (Tables 6 and 8). Crosses of Ack-11 to 2N12-2 produced only resistant F_2 plants indicating that the genes controlling resistance to SS3 in these two lines appear to be either identical, allelic or tightly linked. Certain F_1 and F_2 progenies of several crosses have resistance to SP51 that was not detected in the parental lines (Table 6).

The F_2 data from crosses of Erf-1-4 to susceptible lines consistently fit a 27:5 ratio for resistance to isolate SS3, indicating resistance is controlled by two dominant genes only one of which is homozygous (Table 6). This was confirmed by the fairly good fit of a 5:3 ratio in the backcross populations (Table 8). Crosses between resistant lines indicate that the homozygous resistance gene found in

Erf-1-4 is different from that found in 2N12-2. The homozygous genes for resistance to SS3 in Erf-1-4 and MB-1-2 appear to be the same since when these two lines are crossed, all F_2 progeny are resistant (Table 6).

Line MB-1-2 carries a second gene for resistance to SS3 that appears to be heterozygous (Tables 6 and 8). Resistance in MB-1-2 to rust isolate SP51 also seems to be controlled by two genes, one homozygous and one heterozygous. The homozygous genes controlling resistance to either SS3 or SP51 appear to be different since many F_2 plants are susceptible to only one rust isolate (Table 9). A good fit of a 193:27:27:9 ratio for the combined results from crosses of MB-1-2 to susceptible lines against both isolates, strengthens these conclusions (Table 9). Such a ratio would be expected if one homozygous gene provides resistance only to SS3, a second homozygous gene confers resistance to SP51 and a third heterozygous gene confers resistance to both. The susceptibility of inbred lines in this study to SP51 was always found to be recessive to the resistance of MB-1-2 (Table 6).

Chi square tests for homogeneity revealed that F_2 families within certain crosses involving D-50-7 and HF-13-4 were significantly different. To facilitate interpretation of F_2 data, results from F_2 families found to behave differently are presented separately (Table 7).

Segregation in backcrosses D-50-7 X (D-50-7 X Suscept.) indicates the gene(s) for resistance to isolates SS3 and SP51 are not homozygous (Table 8). The segregation of resistance to SS3 was not anticipated since both parental lines and F_1 progeny were uniformly resistant (Appendices 7 and 8). However, based on lack of uniformity in D-50-7 to SP51 it was expected that the gene(s) conferring resistance to this

isolate would still be heterozygous and F_1 , F_2 and backcross results confirm this (Tables 7 and 8). Resistance in D-50-7 to isolates SS3 and SP51 appears to be controlled by different genes since few F_2 plants are infected by both isolates (Table 9). The relationship of these genes to resistance genes in other lines cannot be determined until they are fixed.

The results for HF-13-4 are the same as that of D-50-7; segregation in F_1 populations and backcrosses to HF-13-4 as well as the lack of similarity between F_2 families from the same crosses, all indicate that the resistance gene(s) in HF-13-4 are not homozygous. The fact that only a small number of F_2 plants were infected by both isolates suggests the gene(s) controlling resistance to each of these isolates are different.

TABLE 6. Segregation of reactions to rust isolates SS3 and SP51 in F₂ populations derived from homozygous parents.

Parents	Isolate SS3				Isolate SP51			
	Parent reaction	F ₂ results** R:S	Expected ratio	P	Parent reaction	F ₂ results** R:S	Expected ratio	P
2N62-6 X 2N447-7	SXS	0:104	0:1		SXS	1:103	0:1	.99-.975
2N62-6 X 2N40-3-5	SXS	0:100	0:1		SXS	4: 96	0:1	.75-.50
Ack-11 X 2N62-6	RXS	142: 42	3:1	.50	SXS	20:161 +	0:1	.25-.10
Ack-11 X 2N447-7	RXS	93: 26	3:1	.50-.25	SXS	15:104	0:1	.25-.10
Erf-1-4 X 2N40-3-5	RXS	179: 23	27:5	.10	SXS	24:177 +	0:1	.10-.05
Erf-1-4 X 2N62-6	RXS	166: 29	27:5	.90-.75	SXS	21:174	0:1	.25-.10
2N12-2 X 2N40-3-5	RXS	75: 27	3:1	.75-.50	SXS	16:86	0:1	.50-.25
2N12-2 X 2N62-6	RXS	132: 31	3:1	.10	SXS	26:147	0:1	.25-.10
MB-1-2 X 2N40-3-5	RXS	178: 21	27:5	.05	RXS	177: 22	27:5	.10-.05
MB-1-2 X 2N62-6	RXS	170: 25	27:5	.50-.25	RXS	168: 27	27:5	.50-.25
Ack-11 X 2N12-2	RXR	230: 0	1:0		SXS	60:156 +	0:1	<.001
Erf-1-4 X 2N12-2	RXR	68: 7	15:1	.50-.25	SXS	13: 62	0:1	.25-.10
MB-1-2 X 2N12-2	RXR	175: 1	63:1	.50-.25	RXS	146: 30	55:9	.50-.25
MB-1-2 X Erf-1-4	RXR	315: 0	1:0		RXS	289: 26	55:9	<.001

* χ^2 probability value

** Only homogeneous families are combined

† Some plants damaged.

TABLE 7. Segregation of reactions to rust isolates SS3 and SP51 in F₂ populations derived from crosses involving at least one heterozygous parent.

Parents	Isolate SS3					Isolate SP51				
	** No. of families	Parent reaction	F ₂ Results R:S	Expected ratio	P*	Parent reaction	** No. of families	F ₂ Results R:S	Expected ratio	P*
D-50-7 X 2N62-6	3	RXS	112:43	3:1	.50-.25	RXS	3	96:59	3:1	< .001
D-50-7 X 2N40-3-5	1	RXS	55:4	15:1	.90-.75	RXS	2	58:42	7:9	< .001
D-50-7 X 2N40-3-5	1	RXS	30:11	3:1	.90-.75					
D-50-7 X 2N12-2	3	RXR	83:12	27:5	.50-.25	RXS	3	53:42	7:9	.025-.01
D-50-7 X Erf-1-4	3	RXR	215:4	63:1	.90-.75	RXS	3	120:99	7:9	< .001
D-50-7 X MB-1-2	3	RXR	211:1	63:1	.25-.10	RXR	3	202:10	15:1	.50-.25
D-50-7 X HF-13-4	3	RXR	212:3	63:1	.90-.75	RXR	3	200:15	15:1	.75-.25
HF-13-4 X 2N62-6	2	RXS	79:20	3:1	.50-.25	RXS	2	80:19	3:1	.25-.10
HF-13-4 X 2N62-6	2	RXS	40:53	7:9	.90	RXS	2	59:34	3:1	.01
HF-13-4 X 2N40-3-5	2	RXS	54:42	7:9	.025-.01	RXS	2	62:34	3:1	.025-.01
HF-13-4 X 2N12-2	2	RXR	83:20	27:5	.50-.25	RXS	2	60:43	3:1	< .001
HF-13-4 X 2N12-2	3	RXR	177:15	15:1	.50-.25	RXS	1	16:32	7:9	.25-.10
							1	69:25	3:1	.75-.50
							1	43:7	27:5	.75
HF-13-4 X 2N12-2	1	RXR	50:0	0:1	1.0	RXS	1	39:17	3:1	.10-.05
HF-13-4 X Ack-11	4	RXR	154:5	15:1	.10-.75	RXS	3	76:45	3:1	< .001
							1	34:4	15:1	.50-.25
HF-13-4 X Erf-1-4	4	RXR	265:4	247:9	.10-.05	RXS	3	172:48	3:1	.50-.25
							1	27:22	7:9	.25-.10
HF-13-4 X MB-1-2	2	RXR	88:13	27:5	.50-.25	RXS	3	147:12	15:1	.50
HF-13-4 X MB-1-2	1	RXR	57:1	247:9	.50-.25					

*x² Probability values.

** Only homogeneous families are combined.

TABLE 8. Segregation of reactions torust isolates SS3 and SP51 in backcross populations.

Parents	Isolate SS3				Isolate SP51			
	Parent reaction	B.C. Results R:S	Expected ratio	p*	Parent reaction	B.C. Results R:S	Expected ratio	p*
2N62-6 X (Ack-11 X 2N62-6)	SXR	37:14	1:1	<.001	SXS	2:49	0:1	.90-.75
Ack-11 X (Ack-11 X 2N62-6)	RXR	27: 0	1:0		SXS	2:24 +	0:1	.75-.50
2N40-3-5 X (Erf-1-4 X 2N40-1-5)	SXR	44:16	5:3	.10-.05	SXS	6:54	0:1	.50-.25
Erf-1-4 X (Erf-1-4 X 2N40-3-5)	RXR	52: 0	1:0		SXS	15:32 +	0:1	.05-.025
2N62-6 X (Erf-1-4 X 2N62-6)	SXR	32:14	5:3	.50-.25	SXS	11:34 +	0:1	.10
Erf-1-4 X (Erf-1-4 X 2N62-6)	RXR	44: 0	1:0		SXS	9:35	0:1	.25-.10
2N62-6 X (2N12-2 X 2N62-6)	SXR	23:23	1:1		SXS	5:40 +	0:1	.50-.25
2N12-2 X (2N12-2 X 2N62-6)	RXR	29: 0	1:0		SXS	2:27	0:1	.75-.50
2N40-3-5 X (MB-1-2 X 2N40-3-5)	SXR	21:16	5:3	.50-.25	SXR	27: 7 +	5:3	.05-.025
MB-1-2 X (MB-1-2 X 2N40-3-5)	RXR	58: 0	1:0		RXR	58: 0	1:0	
2N62-6 X (MB-1-2 X 2N62-6)	SXR	26:19	5:3	.75-.50	SXR	22:23	5:3	.10-.05
MB-1-2 X (MB-1-2 X 2N62-6)	RXR	27: 0	1:0		RXR	27: 0	1:0	
2N40-3-5 X (D-50-7 X 2N40-3-5)	SXR	12:23	1:3	.25-.10	SXR	13:22	1:3	.10
D-50-7 X (D-50-7 X 2N40-3-5)	RXR	29: 3	3:1	.05-.025	RXR	21:11	5:3	.75
2N62-6 X (D-50-7 X 2N62-6)	SXR	16:28	1:3	.10-.05	SXR	16:27 +	1:3	.10-.05
D-50-7 X (D-50-7 X 2N62-6)	RXR	17: 4	3:1	.75-.50	RXR	14: 6 +	5:3	.50-.25
2N62-6 X (HF13-4 X 2N62-6)	SXR	33:36	1:1	.75-.50	SXR	17:51	1:3	.95-.90
HF13-4 X (HF-13-4 X 2N62-6)	RXR	52:15	3:1	.50-.25	RXS	43: 0	1:0	
						15: 9	3:1	.25-.10

* χ^2 Probability values.

+ Some plants damaged.

TABLE 9. Relation of resistance to rust isolates SS3 and SP51 in F₂ populations derived from resistant X susceptible crosses.

Cross	No. of pl. not inf.	No. of pl. inf. by SS3 only	No. of pl. inf. by SP51 only	No. of pl. inf. by both isol.	Expected ratio	P*
MB-1-2 X 2N40-3-5	158	20	18	3	193:27:27:9	.50-.25
MB-1-2 X 2N62-6	150	20	20	7	193:27:27:9	.995-.99
D-50-7 X 2N40-3-5	52	6	33	9		
D-50-7 X 2N62-6	68	28	44	15		
HF-13-4 X 2N40-3-5	35	27	19	15		
HF-13-4 X 2N62-6	99	40	20	35		

*X² probability values.

DISCUSSION

Three different genes for resistance were identified based on the results of crosses between different resistant lines (Table 10). The lines 2N12-2 and Ack-11 carry the same single dominant gene for resistance to SS3 which has been designated Fr1. A second gene for resistance to SS3, found in Erf-1-4 and MB-1-2 has been designated Fr2. MB-1-2 carries an additional homozygous dominant gene Fr3, which provides resistance only to SP51.

In addition to these homozygous genes, Erf-1-4 and MB-1-2 both carry heterozygous resistance genes. The presence of these heterozygous genes is masked in both lines by the homozygous resistance genes.

The low number of resistant F_2 plants found in crosses between parents susceptible to SP51, is somewhat difficult to explain (Table 6). Certain of these plants might have escaped infection and in other cases it may be that one of the parents carried heterozygous gene(s) for resistance which were not detected in earlier tests.

Genes controlling resistance to both rust isolates in D-50-7 and HF-13-4 were not homozygous after three generations of inbreeding and selection for resistance. Further work is needed to complete the identification of their respective genes.

TABLE 10. Proposed genotypes of inbred faba bean lines based on their response to isolates SS3 and SP51.

Host	Proposed genotype ^a
2N447-7	fr1fr1, fr2fr2, fr3fr3
2N40-3-5	fr1fr1, fr2fr2, fr3fr3
2N62-6	fr1fr1, fr2fr2, fr3fr3
2N12-2	Fr1Fr1, fr2fr2, fr3fr3
Ack-11	Fr1Fr1, fr2fr2, fr3fr3
Erf-1-4	fr1fr1, Fr2Fr2, fr3fr3, Rr
MB-1-2	fr1fr1, Fr2Fr2, Fr3Fr3, Rr
D-50-7	Rr, Rr
HF-13-4	Rr, Rr

^aFr1 Fr2 - provide resistance against isolate SS3.

Fr3 - provides resistance against isolate SP51 only.

R represents unidentified resistance genes.

4. Slow Rusting Resistance in *Vicia faba*

ABSTRACT

The relative rate of rust buildup on lines of *Vicia faba* L. susceptible to *Uromyces viciae-fabae* (Pers.) Schroet. was examined in field studies over three years. Significant differences were found between lines for area under the disease progress curve (AUDPC) values, although variation within lines was high. Three lines consistently had low AUDPC values and another six lines had AUDPC values that shifted from low to intermediate. The other 19 lines included in this study were either fast rusters or behaved inconsistently.

Final rust severity was well correlated to AUDPC in each year of this study. Measurements summarizing the incidence of infected plants over time and the percentage of infected plants at the end of the season were poorly correlated to AUDPC.

INTRODUCTION

The often transitory nature of specific resistance to a disease has led to an increased interest in slow rusting resistance. This type of resistance has been studied in several host parasite systems and may provide a more stable form of disease control (Knott 1972).

Slow rusting resistance retards rust development in the field resulting in a general delay of a severe epiphytotic (Milu and Line 1980, Shaner et al. 1978, Wilcoxson et al. 1975). The significance of this type of resistance can only be seen over time. Several measurements such as area under the disease progress curve, infection rate and time needed to reach a certain level of disease severity have been used to study this or similar phenomena (Shaner and Finney 1977, Wilcoxson et al. 1975). The relative merits of such measurements have been reviewed and the area under the disease progress curve is generally considered to be the most reliable value (Shaner and Finney 1977, Wilcoxson et al. 1975). Studies with wheat stem and leaf rust have shown this value to be correlated to yield (Buchenau 1975).

Slow rusting studies have generally been limited to self-pollinating crops or to inbred lines of outcrossing crops (Hooker 1969, Kim and Brewbaker 1977). Kuhn and Smith (1977) proposed a host index system to measure differences in the incidence of maize dwarf mosaic virus in various lines of corn (Zea mays L.). This system could have application in other host systems where selections may not be completely uniform.

The purpose of this study was to develop a system that would allow the detection of differences in levels of the slow rusting resistance in open-pollinated lines of Vicia faba L.

MATERIALS AND METHODS

A total of 25 faba bean accessions were used in this study (Appendix 10). These lines were known to differ for final rust severity when exposed to natural sources of rust inoculum in field trials at the University of Manitoba (C. Bernier, unpublished data).

In a preliminary study to assess the slow rusting phenomenon in selected lines, the coefficients of variation were found to be very high for measurements of disease buildup (Appendix 12) when plot size was small (12-15 plants) and replications were few (three replications in a randomized complete block design). Thus, in 1978 these lines were arranged in a balanced incomplete block design. Each line was replicated nine times in single row plots one meter in length, containing between 12-15 plants.

In 1979, with more seed available, the lines were planted in three row plots, 2 meters in length, containing 40-50 plants per plot. Plots were set out in a partially balanced incomplete block design with five replications. Two sites were used, one at the University of Manitoba and the second at the Glenlea research farm 15 km south of Winnipeg. The trial at the University of Manitoba was so severely damaged by root rot that it could not be used in the slow rusting study.

In 1980, five faba bean lines were removed from the study reducing the number of lines to 20. Lines were planted in three row plots, two and a half meters in length, containing 60-70 plants. Plots were arranged in a randomized complete block design with five replications,

The experiment was conducted at two locations 0.8 km apart on the Glenlea research farm.

Each year, two rows of the rust susceptible line PI222128 were planted at the ends of each plot to act as rust spreader rows. In 1978 the spreader rows were inoculated with uredospores from a mixture of rust isolates suspended in water to which several drops of Tween 20 (Polyoxyethylene sorbitan monolaurate) were added. In 1979 and 1980 two rust isolates SS3 and SP51, with distinct virulence patterns (Thesis Section 2) were used, one at each location. In all three years, rust spreader rows were inoculated 4-5 weeks after emergence and all locations were inoculated the same evening.

Starting two weeks after inoculation, the number of plants infected and average percentage of leaf area infected for each infected plant within a plot were recorded at weekly intervals for 4 or 5 weeks when they began to senesce.

Assessment keys of 1, 5 and 10% leaf area covered by sporulating pustules were developed and used to visually estimate the leaf area infected. Values of leaf area infected over time were summarized as area under the disease progress curve (AUDPC) values using the formula described by Wilcoxson et al (1975):

$$AUDPC = \sum_{i=1}^K \frac{1}{2} (S_i + S_{i+1})$$

where S_i equals rust severity at the end of week i and K is the number of successive evaluations.

Data on the number of plants infected over time were summarized as host index (H.I.) (Kuhn and Smith 1977) and modified host index (Mod. H.I.) values using the formulas:

$$H.I. = K \cdot W + K-1 \cdot X + K-2 \cdot Y + \dots 1 \cdot Z$$

$$\text{Mod. H.I.} = K \cdot W^1 + K-1 \cdot X^1 + K-2 \cdot Y^1 + \dots 1 \cdot Z^1$$

where K equals the number of successive evaluations, and W, X, Y and Z represent the percentage of infected plants out of the total plants tested by the first, second, third and last week respectively. The symbols W^1 , X^1 , Y^1 and Z^1 equal the percentage of susceptible plants infected out of all susceptible plants in a season in the first, second, third and last weeks respectively.

Analysis of variance were carried out on these values, the percent plants infected and the final rust severity. The means of each line were compared using Tukey's w-procedure.

RESULTS

Significant differences between lines were found in all three years for AUDPC values (Appendices 13-16). In each year there was a continuum of AUDPC values ranging from high to relatively low levels of rust build up (Tables 11, 13, 15 and 17). However, it was still possible to identify lines such as 2N34, 2N52 and 2N430 which consistently behaved as slow rusters. Other lines including 2N35, 2N45, 2N58, 2N293, 2N414 and 2N451 had AUDPC values that varied from year to year from low to intermediate. Line 2N62 appeared to carry specific resistance to the rust isolates used in this study since only low infection types were observed and a low percentage of plants was infected. Fast rusting lines included 2N3, 2N4, 2N40, 2N42 and 2N59, all remaining lines had AUDPC values that shifted between intermediate and high from year to year.

High precipitation early in the development of the epidemic appeared to be responsible for the relatively higher AUDPC values in 1978 (Appendix 11) than in other years. However, differences in the number of weeks over which disease was assessed makes direct comparisons of results from different years difficult.

Final rust severity was found to be fairly well correlated to AUDPC (Table 19). Host index was always better correlated to AUDPC than either modified host index or percent plants infected, however, these differences were not always statistically significant. In both

1978 and 1980 at site 2 the correlation of host index to AUDPC and the correlation of modified host index to AUDPC were not significantly different. The correlation of host index to AUDPC in 1979 and 1980 at site 1 were not significantly different from the correlation of percent plants infected to AUDPC.

High coefficients of variation indicated there was a great deal of variation within lines associated with each of these measurements (Tables 11-18).

The analysis of variance for the 1978 data showed a significant block effect was present even after adjusting for line effects (Appendix 13). An adjacent rust experiment appeared to cause the blocks closest to it to rust more rapidly. In most cases equal numbers of plots from each line were influenced by this block effect. This, together with the adjustment of line means for block effects, should likely minimize the importance of the block effect.

Subsequently rust experiments were isolated to avoid this problem. However, in 1979 and 1980 at site 1, replications were significantly different (Appendices 14 and 15). Certain replications had much poorer overall rust development but because rust build up within replications was fairly uniform, line values were not biased by this effect.

TABLE 11. Average values of area under the disease progress curve and final rust severity for faba bean lines in 1978.

Accession no.	R A N K	AUDPC ^{a,b}	R A N K	Final rust ^a severity
2N42	1	13.07 a*	1	5.96 a*
2N3	2	12.41 a	2	5.72 ab
2N12	3	10.32 ab	5	5.03 abc
2N59	4	9.59 abc	7	4.42 abcd
2N4	5	9.13 abcd	4	5.14 abc
2N14	6	8.90 abcd	6	4.93 abc
2N116	7	8.79 abcde	3	5.64 ab
2N40	8	8.27 abcde	9	4.37 abcd
2N313	9	7.29 bcde	16	3.48 bcde
2N413	10	7.05 bcde	8	4.41 abcd
2N447	11	6.96 bcdef	21	2.89 cde
2N45	12	6.77 bcdef	13	3.80 abcd
2N58	13	6.46 bcdef	12	3.82 abcd
2N44	14	6.37 bcdef	17	3.42 bcde
2N125	15	6.36 bcdef	20	3.03 cde
2N83	16	6.11 bcdef	15	3.48 bcde
2N35	17	5.66 bcdef	11	3.98 abcd
2N414	18	5.58 bcdef	19	3.08 cde
2N293	19	5.57 bcdef	23	2.80 cde
2N52	20	5.50 bcdef	14	3.77 abcd
2N124	21	5.26 cdef	10	4.29 abcd
2N451	22	5.01 cdef	24	2.44 de
2N430	23	4.49 def	18	3.23 cde
2N34	24	3.82 ef	22	2.86 cde
2N62	25	1.98 f	25	1.22 e
C.V.		43.67%		37.22%

*Tukey's w procedure of 10% level of significance, values followed by the same letter are not significantly different.

^aValues adjusted for block effects as described in Cochran and Cox 1966.

^bResults based on five weeks data.

TABLE 12. Average values of host index, modified host index and percent plants infected for faba bean lines in 1978.

Accession no.	R A N K	Host Index ^{a,b}	R A N K	Mod. Host Index ^{a,b}	R A N K	Percent plants infected ^a
2N42	2	1179.51 ab*	2	1196.86 ab*	4	98.84 ab*
2N3	1	1399.93 a	1	1353.56 a	2	100.00 a
2N12	3	1164.41 ab	3	1176.46 ab	2	100.00 a
2N59	4	1085.89 abc	4	1141.35 abc	15	92.20 abc
2N4	7	1017.86 bcd	8	1017.86 abcde	2	100.00 a
2N14	11	761.81 cdefg	15	772.92 cdefg	10	94.08 abc
2N116	9	936.54 bcdef	9	1013.43 abcde	12	93.51 abc
2N40	5	1067.89 abc	5	1100.95 abc	6	97.78 abc
2N313	8	975.72 bcde	10	987.11 abcdef	5	98.40 ab
2N413	10	918.99 bcdef	7	1029.69 abcde	13	93.44 abc
2N447	6	1055.34 abcd	6	1070.35 abcd	8	96.69 abc
2N45	21	490.69 gh	17	691.10 defgh	22	77.44 ab
2N58	23	484.49 gh	24	560.90 gh	14	93.16 abc
2N44	16	591.91 fgh	23	605.52 fgh	9	96.22 abc
2N125	17	587.04 fgh	13	807.78 bcdefg	21	77.16 abc
2N83	22	487.64 gh	21	636.73 efgh	16	83.31 abc
2N35	20	523.55 gh	19	655.89 efgh	17	83.30 abc
2N414	13	694.63 defg	11	952.88 bcdefg	19	79.77 abc
2N293	18	585.67 fgh	14	781.73 cdefg	23	71.99 bc
2N52	12	704.74 defg	16	705.91 defgh	11	93.80 abc
2N124	15	644.26 efgh	18	673.61 efgh	7	97.52 abc
2N451	14	655.47 efgh	12	902.19 bcdefg	24	70.88 c
2N430	19	528.70 gh	20	644.70 efgh	16	83.31 abc
2N34	24	422.17 gh	22	618.55 fgh	20	79.52 abc
2N62	25	324.07 h	25	322.80 h	25	37.20 d
C.V.		29.84%		28.31%		19.32%

*Tukey's w procedure of 10% level of significance, values followed by the same letter are not significantly different.

^aValues adjusted for block effects as described in Cochrane and Cox 1966.

^bResults based on five weeks data.

TABLE 13. Average values for area under the disease progress curve and final rust severity for faba bean lines inoculated with rust isolate SS3 in 1979.

Accession no.	R	AUDPC ^{a,b}	R	Final rust ^a severity
	A		A	
	N		N	
	K		K	
2N40	1	5.49 a*	1	2.87 a*
2N447	2	4.49 ab	2	2.38 ab
2N59	3	4.09 abc	3	2.36 ab
2N3	4	4.00 abcd	5	2.00 abc
2N42	5	3.69 abcde	4	2.06 abc
2N4	6	3.60 bcde	9	1.79 abcde
2N116	7	3.22 bcdef	6	1.95 abcd
2N83	8	3.06 bcdef	7	1.81 abcde
2N14	9	3.02 bcdef	8	1.80 abcde
2N44	10	2.98 bcdef	10	1.49 bcde
2N313	11	2.72 bcdefg	12	1.43 bcde
2N413	12	2.47 cdefg	11	1.48 bcde
2N451	13	2.40 cdefg	15	1.27 bcde
2N58	14	2.33 cdefg	13	1.32 bcde
2N414	15	2.25 defg	16	1.21 bcdef
2N45	16	2.17 efg	14	1.27 bcdef
2N293	17	1.98 efg	18	1.13 bcdef
2N35	18	1.93 efg	17	1.15 bcdef
2N52	19	1.75 fg	19	1.06 cdef
2N12	20	1.75 fg	22	0.82 cdef
2N34	21	1.59 fg	21	0.87 cdef
2N124	22	1.48 fg	20	0.89 cdef
2N125	23	1.47 fg	23	0.71 def
2N430	24	1.11 g	24	0.68 ef
2N62	25	0.09 g	25	0.05 f
C.V.		30.9%		38.7%

*Tukey's w procedure at 10% level of significance, values followed by the same letter are not significantly different.

^aValues adjusted for block effects as described in Cochran and Cox 1966.

^bResults based on four weeks data.

TABLE 14. Average values for host index, modified host index and percent infected plants for faba bean lines inoculated with rust isolate SS3 in 1979.

Accession no.	R A N K	Host Index ^{a,b}	R A N K	Mod. Host Index ^{a,b}	R A N K	Percent plants ^a infected
2N40	1	752.4 a*	1	874.6 a*	2	88.5 a*
2N447	6	314.2 cd	5	701.4 abcd	9	43.7 def
2N59	4	516.3 b	6	675.8 abcd	4	72.9 ab
2N3	2	736.8 a	3	822.2 ab	1	89.8 a
2N42	3	604.8 ab	4	708.4 abcd	3	82.2 a
2N4	5	459.1 bc	7	661.9 abcde	5	68.3 abc
2N116	11	214.9 def	20	446.6 defgh	7	46.7 cde
2N83	14	167.5 defg	16	471.4 defgh	12	34.9 defgh
2N14	7	288.8 cde	11	570.3 bcdefg	8	46.7 cde
2N44	15	150.3 defg	10	589.0 abcdefg	19	24.7 efghi
2N313	9	247.1 def	9	607.7 abcdef	11	37.2 defgh
2N413	12	214.4 def	14	528.0 cdefgh	10	39.4 defg
2N451	10	222.4 def	2	846.2 ab	17	26.6 efgh
2N58	13	194.8 defg	8	652.1 abcdefg	13	29.4 defgh
2N414	16	136.2 defg	17	467.6 defgh	16	27.6 efgh
2N45	23	102.0 efg	19	463.0 defgh	20	24.0 efghi
2N293	19	107.5 efg	22	375.2 efgh	14	28.8 defgh
2N35	17	118.9 defg	12	559.8 bcdefg	22	21.6 fghi
2N52	18	118.2 defg	21	426.7 defgh	18	26.0 efgh
2N12	8	247.4 defg	15	507.8 defgh	6	52.3 bcd
2N34	22	104.3 efg	13	551.8 bcdefg	24	15.1 hi
2N124	21	104.9 efg	23	358.7 fgh	15	27.7 efgh
2N125	20	105.9 efg	18	463.6 defgh	21	23.4 efghi
2N430	24	58.2 fg	24	294.0 gh	23	16.9 ghi
2N62	25	6.2 g	25	241.2 h	25	1.6 i
C.V.		35.2%		23.6%		26.9%

*Tukey's w procedure at 10% level of significance, values followed by the same letter are not significantly different.

^aValues adjusted for block effects as described in Cochran and Cox 1966.

^bResults based on four weeks data.

TABLE 15. Average values for area under the disease progress curve and final rust severity for faba bean lines inoculated with rust isolate SS3 at site 1 in 1980.

Accession no.	R A N K	AUDPC ^a	R A N K	Final rust severity
2N40	1	3.24 a*	1.5	2.08 a*
2N3	2	2.86 ab	3	1.93 a
2N12	3	2.81 ab	1.5	2.08 a
2N42	4	2.66 abc	4	1.72 ab
2N44	5	2.37 abc	5	1.50 abc
2N59	6	2.24 abc	7.5	1.45 abcd
2N124	7	2.22 abc	6	1.46 abcd
2N83	8	2.15 abc	9.5	1.16 bcd
2N35	9	2.09 abc	11	1.15 bcd
2N313	10	2.04 bc	7.5	1.45 abcd
2N45	11	1.99 bc	9.5	1.16 bcd
2N58	12	1.94 bc	14	1.07 bcd
2N451	13	1.89 bc	17	0.92 cd
2N414	14.5	1.80 bc	13	1.09 bcd
2N14	14.5	1.80 bc	12	1.12 bcd
2N116	16	1.75 bc	18	0.89 cd
2N293	17	1.65 bc	15	0.96 cd
2N430	18	1.62 c	19	0.87 cd
2N52	19	1.58 c	20	0.77 d
2N34	20	1.51 c	16	0.95 cd
C.V.		25.78%		25.29%

* Tukey's w procedure at the 10% level of significance, values followed by the same letter are not significantly different.

^a Results based on four weeks data.

TABLE 16. Average values for host index, modified host index and percent plants infected for faba bean lines inoculated with rust isolate SS3 at site 1 in 1980.

Accession no.	R	Host Index ^a	R	Mod. Host Index ^a	R	Percent plants infected
	A N K		A N K		A N K	
2N40	1	432.02 a*	4	474.03 a*	1	93.49 a*
2N3	2	370.17 ab	3	477.37 a	4	72.10 a
2N12	3	366.44 ab	12	443.64 a	2	91.46 a
2N42	4	336.76 abc	6	468.59 a	3	73.37 ab
2N44	11	164.44 de	11	445.94 a	11	34.41 cd
2N59	5	277.98 abcd	5	471.69 a	5	56.16 bc
2N124	7	235.83 bcdef	16	436.36 a	6	54.42 bc
2N83	9	167.40 def	7	457.63 a	14	32.88 cd
2N35	15	141.69 def	13	441.31 a	16	31.06 cd
2N313	6	252.10 bcde	8	451.09 a	7	53.79 bcd
2N45	13	151.94 def	15	438.18 a	10	35.12 cde
2N58	12	152.98 def	14	440.32 a	12	34.15 cde
2N451	14	144.22 def	9	450.88 a	15	32.80 cde
2N414	10	164.88 def	2	484.73 a	13	33.15 cde
2N14	17	132.92 def	1	489.59 a	18	27.07 de
2N116	8	193.31 cdef	10	449.30 a	8	41.71 cde
2N293	19	81.89 f	17	430.64 a	19	18.32 e
2N430	18	117.09 ef	19	336.12 a	17	30.87 cde
2N52	16	134.46 def	20	327.82 a	9	35.28 cde
2N34	20	79.26 f	18	410.19 a	20	18.05 e
C.V.		35.74%		19.64%		29.14%

*Tukey's w procedure at the 10% level of significance, values followed by the same letter are not significantly different.

^aResults based on four weeks data.

TABLE 17. Average values for area under the disease progress curve and final rust severity for faba bean lines inoculated with rust isolate SP51 at site 2 in 1980.

Accession no.	R	AUDPC	^a	R	Final rust severity
	A			A	
	N			N	
	K			K	
2N12	1	7.20 a*		3	5.41 abc*
2N40	2	7.11 ab		2	5.44 ab
2N313	3	6.53 abc		1	5.68 a
2N3	4	6.17 abcd		4	5.12 abcd
2N42	5	5.46 bcde		6	4.73 abcde
2N59	6	5.31 cde		5	4.79 abcde
2N116	7	4.69 def		8	4.20 abcdef
2N44	8	4.68 def		9	3.94 abcdef
2N124	9	4.62 def		7	4.44 abcdef
2N35	10	4.54 def		12	3.54 def
2N14	11	4.32 ef		17	3.25 ef
2N293	12	4.26 ef		10	3.77 bcdef
2N430	13.5	4.17 ef		16	3.28 ef
2N414	13.5	4.17 ef		11	3.67 cdef
2N83	15	4.11 ef		18	3.12 ef
2N45	16	4.04 ef		13	3.52 def
2N34	17	3.96 ef		15	3.31 ef
2N58	18.5	3.84 ef		14	3.44 def
2N451	18.5	3.84 ef		19	3.08 ef
2N52	20	3.61 f		20	2.77 f
C.V.		16.04%			20.06%

* Tukey's w procedure at the 10% level of significance, values followed by the same letter are not significantly different.

^a Results based on five weeks data.

TABLE 18. Average values for host index, modified host index and percent plants infected for faba bean lines inoculated with rust isolate SP51 at site 2 in 1980.

Accession no.	R A N K	Host Index ^a	R A N K	Mod. Host Index ^a	R A N K	Percent plants infected
2N12	3	932.24 ab*	2	956.07 a*	3	97.08 abc*
2N40	1	964.51 a	1	966.73 a	1	99.75 a
2N313	5	762.51 abcd	4	813.36 ab	6.5	93.87 abcde
2N3	2	944.60 ab	3	951.89 a	2	99.33 ab
2N42	4	797.98 abc	6	792.92 abc	5	96.29 abcd
2N59	7	715.40 cde	7	781.49 abc	12	91.75 abcde
2N116	6	754.86 bcd	5	809.89 ab	11	92.72 abcdef
2N44	8	665.30 cdef	8	726.02 bcd	10	92.86 abcdef
2N124	10	592.40 cdefg	14	618.29 bcd	4	96.69 abcd
2N35	11	567.35 defgh	11	645.15 bcd	14	87.03 abcdef
2N14	20	385.85 h	9	720.00 bcd	20	54.77 g
2N293	9	593.27 cdefg	12	628.95 bcd	8	93.73 abcdef
2N430	15	511.78 efgh	13	618.32 bcd	18	82.47 ef
2N414	18	487.95 fgh	20	520.86 d	6.5	93.87 abcdef
2N83	14	519.52 efgh	15	617.60 bcd	17	84.58 def
2N45	12	532.55 efgh	10	656.82 bcd	19	80.72 f
2N34	16	509.75 efgh	17	585.47 cd	13	87.13 bcdef
2N58	17	503.94 efgh	16	587.25 cd	16	85.57 def
2N451	13	487.95 fgh	18	564.60 d	9	93.39 abcdef
2N52	19	454.72 gh	19	541.22 d	15	87.02 bcdef
C.V.		14.92%		13.99%		6.38%

*Tukey's w procedure at the 10% level of significance values, followed by the same letter are not significantly different.

^aResults based on five weeks data.

TABLE 19. R^2 values for correlations of area under the disease progress curve with other measurements of disease in the years 1978, 1979 and 1980.

	1978	1979	1980 (SS3)	1980 (SP51)
AUDPC vs HI	0.45**	0.60**	0.64**	0.65**
AUDPC vs Mod HI	0.34**	0.42**	0.24*	0.61**
AUDPC vs Percent	0.20	0.53**	0.52**	0.17
AUDPC vs Final Sev.	0.62**	0.86**	0.70**	0.76**

*,** Correlation significant at the 5% and 1% level respectively.

DISCUSSION

Final rust severity values were well correlated to AUDPC (Table 19). This was to be expected since final rust severity contributes in part to AUDPC. Since these two values are closely associated, it would appear that final rust severity could be used as a criterion for selecting potential slow rusting lines in preliminary tests. Final rust severity has the advantage of being much less labor intensive than recording rust development over time as required in order to obtain AUDPC values.

The poor correlations of host index, modified host index and percent infected plants (Table 19) to AUDPC indicates that these measurements do not account for differences between lines in ability to reduce rust development. Additional factor(s) in these lines must be contributing to their ability to retard rust development. It may be that these factors influence measurements such as host index or modified host index.

The outcrossing nature of V. faba accounts for some of the variability found within lines. In addition, a certain amount of variability is associated with any trait that is visually estimated. Notwithstanding the variability found within lines for AUDPC values, it is possible to identify slow rusting lines in small field plots. Lines such as 2N34, 2N52 and 2N430 consistently had low AUDPC values and other lines including 2N35, 2N45, 2N58, 2N293, 2N414 and 2N451 had low to intermediate values. These lines may carry some level of slow rusting resistance. The poor performance of other lines in this study would

not seem to warrant their further evaluation.

Although this experiment was not designed to examine the influence of plot size on the evaluation of slow rusting in V. faba, it was found that the coefficient of variation was reduced as plot size increased. In view of the heterogeneity that exists within lines for their ability to retard rust, it would seem that the smaller plots are less representative of the population from which they were taken. Larger plots tend to reduce the effects of a few fast rusting plants within a plot allowing more reproducible results.

The reduction of within-line variation observed after 1978 could also be due in part to the use of a single isolate per test site rather than a mixture of isolates. The use of single isolates would avoid problems associated with race specificity that make certain lines appear to be slow rusters only because of a low frequency of virulent races in the mixture.

GENERAL DISCUSSION

Results of the host range study demonstrate that isolates of Uromyces viciae-fabae have a much wider host range than previous studies had indicated. The wide host range of the rust isolates from Canada, together with the previously reported host range studies from other parts of the world (Gaumann 1934, Kapooria and Sinha 1969, 1971, Kispatic 1949) demonstrates that a great deal of variability exists within Uromyces viciae-fabae. Rust isolates from various hosts and geographic regions differ in both pattern and scope of host range on members of the tribe Viciae. Wahl (1978) suggests that variability in host range is usually greatest in areas of host diversity. Thus, areas of North America where native species of Vicia and Lathyrus occur may be primary or secondary centers of diversity.

The use of inbred faba bean lines selected for their reaction to specific rust isolates eliminates the problem of cultivar heterogeneity that has prevented race identification in earlier studies (Kispatic 1944, 1949). Based on knowledge of the homozygous genes for resistance in these inbred faba bean lines, a maximum of six races could theoretically be identified. If the resistance gene Fr3 was separated from the gene Fr2, then an additional two races could also be identified.

This is the first time that the genetics of rust resistance has been successfully studied in V. faba. Although three genes for rust resistance were identified, additional genes are known to be present in

the inbred lines used in this study. The heterozygous resistance gene in line MB-1-2 differs from the other resistance genes described in this study since it confers resistance to both rust isolates SS3 and SP51. The resistance gene(s) in D-50-7 and HF-13-4 still remain to be identified.

Experience with rust diseases in other crops suggests that using single resistance genes in cultivars would not result in long term rust control. Gene pyramiding might be more successful, provided the resistance genes involved are not released singly. Complex races would have to be developed under greenhouse conditions to ensure that all resistance genes were homozygous before a cultivar is released. Safeguards would also be needed during cultivar development and seed increase phases to prevent outcrossing. This would ensure that virulent races do not develop by a stepwise pattern of changes in virulence on outcrossed progeny that possess a reduced number of resistance genes.

Multilines could be even better suited for use in a partially outcrossing crop such as faba beans. Since quality requirements are not very stringent, multiline cultivars could likely be released following a few generations of backcrossing the resistance genes into several agronomically suitable lines.

Faba bean lines were found to differ in their ability to retard the development of a rust epiphytotic. The use of larger isolated plots and inoculations from a point source might more realistically simulate field conditions and allow the rate limiting capability of slow rusting lines to be better expressed. The nature of this resistance and its stability against additional races requires further study. Identifi-

cation of the mechanism(s) responsible for this resistance could aid plant breeders in visually selecting slow rusting resistance and facilitate its incorporation into suitable cultivars.

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APPENDIX 1. (Continued)

Host species	Uromyces fabae (Pers.) de Bary.					Uromyces orobi (Pers.) Lev.			Uromyces ervi (Wallr.) West.	
	f.sp. vicia-fabae	f.sp. pisi-sativi	f.sp. cracca	f.sp. viciae-nipponicae	f.sp. viciae-unifugae	f.sp. viciae-sepium	f.sp. orobi	f.sp. lathyr. verni	f.sp. lathyr. maritimi	f.sp. viciae-hirsutae
<i>Vicia amoena</i> Fisch. var. <i>Sachalinensis</i> Fr.	-									
<i>V. angustifolia</i> All.	-		-			-	-	-		
<i>V. atropurpurea</i> Desf.	-			-						
<i>V. cracca</i> L.	-		+			-				-
<i>V. cracca</i> L. var. <i>japonica</i>	-			-					-	-
<i>V. ervilia</i> (L.) Willd.	-					-				-
<i>V. faba</i> L.	+	-	-	-	-	+	-	-	-	-
<i>V. hirsuta</i> Koch	-	-	+	-	-	-	-	-	-	+
<i>V. hybrida</i> L.						-				
<i>V. manantha</i> (L.) Desf.	-			-		+				
<i>V. nipponica</i> Mats. var. <i>capitata</i> Nak.	-			+	-					-
<i>V. onobrychioides</i> L.	-		-			-		-		
<i>V. pannonica</i> Crantz.	-	-		-		-				
<i>V. pisiformis</i> L.										
<i>V. sativa</i> L.	-		-	-		-	-	-	-	+
<i>V. sepium</i> L.	-		-			+		-		+
<i>V. tetrasperma</i> Moench.	-	-				-		-	-	+
<i>V. uniflora</i> A. Br.	-			-	+					-
<i>V. villosa</i> Roth	-			-		-				
<i>Vigna sinensis</i> Endl.	-			-						

P = Smaller than normal pustule.

APPENDIX 2. Results of host range studies on Uromyces
viciae-fabae conducted after 1934.

Host	Kispatic (1949)	Kapooria and Sinha (1969)	Kapooria and Sinha (1971)
<u>Vicia faba</u>	+	+	
<u>V. disperma</u>	+		
<u>V. bithynica</u>	+		
<u>V. calcarata</u>	+		
<u>V. biennis</u>	+	+	
<u>V. pisiformis</u>	+		
<u>V. narbonensis</u>	+	+	
<u>V. monantha</u>	-		
<u>V. articulata</u>	-		
<u>Pisum sativum</u>	+		+
<u>Lens culinaris</u>	-		
<u>V. tetrasperma</u>		+	
<u>V. ervilia</u>		+	
<u>V. gracilis</u>		+	
<u>V. sativa</u>		-	
<u>V. hirsuta</u>		+	
<u>Lathyrus sphaenicus</u>			+
<u>L. aphaca</u>		-	+
<u>P. abyssinicus</u>			+
<u>P. elatus</u>			+
<u>P. jomardi</u>			+
<u>L. cicera</u>			-
<u>L. ochrus</u>			-
<u>L. odoratus</u>			-
<u>L. sativus</u>			-

APPENDIX 3. Source of rust isolates used
in the host range study.

Host plant	Collection site	No. of isolates
<u>Vicia faba</u> L.	Winnipeg	13
<u>Pisum sativum</u>	Quebec	2
	Winnipeg	4
<u>Vicia americana</u>	Winnipeg	1
	Falcon Lake	1
	Steinbach	1
<u>Lathyrus ochroleucus</u>	Winnipeg	1
	Arborg	1
	Oak Point	1
<u>Lathyrus venosus</u>	Killarney	1
	Brandon	1
	Haddashville	1
	Poplarfield	1
	Brokenhead Rv.	1

APPENDIX 4. Procedures for increasing rust isolates.

Inoculation of host plants for rust increase was carried out in a plexiglass inoculation cabinet equipped with a set of fine spray nozzles. Between inoculations, these nozzles sprayed a fine mist of water from the top of the cabinet washing the air within the cabinet free of uredospores after four to five minutes. After spraying, the walls of the cabinet were hosed down. The inoculated plants were covered with a tubular polyvinyl isolation cage. Filtered air was circulated through the cage by a rubber hose attached at the base of the chamber. This slight flow of air through the isolation cages prevented contaminating spores from floating in and also prevented an excessive build-up of moisture. Rust was later collected with a cyclone spore collector or by the leaf-tap method (Browder 1971). Uninoculated check plants were always included to ensure that any possible contamination could be detected.

APPENDIX 5. Source of host selections used in the
host range study.

Accession no.	Name	Source*
PCR999	<i>Vicia americana</i> Muhl.	Ottawa, Canada
UM-1	<i>V. americana</i> Muhl.	Winnipeg, Canada
OS1	<i>V. angustifolia</i> Reich.	Iran via U.S.A.
OS297	<i>V. angustifolia</i> Reich.	Denmark
OS107	<i>V. bithynica</i> (L.) L.	Upsala, Sweden
OS175	<i>V. calcarata</i> Desf.	Ottawa, Canada
OS405	<i>V. calcarata</i> Desf.	U.S.S.R.
UM-2	<i>V. cracca</i> L.	Winnipeg, Canada
OS54	<i>V. cracca</i> L.	Besancon, France
OS413	<i>V. disperma</i> DC.	Montevideo, Uruguay
OS24	<i>V. ervilia</i> (L.) Willd.	Vermilles, France
OS108	<i>V. ervilia</i> (L.) Willd.	Upsala, Sweden
2M40	<i>V. faba</i> L.	Afghanistan
OS9	<i>V. hirsuta</i> (L.) S.F. Gray	Australia via U.S.A.
OS28	<i>V. hirsuta</i> (L.) S.F. Gray	Vermilles, France
OS33	<i>V. lutea</i> L.	Vermilles, France
OS111	<i>V. monantha</i> Retz.	Upsala, Sweden
OS12	<i>V. parbonensis</i> L.	Turkey via U.S.A.
OS94	<i>V. pisiformis</i> L.	Basel, Switzerland
OS147	<i>V. pisiformis</i> L.	Liverpool, England
OS17	<i>V. sativa</i> L.	Turkey via U.S.A.
OS44	<i>V. sativa</i> L.	Vermilles, France
OS257	<i>V. sativa</i> L.	Sofia, Hungary
OS61	<i>V. sepium</i> L.	Besancon, France
OS46	<i>V. tetrasperma</i> (L.) Schreber	Vermilles, France
OS382	<i>V. tetrasperma</i> (L.) Schreber	Hamburg, Germany
OS114	<i>V. unijuga</i> A. Br.	Upsala, Sweden
OS19	<i>V. villosa</i> Roth.	Hungary via U.S.A.
OS234	<i>V. villosa</i> Roth.	Larissa, Greece
	<i>Lens culinaris</i> Medic. Cowboy	
	<i>L. culinaris</i> Medic. Larissa	
	<i>Pisum sativum</i> L. Little Marvel	
2R17	<i>Lathyrus hirsutus</i> L.	France via U.S.A.
2R125	<i>L. japonicus</i> Willd.	Upsala, Sweden
2R473	<i>L. japonicus</i> Willd.	Hamburg, Germany
2R579	<i>L. japonicus</i> Willd.	Paris, France
UM-3	<i>L. japonicus</i> Willd.	Winnipeg Beach, Canada
	<i>L. latifolius</i> L.	Georgetown, Ont., Canada
2R79	<i>L. latifolius</i> L.	Bern, Switzerland
2R173	<i>L. montanus</i> Bernh.	Freiburg, Germany
PCR1000	<i>L. ochroleucus</i> Hook.	Ottawa, Canada
UM-4	<i>L. ochroleucus</i> Hook.	Winnipeg, Canada
	<i>L. odoratus</i> L.	Canada
2R184	<i>L. pratensis</i> L.	Helsinki, Finland
2R205	<i>L. pratensis</i> L.	Liverpool, England
2R7	<i>L. sativus</i> L.	Turkey via U.S.A.
2R133	<i>L. sativus</i> L.	Hamburg, Germany
2R189	<i>L. tuberosus</i> L.	Poland
2R287	<i>L. tuberosus</i> L.	Ottawa, Canada
UM-5	<i>L. yencous</i> Muhl.	Deerfield, Man., Canada

* Source may not correspond to origin.

APPENDIX 6. Analysis of variance for the
heterogeneity test of faba bean cultivars.

Source of variation	D.F.	S.S.	M.S.	F
Reps	9	9,660.1250	1,073.3472	4.2573**
Time	1	1,725.1250	1,725.1250	6.8425**
Race	1	142,278.1250	142,278.1250	564.3272**
Host lines	4	66,195.7450	16,548.9363	65.6392**
T X R	1	174.875	174.8750	0.6936
T X H.L.	4	1,341.755	335.4388	1.3305
R X H.L.	4	42,018.705	10,504.6760	41.6654**
T X R X H.L.	4	1,086.92	271.7300	1.0778
Error	171	43,112.500	252.1199	
Total	199	307,593.875		

**Significant at the 1% level.

APPENDIX 7. Parentage of field pea cultivars used
to study pathogenic variability of Uromyces
viciae-fabae.

Cultivar	Parents
*Century	(Chancellor X Early Raymond) X Stirling
*Tara	7585-5B X PI162567
7585-SB	Century X Chancellor
**Trapper	Chancellor X Weibulls 700
*Triumph	PI206852

*Described in Agriculture Canada (Production and
Marketing Branch, Plant Products Div.) Description
of Varieties.

**Described in the Seed Scoop 1970, p. 5.

APPENDIX 8. Reactions of inbred faba bean lines
to rust isolates SS3 and SP51.

Inbred line	Isolate SS3		Isolate SP51	
	Infection type	R:S	Infection type	R:S
2N447-7	3-4	0:18	3-4	0:18
2N62-6	3-4	0:23	3-4	0:23
2N40-3-5	3-4	0:23	3-4	0:23
2N12-2	0	22:0	3-4	0:22
Ack-11	0	17:0	3	0:17
Erf-1-4	0	30:0	3	0:30
D-50-7	0	11:0	0-3	10:1
HF-13-4	0-3	35:4	0	39:0
MB-1-2	0-1	12:0	0	12:0

APPENDIX 9. Reactions of F₁ lines to rust isolates
SS3 and SP51.

Cross	SS3		SP51	
	Parent reactions	F ₁ R:S	Parent reactions	F ₁ R:S
2N62-6 X 2N447-7	SXS	0:15	SXS	0:15
2N62-6 X 2N40-3-5	SXS	0:37	SXS	0:37
Ack-11 X 2N62-6	RXS	58:0	SXS	5:53
Ack-11 X 2N447-7	RXS	33:0	SXS	7:26
Ack-11 X 2N12-2	RXR	81:0	SXS	10:71
Ack-11 X HF-13-4	RXR	34:0	SXR	33:1
2N12-2 X 2N62-6	RXS	63:0	SXS	5:58
2N12-2 X 2N40-3-5	RXS	24:0	SXS	0:24
2N12-2 X Erf-1-4	RXR	20:0	SXS	0:20
2N12-2 X MB-1-2	RXR	46:0	SXR	46:0
2N12-2 X HF-13-4	RXR	79:0	SXR	79:0
2N12-2 X D-50-7	RXR	47:9	SXR	23:23
Erf-1-4 X 2N62-6	RXS	65:0	SXS	9:56
Erf-1-4 X 2N40-3-5	RXS	58:0	SXS	3:55
Erf-1-4 X MB-1-2	RXR	82:0	SXR	82:0
Erf-1-4 X HF-13-4	RXR	33:0	SXR	33:0
Erf-1-4 X D-50-7	RXR	52:0	SXR	31:21
MB-1-2 X 2N62-6	RXS	47:0	RXS	47:0
MB-1-2 X 2N40-3-5	RXS	62:0	RXS	62:0
MB-1-2 X HF-13-4	RXR	51:0	RXR	51:0
MB-1-2 X D-50-7	RXR	48:0	RXR	48:0
HF-13-4 X 2N62-6	RXS	38:3	RXS	41:0
HF-13-4 X 2N40-3-5	RXS	25:14	RXS	24:15
HF-13-4 X D-50-7	RXR	54:0	RXR	54:0
D-50-7 X 2N62-6	RXS	36:0	RXS	23:9
D-50-7 X 2N40-3-5	RXS	18:8	RXS	15:10

APPENDIX 10. Accession numbers and source of lines
used in the slow rusting study.

Accession no.	Cultivar name or line no.	Source
2N3	PI221517	Minn., U.S.A.
2N4	PI221517	" "
2N12	PI222129	" "
2N14	PI254005	" "
2N34	Baladi	Yassin, Sudan
2N35	BF/212	" "
2N40	PI222128	Afghanistan, via Pullman, Wash.
2N42	PI222216	" " "
2N44	PI223303	" " "
2N45	PI223304	" " "
2N52	PI254001	Syria via Pullman, Wash.
2N58	Dagestanskije Cernye	Russia
2N59		"
2N62		"
2N83	Minuta Pirhaner	Upsala, Sweden
2N116		Denmark
2N124	Selection Inra-1049	
2N125	" "	
2N293	NEB111	India via U.S.A.
2N313	NEB152	Wad Medani, Sudan
2N413	NEB343	Egypt
2N414	NEB344	"
2N430	NEB362	"
2N447	NEB425	Hudaiba, Sudan
2N451	NEB429	" "

APPENDIX 11. Precipitation for the years
1978, 1979 and 1980.

Period	Total precipitation in mm.		
	Winnipeg	Glenlea	
	1978	1979	1980
July 1-15	44	11	6
July 16-31	39	25	16
Aug. 1-15	5	30	57
Aug. 16-31	50	55	60
Sept. 1-7	2	20	32
Total	140	141	171

APPENDIX 12. Analysis of variance for slow rusting results
from 1977.

Source	D.F.	Sum of squares	Mean squares	F Value
<u>AUDPC</u>				
Replications	2	17,4463	8.7232	1.5099
Lines	24	382,4809	15.9367	2.7584**
Error	48	277.3204	5.7775	
Total	74	677.2476		
C.V.=53.14%				
<u>Final Rust Sev.</u>				
Replications	2	0.2219	0.1109	0.0451
Lines	24	99.7048	4.1544	1.6882
Error	48	118.1248	2.4609	
Total	74	218.0515		
C.V.=70.96%				
<u>Host Index</u>				
Replications	2	165,214.4280	82,607.2140	4.6730*
Lines	24	2,505,458.398	104,394.0999	5.9054**
Error	48	848,529.8279	17,677.7047	
Total	74	3,519,202.6540		
C.V.=39.53%				
<u>Mod H.I.</u>				
Replications	2	50,452.5680	25,226.2840	0.8901
Lines	24	1,497,122.725	62,380.1135	2.2011**
Error	48	1,360,357.421	28,340.7796	
Total	74	2,907,932.714		
C.V.=31.76%				
<u>Percent Infected</u>				
Replications	2	1,809.7814	904.8907	2.3419
Lines	24	34,517.9391	1,438.2475	3.7223**
Error	48	18,546.6628	386.3888	
Total	74	54,874.3833		
C.V.=34.53%				

* Significant at the 5% level.

** Significant at the 1% level.

APPENDIX 13. Analysis of variance for slow rusting
results from 1978.

Source	D.F.	Sums of squares	Mean squares	F Value
<u>AUDPC</u>				
Lines	24	1,398.0288	58.2512	6.1097**
Blocks (adj.)	24	227.8938	9.4956	0.9960
Error	176	1,678.0149	9.5342	
Total	224	3,303.9375		
<u>Final Severity</u>				
Lines	24	285.6524	11.9022	5.6804**
Blocks (adj.)	24	106.0048	4.4169	2.1080**
Error	176	368.7716	2.0953	
Total	224	760.4288		
<u>Host Index</u>				
Lines	24	18,214,406.46	758,933.6024	14.5827**
Blocks (adj.)	24	4,274,900.5506	178,120.8562	3.4225**
Error	176	9,159,621.9111	52,043.3063	
Total	224	30,239,628.05		
<u>Modified H.I.</u>				
Lines	24	13,274,967.27	553,123.6360	9.4003**
Blocks (adj.)	24	1,620,508.089	67,521.1704	1.1475
Error	176	10,356,042.841	58,841.1525	
Total	224	25,250,518.20		
<u>Percent Infected</u>				
Lines	24	42,612.4877	1,775.5203	6.2075**
Blocks (adj.)	24	11,750.090	489.5871	1.7117*
Error	176	50,340.96	286.0282	
Total	224	104,703.5344		

* Significant at the 5% level.

** Significant at the 1% level.

APPENDIX 14. Analysis of variance for slow rusting
results from 1979.

Source	D.F.	Sums of squares	Mean squares	F Value
<u>AUDPC</u>				
Replications	4	40.7613	10.1903	15,6750**
Lines	24	1169.8518	7.0772	10.8863**
Blocks (adj.)	20	14.8235	0.7412	1.1401
Error	76	49.4061	0.6501	
Total	124	274.8427		
<u>Final Rust Sev.</u>				
Replications	4	10.6201	2.6550	8.6145**
Lines	24	48.2724	2.0114	6.5263**
Blocks (adj.)	20	7.5103	0.3755	1.2184
Error	76	23.4209	0.3082	
Total	124	89.8237		
<u>Host Index</u>				
Replications	4	815,723.5200	203,930.8800	25.8737**
Lines	24	5,034,938.8800	209,789.1200	26.6170**
Blocks (adj.)	20	117,290.5600	5,864.5275	0.7441
Error	76	599,014.4000	7,881.7681	
Total	124	6,566,968.3200		
<u>Modified H.I.</u>				
Replications	4	1,191,825.9200	297,956.4800	17.3669**
Lines	24	3,217,579.5200	134,065.8100	7.8142**
Blocks (adj.)	20	351,827.6800	17,591.3800	1.0253
Error	76	1,303,902.7200	17,156.6100	
Total	124	6,065,136.6400		
<u>Percent Infected</u>				
Replications	4	9,674.2400	2,418.5600	21.0714**
Lines	24	66,826.5600	2,784.4400	24.2591**
Blocks (adj.)	20	1,977.2500	98.8625	0.8613
Error	76	8,723.2300	114.7793	
Total	124	87,201.2800		

** Significant at the 1% level.

APPENDIX 15. Analysis of variance ~~for~~ ~~slow~~ rusting
results at site 1 (SS3) 1980.

Source	D.F.	Sums of squares	Mean squares	F Value
<u>AUDPC</u>				
Replications	4	17.6893	4.4223	14.9503**
Lines	19	21.1750	1.1114	3.7573**
Error	76	22.4853	0.2958	
Total	99	61.3496		
<u>Final Rust Sev.</u>				
Replications	4	6.3375	1.5843	14.8900**
Lines	19	15.4236	0.8118	7.6297**
Error	76	8.0828	0.1064	
Total	99	29.8439		
<u>Host Index</u>				
Replications	4	486,342.9117	121,885.7279	22.6698**
Lines	19	988,191.2350	52,010.0650	9.6973**
Error	76	407,614.2381	5,363.3452	
Total	99	1,882,148.3848		
<u>Modified H.I.</u>				
Replications	4	326,712.4011	81,678.1003	10.8697**
Lines	19	169,301.9482	8,910.6289	1.1858
Error	76	571,083.8457	7,514.2611	
Total	99	1,067,098.9500		
<u>Percent Infected</u>				
Replications	4	11,366.5883	2,841.6471	18.3814**
Lines	19	46,811.9252	2,463.7855	15.9372**
Error	76	11,749.1177	154.5937	
Total	99	69,927.6312		

** Significant at the 1% level.

APPENDIX 16. Analysis of variance for slow rusting
results at site 2 (SP51) 1980.

Source	D.F.	Sums of squares	Mean squares	F Value
<u>AUDPC</u>				
Replications	4	12.7872	3.1968	5.3289**
Lines	19	116.3770	6.1251	10.2102**
Error	76	45.5930	0.5999	
Total	99	174.7572		
<u>Final Rust Sev.</u>				
Replications	4	12.8677	3.2169	4.9415**
Lines	19	76.7488	4.0394	6.2049**
Error	76	49.4776	0.6510	
Total	99	139.0941		
<u>Host Index</u>				
Replications	4	140,280.6668	35,070.1667	3.8952**
Lines	19	2,826,187.6896	148,746.7205	16.5213**
Error	76	684,253.8293	9,003.3399	
Total	99	3,650,722.1857		
<u>Modified H.I.</u>				
Replications	4	161,780.2208	40,445.0552	4.1695**
Lines	19	1,877,399.4815	98,810.4990	10.1865**
Error	76	737,210.1034	9,700.1329	
Total	99	2,776,389.8057		
<u>Percent Infected</u>				
Replications	4	177.6987	44.4247	1.3551
Lines	19	9,214.7404	484.9863	14.7940**
Error	76	2,491.4739	32.7826	
Total	99	11,883.9130		

** Significant at the 1% level.