CROWN RUST RESISTANCE IN WILD OATS:

Inheritance and Screening study

By

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CROWN RUST RESISTANCE IN WILD OATS:

INHERITANCE AND SCREENING STUDY

BY

TAYE ZEGEYE

A Thesis submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements for the degree of

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ABSTRACT

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Inheritance of crown rust resistance in three Iberian (IB) accessions of <u>Avena</u> <u>sterilis</u> (IB 2435 from Portugal, IB 3071 and IB 3076 form Spain) to two isolates of <u>Puccinia</u> <u>coronata</u> Corda <u>f</u>. <u>sp</u>. <u>avenae</u> (CR 13 and CR 50) was investigated. Genetic analysis on F_2, F_3, BC_1F_1 and BC_1F_2 populations from Calibre/IB 2435, Calibre/IB 3071 and Calibre/IB 3076 crosses supported the hypothesis that each of the three wild oat accessions possessed a single dominant gene conferring resistance to CR 13 and CR 50. The gene in IB 2435, gene 'A', was different from the genes in IB 3071 (gene 'B') and IB 3076 (gene 'C'). It was also different and independent from eight known Pc genes (Pc 38, Pc 39, Pc 48, Pc 58, Pc 59, Pc 61, Pc 64 and Pc 68). This gene may be new and previously unidentified. Gene 'B' and gene 'C' were the same or allelic or tightly linked to each other and also to Pc 68.

In an effort to identify new sources of crown rust resistance genes, one hundred and eighty four accessions of IB

collections were screened with seven crown rust isolates (CR 13, CR 25, CR 36, CR 50, CR 56, CR 77 and CR 107). Six accessions, three diploids, IB 2100-108, IB 2100-115 and IB 2100-128; one tetraploid, IB 3253 and two hexaploids, IB 2100-121 and IB 2472 were found to be resistant to all seven isolates. Another nine accessions, seven tetraploids (IB 581, IB 866, IB 922, IB 965, IB 980, IB 2394 and IB 3584) and two hexaploids (IB 2061 and IB 2100-132) were resistant to six of the seven isolates. Eleven other accessions which were resistant to five of the seven crown rust isolates were a diploid (IB 2252), five tetraploids (IB 641-A, IB 858, IB 947, IB 979 and IB 3335) and five hexaploids (IB 945, IB 961, IB 1112, IB 1113 and IB 3756). Of all the accessions screened, twelve were found to be susceptible to all seven crown rust isolates.

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1.0   INTRODUCTION

Oats are one of the major crops that have been cultivated for over 2000 years. Out of several species of oats, _Avena strigosa_ Schreb. (diploid), _Avena abyssinica_ Hochst. (tetraploid), _Avena byzantina_ C. Koch. and _Avena sativa_ L. (hexaploids) are the four cultivated species, with _Avena sativa_ L. being the major one. Oats are mostly cultivated in the temperate regions of the world. The former USSR, USA and Canada are the largest producers, contributing 42%, 14% and 8%, respectively, to world oat production (FAO, 1992).

Crown rust, _Puccinia coronata_ Corda f.sp. _avenae_ Eriks., is a major disease of oats causing significant world wide yield and quality reductions. Diversion of plant metabolites into spore production together with uncontrolled water loss through rust pustules, reduction of photosynthetic area and general decreased growth of oat plants due to crown rust leads to low yield and poor quality of oats.

In North America, control of crown rust has been through the use of resistant cultivars. This control measure is effective, efficient, economical and also environmentally friendly. In an effort to develop resistant varieties to crown rust, numerous accessions from several oat species have been tested for their resistance. New resistance genes were

then incorporated into desired cultivated oats to provide protection against the pathogen. However, the evolution of new and virulent race(s) necessitates the use of other resistance genes against the new race(s).

The search for other resistance genes, in addition to improving the yield and quality of oats, has been one of the major objectives of oat breeders. For this reason, identifying new sources of resistance has gone beyond the horizon of cultivated oats. Several expeditions to the Mediterranean region have taken place to collect wild oat species. Presently, Agriculture and Agri-Food Canada has one of the largest oat collections, maintaining over 6000 accessions of wild oats in the Winnipeg Research Centre and also in Plant Gene Resource Centre in Ottawa. About an equal number of Avena spp. are also maintained by USDA, ARS at Beltsville, MD (Briggle et al., 1975). Based on the studies already performed, these accessions are expected to possess a large number of resistance genes. Identifying these gene(s) and incorporating them into already established cultivars with desirable agronomic traits will definitely contribute to the endeavour of improving the quality and yield of oats. In line with the objectives of improving oat production in Canada, a research project was initiated with the following four major objectives:

1. To determine the number and nature of inheritance of resistance genes to crown rust in three hexaploid wild oat accessions.

2. To determine the relationships of the resistance genes in the three wild oat accessions with selected and known Pc genes.

3. To determine the relationships among the resistance genes in the three wild oat accessions.

4. To identify new sources of resistance genes to crown rust by screening wild oat collections with seven crown rust isolates.

2.0 LITERATURE REVIEW

2.1. Oats

2.1.1. Origin of oats

Oats are one of the oldest cereals grown by mankind. They seem to have been man's companion for some 4000 years. Archeological evidence indicated that oats have been known in Europe since 2000 B.C. Oats identified as _Avena strigosa_ Schreb., found in Egypt with belongings of the 12th dynasty in the years between 2000 B.C. and 1788 B.C. were probably the oldest oat grains known. There was no evidence, however, to suggest that they were cultivated (Coffman, 1961). The first written evidence about oats dates back to the time of the Greek philosophers and naturalists Aristotle (384 B.C. - 322 B.C.) and Theophrastus (371 B.C. - 286 B.C.). The physician and medical writer Diascorides (1st century A.D.) and Glan (130 A.D. - 200 A.D.) also wrote about oats.

The origin of cultivated oats has been a subject of debate for over a century. Some early workers suggested that _Avena sativa_ L. originated from _Avena fatua_ L. while others said it originated from _Avena sterilis_ L. Still others believed that _A. sativa_ was derived from _Avena byzantina_ C. Koch. (Coffman, 1961). Jones and Clifford (1983) suggested

the evolution of <u>A</u>. <u>sativa</u> through <u>A</u>. <u>sterilis</u> because of its wider distribution and aggressiveness. It seems that consensus favours this evolution.

It is not known where and when the culture of cultivated oats began. Cultivated <u>A</u>. <u>sativa</u> is thought to have spread from the middle east northward as a weed admixture with emmer wheat (Coffman, 1961). Coffman further suggested that known cultivation of oats goes back to the early period of the Christian era to the north of Greece and Italy as evidenced by Caligula (11 B.C.- 41 A.D.) who was "feeding his favourite horses with oats out of his golden cup". Coffman (1961), citing the 1885 report by Haussknecht, indicated that oat culture came earlier in northwest Europe than anywhere else. This was supported by the evidence that oat meal was being used by Germanic people in the first century A.D. On the other hand, citing Tuncer he also stated that oat cultivation was believed to have been started at an early date in Anatolia (Turkey). According to Baum (1977), the Chinese were cultivating oats between 386 and 534 A.D. or even earlier. It was not known when oats became an established crop in the different parts of the world but oats had become an important crop in the period between 500 and 1000 A.D.

Oats were brought to North America in 1602 (Coffman, 1961; Jones and Clifford, 1983). Russenholt in 1606 indicated

that oats together with wheat, rye, barley, hemp, peas, beans and garden vegetables were doing extraordinarily well. Coffman (1961) citing an 1853 report by Mason, indicated that oats were cultivated in Newfoundland in 1622. The Dutch settlers also grew oats in New Netherlands (Manhattan Island) prior to 1626. Oats were probably brought to the Pacific coast by Spanish priests either from Spain or Mexico since oat kernels of A. byzantina type were found in the ruins of the adobe missions erected in 1811 and 1834.

2.1.2. Classification

Oats are diploid (2n=2x=14), tetraploid (2n=4x=28) or hexaploid (2n=6x=42). Several workers classified oats by different methods and ended up with different classifications. For example, the 1890 report by Hackel cited by Baillie (1986) classified oats into 50 species. Baum (1977) puts them into 28 species while Rajhathy and Thomas (1974), based on ploidy, genome and unit of dispersal, classified them into 19 species (Table 2.1.2.1.). The genus Avena was designated by Linnaeus around 1753. However, the term Avena was first introduced by Bauhin from Plinius and subsequently by Tournefort during the early 17th century (Baum, 1977).

Table 2.1.2.1. _Avena_ species based on ploidy level, genome and
unit of dispersal.

| Ploidy | Genomes | Diaspore | | Cultivated |
		Florets	Spikelet	
Diploids 2n=2x=14	CpCp CvCv ApAp A1A1 AdAd AcAc AsAs	clauda Dur. prostrata Ladizinsky longiglumis Dur. damascena Rajhathy et Baum wiestii Steud. hirtula Lag.	pilosa M.Bieb Ventricosa Bal. canariensis Baum, Rajhathy & Sampson	strigosa Schreb.
Tetraploids 2n=4x=28	AABB AACC	barbata Pott vaviloviana Mordv.	magna Murphy & Terrell murphyi Ladizinsky	abyssinica Hochst.
Hexaploids 2n=6x=42	AACCDD	fatua L.	sterilis L.	byzantina C. Koch sativa L.

From Rajhathy and Thomas (1974)

2.1.3. Adaptation

Oats are one of the major grain crops of the world grown
primarily in the northern hemisphere, north of 40^0 latitude in
North America and north of 50^0 in Europe and Asia (Browning,
1973).   They are widely adapted over a range of climatic
conditions due to varietal diversity thriving best in a rather
cool and humid climate. Highest yields were obtained when the
growing season was long and comparatively cool. But generally
they are best suited to a cool and moist climate since they
are very sensitive to hot and dry weather during heading and

grain filling. Oats require more water than barley or wheat to produce a specific amount of dry matter (Schafer and Gaines, 1916). With the exception of rice, oats required more moisture than any other cereal to produce a given unit of dry matter (Coffman, 1961).

2.1.4. Production

Schafer et al. (1923) stated that in the USA 10% of the land devoted to cereals was planted to oats. Levels of oat production seemed to fluctuate depending on the damage caused by pests in a particular year and also depending on the area sown to oats. World oat production has declined by 18% since 1979 mainly because oat cultivated area was shrinking. In Canada, for example, production in 1992 was 6% lower than that of 1979 because the area under oats, which was 40% of the total oat area in North America (1,238,000 Ha), was 18% of that of 1979 (FAO, 1992). In spite of declining oat areas, improvements in agronomic practices and plant breeding have resulted in yield per hectare increases. Yield in Canada (2.28 tonnes/ha) was consistently higher than the US since 1979 except for 1992 when the summer was unusually cool and wet (FAO, 1992). World oat production in 1992 was almost 34 million tonnes, with 21% (7,199,000 tonnes) being produced in North America. Canada produced approximately 8% (2,823,000 tonnes) of the total world production (Anonymous, 1992; FAO,

1992). This made Canada the third largest producer of oats next to the former USSR and the USA. Canadian oat production for 1992/93 was estimated by the National Grain Bureau (1993) to be 2,821,000 tonnes.

Major oat producing countries seed over 80% of total oat acreage with varieties of A. sativa. Among the eight important cereals, oats consistently ranked fifth. Wheat and barley were always ranked as first and fourth, respectively, while maize and rice interchange the second and the third places, respectively, (Coffman, 1961). The relative area under oat cultivation varies from one country to another. In the USA for example, oats ranked third in total cultivated area, next to corn and wheat. It was, however, the fourth important crop by production preceded by corn, wheat and sorghum (Browning, 1973).

2.1.5. End use of oats

Oats, despite being high in protein, 16% - 19% for A. sativa (Browning, 1973; Jones and Clifford, 1983), 22.1% - 31.4% for A. sterilis (Briggle et, al., 1975) and 16% - 27% for A. fatua (Rines, et al., 1980) and high in fat - 8% (Browning, 1973; Jones and Clifford, 1983), and despite having the best amino acid balance compared to any other grains (Browning, 1973), have only 6% of the total oat production

going directly for human consumption. A primary reason for this is that it has high hull content (20 - 25%). Nevertheless, the National Grain Bureau (1993), indicated that in the years 1990/91, 1991/92 and 1992/93, 86%, 103% and 78%, respectively, of total oat production was used domestically. Oats have been also used as fodder or forage in the Mediterranean area, Asia minor and western Asia and as a forage and for human consumption in Ethiopia as early as the first century A.D.

2.1.6. Oat crossing

Oats are self pollinated crops with little natural out crossing. Morey (1949) found that on the average 0.25% natural out crossing occurred in Clinton oats. Citing the work of others, Morey reported that natural out crossing ranged from 0.07% to 9.82%.

In comparison to wheat and barley, crossing oats is relatively difficult. Seed set failure in interspecific crosses were due to lack of normal cell differentiation and endosperm degeneration (Brown, 1964). Brown and Shands (1956) reported that by removing one anther and by just opening and closing the florets in a couple of days to simulate pollination but without applying foreign pollen, a 30% - 50% reduction in seed set resulted on days where the temperature was over 34.5^{0}C. The same operation on cool days, 21^{0}C - 27^{0}C,

resulted in a 5% - 10% reduction. A negative correlation between seed set and maximum temperature on days of emasculation, pollination and also on number of days between emasculation and pollination was also reported (Brown and Shands, 1956). Coffman (1956) showed that bagging possibly reduced seed set by 12% because of increased temperature under high light intensity. The duration between emasculation and pollination has a significant effect on seed set. There was no seed set when pollination was done four days after emasculation compared to 80% seed set over a two day interval between emasculation and pollination (Brown and Shands, 1956). In addition, more seed set resulted in the top florets after short intervals compared to lower florets that had more seed set at long intervals. Coffman (1937) reported that afternoon pollination resulted in more than double seed set compared to morning pollination. Satisfactory results were also obtained when emasculated in the morning and pollinated in the afternoon.

The most effective period for hand pollination of A. sativa was the time of natural anthesis which under normal field conditions occurs between two and five pm. On hot days, pollen grains were shed late in the day, 5:00 pm to 7:00 pm. When maximum temperature was above 34^{0}C, a 360% increase in seed set was obtained when crossing was done between 4:00 pm and 7:30 pm compared to between 2:00 pm and 5:00 pm (Coffman and Stevens, 1951).

The success of oat pollination can be determined two to three days after pollination by noting a wilted stigma and an enlarged ovary (Fehr, 1980). In common oats, pollen germinates and germ tube enters the style within five minutes after pollination. Temperature has a marked influence on the rate of tube growth, $30^{\circ}C$ resulting in the fastest tube growth and cell division (Brown and Shands, 1957). Lower pollen germination could probably account for lower seed set after hand pollination because more pollen lands on the stigmas in natural pollination from the three anthers in a floret.

Other factors like evaporation, time of the day and high wind velocity are also important factors that would possibly result in seed set failure. High evaporation days are days of lowest seed set. Very high wind velocity may cause excessive whipping about and drying of the flowers (Coffman, 1937). Floret position also affect success of pollination. The top three to four florets show much more receptivity on the day of pollination than the lower florets. Under field conditions, if crossing is to be completed on the same day, the top three to five florets could be used (Brown and Shands, 1956). Also, no more than five spikelets are in a condition to be pollinated on the same day (Coffman, 1937).

2.1.7. A differential set

Differential rust reactions to races of P.coronata was first observed by Hoerner (1919) on seven oat cultivars. A set of cultivars or lines showing different reactions to crown rust races that helps to differentiate between races is useful. Such a set initially consisting of 11 cultivars and later on increased to 13 (Simons, 1970) has been in use since 1935. Weetman (1942) reported the differential reaction of three oat varieties. By late 1940's, due to the appearance of new crown rust races and also due to increased virulence on the standard differential set of 1935, asubsequent set of ten cultivars was introduced and has been in use since 1954. The ten varieties of the differential set were: Anthony, Victoria, Appler, Bond, Landhafer, Santa Fe, Ukraine, Trispernia, Bondvic and Saia (Harder, 1979). On these new differentials, 77% of the isolates were avirulent on all ten cultivars or virulent on one only. About 8% of the isolates were virulent on two or more cultivars. Most of these cultivars became susceptible by 1970. Once again a new differential set containing single crown rust resistance gene Pc lines, all derived from A. sterilis were produced by backcrossing to an A. sativa cultivar with Pendek being the cultivar commonly used. This set has been in use since 1974 (Harder, 1975; Harder, 1979; Chong and Kolmer, 1993).   The number of differential lines was increased from 12 in 1978 to 19 in 1979 so as to help identify more virulence combinations (Harder,

1980). Presently the differential set used at the Winnipeg Research Centre consists of 20 single gene Pc lines, Pc 35, Pc 38, Pc 39, Pc 40, Pc 45, Pc 46, Pc 48, Pc 50, Pc 54, Pc 55, Pc 56, Pc 58, Pc 59, Pc 60, Pc 61, Pc 62, Pc 63, Pc 64, Pc 67 and Pc 68, (Chong and Seaman, 1993).

2.2. Crown rust

2.2.1. History

Archeological findings in Israel showed that the oldest rust spores ever reported, 3000 years old, were from wheat. The Romans believed that a pair of Gods, Robigo and Robigus, punished human beings with rust infected crops. Even though rust was observed by farmers for over 2000 years, it was Aristotle (384 B.C. - 322 B.C.) and Theophrastus (371 B.C. - 286 B.C.) who described rust diseases on crops. Robert Hooke in 1665 published the first illustrations of rust on a rose leaf (Schumann, 1991).

Simons (1970) reported that Tozzetti in 1767, was the first to publish observations on crown rust. In 1791, Persoon described the aecial stage and established the genus *Puccinia* while Corda in 1837 described the telial stage and named it *Puccinia coronata*. DeBary found the aecial stage of *P. coronata* on *Rhamnus* in 1867. In 1875, Nielson proved *P. coronata* to be heteroecious by using aeciospores from *Rhamnus*

cathartica to infect oat plants. It was Cornu in 1880 who conclusively demonstrated the connection between _P. coronata_ on _Rhamnus_ and on oats by infecting seedling oats with aeciospores from buckthorn. By 1889 it was reported that _P. coronata_ was on 22 species of wild grasses and three species of _Rhamnus_. It was at this time that Rostrup, cited by Simons (1970), observed differential susceptibility of varieties of rye grass which indicated that there was varietal resistance. Though it was not established whether it was due to stem rust or crown rust, the first record of rust in North America was in 1858. In 1872, Peck specifically listed _P. coronata_ as a new species for New York (Simon, 1970).

2.2.2. Classification

Crown rust fungus belongs to the division _Eumycota_, Subdivision _Basidiomycotina_, class _basidiomycetes_, subclass _Teliomycetidae_, order _Uredinales_, family _Pucciniaceae_ and genus _Puccinia_ (Littlefield, 1981). Rust is not a single unit pathologically. Rusts attacking one crop or one variety might not necessarily attack other crops or varieties. Yet the morphological differences are small and insignificant, thus these pathogenic variants were regarded as _formae speciales_ (Johnson, 1961 citing J. Eriksson, 1894). Therefore, crown rust fungus was identified as _Puccinia coronata_ Cda _f. sp. avenae_ Eriks.

2.2.3. Life cycle

Crown rust is an obligate parasite. It is also a heteroecious fungus spending part of its life cycle (uredial and telial stage) on oats and grasses and the other part (aecial stage) on the alternate host, common buckthorn, Rhamnus cathartica L. (Figure 1). The uredial stage is dikaryotic (N + N), with each cell containing two nuclei. As the host matures the black teliospores (N + N), the overwintering stage, are formed. Nuclear fusion occurs in maturing teliospores to form a temporary diploid (2N) cells. This will then be followed by meiosis resulting in four haploid basidiospores belonging to two different mating types, (+) and (-). After about two weeks, the basidiospores produce mycelium which can infect the alternate host to produce pycnia. The pycnia of opposite mating types fuse to form dikaryotic (N + N) hyphae once again. These dikaryotic hyphae infect oats and grasses and can produce urediospores indefinitely if weather conditions permit. As the host matures, teliospores are produced completing the life cycle. Under favourable temperature and moisture conditions, repeated cycles of infection by urediospores can occur in as little as seven days (Johnson, 1961; Frankton, 1967; Littlefield, 1981).

The fungus is adapted to a fairly wide temperature range but the generation time becomes correspondingly longer as

temperature decrease below the optimum of about 25°C (Jones and Clifford, 1983). It also persists from season to season in the warmer climate by continuous production of urediospores which could also overwinter and germinate on grasses. Johnson and Green (1952) reported that 2%-20% germination was recorded on couch grass, <u>Agropyron</u> <u>repens</u> L. In areas where the fungus does not overwinter due to harsh cold winter, the disease is initiated by spores carried by wind currents from warmer areas, or from aeciospores originated from the alternate host.
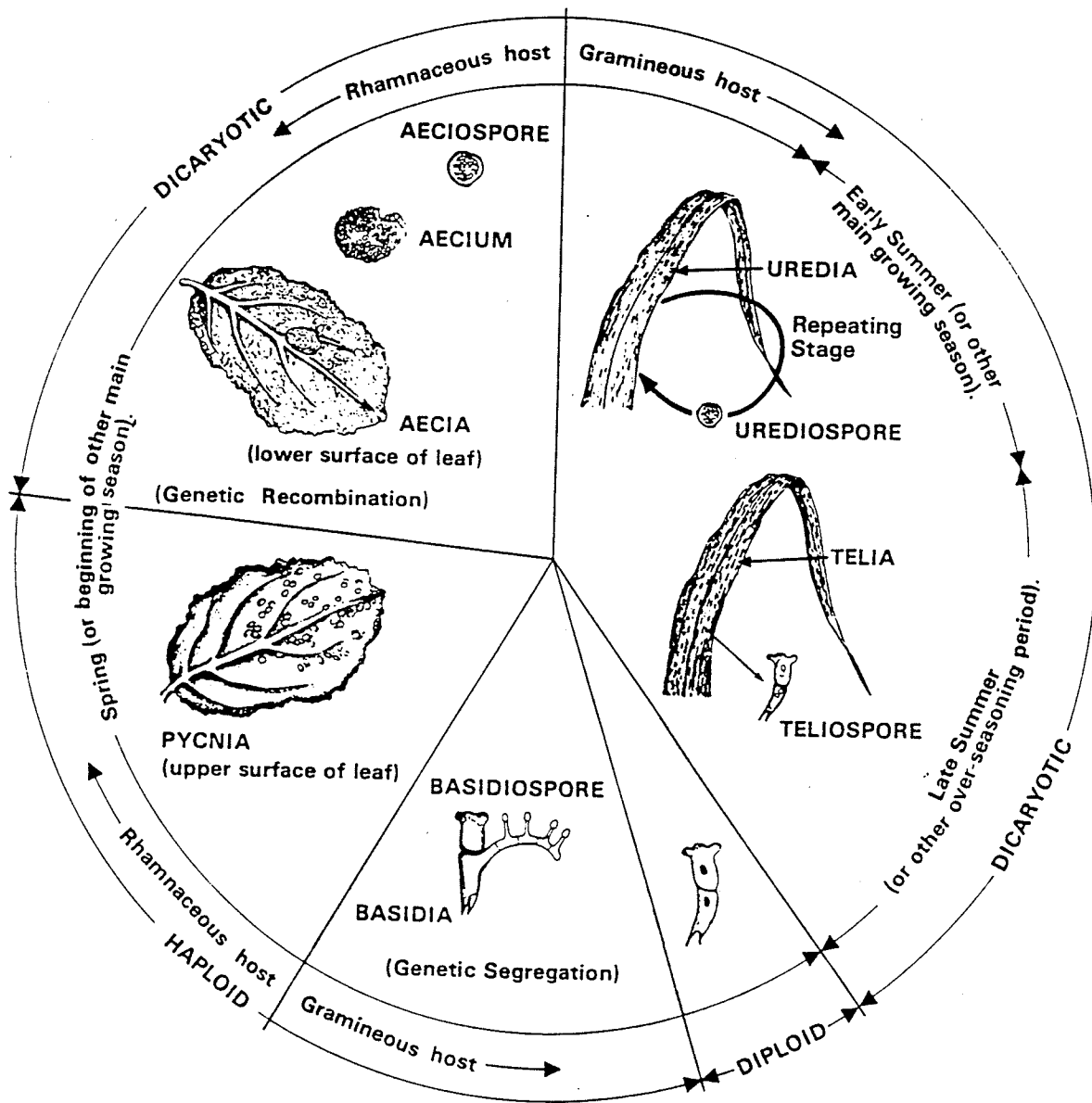
Figure 1. Life cycle of *Puccinia coronata*
(Simons, 1970)

2.2.4. Distribution

Crown rust, caused by the fungus _Puccinia coronata_ Cda _f._ _sp._ _avenae_ Eriks., is the most serious disease of oats occurring nearly world wide. The distribution includes islands far from any major land mass (Simons, 1985b). It is important in temperate and humid regions of oat culture. In North America crown rust is widespread in the south, mid and north central USA, up to Canada making a broad band of _Puccinia_ path (Figure 2). The aecial stage of this fungus has been reported from all major oat producing areas of the northern hemisphere where _Rhamnaceous_ hosts occur in proximity to oats (Harder, 1978; Simons, 1985b). Harder (1978) further reported that a single large buckthorn shrub was sufficient to generate a moderately severe infections in an adjacent oat field. From yearly crown rust surveys, crown rust infection in the Prairies and eastern Canada was reported to range from trace amounts in some fields to near destruction of the crop in others (Harder, 1978). Even though susceptible _Rhamnus_ species are rare or non-existent in South America and Australia, thus lacking the presence of the aecial stage (Simons, 1985b), the disease is still important in these areas, particularly in the cool and moist coastal regions (Jones and Clifford, 1983).
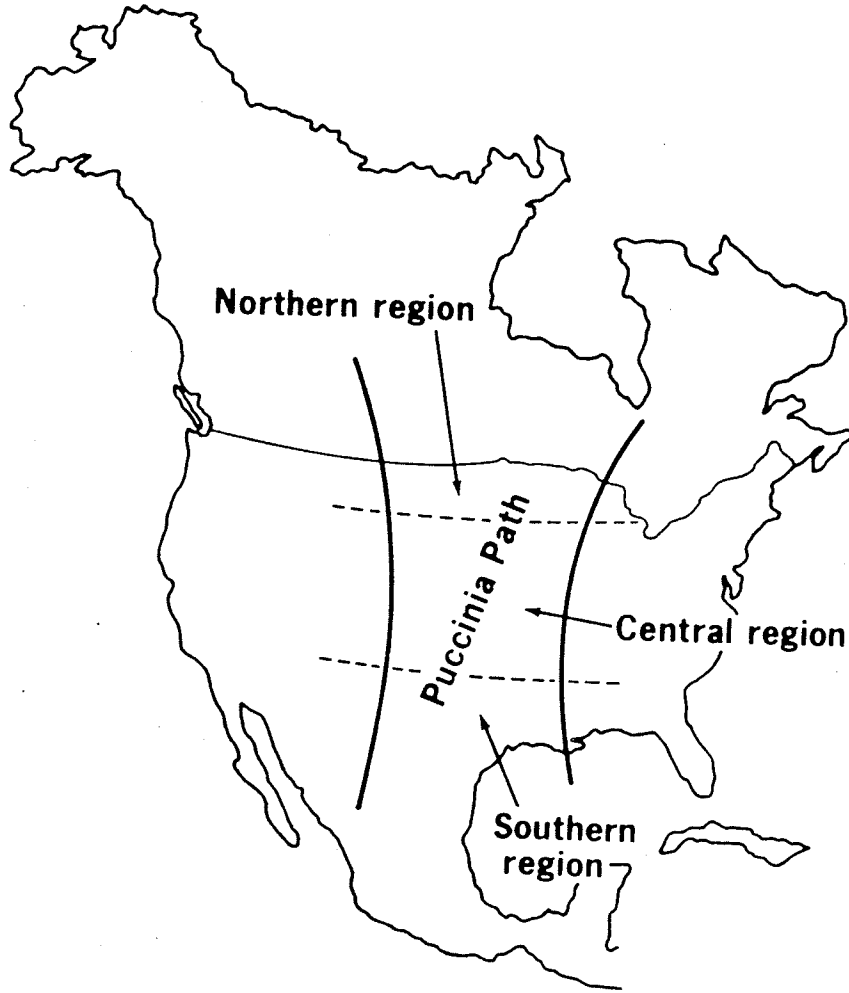
Figure 2. _Puccinia_ path in North America
(Frey et al., 1973)

2.2.5. Effects of crown rust

Crown rust has been recognized as a serious oat disease for over two centuries. It affects oat plants by decreasing photosynthetic leaf area, increasing transpiration rate and increasing diversion of plant metabolites from grain to spore production. In addition to rust pustules permitting uncontrolled water loss, they also weaken the stem causing breakage or lodging. Murphy (1935a) reported that water requirements of plants inoculated at the seedling stage was four times greater than their respective checks. When plants were inoculated at anthesis, their water requirements were 42% higher than their checks. The longer the duration and the greater the degree of infection, the higher will be the water requirement.

Early infection (seedling to early boot stage) of a susceptible variety resulted in failure to head while plants inoculated at early anthesis matured four days later than rust free plants. Plants from a resistant variety inoculated at different stages headed but date of ripening was delayed. Infected plants produce fewer tillers and fewer seeds/tiller, resulting in reduced grain yield of poor quality. Early report on crop loss due to the abundance of crown rust was reported by Carleton (1905) by comparing the yield of two varieties. He found that the yield of the variety Swedish

Select, a variety that normally outweigh the variety Sixty Day, was lower by 1257 kilograms ha^{-1}. Early and severe infection was negatively correlated with yield, plumpness, date of heading and height of oats (Murphy, 1935a). Mung, cited by Osler and Hays (1953), however, did not find an association between rust reactions and date of heading. Harder and Clark (1981) reported mean reductions for yield and kernel weight to be 40% and 23%, respectively, while the relative increase of hull to groat was 31%.

Yield losses to crown rust have been well documented over the years. In the USA, over a 20 year period (1919 - 1939) yield reduction due to crown rust was more than twice of that caused by stem rust. In the years 1934 and 1938, yield reductions were 20% and 24%, respectively (Murphy et al., 1940). For early and late seeding, 28% and 50% losses, respectively, have been reported (Fleischmann and McKenzie, 1965). Yield losses of 20% (Jones and Clifford, 1983) and 30% (Martens et al., 1972; Simons, 1985a) have also been reported. The estimated average annual loss from all oat diseases in the US during 1951 - 60 was 21% (Zillinsky and Murphy, 1967). Even though crown rust has little effect on the crude chemical composition of either kernels or hulls (Murphy, 1936), the end result of severe infection is often grain of poor quality due to lack of starch and abundance of cellulosic material (Agrios, 1988).

2.2.6. Host-pathogen interaction

In the study of host-pathogen interactions, postulating the concept that a gene for virulence has a corresponding gene for resistance in the host was a significant step forward. Flor (1956) was the first to show these relationships in genes for resistance in flax (<u>Linum</u> <u>usitatissimum</u> L.) and genes for pathogenicity in flax rust (<u>Melampsora</u> <u>lini</u> Desm.). The host plant was resistant if it had a given gene for resistance and was only rusted by a rust race that had a corresponding gene for virulence. If the host had two genes for resistance, it would only be rusted by a race or races with two corresponding genes for virulence (Johnson, 1961). The host-parasite interaction has a genetic basis in both the host and the pathogen. Host varieties differ greatly in their physiological processes, which are conditioned by genes. Some of these gene-conditioned processes were incompatible with the growth of a particular rust on the host. Rust races also differed in their physiological processes, which likewise are gene-conditioned. Johnson (1961) explaining the reasons for susceptibility, said "compatibility of the gene-conditioned physiology of the host with the gene conditioned-physiology of the rust leads to susceptibility hence the physiological interlocking of the genes of host and parasite".

A single rust resistance gene might condition resistance to a large group of rust races. There are two types of resistance, seedling resistance and adult/field resistance. These two types of resistance are independently inherited, as plants which were susceptible to some races in the seedling stage were found to be resistant to the same races in the adult stage and vice versa. Major genes were found to be inherited in accordance with the Mendelian laws (Johnson, 1961). Adult resistance is usually controlled by several genes making gene transfer difficult. The mechanism of infection both in seedling and adult resistance is the same except there is a significant difference with respect to hyphal length, uredia cm^{-1} and latent period (Luke, et al., 1984). There is another form in which resistance was also expressed in cultivars with Victoria parentage. These cultivars were susceptible to leaf blight which was caused by the fungus, Helminthosporium victoriae. The dead or injured cells in reaction to Puccinia coronata were readily available food for the H. victoriae thereby becoming a physiological barrier for P. coronata thus making it resistant to crown rust (Litzenberger, 1949; Osler and Hayes, 1953; Simons, 1956b).

2.2.7. Rust scoring scale

A disease is expressed when a disease causing pathogen is present in its host under favourable environmental conditions.

Temperature and relative humidity are important environmental components for disease development. For example, Pc 67 is temperature sensitive (Chong and Kolmer, 1993) and its resistance becomes ineffective above 25°C (Chong personal communication). Martens et al., (1979) reported that resistances of two stem rust resistance genes (Pg 13 and Pg 15) were expressed within 20-25°C while resistance of Pg 16 was not fully expressed at a temperature above 22°C.

Table 2.2.7.1. Infection types of crown rust (Murphy, 1935b)

Symbol	Host reaction	Infection type
0	Immune	No macroscopic evidence of infection
;	Nearly immune	No uredia formed; necrotic areas or chlorotic flecks present
1	Highly resistant	Uredia few, small, always in necrotic areas; also more less necrotic areas produced without the development of uredia
2	Moderately resistant	Uredia fairly abundant, small and mid sized, always in necrotic areas seldom without uredia
3	Moderately susceptible	Uredia abundant, mid sized and surrounded by chlorotic areas; necrotic areas entirely absent
4	Completely susceptible	Uredia abundant, large; no necrosis or chlorosis immediately surrounding uredia

A rust rating scale, taking the different reactions into account, is quite useful. Such a scale was developed by Murphy (1935b) and is still in use with some changes. The scale used to evaluate rust reactions in this study is shown on table 2.2.7.1. Reaction types 0 - 2 were grouped as resistant while reaction types 3 and 4 were susceptible. Reaction types ;, 1 and 2 were always associated with necrotic tissues, which is absent in reaction types 3 and 4.

2.2.8. Virulence

Different geographical locations support different crown rust isolates. Kolmer and Chong (1993) reported two distinct populations of crown rust fungus in Canada. One of the populations is in Ontario and Quebec while the other one is in the Prairie region of southern Manitoba and eastern Saskatchewan. Previous reports also indicated that different frequencies of virulences occurred in the west (on Pc 35, Pc 40 and Pc 46) and in the east (on Pc 35 and Pc 56) (Harder, 1978; Chong 1988b). In western Canada, crown rust inoculum originates, in addition to buckthorn, from over-wintering rust in Mexico and the southern USA and arrives in Canada as showers of urediospores carried by air currents. In Ontario, the source of inoculum was mostly from buckthorn (Harder, 1978; Harder, 1979; Chong, 1988a; Chong and Seaman, 1989).

Apart from geographical locations, other factors could also account for independent epidemiological units. North and South islands of New Zealand are separated by 100 kilometres only and yet the two islands have different virulence combinations. All year-round availability of host plants, north to south crop maturity progression, few winds in the same direction and dissimilar climatic conditions may account for the difference (Martens, et al., 1977).

In the 1980's, reduced crown rust incidence was observed in western Canada. This could have been due to less air-borne inoculum from the USA, reduced oat cultivation, hot and dry weather (Chong and Seaman, 1989) and also due to wide use of resistant cultivars (Chong, 1988a). In the east, favourable weather conditions, the presence of buckthorn and the use of susceptible cultivars resulted in a high level of infection that led to higher yield loss (Harder, 1982; Chong and Seaman, 1991).

Crown rust virulence has changed over time. In the 1940's susceptibility of variety Bond to races of crown rust was on the increase (Osler and Hayes, 1953). The variety Garry which was released as a resistant cultivar in 1947 was withdrawn in 1952 because crown rust races virulent to the resistance genes in Garry became predominant. A new race virulent on varieties Anthony and Saia was identified in 1955

(Simons, 1956a). The resistant varieties Landhafer and Santa Fe, introduced in 1938 and 1945, respectively, (Osler and Hayes, 1953) no longer adequately provided resistance to North American crown rust races by 1961 (McKenzie, 1961; Dyck and Zillinsky, 1963). By 1964, 50% of the races of the area were capable of attacking the two varieties (McKenzie and Fleischmann, 1964).

In the late 1960's Pc 38 and Pc 39 were highly resistant to crown rust isolates of the Prairies (Fleischmann, 1972; Samborski and McKenzie, 1972; Harder, 1976; Harder, 1978). However, isolates that were virulent on Pc 38 were on the increase by the mid 1970's (Harder and McKenzie, 1974). Increase in virulence on Pc 40 from 4.2% and 1.2% in eastern and western Canada, respectively, in 1973 to 48.3% and 14.5% in 1974 was observed (Harder, 1975). Increased virulence was also observed on Pc 50 and Pc 56 (Harder, 1976; Harder, 1978).

The variety TAM 0-301 (Pc 58) which was 100% resistant to all isolates in Texas area in 1974 became 19% susceptible, 64% moderately resistant and only 17% resistant to the isolates of the area by 1980 (Simons and McDaniel, 1983). In Manitoba, virulence on Pc 46 increased from 12.4% in 1979 to 73.6% in 1985 (Chong and Kolmer, 1993). Hudson, the new and most resistant commercial cultivar introduced in Manitoba in the mid 1970's was attacked by 10% and 28% crown rust isolates in

western and eastern Canada, respectively, (Harder, 1976).
Virulence on this cultivar increased from 10% in 1980 to 31%
in 1981 (Harder, 1982) and in 1984 it was removed from the
recommended cultivars for Manitoba (Chong, 1984). In eastern
Canada, virulence on Pc 67 was up to 24.2% in 1985 compared to
1% in 1984 (Chong, 1986).

Cultivars with Pc 39 were resistant to crown rust for a
long time. However, Fidler (Pc 39) was first attacked in 1982
(Harder and Chong, 1983) and virulence on Pc 39 became wide
spread by 1987 (Chong 1988a). In eastern Canada, the
resistant variety Woodstock (Pc 39) that was released in 1982
was first attacked by an isolate in 1985. In 1989, this
variety was withdrawn because 87% of the isolates of the area
in 1988 were virulent on Pc 39 (Chong, 1988a; Chong and
Seaman, 1989). The build up and wider distribution of races
virulent to Pc 39 represent a significant threat to any
cultivar relying on this gene for protection (Chong, 1988b).
By 1990, virulence on Pc 39 has remained high at 75% in
eastern Canada while in the west it increased to 44% from 0%
in 1974 (Chong and Seaman, 1991).

No virulence was detected on lines containing both Pc 38
& Pc 39 (Harder and McKenzie, 1974; Harder, 1976; Harder and
Clark, 1981) until 1987 when two virulent isolates were found
(Chong, 1988a). By 1989, 21.3% of crown rust isolates

isolated in the Prairies became capable of attacking the two genes (Pc 38 and Pc 39), 28 of the isolates were virulent on Dumont (Chong, 1990; Chong and Seaman, 1990). Virulence to this two gene combination went up to 43% and 47% in 1990 and 1991, respectively (Chong and Seaman, 1993). Cultivars Fidler (Pc 39) and Dumont (Pc 38 and Pc 39) which are very important cultivars in western Canada because the two cultivars account for 75%, 81%, 79% and 70% of the total area under oats for the years 1984, 1985, 1987 and 1990, respectively (Chong, 1985; Chong 1986; Chong and Kolmer, 1993), were attacked by crown rust isolates in 1987 (Chong 1988a; Chong 1988b). Considering isolates with virulence to this gene combination were first isolated in 1987 (Chong 1988a) and also considering cultivars like Dumont, Robert and Riel that are widely grown in western Canada rely on Pc 38 and Pc 39 for their protection, it is a very serious concern and even a threat for oat production in western Canada. The concern becomes even more alarming when the dominant gene Pc 68, a gene which conferred resistance to all isolates in Canada for many years (Harder, et al., 1984) was , for the first time, found to be attacked by some isolates in 1991 (Chong and Seaman, 1993).

Decrease of virulence for certain races, for example, race 264 on varieties Victoria, Trispernia and Bondvic was observed (Fleischmann, 1972). Chong (1988b) also reported the change in frequency of virulence from Pc 35 and Pc 56 to Pc 39

and Pc 55. This decrease of virulence and change of frequency may be due to the lesser competitive ability of the existing races compared to the newer and virulent ones that were results of selection pressure. Selection pressure favours the most virulent races on the prevailing varieties (Fleischmann, 1963). For example, races 202, 203 and 213 were 45%, 15% and 14% prevalent, respectively, in 1953 (Simons, 1954) and 30%, 21% and 8%, respectively, in 1954 (Simons, 1955). These races predominate mainly because they were aggressive and more competitive on the cultivars that were grown compared to the low prevalence (0.2%-6.2%) of the other races. It has been observed that virulence increased with the introduction of resistance genes eventually making the resistance genes ineffective. For example, wide spread use of cultivars with Pc 59 and Pc 60 resulted in increased virulence on Pc 59 and Pc 60 (Chong and Kolmer, 1993). Virulence on other genes that were not included in commercial cultivars remained low.

Therefore, since changes of virulence and frequency of crown rust races was observed over the years, it was necessary to monitor these changes. For this reason, crown rust surveys were conducted annually beginning in 1927 in the US and 1929 in Winnipeg, Canada (Chong and Kolmer, 1993).

2.2.9. New races

New crown rust races continually appear either through genetic recombination, mutation, somatic hybridization or heterokaryosis (Johnson, 1961). In this process, cultivars with no crown rust resistance could favour simple races to predominate while cultivars with resistance genes might favour complex races (Brouwer and Oates, 1986). Buckthorn was of a special concern not only because it accelerates epiphytotics but also because of the possibility of sexual recombination of fungi occurring on it. There was a higher probability of a new race capable of attacking resistant cultivars appearing in areas where buckthorn was present (Harder and McKenzie, 1974). Even though the alternate host largely contributes to greater variability due to hybridization and genetic recombination (Fleischmann, 1965), genetic variability also occurred in the absence of the alternate host (Brouwer and Oates, 1986). Fleischmann (1963,1964) also attributed the evolution of a race to mutation (race 332 from race 216) as there was no buckthorn in nearby oat fields.

The work of Fleischmann (1964) on genetic variability as expressed by race to isolate ratio, showed that eastern Canada has higher variability (1:3.8) than the Prairies (1:5.5). This was due to hybridization and recombination of the crown rust fungus occurring on the alternate host. Higher

variability as expressed by low race to isolate ratio with aecial culture (1:1.8) was obtained from the alternate host as opposed to the high ratio (1:3) from the uredial culture. Contrary to the finding of Fleischmann (1964), Chong and Kolmer (1993) reported that phenotypic diversity in the Prairie population to have been higher than eastern population for the period between 1974 - 1990 except for 1977. It was explained that this higher phenotypic diversity might have been due to P. coronata found in grasses and wild oat species and contributing through the sexual cycle. In addition, it could have been due to urediospores originating from the aeciospores of buckthorn in the USA and carried by wind current to the Prairies (Chong and Kolmer, 1993; Kolmer and Chong, 1993).

2.2.10. Control

One of the control measures used against crown rust was the practice of cultural control, ie, early seeding and/or the use of early maturing varieties. Eradication of the alternate host was also another cultural control measure (Jones and Clifford, 1983). However, fields in remote islands with no alternate hosts were found infected with crown rust (Simons, 1970).

The use of multilines was another cultural control measure that resulted in little or no yield loss (Browning and Frey, 1981). Multilines consist of mechanically mixed isolines each containing a single or few major genes conferring resistance to crown rust. It is a convenient way of using diversity in commercial crops for which agronomic uniformity is required. In fact, it is the deliberate introduction of heterogeneity without compromising for yield and quality by introducing resistance from a unique source into a common parent line by backcrossing. In addition to countering a known threat from one parasite, multiline varieties have the potential of countering unpredicted threats (Day, 1974).

There are advantages in using multilines as a control measure. Firstly, if one isoline is susceptible to a specific race, the initial inoculum and thus the disease will be lower than a field seeded to a single susceptible variety. Secondly, the rate of infection decreases because of the likelihood of a resistant isoline growing next to a susceptible one. Thirdly, if one of the isolines was found to be susceptible and significantly affecting yield, then that isoline could be replaced with another isoline the following year (Browning and Frey, 1981; Michel and Simons, 1983; Chong, 1988a).

Chemical control has also been used to control crown rust. Clark (1968) reported that a rusted plot yielded 20% less than plots treated with Daconil 2787 (sprayed four times at weekly intervals at a rate of 900g 75% WP/380 litres of water).

The use of crown rust resistant varieties is by far the most widely used method of controlling the disease in North America because it is economical, effective, efficient and environmentally acceptable (Sanderson, 1960; Harder, 1982; Michel and Simons, 1983; Martens et al., 1988; Chong and Seaman, 1993).

2.2.11. Source of resistance genes

The development of a superior cultivar requires as long as 10-12 years. It could even be longer when several different types of resistance to a number of diseases has to be combined with all the desirable agronomic traits (Martens et al., 1988). Desirable traits like high yield, disease resistance, kernel quality, etc., are not always available in the world collection of cultivated oats. Genetic variability is required to overcome major challenges in oat improvement. Improvement in yield and other agronomic characters of cultivated oats have been accomplished primarily by utilizing the genetic variations within A. sativa (Sharma and Forsberg,

1977). However, there were also other sources of such variations in diploid and tetraploid species (Dyck and Zillinsky 1963; McKenzie and Fleischmann, 1964). Several crown rust resistant strains were found in A. strigosa (diploid), A. abyssinica (tetraploid) and Avena barbata Pott. (tetraploid) (Simons et al., 1959; McKenzie and Fleischmann, 1964). The difficulty of transferring resistance genes, due to mitotic instability and/or incompatibility (Dyck and Zillinsky, 1963) and lack of pairing between chromosomes of hexaploid and the donor species (Sharma and Forsberg, 1977) from lower ploidy level into commercially acceptable hexaploid varieties have been reported. Instability for resistance due to monosomic alien substitution (MAS) lines, which may have resulted from lack of chromosome transmission through the pollen, was also reported (Sharma and Forsberg, 1977). Nevertheless, Zillinsky & Derick (1960) and Dyck & Zillinsky (1963) successfully transferred two genes for crown rust resistance from diploid to hexaploid oats, while Sharma and Forsberg (1977) transferred a crown rust resistance gene from a tetraploid into A. sativa.

In the 1960's it became increasingly difficult to obtain a cultivated hexaploid germplasm possessing a satisfactory level of crown rust resistance. Varieties known to possess resistance genes that were considered to be of marginal value were even considered (Martens, et al., 1968). This did not

help in identifying new sources of crown rust resistance genes. Oat breeding has reached the stage where all variations in oats including wild oats have to be taken into account if further improvement is to be made (Rajhathy et al., 1966).

Since populations of wild species exist in equilibrium with pathogens, they must possess an effective protection mechanism which enables them to survive and reproduce (Zillinsky and Murphy, 1967). Therefore, wild species have been collected in different expeditions with the primary objective of looking for new sources of disease resistance (Martens et al., 1980). Also, A. byzantina and A. sterilis , which are fully compatible with A. sativa , became viable components of progressive oat breeding programs because of ease of genetic transfer (Sharma and Forsberg, 1977).

Crown rust resistance in wild oats has not been widely exploited for breeding programs until 1962 when Suneson and Miller released a variety, the improved Sierra variety, from a cross between A. sativa and A. fatua (McKenzie and Fleischmann, 1964). Differential reactions to cultures of several crown rust races among selections of A. sterilis suggested the presence of several genes conditioning resistance to crown rust. The genetic variability for reaction to disease, displayed by natural populations from diverse geographical areas, suggested that these populations

have new genes useful in improvement of cultivated oat varieties (Zillinsky and Murphy, 1967). <u>A</u>. <u>sterilis</u> , which is the second most common oat species next to <u>A</u>. <u>barbata</u> (Rajhathy et al. 1966), represents a vast living gene pool, that is extremely variable, self reproducing and continually changing with pressure of environment (Zillinsky and Murphy ,1967). Wahl (1970) also expressed the view that the aggressive <u>A</u>. <u>sterilis</u> constituted a rich and heterogeneous reservoir of new and readily usable genes for resistance to crown rust, both in seedling and adult stages. He reported that 16% of seeds of adult plants grown from seeds collected in Israel had field resistance to virulent races of <u>Puccinia</u> <u>coronata</u>.

Populations surviving generations of rust infestations, frequently in the presence of the alternate hosts, are more likely to be rich in genes for resistance and/or tolerance to races of rust (Kiehn, 1976). Accessions of <u>A</u>. <u>sterilis</u> from Iran, North Africa, Israel and the Mediterranean region provided resistance to North American crown rust races (Simons, 1965; Fleischmann et al., 1971a; Kiehn, et al., 1976; Harder et al., 1980). As a result several resistance genes were transferred from <u>A</u>. <u>sterilis</u> into common oats (Martens, et al., 1980). Harder et al., (1992) reported that thus far thirty five crown rust resistance genes have been isolated from wild oats. <u>A</u>. <u>sterilis</u> derived resistance genes were not known to be present in <u>A</u>. <u>fatua</u>.

2.2.12. Linkage of resistance genes

Linkage, association and allelism of crown rust resistance genes and also crown rust resistance genes to certain agronomic traits is known to exist. For example, Sanderson (1960) reported the existence of two closely linked genes with considerable variation of rust reactions due to environment in two crosses involving the varieties Fortune/ Ukraine and Garry/Ukraine. Allelism or close linkage for Pc 46 and Pc 50 (Fleischmann, et al., 1971a) and Pc 35 and Pc 54 (Martens et al., 1980) were also reported. Kiehn et al. (1976) and Brouwer and Oates (1986) reported Pc 39 and Pc 55 to be allelic to each other or closely linked. All isolates that were virulent on Pc 39 were also found to be virulent on Pc 55 (Chong, 1988a; Chong and Kolmer, 1993). Chong and Seaman (1993) and Chong and Kolmer (1993) also reported that all isolates virulent on Pc 63 to be virulent on Pc 38. It appeared that these two pairs of genes were associated which may be due to close linkage in coupling phase (Chong and Kolmer, 1993).

Forsberg & Nishiyama (1969) and Sharma and Forsberg (1977) found association between dark kernel colour and crown rust resistance. They suggested the two genes being located on the same chromosome. In other studies the genes for seed colour and awn character did not appear to be linked to crown

rust resistance genes while genes, for grey colour and awn type appear to be closely linked (Kiehn et al., 1976). In addition, lack of linkages was reported between crown rust resistance and characteristics of wild oats (Fleischmann and McKenzie (1964, 1968) and also between crown rust resistance and awn development, lemma pubescence and lemma colour (Wong et al., 1983).

3.0 INHERITANCE OF CROWN RUST RESISTANCE GENES IN THREE <u>Avena</u>
     <u>sterilis</u> L. ACCESSIONS.

3.1 Introduction

Crown rust is one of the major diseases of oats. Using
crown rust resistant varieties has been the practical method
of controlling this disease. Developing commercially
acceptable resistant varieties requires incorporating
resistance genes into cultivated varieties. Several wild oat
collecting expeditions have been made with the major objective
of obtaining new sources of crown rust resistance. Based on
a preliminary screening trial, three <u>Avena</u> <u>sterilis</u> L.
accessions (IB 2435, IB 3071 and IB 3076) were reported (Fox,
1989) to be resistant to six crown rust isolates (CR 25, CR
36, CR 50, CR 56, CR 77 and CR 107). However, the number of
genes in each of the three accessions conferring resistance to
one or more of the six isolates, the genetic relationships
among these genes and also with known Pc genes was not
investigated.

To determine the usefulness of the resistance in these
accessions, it was necessary to characterize the resistance
genes that were available in the three <u>A</u>. <u>sterilis</u> accessions
with the major aim of using them in the oat breeding programme
in the Winnipeg Research Centre. For this purpose, a study
was initiated with the following three objectives:

1. To determine the number and nature of inheritance of resistance genes in IB 2435, IB 3071 and IB 3076.

2. To determine the relationships of the resistance gene(s) in the three accessions with selected Pc genes.

3. To determine the relationships among the resistance gene(s) in the three wild oat accessions.

3.2 Materials

3.2.1 Oats - Parental materials

The three hexaploid (2n=42) crown rust resistant Iberian wild oat accessions used in this study, IB 2435 from Portugal, IB 3071 and IB 3076 from Spain were used as male parents in crosses with the Canadian oat cultivar, Calibre. Calibre also hexaploid, is susceptible to most crown rust isolates of the Prairies. The species, chromosome number, country and location of collection, and the reactions of parental materials to CR 13 and CR 50 is shown in Table 3.2.1.1.

Table 3.2.1.1. Avena species, chromosome number, country & location of collection and rust reactions to CR 13 and CR 50 of three wild oat accessions (IB 2435, IB 3071 and IB 3076) and the cultivated oat cultivar, Calibre.

Species	IB #/ cultiv.	Country	Location	Chr #	CR 13	CR 50
A. sterilis	2435	Portugal	Figueira	42	0	0
A. sterilis	3071	Spain	Jereze de la Fontera	42	;	;
A. sterilis	3076	Spain	Jereze de la Fontera	42	0	0
A. sativa	Calibre	Canada	Saskatoon	42	4	4

Eight single gene Pc lines (Table 3.2.1.2) from the currently used differential set in the Winnipeg Research Centre (Appendix A), were selected to identify the resistance genes in the three A. sterilis accessions. The basis of selection of the Pc lines was their present use in oat cultivars grown in the Prairies (Pc 38 and Pc 39), their potential use (Pc 68) and also because of the similarity of reaction types to crown rust isolates with the three wild oat accessions.

Table 3.2.1.2.   Reactions of selected single gene Pc lines to seven crown rust isolates.

PC gene	CR 13	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
38	; 1	;	4	;	;	; 1	;
39	4	4	;	;	;	;	;
48	4	4	;	0 ;	;	;	;
58	;	;	;	;	0 ;	; 1	;
59	;	;	;	;	; 1	4	;
61	;	;	;	;	0 ;	;	;
64	;	;	; 1	;	;	;	;
68	0	0	0	0	0	0	0

3.2.2.   Crown rust

Crown rust isolates CR 13 and CR 50, obtained from the Winnipeg Research Centre rust laboratory, were used throughout the inheritance study.   These two isolates were selected because:

1) They were used in the initial screening of the wild oat accessions used in this study.

2) CR 13 is a virulent isolate which attacks eight Pc genes (Pc 39, Pc 40, Pc 45, Pc 46, Pc 48, Pc 54, Pc 55 and Pc 60) and is also virulent on one of the genes (Pc 39) that confers resistance to cultivars which are widely cultivated in the prairies such as Dumont, Robert and Riel. It is, therefore, important to understand the inheritance of resistance genes in the three wild oat accessions in relation to CR 13.

3) CR 50 is a less virulent isolate which attacks three Pc genes (Pc 35, Pc 50 and Pc 56) different from the ones attacked by CR 13. The two isolates have a combined virulence on eleven Pc genes.

4) The use of two isolates with different virulent combinations offers the opportunity of identifying more than one gene in each accession.

5) A similar inheritance study was conducted using these two isolates (Fox, 1989). Thus to maintain consistency with similar studies in the oat breeding programme in Winnipeg Research Centre it was decided to use the two isolates.

3.3   Methods

3.3.1.   Oats

3.3.1.1.   Planting

The three wild oat accessions and Calibre were planted at the rate of four seeds/pot in 15-cm diameter fibre pots. The seedlings were thinned to two/pot. A 5:1:1 soil mix of soil, sand and turface, respectively, was used. When seedlings were inoculated with rust and there was no need to grow them to maturity, ten to twelve seeds/pot were planted in a 13-cm diameter fibre pot using soil only.

To obtain uniform germination and uniformly expanded seedling leaves, the following steps were adhered to during planting:

   a)   Seeds were dehulled and put into labelled envelops,
   b)   The envelopes were soaked in GA_3 with acetone (1 g of GA_3 in 4 litres of acetone) for 15-20 minutes,
   c)   Acetone was allowed to evaporate from the envelop,
   d)   Seeds were then treated with Agrox N-M 50% Maneb and
   e)   Seeds were planted in their respective pots.

After planting, Maneb was applied on the soil surface to supplement the soil with the micronutrient manganese in

addition to its fungicidal effect. To give germinating seedlings a good start, the pots were fertilized with 16-20-0 (NPK) at the rate of about five grams per pot.

Plants were grown in growth cabinets with a photoperiod of 16 hours and a temperature regime of $20^0C/15^0C$ (day/night). Insects were controlled using different insecticides including RAID (Johnson & Sons Ltd.), Malathion 50% EC (Later chemicals, Ltd., Richmond, B.C.), Temik (R) (Union Carbide Agricultural Products Co. Inc.), and Cygon (1.5ml/ litre). In addition, pots were watered weekly with a fertilizer solution containing 5g/litre of 20-20-20 (NPK) (Plant Products Co. Ltd., Bramalea, Ontario).

3.3.1.2. Crossing

Obtaining matching male and female parents, that is, a male parent shedding its pollen at the time when the stigma of the female parent is receptive was one of the difficult tasks faced in oat crossing. This is mainly because the two parents head at different times and there is only a little window to pollinate at the right moment. It is even more difficult when it is not possible to predict as to when the wild oat parent will head. This problem was addressed by planting both parents at different planting dates separated by weekly intervals. Both parents were kept in the same cabinet until

heading time when the male and female parents were kept in separate growth cabinets in which day/night cycle of the male cabinet was six hours ahead of the female cabinet.

For successful crossing, Coffman's (1937) technique was used on healthy and sturdy oat plants. Pollen was obtained by opening the glume and the pilea of the male parent and removing the bright creamy anthers with a pair of tweezers. The anthers were placed on the palm and carefully the floret of the female parent opened. Only the primary florets of the female parent were used in hybridization to ensure viable seed production. Anthers, the secondary and the tertiary florets of the female parent were discarded. The pollen from the male parent was shed on the feathery stigma and the pilea and the glume were carefully pushed back to their original positions. Fertilization was checked by examining the stigma two days after pollination. If the stigma remained feathery, a second round of pollination was made. Pollinated florets were covered with a wet glassine bag which helps to improve seed set (Marshall, 1962) and also to protect the pollinated florets from pollen of other plants in a cabinet. In addition, to avoid unwanted crossing by carrying pollen from a different male parent when more than one parent was involved in crossing, the tip of the pair of tweezers were licked and then dipped into a jar of 70% ethanol. Also, the palm and the fingers were cleaned with alcohol.

3.3.2. Crown rust screening

3.3.2.1. Inoculation

For inoculation to be successful, viable crown rust spores are required. Fresh spores collected from susceptible hosts or spores dried on a desiccator for two hours, stored at -75^0C +/- 5^0C and heat shocked in a 40^0C water bath for five minutes were used.

In this study, three methods of inoculation were used. These differed in the spore carrier - oil, talc or finger. In oil inoculation, a very small amount of urediosopres of each crown rust was put in a small disposable capsule into which mineral oil was added. The leaves were inoculated with the rust and the oil was allowed to evaporate (20-30 minutes) before the plants were incubated. With the talc method, a spore - talc mixture at the ratio of about 1:50, respectively, was used. A mist of 0.01% solution of Tween 20 (five drops of polyoxyethylene 20 Sorbitan monlaurate per litre of water) was sprayed on the leaves. The seedlings were then inoculated using a compressed air duster. With the finger inoculation method, a small amount of crown rust isolate was put between the thumb and the index finger and the leaves were gently rubbed from the base towards the tip. In talc and finger inoculation methods the inoculated plants were put in an incubating chamber as soon as inoculation was completed.

Inoculated plants were incubated at high relative humidity for 20-24 hours. Most incubations were made in growth cabinets after which the plants were kept in the greenhouse or growth cabinets for normal growth to resume. Rust reactions were scored 12-14 days post inoculation. A "0" to "4" scoring scale described by Murphy (1935b) (Table 2.2.7.1) was used where ";", "1" and "2" ratings represented varying degrees of resistance and "3" and "4" represent susceptible ratings.

3.3.4. Populations developed for genetic analysis

Four different populations were developed for genetic analysis. The purposes were to determine the number and nature of inheritance of resistance genes, to determine the relationships of these genes between themselves and with selected Pc genes. Similar methods of rust testing and chi-square analysis were used in all the populations.

1. a) The three wild oat accessions were crossed with Calibre. The parents and F_1 seedlings from each of the crosses were tested with CR 13 and CR 50 on their first and second leaves, respectively. Resistant reactions of the three accessions (Table 3.3.4.1.) reported in a preliminary screening (Fox, 1989) were confirmed. F_1 seedlings were selfed and were grown to maturity. F_2 seeds were collected

from individual F_1 plants and then six F_2 seeds/pot were planted. The seedlings were tested for their reactions to CR 13 and CR 50. The number of resistant and susceptible seedlings for each of the isolates and crosses was scored. Chi-square test was used to test the goodness-of-fit of the ratios of the segregating populations to the proposed models.

Table 3.3.4.1. Three Iberian wild oat accessions (IB 2435, IB 3071 and IB 3076) and their reactions to six crown rust isolates (CR 25, CR 36, CR 50, CR 56, CR 77, and CR 107).

IB Number	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
IB 2435	0	0	;	0	0	0, ;
IB 3071	;	;	0	;	0	0, 4
IB 3076	0	0	0	0	0	0

b) Randomly selected F_2 plants were grown to maturity. To avoid bias, all even numbered seedlings were removed by numbering the seedlings from 1 - 6, the seedling nearest to the pot label being plant number one. F_3 seeds of individual F_2 plants were collected separately. A minimum of 20 F_3 seedlings/F_2 plant was grown and tested for reactions to CR 13 and CR 50 on the first and second leaves, respectively. Chi-square test was used to test whether the segregating F_2 populations fit into proposed models.

2 a) The F_1 plants of the three wild oat accessions crossed with Calibre were backcrossed to Calibre and grown to maturity. Seeds from individual BC_1F_1 plants were collected separately and then six BC_1F_1 seeds/pot were planted. The reactions of the seedlings to the two test isolates were scored. The seedlings were randomly thinned to three seedlings/pot and grown to maturity. Chi-square test was done on the segregating BC_1F_1 population.

b) Seeds from randomly selected individual BC_1F_1 plants were collected and 20-25 BC_1F_2 seedlings, planted as 10-13 seeds/pot, were grown and tested with CR 13 and CR 50 on their first and second leaves, respectively. Chi-square test was used to determine the goodness-of-fit of the proposed model. The result will help to support the model proposed in the F_2, F_3, and BC_1F_1 segregating populations.

3) Each of the three IB accessions were crossed with selected single gene Pc lines. The purpose of these crosses was to produce F_2 populations for testing with CR 13 and CR 50 to determine whether or not the resistance genes in the wild oat accessions were already known genes. For these crosses eight Pc lines (Pc 38, Pc 39, Pc 48, Pc 58, Pc 59, Pc 61, Pc 64 and Pc 68) were selected based on their resistant reactions (Table 3.2.1.2) and also based on their present and potential use as crown rust resistant genes.

4) To determine whether the resistance genes in each of the three IB accessions were the same or different, intercrosses

between the three unknown wild oat accessions were made. Chi-square test was used to test whether the segregating F_2 population of each of the crosses would fit to a proposed model.

In some cases rating rust reactions was difficult due to seedling deaths. This was presumably caused due to soil toxicity. However, some F_2 and BC_1F_1 plants survived and were carried to the next generation even without scoring their rust reactions. As a result, the BC_1F_2 population sizes were more than 50% of the BC_1F_1 population.

3.3. Results and discussion

3.4.1. IB 2435 study

3.4.1.1 Inheritance of crown rust resistance in Calibre/IB 2435

Segregation of the Calibre/IB 2435 F_2 and F_3 populations and of Calibre*2/IB 2435 BC_1F_1 and BC_1F_2 populations when tested with CR 13 and CR 50 are shown in Table 3.4.1.1.1.

The F_2 population of the Calibre/IB 2435 cross segregated into 245 resistant and 72 susceptible seedlings when tested with CR 13. Chi-square test supported that the segregation ratio fitted (P=0.30-0.50) the proposed 3:1 (R:S) model for a

single dominant gene. When tested with CR 50, the same population segregated for 250R and 67S seedlings. This segregation ratio also fitted (P=0.10-0.20) the proposed model for a single dominant gene, 3R:1S.

Table 3.4.1.1.1. Segregation of the F_2, F_3, BC_1F_1 and BC_1F_2 populations from the Calibre/IB 2435 cross to crown rust isolates CR 13 and CR 50.

Populations	CR isolates	R : S	Proposed model	X^2	P of X^2
F_2	CR 13 CR 50	245 : 72 250 : 67	3 : 1 3 : 1	0.884 2.525	0.30 - 0.50 0.10 - 0.20
BC_1F_1	CR 13 CR 50	41 : 28 40 : 29	1 : 1 1 : 1	2.449 1.754	0.10 - 0.20 0.10 - 0.20
BC_1F_2*	CR 13 CR 50	38 : 41 38 : 41	1 : 1 1 : 1	0.114 0.114	0.70 - 0.90 0.70 - 0.90
F_3**	CR 13 CR 50	44:116:42 44:116:42	1: 2: 1 1: 2: 1	4.495 4.495	0.10 - 0.20 0.10 - 0.20

R = Resistant , S = Susceptible ; * Resistant : Segregating families ;
** Resistant : Segregating : Susceptible families

In the resistant class of reactions (described in Table 2.2.7.1) the majority of the segregating F_2 seedlings (except for 7 plants for CR 13 and 14 plants for CR 50 out of 317 plants) showed '0' or ';' reaction types for both CR 13 and CR 50. The ratio of the seedlings in the different reaction types did not fit the 1:2:1 model for a partial dominant gene. This supported the hypothesis that the gene conferring resistance to CR 13 and also the gene conferring resistance to CR 50 was a dominant gene. Plants resistant/susceptible to CR 13 when inoculated on their first leaves were also resistant/ susceptible, respectively, to CR 50 when inoculated on their

second leaves. This suggests that the same gene was conferring resistance to both isolates. For the purpose of this study the gene conferring resistance to CR 13 and CR 50 in IB 2435 was designated as gene 'A'.

The F_3 families from the population of randomly selected F_2 plants resulted in 44 resistant , 116 segregating and 42 susceptible families when tested with CR 13 and CR 50 on their first and second leaves, respectively. This segregation ratio fitted (P=0.10-0.20) the proposed one dominant gene model, one resistant : two segregating : one susceptible families. Within the segregating F_3 families, seedlings segregated with a 3R:1S ratio (Appendix B). All the 202 families segregated for CR 13 and CR 50 in the same way. That is, homozygous resistant, heterozygous and homozygous susceptible families to CR 13 were also homozygous resistant, heterozygous and homozygous susceptible, respectively, to CR 50. The results from the F_3 families, that is, segregation of the F_3 families with a one resistant : two segregating : one susceptible and also segregation within the segregating families with a 3R:1S seedling ratio for a single dominant gene, was in agreement with the results obtained in the F_2 segregating population, suggesting that IB 2435 possesses a single dominant gene conferring resistance to both CR 13 and CR 50.

In the backcross populations, the BC_1F_1 population segregated for 41R and 28S seedlings when tested with CR 13 on their first leaves. The same population segregated into 40R

and 29S seedlings when their second leaves were tested with CR 50. In both populations, the resistant : susceptible ratio fitted (P=0.10-0.20) the proposed 1:1 model. The population from randomly selected BC_1F_1 plants that were carried to the next generation, BC_1F_2, resulted in 38 segregating and 41 susceptible families when tested with both CR 13 and CR 50 on their first and second leaves, respectively. This segregation ratio in BC_1F_2 also fitted (P=0.70-0.90) the proposed 1:1 model for segregating : susceptible families. The seedlings within the segregating BC_1F_2 families segregated with a 3R:1S seedling ratio for a single dominant gene (Appendix C). These results from the backcross method were strong evidences supporting the results obtained from the F_2 populations and F_3 families suggesting IB 2435 possessed a single dominant gene.

3.4.1.2. Relationships of the resistance genes in IB 2435 with selected Pc genes.

Segregation to CR 13 and CR 50 of F_2 seedlings from crosses between selected Pc lines with single genes for crown rust resistance and IB 2435 are shown in Table 3.4.1.2.1.

In the F_2 populations from crosses of Pc 61 and Pc 68 with IB 2435, the resistant to susceptible segregation ratio fitted the proposed model for two dominant genes, 15R:1S, when tested with CR 13. This was to be expected because the three Pc lines, each possessing a single gene, were resistant to CR 13.

Table 3.4.1.2.1. Segregation of the F_2 populations from selected Pc lines/IB 2435 crosses to crown rust isolates CR 13 and CR 50.

Crosses	CR isolates	R : S	Proposed Model	X^2	P of X^2
Pc 38/IB 2435	CR 13	169 : 31	13 : 3	1.387	0.20 - 0.30
	CR 50	185 : 15	15 : 1	0.533	0.30 - 0.50
Pc 39/IB 2435	CR 13	148 : 52	3 : 1	0.107	0.70 - 0.90
	CR 50	187 : 13	15 : 1	0.021	0.70 - 0.90
Pc 48/IB 2435	CR 13	161 : 41	3 : 1	2.383	0.10 - 0.20
	CR 50	193 : 9	15 : 1	1.110	0.20 - 0.30
Pc 58/IB 2435	CR 13	169 : 31	13 : 3	1.387	0.20 - 0.30
	CR 50	192 : 8	15 : 1	1.728	0.10 - 0.20
Pc 59/IB 2435	CR 13	123 : 31	13 : 3	0.192	0.50 - 0.70
	CR 50	140 : 14	15 : 1	2.122	0.10 - 0.20
Pc 61/IB 2435	CR 13	178 : 11	15 : 1	0.059	0.70 - 0.90
	CR 50	188 : 1	63 : 1	1.313	0.20 - 0.30
Pc 64/IB 2435	CR 13	165 : 33	13 : 3	0.564	0.30 - 0.50
	CR 50	163 : 35	13 : 3	0.151	0.50 - 0.70
Pc 68/IB 2435	CR 13	181 : 14	15 : 1	0.288	0.50 - 0.70
	CR 50	181 : 14	15 : 1	0.288	0.50 - 0.70

R = Resistant, S = Susceptible

For each of the two crosses, gene A from IB 2435 together with the single gene from the Pc lines segregated following a simple Mendelian segregation ratio for two independent dominant genes, 15R:1S. Populations from Pc 39/Ib 2435 and Pc 48/IB 2435 crosses resulted in a segregation ratio for one dominant gene. Since CR 13 is virulent to Pc 39 and Pc 48, F_2 populations derived from crosses involving Pc 39 and Pc 48 would show only a single gene segregation, that is, the gene derived from IB 2435. Populations of crosses from Pc 38, Pc 58, Pc 59 and Pc 64 with IB 2435 also fitted the two gene model acting in a dominant-recessive epistatic manner when tested with CR 13.

When tested with CR 50, the populations from all the crosses, except for Pc 61/IB 2435 and Pc 64/IB 2435 crosses, segregated with a resistant to susceptible ratio that fitted the two dominant gene model, 15R:1S. The segregating population from Pc 64/IB 2435 cross fitted a two gene model acting in a dominant-recessive epistatic manner, 13R:3S, while the segregating population from Pc 61/IB 2435 cross fitted a three gene model, 63R:1S.

In all the populations involving Pc lines and IB 2435 crosses, segregation for two genes was expected when tested with CR 50. This is because all the Pc lines were resistant to CR 50. Therefore, in addition to the single genes in each of the Pc lines, gene A from IB 2435 was also expected to confer resistance to CR 50 in the segregating F_2 populations. The population from Pc 61/IB 2435 cross, however, resulted in the unexpected resistant to susceptible segregation ratio that fitted a three gene model. One possibility as to why the unexpected segregation ratio resulted could be due to the expression of a resistance gene(s), in one or both parents, which was not able to express its resistance in the absence of the second gene from the second parent. This phenomenon was observed by Nelson (1981) where a number of defeated genes (susceptible) for stem rust resistance resulted in improved resistance when combined together. Even though Nelson's observation does not fully explain the results obtained in

this cross, it suggests the possibility of the presence of a defeated/minor/secondary gene(s) in either of the parents that might have expressed resistance when combined with the other.

Another possible explanation for the three gene ratio involving Pc 61 and CR 50 is that the Pc 61 line may not be a single gene line. This is because results from all the populations, except for the population from Pc 61/IB 2435 cross, when tested with CR 50 suggest that IB 2435 to possess a single dominant gene. Therefore, it follows from this that it might be the Pc 61 line that possesses the second gene. The other possibility is that scoring of rust reactions might have been inaccurate. Resistant reaction depends on the host-pathogen interaction which means not all hosts express similar reaction types when inoculated with the same isolate. Therefore, if some seedlings were scored as resistant when they were really susceptible, then the segregating population could fit the model for a three gene segregation ratio. Still an other possibility, though very unlikely, is that some seedlings could have been escapes during inoculation thereby affecting the result. Whether they were escapes or not and whether the results obtained were indeed correct could be verified by growing the F_2 population to F_3 and testing the segregating families with CR 50.

In all the segregation ratios, with few exceptions, all seedlings and families resistant to CR 13 were also found to be resistant to CR 50. Also, those that were susceptible to

CR 13 were susceptible to CR 50. This is indicative that the same gene, gene 'A', was conferring resistance to both CR 13 and CR 50 further supporting the hypothesis made based on results of previous populations.

3.4.2 IB 3071 study

3.4.2.1. Inheritance of crown rust resistance in Calibre/IB 3071

Segregation of the Calibre/IB 3071 F_2 and F_3 seedlings and also segregation of Calibre*2/IB 3071 BC_1F_1 and BC_1F_2 seedlings tested with CR 13 and CR 50 are shown in Table 3.4.2.1.1.

Table 3.4.2.1.1. Segregation of the F_2, F_3, BC_1F_1 and BC_1F_2 populations from the Calibre/IB 3071 cross to crown rust isolates CR 13 and CR 50.

Populations	CR isolates	R : S	Proposed Model	X^2	P of X^2
F_2	CR 13	100 : 37	3 : 1	0.295	0.50 - 0.70
	CR 50	103 : 34	3 : 1	0.002	0.95 - 1.00
BC_1F_1	CR 13	43 : 31	1 : 1	1.946	0.10 - 0.20
	CR 50	36 : 38	1 : 1	0.054	0.70 - 0.90
BC_1F_2*	CR 13	61 : 91	1 : 1	5.570	0.01 - 0.05
	CR 50	61 : 91	1 : 1	5.570	0.01 - 0.05
F_3**	CR 13	52:86:57	1: 2: 1	2.969	0.20 - 0.30
	CR 50	52:86:57	1: 2: 1	2.969	0.20 - 0.30

R = Resistant, S = Susceptible; * Segregating : Susceptible families;
** Resistant: Segregating:Susceptible families

The F_2 populations from the Calibre/IB 3071 cross when tested with CR 13 segregated for 100R and 37S seedlings. The chi-square test supported that this ratio fitted (P=0.50-0.70)

the single dominant gene model, 3R:1S. The same population when tested with CR 50 resulted in 103R and 34S seedlings. This ratio fitted (P=0.95-1.00) the single dominant gene model, 3R:1S. In addition, most of the resistant reaction types for the 137 F_2 seedlings were '0' or ';' except for 10 plants for CR 13 and 34 plants for CR 50. The ratio of the populations in the different reaction types did not fit the 1:2:1 model for a partial dominant gene indicating that the gene in IB 3071 was a dominant gene. Here again, like in the populations derived from IB 2435 crosses, most of the seedlings resistant/susceptible to CR 13 on the first leaves were resistant/susceptible, respectively, to CR 50 on the second. This suggests that the same resistance gene confers resistance to both CR 13 and CR 50. For the purpose of this study, the gene conferring resistance to both CR 13 and CR 50 in IB 3071 was designated as gene 'B'.

The F_3 families from randomly selected F_2 plants resulted in 52 resistant, 86 segregating and 57 susceptible families when tested with CR 13 and CR 50 on their first and second leaves, respectively. The chi-square test supported that the ratio fitted (P=0.20-0.30) the proposed model for a single dominant gene, one resistant : two segregating : one susceptible families. Within the segregating families, seedlings segregated with a 3R:1S ratio (Appendix D). In addition to the result from F_2 plants where 191 out of 195

showed reaction types that were the same for CR 13 and CR 50, the result in F_3 families, that is, segregation of the F_3 families with a one resistant : two segregating : one susceptible ratio and also segregation within the segregating families with a 3R:1S ratio supported the hypothesis that IB 3071 carries a dominant gene conferring resistance to both CR 13 and CR 50. Further, the fact that 185 families out of 195 showed the same reaction for the two isolates is a supporting evidence that a single dominant gene conferred resistance to both CR 13 and CR 50.

In the backcross populations, the BC_1F_1 population segregated for 43R:31S when the first leaves were inoculated with CR 13. The same population when tested with CR 50 on the second leaves resulted in 36R and 38S seedlings. In both cases, chi-square test supported the hypothesis that the resistant : susceptible ratio fitted ($P=0.10-0.20$ for CR 13 and $P=0.70-0.90$ for CR 50) the proposed single gene model. Randomly selected BC_1F_1 plants that were advanced to BC_1F_2 were also tested with CR 13 and CR 50 on their first and second leaves, respectively. The result was 61 families segregating and 91 families susceptible to both isolates. Chi-square test supported proposal that the ratio of the segregating to susceptible seedlings weakly fitted (0.01-0.05) 1:1 model. The seedlings within the segregating BC_1F_2 families segregated with a 3R : 1S ratio for a single dominant gene (Appendix E).

The results from the BC_1F_1 and BC_1F_2 were additional evidences supporting the results obtained in F_2 populations and F_3 families suggesting the presence of a single dominant gene in IB 3071 conferring resistance to both isolates.

3.4.2.2. Relationships of the resistance gene in IB 3071 with selected single gene Pc lines.

Segregation of F_2 seedlings from crosses between selected Pc lines with single genes for crown rust resistance and IB 3071 when tested with CR 13 and CR 50 are shown in Table 4.4.2.2.1. The segregating F_2 populations in crosses between Pc lines 38, Pc 58, Pc 59, Pc 61 and Pc 64 with IB 3071 when tested with CR 13, resulted in a 15R:1S ratio that fitted the proposed model for two dominant genes.

Since the Pc 38, Pc 58, Pc 59 Pc 61 and Pc 64 lines were all resistant to CR 13 and also since each one of them carries a single gene, a two gene segregation ratio was to be expected in the segregating F_2 population. The segregating populations from crosses between Pc 39 and Pc 48 with IB 3071 also fitted the proposed single dominant gene model, 3R:1S, when tested with CR 13. Since both Pc 39 and Pc 48 were susceptible to CR 13, test with this isolate could only detect one gene segregation. All of the crosses involving single gene Pc lines with IB 3071 and tested with CR 13 support the

hypothesis and agree with the evidences from F_2, F_3, BC_1F_1 and BC_1F_2 results that IB 3071 had a single crown rust resistance gene.

Table 3.4.2.2.1. Segregation of the F_2 populations from selected Pc lines/IB 3071 crosses to crown rust isolates CR 13 and CR 50.

Crosses	CR isolates	R : S	Proposed Model	X^2	P of X^2
Pc 38/IB 3071	CR 13	185 : 14	15 : 1	0.209	0.50 - 0.70
	CR 50	190 : 9	15 : 1	1.013	0.30 - 0.50
Pc 39/IB 3071	CR 13	154 : 45	3 : 1	0.605	0.30 - 0.50
	CR 50	188 : 11	15 : 1	0.177	0.50 - 0.70
Pc 48/IB 3071	CR 13	156 : 39	3 : 1	2.600	0.10 - 0.20
	CR 50	189 : 6	15 : 1	3.350	0.05 - 0.10
Pc 58/IB 3071	CR 13	190 : 14	15 : 1	0.131	0.70 - 0.90
	CR 50	204 : 0	-	-	-
Pc 59/IB 3071	CR 13	179 : 13	15 : 1	0.089	0.70 - 0.90
	CR 50	177 : 15	15 : 1	0.800	0.30 - 0.50
Pc 61/IB 3071	CR 13	174 : 11	15 : 1	0.029	0.70 - 0.90
	CR 50	182 : 3	63 : 1	0.007	0.95 - 0.99
Pc 64/IB 3071	CR 13	179 : 18	15 : 1	2.802	0.05 - 0.10
	CR 50	189 : 8	15 : 1	1.611	0.20 - 0.30
Pc 68/IB 3071	CR 13	201 : 0	-	-	-
	CR 50	201 : 0	-	-	-

R = Resistant, S = Susceptible

When tested with CR 50, the populations from crosses between Pc 38, Pc 39, Pc 48, Pc 59 and Pc 64 with IB 3071 resulted in a resistant to susceptible segregation ratio fitting the proposed model for two dominant genes, 15R:1S. This was expected because the above five Pc lines were all resistant to CR 50. Therefore, the F_2 populations must have obtained their protection from each of the parents that contributed one gene each.

The populations from the Pc 61/IB 3071 cross resulted in a three gene segregation ratio, 63R:1S. This result was consistent with the findings in the Pc 61/IB 2435 cross. A two gene segregation ratio was expected because each parent was expected to provide a single gene conferring resistance to CR 50. The same reasoning explained for the cross between Pc 61/IB 2435 (expression of defeated gene(s), presence of two genes in Pc 61, incorrect scoring of rust reactions or escapes) might be the reason for the result obtained in this cross.

The population from the Pc 68/IB 3071 cross when tested with both CR 13 and CR 50 did not segregate at all. This suggests the possibility that gene B from IB 3071 to be the same or allelic or tightly linked to Pc 68.

The population from the Pc 58/IB 3071 cross when tested with CR 50 did not segregate. This suggests the possibility that gene B to be the same or allelic or tightly linked to Pc 58. Since Pc 58 and Pc 68 are two different and independent genes, gene B can not be the same as the two genes at the same time. It is possible to suggest that gene B is located between Pc 58 and Pc 68 on the same chromosome thereby linked to Pc 58 on one side and to Pc 68 on the other. However, this is unlikely because the same population, when tested with CR 13, segregated for two independent genes. Therefore, because

this result can not be explained properly it would be useful to grow out F_3 families with a larger population size and test them with CR 50.

3.4.3. IB 3076 study

3.4.3.1. Inheritance of crown rust resistance in Calibre/IB 3076

Segregation of the Calibre/IB 3076 F_2 and F_3 seedlings and also segregation of Calibre*2/IB 3076 BC_1F_1 and BC_1F_2 seedlings tested with CR 13 and CR 50 are shown in Table 3.4.3.1.1.

Table 3.4.3.1.1. Segregation of the F_2, F_3, BC_1F_1 and BC_1F_2 populations from the Calibre/IB 3076 cross to crown rust isolates CR 13 and CR 50.

Populations	CR isolates	R : S	Proposed Model	X^2	P of X^2
F_2	CR 13	229 : 66	3 : 1	1.085	0.20 - 0.30
	CR 50	228 : 67	3 : 1	0.824	0.30 - 0.50
BC_1F_1	CR 13	90 : 68	1 : 1	3.064	0.05 - 0.10
	CR 50	91 : 67	1 : 1	3.646	0.05 - 0.10
$BC_1F_2^*$	CR 13	94 : 68	1 : 1	4.173	0.01 - 0.05
	CR 50	94 : 68	1 : 1	4.173	0.01 - 0.05
F_3^{**}	CR 13	53:85:44	1: 2: 1	1.681	0.30 - 0.50
	CR 50	53:85:44	1: 2: 1	1.681	0.30 - 0.50

R = Resistant, S = Susceptible; * Segregating : Susceptible families;
** Resistant : Segregating : Susceptible families

The F_2 populations of the Calibre/IB 3076 cross resulted in 229R and 66S seedlings when tested with CR 13. The same population tested with CR 50 resulted in 228R and 67S seedlings. These segregation ratios for each of the two crown

rust isolates fitted (P=0.20-0.30 for CR 13 and P=0.30-0.50 for CR 50) the proposed model for a single dominant gene, 3:1. Only seven plants for CR 13 and 14 plants for CR 50, out of a population of 295, showed a type '1' or '2' reaction. The ratio of the seedlings in these reaction types did not fit the 1:2:1 model for a partial dominant gene. This indicated that the resistance gene was a dominant gene. Once again as in IB 2435 and IB 3071, the plants resistant/susceptible to CR 13 on the first leaf were also resistant/susceptible, respectively, to CR 50 on the second. There were only three out of 295 F_2 plants where reactions for CR 13 and CR 50 were not the same. This is indicative that the same gene provides protection to the two isolates. For the purpose of this study the gene conferring resistance to CR 13 and CR 50 in IB 3076 was designated as gene 'C'.

The F_3 families from randomly selected F_2 plants resulted in 53 resistant, 85 segregating and 44 susceptible families when tested with CR 13 and CR 50 on their first and second leaves, respectively. This segregation ratio also fitted (P=0.30-0.50) the single gene model for one homozygous resistant : two segregating : one homozygous susceptible families. Within the segregating families seedlings segregated with a 3R:1S ratio (Appendix F). Only in six of the 183 F_3 families were the reactions for CR 13 and CR 50 different. This is a further evidence supporting the hypothesis that a single gene conferred resistance to both CR

13 and CR 50. The results from the F_3 families, that is, segregation of the F_3 families with a one resistant : two segregating : one susceptible ratio and also segregation within the segregating families with a 3R : 1S ratio expected for a single dominant gene, was in agreement with the results obtained in the F_2 segregating population that suggests IB 3076 possesses a single dominant gene conferring resistance to both CR 13 and CR 50.

In the backcross populations, the seedlings from BC_1F_1 segregated for 90R and 68S seedlings when tested with CR 13 on their first leaves. This same population when tested with CR 50 on their second leaves segregated with 91R:67S seedlings. These segregation ratios fitted (P=0.05-0.10) the proposed 1R:1S ratio which is a model for a single dominant gene for backcrossed seedlings. Populations from randomly selected BC_1F_1 plants that were grown to BC_1F_2 resulted in 94 segregating families and 68 susceptible families when tested with CR 13 and CR 50 on their first and second leaves, respectively. These segregation ratios weakly fitted (P=0.01-0.05) the proposed single gene model of one segregating to one susceptible. The seedlings from the segregating BC_1F_2 families segregated with 3R : 1S ratio for a single dominant gene (Appendix G). The results from BC_1F_1 and BC_1F_2 supported the results obtained from F_2 population and F_3 families suggesting IB 3076 possessed a single dominant gene against both CR 13 and CR 50.

3.4.3.2 Relationships of resistance genes in IB 3076 with selected single gene Pc lines

Segregation of F_2 Seedlings from crosses between selected Pc lines with single genes and IB 3076 when tested with CR 13 and CR 50 are shown in Table 3.4.3.2.1.

Table 3.4.3.2.1. Segregation of the F_2 populations from selected Pc lines/IB 3076 crosses to crown rust isolates CR 13 and CR 50.

Crosses	CR isolates	R : S	Proposed Model	X^2	P of X^2
Pc 38/IB 3076	CR 13 CR 50	175 : 8 176 : 7	15 : 1 15 : 1	1.102 1.837	0.20 - 0.30 0.10 - 0.20
Pc 39/IB 3076	CR 13 CR 50	155 : 45 190 : 10	3 : 1 15 : 1	0.667 0.533	0.30 - 0.50 0.30 - 0.50
Pc 48/IB 3076	CR 13 CR 50	156 :41 188 : 9	3 : 1 15 : 1	1.843 0.950	0.10 - 0.20 0.30 - 0.50
Pc 58/IB 3076	CR 13 CR 50	176 : 11 179 : 8	15 : 1 15 : 1	0.043 1.241	0.70 - 0.90 0.20 - 0.30
Pc 59/IB 3076	CR 13 CR 50	190 : 15 196 : 9	15 : 1 15 : 1	0.398 1.210	0.50 - 0.70 0.20 - 0.30
Pc 61/IB 3076	CR 13 CR 50	196 : 9 201 : 4	15 : 1 63 : 1	1.210 0.201	0.20 - 0.30 0.50 - 0.70
Pc 64/IB 3076	CR 13 CR 50	164 : 43 163 : 44	13 : 3 13 : 3	0.556 0.853	0.30 - 0.50 0.30 - 0.50
Pc 68/IB 3076	CR 13 CR 50	171 : 0 171 : 0	- -	- -	- -

R = Resistant, S = Susceptible

The F_2 seedlings from crosses between Pc 38, Pc 58, Pc 59 and Pc 61 with IB 3076 when tested with CR 13 segregated with a 15R : 1S ratio fitting the proposed two dominant genes model. Also the population from the Pc 64/IB 3076 cross

fitted a two gene model acting in a dominant-recessive epistatic manner when tested with CR 13. Since the parents (the single Pc genes and IB 3076) each had single dominant genes for protection against CR 13, the F_2 populations segregated as expected with a resistant to susceptible ratio that fitted a two gene model. The populations from crosses of Pc 39 and Pc 48 with IB 3076 resulted in a model that fitted a one gene segregation ratio, 3R:1S. This was also expected because both Pc 39 and Pc 48 were susceptible to CR 13 and the F_2 populations would only obtain their protection against CR 13 from gene C in IB 3076.

The populations involving Pc 38, Pc 39, Pc 48, Pc 58 and Pc 59 crossed with IB 3076 all segregated in the ratio that fitted the proposed two dominant genes model when tested with CR 50. This was expected since the five Pc lines were all resistant to CR 50 and the populations from the Pc lines/IB 3076 crosses would obtain one gene for resistance from each of the parents resulting in a two gene segregation.

The population from the Pc 61/IB 3076 cross segregated in a ratio that fitted a three gene model when tested with CR 50. This was not expected simply because each of the parents were expected to provide one gene each effective against CR 50. Once again the possible explanations for this unexpected segregation ratio could be the ones given for Pc 61/IB 2435

cross (defeated gene(s), the presence of two genes in Pc 61, incorrect scoring of rust reactions or escapes). Growing the F_2 plants to maturity and testing F_3 families with a larger population would verify whether Pc 61 indeed possess one or two genes.

A two gene segregation ratio acting in a dominant-recessive epistatic manner resulted when the population from Pc 64/IB 3076 cross when tested with CR 50. The result showed that each of the two parents contributed, as expected, a single gene of resistance.

The population from Pc 68/IB 3076 cross did not segregate when tested with either one of the two test isolates. Here again, like the Pc 68/IB 3071 cross, this result suggests that the resistance gene in IB 3076 to be the same or allelic or tightly linked to Pc 68.

3.4.4. Relationships among the resistance genes in the three wild oat accessions

Segregation of the F_2 Seedlings from intercrosses between the three A. sterilis accessions (IB 2435, IB 3071 and IB 3076) when tested with CR 13 and CR 50 are shown in Table 3.4.4.1.

Table 3.4.4.1. Segregation of F_2 populations from the crosses among the three wild oat accessions (IB 2435, IB 3071 and IB 3076) to crown rust isolates CR 13 and CR 50.

Crosses	CR isolates	R : S	Proposed Model	X^2	P of X^2
IB 2435/IB 3071	CR 13 CR 50	335 : 30 345 : 20	15 : 1 15 : 1	2.416 0.370	0.10 - 0.20 0.50 - 0.70
IB 2435/IB 3076	CR 13 CR 50	535 : 26 536 : 25	15 : 1 15 : 1	2.498 3.081	0.10 - 0.20 0.05 - 0.10
IB 3071/IB 3076	CR 13 CR 50	436 : 0 436 : 0	- -	- -	- -

R = Resistant, S = Susceptible

The population from the IB 2435/IB 3071 cross resulted in 335R and 30S seedlings when tested with CR 13. The same population when tested with CR 50 resulted in 345R and 20S seedlings. The Chi-square test for both populations supported the hypothesis that the segregating populations fitted (P=0.10-0.20 for CR 13 and P=0.50-0.70 for CR 50) the ratio for two dominant genes, 15R:1S. This result was to be expected because F_2, F_3, BC_1F_1 and BC_1F_2 results in the crosses involving IB 2435 and IB 3071 showed that each of the two wild oat accessions possessed a single dominant gene conferring resistance to CR 13 and CR 50. The result, 15R:1S ratio, clearly showed that each of the parents (IB 2435 and IB 3071) contributed a single dominant gene each conferring resistance to CR 13 and CR 50 further supporting the results in F_2, F_3, BC_1F_1 and BC_1F_2. Therefore, gene 'A' in IB 2435 and gene 'B' in IB 3071 must be two different and independent genes. Had the genes from the two wild oat accessions been the same, allelic

to or tightly linked to each other, there would have been no segregation in the population of the IB 2435/IB 3071 cross.

The population from the IB 2435/IB 3076 cross resulted in 535R and 26S seedlings when tested with CR 13. The same population when tested with CR 50 resulted in 536R and 25S seedlings. Here again, Chi-square supported the proposal that the segregating population fitted (P=0.10-0.20 for CR 13 and P=0.05-0.10 for CR 50) the model for two dominant genes, 15R:1S. The result supported the one dominant gene hypothesis based on the results from the segregating populations of F_2, F_3, BC_1F_1 and BC_1F_2. It was also a strong evidence that gene 'A' from IB 2435 and gene 'C' from IB 3076 were two different and independent genes.

The F_2 population from IB 3071/IB 3076 cross did not segregate at all with all 436 seedlings being resistant to both CR 13 and CR 50. This result suggested that the gene in IB 3071 (gene B) conferring resistance to CR 13 and CR 50 was the same as, allelic to or tightly linked to the gene in IB 3076 (gene C) conferring resistance to the two isolates. Had the genes in the two accessions conferring resistance to the two isolates been different or independent, a two gene segregation ratio would have resulted. Since the population size tested was large enough to detect the segregation of two dominant genes (Hunson, 1959), the result obtained in this

cross would lead to the conclusion that the two genes were neither different nor independent. Therefore, gene B from IB 3071 which condition resistance to CR 13 and CR 50 may be the same as, allelic to or tightly linked to gene C from IB 3076 which was also resistant to the two isolates.

3.6 Conclusions

The susceptible cultivar Calibre was crossed with each of the three accessions. The resulting F_1 generation was either permitted to self and produce F_2 and F_3 or backcrossed to Calibre to produce BC_1F_1 and permitted to self to produce BC_1F_2. The results from all the four genetic populations showed that each of the three accessions possessed a single dominant gene conferring resistance to both CR 13 and CR 50.

The cross between eight selected single gene Pc lines with each of the three accessions showed, with few exceptions, that they segregated for two genes. The populations from the cross between Pc 61 and each of the three accessions segregated for the unexpected three gene segregation ratio when tested with CR 50. Based on this study and the literature this unexpected result can not readily be explained. It appears that the Pc 61 line possessed a second gene of resistance against CR 50. The population from the Pc 58/IB 3071 cross also did not segregate when tested with CR

50. This also was an unexpected result that can not readily be explained. The single gene Pc lines/IB 2435 cross showed that the resistance gene in IB 2435 was different and independent from the eight Pc genes (Pc 38, Pc 39, Pc 48, Pc 58, Pc 59, Pc 61, Pc 64 and Pc 68). The Pc lines/IB 3071 and Pc lines/IB 3076 crosses suggested that the genes in IB 3071 and IB 3076 to be the same or allelic or tightly linked to Pc 68.

The results from the F_2 populations of the intercrosses among the three Iberian accessions, when tested with CR 13 and CR 50, showed that the resistance gene in IB 2435, gene 'A', to be different and independent from gene 'B' in IB 3071 and gene 'C' in IB 3076. Gene B and gene C were found to be the same as or allelic or tightly linked to each other. Results of this study indicated that gene B, gene C and Pc 68 were either the same or allelic or tightly linked. Gene A may be new and useful gene. Therefore, further study with the known crown rust races is required to substantiate this conclusion.

4.0 SCREENING FOR CROWN RUST RESISTANCE

4.1 Introduction

A few cultivars dominate oat production because of their high agronomic performance. In developing and improving commercial cultivars, oat lines that were not as good as the selected few, with respect to yield and quality, were often taken out of production even though they might posses some desirable traits such as disease resistance. Improving disease resistance, however, requires sources of parental materials possessing resistance genes other than those in the few cultivars in use which are of limited genetic background. Consequently, additional sources of resistance to crown rust are needed.

Several thousand wild oat accessions of all ploidy levels (diploids, teraploids and hexaploids) have been collected from the Mediterranean region (Spain, Portugal, Morocco and the Canary Islands) in the late sixties and early seventies. One of the expected uses of these accessions was to obtain additional resistance genes. Most of the accessions in the collections have been screened for reactions to several crown rust races. Several accessions, especially of Avena sterilis L., were found to be sources of resistance genes for crown rust. Some of these resistance genes were incorporated into

cultivated oats. With the objective of identifying new sources of crown rust resistance genes wild oat accessions that were not previously screened for crown rust resistance, were screened with seven crown rust races (CR 13, CR 25, CR 36, CR 50, CR 56, CR 77 and CR 107). These races attack a wide range of resistance genes some of which are the genes that are present in the currently grown cultivars in the Prairies.

4.2 Materials

4.2.1 Oats

A total of one hundred and eighty four accessions of wild oats of all ploidy levels were grown. There were thirty nine diploids (2n=14), forty four tetraploids (2n=28), ninety six hexaploids(2n=42) and also another five accessions whose ploidy levels were not determined (chromosome counting was not done). Chromosome number counting and rating for rust reactions to some isolates of crown rust were previously made by several people. Along with the wild oats, the susceptible hexaploid cultivar Calibre was also grown as a susceptible check. Two flats of differential sets, each containing twenty single gene Pc lines (Appendix A), were also planted with each batch of wild oats.

For the purpose of increasing rust, twenty 15 cm fibre pots with five plants/pot for each of the single gene Pc host

lines (Pc 35, Pc 38, Pc 39, Pc 40, Pc 46 and variety Bond) were grown in separate green houses. When crown rust was to be increased on seedlings, eight to ten seeds were planted in a hill at the centre of a 13 cm fibre pot.

4.2.2. Crown rust

Seven crown rust isolates were used to screen one hundred and eighty four wild oat accessions. The isolates and the ineffective single gene Pc lines against these isolates is shown in Table 4.2.2.1. These isolates in combination attack eighteen different Pc genes, with a range of virulence from an isolate attacking one gene only (Pc 56) to a highly virulent isolate attacking eight different Pc genes (CR 13). Using this wide range of virulences will permit screening the accessions for a range of effective crown rust resistance genes.

Table 4.2.2.1 Ineffective Pc genes against
seven crown rust test isolates

Isolates	Ineffective Pc genes
13	39, 40, 45, 46, 48, 54, 55, 60
25	39, 40, 45, 46, 48, 54, 55, 60
36	38, 40, 63, 65, 67
50	35, 50, 56
56	56
77	35, 40, 46, 47, 59
107	40, 46, 62

(Chong unpublished)

4.3 Methods

4.3.1 Oats

To obtain healthy and vigorously growing seedlings, seeds from each of the wild oat accessions and Calibre were dehulled, soaked in GA_3, treated with Maneb and planted as explained in chapter 3 (3.3.1.1). Seeds of the differential set and also the single gene Pc lines used for increasing rust were neither dehulled nor soaked in GA_3 because they germinated uniformly. For each wild oat accessions six seeds/pot were planted and seedlings were grown in greenhouses. Attempts were made to maintain the day/night temperature at 20^0C during the day and 15^0C during the night. The light/dark cycle was 16 hours light and eight hours dark. Oat plants that were grown to maturity were staked for support and the panicles were bagged to collect the seeds that disarticulate which is typical of some wild oat species.

4.3.2. Crown rust

4.3.2.1. Inoculation

The method used to inoculate both seedlings and adult plants was talc inoculation described in chapter 3 (3.3.2.1.2). Crown rust races CR 13, CR 56 and CR 107 were inoculated on

the first leaf (Zadoks scale 11), CR 36 and CR 50 on the second leaf (Zadoks scale 12) and CR 25 and CR 77 on the flag leaf (Zadoks scale 39).

4.3.2.2. Increasing rust

Crown rust inoculum was increased in the greenhouse. Increasing a particular crown rust race on a specific host, a host which served as a biological screen, was necessary to maintain the purity of each particular race. Rust increases were not made on universal suscepts such as Makuru, which would equally favour the growth of all races. The seven crown rust isolates used in this study and their respective hosts on which rusts were increased is shown in Table 4.3.2.2.1.

Table 4.3.2.2.1. Crown rust isolates and the hosts on which they were increased.

Isolate	Host
CR 13	Pc 39
CR 25	Pc 39
CR 36	Pc 38
CR 50	Pc 35
CR 56	Bond
CR 77	Pc 46
CR 107	Pc 40

To increase the seven isolates that were obtained from the rust laboratory of Agriculture and Agri-food Canada, Winnipeg Research Centre, twenty pots with five host plants/pot for each of the seven races were grown in 15 cm fibre pots in separate greenhouses. When the flag leaves were

fully developed (Zadoks scale 39), the plants were inoculated with the appropriate race using talc as a spore carrier and incubated in high humidity chamber. Infected plants were allowed to grow by removing their panicles so that seed setting and grain filling would not compete with rust production. In addition, the amount and frequency of watering was also increased since rusted plants required a substantially more water compared to healthy plants.

When a sufficient number of rust spores was produced (about two to three weeks post inoculation), a relatively dry pot with the host plants was carefully lifted, tilted and the plants were gently tapped with a bamboo stick so that rust spores would land on a dry wax paper. Spores were then collected and sieved with a 250μm sieve, Canadian Standard Sieve # 60, meant for this purpose. A desired amount of spores were then put into labelled small tubes. The tubes were then put into a desiccator for at least three hours so that the spores would dry. Finally the tubes were sealed and stored in a deep freezer at a temperature of -75^0C $^+/-5^0C$ until required.

Crown rust was also increased on seedlings. By this method, eight to ten seeds were planted in a clump at the centre of a 13-cm fibre pot. When the seedlings just emerged, 40ml Maleic Hydrazide solution (1.5 litres concentrated Maleic

Hydrazide into 18 litres of water) was added to each of the pots to arrest development of the second leaf. When the first leaf was fully expanded, seedlings were inoculated with the desired race using talc as a carrier. Two to three weeks later, spores were collected and stored as described above.

4.4 Results and discussion

Rust reactions to the seven isolates were scored (Appendix H). Repeating some of the tests was necessary to score rust reactions for all of the accessions and all of the isolates. However, when an accession was found to be susceptible to more than three races, repeats with other races was not done since the objective of the screening was to identify those accessions possessing resistance gene(s) to a wide range of crown rust races that might be useful in future oat breeding programs.

Table 4.4.1. IB accessions resistant to all the seven crown rust isolates (CR 13, CR 25, CR 36, CR 50, CR 56, CR 77 and CR 107).

IB Number	Chromosome Number	Origin	
		Country	Location
2100-108	14	Portugal	Reguengos Mo De
2100-115	14	"	" " "
2100-121	42	"	" " "
2100-128	14	"	Elvora
2472	42	"	Sao Maktinho
3253	28	Spain	La Carlata

Out of the 184 accessions, only six accessions were found to be resistant to all the seven test races (Table 4.4.1). Three accessions (IB 2100-108, IB 2100-115, and IB 2100-128) were diploids, one accession (IB 3253) was tetraploid and the other two (IB 2100-121 and IB 2472) were hexaploid. The two hexaploid accessions are potentially useful in that the resistance genes could easily be transferred into cultivated oats unlike the difficulty of transferring resistance genes from lower ploidy levels. Diploid and tetraploid accessions possessing crown rust resistance genes could be useful despite the difficulty of interspecific transfer. Therefore, since they are sources of resistance for the seven races, they should be maintained in the germplasm collection to be used in oat improvement when the need arises.

If the resistance observed in each of these six accessions is due to just one gene (instead of an accession carrying several crown rust resistance genes), the genes in these six accessions could be previously unidentified genes. However, just as Pc 58, Pc 61, Pc 64 and Pc 68 are resistant to the seven tester races used, the resistance genes in these six accessions could be one of these Pc genes. Additional studies would be required to determine whether the resistance in these accessions is mono- or multi-genic and whether the genes have been previously described.

When the accessions were screened with the seven races, seven tetraploid and two hexaploid accessions were found to be resistant to six of the seven test races (Table 4.4.2).

Table 4.4.2 IB accessions resistant to six crown rust isolates

IB Number	Chromosome Number	Origin		Susceptible to isolate
		Country	Location	
581	28	Canary Islands	La Palma Is.	13
866	28	" "	Tenerife	77
922	28	" "	Gran Canaria	13
965	28	" "	" "	50
980	28	" "	" "	36
2061	42	Portugal	Alandroal	36
2100-132	42	"	Elvora	36
2394	28	"	Sines	107
3584	28	Spain	Alcaudet	25

By a process of elimination, the genes in the accessions shown in Table 4.2.2 could be identified as known Pc genes or might be unidentified genes. One assumption of these identifications is that there is only one gene for resistance in each of these accessions. More isolates and genetic testing is required to provide further guidance as to the proper identity.

Tetraploid accessions, IB 581 and IB 922 were resistant to all the test races except CR 13. CR 13 is virulent on eight Pc genes (Pc 39, Pc 40, Pc 45, Pc 46, Pc 48, Pc 54, Pc 55 and Pc 60). If each of these accessions is carrying only one crown rust resistance gene, it is possible that the

resistance gene(s) in the above accessions might be one of the eight Pc genes. However, since both CR 13 and CR 25 are virulent on these same eight Pc genes and also because the two tetraploid accessions were not susceptible to race 25 (the race that attacks the same eight Pc genes as CR 13), it is unlikely that the two tetraploid accessions possess one of the above eight Pc genes. Therefore, the resistance gene(s) in accessions IB 581 and IB 922 must be a gene other than Pc 39, Pc 40, Pc 45, Pc 46, Pc 48, Pc 54, Pc 55 and Pc 60. It could also be another unidentified gene(s).

Tetraploid IB 3584 was resistant to all isolates except CR 25. The same reasoning given for accessions IB 581 and IB 922 when tested with CR 13 could be applicable for IB 3584. Therefore, if there is a single crown rust resistance gene in this accession it must be a gene other than the eight Pc genes that both CR 13 and CR 25 attack.

Tetraploid IB 980 and two hexaploids, IB 2061 and IB 2100-132, were resistant to all the test races except for CR 36. This suggests that the gene(s) giving protection against the other races could possibly be either Pc 38, Pc 63, Pc 67 or another known crown rust resistance gene not used in the Winnipeg Research Centre differential set or another unidentified gene(s). CR 36 is virulent on the above three Pc genes as well as including Pc 40. However, the gene(s) in

these accessions can not be Pc 40 because the above accessions were resistant to CR 13, CR 25, CR 77 and CR 107 which are also virulent on Pc 40. Thus, IB 980, IB 2061 and IB 2100-132 could possibly be another source of Pc 38, Pc 63 or Pc 67 or could be a new , previously unidentified gene.

IB 965, a tetraploid, was found to be resistant to all the test races except for CR 50. CR 50 is virulent on Pc 35, Pc 50 and Pc 56 but the resistance gene(s) in this accession can not be Pc 35 since IB 965 was not susceptible to CR 77 which also attacks Pc 35. It is possible that the gene(s) in IB 965 might be Pc 50, Pc 56 or another unidentified gene(s).

Another tetraploid, IB 866, was susceptible to CR 77 only. Even though CR 77 is virulent on Pc 35, Pc 40 and Pc 46 in addition to Pc 59, the resistance gene(s) in this accession could possibly be Pc 59 or unidentified resistance gene(s). Using the same process of elimination as with previously discussed accessions, IB 866 was resistant to CR 50 which is virulent on Pc 35, resistant to CR 13 and CR 25 that are virulent on Pc 40 and Pc 46, and also resistant to CR 36 that is also virulent on Pc 40. Thus, IB 866 could possibly be another source of Pc 59 or it could be a new, previously unidentified resistance gene.

IB 2394, a tetraploid, was found to be resistant to all the test races except for CR 107. Based on the differential set reactions, Pc 40, Pc 46 and Pc 62 are susceptible to CR 107. The gene(s) in IB 2394 could neither be Pc 40 nor Pc 46 because this accession was resistant to CR 13 and CR 25 that are virulent on Pc 40 and Pc 46. In addition, the accession was also resistant to CR 36 which is also virulent on Pc 40. Therefore, the resistance gene(s) in IB 2394 could possibly be Pc 62 or another unidentified gene(s).

Table 4.4.3. IB accessions resistant to five crown rust isolates.

IB Number	Chromosome Number	Origin		Susceptible to isolates
		Country	Location	
641-A	28	Canary Islands	La Palma Is.	13, 25
858	28	" "	Tenerife	36, 77
945	42	" "	Gran Canaria	56, 107
947	28	" "	" "	36, 50
961	42	" "	" "	13, 50
979	28	" "	" "	50, 77
1112	42	Morocco	Talmest	50, 56
1113	42	"	"	50, 56
2252	14	Portugal	Ponte De Sor	36, 107
3335	28	Spain	Venta Nueva	13, 25
3756	42	"	Orihuela	56, 107

Out of the one hundred and eighty four accessions, eleven accessions (a diploid, five tetraploids and five hexaploids) were resistant to five of the seven test races (Table 4.3.3). Two tetraploids, IB 641-A and IB 3335 were susceptible to CR 13 and CR 25. CR 13 and CR 25 are virulent on the same eight

Pc genes (Pc 39, Pc 40, Pc 45, Pc 46, Pc 48, Pc 54, Pc 55 and Pc 60). The resistance gene(s) in the above accessions can not be Pc 40 because the accessions were resistant to CR races that are virulent on Pc 40 (CR 36, CR 77 and CR 107). Similarly it can not be Pc 46 since the accessions were resistant to CR 77 and CR 107. Therefore, the resistance gene(s) in the above accessions could be any one of the remaining six Pc genes (Pc 39, Pc 45, Pc 48, Pc 54, Pc 55, Pc 60) or an other unidentified gene(s).

IB 961, a hexaploid, was susceptible to CR 13 and CR 50. CR 13 and CR 50 in combination are virulent on eleven Pc genes (Pc 39, Pc 40, Pc 45, Pc 46, Pc 48, Pc 54, Pc 55, Pc 60, Pc 35, Pc 50 and Pc 56). The resistance gene(s) in IB 961 can not be one of the first eight Pc genes listed above since IB 961 was resistant to CR 25 which is virulent on the first eight Pc genes. Similarly since the above accession was resistant to CR 77, the resistance gene(s) in IB 961 can not be Pc 35 but could possibly be Pc 50, Pc 56 or another unidentified gene(s).

A tetraploid, IB 947, was resistant to all of the test races except for CR 36 and CR 50. CR 36 and CR 50 in combination are virulent on a total of seven Pc genes (Pc 35, Pc 38, Pc 40, Pc 50, Pc 56, Pc 63, and Pc 67). Since IB 947 was susceptible to the two races and since the two races do

not attack any one of the known Pc genes in common, then the resistance gene(s) in this accession can not be one of the above seven Pc genes. Therefore, the genes giving protection to these two accessions must be an unidentified gene.

IB 858, a tetraploid, was susceptible to CR 36 and CR 77. These two races together are virulent on seven Pc genes attacking Pc 40 in common. The resistance gene(s) in this accession can not be Pc 40 because CR 13, CR 25 and CR 107 which are virulent on Pc 40, did not attack IB 858. Therefore, the gene(s) in this accession must also be unidentified gene(s).

The diploid, IB 2252, was susceptible to CR 36 and CR 107. CR 36 is virulent on Pc 38, Pc 40, Pc 63 and Pc 67 while CR 107 is virulent on Pc 40, Pc 46 and Pc 62, both races attacking Pc 40 in common. However, since IB 2252 was resistant to CR 13 and CR 25 which are virulent on Pc 40, the resistance gene(s) in IB 2252 can not be Pc 40. This means that the resistance gene(s) in IB 2252 is other than the identified Pc gene(s).

CR 50 and CR 56 were virulent on the two hexaploid accessions, IB 1112 and IB 1113. The two crown rust races in combination are virulent on three Pc genes only, Pc 35, Pc 50 and Pc 56, attacking Pc 56 in common. The resistance gene(s)

in the two accessions could possibly be Pc 56 or another unidentified gene(s) because the two races were virulent on IB 1112 and IB 1113 and also the two accessions were resistant to the other five test races.

CR 50 and CR 77 were virulent on the tetraploid IB 979. The two races in combination are virulent on six races in total (Pc 35, Pc 40, Pc 46, Pc 50, Pc 56 and Pc 59) attacking Pc 35 in common. Based on the process of elimination, the resistance gene in IB 979 could possibly be Pc 35 or an other unidentified gene(s).

IB 945 and IB 3756, both hexaploids, were susceptible to CR 56 and CR 107. The two test races are virulent on four Pc genes (Pc 40, Pc 46, Pc 56 and Pc 62). The resistance gene(s) in the two hexaploids can not be any one of the above Pc genes simply because CR 56 and CR 107 do not attack any of the known Pc genes in common. Thus the gene(s) conferring resistance in the two accessions must be unidentified gene(s).

Eighteen accessions were found to be susceptible to six of the seven test races. Seven hexaploids (IB 939, IB 995, IB 1461, IB 2001, IB 2010, IB 2100-103 and IB 3336) and an accession whose chromosome number has not been determined (IB 2331) were resistant to CR 13 but susceptible to all the rest. Accessions resistant to CR 36 but susceptible to the other six

races were the four hexaploids (IB 990, IB 2100-116, IB 2100-120 and IB 3568). IB 218 and IB 1000-112 (diploids), IB 1242 (a tetraploid) and IB 665 and IB 2200 (hexaploids) were susceptible to all the test races except for CR 77. Also, IB 1167, a tetraploid, was resistant to CR 107.

All of the above eighteen accessions that were resistant to only one of the seven test races did not posses any of the resistance gene(s) presently being in use in the differential set. This is simply because when one of the eighteen accessions was resistant to one of the test races, the same accession was susceptible to the other six races. There is no single gene from the known Pc lines in the differential set that behaves in the manner observed when the eighteen accessions were screened with the seven test races. This suggests that the resistance gene(s) carried in any one of the eighteen accessions for their protection against the particular race to which they were resistant, to be other than the twenty genes used in the current Winnipeg Research Centre differential set. There are many additional known crown rust resistance genes and the genes in these accessions may be previously described crown rust resistance genes.

Twelve accessions, out of which four diploids (IB 336, IB 1275, IB 1309 and IB 2320) and eight hexaploids (IB 991, IB 2013, IB 2028, IB 2100-118, IB 2429-103, IB 2429-117, IB 3530

and IB 3745) were susceptible to all the seven test races. These accessions did not have any protection against the seven test races used to screen the wild oats with. Even though these accessions did not posses resistance genes against the seven crown rust races, they could have other important and unidentified genes conferring resistance to other races. They could also posses other desirable qualities that were not presently recognized. Thus, it is important to make sure that the germplasm is maintained. For this purpose, therefore, seeds from these and other accessions were collected and the seed source was replenished.

4.5 Conclusion

Out of one hundred and eighty four wild oat accessions screened with seven crown rust isolates (CR 13, CR 25, CR 36, CR 50, CR 56, CR 77 and CR 107), six of them , three diploids (IB 2100-108, 2100-115 and 2100-128), a tetraploid (IB 3253) and two hexaploids (IB 2100-121 and IB 2472) were found to be resistant to all the seven races. Another nine accessions, seven tetraploids (IB 581, IB 866, IB 922, IB 965, IB 980, IB 2394 and IB 3584) and two hexaploids (IB 2061 and IB 2100-132) were resistant to six of the seven races. In addition, eleven accessions, a diploid (IB 2252), five tetraploids (IB 641-A, IB 858, IB 947, IB 979 and IB 3335) and five hexaploids (IB 945, IB 961, IB 1112, IB 1113 and IB 3756) were resistant to five of the seven races.

There were twelve accessions that were susceptible to all seven races while eighteen accessions were susceptible to six of the seven races. The rest of the accessions, one hundred and twenty eight of them, were susceptible to three or four of the seven races.

The resistant accessions identified could possess new sources of resistance gene(s) that might be useful in oat breeding programs. The two resistant hexaploids, IB 2100-121 and IB 2472, are of a particular importance to breeding programs due to the expected ease of transferring their resistance gene(s) into cultivated hexaploid oats.

5.0 GENERAL CONCLUSION

The project had two parts. The first one was the study of the inheritance of crown rust resistance in three Iberian wild oat accessions. In this study it was found that each of the three accessions possess a single dominant gene conferring resistance to the two crown rust isolates used, CR 13 and CR 50. The resistance genes in the three accessions were simply inherited. The relationships of the gene in IB 2435, gene 'A', with the known Pc genes and also with the gene in the other two accessions was studied. It was found that gene 'A' was independent of the eight Pc genes (Pc 38, Pc 39, Pc 48, Pc 58, Pc 59, Pc 61 Pc 64, and Pc 68). Gene 'A' was also independent of the resistance gene in IB 3071 and the gene in IB 3076. A further study with other known Pc genes is required to establish whether or not this gene is new. Gene 'A' may be new and previously unidentified and it could potentially be useful in oat breeding programs. Gene 'B' and Gene 'C' were the same as or allelic or tightly linked to each other and also to Pc 68.

Understanding the number and the nature of inheritance of crown rust resistance genes is useful. This helps in deciding how to utilize resistance genes in improving crown rust resistance in oats. Gene 'A' could be incorporated into the currently cultivated cultivars with known genes. This

process, pyramiding of genes (Fleischmann and McKenzie, 1968; Chong, 1988b) will help to overcome the rapid breakdown of resistance which is likely to happen if resistance genes were singly incorporated into a cultivar.

Crown rust fungus, like all disease causing organisms has no national boundaries. No one country could permanently control the disease alone. This is true if two or more countries are located in the path of the fungus such as the Puccinia path in North America. Therefore, a joint effort of control strategy by the countries involved in the Puccinia path is needed. In North America, for example, since the disease progresses northwards from warmer to colder climate, regional release of varieties with different genetic backgrounds (Fleischmann and McKenzie, 1968; Kiehn et al., 1976; Chong, 1988b) could be used to contain the disease in a certain area along the Puccinia path. This is where gene 'A' could be utilized in addition to its usefulness in a multiline program.

The second part of the project was screening for crown rust resistance in one hundred and eighty four IB accessions. There were thirty nine diploids, forty four tetraploids, ninety six hexaploids and also five accessions whose chromosome number were not determined. It was found that six accessions were resistant to seven crown rust isolates (CR 13,

CR 25, CR 36, CR 50, CR 56, CR 77 and CR 107). Another nine accessions were resistant to six of the isolates.

The process of screening is the initial step in the long process of improving crown rust resistance in oats. The six IB accessions, the two hexaploids in particular, are quite useful since potentially they could be a new source of crown rust resistance gene(s) that could easily be transferred into cultivated oats. The next step is to conduct inheritance studies to determine the number and nature of inheritance of the resistance genes.

The search for and collection of new sources of resistance genes should continue. The potential source is only from the diversity of oat species from the centre of origin of the crop-the Mediterranean region and the Ethiopian highlands. The benefit of germplasm collection and screening for resistance should not be underestimated. It is only because of the effort of several researchers in the past that wild oat accessions were collected, screened and new sources of crown rust resistance were found that could be used to develop crown rust resistant cultivars that are presently cultivated. Similar effort must be continued.

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APPENDICES

Appendix A. The differential set used in this study and its reaction to the seven crown rust races (CR 13, CR 25, CR 36, CR 50, CR 56, CR 77 and CR 107).

PC gene	CR 13	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
35	;	;	;	4	4	4	;
38	; 1	;	4	;	;	; 1	;
39	4	4	;	;	;	;	;
40	4	4	4	;	; 1	4	4
45	4	4	;	; 1	;	;	;
46	4	4	; 1	1 2	;	4	4
48	4	4	;	0 ;	;	;	;
50	0 ;	;	;	4	;	0 ;	;
54	4	4	; 1	; 1	; 1	;	;
55	4	4	; 1	;	;	;	;
56	;	;	; 1	4	4	;	;
58	;	;	;	;	0 ;	; 1	;
59	;	;	;	;	; 1	4	;
60	4	4	; 1	;	; 1	;	;
61	;	;	;	;	0 ;	;	;
62	;	;	;	;	;	;	4
63	;	;	4	;	;	;	;
64	;	;	; 1	;	;	;	;
67	4	4	4	;	4	4	4
68	0	0	0	0	0	0	0

Appendix B. Segregating seedlings from F_3 families of
Calibre/IB2435 cross

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
1	CR 13	15	4	3:1	0.424	0.50 - 0.70
	CR 50	15	4	3:1	0.424	0.50 - 0.70
2	CR 13	12	8	3:1	2.067	0.10 - 0.20
	CR 50	12	8	3:1	2.067	0.10 - 0.20
3	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
4	CR 13	17	3	3:1	1.400	0.20 - 0.30
	CR 50	17	3	3:1	1.400	0.20 - 0.30
5	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	17	5	3:1	0.182	0.50 - 0.70
6	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
7	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
8	CR 13	16	3	3:1	1.175	0.30 - 0.50
	CR 50	16	3	3:1	1.175	0.30 - 0.50
9	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
10	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	16	6	3:1	0.061	0.70 - 0.90
11	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	18	6	3:1	0.056	0.70 - 0.90
12	CR 13	16	4	3:1	0.467	0.50 - 0.70
	CR 50	16	4	3:1	0.467	0.50 - 0.70
13	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	13	8	3:1	1.635	0.10 - 0.20
14	CR 13	14	8	3:1	1.273	0.20 - 0.30
	CR 50	14	8	3:1	1.273	0.20 - 0.30
15	CR 13	17	4	3:1	0.619	0.30 - 0.50
	CR 50	17	4	3:1	0.619	0.30 - 0.50
16	CR 13	14	8	3:1	1.273	0.20 - 0.30
	CR 50	14	8	3:1	1.273	0.20 - 0.30
17	CR 13	16	3	3:1	1.175	0.30 - 0.50
	CR 50	16	3	3:1	1.175	0.30 - 0.50
18	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	16	5	3:1	0.111	0.70 - 0.90
19	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
20	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	19	3	3:1	1.879	0.10 - 0.20
21	CR 13	12	6	3:1	0.519	0.30 - 0.50
	CR 50	12	6	3:1	0.519	0.30 - 0.50

Appendix B. cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
22	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 50	16	6	3:1	0.061	0.70 - 0.90
23	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	19	3	3:1	1.879	0.10 - 0.20
24	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	14	7	3:1	0.619	0.30 - 0.50
25	CR 13	14	7	3:1	0.619	0.30 - 0.50
	CR 50	15	6	3:1	0.111	0.70 - 0.90
26	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 50	16	6	3:1	0.061	0.70 - 0.90
27	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	19	3	3:1	1.879	0.10 - 0.20
28	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
29	CR 13	21	6	3:1	0.235	0.50 - 0.70
	CR 50	21	6	3:1	0.235	0.50 - 0.70
30	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	18	6	3:1	0.056	0.70 - 0.90
31	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
32	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
33	CR 13	21	4	3:1	1.373	0.70 - 0.90
	CR 50	21	4	3:1	1.373	0.70 - 0.90
34	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	18	6	3:1	0.056	0.70 - 0.90
35	CR 13	12	4	3:1	0.083	0.70 - 0.90
	CR 50	12	4	3:1	0.083	0.70 - 0.90
36	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
37	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
38	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
39	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
40	CR 13	16	8	3:1	0.722	0.30 - 0.50
	CR 50	16	8	3:1	0.722	0.30 - 0.50
41	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
42	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	19	5	3:1	0.389	0.50 - 0.70

Appendix B.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
43	CR 13	14	4	3:1	0.222	0.50 - 0.70
	CR 50	15	3	3:1	0.963	0.30 - 0.50
44	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	21	4	3:1	1.373	0.20 - 0.30
45	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
46	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
47	CR 13	21	4	3:1	1.373	0.30 - 0.50
	CR 50	21	4	3:1	1.373	0.30 - 0.50
48	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 50	17	8	3:1	0.520	0.30 - 0.50
49	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
50	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
51	CR 13	15	7	3:1	0.424	0.50 - 0.70
	CR 50	15	7	3:1	0.424	0.50 - 0.70
52	CR 13	13	3	3:1	0.583	0.30 - 0.50
	CR 50	13	3	3:1	0.583	0.30 - 0.50
53	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
54	CR 13	15	4	3:1	0.333	0.50 - 0.70
	CR 50	16	3	3:1	1.175	0.20 - 0.30
55	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
56	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
57	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
58	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
59	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	18	5	3:1	0.275	0.50 - 0.70
60	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	21	3	3:1	2.389	0.30 - 0.50
61	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
62	CR 13	18	3	3:1	1.635	0.20 - 0.30
	CR 50	18	3	3:1	1.635	0.20 - 0.30
63	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 50	16	9	3:1	1.371	0.20 - 0.30

Appendix B.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
64	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	19	4	3:1	0.971	0.30 - 0.50
65	CR 13	15	2	3:1	2.020	0.10 - 0.20
	CR 50	15	2	3:1	2.020	0.10 - 0.20
66	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 50	18	7	3:1	0.093	0.70 - 0.90
67	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
68	CR 13	21	4	3:1	1.080	0.20 - 0.30
	CR 50	21	4	3:1	1.080	0.20 - 0.30
69	CR 13	18	3	3:1	1.635	0.20 - 0.30
	CR 50	18	3	3:1	1.635	0.20 - 0.30
70	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
71	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
72	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
73	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
74	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	20	4	3:1	1.167	0.20 - 0.30
75	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	17	5	3:1	0.182	0.50 - 0.70
76	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	21	4	3:1	1.373	0.20 - 0.30
77	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
78	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
79	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90
80	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
81	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
82	CR 13	15	2	3:1	2.020	0.10 - 0.20
	CR 50	15	2	3:1	2.020	0.10 - 0.20
83	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	20	3	3:1	2.130	0.10 - 0.20
84	CR 13	15	2	3:1	2.020	0.10 - 0.20
	CR 50	15	2	3:1	2.020	0.10 - 0.20

Appendix B.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
85	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	19	4	3:1	0.971	0.70 - 0.90
86	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	19	4	3:1	0.971	0.30 - 0.50
87	CR 13	21	4	3:1	1.080	0.20 - 0.30
	CR 50	21	4	3:1	1.080	0.20 - 0.30
88	CR 13	21	4	3:1	1.080	0.20 - 0.30
	CR 50	21	4	3:1	1.080	0.20 - 0.30
89	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
90	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	20	4	3:1	1.167	0.20 - 0.30
91	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	18	5	3:1	0.275	0.50 - 0.70
92	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 50	16	9	3:1	1.373	0.20 - 0.30
93	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	19	6	3:1	0.093	0.70 - 0.90
94	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	15	6	3:1	0.111	0.70 - 0.90
95	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
96	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	21	4	3:1	1.373	0.20 - 0.30
97	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
98	CR 13	22	4	3:1	1.590	0.20 - 0.30
	CR 50	21	5	3:1	0.667	0.70 - 0.90
99	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
100	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90
101	CR 13	10	6	3:1	1.083	0.20 - 0.30
	CR 50	12	4	3:1	0.083	0.70 - 0.90
102	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	20	3	3:1	2.130	0.10 - 0.20
103	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	19	4	3:1	0.971	0.70 - 0.90
104	CR 13	20	6	3:1	0.154	0.50 - 0.70
	CR 50	18	8	3:1	0.154	0.50 - 0.70
105	CR 13	19	9	3:1	1.373	0.20 - 0.30
	CR 50	19	9	3:1	1.373	0.20 - 0.30

Appendix B.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
106	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90
107	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
108	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
109	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
110	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	19	4	3:1	0.971	0.30 - 0.50
111	CR 13	15	4	3:1	0.333	0.50 - 0.70
	CR 50	15	4	3:1	0.333	0.50 - 0.70
112	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
113	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
114	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
115	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	18	6	3:1	0.056	0.70 - 0.90
116	CR 13	15	7	3:1	0.424	0.50 - 0.70
	CR 50	15	7	3:1	0.424	0.50 - 0.70

Appendix C. Segregating seedlings from BC_1F_2 families of Calibre/IB 2435 backcross

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
1	CR 13	17	4	3:1	0.619	0.30 - 0.50
	CR 13	17	4	3:1	0.619	0.30 - 0.50
2	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 13	15	7	3:1	0.424	0.50 - 0570
3	CR 13	16	7	3:1	0.275	0.50 - 0.70
	CR 13	15	8	3:1	0.971	0.30 - 0.50
4	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 13	16	6	3:1	0.061	0.70 - 0.90
5	CR 13	17	4	3:1	0.619	0.30 - 0.50
	CR 13	16	5	3:1	0.111	0.70 - 0.90
6	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 13	16	5	3:1	0.111	0.70 - 0.90
7	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 13	16	6	3:1	0.061	0.70 - 0.90
8	CR 13	14	8	3:1	1.273	0.20 - 0.30
	CR 13	14	8	3:1	1.273	0.20 - 0.30
9	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 13	18	7	3:1	0.093	0.70 - 0.90
10	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 13	19	5	3:1	0.389	0.50 - 0.70
11	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 13	17	7	3:1	0.167	0.50 - 0.70
12	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 13	16	9	3:1	1.373	0.20 - 0.30
13	CR 13	16	8	3:1	0.722	0.30 - 0.50
	CR 13	17	7	3:1	0.167	0.50 - 0.70
14	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 13	21	4	3:1	1.373	0.20 - 0.30
15	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 13	17	7	3:1	0.167	0.50 - 0.70
16	CR 13	28	5	3:1	2.010	0.10 - 0.20
	CR 13	25	8	3:1	0.071	0.70 - 0.90
17	CR 13	26	6	3:1	0.875	0.30 - 0.50
	CR 13	24	8	3:1	0.042	0.70 - 0.90
18	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 13	19	5	3:1	0.389	0.50 - 0.70
19	CR 13	13	6	3:1	0.333	0.50 - 0.70
	CR 13	13	6	3:1	0.333	0.50 - 0.70
20	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 13	21	3	3:1	2.389	0.10 - 0.20
21	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 13	20	5	3:1	0.520	0.30 - 0.50

Appendix C.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
22	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 13	20	5	3:1	0.520	0.30 - 0.50
23	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 13	19	6	3:1	0.093	0.70 - 0.90
24	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 13	18	6	3:1	0.056	0.70 - 0.90
25	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 13	20	4	3:1	1.167	0.20 - 0.30
26	CR 13	16	4	3:1	0.467	0.30 - 0.50
	CR 13	14	6	3:1	0.200	0.50 - 0.70
27	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 13	21	3	3:1	2.389	0.10 - 0.20
28	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 13	16	8	3:1	0.722	0.30 - 0.50
29	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 13	22	3	3:1	2.653	0.10 - 0.20
30	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 13	17	7	3:1	0.167	0.50 - 0.70
31	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 13	20	5	3:1	0.520	0.30 - 0.50
32	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 13	19	5	3:1	0.389	0.50 - 0.70
33	CR 13	23	4	3:1	1.815	0.20 - 0.30
	CR 13	23	4	3:1	1.815	0.20 - 0.30
34	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	18	6	3:1	0.056	0.70 - 0.90
35	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
36	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
37	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
38	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90

Appendix D. Segregating seedlings from F_3 families of
Calibre/IB 3071 cross

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
1	CR 13	16	4	3:1	0.467	0.30 - 0.50
	CR 50	17	3	3:1	1.400	0.20 - 0.30
2	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	18	3	3:1	1.635	0.20 - 0.30
3	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	20	2	3:1	3.455	0.05 - 0.10
4	CR 13	10	6	3:1	1.083	0.20 - 0.30
	CR 50	13	3	3:1	0.583	0.30 - 0.50
5	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	20	2	3:1	3.455	0.05 - 0.10
6	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 50	19	3	3:1	1.879	0.10 - 0.20
7	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	20	2	3:1	3.455	0.05 - 0.10
8	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
9	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
10	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
11	CR 13	16	4	3:1	0.467	0.30 - 0.50
	CR 50	17	3	3:1	1.400	0.20 - 0.30
12	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	17	4	3:1	0.619	0.30 - 0.50
13	CR 13	17	3	3:1	1.400	0.20 - 0.30
	CR 50	16	4	3:1	0.467	0.30 - 0.50
14	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	15	6	3:1	0.111	0.70 - 0.90
15	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
16	CR 13	18	3	3:1	1.635	0.20 - 0.30
	CR 50	18	3	3:1	1.635	0.20 - 0.30
17	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	16	6	3:1	0.061	0.70 - 0.90
18	CR 13	14	4	3:1	0.222	0.50 - 0.70
	CR 50	16	2	3:1	2.296	0.10 - 0.20
19	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
20	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 50	17	7	3:1	0.167	0.50 - 0.70
21	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	23	4	3:1	1.373	0.20 - 0.30

Appendix D. cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
22	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	15	8	3:1	0.971	0.70 - 0.90
23	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
24	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90
25	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	21	4	3:1	1.373	0.20 - 0.30
26	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	20	2	3:1	3.455	0.05 - 0.10
27	CR 13	13	3	3:1	0.583	0.30 - 0.50
	CR 50	13	3	3:1	0.583	0.30 - 0.50
28	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
29	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	19	4	3:1	0.971	0.70 - 0.90
30	CR 13	14	4	3:1	0.222	0.50 - 0.70
	CR 50	14	4	3:1	0.222	0.50 - 0.70
31	CR 13	21	6	3:1	0.235	0.50 - 0.70
	CR 50	21	6	3:1	0.235	0.50 - 0.70
32	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
33	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	19	5	3:1	0.389	0.50 - 0.70
34	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 50	20	4	3:1	1.167	0.20 - 0.30
35	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	20	4	3:1	1.167	0.20 - 0.30
36	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
37	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
38	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	19	5	3:1	0.389	0.50 - 0.70
39	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	21	4	3:1	1.373	0.20 - 0.30
40	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	20	2	3:1	3.455	0.05 - 0.10
41	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	19	4	3:1	0.971	0.70 - 0.90
42	CR 13	17	4	3:1	0.619	0.30 - 0.50
	CR 50	17	4	3:1	0.619	0.30 - 0.50

Appendix D.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
43	CR 13	15	4	3:1	0.333	0.50 - 0.70
	CR 50	16	3	3:1	1.175	0.20 - 0.30
44	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
45	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90
46	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
47	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	19	4	3:1	0.971	0.30 - 0.50
48	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 50	17	7	3:1	0.167	0.50 - 0.70
49	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	20	5	3:1	0.520	0.30 - 0.50
50	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.34
51	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	21	4	3:1	1.373	0.20 - 0.30
52	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
53	CR 13	15	5	3:1	0.067	0.70 - 0.90
	CR 50	18	2	3:1	2.867	0.05 - 0.10
54	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
55	CR 13	16	7	3:1	0.275	0.50 - 0.70
	CR 50	20	3	3:1	2.130	0.10 - 0.20
56	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 50	17	7	3:1	0.167	0.50 - 0.70
57	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 50	17	7	3:1	0.167	0.50 - 0.70
58	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
59	CR 13	18	3	3:1	1.635	0.20 - 0.30
	CR 50	18	3	3:1	1.635	0.20 - 0.30
60	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
61	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 50	17	7	3:1	0.167	0.50 - 0.70
62	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
63	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	21	4	3:1	1.373	0.20 - 0.30

Appendix D.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
64	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	20	3	3:1	2.130	0.10 - 0.20
65	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
66	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
67	CR 13	17	4	3:1	0.619	0.30 - 0.50
	CR 50	18	3	3:1	1.635	0.20 - 0.30
68	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
69	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	20	3	3:1	2.130	0.10 - 0.20
70	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	18	5	3:1	0.275	0.50 - 0.70
71	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
72	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
73	CR 13	24	5	3:1	1.184	0.10 - 0.20
	CR 50	24	5	3:1	1.184	0.10 - 0.20
74	CR 13	20	3	3:1	2.130	0.10 - 0.20
	CR 50	20	3	3:1	2.130	0.10 - 0.20
75	CR 13	20	3	3:1	2.130	0.10 - 0.20
	CR 50	21	2	3:1	3.754	0.05 - 0.10
76	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
77	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	16	5	3:1	0.111	0.70 - 0.90
78	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
79	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
80	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 50	17	7	3:1	0.167	0.50 - 0.70
81	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
82	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	15	6	3:1	0.111	0.70 - 0.90
83	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
84	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	19	4	3:1	0.971	0.30 - 0.50

Appendix D.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
85	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
86	CR 13	16	4	3:1	0.467	0.30 - 0.50
	CR 50	17	3	3:1	1.400	0.20 - 0.30

Appendix E. Segregating seedlings from BC_1F_2 families of
Calibre/IB 3071 backcross

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
1	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
2	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	14	7	3:1	0.619	0.30 - 0.50
3	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
4	CR 13	17	3	3:1	1.400	0.20 - 0.30
	CR 50	18	2	3:1	2.867	0.05 - 0.10
5	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
6	CR 13	13	8	3:1	1.635	0.20 - 0.30
	CR 50	13	8	3:1	1.635	0.20 - 0.30
7	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	15	6	3:1	0.111	0.70 - 0.90
8	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	17	4	3:1	0.619	0.30 - 0.50
9	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
10	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
11	CR 13	15	7	3:1	0.424	0.50 - 0.70
	CR 50	15	7	3:1	0.424	0.50 - 0.70
12	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
13	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
14	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	19	4	3:1	0.971	0.30 - 0.50
15	CR 13	17	4	3:1	0.619	0.70 - 0.90
	CR 50	16	5	3:1	0.111	0.30 - 0.50
16	CR 13	18	6	3:1	0.056	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
17	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
18	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	15	8	3:1	0.971	0.30 - 0.50
19	CR 13	15	7	3:1	0.424	0.50 - 0.70
	CR 50	15	7	3:1	0.424	0.50 - 0.70
20	CR 13	17	3	3:1	1.400	0.20 - 0.30
	CR 50	17	3	3:1	1.400	0.20 - 0.30
21	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30

Appendix E. cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
22	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
23	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
24	CR 13	13	8	3:1	1.635	0.20 - 0.30
	CR 50	13	8	3:1	1.635	0.20 - 0.30
25	CR 13	17	8	3:1	0.520	0.30 - 0.50
	CR 50	16	9	3:1	1.373	0.30 - 0.50
26	CR 13	17	7	3:1	0.167	0.50 - 0.70
	CR 50	17	7	3:1	0.167	0.50 - 0.70
27	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
28	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
29	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	19	6	3:1	0.093	0.70 - 0.90
30	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	17	6	3:1	0.043	0.70 - 0.90
31	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	16	5	3:1	0.111	0.70 - 0.90
32	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
33	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
34	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
35	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	16	9	3:1	1.373	0.20 - 0.30
36	CR 13	14	4	3:1	0.222	0.50 - 0.70
	CR 50	14	4	3:1	0.222	0.50 - 0.70
37	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	17	8	3:1	0.520	0.30 - 0.50
38	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
39	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
40	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
41	CR 13	16	8	3:1	0.722	0.30 - 0.50
	CR 50	16	8	3:1	0.722	0.30 - 0.50
42	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	20	5	3:1	0.520	0.30 - 0.50

Appendix E. cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
43	CR 13	17	8	3:1	0.520	0.30 - 0.50
	CR 50	17	8	3:1	0.520	0.30 - 0.50
44	CR 13	16	8	3:1	0.722	0.30 - 0.50
	CR 50	16	8	3:1	0.722	0.30 - 0.50
45	CR 13	17	9	3:1	1.077	0.20 - 0.30
	CR 50	17	9	3:1	1.077	0.20 - 0.30
46	CR 13	18	9	3:1	0.827	0.30 - 0.50
	CR 50	18	9	3:1	0.827	0.30 - 0.50
47	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
48	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
49	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 50	17	8	3:1	0.520	0.30 - 0.50
50	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
51	CR 13	23	3	3:1	2.923	0.05 - 0.10
	CR 50	23	3	3:1	2.923	0.05 - 0.10
52	CR 13	17	8	3:1	0.520	0.30 - 0.50
	CR 50	16	9	3:1	1.373	0.20 - 0.30
53	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
54	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	16	5	3:1	0.111	0.70 - 0.90
55	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
56	CR 13	14	6	3:1	0.200	0.30 - 0.50
	CR 50	16	4	3:1	0.467	0.30 - 0.50
57	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 50	20	5	3:1	0.520	0.30 - 0.50
58	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	17	6	3:1	0.043	0.70 - 0.90
59	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	16	5	3:1	0.111	0.70 - 0.90
60	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
61	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	16	9	3:1	1.373	0.20 - 0.30

Appendix F. Segregating seedlings from F_3 families of
Calibre/IB 3076 cross

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
1	CR 13	14	8	3:1	1.273	0.20 - 0.30
	CR 50	16	6	3:1	0.061	0.70 - 0.90
2	CR 13	14	8	3:1	1.273	0.20 - 0.30
	CR 50	15	7	3:1	0.424	0.50 - 0.70
3	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 50	17	5	3:1	0.182	0.50 - 0.70
4	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	18	4	3:1	0.788	0.30 - 0.50
5	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	18	4	3:1	0.788	0.30 - 0.50
6	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	20	2	3:1	3.455	0.05 - 0.10
7	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
8	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
9	CR 13	18	3	3:1	1.440	0.20 - 0.30
	CR 50	18	3	3:1	1.440	0.20 - 0.30
10	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	19	4	3:1	0.971	0.30 - 0.50
11	CR 13	14	6	3:1	0.200	0.50 - 0.70
	CR 50	14	6	3:1	0.200	0.50 - 0.70
12	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	20	5	3:1	0.520	0.30 - 0.50
13	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
14	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
15	CR 13	13	6	3:1	0.333	0.50 - 0.70
	CR 50	13	6	3:1	0.333	0.50 - 0.70
16	CR 13	17	3	3:1	1.400	0.20 - 0.30
	CR 50	18	2	3:1	2.867	0.10 - 0.20
17	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	19	4	3:1	0.971	0.30 - 0.50
18	CR 13	17	3	3:1	1.400	0.20 - 0.30
	CR 50	16	4	3:1	0.467	0.30 - 0.50
19	CR 13	20	3	3:1	2.130	0.10 - 0.20
	CR 50	18	5	3:1	0.275	0.50 - 0.70
20	CR 13	23	3	3:1	2.923	0.05 - 0.10
	CR 50	22	4	3:1	1.590	0.20 - 0.30
21	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30

Appendix F. cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
22	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	16	5	3:1	0.111	0.70 - 0.90
23	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	.070 - 0.90
24	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
25	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
26	CR 13	17	6	3:1	0.043	0.70 - 0.90
	CR 50	17	6	3:1	0.043	0.70 - 0.90
27	CR 13	20	3	3:1	2.132	0.10 - 0.20
	CR 50	20	3	3:1	2.132	0.10 - 0.20
28	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	17	5	3:1	0.182	0.50 - 0.70
29	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	20	2	3:1	3.455	0.05 - 0.10
30	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
31	CR 13	11	5	3:1	0.250	0.50 - 0.70
	CR 50	11	5	3:1	0.250	0.50 - 0.70
32	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
33	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
34	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
35	CR 13	16	8	3:1	0.722	0.30 - 0.50
	CR 50	17	7	3:1	0.167	0.20 - 0.30
36	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	18	6	3:1	0.056	0.70 - 0.90
37	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
38	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
39	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	21	4	3:1	1.373	0.20 - 0.30
40	CR 13	16	8	3:1	0.722	0.30 - 0.50
	CR 50	16	8	3:1	0.722	0.30 - 0.70
41	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90
42	CR 13	15	7	3:1	0.424	0.50 - 0.70
	CR 50	16	6	3:1	0.061	0.70 - 0.90

Appendix F. cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
43	CR 13	20	3	3:1	2.130	0.10 - 0.20
	CR 50	20	3	3:1	2.130	0.10 - 0.20
44	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	21	4	3:1	1.373	0.20 - 0.30
45	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
46	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
47	CR 13	18	3	3:1	1.635	0.20 - 0.30
	CR 50	18	3	3:1	1.635	0.20 - 0.30
48	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
49	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
50	CR 13	14	7	3:1	0.619	0.30 - 0.50
	CR 50	16	5	3:1	0.111	0.70 - 0.90
51	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	17	7	3:1	0.167	0.50 - 0.70
52	CR 13	20	4	3:1	1.167	0.50 - 0.70
	CR 50	21	4	3:1	1.167	0.50 - 0.70
53	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	18	7	3:1	0.093	0.70 - 0.90
54	CR 13	16	5	3:1	0.111	0.70 - 0.90
	CR 50	18	4	3:1	0.788	0.30 - 0.50
55	CR 13	21	2	3:1	3.754	0.05 - 0.10
	CR 50	21	2	3:1	3.754	0.05 - 0.10
56	CR 13	14	7	3:1	0.619	0.30 - 0.50
	CR 50	14	7	3:1	0.619	0.30 - 0.50
57	CR 13	21	2	3:1	3.754	0.05 - 0.10
	CR 50	21	2	3:1	3.754	0.05 - 0.10
58	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
59	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	20	4	3:1	1.167	0.20 - 0.30
60	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
61	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90
62	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	20	5	3:1	0.520	0.30 - 0.50
63	CR 13	15	5	3:1	0.067	0.70 - 0.90
	CR 50	15	5	3:1	0.067	0.70 - 0.90

Appendix F. cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
64	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	20	4	3:1	1.167	0.20 - 0.30
65	CR 13	13	6	3:1	0.333	0.50 - 0.70
	CR 50	16	3	3:1	1.175	0.20 - 0.30
66	CR 13	20	3	3:1	2.130	0.10 - 0.20
	CR 50	20	3	3:1	2.130	0.10 - 0.20
67	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	21	4	3:1	1.373	0.20 - 0.30
68	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
69	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
70	CR 13	19	7	3:1	0.051	0.70 - 0.90
	CR 50	20	6	3:1	0.154	0.50 - 0.70
71	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	22	3	3:1	2.653	0.10 - 0.20
72	CR 13	18	7	3:1	0.093	0.70 - 0.90
	CR 50	18	7	3:1	0.093	0.70 - 0.90
73	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
74	CR 13	15	5	3:1	0.067	0.70 - 0.90
	CR 50	16	4	3:1	0.467	0.30 - 0.50
75	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
76	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
77	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
78	CR 13	17	4	3:1	0.619	0.30 - 0.50
	CR 50	17	4	3:1	0.619	0.30 - 0.50
79	CR 13	20	3	3:1	2.130	0.10 - 0.20
	CR 50	20	3	3:1	2.130	0.10 - 0.20
80	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	18	6	3:1	0.056	0.70 - 0.90
81	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
82	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
83	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	21	4	3:1	1.373	0.20 - 0.30
84	CR 13	19	4	3:1	0.971	0.30 - 0.50
	CR 50	19	4	3:1	0.971	0.30 - 0.50

Appendix F.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
85	CR 13	17	3	3:1	1.400	0.20 - 0.30
	CR 50	16	4	3:1	0.467	0.30 - 0.50

Appendix G. Segregating seedlings from BC_1F_2 families of
Calibre/IB 3076 backcross

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
1	CR 13	18	3	3:1	1.440	0.20 - 0.30
	CR 50	16	5	3:1	0.111	0.70 - 0.90
2	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	20	2	3:1	3.455	0.05 - 0.10
3	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 50	17	5	3:1	0.182	0.50 - 0.70
4	CR 13	20	2	3:1	3.455	0.05 - 0.10
	CR 50	20	2	3:1	3.455	0.05 - 0.10
5	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	17	5	3:1	0.182	0.50 - 0.70
6	CR 13	17	5	3:1	0.182	0.50 - 0.70
	CR 50	18	4	3:1	0.788	0.30 - 0.50
7	CR 13	16	6	3:1	0.061	0.70 - 0.90
	CR 50	16	6	3:1	0.061	0.70 - 0.90
8	CR 13	19	3	3:1	1.879	0.10 - 0.20
	CR 50	19	3	3:1	1.879	0.10 - 0.20
9	CR 13	15	2	3:1	2.020	0.10 - 0.20
	CR 50	12	2	3:1	2.020	0.10 - 0.20
10	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	14	7	3:1	0.619	0.30 - 0.50
11	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	19	3	3:1	1.879	0.10 - 0.20
12	CR 13	27	6	3:1	1.040	0.30 - 0.50
	CR 50	28	5	3:1	2.010	0.10 - 0.20
13	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	15	6	3:1	0.111	0.70 - 0.90
14	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	22	3	3:1	2.653	0.10 - 0.20
15	CR 13	15	7	3:1	0.424	0.50 - 0.70
	CR 50	15	7	3:1	0.424	0.50 - 0.70
16	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 50	16	9	3:1	1.373	0.20 - 0.30
17	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	18	5	3:1	0.275	0.50 - 0.70
18	CR 13	11	7	3:1	1.556	0.20 - 0.30
	CR 50	11	7	3:1	1.556	0.20 - 0.30
19	CR 13	13	5	3:1	0.074	0.70 - 0.90
	CR 50	13	5	3:1	0.074	0.70 - 0.90
20	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
21	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	18	6	3:1	0.056	0.70 - 0.90

Appendix G.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
22	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	18	4	3:1	0.788	0.30 - 0.50
23	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
24	CR 13	21	2	3:1	3.754	0.05 - 0.10
	CR 50	21	2	3:1	3.754	0.05 - 0.10
25	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	18	6	3:1	0.056	0.70 - 0.90
26	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	18	6	3:1	0.056	0.70 - 0.90
27	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
28	CR 13	19	7	3:1	0.056	0.70 - 0.90
	CR 50	19	7	3:1	0.056	0.70 - 0.90
29	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	17	8	3:1	0.520	0.30 - 0.50
30	CR 13	20	3	3:1	2.130	0.10 - 0.20
	CR 50	18	5	3:1	0.275	0.30 - 0.50
31	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
32	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	20	5	3:1	0.520	0.30 - 0.50
33	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
34	CR 13	22	4	3:1	1.590	0.20 - 0.30
	CR 50	21	5	3:1	0.667	0.30 - 0.50
35	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	18	6	3:1	0.056	0.70 - 0.90
36	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
37	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	18	5	3:1	0.275	0.50 - 0.70
38	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
39	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
40	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
41	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
42	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	20	5	3:1	0.520	0.30 - 0.50

Appendix G. cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
43	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	18	6	3:1	0.056	0.70 - 0.90
44	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	21	4	3:1	1.373	0.20 - 0.30
45	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	19	6	3:1	0.093	0.70 - 0.90
46	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
47	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	20	5	3:1	0.520	0.30 - 0.50
48	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
49	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
50	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	18	6	3:1	0.056	0.70 - 0.90
51	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
52	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
53	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	21	4	3:1	1.373	0.20 - 0.30
54	CR 13	17	4	3:1	0.619	0.30 - 0.50
	CR 50	15	6	3:1	0.111	0.70 - 0.90
55	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
56	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
57	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
58	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
59	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	17	7	3:1	0.167	0.50 - 0.70
60	CR 13	22	7	3:1	0.827	0.30 - 0.50
	CR 50	20	5	3:1	0.037	0.70 - 0.90
61	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
62	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
63	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	21	3	3:1	2.389	0.10 - 0.20

Appendix G.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
64	CR 13	20	4	3:1	1.167	0.20 - 0.30
	CR 50	20	4	3:1	1.167	0.20 - 0.30
65	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
66	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	20	5	3:1	0.520	0.30 - 0.50
67	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	19	5	3:1	0.389	0.50 - 0.70
68	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	20	5	3:1	0.520	0.30 - 0.50
69	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	21	4	3:1	1.373	0.20 - 0.30
70	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
71	CR 13	21	3	3:1	2.389	0.10 - 0.20
	CR 50	21	3	3:1	2.389	0.10 - 0.20
72	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
73	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
74	CR 13	18	5	3:1	0.275	0.50 - 0.70
	CR 50	18	5	3:1	0.275	0.50 - 0.70
75	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	22	3	3:1	2.653	0.10 - 0.20
76	CR 13	17	8	3:1	0.520	0.30 - 0.50
	CR 50	16	9	3:1	1.373	0.20 - 0.30
77	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
78	CR 13	19	6	3:1	0.093	0.70 - 0.90
	CR 50	19	6	3:1	0.093	0.70 - 0.90
79	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
80	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	20	5	3:1	0.520	0.30 - 0.50
81	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	19	5	3:1	0.389	0.50 - 0.70
82	CR 13	18	4	3:1	0.788	0.30 - 0.50
	CR 50	19	3	3:1	1.879	0.10 - 0.20
83	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
84	CR 13	21	2	3:1	3.754	0.05 - 0.10
	CR 50	21	2	3:1	3.754	0.05 - 0.10

Appendix G.   cont'd

Family	Isolate	Resis.	Sus.	Model	X^2	P of X^2
85	CR 13	22	3	3:1	2.653	0.10 - 0.20
	CR 50	22	3	3:1	2.653	0.10 - 0.20
86	CR 13	21	4	3:1	1.373	0.20 - 0.30
	CR 50	21	4	3:1	1.373	0.20 - 0.30
87	CR 13	19	5	3:1	0.389	0.50 - 0.70
	CR 50	21	3	3:1	2.389	0.10 - 0.20
88	CR 13	20	5	3:1	0.520	0.30 - 0.50
	CR 50	20	5	3:1	0.520	0.30 - 0.50
89	CR 13	18	4	3:1	1.167	0.20 - 0.30
	CR 50	16	6	3:1	0.061	0.70 - 0.90
90	CR 13	20	6	3:1	0.154	0.50 - 0.70
	CR 50	20	6	3:1	0.154	0.50 - 0.70
91	CR 13	15	6	3:1	0.111	0.70 - 0.90
	CR 50	15	6	3:1	0.111	0.70 - 0.90
92	CR 13	16	9	3:1	1.373	0.20 - 0.30
	CR 50	16	9	3:1	1.373	0.20 - 0.30
93	CR 13	18	3	3:1	1.440	0.20 - 0.30
	CR 50	16	5	3:1	0.111	0.50 - 0.90
94	CR 13	18	6	3:1	0.056	0.70 - 0.90
	CR 50	18	6	3:1	0.056	0.70 - 0.90

Appendix H. Country and site of collection of selected accessions from the Iberian wild oat collections, chromosome number and rust reactions to seven crown rust races
(CR 13, CR 25, CR 36, CR 50, CR 56, CR 77 and CR 107)

IB Number	Country	Location	chr. #	CR 13	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
180	Canary	Fuerteventura	28	0	2	4	4	4	4	4
181	"	"	14	3	4	0	4	4	2	4
183	"	"	28	4	0	0	4	04	4	4
197	"	"	14	0	-	4	4	4	-	4
200	"	"	14	0	3	0	4	4	3	4
201	"	"	14	0	-	4	4	4	-	4
218	"	"	14	4	4	4	4	4	2	4
336	"	"	14	4	4	4	4	3	4	4
449	"	Lanzarote	42	4	4	4	-	2	3	4
456	"	"	14	3	-	-	3	4	3	4
468	"	"	14	0	4	-	4	4	4	4
477	"	"	14	-	-	-	4	4	-	4
487	"	"	14	0	-	-	4	4	4	4
488	"	"	14	2	-	-	4	3	3	4
581	"	La Palma Is.	28	4	0	0	2	1	0	2
638	"	"	42	0	4	4	4	2	2	4
641-A	"	"	28	4	4	;1	;	;	1	2
660	"	"	28	3	-	-	3	4	2	4
664	"	"	28	0	4	4	4	4	-	24
665	"	"	42	4	3	4	4	3	2	4
722	"	Gomera	42	4	-	-	4	4	2	4
737	"	"	42	;	0	2	4	4	;	4
738	"	"	28	0	3	4	2	4	2	3
750	"	"	42	0;	-	0	4	4	3	4
812	"	Tenerife	-	4	0	4	4	2	3	23
858	"	"	28	0	0	4	1	2	4	2
861	"	"	28	0	4	4	2	4	1	23
864	"	"	28	3	-	4	4	4	-	3
866	"	"	28	0	1	0	;1	;	3	;
922	"	Gran Canaria	28	4	;	0	1	;2	2	;2
939	"	"	42	2	4	4	4	3	4	4
941	"	"	42	0;	1	4	3	4	0	4

Appendix H.   cont'd

IB Number	Country	Location	Chr. #	CR 13	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
944	Canary	Gran Canaria	42	2	4	;1	4	4	4	4
945	"	"	42	2	1	0	;	;3	;	3
946	"	"	28	4	4	4	4	2	4	-
947	"	"	28	0	0	4	3	;	-	1
948	"	"	42	4	4	0	4	4	0	4
949	"	"	28	3	4	4	2	3	2	2
958	"	"	28	0	4	2	4	2	3	4
960	"	"	42	4	;	4	4	1	3	1
961	"	"	42	3	0	0	4	2	2	-
962	"	"	28	0	4	0	4	;	3	;
965	"	"	28	0	2	0	4	;2	2	;
966	"	"	42	2	4	0	4	3	3	4
967	"	"	28	0	4	0	4	4	2	4
968	"	"	28	4	0	3	3	3	1	4
970	"	"	28	3	;	4	4	4	1	4
979	"	"	28	0	;	0	4	;	4	;
980	"	"	28	0;	0	4	;	;1	0	;
981	"	"	28	3	0	4	3	;	-	2
989	"	"	42	4	;	4	3	2	3	4
990	"	"	42	3	4	0	3	4	3	3
991	"	"	42	4	4	4	4	4	3	4
992	"	"	42	4	4	4	2	0;	3	;
993	"	"	28	4	0	4	4	3	1	3
994	"	"	42	4	0	0;	4	4	4	4
995	"	"	28	0;	4	3	4	3	3	3
996	"	"	42	3	4	0	4	04	3	4
1000-112	"	"	28	4	4	4	4	4	;	4
1000-129	"	"	42	0	4	0	4	4	-	4
1000-144	"	"	42	12	-	4	4	4	-	4
1044	Morocco	Casablanca	14	0	2	0	3	4	0	3
1071	"	Sidi Bennour	42	4	4	;	4	4	3	4
1094	"	Tleta Sidi Boug	42	2	4	4	3	4	2	2
1101	"	Sebt Des Gzoula	28	0	;	0;	4	4	-	4

Appendix H.   cont'd

IB Number	Country	Location	Chr #	CR 13	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
1112	Morocco	Talmest	42	0;	;	0	4	04	;	;
1113	"	"	42	;	0;	;	4	4	;1	;
1128	"	Smimou	14	0	-	4	4	4	-	3
1140	"	Agadir	28	2	4	0	3	4	0	4
1167	"	Tiznit	28	4	4	4	4	4	4	2
1203	"	Tiznit	14	;	4	4	4	4	2	4
1205	"	"	-	4	4	3	;	;	;1	;
1230	"	"	14	;	-	-	4	4	0	4
1242	"	"	14	4	4	4	3	4	-	4
1266	"	Ait Baha	14	;	-	-	4	4	0	4
1275	"	"	14	3	4	4	4	4	4	4
1296	"	Taliouine	14	4	0	-	4	4	0	4
1309	"	Addouz	14	4	3	4	4	4	4	4
1310	"	Taliouine	14	4	-	4	4	4	4	4
1330	"	"	14	3	4	-	3	1	3	4
1350	"	Tazenakht	14	0	4	0	4	4	4	4
1374	"	Agdz	42	4	-	-	4	-	2	4
1461	"	Marrakech	42	0	4	4	4	4	4	4
1473	"	Tamelelt	14	;	-	4	4	4	;	;
2001	Portugal	Lisboa	42	0	3	4	4	4	3	4
2002	"	Elvas	42	4	3	0	4	3	2	4
2003	"	Lisboa	28	2	3	3	3	2	1	3
2008	"	Elvas	42	4	-	-	4	4	4	4
2009	"	"	42	0	4	0;	4	4	3	2
2010	"	"	42	0	4	4	4	3	3	3
2011	"	"	42	2	3	0	4	4	2	;2
2013	"	"	42	4	4	4	4	3	4	3
2028	"	"	42	4	4	4	4	4	3	4
2034	"	"	42	4	2	4	4	4	2	4
2044	"	Alandroal	42	0	-	4	4	4	4	4
2045	"	"	42	0	0	4	4	2	1	2
2061	"	"	42	0	;	4	;2	;	;	;
2100-100	"	Reguengos Mo De	42	3	4	1	;	2	2	4

Appendix H. cont'd

IB Number	Country	Location	Chr. #	CR 13	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
2100-101	Portugal	Reguengos Mo De	42	3	-	-	4	4	4	4
2100-102	"	"	14	2	2	4	4	3	2	3
2100-103	"	"	42	1	3	4	4	4	4	4
2100-104	"	"	42	0	4	0	2	2	0	3
2100-105	"	"	42	0	4	0	3	2	3	2
2100-106	"	"	42	0	4	;	4	4	4	4
2100-107	"	Reguengos Mo De	42	4	4	0	4	4	2	4
2100-108	"	"	14	;	0	0	;1	;	0	2
2100-109	"	"	42	4	-	4	4	4	3	4
2100-111	"	"	42	0	4	0	4	4	3	4
2100-112	"	"	14	0	4	4	;	;	;1	;
2100-113	"	"	14	2	4	;	4	4	4	4
2100-114	"	"	42	4	4	3	;	;	2	;1
2100-115	"	"	14	;	0	0	0	0	;	0
2100-116	"	"	42	3	3	0	4	4	3	4
2100-117	"	"	42	3	4	0;	24	2	4	;
2100-118	"	"	42	3	4	4	4	4	4	4
2100-120	"	"	42	4	3	0	3	3	3	4
2100-121	"	"	42	2	2	0	;	12	0	1
2100-122	"	Elvora	14	2	;	2	3	3	;1	4
2100-128	"	"	14	;	0;	;	;	;	;1	;
2100-132	"	"	42	0	;	4	;	1	0	;2
2100-136	"	Elvas	28	3	2	4	4	4	12	4
2162	"	Santa Eulalia	28	3	-	-	3	4	3	4
2166	"	Elvas	42	4	4	0;	4	2	3	3
2193	"	Vila Velha de R	28	4	3	-	4	4	2	4
2194	"	"	42	0	3	0	4	4	2	4
2200	"	Costelo Bronco	42	4	4	4	4	4	2	4
2203	"	"	42	4	-	4	4	4	-	4
2224	"	"	14	4	-	-	4	-	4	4
2252	"	Ponte de Sor	14	0	0	3	0	1	1	3
2266	"	Sobriera Formos	42	4	-	0	-	4	4	4
2267	"	"	42	4	-	-	4	4	4	4

Appendix H.   cont'd

IB Number	Country	Location	Chr. #	CR 13	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
2272	Portugal	Abrantes	42	1	-	0	4	4	3	4
2275	"	-	42	0	4	4	4	4	-	4
2289	"	Sobreira Formos	14	3	-	0	4	4	3	4
2320	"	Seia	14	4	4	4	4	3	-	4
2331	"	Rio Major	-	0	4	4	4	4	3	4
2342	"	"	42	;	-	-	4	4	2	4
2345	"	Lisboa	14	;	;	0	0	4	4	;
2383	"	Setubal	14	;	;	-	;	;	;	;
2391	"	Senera	42	0;	-	-	4	4	2	3
2394	Portugal	Sines	28	0	0	;1	1	2	0	3
2429-100	"	Loule	42	4	-	0;	4	4	3	4
2429-103	"	Castro Marim	42	4	4	4	4	4	4	4
2429-113	"	Odeleite	42	0	-	-	3	2	3	4
2429-117	"	Tavira	42	4	4	4	4	4	4	3
2437	"	-	-	2	4	0	4	4	2	4
2443	"	Olhao	42	4	-	0;	4	4	2	3
2472	"	Sao Maktinho	42	0	0	0	0	0	0	0
2503	"	Portimao	42	0	4	0	1	;1	2	1
3032	Spain	Carmona	42	-	-	-	3	4	-	3
3034	"	"	42	3	4	0	2	4	3	4
3121	"	Sevilla	42	0	-	0	4	4	-	4
3124	"	"	42	0	4	0	2	3	2	4
3125	"	El Ronquillo	42	4	4	0	4	3	;	4
3134	"	Sevilla	42	0	4	0	4	4	3	4
3193	"	La Albuera	42	4	3	4	;	;	;	;
3219	"	Santa olalla	14	0;	0	3	1	;	;	;
3239	"	Carmona	28	4	-	-	4	4	4	4
3253	"	La carlata	28	0	2	0	0;	;	0	;
3301	"	La peuble Lo De	42	-	-	-	4	4	-	4
3314	"	Constantin	42	;1	4	4	2	4	;	4
3335	"	Venta Nueva	28	4	4	;	;	;	;1	;
3336	"	"	42	;1	4	4	4	4	4	4
3349	"	Espera	42	3	-	4	4	4	-	4

Appendix H. cont'd

IB Number	Country	Location	Chr. #	CR 13	CR 25	CR 36	CR 50	CR 56	CR 77	CR 107
3390	Spain	Arcos De La Fru	28	4	3	0	-	3	3	23
3414	"	Medina Sidonia	42	4	;	0	3	3	3	4
3421	"	Tahivilla	28	2	-	-	4	4	1	3
3442	"	"	42	01	1	4	4	4	2	4
3449	"	Tarifa	42	2	4	4	4	3	;	2
3482	"	Marbella	28	;	0	4	;	1	3	1
3485	"	Coin	28	0	4	4	2	2	4	4
3522	"	Antequera	42	1	4	4	4	4	2	4
3528	"	"	-	0	-	-	4	4	3	3
3530	"	Cartama	42	4	4	4	4	4	4	4
3568	"	Granada	42	3	3	0;	4	4	3	4
3584	"	Alcaudete	28	0	4	;	1	;2	1	;
3618	Spain	Cordoba	42	3	;	0	4	4	4	4
3660	"	Lanjaron	14	;	2	0	4	3	0	4
3691	"	Almeria	42	4	4	0	4	4	1	4
3745	"	Murcia	42	4	4	4	4	4	4	4
3756	"	Orihuela	42	0	2	0	2	34	;1	3
3782	"	Yecla	28	0	4	0	2	34	3	4
3823	"	La Roda	42	2	-	-	4	4	2	3
3842	"	Ocana	42	4	-	4	3	4	4	4